Annex E. Epidemiologic Studies[a]

E.1. Short-Term Exposure and Cardiovascular Outcomes

E.1.1. Panel Studies

Table E-1. Short-term exposure to PM_{10} and cardiovascular morbidity outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Baccarelli et al. (2007a) **Period of Study:** Jan 1995 – Aug 2005 **Location:** Lombardia region, Italy	**Outcome:** Fasting and postmethionine-load total homocysteine (tHcy) **Age Groups:** 11-84 yrs **Study Design:** Cross-sectional / Panel **N:** 1,213 participants **Statistical Analyses:** Generalized additive models **Covariates:** age, sex, BMI, smoking, alcohol, hormone use, temperature, day of the year, and long-term trends **Season:** Adjusted for long-term trends to account for season **Dose-response Investigated?** No **Statistical Package:** R v2.2.1 **Lags Considered:** 1d, 7d moving avg.	**Pollutant:** PM_{10} (some TSP measures used to predict PM_{10}) **Averaging Time:** 24 h **Mean (SD):** NR **Percentiles:** 25th: 20.1; 50th: 34.1; 75th: 52.6 **Max:** 390.0 **Monitoring Stations:** 53 **Copollutant:** CO, NO_2, SO_2, O_3	**PM Increment:** IQR **Percent Change: [Lower CI, Upper CI]:** Homocysteine, fasting: 0.4 (-2.4, 3.3) Homocysteine, postmethionine-load: 1.1 (-1.5, 3.7) **Percent Change: per 25.7m^3 increase in 7-day moving avg of PM_{10}** Homocysteine, fasting: 1.0 (-1.9, 3.9) Homocysteine, postmethionine-load: 2.0 (-0.6, 4.7) **Percent Change:on fasting homocysteine per IQR increase in 24-h PM_{10} levels** Among smokers: 6.2 (0.0, 12.7) Among non-smokers: -1.6 (-5.5, 2.5) **Percent Change:on postmethionine-load homocysteine per IQR increase in 24-h PM_{10} levels** Among smokers: 6.0 (0.5, 11.8) Among non-smokers: -0.1 (-3.6, 3.5)
Reference: Baccarelli et al. (2007a) **Period of Study:** Jan 1995–Aug 2005 **Location:** Lombardia region, Italy	**Outcome:** Fasting and postmethionine-load total homocysteine (tHcy) **Age Groups:** 11-84 yrs **Study Design:** Cross-sectional / Panel **N:** 1,213 participants **Statistical Analyses:** Generalized additive models **Covariates:** age, sex, BMI, smoking, alcohol, hormone use, temperature, day of the year, and long-term trends **Season:** Adjusted for long-term trends to account for season **Dose-response Investigated?** No **Statistical Package:** R software v2.2.1	**Pollutant:** PM_{10} (some TSP measures used to predict PM_{10}) **Averaging Time:** Hourly concentrations used to calculate 24-h moving averages and 7-day moving averages **Mean (SD):** NR **Percentiles:** 25th: 20.1; 50th: 34.1; 75th: 52.6 **Range (Min, Max):** Max: 390.0 **Monitoring Stations:** 53 sites **Copollutant:** CO; NO_2; SO_2; O_3	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** Estimates (%) per 32.5 µg/m^3 increase in 24-h moving avg of PM_{10} Homocysteine, fasting: 0.4 (-2.4, 3.3) Homocysteine, postmethionine-load: (-1.5, 3.7) Estimates (%) per 25.7m^3 increase in 7-day moving avg of PM_{10} Homocysteine, fasting: 1.0 (-1.9, 3.9) Homocysteine, postmethionine-load: 2.0 (-0.6, 4.7) Estimates of effect (%) on fasting homocysteine per IQR increase in 24-h PM_{10} levels Among smokers: 6.2 (0.0, 12.7) Among non-smokers: -1.6 (-5.5, 2.5) Estimates of effect (%) on postmethionine-load homocysteine per IQR increase in 24-h PM_{10} levels Among smokers: 6.0 (0.5, 11.8) Among non-smokers: -0.1 (-3.6, 3.5)

[a] All units expressed in µg/m^3 unless otherwise specified.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Baccarelli et al. (2007b) **Period of Study:** Jan 1995 – Aug 2005 **Location:** Lombardia region, Italy	**Outcome:** Prothrombin time (PT); Activated partial thromboplastin time (APTT); Fibrinogen; Functional antithrombin; Functional protein C; Protein C, antigen; Functional protein S; Free protein S **Age Groups:** 11-84 yrs **Study Design:** Cross-sectional / Panel **N:** 1,218 participants **Statistical Analyses:** Generalized additive models **Covariates:** Age, sex, BMI, smoking, alcohol, hormone use, temperature, day of the year, and long-term trends **Season:** Adjusted for long-term trends to account for season **Dose-response Investigated?** No **Statistical Package:** R software v2.2.1	**Pollutant:** PM_{10} (some TSP measures used to predict PM_{10}) **Averaging Time:** Hourly concentrations used to calculate lags of same day, 7-day, 30-day, and h 0-6 **Mean (SD):** NR **Percentiles:** Sep-Nov: 25th: 33.1; 50th: 51.2 75th: 76.5; Max: 148.9 Dec-Feb: 25th: 47.9; 50th: 68.5 75th: 95.3; Max: 238.3 Mar-May: 25th: 30.0; 50th: 64.1 75th: 64.8; Max: 158.5 Jun-Aug: 25th: 28.0; 50th: 44.3 75th: 61.3; Max: 94.7 **Monitoring Stations:** 53 sites **Copollutant:** CO, NO_2 , SO_2, O_3	**PM Increment:** SD **Effect Estimate [Lower CI, Upper CI]:** Estimated changes in endpoint **PT (international normalized ratio):** At time of blood sample: -0.06 (-0.12, 0.00) Avg levels 7 days prior: -0.03 (-0.10, 0.04) Avg levels 30 days prior: -0.08 (-0.14, -0.01) (Hourly moving Avgs presented in Fig 2) **APTT (ratio to reference plasma):** At time of blood sample: 0.02 (-0.04, 0.08) Avg levels 7 days prior: 0.00 (-0.07, 0.06) Avg levels 30 days prior: 0.01 (-0.06, 0.08) **Fibrinogen:** At time of blood sample: 0.01 (-0.05, 0.07) Avg levels 7 days prior: -0.03 (-0.09, 0.04) Avg levels 30 days prior: -0.02 (-0.09, 0.05) **Functional antithrombin:** At time of blood sample: -0.02 (-0.09, 0.04) Avg levels 7 days prior: -0.06 (-0.13, 0.01) Avg levels 30 days prior: -0.06 (-0.13, 0.02) **Functional protein C:** At time of blood sample: 0.00 (-0.06, 6.1) Avg levels 7 days prior: -0.06 (-0.12, 0.01) Avg levels 30 days prior: -0.06 (-0.14, 0.01) **Protein C, antigen:** At time of blood sample: 0.00 (-0.06, 6.0) Avg levels 7 days prior: -0.04 (-0.10, 0.03) Avg levels 30 days prior: -0.06 (-0.14, 0.01) **Functional protein S:** At time of blood sample: 0.04 (-0.03, 0.10) Avg levels 7 days prior: -0.03 (-0.11, 0.06) Avg levels 30 days prior: -0.14 (-0.23, -0.05) **Free protein S:** At time of blood sample: 0.05 (-0.01, 0.10) Avg levels 7 days prior: 0.01 (-0.05, 0.07) Avg levels 30 days prior: -0.01 (-0.08, 0.06)
Reference: Choi et al (2007) **Period of Study:** 2001-2003 **Location:** Incheon, South Korea	**Outcome:** Blood pressure **Study Design:** Cross-sectional **N:** 10459 subjects with a hospital health examination **Statistical Analyses:** Linear regression **Covariates: Season:** Effect modification by season	**Pollutant:** PM_{10} **Averaging Time:** Measured hourly and calculated 24-h means **Percentiles:** Warm season: Median: 36.7 Cold season: Median: 45.7 **Monitoring Stations:** 9 stations **Copollutant:** NO_2, SO_2	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Estimate (p-value) for the relationship between systolic blood pressure (SBP) and diastolic blood pressure (DBP) and an increase in PM_{10} on lag day 1 SBP: Warm season: 0.0798 (p<0.001) DBP: Warm season: 0.0240 (p<0.001) **Note:** No evidence of associations between PM_{10} and BP during the cold season
Reference: Chuang et al. (2007a) **Period of Study:** Between Apr-Jun 2004 or 2005 **Location:** Taipei, Taiwan	**Outcome:** High-sensitivity C-reactive protein (hs-CRP); Fibrinogen, plasminogen activator fibrinogen inhibitor-1 (PAI-1), tissue-type plasminogen activator (tPA), 8-hydroxy-2'-deoxyguanosine (8-OHdG), and log-transformed HRV indices (SDNN = standard deviation of NN intervals, r-MSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, LF = low frequency [0.04-0.15Hz], and HF = high frequency [0.15-0.40Hz]) **Age Groups:** 18-25 yrs **Study Design:** Panel (cross-sectional) **N:** 76 students **Statistical Analyses:** linear mixed-effects models **Covariates:** Age, sex, BMI, weekday, temperature of previous day, relative humidity **Season:** Only 1 season of data collection **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Hourly data used to calculate averages over 1-3 day periods **Mean (SD):** 1-day avg: 49.2 (18.0) 2-day avg: 55.3 (18.6) 3-day avg: 54.9 (18.2) **Range (Min, Max):** 1-day avg: 29.5, 83.4 2-day avg: 25.5, 85.1 3-day avg: 22.2, 87.2 **Monitoring Stations:** 2 sites (each pollutant measured at one site only) **Copollutant:** $PM_{2.5}$, Sulfate, Nitrate, OC, EC, NO_2, CO, SO_2, O_3	**PM Increment:** IQR (1-d avg: 32.7; 2-day avg: 34.5; 3-day avg: 26.0) **Effect Estimate [Lower CI, Upper CI]:** % change in health endpoint per increase in IQR of PM_{10} (1-3 day averaging period; single pollutant models) **hs-CRP:** 1-d: 135.8 (1.8, 269.7); 2-d: 108.2 (-10.9, 227.3); 3-d: 109.6 (2.5, 216.7) **8-OHdG:** 1-d: -9.2 (-21.5, 3.2); 2-d: -6.1 (-17.0, 4.8); 3-d: -5.6 (-13.8, 2.6) **PAI-1:** 1-d: 30.0 (12.4, 47.7); 2-d: 19.1 (3.6, 34.7); 3-d: 21.2 (9.7, 32.8) **tPA:** 1-d: 16.0 (-4.1, 36.2); 2-d: 10.4 (-6.3, 27.2); 3-d: 8.8 (-2.8, 20.5) **Fibrinogen:** 1-d: 5.3 (1.5, 15.2); 2-d: 1.5 (-4.4, 7.5); 3-d: 3.3 (-1.1, 7.7) **Heart Rate Variability** **SDNN:** 1-d: -4.9 (-7.8, -2.1); 2-d: -4.0 (-6.6, -1.4); 3-d: -4.1 (-6.1, -2.2) **r-MSSD:** 1-d: -4.8 (-12.3, 2.7); 2-d: -2.2 (-9.0, 4.7); 3-d: -4.0 (-9.0, 0.9) **LF:** 1-d: -6.1 (-10.1, -2.1); 2-d: -3.0 (-7.2, 1.2); 3-d: -4.3 (-7.0, -1.6) **HF:** 1-d: -5.5 (-13.0, 2.1); 2-d: -2.7 (-9.5, 4.1); 3-d: -2.0 (-7.2, 3.2)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Liao et al. (2004) **Period of Study:** 1996-1998 **Location:** ARIC study cohort (Washington County, MD; Forsyth County, NC; and selected suburbs of Minneapolis, MN). The 4th quarter of the ARIC cohort was sampled exclusively from black residents of Jackson, MS.	**Outcome:** 5-min HR, HRV indices (HF, LF, SDNN) **Study Design:** Cross-sectional **Statistical Analyses:** Linear regression	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 24.3 (11.5) **Copollutant:** O_3; CO; SO_2; NO_2	**PM Increment:** SD **Effect Estimate [Lower CI, Upper CI]:** Estimate (SE) HF: -0.06 ms^2 (0.018) SDNN: -1.03 ms (0.31) H: 0.32 beats/min (0.158)
Reference: Liao et al. (2005) **Period of Study:** 1987-1989 baseline health exam **Location:** 3 centers in the US (Forsyth County, NC; suburbs of Minneapolis, MN; black residents of Jackson, MS)	**Outcome:** Fibrinogen, factor VIII coagulant activity (VIII-C), von Willebrand factor (vWF), white blood cell count (WBC), and serum albumin **Age Groups:** 45-64 yrs **Study Design:** Cross-sectional **N:** 10,208 participants (7705 for PM) **Statistical Analyses:** Multiple linear regression **Covariates:** Age, sex, ethnicity-center, education, smoking, drinking status, BMI, history of chronic respiratory disease, humidity, season, cloud cover, and temperature **Dose-response Investigated?** Yes, examined higher-ordered terms for each pollutant **Statistical Package:** SAS v8.2	**Pollutant:** PM_{10} **Averaging Time:** 24-h averages (1, 2, and 3 days prior to the exam) **Mean (SD):** 29.9 (29.9) **Mean (SD) within Quartiles:** Q1-3: 24.0 (6.96) Q4: 47.3 (10.11) **Copollutant:** CO, SO_2, NO_2, O_3	**PM Increment:** 1 SD (12.8 $\mu g/m^3$) **Effect Estimate:** Adjusted regression coefficient (SE): Fibrinogen (mg/dl): 0.163 (0.755) Factor VIII-C (%): Non-linear association: β (PM_{10}) = -5.30, $p<0.01$ β $(PM_{10})^2$ = 0.80, $p<0.05$ vWF (%): Diabetics: 3.93 (1.80) Nondiabetics: -0.54 (0.58) Albumin (g/dl): CVD: -0.006 (0.003) Non-CVD: 0.029 (0.017) $p<0.05$

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Liu et al. (2007) **Period of Study:** May 24, 2005–Jul 8, 2005 **Location:** Windsor, Ontario, Canada	**Outcome:** Heart rate, blood pressure, brachial arterial diameter, flow-mediated vasodilation (FMD), plasma cytokines, and thiobarbituric acid reactive substances (TBARS) **Age Groups:** 18-65 yrs **Study Design:** Panel **N:** 24 nonsmoking subjects with type I or II diabetes over a 7 week period (2-14 visits for subjects); 170 total vascular measurements and 134 total blood samples collected **Statistical Analyses:** Mixed effects regression models **Covariates:** (time-dependent covariates) Daily temperature, relative humidity, blood glucose level, also checked for confounding by ambient air pollutant concentrations (controlled for ambient $PM_{2.5}$) **Season:** No adjustment since testing was completed within a 7 week period during early summer **Dose-response Investigated?** No **Statistical Package:** S-Plus	**Pollutant:** PM_{10} (personal) **Averaging Time:** Real-time monitor measured exposure during 24-h period prior to clinic measures **Median (5th-95th percentile):** 0-24 hrs: 25.5 (9.8-133.0) 0-6hrs: 15.3 (5.3-83.2) 7-12hrs: 17.0 (7.1-186.3) 13-18hrs: 28.5 (11.4-167.0) 19-24 hrs: 30.5 (10.1-148.2) **Monitoring Stations:** Personal monitoring **Copollutant (correlation):** Ambient $PM_{2.5}$ (r = 0.34)	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** $^{**}p < 0.05$; $^{*}p < 0.10$. Regression coefficients (SE) **End-diastolic basal diameter (µm):** All subjects (n=24): -2.52 (3.27); subjects not taking vasoactive meds (n=17): -3.93 (3.66); subjects w/BMI ≤ 29kg/m^2 (n=14): 8.85 (5.85) **End-systolic basal diameter (µm):** All subjects (n=24): -9.02 (3.58)**; subjects not taking vasoactive meds (n=17): -10.59 (4.36)**; subjects w/BMI ≤29kg/m^2 (n=14): 3.85 (5.49) **End-diastolic FMD (%):** All subjects (n=24): 0.20 (0.08)**; subjects not taking vasoactive meds (n=17): 0.23 (0.09)**; subjects w/BMI ≤29kg/m^2 (n=14): 0.12 (0.05)** **End-systolic FMD (%):** All subjects (n=24): 0.38 (0.18)**; subjects not taking vasoactive meds (n=17): 0.51 (0.22)**; subjects w/BMI ≤ 29kg/m^2 (n=14): 0.18 (0.10)* **Flow (cm/s):** All subjects (n=24): -0.16 (0.19); subjects not taking vasoactive meds (n=17): -0.48 (0.21)**; subjects w/BMI ≤ 29kg/m^2 (n=14): -0.39 (0.23)* **Heart rate (bpm):** All subjects (n=24): 0.01 (0.11); subjects not taking vasoactive meds (n=17): -0.06 (0.12); subjects w/BMI ≤ 29kg/m^2 (n=14): 0.15 (0.12) **Diastolic blood pressure (mm Hg):** All subjects (n=24): 0.19 (0.16); subjects not taking vasoactive meds (n=17): 0.40 (0.18)**; subjects w/BMI ≤ 29kg/m^2 (n=14): 0.27 (0.21) **Systolic blood pressure (mm Hg):** All subjects (n=24): 0.17 (0.19); subjects not taking vasoactive meds (n=17): 0.43 (0.24)*; subjects w/ BMI ≤ 29kg/m^2 (n=14): 0.38 (0.24) **CRP (µg/mL):** All subjects (n=24): 0.11 (0.07); subjects not taking vasoactive meds (n=17): 0.10 (0.09); subjects w/ BMI ≤ 29kg/m^2 (n=14): 0.02 (0.03) **ET-1 (pg/mL):** All subjects (n=24): 0.00 (0.00); subjects not taking vasoactive meds (n=17): 0.00 (0.00); subjects w/BMI ≤ 29kg/m^2 (n=14): 0.00 (0.01) **IL-6 (pg/mL):** All subjects (n=24): 0.00 (0.05); subjects not taking vasoactive meds (n=17): 0.01 (0.05); subjects w/BMI ≤ 29kg/m^2 (n=14): -0.00 (0.03) **TNF-α (pg/mL):** All subjects (n=24): 0.03 (0.05); subjects not taking vasoactive meds (n=17): 0.02 (0.05); subjects w/ BMI ≤ 29kg/m^2 (n=14): 0.03 (0.08) **TBARS (pmol/mL)** All subjects (n=24): 16.12 (4.00)**; subjects not taking vasoactive meds (n=17): 8.10 (9.18); subjects w/ BMI ≤ 29kg/m^2 (n=14): -0.28 (6.60)**;** regression coefficients (SE) among subjects not taking vasoactive medications, with lag time **End-diastolic basal diameter (µm):** 0-6 h: 29.91 (10.64)****;** 7-12 h: 0.72 (3.95)**;** 13-18 h: -3.62 (2.80)**;** 19-24 h: -0.57 (1.7) **End-systolic basal diameter (µm):** 0-6 h: 28.88 (11.22)****;** 7-12 h: -0.78 (4.58)**;** 13-18 h: -7.70 (3.30)****;** 19-24 h: -2.87 (2.05) **End-diastolic FMD (%):** 0-6 h: -0.12 (0.10)**;** 7-12 h: 0.04 (0.05)**;** 13-18 h: 0.11 (0.03)****;** 19-24 h: 0.12 (0.04)** **End-systolic FMD (%):** 0-6 h: 0.36 (0.08)****;** 7-12 h: 0.48 (0.32)**;** 13-18 h: 0.19 (0.06)****;** 19-24 h: 0.34 (0.13)** **Flow (cm/s):** 0-6 h: -0.34 (0.22)**;** 7-12 h: -0.26 (0.27; 13-18 h: -0.27 (0.15)***;** 19-24 h: -0.30 (0.11)** **Heart rate (bpm):** 0-6 h: 0.31 (0.13)****;** 7-12 h: 0.26 (0.12)****;** 13-18 h: 0.01 (0.09)**;** 19-24 h: -0.08 (0.05) **Diastolic blood pressure (mm Hg):** 0-6 h: -0.29 (0.12)****;** 7-12 h: 0.24 (0.12)****;** 13-18 h: 0.46 (0.17)****;** 19-24 h: 0.18 (0.14) **Systolic blood pressure (mm Hg):** 0-6 h: -0.65 (0.18)**; 7-12 h: 0.17 (0.19)**;** 13-18 h: 0.86 (0.24)****;** 19-24 h: 0.11 (0.10) **CRP (µg/mL):** 0-6 h: 0.15 (0.13)**;** 7-12 h: 0.15 (0.13)**;** 13-18 h: 0.03 (0.06)**;** 19-24 h: 0.04 (0.03) **ET-1 (pg/mL):** 0-6 h: 0.02 (0.00)**: 7-12 h: -0.00 (0.00)**;** 13-18 h: -0.00 (0.00)**;** 19-24 h: 0.00 (0.00) **IL-6 (pg/mL):** 0-6 h: 0.03 (0.06)**;** 7-12 h: 0.00 (0.06)**;** 13-18 h: 0.02 (0.03)**;** 19-24 h: 0.00 (0.02) **TNF-α (pg/mL):** 0-6 h: 0.01 (0.07)**;** 7-12 h: 0.09 (0.04)****;** 13-18 h: 0.01 (0.04)**;** 19-24 h: -0.00 (0.03) **TBARS (pmol/mL):** 0-6 h: -4.44 (6.72)**;** 7-12 h: 11.94 (5.08)****;** 13-18 h: 5.06 (4.03)**;** 19-24 h: 1.06 (4.64) **Note:** Adding ambient $PM_{2.5}$ data as a covariate in the model yielded similar regression coefficients for personal PM_{10}

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lipsett et al. (2006) **Period of Study:** February–May 2000 **Location:** Coachella Valley, CA	**Outcome:** HRV parameters: SDNN, SDANN, r-MSSD, LF, HF, total power, triangular index (TRII). **Study Design:** Panel study **N:** 19 non-smoking adults with coronary artery disease **Statistical Analysis:** Mixed linear regression models with random effects parameters	**Pollutant:** PM_{10} **Averaging Time:** 2 h **Mean (range):** Indio: 23.2 (6.3-90.4); Palm Springs: 14 (4.7-52) **Monitoring Stations:** 2 **Copollutant:** O_3	**PM Increment:** SE*1000 **Effect Estimate (change in HRV per unit increase in PM concentration):** SDNN: -0.71 msec (SE = 0.268) **Notes:** Weekly ambulatory 24 h ECG recordings (once per week for up to 12 weeks), using Holter monitors, were made. Subjects' residences were withinn 5 miles of one of two PM monitoring sites. Regressed HRV parameters against 18: 00–20: 00 mean particulate pollution.
Reference: Mar et al. (2005b) **Period of Study:** 1999–2001 **Location:** Seattle, WA	**Outcome:** Change in arterial O_2 saturation, heart rate, and blood pressure (SBP and DBP) **Age Groups:** >75 years **Study Design:** Panel study **N:** 88 elderly subjects **Statistical Analysis:** GEE	**Pollutant:** PM_{10} **Averaging Time:** 24-hs **Mean (SD):** Indoor: 12.6 (7.8) Outdoor: 14.5 (7.0)	**PM Increment:** 10 µg/m^3 **Unit change in measure(95% CI): Among all subjects:** Each increase in outdoor same day PM_{10} was associated with: SBP: -0.10 mmHg (95% CI: -1.37, 1.18) DBP: -0.03 mmHg (95% CI: -0.79, 0.73) HR: -0.48 beats/min (95% CI: -1.03, 0.06) **Each increase in indoor same day** $PM_{2.5}$ **was associated with:** SBP: 0.92 mmHg (95% CI: -0.95, 2.78) DBP: 0.63 mmHg (95% CI: -0.29, 1.56) HR: 0.02 beats/min (95% CI: -0.54, 0.58) **Notes:** Results by health status presented in Fig 1. Used 2 sessions that each were 10 consecutive days of measurement. Used personal, indoor, and outdoor measures of $PM_{2.5}$
Reference: Metzger et al. (2007) **Period of Study:** January 1993–December 2002 **Location:** Atlanta, GA	**Outcome:** Days with any event recorded by the ICD, days with ICD shocks/defibrillation and days with either cardiac pacing or defibrillation **Study Design:** Repeated measures **N:** 884 subjects **Statistical Analysis:** Logistic regression with GEE to account for residual autocorrelation within subjects	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 28.0 (12.2) **Median:** 26.4 **Copollutant:** O_3, NO_2, CO, SO_2. Aug1998-Dec2002: Oxygenated hydrocarbons	**PM Increment: OR (95% CI):** Outcome = Any event recorded by ICD OR = 1.00 (95% CI: 0.97, 1.03)
Reference: Ruckerl et al. (2006) **Period of Study:** May 2003–Jul 2004 **Location:** Athens, Augsburg, Barcelona, Helsinki, Rome, and Stockholm	**Outcome:** Interleukin-6 (IL-6), fibrinogen, C-reactive protein (CRP) **Age Groups:** 35-80 yrs **Study Design:** Repeated measures / longitudinal **N:** 1003 MI survivors **Statistical Analyses:** Mixed-effect models **Covariates:** City-specific confounders (age, sex, BMI); long-term time trend and apparent temperature; RH, time of day, day of week included if adjustment improved model fit **Season:** Long-term time trend **Dose-response Investigated?** Used p-splines to allow for nonparametric exposure-response functions **Statistical Package:** SAS v9.1	**Pollutant:** PM_{10} **Averaging Time:** Hourly and 24-h (lag 0-4, mean of lags 0-4, mean of lags 0-1, mean of lags2-3, means of lags 0-3) **Mean (SD):** Presented by city only **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** Central monitoring sites in each city **Copollutant:** SO_2; O_3; NO; NO_2	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change in mean blood markers per increase in IQR increase of air pollutant. **IL-6:** Lag (IQR): % change in GM (95%CI); Lag 0 (17.4): -0.34 (-1.66, 0.99); Lag 1 (17.4): -0.69 (-1.95, 0.58); Lag 2 (17.4): -1.59 (-3.99, 0.88); 5-d avg (13.5): -0.87 (-2.28, 0.55) **Fibrinogen:** Lag (IQR): % change in AM (95%CI); Lag 0 (17.4): 0.06 (-0.43, 0.55); Lag 1 (17.4): 0.14 (-0.35, 0.63); Lag 2 (17.4): 0.24 (-0.24, 0.72); 5-d avg (13.5): 0.60 (0.10, 1.09) **CRP:** Lag (IQR): % change in GM (95%CI); Lag 0 (17.4): -0.71 (-2.75, 1.37); Lag 1 (17.4): -0.63 (-2.61, 1.39); Lag 2 (17.4): -1.42 (-4.23, 1.47); 5-d avg (13.5): -1.35 (-3.45, 0.79)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ruckerl et al. (2006) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** C-reactive protein (CRP); serum amyloid A (SAA); E-selectin; vWF; intercellular adhesion molecule-1 (ICAM-1); fibrinogen; Factor VII; prothrombin fragment 1+2; D-dimer **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear and logistic regression models **Covariates:** Models adjusted for different factors based on health endpoint; CRP: RH, temperature, trend, ID; ICAM-1: temperature, trend, ID; vWF: air pressure, RH, temperature, trend, ID; FVII: air pressure, RH, temperature, trend, ID, weekday **Season:** Time trend as covariate **Dose-response Investigated?** Sensitivity analyses examined nonlinear exposure-response functions **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 20.0 (13.0) **Percentiles:** 25th: 10.8 50th: 15.6 75th: 26.0 **Range (Min, Max):** 5.4, 74.5 **Monitoring Stations:** 1 site **Copollutant:** UFPs (ultrafine particles) AP (accumulation mode particles) $PM_{2.5}$ PM_{10} OC (organic carbon) EC (elemental carbon) NO_2 CO	**PM Increment:** IQR (15.2; 5-d avg: 12.8) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as OR (95%CI) for an increase in the blood marker above the 90th percentile per increase in IQR air pollutant. **CRP:** Time before draw: 0 to 23 h: 1.2 (0.8, 1.9); 24 to 47 h: 2.0 (1.1, 3.6); 48 to 71 h: 2.2 (1.2, 3.8); 5-d mean: 2.0 (1.2, 3.7) **ICAM-1:** Time before draw: 0 to 23 h: 1.3 (0.9, 1.8); 24 to 47 h: 3.1 (2.0, 4.8); 48 to 71 h: 3.4 (2.2, 5.2); 5-d mean: 3.4 (2.2, 5.3) Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **vWF:** Time before draw: 0 to 23 h: 4.0 (-0.6, 8.5); 24 to 47 h: 6.0 (0.6, 11.5); 48 to 71 h: 1.1 (-4.9, 7.0); 5-d mean: 6.1 (-0.6, 12.8) **FVII:** Time before draw: 0 to 23 h: -6.6 (-10.4 to -2.5); 24 to 47 h: -8.4 (-12.3 to -4.3); 48 to 71 h: -5.9 (-9.6, -2.0); 5-d mean: -8.0 (-12.4, -3.4) **Note:** summary of results presented in figures. SAA results indicate increases in association with PM (not as strong and consistent as with CRP); no association observered between E-selectin and PM; an increase in prothrombin fragment 1+2 was consistently observed, particularly with lag 4; fibrinogen results revealed few significant associations, potentially due to chance; D-dimer results revealed null associations in linear and logistic analyses
Reference: Ruckerl et al. (2007a) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** Soluble CD40 ligand (sCD40L), platelets, leukocytes, erythrocytes, hemoglobin **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear regression models **Covariates:** Long-term time trend, weekday of the visit, temperature, RH, barometric pressure **Season:** Time trend as covariate **Dose-response Investigated?** No **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 20.0 (13.0) **Percentiles:** 25: 10.8 50: 15.6 75: 26.0 **Range (Min, Max):** 5.4, 74.5 **Monitoring Stations:** 1 site **Copollutant:** UFPs (ultrafine particles), AP (accumulation mode particles), $PM_{2.5}$, PM_{10}, NO	**PM Increment:** IQR (15.2; 5-d avg: 12.8) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **sCD40L, % change GM (pg/mL):** lag0: 1.6 (-3.5, 7.0); lag1: 1.1 (-5.4, 7.9); lag2: -3.5 (-8.9, 2.2); lag3: -1.4 (-6.0, 3.4); 5-d mean: -1.2 (-7.8, 5.8) **Platelets, % change mean (10^3/µl):** lag0: -0.4 (-1.9, 1.0); lag1: 0.4 (-1.4, 2.3); lag2: 0.5 (-1.4, 2.3); lag3: -0.1 (-1.6, 1.4); 5-d mean: 0.0 (2.1, 0.0) **Leukocytes, % change in mean (10^3/µl):** lag0: -1.1 (-2.8, 0.7); lag1: -0.5 (-2.6, 1.5); lag2: 0.1 (-2.1, 2.4); lag3: -0.7 (-2.6, 1.2); 5-d mean: -1.1 (-3.6, 1.4) **Erythrocytes, % change mean (10^6/µl):** lag0: 0.0 (-0.4, 0.5); lag1: -0.4 (-1.0, 0.1); lag2: -0.7 (-1.2, -0.2); lag3: -0.4 (-0.8, 0.0); 5-d mean: -0.6 (-1.2, -0.1) **Hemoglobin, % change mean (g/dl):** lag0: -0.1 (-0.7, 0.6); lag1: -0.4 (-1.2, 0.3); lag2: -0.7 (-1.3, 0.0); lag3: -0.3 (-0.9, 0.2); 5-d mean: -0.7 (-1.5, 0.1)
Reference: Su et al. 2003 **Period of Study:** Feb–Apr 2002 **Location:** Taipei, Taiwan	**Outcome:** Total Cholesterol, HDL, tryglycerides, LDL, C-reactive protein (hs-CRP), interleukin-g (IL-6), tumor necrotic factor- α (TNF-α), plasma tissue-type plasminogen activator (tPA), plasminogen activator inhibitor-1 (PAI-1), fibrinogen **Age Groups:** 40-75 yrs **Study Design:** Panel (subjects provided blood samples/health endpoints during a low and a high pollution day) **N:** 49 subjects with coronary heart disease or multiple CHD risk factors **Statistical Analyses:** Paired t-test used for primary analysis; also performed linear mixed-effects models to assess confounding **Covariates:** Sex, age, temperature, humidity **Season:** Only 1 season **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} (High pollution day >100 µg/m^3) **Averaging Time:** Daily **Mean (SD):** Low pollution day: High pollution day: **Monitoring Stations:** 1 monitor **Copollutant:** PM_{10}; $PM_{2.5}$; Ozone; OC; EC; Nitrate; Sulfate	**PM Increment:** High vs. Low pollution days **Effect Estimate [Lower CI, Upper CI]: CHD patients (n = 23):** P-value for paired t-test comparing health endpoint means on high and low pollution days hs-CRP: p = 0.568; IL-6: p = 0.856 TNF-α: p = 0.246 PAI-1: p = 0.008 tPA: p = 0.322 Fibrinogen: p = 0.189 P-value for health endpoint in mixed-effects models PAI-1: p = 0.010 tPA: p = 0.329 Fibrinogen: p = 0.747 **Patients with multiple CHD risk factors (n = 26):** P-value for paired t-test comparing health endpoint means on high and low pollution days hs-CRP: p = 0.475 IL-6: p = 0.561; TNF-α: p = 0.572; PAI-1: p = 0.098; tPA: p = 0.260 Fibrinogen: p = 0.087; P-value for health endpoint in mixed-effects models; PAI-1: p = 0.891; tPA: p = 0.789 Fibrinogen: p = 0.923

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Su et al. (2006) **Period of Study:** February–April 2002 **Location:** Taipei, Taiwan	**Outcome:** Total cholesterol, HDL, tryglycerides, LDL, hs-CRP, IL-6, TNF-α, tPA, PAI-1, and fibrinogen **Age Groups:** 40-75 years **Study Design:** Panel study **N:** 49 subjects (31 males and 18 females) with coronary heart disease or multiple risk factors for CHD **Statistical Analysis:** Linear mixed effects regression	**Pollutant:** PM_{10} **Averaging Time:** 1 h (High pollution day = PM_{10} from 08: 00 to 18: 00 >100) **Copollutant:** O_3	**Effect Estimate:** On high air pollution days, PAI-1 levels (63.9 ng/mL[SD = 29.0]) were significantly higher than on low pollution days (51.1[27.1]). There were not clear differences for any of the other markers, although tPA and fibrinogen each had higher mean levels on high air pollution days than on low air pollution days. **Notes:** Subjects had paired fasting blood samples taken during high and low air pollution days.
Reference: Vedal et al., (2004) **Period of Study:** 1997-2000 **Location:** Vancouver, British Columbia	**Outcome:** Implantable cardioverter defibrillator (ICD) discharge **Age Groups:** All **Study Design:** Time series (Retrospective, longitudinal panel study) **N:** 50 ICD patients with 1+ discharges (40,328 person-days and 257 arrhythmia event days) **Statistical Analyses:** Multiple logistic regression with GEE **Covariates:** Temperature, relative humidity, barometric pressure, rainfall, wind direction and speed **Season:** Summer (May-Sep) and winter (Oct-Apr) **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** -3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 12.9 (3.8-49.3); SD = 5.6 **Monitoring Stations:** 8 **Copollutant (correlation):** O_3: r = 0.11 SO_2: r = 0.70 NO_2: r = 0.49 CO: r = 0.43 **Other variables:** Temp: r = 0.43 Humidity: r = -0.35 Baro Pressure: r = 0.26 Rain: r = -0.63 Wind: r = -0.53	**PM Increment:** 5.6 μg/m^3 (SD) **Percent Change [CI]:** Values NR **Notes:** The author states that significant negative associations were found for ICD discharge with same-day lag, and also for 3-day lag with more arrhythmia-prone patients. All other non-significant percent change estimates are shown in Fig 3 and 4.
Reference: Vedal et al. (2004) **Period of Study:** 1997–2000 **Location:** Vancouver, British Columbia, Canada	**Outcome:** ICD discharges (arrhythmias) **N:** 150 patients w/ICD, 4 yrs **Statistical Analysis:** Logistic regression, GEE **Covariates:** Temporal trends, temperature, relative humidity, wind speed, rain **Season:** Summer, Winter **Dose-response Investigated?** No **Lags Considered:** 0.1.2.3d	**Pollutant:** PM_{10} **Mean:** 12.9 (SD = 5.6) **Copollutant):** O_3, SO_2, NO_2, CO	**Increment:** 1 SD Effect Estimates, e.g., % change in the rate of arrhythmia, were presented in Figure 3. No association with PM_{10} was observed while SO_2 was associated with an increase in the rate of arrhythmia among 16 patients with at least 2 discharges per year.

Table E-2. Short-term exposure $PM_{10-2.5}$ and cardiovascular morbidity outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lipsett et al. (2006) **Period of Study:** February–May 2000 **Location:** Coachella Valley, CA	**Outcome:** HRV parameters, specifically SDNN, SDANN, r-MSSD, LF, HF, total power, triangular index (TRII). **Study Design:** Panel study **N:** 19 non-smoking adults with coronary artery disease **Statistical Analysis:** Mixed linear regression models with random effects parameters	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 2 h **Monitoring Stations:** 2 **Copollutant:** O_3	**PM Increment:** SE*1000 **Effect Estimate (change in HRV per unit increase in PM concentration):** SDNN: -0.72 msec (SE = 0.296) **Notes:** $PM_{10-2.5}$ calculated by subtracting $PM_{2.5}$ concentration from PM_{10} concentration. Weekly ambulatory 24 h ECG recordings (once per week for up to 12 weeks), using Holter monitors, were made.Subjects' residences were withinn 5 miles of one of two PM monitoring sites. Regressed HRV parameters against 18: 00–20: 00 mean particulate pollution

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Metzger et al. (2007) **Period of Study:** August 1998–December 2002 **Location:** Atlanta, GA	**Outcome:** Days with any event recorded by the ICD, days with ICD shocks/defibrillation and days with either cardiac pacing or defibrillation **Study Design:** Repeated measures **N:** 884 subjects between 1993 and 2002 **Statistical Analysis:** Logistic regression with GEE to account for residual autocorrelation within subjects	**Pollutant:** $PM_{10-2.5}$ (ng/cm^3) **Averaging Time:** 24-hs **Mean (SD):** 9.6 (5.4) **Median:** 8.7 **Copollutant:** O_3, NO_2, CO, SO_2, oxygenated hydrocarbons	**PM Increment: OR (95% CI):** OR = 1.03 (95% CI: 1.00, 1.07)
Reference: Pekkanen et al. (2002) **Period of Study:** Winter 1998 to 1999 **Location:** Helsinki, Finland	**Outcome**: ST Segment Depression (>0.1mV) **Study Design**: Panel of ULTRA Study participants **N**: 45 subjects, 342 biweekly submaximal exercise tests, 72 exercise induced ST Segment Depressions **Statistical Analysis:** Logistic regression / GAM	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Median**: 4.8 **IQR**: 5.5 **Monitoring Stations**: 1 **Copollutant:** NO_2, CO, $PM_{2.5}$, PM1, ACP, ultrafine	**PM Increment:** IQR **Effect Estimate(s):** $PM_{10-2.5}$: OR = 1.99 (0.70, 5.67), lag 2 **Notes**: The effect was strongest for ACP and $PM_{2.5}$, which in two pollutant models appeared independent. Increases in NO_2 and CO were also associated with increased risk of ST segment depression, but not with coarse particles.
Reference: Timonen et al. (2006) **Period of Study:** 1998–1999 **Location:** Amsterdam, Netherlands; Erfurt, Germany; Helskinki, Finland	**Outcome:** HRV measurements: [LF, HF, LFHFR, NN interval, SDNN, r-MSSD] **Study Design:** Panel study **N:** 131 elderly subjects with stable coronary heart disease **Statistical Analysis:** Linear mixed models	**Pollutant:** $PM_{10-2.5}$ **Means:** Amsterdam: 15.3 Erfurt: 3.7 Helsinki: 6.7 **Copollutant:** NO_2, CO	**PM Increment:** 10 $\mu g/m^3$ **Effect Estimate:** SDNN; 0.69ms (95% CI: -1.24, 2.63) HF: 2.9% (95% CI: -7.3, 13.1) LFHFR: -3.3 (95% CI: -12.7, 6.1) **Notes:** Followed for 6 months with biweekly clinic visits 2 day lag. ULTRA Study

Table E-3. Short-term exposure to $PM_{2.5}$ (including PM components/sources) and cardiovascular morbidity outcomes.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Adar et al. (2007) **Period of Study:** Mar–Jun 2002 **Location:** St. Louis, Missouri	**Outcome:** Heart rate variability: heart rate, standard deviation of all normal-to-normal intervals (SDNN), square root of the mean squared difference between adjacent normal-to-normal intervals (rMSSD), percentage of adjacent normal-to-normal intervals that differed by more thean 50 ms (pNN50), high frequency power (HF; in the range of 0.15-0.4Hz), low frequency power (LF, in the range of 0.04-0.15Hz), and the ratio of LF/HF **Age Groups:** ≥ 60 yrs **Study Design:** Panel (4 planned repeated measures surrounding bus trips with a total of 158 person-trips; 35 participating in all 4 trips) **N:** 44 participants **Statistical Analyses:** Generalized additive models **Covariates:** Subject, weekday, time, apparent temperature, trip type, activity, medications, and autoregressive terms **Season:** Limited data collection period **Dose-response Investigated?** No **Statistical Package:** SAS v8.02, R v2.0.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Measurements collected over 48 h period surrounding the bus trip (during which health endpoints were measured) used to calculate 5-, 30-, 60-minute, 4-h, 24-h moving averages **Median (IQR):** All: 7.7 (6.8) Facility: 6.8 (5.1) Bus: 17.2 (10.3) Activity: 8.2 (16.1) Lunch: 11.2 (5.9) **Monitoring Stations:** 2 portable carts **Copollutant:** $PM_{2.5}$; BC; Fine particle counts; coarse particle counts **Correlation notes:** 24-h mean $PM_{2.5}$, BC, and fine particle count concentrations ranged from 0.80 to 0.98; r = 0.76 to 0.97 when limited to time spent on the bus; r = 0.55 to 0.86 when comparing bus concentrations to 24-h moving averages; r = -0.003 to 0.51 when comparing 5-min averages and 24-h moving averages Poor correlations found between coarse particle count concentrations and all fine particulate measures during all times periods	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change (95%CI) in HRV per IQR in the 24-h moving avg of the microenvironmental pollutant (IQr = 4.5 μg/m³) **Single-pollutant models:** SDNN: -5.5 (-6.3, -4.8); rMSSD: -9.1 (-9.8, -8.4); pNN50 + 1: -12.2 (-13.3, -11.1) LF: -10.8 (-12.3, -9.3); HF: -15.1 (-16.7, -13.7); LF/HF: 5.1 (3.9, 6.4); H: 1.0 (0.9, 1.2) **Two-pollutant models (with particle number count coarse):** SDNN: -5.7 (-6.5, -4.9); rMSSD: -9.4 (-10.1, -8.6); pNN50 + 1: -13.1 (-14.3, -11.9) LF: -10.7 (-12.4, -9.1); HF: -14.9 (-16.5, -13.3); LF/HF: 4.9 (3.6, 6.2)' H: 0.9 (0.7, 1.1) Independent short- and medium-term associations with HRV across all time periods; % change per IQR (95%CI); IQR 5-min means = 6.8 μg/m³ and 23: 55-h means = 4.2 μg/m³ SDNN: 5-min mean: -0.5 (-0.8, -0.1); 23: 55-h mean: -4.6 (-5.3, -4.0) rMSSD: 5-min mean: -0.9 (-1.3, -0.5); 23: 55-h mean: -7.5 (-8.1 to -6.8) pNN50 + 1 ; 5-min mean: -1.1 (-1.7 to -0.5); 23: 55-h mean: -9.9 (-10.9 to -8.9) LF; 5-min mean: 0.4 (-0.5, 1.2); 23: 55-h mean: -10.0 (-11.4 to -8.6) HF; 5-min mean: -1.5 (-2.3 to -0.6); 23: 55-h mean: -12.9 (-14.2 to -11.5) LF/HF; 5-min mean: 1.9 (1.3, 2.4); 23: 55-h mean: 3.2 (2.1, 4.3) H: 5-min mean: 0.1 (0.1, 0.2); 23: 55-h mean: 0.8 (0.7, 0.9) Independent associations of short-term averages (5-min means) of PM with HRV by bus and nonbus periods; IQR for bus = 10 μg/m³) and nonbus = 5.6 μg/m³) % change (95%CI); p-value of interaction SDNN; Bus: -5.0 (-6.3 to -3.7); Nonbus: -0.5 (-0.9 to -0.2); p-value for interaction: <0.0001 rMSSD; Bus: -4.8 (-6.2 to -3.5); Nonbus: -0.7 (-1.1 to -0.4 p-value for interaction: <0.0001; pNN50 + 1 ; Bus: -6.3 (-8.4 to -4.2); Nonbus: -0.8 (-1.4 to -0.3); p-value for interaction: <0.0001 LF: Bus: -7.0 (-9.8 to -4.1) Nonbus: 0.6 (-0.1, 1.4); p-value for interaction: <0.0001 HF: Bus: -10.7 (-13.5 to -7.9)' Nonbus: -0.7 (-1.5, 0.04) p-value for interaction: <0.0001 LF/HF: Bus: 3.9 (1.7, 6.0); Nonbus: 1.4 (0.8, 1.9); p-value for interaction: 0.39 H: Bus: 0.7 (0.5, 1.0); Nonbus: -0.01 (-0.08, 0.1); p-value for interaction: <0.0001 **Note:** Exposure to health associations by all lag periods presented in Figure 2 (magnitude of associations increased with averaging period, with the largest associations consistently found for 24-h moving averages)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Auchincloss et al. (2008) **Period of Study:** Jul 2000–Aug 2002 **Location:** 6 US communities (Baltimore City and Baltimore County, Maryland; Chicago, Illinois; Forsyth County, North Carolina; Los Angeles, California; Northern Manhattan and the Bronx, New York; and St. Paul, Minnesota); part of MESA (Multi-ethnic Study of Atherosclerosis)	**Outcome:** Blood pressure: systolic (SBP), diastolic (DBP), mean arterial (MAP), pulse pressure (PP); Avg of 2nd and 3rd BP measurement used for analyses **Age Groups:** 45-84 years **Study Design:** Cross-sectional (Multi-Ethnic Study of Atherosclerosis baseline examination) **N:** 5,112 persons (free of clinically apparent cardiovascular disease) **Statistical Analyses:** Linear regression; secondary analyses used log binomial models to fit a binary hypertension outcome **Covariates:** Age, sex, race/ethnicity, per capita family income, education, BMI, diabetes status, cigarette smoking status, exposure to ETS, high alcohol use, physical activity, BP medication use, meteorology variables, and copollutants; examined site as a potential confounder and effect modifier; heterogeneity of effects also examined by traffic-related exposures, age, sex, type 2 diabetes, hypertensive status, cigarette use **Season:** Adjusted for temperature and barometric pressure to adjust for seasonality (because seasons vary by the study sites); Also performed sensitivity analyses adjusting for season to examine the potential for residual confounding not accounted for by weather variables **Dose-response Investigated?** Assessed nonlinear relationships–no evidence of strong threshold/nonlinear effects for $PM_{2.5}$ **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 5 exposure metrics constructed: prior day, avg of prior 2 days, prior 7 days, prior 30 days, and prior 60 days **Mean (SD):** Prior day: 17.0 (10.5) Prior 2 days: 16.8 (9.3) Prior 7 days: 17.0 (6.9) Prior 30 days: 16.8 (5.0) Prior 60 days: 16.7 (4.4) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** Used monitor nearest the participant's residence to calculate exposure metrics **Copollutant:** SO_2; NO_2; CO Traffic-related exposures (straight-line distance to a highway; total road length around a residence)	**PM Increment:** 10 μg/m3 (approx. equivalent to difference between 90th and 10th percentile for prior 30 day mean) **Effect Estimate [Lower CI, Upper CI]:** Adjusted mean difference (95% CI) in PP and SBP (mmHg) per 10 μg/m3 increase in $PM_{2.5}$ (averaged for the prior 30 days) **Pulse Pressure** Adjustment variables: Person-level **Covariates:** 1.04 (0.25, 1.84) Person-level cov., weather: 1.12 (0.28, 1.97) Person-level cov., weather, gaseous copollutants: 2.66 (1.61, 3.71) Person-level cov., study site: 0.93 (-0.04, 1.90) Person-level cov., study site, weather: 1.11 (0.01, 2.22) Person-level cov., study site, weather, gaseous copollutants: 1.34 (0.10, 2.59) **Systolic Blood Pressure** Adjustment variables: Person-level **Covariates:** 0.66 (-0.41, 1.74) Person-level cov., weather: 0.99 (-0.15, 2.13) Person-level cov., weather, gaseous copollutants: 2.8 (1.38, 4.22) Person-level cov., study site: 0.86 (-0.45, 2.17) Person-level cov., study site, weather: 1.32 (-0.18, 2.82) Person-level cov., study site, weather, gaseous copollutants: 1.52 (-0.16, 3.21) **Additional results:** Associations became stronger with longer averaging periods up to 30 days. For example: Adjusted (personal covariates and weather) mean differences in PP: Prior day: -0.38 (-0.76, 0.00) Prior 2 days: -0.22 (-0.65, 0.21) Prior 7 days: 0.52 (-0.08, 1.11) Prior 30 days: 1.12 (0.28, 1.97) Prior 60 days: 1.08 (0.11, 2.05) (Pattern held for additional adjustments and for SBP results; therefore, only results for 30-day mean differences were presented) **Additional results (not presented):** None of DBP results were statistically significant; results for MAP were similar to SBP, though weaker and generally not significant **Effect modification:** associations between $PM_{2.5}$ and BP were stronger for persons taking medications, with hypertension, during warmer weather, in the presence of high NO_2, residing ≤ 300m from a highway, and surrounded by a high density of roads (Fig 1); associations were not modified for age, sex, diabetes, cigarette smoking, study site, high levels of CO or SO_2, season , nor residence ≤ 400m fro a highway **Note:** supplementary material available on-line

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chuang et al. (2007b) **Period of Study:** Between Apr-Jun 2004 or 2005 **Location:** Taipei, Taiwan	**Outcome:** High-sensitivity C-reactive protein (hs-CRP); Fibrinogen, plasminogen activator fibrinogen inhibitor-1 (PAI-1), tissue-type plasminogen activator (tPA), 8-hydroxy-2'-deoxyguanosine (8-OHdG), and log-transformed HRV indices (SDNN = standard deviation of NN intervals, r-MSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, LF = low frequency [0.04-0.15Hz], and HF = high frequency[0.15-0.40Hz]) **Age Groups:** 18-25 yrs **Study Design:** Panel (cross-sectional) **N:** 76 students **Statistical Analyses:** linear mixed-effects models **Covariates:** Age, sex, BMI, weekday, temperature of previous day, relative humidity **Season:** Only 1 season of data collection **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$, nitrate, sulfate **Averaging Time:** Hourly data used to calculate averages over 1-3 day periods **Mean (SD):** 1-day avg: 31.8 (10.6) 2-day avg: 36.4 (12.6) 3-day avg: 36.5 (12.6) **Range (Min, Max):** 1-day avg: 16.2, 50.1 2-day avg: 15.0, 53.4 3-day avg: 12.7, 59.5 **Monitoring Stations:** 2 sites (each pollutant measured at one site only) **Copollutant:** PM_{10}; Sulfate; Nitrate; OC; EC; NO_2; CO; SO_2 ; O_3	**$PM_{2.5}$ Increment:** IQR (1-d avg: 20.4; 2-day avg: 25.2; 3-day avg: 20.0) **Effect Estimate [Lower CI, Upper CI]:** % change in health endpoint per increase in IQR of $PM_{2.5}$ (1-3 day averaging period; single pollutant models) hs-CRP: 1-d: 90.2 (-10.2, 190.1); 2-d: 99.1 (-26.1, 224.3) 3-d: 100.4 (-2.9, 203.7) 8-OHdG: 1-d: -5.0 (-14.3, 4.4); 2-d: -5.5 (-15.6, 4.6); 3-d: -5.6 (-13.8, 2.6) PAI-1: 1-d: 20.4 (17.3, 33.5); 2-d: 16.2 (1.9, 30.5); 3-d: 20.0 (18.5, 31.5) tPA: 1-d: 12.0 (-2.4, 26.3); 2-d: 12.0 (-2.9, 26.9); 3-d: 12.0 (-2.7, 26.6) Fibrinogen: 1-d: 2.6 (-2.7, 7.8); 2-d: 1.5 (-4.1, 7.1); 3-d: 3.6 (-0.8, 8.1) **Heart Rate Variability** SDNN: 1-d: -4.0 (-6.1 to -1.9); 2-d: -2.5 (-4.6 to -0.4); 3-d: -3.0 (-5.0 to -1.1) r-MSSD: 1-d: -3.0 (-8.7, 2.7); 2-d: -2.0 (-8.4, 4.4); 3-d: -3.6 (-8.8, 1.6) LF: 1-d: -3.1 (-6.1 to -0.1); 2-d: -3.2 (-4.6, 0.1); 3-d: -3.4 (-6.1 to -0.6) HF: 1-d: -3.7 (-9.4, 2.1); 2-d: -2.1 (-8.4, 4.3); 3-d: -4.0 (-9.3, 1.2)
Reference: Chuang et al. (2007b) **Period of Study:** Between Apr-Jun 2004 or 2005 **Location:** Taipei, Taiwan	**Outcome:** High-sensitivity C-reactive protein (hs-CRP); Fibrinogen, plasminogen activator fibrinogen inhibitor-1 (PAI-1), tissue-type plasminogen activator (tPA), 8-hydroxy-2'-deoxyguanosine (8-OHdG), and log-transformed HRV indices (SDNN = standard deviation of NN intervals, r-MSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, LF = low frequency [0.04-0.15Hz], and HF = high frequency[0.15-0.40Hz]) **Age Groups:** 18-25 yrs **Study Design:** Panel (cross-sectional) **N:** 76 students **Statistical Analyses:** Linear mixed-effects models **Covariates:** Age, sex, BMI, weekday, temperature of previous day, relative humidity **Season:** Only 1 season of data collection **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** Nitrate **Averaging Time:** Hourly data used to calculate averages over 1-3 day periods **Mean (SD):** 1-day avg: 4.5 (2.7) 2-day avg: 4.7 (2.4) 3-day avg: 4.4 (2.2) **Range (Min, Max):** 1-day avg: 0.7, 10.6 2-day avg: 0.7, 8.9 3-day avg: 0.8, 7.5 **Monitoring Stations:** 2 sites (each pollutant measured at one site only) **Copollutant:** $PM_{10;}$ Sulfate; $PM_{2.5}$; OC; EC; NO_2; CO; SO_2 ; O_3	**Nitrate Increment:** IQR (1-d avg: 2.5; 2-day avg: 4.0; 3-day avg: 3.4) **Effect Estimate [Lower CI, Upper CI]:** % change in health endpoint per increase in IQR of nitrate (1-3 day averaging period; single pollutant models) hs-CRP: 1-d: -2.1 (-21.9, 17.8); 2-d: -11.6 (-58.6, 35.5) 3-d: -18.7 (-69.9, 32.5) 8-OHdG: 1-d: 9.0 (4.0, 14.1); 2-d: 15.1 (5.9, 24.3) 3-d: 15.0 (4.9, 25.0) PAI-1: 1-d: 4.0 (-2.5, 10.4); 2-d: 11.6 (0.1, 23.1) 3-d: 16.9 (4.3, 29.4) tPA: 1-d: 2.0 (-6.2, 10.3); 2-d: 12.9 (-1.6, 27.5) 3-d: 10.0 (-5.8, 25.8) Fibrinogen: 1-d: 1.6 (-1.3, 4.5); 2-d: 1.3 (-3.9, 6.5) 3-d: 1.0 (-4.6, 6.6) **Heart Rate Variability** SDNN: 1-d: -1.5 (-2.6 to -0.3); 2-d: -2.6 (-4.7 to -0.5) 3-d: -3.0 (-5.3 to -0.7) r-MSSD: 1-d: -5.5 (-8.7 to -2.2); 2-d: -7.1 (-14.0 to -0.2) 3-d: -8.1 (-14.5 to -1.8) LF: 1-d: -1.0 (-1.6 to -0.5); 2-d: -2.0 (-5.6, 1.6) 3-d: -2.0 (-5.2, 1.2) HF: 1-d: -2.0 (-5.3, 14[potential typo, possibly 1.4]) 2-d: -4.9 (-10.9, 0.9); 3-d: -6.9 (-13.4 to -0.3)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chuang et al. (2007b) **Period of Study:** Between Apr-Jun 2004 or 2005 **Location:** Taipei, Taiwan	**Outcome:** High-sensitivity C-reactive protein (hs-CRP); Fibrinogen, plasminogen activator fibrinogen inhibitor-1 (PAI-1), tissue-type plasminogen activator (tPA), 8-hydroxy-2'-deoxyguanosine (8-OHdG), and log-transformed HRV indices (SDNN = standard deviation of NN intervals, r-MSSD = square root of the mean of the sum of the squares of differences between adjacent NN intervals, LF = low frequency [0.04-0.15Hz], and HF = high frequency[0.15-0.40Hz]) **Age Groups:** 18-25 yrs **Study Design:** Panel (cross-sectional) **N:** 76 students **Statistical Analyses:** Linear mixed-effects models **Covariates:** Age, sex, BMI, weekday, temperature of previous day, relative humidity **Season:** Only 1 season of data collection **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** Sulfate **Averaging Time:** Hourly data used to calculate averages over 1-3 day periods **Mean (SD):** 1-day avg: 4.1 (3.6) 2-day avg: 4.1 (3.7) 3-day avg: 3.9 (3.5) **Range (Min, Max):** 1-day avg: 0.4, 10.9 2-day avg: 0.4, 11.9 3-day avg: 0.4, 11.5 **Monitoring Stations:** 2 sites (each pollutant measured at one site only) **Copollutant:** $PM_{10;}$ $PM_{2.5;}$ Nitrate; OC; EC; NO_2; CO; SO_2 ; O_3	**Sulfate Increment:** IQR (1-d avg: 3.9; 2-day avg: 4.3; 3-day avg: 3.8) **Effect Estimate [Lower CI, Upper CI]:** % change in health endpoint per increase in IQR of sulfate (1-3 day averaging period; single pollutant models) hs-CRP: 1-d: 80.0 (9.8, 150.2); 2-d: 87.1 (14.9, 159.4) 3-d: 71.1 (13.0, 129.2) 8-OHdG: 1-d: 1.0 (0.3, 1.3); 2-d: -0.4 (-5.4, 4.7) 3-d: -0.3 (-4.3, 3.7) PAI-1: 1-d: 12.0 (5.4, 18.7); 2-d: 13.3 (6.6, 19.9) 3-d: 11.2 (5.7, 16.6) tPA: 1-d: 2.0 (-4.6, 8.7); 2-d: 3.8 (-2.8, 10.3) 3-d: 3.0 (-2.3, 8.2) Fibrinogen: 1-d: 2.9 (0.2, 5.5); 2-d: 2.8 (0.1, 5.5) 3-d: 2.2 (0.4, 4.7) **Heart Rate Variability** SDNN: 1-d: -3.1 (-4.1 to -2.1); 2-d: -4.1 (-5.2 to -3.1) 3-d: -2.0 (-2.9 to -1.2) r-MSSD: 1-d: -5.0 (-8.0 to -2.0); 2-d: -6.0 (-8.9 to -2.9) 3-d: -5.7 (-8.2 to -3.2) LF: 1-d: -3.4 (-4.9 to -1.8); 2-d: -3.0 (-4.5 to -1.5) 3-d: -3.0 (-4.3 to -1.7) HF: 1-d: -3.5 (-6.5 to -0.4); 2-d: -3.9 (-7.0 to -0.8) 3-d: -3.0 (-5.5 to -0.5)
Reference: Diez Roux et al. (2006) **Period of Study:** Baseline data collected June 2000–Aug 2002; **Location:** USA (6 field centers: Baltimore, MD; Chicago, IL; Forsyth Co, NC; Los Angeles, CA; New York, NY; St. Paul, MN	**Outcome:** C-reactive protein (CRP) assessed continuously and as a dichotomous variable (cutpoint, 3 mg/L); interleukin-6 (IL-6) **Age Groups:** 45-84 yrs **Study Design:** Cross-sectional **N:** 5634 persons **Statistical Analyses:** Linear regression & logistic regression **Covariates:** Age, sex, race/ethnicity, general health status, BMI, diabetes, cigarette status, socindhand smoke, physical activity, arthritis flare in last 2 weeks, medications, infections in last 2 weeks (also ran models including site, copollutants, and weather) **Season:** Examined seasonal patterns in the residuals of fully adjusted models; stratified by season **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Prior day, prior 2 days, prior week, prior 30 days, and prior 60 days **Mean (SD):** Presented in Fig 1 by site **Percentiles:** Presented in Fig 1 by site **Range:** NR **Monitoring Stations:** NR; Long-term exposure to PM estimated based on residential history reported retrospectively; all addresses geocoded; ambient AP obtained from US EPA **Copollutant:** SO_2; NO_2; CO; O_3	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Adjusted (all personal-level covariates) relative difference in CRP (mg/L) per 10 µg/m^3 increase in $PM_{2.5}$ Prior day: 0.99 (0.96, 1.01) Prior 2 days: 0.99 (0.96, 1.01) Prior 7 days: 1.00 (0.96, 1.04) Prior 30 days: 1.03 (0.98, 1.10) Prior 60 days: 1.04 (0.97, 1.11) Odds Ratios of CRP of ≥ 3 mg/L per 10 µg/m^3 increase in $PM_{2.5}$ (adjusted for all personal-level covariates) Prior day: 0.98 (0.92, 1.04) Prior 2 days: 0.99 (0.93, 1.06) Prior 7 days: 1.05 (0.96, 1.15) Prior 30 days: 1.12 (0.98, 1.29) Prior 60 days: 1.12 (0.96, 1.32)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Dubowsky et al. (2006) **Period of Study:** March–Jun 2002 **Location:** St. Louis, Missouri	**Outcome:** White blood cells (WBC), C-reactive protein (CRP), interleukin-6 (IL-6) **Age Groups:** ≥ 60 yrs **Study Design:** Panel (4 planned repeated measures; n = 35 participated in 4 trips) **N:** 44 participants **Statistical Analyses:** Linear mixed models **Covariates:** Sex, obesity, diabetes, smoking history, time-varying parameters (apparent temperature, h, day, trip, residence, mold, pollen, illness, and juice intake), medication and vitamin consumption (day of blood draw) **Season:** Limited data collection period **Dose-response Investigated?** No **Statistical Package:** SAS v8.02	**Pollutant:** $PM_{2.5}$ (ambient) **Averaging Time:** Hourly data used to calculate avg concentrations over 1-7 days preceding the blood draw (ambient $PM_{2.5}$); microenvironmental $PM_{2.5}$ measures were averaged over the 1-2 days preceding the blood draw **Mean (SD) (1-day):** 16 (6.0) **Percentiles (1-day):** 0: 6.5; 25th: 12; 75th: 22; 100th: 28 **Monitoring Stations:** 1 ambient monitor **Copollutant:** $PM_{2.5}$ (ambient); BC (ambient); $PM_{2.5}$ (microenvironment); CO; NO_2; SO_2; O_3	**PM Increment:** 6.1 μg/m^3 (5-d mean) **Effect Estimate [Lower CI, Upper CI]:** **Note:** Most results presented in figures. Selected result in abstract text: % change in WBC per increase in IQR (5.4 μg/m^3) of $PM_{2.5}$ averaged over the previous week: 5.5 (0.1, 11) Associations (% changes and 95%CI) between 5-day mean ambient concentrations and markers of inflammation per increase (IQR) in pollutant. CRP: All participants: 14 (-5.4, 37); Among those with all 3 conditions (diabetes, obesity, and hypertension): 81 (21, 172); Among those with at least 2 of the conditions: 11 (-7.3, 33) IL-6: All participants: -2.1 (-13, 11); Among those with all 3 conditions (diabetes, obesity, and hypertension): 23 (-5.3, 59); Among those with at least 2 of the conditions: -3.1 (-14, 9.7) WBC (x10^9/L): All participants: 3.4 (-1.8, 8.9); Among those with all 3 conditions (diabetes, obesity, and hypertension): 0.4 (-8.8, 11); Among those with at least 2 of the conditions: 3.6 (-1.7, 9.1)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lanki et al. (2006b) **Period of Study:** Autumn 1998–spring 1999 **Location:** Helsinki, Finland	**Outcome:** ST segment depressions (2 endpoints: >0.1mV regardless of the direction of the ST slope and >0.1mV with horizontal or downward slope [stricter criteria]) **Age Groups:** Mean = 68.2 (6.5) yrs **Study Design:** Panel **N:** 45 elderly nonsmoking persons with stable coronary heart disease; 342 total exercise tests for analyses **Statistical Analyses:** Generalized additive models with penalized splines (logistic regression); principal components analysis and linear regression of 13 measured elements used to apportion $PM_{2.5}$ mass between different sources **Covariates:** Subject, linear terms for time trend, temperature, relvative humidity, penalized spline for change in heart rate during the exercise test **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-plus 2000 and R	**Pollutant:** $PM_{2.5}$ (Analyses conducted for source specific $PM_{2.5}$) **Averaging Time:** Daily filter samples **Mean:** Crustal: 0.6 Long-range transported: 6.4 Oil combustion: 1.6 Salt: 0.9 Local traffic: 2.9 Total: 12.8 **Percentiles:** Crustal 25: 0.0; 50: 0.4; 75: 1.1; Max: 5.3 Long-range transported 25: 2.2; 50: 5.5; 75: 9.8; Max: 26.5 Oil combustion 25: 0.6; 50: 1.3; 75: 2.3; Max: 12.2 Salt 25: 0.3; 50: 0.8; 75: 1.2; Max: 5.9 Local traffic 25: 1.7; 50: 2.5; 75: 3.4; Max: 12.0 Total 25: 8.3; 50: 10.6; 75: 15.9; Max: 39.8 **Monitoring Stations:** 1 monitor **Copollutant (correlation):** Correlations with $PM_{2.5}$: Crustal: r = -0.01 Long-range transported: r = 0.82 Oil combustion: r = 0.35 Salt: r = 0.19 Local traffic: r = 0.26	**PM Increment:** 1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Adjusted ORs between daily source-specific $PM_{2.5}$ concentrations and ST segment depressions.ST segment depression defined as >0.1 mV (n = 62) **Crustal** Lag0: 0.80 (0.47, 1.36); Lag1: 0.66 (0.40, 1.10) Lag2: 1.18 (0.68, 2.06); Lag3: 1.87 (0.85, 4.09) **Long-range transport** Lag0: 0.94 (0.84, 1.05); Lag1: 1.00 (0.92, 1.08) Lag2: 1.11 (1.02, 1.20); Lag3: 1.06 (0.95, 1.18) **Oil combustion** Lag0: 0.87 (0.57, 1.32); Lag1: 1.04 (0.75, 1.45) Lag2: 1.10 (0.83, 1.46); Lag3: 1.12 (0.79, 1.58) **Salt** Lag0: 1.03 (0.57, 1.85); Lag1: 0.72 (0.37, 1.40) Lag2: 0.66 (0.31, 1.40); Lag3: 1.55 (0.83, 2.89) **Local traffic** Lag0: 0.91 (0.69, 1.21); Lag1: 1.22 (0.88, 1.69) Lag2: 1.53 (1.19, 1.97); Lag3: 0.98 (0.78, 1.23) ST segment depression defined as >0.1 mV with horizontal or downward slope (n = 46) **Crustal** Lag0: 0.76 (0.42, 1.35); Lag1: 0.41 (0.22, 0.79) Lag2: 1.17 (0.65, 2.09); Lag3: 1.60 (0.72, 3.59) **Long-range transport** Lag0: 0.98 (0.86, 1.10); Lag1: 1.03 (0.95, 1.12) Lag2: 1.11 (1.02, 1.21); Lag3: 1.02 (0.95, 1.10) **Oil combustion** Lag0: 0.95 (0.61, 1.49); Lag1: 1.13 (0.76, 1.68) Lag2: 1.33 (0.98, 1.80); Lag3: 1.29 (0.90, 1.86) **Salt** Lag0: 1.15 (0.56, 2.38); Lag1: 0.90 (0.44, 1.81) Lag2: 1.39 (0.63, 3.08); Lag3: 1.93 (1.00, 3.72) **Local traffic** Lag0: 0.89 (0.64, 1.23); Lag1: 1.21 (0.86, 1.71) Lag2: 1.37 (1.03, 1.83); Lag3: 1.03 (0.80, 1.32) Adjusted ORs for the association of indicator elements of $PM_{2.5}$ sources and ST segment depressions in multipollutant models (models include all 5 indicator elements). ST segment depression defined as >0.1 mV (n = 62) **Si (Crustal)** Lag0: 0.73 (0.39, 1.38); Lag1: 0.48 (0.25, 0.93) Lag2: 0.78 (0.35, 1.71); Lag3: 1.95 (0.69, 5.48) **S (Long-range transport)** Lag0: 0.70 (0.25, 1.95); Lag1: 0.58 (0.23, 1.47) Lag2: 1.08 (0.44, 2.63); Lag3: 1.60 (0.73, 3.48) **Ni (Oil combustion)** Lag0: 0.78 (0.30, 2.04); Lag1: 1.20 (0.58, 2.46) Lag2: 1.15 (0.61, 2.18); Lag3: 1.02 (0.41, 2.54) **Cl (Salt)** Lag0: 1.03 (0.79, 1.34); Lag1: 0.88 (0.56, 1.38) Lag2: 1.02 (0.62, 1.69); Lag3: 1.27 (0.85, 1.91) **ABS (Local traffic)** Lag0: 0.92 (0.36, 2.37); Lag1: 1.83 (0.73, 4.59) Lag2: 4.46 (1.69, 11.79); Lag3: 0.92 (0.40, 2.12) ST segment depression defined as >0.1 mV with horizontal or downward slope (n = 46) **Si (Crustal)** Lag0: 0.67 (0.33, 1.36); Lag1: 0.34 (0.15, 0.81) Lag2: 0.81 (0.33, 2.00); Lag3: 1.90 (0.64, 5.65) **S (Long-range transport)** Lag0: 0.84 (0.29, 2.47); Lag1: 0.89 (0.34, 2.32) Lag2: 1.36 (0.54, 3.45); Lag3: 1.12 (0.53, 2.40) **Ni (Oil combustion)** Lag0: 1.10 (0.36, 3.37); Lag1: 1.16 (0.45, 2.96) Lag2: 1.64 (0.84, 3.20); Lag3: 1.63 (0.64, 4.14) **Cl (Salt)** Lag0: 1.13 (0.80, 1.62); Lag1: 0.99 (0.58, 1.68) Lag2: 1.55 (0.87, 2.76); Lag3: 1.45 (0.94, 2.25) **ABS (Local traffic)** Lag0: 0.74 (0.25, 2.23); Lag1: 1.76 (0.62, 5.00) Lag2: 4.86 (1.55, 15.26); Lag3: 0.97 (0.39, 2.41)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lipsett et al. (2006) **Period of Study:** February–May 2000 **Location:** Coachella Valley, CA	**Outcome:** HRV parameters, specifically SDNN, SDANN, r-MSSD, LF, HF, total power, triangular index (TRII). **Study Design:** Panel study **N:** 19 non-smoking adults with coronary artery disease **Statistical Analysis:** Mixed linear regression models with random effects parameters	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2 h **Mean (range)** Indio: 23.2 (6.3-90.4) Palm Springs: 14 (4.7-52) **Monitoring Stations:** 2 **Copollutant:** O_3	**PM Increment:** SE*100 **Effect Estimate (change in HRV per unit increase in PM concentration):** SDNN: -0.37 msec (SE = 1.01) **Notes:** Weekly ambulatory 24 h ECG recordings (once per week for up to 12 weeks), using Holter monitors, were made.Subjects' residences were withinn 5 miles of one of two PM monitoring sites.Decreased HRV was associated with $PM_{2.5}$, but these effects were not statistically significant. Regressed HRV parameters against 18: 00–20: 00 mean particulate pollution.
Reference: Luttman-Gibson et al. (2006) **Period of Study:** June–December 2000 **Location:** Steubenville, OH	**Outcome:** Heart rate variability **Age Groups:** **Study Design:** Panel study **N:** 32 participants **Statistical Analysis:** Linear mixed models	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h; 24 h **Mean (IQR)** $PM_{2.5}$: 20.0 (15.2) Sulfate: 6.9 (5.1) EC: 1.1 (0.6) **Copollutant:** NO_2, SO_2, O_3	**PM Increment:** IQR **Percent change (95% CI):** Each 13.4 µg/m^3 increase in 24 hour mean $PM_{2.5}$ concentration was associated with: SDNN: -4.0% (95% CI: -7.0% to -0.9%) r-MSSD: -6.5% (95% CI: -12.1% to -0.6%) HF: -11.4% (95% CI: -21.5% to -0.1%) Each 5.1 µg/m^3 increase in suflates on the previous day was associated with: SDNN: -3.3% (95% CI: -6.0% to -0.5%) r-MSSD: -5.6% (95% CI: -10.7%, 0.2%) HF: -10.3% (95% CI: -19.5% to -0.1%) **Notes:** The authors conclude that increases in both traffic related particles and sulfates may adversely effect autonomic function.
Reference: Mar et al. (2005b) **Period of Study:** 1999–2001 **Location:** Seattle, WA	**Outcome:** Change in arterial O_2 saturation, heart rate, and blood pressure (SBP and DBP) **Age Groups:** >75 years **Study Design:** Panel study **N:** 88 elderly subjects **Statistical Analysis:** GEE	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Personal: 9.3(8.4) Indoor: 7.4 (4.8) Outdoor: 9.0 (4.6)	**PM Increment:** 10 µg/m^3 **Unit change in measure (95% CI):** Among all subjects: Each increase in outdoor same day $PM_{2.5}$ was associated with: SBP: -0.81 mmHg (95% CI: -2.34, 0.73) DBP: -0.46 mmHg (95% CI: -1.49, 0.57) H: -0.75 beats/min (95% CI: -1.42 to -0.07) Each increase in indoor same day $PM_{2.5}$ was associated with: SBP: 0.92 mmHg (95% CI: -2.04, 3.87) DBP: 0.38 mmHg (95% CI: -1.43, 2.20) H: 0.22 beats/min (95% CI: -0.71, 1.16) Each increase in personal same day $PM_{2.5}$ was associated with: SBP: 0.37 mmHg (95% CI: -0.93, 1.67) DBP: -0.20 mmHg (95% CI: -0.85, 0.46) H: 0.44 beats/min (95% CI: 0.04, 0.84) **Notes:** Results by health status presented in Figure 1 Used 2 sessions that each were 10 consecutive days of measurements; Used personal, indoor, and outdoor measures of $PM_{2.5}$
Reference: Metzger et al. (2007) **Period of Study:** August 1998–December 2002 **Location:** Atlanta, GA	**Outcome:** Days with any event recorded by the ICD, days with ICD shocks/defibrillation and days with either cardiac pacing or defibrillation **Study Design:** Repeated measures **N:** 884 subjects between 1993 and 2002 **Statistical Analysis:** Logistic regression with GEE to account for residual autocorrelation within subjects	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** $PM_{2.5}$: 17.8 (8.6) $PM_{2.5}$ sulfates: 5.0 (3.4) $PM_{2.5}$ EC: 1.7 (1.2) $PM_{2.5}$ OC: 4.4 (2.4) $PM_{2.5}$ water-soluble metals: 0.029 (0.024) **Percentiles:** $PM_{2.5}$: Median: 16.2 $PM_{2.5}$ sulfates: Median: 4.1 $PM_{2.5}$ EC: Median: 1.4 $PM_{2.5}$ OC: Median: 3.9 $PM_{2.5}$ water-soluble metals: Median: 0.022 **Copollutant:** O_3; NO_2; CO; SO_2; oxygenated hydrocarbons	**PM Increment: OR (95% CI):** Outcome = Any event recorded by ICD $PM_{2.5}$ OR = 1.00 (95% CI: 0.95, 1.04) $PM_{2.5}$ EC OR = 1.01 (95% CI: 0.98, 1.05) $PM_{2.5}$ OC OR = 1.01 (95% CI: 0.98, 1.03) $PM_{2.5}$ Sulfates OR = 0.99 (95% CI: 0.93, 1.06) $PM_{2.5}$ Water soluble metals OR = 0.95 (95% CI: 0.90, 1.00

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: O'Neill et al. (2007) **Period of Study:** May 1998–Dec 2002 **Location:** Boston, MA	**Outcome:** Soluble intercellular adhesion molecule 1 (ICAM-1); vascular cell adhesion molecule 1 (VCAM-1); von Willebrand factor (vWF) **Age Groups:** Mean (SD): 56.6 (10.6) **Study Design:** Cross-sectional **N:** 92 participants (type 2 diabetic patients) **Statistical Analyses:** linear regression **Covariates:** Apparent temperature, season, age, race, sex, glycosylated hemoglobin, cholesterol, smoking history, BMI **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h (lagged moving averages of days 0 to 1, 2, 3, 4, and 5) **Mean (SD):** 11.4 (5.9); descriptive statistics represent entire study period **Percentiles:** IQR range: 7.6 **Range (Min, Max):** 0.07, 33.7) **Monitoring Stations:** 1 site **Copollutant:** $PM_{2.5}$; BC; SO_4^{2-}	**PM Increment:** IQR (specific to lag period) **Effect Estimate [Lower CI, Upper CI]:** % change per IQR of $PM_{2.5}$ **ICAM-1 -** All subjects Lag 0: 2.87 (-4.63, 10.95); 2 dma: 2.25 (-5.15, 10.22); 3 dma: 1.48 (-5.63, 9.11); 4 dma: 1.80 (-4.98, 9.07); 5 dma: 1.51 (-5.30, 8.80); 6 dma: 2.12 (-4.23, 8.89) **Subjects not known to be taking statins** Lag 0: 5.47 (-3.74, 15.57); 2 dma: 5.70 (-3.70, 16.01); 3 dma: 4.57 (-4.31, 14.27); 4 dma: 4.57 (-4.27, 14.23); 5 dma: 3.80 (-4.84, 13.22); 6 dma: 3.79 (-4.49, 12.80) **Subjects who report smoking in the past (but not within 6 months)** Lag 0: 0.9 (-9.56, 12.66); 2 dma: 0.40 (-12.08, 14.65); 3 dma: 1.34 (-9.23, 13.14); 4 dma: 2.29 (-6.84, 12.30); 5 dma: 1.09 (-8.30, 11.44); 6 dma: 3.08 (-6.30, 13.40); **Subjects who did not report smoking in the past** Lag 0: 0.46 (-8.23, 9.97); 2 dma: 1.37 (-7.96, 11.65); 3 dma: -0.96 (-10.01, 9.00); 4 dma: -1.34 (-10.35, 8.58); 5 dma: -0.87 (-10.17, 9.40); 6 dma: -1.78 (-10.64, 7.94) **VCAM-1 -** All subjects Lag 0: 6.88 (-2.88, 17.62); 2 dma: 8.18 (-1.43, 18.72); 3 dma: 6.92 (-1.66, 16.25); 4 dma: 6.46 (-1.16, 14.66); 5 dma: 8.57 (0.05, 17.80); 6 dma: 11.76 (3.48, 20.70) **Subjects not known to be taking statins** Lag 0: 10.26 (-0.64, 22.35); 2 dma: 15.02 (3.76, 27.49); 3 dma: 14.59 (3.94, 26.34); 4 dma: 15.15 (4.54, 26.84); 5 dma: 16.16 (5.77, 27.58); 6 dma: 17.66 (7.77, 28.45) **Subjects who report smoking in the past (but not within 6 months)** Lag 0: 13.2 (-1.30, 29.72); 2 dma: 18.4 (0.69, 39.33); 3 dma: 15.7 (1.19, 32.30); 4 dma: 13.1 (0.88, 26.78); 5 dma: 13.2 (0.49, 27.58); 6 dma: 16.2 (3.76, 30.10) **Subjects who did not report smoking in the past** Lag 0: -3.12 (-12.41, 7.17); 2 dma: -0.34 (-10.57, 11.05); 3 dma: -1.09 (-11.15, 10.12); 4 dma: -0.81 (-10.91, 10.43); 5 dma: 2.07 (-8.59, 13.96); 6 dma: 4.89 (-5.56, 16.50) **vWF -** All subjects Lag 0: 15.16 (-9.79, 47.01); 2 dma: 12.57 (-9.19, 39.55); 3 dma: 25.14 (-9.87, 73.74); 4 dma: 23.42 (-9.47, 68.25); 5 dma: 17.92 (-10.22, 54.87); 6 dma: 20.48 (-8.82, 59.22) **Subjects not known to be taking statins** Lag 0: 7.40 (-19.82, 43.88); 2 dma: 7.10 (-19.09, 41.76); 3 dma: 10.78 (-17.92, 49.52); 4 dma: 11.61 (-16.64, 49.42); 5 dma: 9.15 (-20.32, 49.53); 6 dma: 7.91 (-20.70, 46.85) **Subjects who report smoking in the past (but not within 6 months)** Lag 0: 19.23 (-24.29, 87.77); 2 dma: 19.92 (-29.65,104.41); 3 dma: 29.54 (-17.24, 102.76); 4 dma: 41.98 (-6.95, 116.63); 5 dma: 44.05 (-1.23, 110.07); 6 dma: 50.39 (9.35, 106.82) **Subjects who did not report smoking in the past** Lag 0: -14.21 (-53.20, 57.24); 2 dma: -20.66 (-63.14, 70.77); 3 dma: -28.89 (-68.43, 60.19); 4 dma: -23.51 (-55.11, 30.34); 5 dma: -29.18 (-60.08, 25.66); 6 dma: -30.68 (-55.95, 9.08)
Reference: O'Neill et al. (2005a) **Period of Study:** Baseline period: May 1998–January 2000 Time trial: 2000–2002 **Location:** Boston, MA	**Outcome:** Changes in vascular reactivity, specifically percent change in brachial artery diameter (flow-mediated and nitroglycerin-mediated) **N:** 270 patients with diabetes or at risk of diabetes, who participated in non-air pollution related studies at the Joselyn Diabetes Center in Boston **Statistical Analysis:** Linear regression	**Pollutant:** $PM_{2.5}$ **Mean (SD):** 11.5 (6.4) **Range:** 1.1–40.0 **Monitoring Stations:** 1 **Copollutant:** Sulfates; BC; Ultrafine particle counts	**PM Increment:** IQR (value not given) **Percent change (95% CI):** $PM_{2.5}$ 6-day moving avg Nitroglycerin-mediated reactivity: -7.6% (95% CI: 12.8% to -2.1%) **Notes:** $PM_{2.5}$ was positively associated with nitroglycerin-mediated reactivity; an association was also reported with ultrafine particles. Effect estimates were larger in type II than type I diabetes. BC and sulfate increases were associated with decreased flow-mediated reactivity among those with diabetes. Although the largest associations were with the 6-day moving avg, similar patterns and quantitatively similar results appear in the other lags.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: O'Neill et al. (2007) **Period of Study:** May 1998–Dec 2002 **Location:** Boston, MA	**Outcome:** soluble intercellular adhesion molecule 1 (ICAM-1); vascular cell adhesion molecule 1 (VCAM-1); von Willebrand factor (vWF) **Mean Age:** 56.6 (10.6) **Study Design:** Cross-sectional **N:** 92 participants (type 2 diabetic patients) **Statistical Analyses:** Linear regression **Covariates:** Apparent temperature, season, age, race, sex, glycosylated hemoglobin, cholesterol, smoking history, BMI **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** SO_4^{2-} **Averaging Time:** 24 h (lagged moving averages of days 0 to 1, 2, 3, 4, and 5) **Mean (SD):** 3.0 (2.0); descriptive statistics represent entire study period **Percentiles:** IQR range: 2.2 **Range (Min, Max):** 0.5, 9.6 **Monitoring Stations:** 1 site **Copollutant:** $PM_{2.5}$, BC, SO_4^{2-}	**PM Increment:** IQR (specific to lag period) **Effect Estimate [Lower CI, Upper CI]:** % change per IQR of $PM_{2.5}$ **ICAM-1** All subjects Lag 0: 5.30 (-2.60, 13.83); 2 dma: 4.02 (-3.26, 11.85); 3 dma: 4.03 (-5.34, 14.34); 4 dma: -0.79 (-7.30, 6.18); 5 dma: 1.06 (-7.10, 9.93); 6 dma: 3.15 (-5.66, 12.78) **Subjects not known to be taking statins** Lag 0: 10.14 (0.44, 20.77); 2 dma: 9.39 (-1.28, 21.20); 3 dma: 10.93 (-2.23, 25.85); 4 dma: -0.24 (-9.66, 10.16); 5 dma: 4.03 (-8.66, 18.47); 6 dma: 5.66 (-7.52, 20.72) **Subjects who report smoking in the past (but not within 6 months)** Lag 0: -4.00 (-24.79, 22.52); 2 dma: -4.82 (-18.01, 10.48); 3 dma: -7.19 (-23.66, 12.83); 4 dma: -9.8 (-27.96, 12.97); 5 dma: -10.4 (-29.92, 14.44); 6 dma: -6.8 (-25.72, 17.03) **Subjects who did not report smoking in the past** Lag 0: 6.67 (-4.34, 18.94); 2 dma: 5.65 (-4.67, 17.10); 3 dma: 10.21 (-5.83, 28.99); 4 dma: 0.80 (-9.94, 12.83); 5 dma: 2.80 (-10.85, 18.54); 6 dma: 5.15 (-7.78, 19.89) **VCAM-1** All subjects Lag 0: -0.04 (-3.75, 3.80); 2 dma: 0.94 (-4.79, 7.01); 3 dma: -0.87 (-3.50, 1.82); 4 dma: 0.13 (-2.02, 2.34); 5 dma: -0.47 (-2.67, 1.78); 6 dma: -0.46 (-1.99, 1.09) **Subjects not known to be taking statins** Lag 0: -1.34 (-11.23, 9.66); 2 dma: -0.19 (-11.13, 12.09); 3 dma: -2.84 (-13.90, 9.64); 4 dma: 4.28 (-6.18, 15.90); 5 dma: -0.26 (-13.44, 14.93); 6 dma: -3.44 (-16.51, 11.67) **Subjects who report smoking in the past (but not within 6 months)** Lag 0: 0.07 (-23.40, 30.73); 2 dma: -5.62 (-20.77, 12.43); 3 dma: -26.92 (-33.31 to -19.91); 4 dma: -3.06 (-28.01,30.56); 5 dma: -6.42 (-30.75, 26.47); 6 dma: -6.46 (-28.55, 22.47) **Subjects who did not report smoking in the past** Lag 0: -3.28 (-12.66, 7.12); 2 dma: -3.17 (-11.75, 6.23); 3 dma: -9.67 (-22.07, 4.70); 4 dma: -5.51 (-14.28, 4.15); 5 dma: -12.17 (-22.05 to -1.05); 6 dma: -11.77 (-20.95 to -1.52) **vWF** (sulfate measures not available)
Reference: Park et al. (2008) **Period of Study:** Jan 1995–Jun 2005 **Location:** Greater Boston area, MA	**Outcome:** Total homocysteine (tHcy) **Mean Age:** 73.6 ± 6.9 yrs **Study Design:** Cross-sectional and longitudinal analyses performed **N:** 960 men **Statistical Analyses:** Generalized additive models (also hierarchical mixed-effects regression models to assess repeated measures of tHcy) **Covariates:** Model 1: season, age, long-term trend, apparent temperature; Model 2: further adjustment for BMI, systolic blood pressure, smoking status, pack years of cigarettes, alcohol consumption; Model 3: further adjustment for serum creatinine, plasma folate, vitamin B6, and vitamin B12 **Dose-response Investigated?** Modeled continuous covariates as penalized splines to determine if association with tHcy was linear **Statistical Package:** R software	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h (moving averages up to 7 days prior to blood collection) **Mean (SD):** 12.0 (6.6) **Median:** 10.6 **Range (Min, Max):** 2.0, 62.0 **Monitoring Stations:** 1 site **Copollutant:** $PM_{2.5}$ BC (r = 0.51) OC (r = 0.51) SO_4^{2-} (r = 0.85)	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** Estimated % change in tHcy per IQR increase in pollutant. Lag model Concurrent day. IQR: 7.66 Model 1: 1.32 (-0.83, 3.52); Model 2: 1.55 (-0.77, 3.91); Model 3: 1.57 (-0.38, 3.56) 1-day previous. IQR: 6.91 Model 1: -1.43 (-3.51, 0.69); Model 2: -1.41 (-3.53, 0.76); Model 3: -1.28 (-3.12, 0.60) 2-day moving avg. IQR: 6.47 Model 1: 0.04 (-2.13, 2.26); Model 2: -0.07 (-2.26, 2.17); Model 3: 0.25 (-1.69, 2.22) 3-day moving avg. IQR: 5.83 Model 1: -0.64 (-2.92, 1.69); Model 2: -0.74 (-3.04, 1.61); Model 3: -0.59 (-2.63, 1.49) 4-day moving avg. IQR: 5.21 Model 1: -0.63 (-2.94, 1.72); Model 2: -0.86 (-3.19, 1.52); Model 3: -0.73 (-2.78, 1.37) 5-day moving avg. IQR: 4.68 Model 1: -0.51 (-2.79, 1.83); Model 2: -0.82 (-3.13, 1.54); Model 3: -0.84 (-2.85, 1.22) 6-day moving avg.IQR: 4.50 Model 1: -0.91 (-3.32, 1.56); Model 2: -1.32 (-3.76, 1.17); Model 3: -1.44 (-3.58, 0.74) 7-day moving avg. IQR: 4.20 Model 1: -0.84 (-3.27, 1.64); Model 2: -1.19 (-3.64, 1.33); Model 3: -1.69 (-3.84, 0.51) Stratified analyses: No significant difference in effect of $PM_{2.5}$ among those with high and low levels of vitamins

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Park et al. (2008) **Period of Study:** Jan 1995–Jun 2005 **Location:** Greater Boston area, MA	**Outcome:** Total homocysteine (tHcy) **Mean Age:** 73.6 ± 6.9 yrs **Study Design:** Cross-sectional and longitudinal analyses performed **N:** 960 men **Statistical Analyses:** Generalized additive models (also hierarchical mixed-effects regression models to assess repeated measures of tHcy) **Covariates:** Model 1: season, age, long-term trend, apparent temperature; Model 2: further adjustment for BMI, systolic blood pressure, smoking status, pack years of cigarettes, alcohol consumption; Model 3: further adjustment for serum creatinine, plasma folate, vitamin B6, and vitamin B12 **Dose-response Investigated?** Modeled continuous covariates as penalized splines to determine if association with tHcy was linear **Statistical Package:** R software	**Pollutant:** OC **Averaging Time:** 24 h (moving averages up to 7 days prior to blood collection) **Mean (SD):** 3.5 (1.8) **Median:** 3.1 **Range (Min, Max):** 0.29, 11.8 **Monitoring Stations:** 1 site **Copollutant (correlation):** $PM_{2.5}$ (r = 0.51) BC (r = 0.51) OC SO_4^{2-} (r = 0.41)	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** Estimated % change in tHcy per IQR increase in pollutant. Lag model Concurrent day. IQR: NA Model 1: NA ; Model 2: NA; Model 3: NA 1-day previous. IQR: 2.00 Model 1: 2.12 (-0.98, 5.31); Model 2: 1.69 (-1.51, 5.00); Model 3: 1.87 (-0.81, 4.62) 2-day moving avg. IQR: 1.93 Model 1: -0.39 (-3.67, 3.01); Model 2: -0.88 (-4.26, 2.61); Model 3: 1.05 (-1.86, 4.06) 3-day moving avg. IQR: 1.68 Model 1: 0.53 (-2.66, 3.83); Model 2: 0.14 (-3.15, 3.54); Model 3: 1.32 (-1.44, 4.16) 4-day moving avg. IQR: 1.64 Model 1: 1.57 (-1.89, 5.15); Model 2: 1.42 (-2.14, 5.12); Model 3: 1.89 (-1.15, 5.03) 5-day moving avg, IQR: 1.60 Model 1: 2.27 (-1.49, 6.16); Model 2: 2.11 (-1.77, 6.15); Model 3: 2.12 (-1.29, 5.65) 6-day moving avg. IQR: 1.43 Model 1: 2.83 (-0.74, 6.52); Model 2: 2.78 (-0.90, 6.60); Model 3: 2.53 (-0.59, 5.74) 7-day moving avg. IQR: 1.23 Model 1: 2.75 (-0.41, 6.02); Model 2: 2.55 (-0.71, 5.92); Model 3: 2.55 (-0.21, 5.39) % change in tHcy per IQR increase in OC, 7-d avg. Among those with low B12: 5.23 (1.59, 9.01) nearly null associations among those with high levels
Reference: Park et al. (2005) **Period of Study:** November 2000–October 2003 **Location:** Greater Boston area, MA	**Outcome:** Change in HRV (SDNN, HF, LF, LFHFR) **Mean age:** 72.7 years **Study Design:** Cross-sectional **N:** 497 adult males living in the Greater Boston, MA area	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 4 h 24 h 48 h **Mean (SD):** 11.4 (8.0) **Range:** 6.45–62.9 **Copollutant:** O_3, Particle number count, BC, NO_2, SO_2, CO	**PM Increment:** 8 µg/m^3 **Percent change (95% CI):** 48h mean $PM_{2.5}$: 20.8% decrease in HF (95% CI: 4.6%, 34.2%) 18.6% increase in LFHFR (4.1%, 35.2%). **Notes:** Subjects were monitored during a 4-min rest period between 8 a.m. and 1 p.m. Modifying effects of hypertension, IHD, diabetes, and use of cardiac/antihypertensive medications also examined. Linear regression analyses. This subject group is from the VA Normative Aging Study. The 4-h averaging period was most strongly associated with HRV indices. The PM effect was robust in models including O_3. The HRV change per IQR increase in $PM_{2.5}$ were larger in subjects with hypertension (n = 335) IHD (n = 142), and diabetes (n = 72). In addition, those who did not use calcium-channel blockers had a greater decline in LF associated with each IQR increase in $PM_{2.5}$ than did those who did use calcium channel blockers. IQR increases in 48h mean BC concentration were also associated with adverse changes in HRV, suggesting traffic pollution may be particularly toxic.
Reference: Park et al. (2006b) **Period of Study:** November 2000–December 2004 **Location:** Greater Boston area, MA	**Outcome:** Change in HF **Study Design:** Cross-sectional **N: Statistical Analysis:** Linear regression models	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48 h **Mean (SD):** $PM_{2.5}$: 11.7 (7.8); Sulfates: 3.3 (3.3); BC: 0.92 (0.46) **Copollutant:** O_3	**PM Increment:** 10 µg/m^3 **Percent change (95% CI):** Wild-type HFE genotype: 31.7% (95% CI: 10.3, 48.1) Among those with either of the two HFE variants, there was no association between 48h $PM_{2.5}$ and HF (shown in a graph, ~10% non-significant increase). **Notes:** Normative Aging Study. Examining association between PM and HF among those with and without the wild-type HFE genotype.
Reference: Pekkanen et al. (2002) **Period of Study:** Winter 1998 to 1999 **Location:** Helsinki, Finland	**Outcome**: ST Segment Depression (>0.1mV) **Study Design**: Panel of ULTRA Study participants **N**: 45 Subjects, n = 342 biweekly submaximal exercise tests, 72 exercise induced ST Segment Depressions **Statistical Analysis:** Logistic regression / GAM	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h Median: 10.6; IQR: 7.9 **Pollutant:** PM_1 Median: 7.0; IQR: 5.6 **Pollutant:** ACP (100 to 1000nm) (n/cm^3) **Median:** 1200; IQR: 760 **Copollutant:** NO_2, CO, $PM_{10-2.5}$, ultrafine	**PM Increment:** IQR **Effect Estimate(s):** ACP: OR = 3.29 (1.57, 6.92), lag 2 PM1: OR = 4.56 (1.73, 12.03), lag 2 $PM_{2.5}$: OR = 2.84 (1.42, 5.66), lag 2 **Notes**: The effect was strongest for ACP and $PM_{2.5}$, which in two pollutant models appeared independent. Increases in NO_2 and CO were also associated with increased risk of ST segment depression, but not with coarse particles.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Park et al. (2008) **Period of Study:** Jan 1995–Jun 2005 **Location:** Greater Boston area, MA	**Outcome:** Total homocysteine (tHcy) **Mean Age:** 73.6 ± 6.9 yrs **Study Design:** Cross-sectional and longitudinal analyses performed **N:** 960 men **Statistical Analyses:** Generalized additive models (also hierarchical mixed-effects regression models to assess repeated measures of tHcy) **Covariates:** Model 1: season, age, long-term trend, apparent temperature; Model 2: further adjustment for BMI, systolic blood pressure, smoking status, pack years of cigarettes, alcohol consumption; Model 3: further adjustment for serum creatinine, plasma folate, vitamin B6, and vitamin B12 **Dose-response Investigated?** Modeled continuous covariates as penalized splines to determine if association with tHcy was linear **Statistical Package:** R software	**Pollutant:** SO_4^2 **Averaging Time:** 24 h (moving averages up to 7 days prior to blood collection) **Mean (SD):** 3.2 (3.0) **Median:** 2.4 **Range (Min, Max):** 0.39, 29.0 **Monitoring Stations:** 1 site **Copollutant (correlation):** $PM_{2.5}$ (r = 0.85) BC (r = 0.50) OC (r = 0.41) SO_4^{2-}	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** Estimated % change in tHcy per IQR increase in pollutant. Lag model Concurrent day: IQR: NA Model 1: NA; Model 2: NA; Model 3: NA 1-day previous: IQR: 2.61 Model 1: 0.91 (-0.77, 2.62); Model 2: 0.99 (-0.94, 2.95); Model 3: 0.91 (-0.72, 2.57) 2-day moving avg: IQR: 2.10 Model 1: -0.25 (-2.07, 1.60); Model 2: -0.29 (-2.35, 1.82); Model 3: 0.05 (-1.74, 1.86) 3-day moving avg: IQR: 1.73 Model 1: -0.15 (-1.97, 1.69); Model 2: -0.17 (-2.23, 1.93); Model 3: -0.01 (-1.78, 1.80) 4-day moving avg: IQR: 1.64 Model 1: -0.69 (-2.74, 1.41); Model 2: -0.60 (-2.95, 1.81); Model 3: -0.58 (-2.63, 1.51) 5-day moving avg: IQR: 1.60 Model 1: -1.14 (-3.53, 1.30); Model 2: -0.90 (-3.64, 1.92); Model 3: -1.09 (-3.48, 1.36) 6-day moving avg;' IQR: 1.40 Model 1: 0.00 (-2.39, 2.44); Model 2: 0.36 (-2.36, 3.16); Model 3: 0.41 (-2.01, 2.89) 7-day moving avg; IQR: 1.30 Model 1: -0.16 (-2.51, 2.24); Model 2: 0.30 (-2.37, 3.04); Model 3: 0.07 (-2.25, 2.43) Stratified analyses: No significant difference in effect of SO_4^{2-} among those with high and low levels of vitamins
Reference: Pope et al. (2004) **Period of Study:** Winter 1999–2000 (in Wasatch Front, UT). Summer 2000 (in Hawthorne, UT). Winter 2000–2001 (in Bountiful, UT and Lindon, UT) **Location:** Utah: Wasatch Front, Hawthorne, Bountiful, and Lindon	**Outcome:** Change in autonomic function (measured by changes in HRV), C-reative protein (CRP), blood cell counts, platelets, and blood viscosity associated with short-term changes in $PM_{2.5}$ **Age Groups:** Elderly (specific age range not given) **Study Design:** Panel study **N:** 88 elderly subjects **Statistical Analysis:** Linear regression **Season:** Winter, summer **Dose-response Investigated?**No	**Pollutant:** $PM_{2.5}$ (TEOM) **Averaging Time:** 24 h **Mean (SD):** 18.9 (13.4) **Copollutant:** None	**PM Increment:** 100 µg/m^3 **Effect Estimate:** Each 100 µg/m^3 increase associated with: -35 (SE = 8) msec decline in SDNN 0.81 (SE 0.17) mg/dL increase in CRP 0.31 (SE 9.34) k/µL increase in platelets 0.07 (SE 0.21) cP increase in blood viscosity **Notes:** The study observed small but statistically significant adverse associations between daily mean $PM_{2.5}$ and HRV and C-reactive protein (CRP). The authors point out, however, that most of the variability in the temporal deviation of these physiological endpoints was not explained by $PM_{2.5}$. These observations therefore suggest that $PM_{2.5}$ may be one of multiple factors that influence HRV and CRP.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Pope et al. (2004) **Period of Study:** 1999-2001 **Location:** Wasatch Front, Utah	**Outcome:** Heart rate variability (HRV); C-reactive protein (CRP); blood cell counts, whole blood viscosity **Age Groups:** 54-89 yrs **Study Design:** Panel study **N:** 88 participants **Statistical Analyses:** Linear regression **Covariates:** Subject-specific fiexed effects; interactive spline smooths for temp, RH (partial control for H) **Season:** Temperature as covariate **Dose-response Investigated?** Yes, also assessed PM by including cubic smoothing splines with 3 df **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 23.7 (20.2) **Range (Min, Max):** 1.7, 74.0 **Monitoring Stations:** NR **Copollutant:** None	**PM Increment:** 100 µg/m^3 **Effect Estimate [Lower CI, Upper CI]: Regression coefficients (SE) for associations with concurrent day pollutant:** Mean H: -4.49 (1.73) SDNN: -34.94 (8.32) SDANN: -18.98 (8.67) r-MSSD: -42.25 (10.90) CRP: 0.81 (0.18) Whole blood viscosity: 0.07 (0.21) WBC: -0.07 (0.38) Granulocytes: 0.02 (0.37) Lymphocytes: -0.07 (0.14) Monocytes: 0.12 (0.04) Basophils: -0.01 (0.01) Eosinophils: -0.01 (0.02) RBC: 0.03 (0.06) Platelets: 0.31 (9.34)
Reference: Rich et al. (2005) **Period of Study:** July 1995–July 2002 **Location:** Eastern Massachusetts, USA	**Outcome:** Confirmed ventricular arrhythmias **Study Design:** Case-crossover (time-stratified control selection) **N:** 203 patients with implantable cardioverter defibrillators **Statistical Analysis:** Conditional logistic regression	**Pollutant:** $PM_{2.5}$ (TEOM) **Averaging Time:** 1-h avg 24-h avg **Median (IQR):** 1-h avg: Median = 9.2 µg/m^3 24-h avg: Median = 9.8 µg/m^3 IQr = 7.8 **Copollutant:** O_3, BC, CO, NO_2, SO_2	**PM Increment:** 7.8 µg/m^3 **Effect Estimate:** For mean $PM_{2.5}$ in the 24 h before ventricular arrhythmia: OR = 1.19; 95% CI: 1.02, 1.38 **Notes:** 794 ventricular arrhythmias among 84 subjects. **Lag h:** 0-2, 0-6, 0-23, 0-47
Reference: Rich et al. (2006a) **Period of Study:** July 1995–July 2002 **Location:** Eastern Massachusetts, USA	**Outcome:** Confirmed episodes of paroxysmal atrial fibrillation **Study Design:** Case-crossover (time-stratified control selection) **N:** 203 patients with implantable cardioverter defibrillators **Statistical Analysis:** Conditional logistic regression	**Pollutant:** $PM_{2.5}$ (TEOM) **Averaging Time:** 1 h avg 24-h avg **Median (IQR):** 1-h avg: Median = 9.2 µg/m^3 24-h avg: Median = 9.8 µg/m^3 IQr = 7.8 **Copollutant:** O_3, BC, CO, NO_2, SO_2	**PM Increment:** 9.4 µg/m^3 **Effect Estimate:** 0-h lag: OR 1.41 (0.82, 2.42) **Notes:** 91 paroxysmal atrial fibrillation (PAF) episodes among 29 subjects. **Lag h:** 0, 0 - 23 Positive, but not significant increases in the relative odds of PAF associated with $PM_{2.5}$ concentrations in the same h and 24-h before PAF episode onset. Authors note reduced statistical power for $PM_{2.5}$ analyses due to missing data.
Reference: Rich et al. (2006b) **Period of Study:** May 2001–December 2002 **Location:** St. Louis, MO metropolitan area	**Outcome:** Confirmed ventricular arrhythmia **Study Design:** Case-crossover design (time-stratified control selection) **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ (CAMM) **Averaging Time:** 24 h **Median (IQR):** 16.2 µg/m^3 (IQr = 9.7) **Copollutant:** NO_2, SO_2, CO, O_3, EC, OC	**PM Increment:** 9.7 µg/m^3 (IQR) **Effect Estimate:** OR ($PM_{2.5}$) = 0.95 (95% CI: 0.72, 1.27) OR (SO_2) = OR = 1.24 (95% CI: 1.07, 1.44) **Notes:** 139 confirmed ventricular arrhythmia epidsodes among 56 subjects. Lags: 0-2h, 0-6h, 0-11h, 0-23h, 0-47h Authors did not find increased relative odds of VA associated with each IQR increase in 24-h mean $PM_{2.5}$, but did find non-significantly increased relative odds of VA associated with 24-h EC. Shorter and longer lag times' relative odds estimates provided no evidence of immediate ventricular arrhythmic effects of air pollution.
Reference: Rich et al. (2004) **Period of Study:** February–December 2000 **Location:** Vancouver, British Columbia, Canada	**Outcome:** ICD discharges (as a proxy for VT/VF) **Age Groups:** 15-85 years **Study Design:** Case-crossover design (ambidirectional control selection ± 7 days) **N:** 34 patients with implantable cardioverter defibrillators **Statistical Analysis:** Conditional logistic regression **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ (Partisol) **Averaging Time:** 1 h **Mean (SD), IQR:** Mean:: 8.2 µg/m^3 (SD = 10.7) IQr = 5.2 **Copollutant:** O_3, EC, OC, SO_4^{2-}, CO, NO_2, SO_2, PM_{10} PM_{10}: Mean:: 13.3 µg/m^3 (SD = 4.9) IQr = 7.4	**PM Increment: Effect Estimate:** Odds ratios were less than 1.0 at all lags (0, 1, 2, 3) for $PM_{2.5}$. No consistent association between any of the air pollutants and implantable cardioverter defibrillators discharges. **Notes:** Same study as Vedal et al. (2004), except Rich (2004) used data from a shorter time period so as to estimate relative odds of ICD discharge associated with acute increases in more pollutants than Vedal (2004).

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Riediker et al. (2004) **Period of Study:** Fall 2001 **Location:** Wake County, North Carolina	**Outcome:** Heart rate variability (measured 10 h after shift): mean cycle lenth of normal R-R intervals (MCL), the standard deviation of normal R-R intervals (SDNN), and percentage of normal R-R interval differences greater than 50 msec (PNN50), low frequency (0.04-0.15Hz), high frequency (0.15-0.40Hz), the ratio of low to high frequency. **Blood analysis (measured 15 h after shift)**: Uric acid, blood urea nitrogen, gamma glutamyl transpeptidase, white blood cell count, red blood cell count, hematocrit, hemoglobin, mean red blood cell volume (MCV), neutrophils (count and %), lymphocytes (count and %), C-reactive protein, plasminogen, plasminogen activator inhibitor type 1, von Willebrand factor (vWF), endovthzelin-1, protein C, and interleukin-6 **Age Groups:** 23-30 yrs **Study Design:** Panel **N:** 9 healthy male troopers, repeated measures (36 person-days) **Statistical Analyses:** Mixed effects regression models (principal factor analysis for classification of exposure) **Covariates:** Potential confounders: temperature, relative humidity, number of law-enforcement activities during the shift and the avg speed during the shift; controlling had no effect on effect estimates for "crustal" and "speed-change" factors; however, confounder inclusion in the "speed change" and blood urea nitrogen and vWF reduced the effect estimate and the CI included zero **Season:** Only 1 season included **Dose-response Investigated?** No **Statistical Package:** S-Plus 6.1	**Pollutant:** In-vehicle $PM_{2.5}$ components identified with factor analysis (crustal material, wear of steel automotive components, gasoline combustion, speed-changing traffic with engine emissions and brake wear **Averaging Time:** Exposure assessed during 3pm to 12am workshifts **Mean:** $PM_{2.5mass}$ = 23.0 µg/m^3 **Monitoring Stations:** Per vehicle **Copollutant (correlation):** Correlation to $PM_{2.5}$Mass Benzene: r = 0.50 Aldehydes: r = 0.34 CO: r = 0.52 Aluminum: r = 0.58 Silicon: r = 0.66 Sulfur: r = 0.58 Calcium: r = 0.37 Titanium: r = 0.41 Chromium: r = 0.51 Iron: r = 0.71 Copper: r = 0.16 Selenium: r = 0.38 Tungsten: r = 0.37 PM_2 Lightscatter: r = 0.71	**PM Increment:** 1 SD change in source factor **Effect Estimate:** % change in the health outcome per 1 SD change in the "speed change" factor MCL: 7% HRV: 16% supraventricular ectopic beats: 39% % Neutrophils: 7% % lymphocytes: -10% red blood cell volume MCV: 1% vWF: 9% blood urea nitrogen: 7% protein C: -11% % change in the health outcome per 1 SD change in the "crustal" factor MCL: 3% serum uric acid concentrations: 5% **Note:** Results (including CIs) are reported in figures 2 & 3.
Reference: Riojas-Rodriguez et al. (2006) **Period of Study:** December 2001–April 2002 **Location:** Mexico City metropolitan area	**Outcome:** Heart rate variability (5-minute periods) **Study Design:** Panel study **N:** 30 patients from the outpatient clinic of the National Institute of Cardiology of Mexico, where each subject had existing ischemic heart disease. **Statistical Analysis:** Mixed models	**Pollutant:** $PM_{2.5}$ (nephelometry) **Averaging Time:** 5 minutes **Mean (SD), Range:** 46.8 µg/m^3 (SD = 1.82) **Range:** 0–483 µg/m^3 **Copollutant:** CO	**PM Increment:** 10 µg/m^3 **Effect Estimate:** Each 20 µg/m^3 increase in 5 minute $PM_{2.5}$ was associated with a: -0.008 decrease in the ln(HF)(95% CI: -0.015, 0.0004 **Notes:** Population of subjects with known ischemic heart disease (25 men and 5 women who had at least 1 prior MI [not in last 6 months]) Each 10 µg/m^3 increase in 5 minute mean $PM_{2.5}$ was associated with non-significantly decreased HF, and with similar, but smaller changes in LF and VLF.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Romieu et al. (2005) **Period of Study:** 2000–2001 **Location:** Mexico City, Mexico	**Outcome:** Heart rate variability (HF, LF, VLF, PNN50, SDNN, r-MSSD) **Age Groups:** >60 years of age **Study Design:** Double blind randomized controlled trial **N:** 50 elderly residents of a Mexico City nursing home	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Copollutant:** O_3, NO_2, SO_2, PM_{10}	**PM Increment:** 8 µg/m^3 **Effect Estimate:** In the group receiving the fish oil supplement, each 8 µg/m^3 change in 24 h mean total exposure $PM_{2.5}$ was associated with a: a) 54% reduction (95% CI: -72% to -24%) in HF (log transformed) in the pre-supplementation phase b) 7% reduction (95% CI: -20%, 7%) in the supplementation phase. Changes in other HRV parameters were also smaller in the supplementation phase. In the group receiving soy oil supplementation, the % reduction in HF was also smaller in the supplementation phase, but the differences were smaller and not statistically significant. **Notes:** Study of the effect of omega-3-fatty acid supplementation (2 g/day of fish oil versus 2 g/day of soy oil) to mitigate the effect of ambient $PM_{2.5}$ on HRV. Subjects had no cardiac arrhythmias, cardiac pacemakers, allergies to omega-3 fatty acids or fish, treatment with oral anticoagulants, or history of bleeding diathesis. $PM_{2.5}$ was measured and estimated indoors, outdoors, and with regards to total exposure (the same as Holguin et al. (2003)).
Reference: Romieu et al. (2008) **Period of Study:** Sep 2001–Apr 2002 **Location:** Mexico City, Mexico	**Outcome:** Copper/zinc superoxide dismutase activity (Cu/Zn SOD); lipoperoxidation (LPO); reduced glutathione (GSH) **Age Groups:** 60-96 yrs **Study Design:** Intervention (randomly assigned fish oil or soy oil) **N:** 52 participants **Statistical Analyses:** Linear mixed models **Covariates:** Time **Dose-response Investigated?** Assessed possible nonlinearity using generalized additive mixed models with p-splines **Statistical Package:** STATA v8.2 and SAS v9.1	**Pollutant:** $PM_{2.5}$ (indoor) **Averaging Time:** 24 h (same day) **Mean (SD):** 38.7 (14.7) **Percentiles:** 25th: 30.62 50th: 35.11 75th: 41.10 **Range (Min, Max):** 14.8, 70.9 **Monitoring Stations:** Indoor measured inside nursing home **Copollutant:** O_3	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** **Regression coefficient (SE; p-value):** Cu/Zn SOD: -0.05 (0.02; 0.001) LPO (square root transformed): 0.08 (0.09; 0.381) GSH (log-transformed; quadratic term for PM): -0.05 (0.01; 0.002) Regression coefficient (SE; p-value) by supplemention groups (same transformations as above): Cu/Zn SOD Soy Oil: -0.06 (0.02; <0.001) Fish Oil: * 0.04 (0.02; 0.009) LPO Soy Oil: -0.02 (0.14; 0.904) Fish Oil: * 0.16 (0.07; 0.024) GSH Soy Oil: -0.03 (0.04; 0.406) Fish Oil: -0.09 (0.04; 0.017) *Quadratic term for PM
Reference: Ruckerl et al. (2007b) **Period of Study:** May 2003–Jul 2004 **Location:** Athens, Augsburg, Barcelona, Helsinki, Rome, and Stockholm	**Outcome:** Interleukin-6 (IL-6), fibrinogen, C-reactive protein (CRP) **Age Groups:** 35-80 yrs **Study Design:** Repeated measures / longitudinal **N:** 1003 MI survivors **Statistical Analyses:** Mixed-effect models **Covariates:** City-specific confounders (age, sex, BMI); long-term time trend and apparent temperature; RH, time of day, day of week included if adjustment improved model fit **Season:** Long-term time trend **Dose-response Investigated?** Used p-splines to allow for nonparametric exposure-response functions **Statistical Package:** SAS v9.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Hourly and 24-h (lag 0-4, mean of lags 0-4, mean of lags 0-1, mean of lags2-3, means of lags 0-3) **Mean (SD):** Presented by city only **Monitoring Stations:** Central monitoring sites in each city **Copollutant:** SO_2; O_3; NO; NO_2	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change in mean blood markers per increase in IQR of air pollutant. IL-6 Lag (IQR): % change in GM (95%CI); Lag 0 (11.0): 0.46 (-0.89, 1.83); Lag 1 (11.0): -0.39 (-1.69, 0.93); Lag 2 (11.0): -0.23 (-1.53, 1.07); 5-d avg (8.6): 0.05 (-1.37, 1.50) Fibrinogen Lag (IQR): % change in AM (95%CI); Lag 0 (11.0): 0.05 (-0.48, 0.58); Lag 1 (11.0): 0.17 (-0.35, 0.69); Lag 2 (11.0): 0.20 (-0.32, 0.71); 5-d avg (8.6): 0.38 (-0.21, 0.96) CRP Lag (IQR): % change in GM (95%CI); Lag 0 (11.0): 0.11 (-1.95, 2.21); Lag 1 (11.0): -0.06 (-1.98, 1.90); Lag 2 (11.0): 0.11 (-1.80, 2.06); 5-d avg (8.6): -0.13 (-2.15, 1.92)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ruckerl et al. (2006) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** C-reactive protein (CRP); serum amyloid A (SAA); E-selectin; von Willebrand Factor (vWF); intercellular adhesion molecule-1 (ICAM-1); fibrinogen; Factor VII; prothrombin fragment 1+2; D-dimer **Age Groups:** 50+ **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear and logistic regression models **Covariates:** Models adjusted for different factors based on health endpoint; CRP: RH, temperature, trend, ID; ICAM-1: temperature, trend, ID; vWF: air pressure, RH, temperature, trend, ID; FVII: air pressure, RH, temperature, trend, ID, weekday **Season:** Time trend as covariate **Dose-response Investigated?** Sensitivity analyses examined nonlinear exposure-response functions **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 20.0 (15.0) **Percentiles:** 2th5: 9.7 50th: 14.9 75th: 26.1 **Range (Min, Max):** 2.6, 83.7 **Monitoring Stations:** 1 site **Copollutant:** UFPs (ultrafine particles) AP (accumulation mode particles) $PM_{2.5}$ PM_{10} OC (organic carbon) EC (elemental carbon) NO_2 CO	**PM Increment:** IQR (16.4; 5-d avg: 12.2) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as OR (95%CI) for an increase in the blood marker above the 90th percentile per increase in IQR air pollutant. **CRP** Time before draw: 0 to 23 h: 1.1 (0.7, 1.8); 24 to 47 h: 1.5 (0.9, 2.5); 48 to 71 h: 1.2 (0.8, 1.9); 5-d mean: 1.4 (0.9, 2.3) **ICAM-1** Time before draw: 0 to 23 h: 0.7 (0.4, 0.9); 24 to 47 h: 1.3 (0.8, 1.8); 48 to 71 h: 1.8 (1.2, 2.7); 5-d mean: 1.1 (0.8, 1.5) Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **vWF** Time before draw: 0 to 23 h: 3.9 (-0.3, 8.1); 24 to 47 h: 3.1 (-1.6, 7.8); 48 to 71 h: 3.6 (-1.1, 8.3); 5-d mean: 5.6 (0.5, 10.8) **FVII** Time before draw: 0 to 23 h: -2.5 (-6.2, 1.4); 24 to 47 h: -2.8 (-6.1, 0.6); 48 to 71 h: -2.3 (-5.0, 0.6); 5-d mean: -3.5 (-6.4 to -0.4) **Note:** Summary of results presented in figures. SAA results indicate increase in association with PM (not as strong and consistent as with CRP); no association observered between E-selectin and PM; an increase in prothrombin fragment 1+2 was consistently observed, particularly with lag 4; fibrinogen results revealed few significant associations, potentially due to chance; D-dimer results revealed null associations in linear and logistic analyses
Reference: Ruckerl et al. (2006) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** C-reactive protein (CRP); serum amyloid A (SAA); E-selectin; von Willebrand Factor (vWF); intercellular adhesion molecule-1 (ICAM-1); fibrinogen; Factor VII; prothrombin fragment 1+2; D-dimer **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear and logistic regression models **Covariates:** Models adjusted for different factors based on health endpoint; CRP: RH, temperature, trend, ID; ICAM-1: temperature, trend, ID; vWF: air pressure, RH, temperature, trend, ID; FVII: air pressure, RH, temperature, trend, ID, weekday **Season:** Time trend as covariate **Dose-response Investigated?** Sensitivity analyses examined nonlinear exposure-response functions **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** EC **Averaging Time:** 24 h **Mean (SD):** 2.6 (2.4) **Percentiles:** 25th: 1.0 50th: 1.8 75th: 3.2 **Range (Min, Max):** 0.2, 12.4 **Monitoring Stations:** 1 site **Copollutant:** UFPs (ultrafine particles) AP (accumulation mode particles) $PM_{2.5}$ PM_{10} OC EC NO_2 CO	**PM Increment:** IQR (2.3; 5-d avg: 1.8) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as OR (95%CI) for an increase in the blood marker above the 90th percentile per increase in IQR air pollutant. **CRP** Time before draw: 0 to 23 h: 1.2 (0.7, 2.0); 24 to 47 h: 1.3 (0.7, 2.4); 48 to 71 h: 1.6 (0.9, 2.7); 5-d mean: 1.2 (0.7, 2.1) **ICAM-1** Time before draw: 0 to 23 h: 1.0 (0.7, 1.6); 24 to 47 h: 2.6 (1.7, 3.8); 48 to 71 h: 4.0 (2.5, 6.1); 5-d mean: 2.2 (1.4, 3.3) Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **vWF** Time before draw: 0 to 23 h: 5.0 (0.0, 10.1); 24 to 47 h: 7.6 (1.4, 13.7); 48 to 71 h: 1.1 (-5.2, 7.4); 5-d mean: 5.7 (-0.5, 12.0) **FVII** Time before draw: 0 to 23 h: -5.7 (-10.5 to -0.7); 24 to 47 h: -6.9 (-11.2 to -2.3); 48 to 71 h: -4.2 (-8.4, 0.2); 5-d mean: -6.0 (-10.5 to -1.2) **Note:** Summary of results presented in figures. SAA results indicate increase in association with PM (not as strong and consistent as with CRP); no association observered between E-selectin and PM; an increase in prothrombin fragment 1+2 was consistently observed, particularly with lag 4; fibrinogen results revealed few significant associations, potentially due to chance; D-dimer results revealed null associations in linear and logistic analyses

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ruckerl et al. (2006) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome (ICD9 and ICD10):** C-reactive protein (CRP); serum amyloid A (SAA); E-selectin; von Willebrand Factor (vWF); intercellular adhesion molecule-1 (ICAM-1); fibrinogen; Factor VII; prothrombin fragment 1+2; D-dimer **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear and logistic regression models **Covariates:** Models adjusted for different factors based on health endpoint; CRP: RH, temperature, trend, ID; ICAM-1: temperature, trend, ID; vWF: air pressure, RH, temperature, trend, ID; FVII: air pressure, RH, temperature, trend, ID, weekday **Season:** Time trend as covariate **Dose-response Investigated?** Sensitivity analyses examined nonlinear exposure-response functions **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** OC **Averaging Time:** 24 h **Mean (SD):** 1.5 (0.6) **Percentiles:** 25th: 1.1 50th: 1.4 75th: 1.8 **Range (Min, Max):** 0.3, 3.4 **Monitoring Stations:** 1 site **Copollutant:** UFPs AP $PM_{2.5}$ PM_{10} OC EC NO_2 CO	**PM Increment:** IQR (0.7; 5-d avg: 0.5) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as OR (95%CI) for an increase in the blood marker above the 90th percentile per increase in IQR air pollutant. **CRP** Time before draw: 0 to 23 h: 1.2 (0.7, 1.9); 24 to 47 h: 1.3 (0.8, 2.1); 48 to 71 h: 1.4 (0.8, 2.4); 5-d mean: 1.2 (0.7, 1.8) **ICAM-1** Time before draw: 0 to 23 h: 0.9 (0.6, 1.3); 24 to 47 h: 2.0 (1.3, 3.2); 48 to 71 h: 3.0 (1.8, 4.8); 5-d mean: 1.3 (0.8, 2.0) Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **vWF** Time before draw: 0 to 23 h: 5.5 (0.2, 10.8); 24 to 47 h: 8.0 (2.1, 13.9); 48 to 71 h: 3.5 (-2.6, 9.6); 5-d mean: 7.4 (2.0, 12.8) **FVII** Time before draw: 0 to 23 h: -6.1 (-10.6 to -1.4); 24 to 47 h: -7.2 (-11.4 to -2.8); 48 to 71 h: -3.8 (-8.2, 0.9); 5-d mean: -5.6 (-9.8 to -1.1) **Note:** Summary of results presented in figures. SAA results indicate increase in association with PM (not as strong and consistent as with CRP); no association observered between E-selectin and PM; an increase in prothrombin fragment 1+2 was consistently observed, particularly with lag 4; fibrinogen results revealed few significant associations, potentially due to chance; D-dimer results revealed null associations in linear and logistic analyses
Reference: Ruckerl et al. (2007a) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** Soluble CD40 ligand (sCD40L), platelets, leukocytes, erythrocytes, hemoglobin **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear regression models **Covariates:** Long-term time trend, weekday of the visit, temperature, RH, barometric pressure **Season:** Time trend as covariate **Dose-response Investigated?** No **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 20.0 (15.0) **Percentiles:** 25th: 9.7 50th: 14.9 75th: 26.1 **Range (Min, Max):** 2.6, 83.7 **Monitoring Stations:** 1 site **Copollutants:** UFPs AP PM_{10} NO	**PM Increment:** IQR (16.4; 5-d avg: 12.2) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **sCD40L, % change GM (pg/mL)** lag0: 1.5 (-4.0, 7.3); Lag1: 0.2 (-5.4, 6.2); Lag2: -2.6 (-8.0, 3.1); Lag3: 0.5 (-3.9, 5.0); 5-d mean: 0.2 (-5.4, 6.2) **Platelets, % change mean (10^3/µl)** Lag0: -0.6 (-1.9, 0.7); Lag1: 0.1 (-1.3, 1.5); Lag2: 0.5 (-0.9, 1.9); Lag3: 0.2 (-1.1, 1.5); 5-d mean: -0.4 (-1.9, 1.2) **Leukocytes, % change in mean (10^3/µl)** Lag0: -1.6 (-3.2, 0.0); Lag1: -0.4 (-2.2, 1.4); Lag2: -0.2 (-2.1, 1.7); Lag3: -0.8 (-2.4, 0.7); 5-d mean: -1.6 (-3.5, 0.3) **Erythrocytes, % change mean (10^6/µl)** Lag0: -0.1 (-0.5, 0.3); Lag1: -0.3 (-0.7, 0.2); Lag2: -0.4 (-0.8, 0.0); Lag3: -0.2 (-0.5, 0.1); 5-d mean: -0.4 (-0.8, 0.0) **Hemoglobin, % change mean (g/dl)** Lag0: 0.0 (-0.6, 0.5); Lag1: -0.2 (-0.8, 0.3); Lag2: -0.5 (-1.1, 0.0); Lag3: -0.2 (-0.7, 0.2); 5-d mean: -0.5 (-1.0, 0.1)
Reference: Sarnat et al. (2006) **Period of Study:** summer and Autumn 2000 **Location:** Steubenville, OH	**Outcome:** Supraventricular ectopy (SVE) or ventricular ectopy (VE) **N:** 32 nonsmoking older adults **Statistical Analysis:** Logistic mixed effects regression **Season:** Summer, Autumn **Dose-response Investigated?**No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 5 days **Median (IQR):** $PM_{2.5}$: Median: 19.0 µg/m^3 IQr = 10.0 Sulfate: Median: 6.1. IQR: 4.2 EC: Median: 0.9. IQR: 0.5 **Copollutants:** O_3, NO_2, SO_2	**PM Increment:** IQR **Effect Estimate:** $PM_{2.5}$: SVE: OR = 1.42 (95% CI: 0.99, 2.04); VE: OR = 1.02 (95% CI: 0.63-1.65) Sulfate: SVE: OR = 1.70 (95% CI: 1.12, 2.57); VE: OR = 1.08 (95% CI: 0.65, 1.80) EC: SVE: OR = 1.15 (95% CI: 0.73, 1.81); VE: OR = 1.00 (95% CI: 0.57, 1.75) **Notes:** Longitudinal study of 32 nonsmoking older adults who had ECG measurements made every week for 24 weeks. PM measured within 1 mile of subjects' residences, and central site pollutant measurements were also made.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Schwartz et al. (2005b) **Period of Study:** 12 weeks during the summer of 1999 **Location:** Boston, MA	**Outcome:** Heart rate variability (HRV), ((SDNN, r-MSSD, PNN50, LFHFR) **Age Groups:** 61–89 years **Study Design:** Panel study **N:** 28 elderly subjects **Statistical Analysis:** Mixed models. To examine heterogeneity of effects, hierarchical modeling was used. **Season:** Summer **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h; 24 h **Median:** 24-hs: 10 µg/m^3 **Monitoring Stations:** 1 **Copollutant:** BC, O_3, CO, SO_2, NO_2	**PM Increment:** IQR (not given) **Effect Estimate:** 24 h 2.6 ms decrease in SDNN (95% CI: 0.8 to -6.0) 10.1 ms decrease in r-MSSD (95% CI: -2.8 to -16.9). 1 h 3.4 ms decrease in SDNN (95% CI: 0.6 to -7.3) 7.4 ms decrease in r-MSSD (95% CI: 1.6 to -15.5). **Notes:** Various log-transformed HRV parameters were measured for 30 minutes once a week. The random effects model indicated that the negative effect of BC on HRV was not restricted to a few subjects. Same study population as Gold et al. (2005). Boston Elders Study For each pollutant/averaging time, similarly sized changes were observed for PNN50 (%) and LFHFR.
Reference: Schwartz et al. (2005a) **Period of Study:** 2000 **Location:** Boston, Massachusetts	**Outcome:** HF (high frequency component of heart rate variability) **Study Design:** Cross-sectional **N:** 497 subjects **Statistical Analysis:** Linear regression, controlling for covariates	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48 h **Mean (SD):** 11.4 (8.0) **Copollutant:** None	**PM Increment:** 10 µg/m^3 **Effect Estimate:** 34% decrease in HF (95% CI: -9% to -52%) in subjects without the GSTM1 allele. In subjects with the allele, no effect was noted. Similar findings for obese subjects and those with high neutrophil counts. **Notes:** Study population: Normative Aging Study. Effects of $PM_{2.5}$ appear to be mediated by ROS.
Reference: Sorensen et al. (2005) **Period of Study:** Nov 1999–Aug 2000 **Location:** Copenhagen, Denmark	**Outcome:** 7-Hydro-8-Oxo-2'-Deoxyguanosine (8-oxodG) (measured in lymphocytes and urine) **Age Groups:** 20-33 yrs **Study Design:** Panel (repeated measures) **N:** 49 students living and studying in central Copenhagen; 50 students examined each season (66 subjects total; 32 participated in each season; total of 98 measurements) **Statistical Analyses:** Mixed models repeated measures **Covariates:** $PM_{2.5}$, season, subject (random factor) **Dose-response Investigated?** No **Statistical Package:** SAS v8e	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48 h **Mean (SD):** Autumn: 20.7 Summer: 12.6 **Percentiles:** IQR Autumn: 13.1-27.7 IQR summer: 9.4-24.3 **Range (Min, Max):** NR **Monitoring Stations:** NA (personal assessment) **Copollutant (correlation):** Spearman correlations with $PM_{2.5}$ mass: chromium (r = 0.22) copper (r = 0.33) iron (r = 0.29) vanadium (p>0.5) nickel (p>0.5) platinum (p>0.5)	**PM Increment:** see below **Effect Estimate [Lower CI, Upper CI]:** Association between 8-oxodG in lymphocytes and personal exposure to transition metals in $PM_{2.5}$. % increase in 8-oxodG per increase in metal concentration indicated Vanadium: 1.9% per 1 µg/L (0.6, 3.3) Chromium: 2.2% per 1 µg/L (0.8, 3.5) Platinum: 6.1% per 1 ng/L (-0.6, 13.2) Nickel: 0.8% per 10 µg/L (-2.1, 3.7) Copper: -0.8% per 10 µg/L (-2.7, 1.0) Iron: 0.6% per 10 µg/L (-1.4, 2.6) **Note:** $PM_{2.5}$ mass was independently associated with 8-oxodG in 5 of 6 transition metal models (p<0.02 in models with vanadium, chromium, nickel, copper, and iron; p = 0.07 in platinum model). No transition metals were associated with 8-oxodG measured in urine

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Sorensen et al. (2003) **Period of Study:** Nov 1999–Aug 2000 **Location:** Copenhagen, Denmark	**Outcome:** RBC count, hemoglobin, platelet count, fibrinogen, PLAAS (2-aminoadipic semialdehyde in plasma proteins), HBGGS (γ-glutamyl semialdehyde in hemoglobin), HBAAS (2-aminoadipic semialdehyde in hemoglobin), MDA (malondialdehyde) **Age Groups:** 20-33 yrs **Study Design:** Panel (repeated measures) **N:** 50 students living and studying in central Copenhagen; 50 students examined each season (68 subjects total; 31 participated in each season; total of 195 measurements) **Statistical Analyses:** Mixed model repeated-measures analysis **Covariates: S**eason, avg outdoor temperature, and sex **Season:** Repeated measures 4 times (once per season) **Dose-response Investigated?** No **Statistical Package:** SAS v8e	**Pollutant:** $PM_{2.5}$ (personal) **Averaging Time:** 48 h **Median:** 16.1 μg/m^3 **Percentiles:** Q25-Q75: 10.0-24.5 **Copollutant:** Urban background $PM_{2.5}$ Personal $PM_{2.5}$	**PM Increment:** 1 μg/m^3 **Effect Estimate [Lower CI, Upper CI]:** **Relationship between exposure and biomarkers** Estimate (p-value): Platelet count (x 10^6/g protein): 0.0008 (0.37) Fibrinogen (nmol/g protein): 0.0006 (0.69) PLAAS (pmol/mg protein): 0.0016 (0.061) HBGGS (pmol/mg protein): 0.0001 (0.94) HBAAS (pmol/mg protein): 0.0006 (0.64) **Increase (95%CI) in biomarkers per 10 μg/m^3 increase in $PM_{2.5}$** RBC Men: 0% (-1.6, 1.6) Women: 2.3% (0.5, 4.1) Hemoglobin Men: 0.0% (-1.7, 1.5) Women: 2.6% (0.8, 4.5)
Reference: Sorensen et al. (2003) **Period of Study:** Nov 1999–Aug 2000 **Location:** Copenhagen, Denmark	**Outcome:** RBC count, hemoglobin, platelet count, fibrinogen, PLAAS (2-aminoadipic semialdehyde in plasma proteins), HBGGS (γ-glutamyl semialdehyde in hemoglobin), HBAAS (2-aminoadipic semialdehyde in hemoglobin), MDA (malondialdehyde) **Age Groups:** 20-33 yrs **Study Design:** Panel (repeated measures) **N:** 50 students living and studying in central Copenhagen; 50 students examined each season (68 subjects total; 31 participated in each season; total of 195 measurements) **Statistical Analyses:** Mixed model repeated-measures analysis **Covariates: S**eason, avg outdoor temperature, and sex **Season:** Repeated measures 4 times (once per season) **Dose-response Investigated?** No **Statistical Package:** SAS v8e	**Pollutant:** $PM_{2.5}$ (urban background concentration) **Averaging Time:** 48 h **Median:** 9.2 μg/m^3 **Percentiles:** Q25-Q75: 5.3-14.8 **Copollutant:** Urban background $PM_{2.5}$ Personal carbon black	**PM Increment:** 1 μg/m^3 **Effect Estimate [Lower CI, Upper CI]:** **Relationship between exposure and biomarkers** Estimate (p-value): RBC count (x 10^9/g protein): 0.0008 (0.36) Hemoglobin (μmol/g protein): 0.0005 (0.53) Platelet count (x 10^6/g protein): -0.0008 (0.49) Fibrinogen (nmol/g protein): 0.0004 (0.84) PLAAS (pmol/mg protein): 0.0004 (0.76) HBGGS (pmol/mg protein): -0.0020 (0.39) HBAAS (pmol/mg protein): -0.0021 (0.29) MDA (pmol/mg protein): 0.0012 (0.52)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Sullivan et al. (2007) **Period of Study:** February 2000–March 2002 **Location:** Seattle, Washington, USA	**Outcome:** Blood CRP, fibrinogen, D-dimer **Age Groups:** >55 years of age **Study Design:** Panel study **N:** 47 elderly subjects	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Median (IQR):** 7.7 µg/m³ (6.4) **Monitoring Stations:** 1 **Copollutant:** Indoor $PM_{2.5}$	**PM Increment:** 10 µg/m³ **Effect Estimate:** Among those with CVD, $PM_{2.5}$ 1 day earlier: CRP: 1.25 (95% CI: 0.97, 1.58); Fibrinogen: 1.01 (95% CI: 0.97, 1.05) D-dimer: 1.04 (95% CI: 0.93, 1.15) With COPD: CRP: 0.69 (95% CI: 0.34, 1.42) Fibrinogen: 1.05 (95% CI: 0.97, 1.13); D-dimer: 1.10 (95% CI: 0.95, 1.28) Healthy: CRP: 1.01 (95% CI: 0.85, 1.19) Fibrinogen: 0.88 (95% CI: 0.81, 0.95); D-dimer: 1.10 (95% CI: 0.75, 1.58) **Notes:** Out of 47 subjects, n = 23 with CVD and n = 24 (n = 16 COPD and 8 healthy) without CVD. Blood markers were measured on 2-3 morning over a 5-10 day period, and outdoor $PM_{2.5}$ was measured at a central monitoring site. These findings are not consistent with and effect of fine PM on markers of inflammation and thrombosis in the elderly.
Reference: Sullivan et al. (2005b) **Period of Study:** February 2000–March 2002 **Location:** Seattle, Washington, USA	**Outcome:** Heart rate variability (H, LF, HF, r-MSSD, SDNN) **Study Design:** Panel study **N:** 34 elderly subjects with (n = 21) and without (n = 13) CVD. **Statistical Analysis: L**inear mixed effects regression	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h **Median (IQR):** 10.7 (7.6) **Copollutant:** CO, NO_2	**PM Increment:** 10 µg/m³ **Effect Estimate:** 1 h: With CVD: HF: (3% increase, 95% CI: -19, 32) Without CVD: HF(5% decrease, 95% CI: -34, 36) Similarly, no association was found for 4-h or 24-h mean $PM_{2.5}$ concentrations. **Notes:** 285 daily 20 minute HRV measures were made in the homes of study subjects over a 10-day period.
Reference: Sullivan et al. (2005b) **Period of Study:** February 2000–March 2002 **Location:** Seattle area, WA	**Outcome (ICD9 and ICD10):** High-sensitivity C-reactive protein (hs-CRP); fibrinogen; D-dimer; endothelin-1 (ET-1); interleukin-6 (IL-6; interleukin-6 receptor (IL-6r); tumor necrosis factor-α (TNF-8- α); tumor necrosis factor-receptors (p55, p75); monocyte chemoattractant protein-1 (MCP-1) **Age Groups:** ≥ 55 yrs **Study Design:** Panel (repeated measures) **N:** 47 participants with (23) and without (10 COPD and 8 healthy) CVD **Statistical Analyses:** Mixed models **Covariates:** Age, gender, medication use, meteorological variables (temperature and RH) **Dose-response Investigated?** No **Statistical Package:** SAS v8.02	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h (0-day and 1-day lags) **Mean (SD):** NR **Percentiles:** For all subject-days: 25th: 5.2 50th: 7.7 75th: 11.5 90th: 19.9 **Range (Min, Max):** 1.3, 33.9 **Monitoring Stations:** NA, measured at participant's residence **Copollutant:** None	**PM Increment:** 10 µg/m³ **Effect Estimate [Lower CI, Upper CI]:** Multiplicative change in mean outcome associated with 10 µg/m³ increase in PM **Among those with different disease status.** **CRP Fold-rise (95%CI)** CV; 0-d lag: 1.21 (0.86, 1.70); CV; 1-d lag: 1.25 (0.97, 1.58); COPD; 0-d lag: 0.93 (0.48, 1.80); COPD; 1-d lag: 0.69 (0.33, 1.46); Healthy; 0-d lag: 0.98 (0.88, 1.08); Healthy; 1-d lag: 1.01 (0.84 1.21) **Fibrinogen Fold-rise (95%CI)** CV; 0-d lag: 1.02 (0.98, 1.06); CV; 1-d lag: 1.0 (0.97, 1.03); COPD; 0-d lag: 1.0 (0.91, 1.09); COPD; 1-d lag: 1.08 (0.99, 1.17); Healthy; 0-d lag: 0.94 (0.87, 1.01); Healthy; 1-d lag: 0.99 (0.88, 1.17) **D-dimer Fold-rise (95%CI)** CV; 0-d lag: 1.02 (0.88, 1.17); CV; 1-d lag: 1.03 (0.93, 1.15); COPD; 0-d lag: 1.04 (0.93, 1.16); COPD; 1-d lag: 1.09 (0.94, 1.27); Healthy; 0-d lag: 0.95 (0.79, 1.14); Healthy; 1-d lag: 0.97 (0.71, 1.31) **Among those with cardiovascular disease** **MCP-1 Fold-rise (95%CI)** 0-d lag: 1.3 (1.1, 1.7); 1-d lag: 1.0 (0.9, 1.3) **ET-1 Fold-rise (95%CI)** 0-d lag: 1.1 (0.8, 1.2); 1-d lag: 1.1 (0.9, 1.2) **Note:** TNF-α and IL-6 measures were below the limit of detection of assays
Reference: Timonen et al. (2006) **Period of Study:** 1998–1999 **Location:** Amsterdam, Netherlands Erfurt, Germany Helskinki, Finland	**Outcome:** Heart variability (HRV) measurements: [LF, HF, LFHFR, NN interval, SDNN, r-MSSD] **Study Design:** Panel study **N:** 131 elderly subjects with stable coronary heart disease **Statistical Analysis:** Linear mixed models	**Pollutant:** $PM_{2.5}$ **Means:** Amsterdam: 20.0 Erfurt: 23.3 Helsinki: 12.7 **Copollutant:** NO_2, CO	**PM Increment:** 10 µg/m³ **Effect Estimate:** SDNN; -0.33ms (95% CI: -1.05, 0.38) HF: -0.3% (95% CI: -10.6, 5.4) LFHFR: -1.4 (95% CI: -5.9, 8.7) **Notes:** Followed for 6 months with biweekly clinic visits 2-day lag. ULTRA Study

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Vallejo et al. (2006) **Period of Study:** April–August 2002 **Location:** Mexico City metropolitan area	**Outcome:** Heart rate variability measures (SDNN, pNN50) **Age Groups:** Mean age 27 yrs **Study Design:** Panel study **N:** 40 young healthy participants (non-smokers, no meds or history of CVD, respiratory, neurological, or endocrine disease) **Statistical Analysis:** Linear mixed effects models	**Pollutant:** $PM_{2.5}$ (pDR nephelometric method-DataRAM) **Copollutant:** None	**PM Increment:** 30 µg/m^3 **Effect Estimate: pNN50:** 0 h lag: -0.01% (95% CI: -0.03, 0.01); 1 h: -0.01% (95% CI: -0.04, 0.02); 2 h: -0.05% (95% CI: -0.09, 0.00); 3 h: -0.07% (95% CI: -0.13 to -0.02); 4 h: -0.08% (95% CI: -0.14 to -0.01); 5 h: -0.06% (95% CI: -0.13, 0.02); 6 h: -0.05% (95% CI: -0.13, 0.04) **SDNN:** 0 h: 0.00% (95% CI: 0.00, 0.01); 1 h: 0.00% (95% CI: -0.01, 0.01); 2 h: 0.00% (95% CI: -0.02, 0.01); 3 h: -0.01% (95% CI: -0.02, 0.00); 4 h: -0.01% (95% CI: -0.02, 0.01); 5 h: -0.01% (95% CI: -0.02, 0.01); 6 h: 0.00% (95% CI: -0.02, 0.02) **Notes:** Subjects underwent 13 h of ECG monitoring and personal $PM_{2.5}$ measurement. HRV measures were regressed against different lags of $PM_{2.5}$ concentration.
Reference: Wellenius et al. (2007) **Period of Study:** February 2002–March 2003 **Location:** Boston, Massachusetts, USA	**Outcome:** Circulating levels of B-type natriuretic peptide (BNP; measured in whole blood at 0, 6, 12 weeks) **Study Design:** Panel study **N:** 28 subjects (each with chronic stable HF and impaired systolic function) **Statistical Analysis:** Linear mixed effects models	**Pollutant:** $PM_{2.5}$ **Copollutant:** NO_2, SO_2, O_3, CO, BC	**PM Increment:** 10 µg/m^3 **Effect Estimate:** Same day $PM_{2.5}$: 0.8% increase in BNP (95% CI: -16.4, 21.5) **Notes:** The study found no association between any pollutant and measures of BNP at any lag. Further, the within subject coefficient of variation was large suggesting the magnitude of effected air pollutant health effects are small in relation to within subject variability in BNP.
Reference: Wellenius et al. (2007) **Period of Study:** February 2002–March 2003 **Location:** Boston, Massachusettes	**Outcome (ICD9 and ICD10):** B-type natriuretic peptide (BNP) (natural-log transformed) **Age Groups:** 33-88 yrs **Study Design:** Panel (blood collected at 0, 6, and 12 weeks) **N:** 28 patients with chronic stable heart failure and impaired systolic function **Statistical Analyses:** Linear mixed-effects models **Covariates:** Temperature, dew point, mean dew point over the past 3 days, calendar month of blood draw, measurement occasion, treatment assignment, measurement occasion by treatment assignment interaction **Season:** Adjusted for calendar month **Dose-response Investigated?** No **Statistical Package:** SAS v9.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily (assessed lags of 0-3 days) **Mean (SD):** 10.9 (8.4) **Percentiles:** 50th: 8.0 µg/m^3 **Range (Min, Max):** 0.7-50.9 µg/m^3 **Monitoring Stations:** 1 monitor **Copollutant (correlation):** CO (r = 0.35) NO_2 (r = 0.31) SO_2 (r = 0.18) O_3 (r = 0.35) BC(r = 0.68)	**PM Increment:** IQr = 8.1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** % change in BNP per IQR increase in $PM_{2.5}$ Lag0: 1.5 (-18.7, 19.2) Lag1: 2.1 (-20.0, 30.3) Lag2: 1.3 (12.3, 17.1) Lag3: 5.6 (-16.8, 34.0) **Note:** No significant associations observed between any pollutant and BNP levels at any lags (presented in Fig 2)
Reference: Wheeler et al. (2006) **Period of Study:** Fall 1999 and spring 2000 **Location:** Atlanta, GA	**Outcome:** Heart rate variability **Age Groups:** 49–76 years **N:** 18 subjects with COPD and 12 subjects with a recent MI **Statistical Analysis:** Linear-mixed effect model **Season:** Fall and spring	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h 4 h 24 h **Mean:** 24-hs: 17.8 µg/m^3 **Copollutant:** O_3, CO, SO_2, NO_2	**PM Increment:** 11.65 µg/m^3 (IQR) in 4 h $PM_{2.5}$ **Effect Estimate:** Among COPD patients: 8.3% increase in SDNN (95% CI: 1.7, 15.3) Among MI patients: 2.9% decrease in SDNN (95% CI: -7.8, 2.3) Results for 1h and 24 h averaging times were similar. **Notes:** Data was collected on 7 days in the Fall of 1999 or spring of 2000. Effects were modified by medication use, baseline pulmonary function, and health status.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Yue et al. (2007) **Period of Study:** October 2000–April 2001 **Location:** Erfurt, Germany	**Outcome:** QT interval and T-wave amplitude for ECG recordings, and vWF, CRP from blood samples **Study Design:** Panel study **N:** 56 patients (male CAD patients with 12 clinical visits) **Statistical Analysis:** Linear and logistic regression models **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$, Particle Number Concentration (PNC) (n/cm^3) **Averaging Time: Mean:** Mass concentrations of PNC (0.1-2.84 n/cm^3) **Monitoring Stations:** 1 **Copollutant:** None	**PM Increment:**. IQR **Effect Estimate:** Each IQR increase in 0-23 h mean traffic particle concentration was associated with: QT interval: 0.6% (95% CI: -0.3, 1.4) T wave amplitude: -1.6% (95% CI: -3.3, 0.1) vWF: 3.2% (95% CI: -0.5, 7.0) CRP: (OR = 1.5; 95% CI 1.0–2.3) Each IQR increase in 0-23 h mean combustion-generated particle concentration was associated with: QT interval: 0.1%(-0.3, 0.6) T wave amplitude: -0.2% (-1.2, 0.7) vWF: 2.8% (0.8, 4.8) CRP (OR = 1.0; 0.8, 1.2) **Notes:** Five sources of particles were identified (airborne soil, local traffic-related ultrafine particles, combustion-generated aerosols, diesel traffic-related particles, and secondary aerosols).
Reference: Yue et al. (2007) **Period of Study:** Oct 12, 2000–Apr 27, 2001 **Location:** Erfurt, Germany	**Outcome:** QT interval, T wave amplitude, von Willebrand factor (vWF), C-reactive protein (CRP; above 90th percentile compared to below) **Age Groups:** >50 yrs **Study Design:** Panel (12 visits; 625 observations for repolarization parameters and 578 observations for inflammatory markers) **N:** 57 male coronary artery disease patients **Statistical Analyses:** Linear and logistic fixed-effects regression models (generalized additive models) **Covariates:** Trend, weekday, and meteorological variables (temperature, relative humidity, barometric pressure) **Dose-response Investigated?** No **Statistical Package:** SAS v9.1 and S-Plus v6.0	**Pollutant:** Five particle source factors (airborne soil, local traffic-related ultrafine particles, combustion-generated aerosols, diesel traffic-related particles, and secondary aerosols); see below for size fractions (factor scores) **Averaging Time:** Used daily factor scores in analyses **Mean (SD):** Factor 1: particles from airborne soil (1.0-2.8 µm): 2390 (1696) Factor 2: ultrafine particles from local traffic (0.01-0.1 µm): 9931 (5858) Factor 3: secondary aerosols from local fuel combustion (0.1-0.5 µm): 3770 (6129) Factor 4: particles from traffic (0.01-0.5 µm): 6865 (5689) Factor 5: secondary aerosols from multiple sources (0.2-1.0 µm): 4732 (3890) **Median:** Factor 1: 2053 Factor 2: 8531 Factor 3: 1348 Factor 4: 5045 Factor 5: 3752 **IQR (5-day avg):** Factor 1: 1110 Factor 2: 5749 Factor 3: 4124 Factor 4: 5000 Factor 5: 3393 **Range (Min, Max):** Factor 1: 284, 12960 Factor 2: 866, 26632 Factor 3: 139, 39097 Factor 4: 283, 27605 Factor 5: 67, 20129 **Monitoring Stations:** 1 monitor **Copollutant:** NA	**PM Increment: IQR** **Effect Estimate [Lower CI, Upper CI]: QT interval, % change (95%CI)** **Factor 1:** 0-5h: -0.1 (-0.6, 0.6); 6-11h: -0.5 (-1.1, 0.2); 12-17h: 0.1 (-0.4, 0.4); 18-23h: -0.2 (-0.7, 0.2); 0-23h: -0.2 (-0.9, 0.4); 1d: -0.1 (-0.7, 0.6); 2d: -0.3 (-0.9, 0.4); 3d: -0.7 (-1.4, 0.1); 4d: -0.2 (-0.9, 0.5); 0-4d avg: -0.7 (-1.8, 0.3) **Factor 2:** 0-5h: 0.2 (-0.4, 0.8); 6-11h: 0.8 (-0.0, 1.7); 12-17h: 0.6 (-0.2, 1.4); 18-23h: 0.5 (-0.4, 1.4); 0-23h: 0.9 (-0.1, 2.0); 1d: 1.5 (0.3, 2.7); 2d: -0.4 (-1.7, 1.0); 3d: 0.5 (-0.9, 1.9); 4d: 0.1 (-1.2, 1.4); 0-4d avg: 1.6 (-0.1, 3.3) **Factor 3:** 0-5h: 0.1 (-0.3, 0.5); 6-11h: 0.2 (-0.3, 0.6); 12-17h: 0.2 (-0.3, 0.6); 18-23h: 0.1 (-0.3, 0.4); 0-23h: 0.1 (-0.3, 0.6); 1d: 0.1 (-0.3, 0.4); 2d: -0.1 (-0.4, 0.3); 3d: -0.2 (-0.5, 0.2); 4d: -0.1 (-0.5, 0.2); 0-4d avg: -0.1 (-0.7, 0.6) **Factor 4:** 0-5h: 0.2 (-0.4, 0.8); 6-11h: 0.8 (0.0, 1.6); 12-17h: 0.5 (-0.2, 1.3); 18-23h: 0.5 (-0.2, 1.2); 0-23h: 0.6 (-0.3, 1.4); 1d: -0.4 (-1.5, 0.7); 2d: -0.9 (-2.0, 0.1); 3d: -0.5 (-1.4, 0.5); 4d: -0.5 (-1.3, 0.2); 0-4d avg: -0.3 (-1.7, 1.1) **Factor 5:** n0-5h: 1.0 (-0.1, 2.1); 6-11h: 0.9 (-0.2, 2.0); 12-17h: 0.3 (-0.7, 1.4); 18-23h: -0.1 (-1.2, 1.0); 0-23h: 0.7 (-0.6, 1.9); 1d: 0.1 (-1.1, 1.3); 2d: -0.2 (-1.5, 1.1); 3d: -0.6 (-1.9, 0.8); 4d: -0.9 (-2.0, 0.2); 0-4d avg: -0.4 (-1.9, 1.2) **T wave amplitude, % change (95%CI)** **Factor 1:** 0-5h: -0.3 (-1.5, 0.9); 6-11h: -0.6 (-1.9, 0.7); 12-17h: 0.1 (-0.8, 0.9); 18-23h: -0.6 (-1.5, 0.4); 0-23h: -0.5 (-1.8, 0.9); 1d: 0.4 (-0.9, 1.7); 2d: 1.2 (-0.3, 2.7); 3d: 0.2 (-1.2, 1.7); 4d: -0.2 (-1.3, 1.0); 0-4d avg: 0.8 (-1.1, 2.6) **Factor 2**: 0-5h: -1.7 (-3.0 to -0.4); 6-11h: -2.6 (-4.5 to -0.6); 12-17h: -1.0 (-2.6, 0.7); 18-23h: -1.1 (-2.8, 0.7); 0-23h: -3.1 (-5.3 to -0.9); 1d: -0.3 (-2.9, 2.2); 2d: -1.2 (-4.1, 1.7); 3d: -0.5 (-3.2, 2.1); 4d: -3.4 (-9.9, 3.1); 0-4d avg: -1.5 (-4.4, 1.5) **Factor 3:** 0-5h: -0.3 (-1.1, 0.6); 6-11h: -0.1 (-0.9, 0.9); 12-17h: 0.1 (-0.9, 1.0); 18-23h: -0.4 (-1.2, 0.4); 0-23h: -0.2 (-1.2, 0.7); 1d: 0.1 (-0.7, 0.8); 2d: -0.1 (-0.7, 0.7); 3d: 0.4 (-0.3, 1.1); 4d: 0.1 (-0.7, 0.7); 0-4d avg: 0.3 (-0.9, 1.5) **Factor 4:** 0-5h: -1.5 (-2.8 to -0.2); 6-11h: -1.3 (-3.0, 0.3); 12-17h: -1.1 (-2.7, 0.4); 18-23h: -0.9 (-2.4, 0.6); 0-23h: -1.6 (-3.3, 0.1); 1d: -1.2 (-3.3, 0.9); 2d: -1.0 (-3.2, 1.2); 3d: 0.2 (-1.5, 1.9); 4d: 0.5 (-1.0, 2.0); 0-4d avg: -1.7 (-4.1, 0.7) **Factor 5:** 0-5h: -1.6 (-3.6, 0.4); 6-11h: -0.1 (-2.1, 2.0); 12-17h: -0.2 (-2.2, 1.8); 18-23h: -1.8 (-3.8, 0.2); 0-23h: -1.2 (-3.4, 1.0); 1d: -1.8 (-4.2, 0.6); 2d: -0.7 (-3.5, 2.1); 3d: 0.8 (-1.5, 3.2); 4d: 0.5 (-1.5, 2.5); 0-4d avg: -1.4 (-4.0, 1.2) **vWF, % change (95%CI)** **Factor 1:** 0-5h: 1.1 (-1.5, 3.6); 6-11h: 1.6 (-1.2, 4.5); 12-17h: 0.4 (-1.4, 2.1); 18-23h: 1.4 (-0.6, 3.5); 0-23h: 1.6 (-1.3, 4.4); 1d: -1.0 (-3.9, 1.9); 2d: -1.8 (-4.8, 1.2); 3d: -2.5 (-5.8, 0.9); 4d: 0.5 (-2.9, 3.9); 0-4d avg: -2.5 (-7.1, 2.2) **Factor 2:** 0-5h: 0.4 (-2.4, 3.2); 6-11h: -0.4 (-4.3, 3.4); 12-17h: 2.1 (-1.4, 5.7); 18-23h: 2.3 (-1.4, 5.9); 0-23h: 1.9 (-2.8, 6.6); 1d: 2.8 (-2.8, 8.3); 2d: 5.1 (-0.8, 11.1); 3d: 11.4 (5.3, 17.6); 4d: 6.6 (0.0, 13.1); 0-4d avg: 11.4 (3.7, 19.1) **Factor 3:** 0-5h: 1.8 (0.1, 3.6); 6-11h: 1.7 (-0.3, 3.7); 12-17h: 2.2 (0.3, 4.2); 18-23h: 2.8 (1.1, 4.5); 0-23h: 2.8 (0.8, 4.8); 1d: 2.7 (1.0, 4.4); 2d: 3.4 (1.8, 5.0); 3d: 2.3 (0.8, 3.8); 4d: 1.4 (-0.2, 2.9); 0-4d avg: 4.8 (2.0, 7.6) **Factor 4:** 0-5h: 1.5 (-1.4, 4.3); 6-11h: 2.0 (-1.7, 5.6); 12-17h: 2.6 (-0.8, 5.9); 18-23h: 3.5 (0.4, 6.6); 0-23h: 3.2 (-0.5, 7.0); 1d: 5.4 (0.6, 10.2); 2d: 4.5 (-0.6, 9.5); 3d: 3.8 (-0.6, 8.1); 4d: 3.0 (-0.6, 6.6); 0-4d avg: 11.3 (5.0, 17.6) **Factor 5:** 0-5h: 1.9 (-2.8, 6.6); 6-11h: 3.2 (-1.6, 8.0); 12-17h: 2.4 (-2.3, 7.1); 18-23h: 1.6 (-3.1, 6.2); 0-23h: 2.9 (-2.5, 8.2); 1d: -2.2 (-7.6, 3.2); 2d: -

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
			1.3 (-7.4, 4.9); 3d: 1.1 (-4.8, 7.1); 4d: 1.3 (-4.2, 6.7); 0-4d avg: 3.3 (-4.1, 10.6) **CRP, Odds Ratio (95%CI)** Factor 1: 0-5h: 0.9 (0.7, 1.1); 6-11h: 1.4 (1.1, 1.8); 12-17h: 1.2 (1.0, 1.4); 18-23h: 1.0 (0.8, 1.3); 0-23h: 1.1 (0.9, 1.5); 1d: 1.4 (1.1, 1.8); 2d: 1.3 (1.0, 1.7); 3d: 1.0 (0.7, 1.4); 4d: 1.1 (0.9, 1.5); 0-4d avg: 1.6 (1.1, 2.2) Factor 2: 0-5h: 0.8 (0.6, 1.0); 6-11h: 1.0 (0.7, 1.4); 12-17h: 1.1 (0.8, 1.5); 18-23h: 1.0 (0.8, 1.4); 0-23h: 0.9 (0.6, 1.4); 1d: 0.9 (0.6, 1.5); 2d: 2.1 (1.3, 3.3); 3d: 1.9 (1.0, 3.6); 4d: 1.4 (0.8, 2.3); 0-4d avg: 1.4 (0.8, 2.6) Factor 3: 0-5h: 1.0 (0.8, 1.1); 6-11h: 0.9 (0.8, 1.1); 12-17h: 1.0 (0.9, 1.2); 18-23h: 1.0 (0.8, 1.2); 0-23h: 1.0 (0.8, 1.2); 1d: 1.1 (1.0, 1.3); 2d: 1.0 (0.9, 1.2); 3d: 1.2 (1.1, 1.4); 4d: 1.1 (1.0, 1.3); 0-4d avg: 1.2 (1.0, 1.5) Factor 4: 0-5h: 0.8 (0.6, 1.1); 6-11h: 0.8 (0.6, 1.1); 12-17h: 1.3 (1.0, 1.8); 18-23h: 1.1 (0.8, 1.5); 0-23h: 1.0 (0.7, 1.4); 1d: 1.5 (1.0, 2.3); 2d: 2.0 (1.3, 3.2); 3d: 1.5 (0.9, 2.3); 4d: 1.3 (0.9, 1.8); 0-4d avg: 1.7 (1.0, 2.9) Factor 5: 0-5h: 0.7 (0.5, 1.1); 6-11h: 1.4 (0.9, 2.1); 12-17h: 1.9 (1.3, 2.8); 18-23h: 1.4 (1.0, 2.0); 0-23h: 1.4 (0.9, 2.2); 1d: 1.6 (1.0, 2.6); 2d: 1.6 (0.9, 2.6); 3d: 2.3 (1.3, 4.1); 4d: 1.6 (0.9, 2.8); 0-4d avg: 2.1 (1.2, 3.8)
Reference: Zanobetti et al. (2004) **Period of Study:** 1999 to 2001 **Location:** Boston, Massachusetts, USA	**Outcome:** Blood pressure (systolic blood pressure, diastolic blood pressure, mean arterial blood pressure) **Age Groups:** Elderly **Study Design:** Panel study **N:** 62 elderly subjects with n = 631 repeated visits for cardiac rehabilitation **Statistical Analysis:** Linear mixed effects models	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Median (10th–90th percentile)** Median: 8.8 10th-90th: 13.4 **Monitoring Stations:** 1 **Copollutant:** SO_2, O_3, CO, NO_2, BC 120-h avg Median: 0.651 10th-90th: 0.376	**PM Increment:**.10.4 µg/m³ for 5 day mean, 13.9 µg/m³ for 2-day mean **Effect Estimate:** Each 10.4 µg/m³ increase in 5 day mean $PM_{2.5}$ concentration was associated with: Systolic BP: 2.8mmHg (95% CI: 0.1, 5.5) Diastolic BP: 2.7mmHg (95% CI: 1.2, 4.3) Mean arterial BP: 2.7mmHg (95% CI: 1.0, 4.5) Each 13.9 µg/m³ increase in 2-day mean $PM_{2.5}$, during exercise in person with H.70bpm Diastolic: 7.0mmHg (95% CI: 2.3, 12.1) Mean arterial BP: 4.7mmHg (95% CI: 0.5, 9.1)
Reference: Zeka et al. (2006a) **Period of Study:** Nov 2000–Dec 2004 **Location:** Greater Boston area (Massachusettes)	**Outcome:** White blood cells (WBC), C-reactive protein (CRP), sediment rate, fibrinogen **Age Groups:** Mean age (SD) = 73.0 (6.7) **Study Design:** Cross-sectional **N:** 710 subjects **Statistical Analyses:** Linear regression **Covariates:** Age, BMI, season (also assessed potential for confounding by temperature, RH, barometric pressure, hypertensive or cardiac medications, hypertension, smoking, alcohol, and fasting glucose levels) **Dose-response Investigated?** No	**Pollutant:** SO_4^{2-} **Averaging Time:** Hourly (PN, BC, $PM_{2.5}$) and 24-h (SO_4^{2-}) measurements used to create 48-h, 1-wk, and 4-wk moving averages **Mean (SD):** 2.29 (1.62) **Percentiles:** 50th: 1.84 75th: 2.81 90th: 4.10 **Monitoring Stations:** 2 sites **Copollutant (correlation):** $PM_{2.5}$ (r = 0.50) BC (r = 0.30) PN (r = -0.15) SO_4^{2-}	**PM Increment:** 1 SD increase **Effect Estimate [Lower CI, Upper CI]:** % increase (95%CI) in biomarker per 1 SD increase in pollutant. **Fibrinogen:** 48 h: 0.60 (-1.23, 2.42); 1 wk: 0.03 (-1.93, 1.99); 4 wk: 1.12 (-0.52, 2.77) **CRP:** 48 h: 1.57 (-7.13, 10.27); 1 wk: 0.21 (-8.27, 8.69); 4 wk: 5.29 (-1.91, 12.49) **Sediment rate:** 48 h: 4.05 (-23.26, 31.36); 1 wk: -5.87 (-32.39, 20.64); 4 wk: -1.60 (-25.24, 22.04) **WBC count:** 48 h: -0.12 (-2.35, 2.11); 1 wk: -0.48 (-2.87, 1.90); 4 wk: 0.75 (-1.30, 2.80) **Note:** No statistically significant difference was reported for any category of effect modifiers (age, obesity, medications, homozygous for the deletion of GSTM1-null, hypertension)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Zeka et al. (2006a) **Period of Study:** Nov 2000–Dec 2004 **Location:** Greater Boston area (Massachusettes)	**Outcome (ICD9 and ICD10):** White blood cells (WBC), C-reactive protein (CRP), sediment rate, fibrinogen **Age Groups:** Mean age (SD) = 73.0 (6.7) **Study Design:** Cross-sectional **N:** 710 subjects **Statistical Analyses:** Linear regression **Covariates:** Age, BMI, season (also assessed potential for confounding by temperature, RH, barometric pressure, hypertensive or cardiac medications, hypertension, smoking, alcohol, and fasting glucose levels) **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Hourly (PN, BC, $PM_{2.5}$) and 24-h (SO_4^{2-}) measurements used to create 48-h, 1-wk, and 4-wk moving averages **Mean (SD):** 11.16 (7.95) **Percentiles:** 50th: 9.39 75th: 14.57 90th: 21.48 **Monitoring Stations:** 2 sites **Copollutant (correlation):** $PM_{2.5}$ BC (r = 0.52) PN (r = -0.02) SO_4^{2-} (r = 0.50)	**PM Increment:** 1 SD increase **Effect Estimate [Lower CI, Upper CI]:** % increase (95%CI) in biomarker per 1 SD increase in pollutant. **Fibrinogen:** 48 h: -0.18 (-1.93, 1.57); 1 wk: -1.39 (-3.46, 0.67); 4 wk: 1.14 (-0.60, 2.88) **CRP:** 48 h: -4.88 (-13.29, 3.53); 1 wk: -1.37 (-10.44, 7.71); 4 wk: 4.36 (-3.25, 11.96) **Sediment rate:** 48 h: -16.91 (-43.66, 9.84); 1 wk: -18.89 (-47.48, 9.70); 4 wk: 24.93 (0.68, 49.18) **WBC count:** 48 h: -3.18 (-5.39 to -0.97); 1 wk: -0.51 (-3.02, 2.00); 4 wk: -0.03 (-2.17, 2.10) **Note:** No statistically significant difference was reported for any category of effect modifiers (age, obesity, medications, homozygous for the deletion of GSTM1-null, hypertension)

Table E-4. Short-term exposure to other PM size fractions and cardiovascular morbitidy outcomes.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Adar et al. (2007) **Period of Study:** March–June 2002 **Location:** St. Louis, Missouri	**Outcome:** Heart rate variability: heart rate, standard deviation of all normal-to-normal intervals (SDNN), square root of the mean squared difference between adjacent normal-to-normal intervals (rMSSD), percentage of adjacent normal-to-normal intervals that differed by more than 50 ms (pNN50), high frequency power (HF; in the range of 0.15-0.4Hz), low frequency power (LF, in the range of 0.04-0.15Hz), and the ratio of LF/HF **Age Groups:** ≥ 60 yrs **Study Design:** Panel (4 planned repeated measures with a total of 158 person-trips; 35 participating in all 4 trips) **N:** 44 participants **Statistical Analyses:** Generalized additive models **Covariates:** Subject, weekday, time, apparent temperature, trip type, activity, medications, and autoregressive terms **Season:** Limited data collection period **Dose-response Investigated?** No **Statistical Package:** SAS v8.02, R v2.0.1	**Pollutant:** Particle count fine (PC fine) (particles/cm^3) **Averaging Time:** Measurements collected over 48 h period surrounding the bus trip (during which health endpoints were measured) used to calculate 5-, 30-, 60-minute, 4-h, 24-h moving averages **Median (IQR):** All: 42 (57) Facility: 36 (45) Bus: 105 (96) Activity: 50 (133) Lunch: 69 (48) **Monitoring Stations:** 2 portable carts **Copollutant:** $PM_{2.5}$; BC; Fine particle counts; Coarse particle counts **Correlation notes:** 24-h mean $PM_{2.5}$, BC, and fine particle count concentrations ranged from 0.80 to 0.98; r = 0.76 to 0.97 when limited to time spent on the bus; r = 0.55 to 0.86 when comparing bus concentrations to 24-h moving averages; r = -0.003 to 0.51 when comparing 5-min averages and 24-h moving averages.Poor correlations found between coarse particle count concentrations and all fine particulate measures during all times periods	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change (95%CI) in HRV per IQR in the 24-h moving avg of the microenvironmental pollutant (IQr = 39 pt/cm^3) **Single-pollutant models** SDNN: -5.1 (-5.8 to -4.4) rMSSD: -8.0 (-8.7 to -7.2) pNN50 + 1: -10.2 (-11.3 to -9.0) LF: -9.9 (-11.4 to -8.4) HF: -13.7 (-15.1 to -12.2) LF/HF: 4.3 (3.1, 5.5) H: 0.9 (0.8, 1.1) **Note:** Exposure to health associations by all lag periods presented in Figure 2 (magnitude of associations increased with averaging period, with the largest associations consistently found for 24-h moving averages)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Adar et al. (2007) **Period of Study:** March–June 2002 **Location:** St. Louis, Missouri	**Outcome:** Heart rate variability: heart rate, standard deviation of all normal-to-normal intervals (SDNN), square root of the mean squared difference between adjacent normal-to-normal intervals (rMSSD), percentage of adjacent normal-to-normal intervals that differed by more than 50 ms (pNN50), high frequency power (HF; in the range of 0.15-0.4Hz), low frequency power (LF, in the range of 0.04-0.15Hz), and the ratio of LF/HF **Age Groups:** ≥ 60 yrs **Study Design:** Panel (4 planned repeated measures with a total of 158 person-trips; 35 participating in all 4 trips) **N:** 44 participants **Statistical Analyses:** Generalized additive models **Covariates:** Subject, weekday, time, apparent temperature, trip type, activity, medications, and autoregressive terms **Season:** Limited data collection period **Dose-response Investigated?** No **Statistical Package:** SAS v8.02, R v2.0.1	**Pollutant:** Particle count coarse (PT coarse) (pt/cm^3) **Averaging Time:** Measurements collected over 48-h period surrounding the bus trip (during which health endpoints were measured) used to calculate 5-, 30-, 60-minute, 4-h, and 24-h moving averages **Median (IQR):** All: 0.02 (0.11) Facility: 0.01 (0.04) Bus: 0.16 (0.13) Activity: 0.29 (0.26) Lunch: 0.16 (0.36) **Monitoring Stations:** 2 portable carts **Copollutant:** $PM_{2.5}$; BC; Fine particle counts; Coarse particle counts **Correlation notes:** 24-h mean $PM_{2.5}$, BC, and fine particle count concentrations ranged from 0.80 to 0.98; r = 0.76 to 0.97 when limited to time spent on the bus; r = 0.55 to 0.86 when comparing bus concentrations to 24-h moving averages; r = -0.003 to 0.51 when comparing 5-min averages and 24-h moving averages. Poor correlations found between coarse particle count concentrations and all fine particulate measures during all times periods	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change (95%CI) in HRV per IQR in the 24-h moving avg of the microenvironmental pollutant (IQr = 0.066 pt/cm^3) **Single-pollutant models** SDNN: 2.4 (1.3, 3.6) rMSSD: 3.9 (2.6, 5.1) pNN50 + 1: 2.9 (1.0, 4.9) LF: 6.4 (3.7, 9.1) HF: 10.2 (7.4, 13.1) LF/HF: -3.3 (-5.0 to -1.6) H: -1.1 (-1.3 to -0.8) **Two-pollutant models (with $PM_{2.5}$):** SDNN: -0.7 (-1.9, 0.6) rMSSD: -1.3 (-2.6 to -0.05) pNN50 + 1: -4.3 (-6.3 to -2.4) LF: 0.2 (-2.5, 3.0) HF: 1.3 (-1.5, 4.1) LF/HF: -0.9 (-2.7, 1.0) H: -0.6 (-0.9 to -0.4) **Note:** Exposure to health associations by all lag periods presented in Figure 2 (magnitude of associations increased with averaging period, with the largest associations consistently found for 24-h moving averages)
Reference: Delfino et al. (2008) **Period of Study:** 2005-2006 **Location:** Los Angeles, Califoria, air basin	**Outcome:** C-reactive protein (CRP); fibrinogen, tumor necrosis factor-α (TNF-α) and its soluble receptor-II (TNF-RII); interleukin-6 (IL-6); and its soluble receptor (IL-6sR); fibrin D-dimer; soluble platelet selectin (sP-selectin); soluble vascular cell adhesion molecule-1 (sVCAM-1); intracellular adhesion molecule-1 (sICAM-1); and myeloperoxidase (MPO); erythrocyte lysates for glutathione peroxidase-1 (GPx-1); copper-zinc superoxide dismutase (cu,Zn-SOD) **Age Groups:** ≥ 65 yrs **Study Design:** Panel (biomarkers measured weekly 12 times) **N:** 29 participants (nonsmoking with history of coronary artery desease) **Statistical Analyses:** Mixed models **Covariates:** temperature (infectious illnesses were excluded by excluding weeks with such observations) **Season:** Collected 6 weeks of data during warm period and 6 weeks of data during cool period **Dose-response**	**Pollutant:** PM (multiple size fractions and components) **Averaging Time:** 24-h avg preceding the blood draw (lag 0) and cumulative averages up to 5 days preceding the draw **Outdoor hourly PM:** EC: Mean (SD): 1.61 (0.62); Median: 1.56; IQR: 0.92; Min, Max: 0.24, 3.94 OC: Mean (SD): 5.94 (2.11); Median: 5.58; IQR: 2.79; Min-Max: 2.51, 13.60 BC: Mean (SD): 2.00 (0.77); Median: 1.89; IQR: 0.96; Min-Max: 0.58, 5.11 OC_{pri}: Mean (SD): 3.37 (1.21); Median: 3.21; IQR: 1.63; Min-Max: 0.99, 7.11 Secondary OC: Mean (SD): 2.49 (1.50); Median: 2.10; IQR: 1.86; Min-Max: 0, 8.10 PN (pt/cm^3): Mean (SD): 16,043 (5886); Median: 13,968; IQR: 7,386; Min-Max: 6837, 31263 **Indoor hourly PM** EC: Mean (SD): 1.31 (0.52); Median: 1.30; IQR: 0.70; Min-Max: 0.19, 2.89 EC of outdoor origin: Mean (SD): 1.11 (0.39); Median: 1.06; IQR: 0.51; Min-Max: 0.41, 2.97 OC: Mean (SD): 5.69 (1.51); Median: 5.60; IQR: 1.96; Min-Max: 2.34, 10.79 OC_{pri} of outdoor origin: Mean	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** **Note:** Nearly all results presented in figures **Results:** We found significant positive associations for CRP, IL-6, sTNF-RII, and sP-selectin with outdoor and/or indoor concentrations of quasi-ultrafine PM ≤ 0.25 µm in diameter, EC, OC_{pri}, BC, PN, CO, and nitrogen dioxide from the current-day and multiday averages. We found consistent positive but largely nonsignificant coefficients for TNF-α, sVCAM-1, and sICAM-1, but not fibrinogen, IL-6sR, or D-dimer. We found inverse associations for erythrocyte Cu,Zn-SOD with these pollutants and other PM size fractions (0.25–2.5 and 2.5–10 µm). Inverse associations of GPx-1 and MPO with pollutants were largely nonsignificant. Indoor associations were often stronger for estimated indoor EC, OC_{pri}, and PN of outdoor origin than for uncharacterized indoor measurements. There was no evidence for positive associations with SOA.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
	Investigated? No **Statistical Package:** NR	(SD): 2.18 (0.82); Median: 2.15; IQR: 1.07; Min-Max: 0.32, 5.21 Secondary OC of outdoor origin: Mean (SD): 2.08 (1.26); Median: 1.75; IQR: 1.45; Min-Max: 0, 6.87 PN (particles/cm^3): Mean (SD): 14,494 (6770); Median: 12,341; IQR: 7,337; Min-Max: 1016, 43027 PN of outdoor origin (p/cm^3): Mean (SD): 10,108 (3108); Median: 9,580; IQR: 3,684; Min-Max: 1016, 17700 **Outdoor PM mass** $PM_{0.25}$: Mean (SD): 9.47 (2.97); Median: 9.4; IQR: 4.2; Min-Max: 3.31, 18.75 $PM_{0.25-2.5}$: Mean (SD): 13.53 (10.67); Median: 11.7; IQR: 11.5; Min-Max: 1.29, 66.77 $PM_{2.5-10}$: Mean (SD): 10.04 (4.07); Median: 9.9; IQR: 5.9; Min-Max: 1.76, 22.38 **Indoor PM mass** $PM_{0.25}$: Mean (SD): 10.45 (6.77); Median: 9.5; IQR: 4.5; Min-Max: 1.42, 69.86 $PM_{0.25-2.5}$: Mean (SD): 7.36 (4.57); Median: 6.5; IQR: 5.7; Min-Max: 0.77, 30.86 $PM_{2.5-10}$: Mean (SD): 4.12 (4.76); Median: 2.8; IQR: 3.5; Min-Max: 0.12, 37.63 **Copollutant:** Outdoor hourly gases (NO_2, CO, O_3) and indoor hourly gases (NO_2, CO)	
Reference: Pekkanen et al. (2002) **Period of Study:** Winter 1998 to 1999 **Location:** Helsinki, Finland	**Outcome**: ST Segment Depression (>0.1mV) **Study Design**: Panel of ULTRA Study participants **N**: 45 Subjects, n = 342 biweekly submaximal exercise tests, 72 exercise induced ST Segment Depressions **Statistical Analysis:** Logistic regression / GAM	**Pollutant:** Ultrafine $NC_{0.01-0.1}$ (n/cm^3) **Averaging Time:** 24 h **Median**: 14,890 **IQR**: 9830 **Monitoring Stations**: 1 **Copollutant:** NO_2, CO, $PM_{2.5}$, $PM_{10-2.5}$, PM1, ACP	**PM Increment:** IQR **Effect Estimate(s):** NC0.01-0.1: OR = 3.14 (1.56, 6.32), lag 2 **Notes**: The effect was strongest for ACP and $PM_{2.5}$, which in two pollutant models appeared independent. Increases in NO_2 and CO were also associated with increased risk of ST segment depression, but not with coarse particles.
Reference: Peters et al. (2005) **Period of Study:** February 1999–July 2001 **Location:** Augsburg, Germany	**Outcome:** Myocardial infarction **Study Design:** Case-crossover **N:** 691 myocardial infarction patients **Statistical Analysis:** Conditional logistic regression **Dose-response investigated (yes/no)?** No	**Pollutant:** Ultrafine (TNC) (n/cm^3) **Averaging Time:** 1 h: Median = 10,001 IQR: 7919 24 h: Median = 10,934 IQR: 6276 **Copollutant:** NO_2, SO_2, CO	**PM Increment: Effect Estimate:** 2-h lag: OR = 0.95; 95% CI: 0.84, 1.06 24-h mean, 2-day lag: OR = 1.04; 95% CI: 0.90, 1.20 **Notes:** Examined triggering for MI at various lags before MI onset (up to 6 h before MI, up to 5 days before MI). No statistically significant increases in lagged ultrafine particle concentration were found.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ruckerl et al. (2006) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome (ICD9 and ICD10):** C-reactive protein (CRP); serum amyloid A (SAA); E-selectin; von Willebrand Factor (vWF); intercellular adhesion molecule-1 (ICAM-1); fibrinogen; Factor VII; prothrombin fragment 1+2; D-dimer **Age Groups:** 50+ yrs **Study Design:** Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear and logistic regression models **Covariates:** Models adjusted for different factors based on health endpoint; CRP: RH, temperature, trend, ID; ICAM-1: temperature, trend, ID; vWF: air pressure, RH, temperature, trend, ID; FVII: air pressure, RH, temperature, trend, ID, weekday **Season:** Time trend as covariate **Dose-response Investigated?** Sensitivity analyses examined nonlinear exposure-response functions **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** AP (n/cm^3) **Averaging Time:** 24 h **Mean (SD):** 1593 (1034) **Percentiles:** 25: 821 50: 1238 75: 2120 **Range (Min, Max):** 328, 4908 **Unit (i.e. μg/m^3):** n/cm^3 **Monitoring Stations:** 1 site **Copollutant:** UFPs AP $PM_{2.5}$ PM_{10} OC EC NO_2 CO	**PM Increment:** IQR (1299; 5-d avg: 1127) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as OR (95%CI) for an increase in the blood marker above the 90th percentile per increase in IQR air pollutant. **CRP** Time before draw: 0 to 23 h: 0.7 (0.5, 1.2); 24 to 47 h: 1.5 (0.9, 2.6) 48 to 71 h: 3.2 (1.7, 6.0); 5-d mean: 1.5 (0.8, 3.0) **ICAM-1** Time before draw: 0 to 23 h: 0.6 (0.4, 0.9); 24 to 47 h: 1.8 (1.2, 2.8) 48 to 71 h: 1.6 (1.0, 2.5); 5-d mean: 0.9 (0.6, 1.5) Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **vWF** Time before draw: 0 to 23 h: 4.8 (0.2, 9.3); 24 to 47 h: 5.9 (0.4, 11.5) 48 to 71 h: 7.0 (0.7, 13.4); 5-d mean: 13.5 (6.3, 20.6) **FVII** Time before draw: 0 to 23 h: 0.0 (-2.9, 3.0); 24 to 47 h: -2.9 (-6.1, 0.4) 48 to 71 h: -3.6 (-6.8 to -0.3); 5-d mean: -4.1 (-7.9 to -0.3) **Note:** summary of results presented in figures. SAA results indicate increase in association with PM (not as strong and consistent as with CRP); no association observered between E-selectin and PM; an increase in prothrombin fragment 1+2 was consistently observed, particularly with lag 4; fibrinogen results revealed few significant associations, potentially due to chance; D-dimer results revealed null associations in linear and logistic analyses
Reference: Ruckerl et al. (2006) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome:** Soluble CD40 ligand (sCD40L), platelets, leukocytes, erythrocytes, hemoglobin **Age Groups:** 50+ yrs **Study Design**: Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear regression models **Covariates:** Long-term time trend, weekday of the visit, temperature, RH, barometric pressure **Season:** Time trend as covariate **Dose-response Investigated?** No **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** AP (n/cm^3) **Averaging Time:** 24 h **Mean (SD):** 1593 (1034) **Percentiles:** 25th: 821 50th: 1238 75th: 2120 **Range (Min, Max):** 328, 4908 **Monitoring Stations:** 1 site **Copollutant:** UFPs AP $PM_{2.5}$ PM_{10} NO	**PM Increment:** IQR (1299; 5-d avg: 1127) **Effect Estimate [Lower CI, Upper CI]:** Effects of air pollution on blood markers presented as % change from the mean/GM in the blood marker per increase in IQR air pollutant. **sCD40L, % change GM (pg/mL)** lag0: 6.9 (0.5, 13.8); lag1: -1.1 (-8.0, 6.4) lag2: -4.9 (-11.9, 2.7); lag3: -3.8 (-10.3, 3.2) 5-d mean: -1.3 (-9.9, 8.1) **Platelets, % change mean (10^3/μl)** lag0: -1.0 (-2.5, 0.5); lag1: -0.4 (-2.1, 1.6) lag2: 0.8 (-1.0, 2.4); lag3: 0.0 (-1.8, 1.7) 5-d mean: -0.9 (-3.0, 1.3) **Leukocytes, % change in mean (10^3/μl)** lag0: -1.9 (-3.8 to -0.1); lag1: -0.6 (-2.9, 1.6) lag2: -0.6 (-3.2, 2.0); lag3: -2.3 (-4.6, 0.1) 5-d mean: -2.7 (-5.5, 0.1) **Erythrocytes, % change mean (10^6/μl)** lag0: -0.1 (-0.5, 0.3); lag1: -0.4 (-0.9, 0.2) lag2: -0.4 (-0.9, 0.2); lag3: -0.4 (-0.6, 0.3) 5-d mean: -0.4 (-1.0, 0.2) **Hemoglobin, % change mean (g/dl)** lag0: -0.2 (-0.7, 0.4); lag1: -0.3 (-1.0, 0.4) lag2: -0.1 (-0.9, 0.7); lag3: -0.1 (-0.8, 0.6) 5-d mean: -0.2 (-1.1, 0.6)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ruckerl et al. (2007b) **Period of Study:** May 2003–Jul 2004 **Location:** Athens, Augsburg, Barcelona, Helsinki, Rome, and Stockholm	**Outcome:** Interleukin-6 (IL-6), fibrinogen, C-reactive protein (CRP) **Age Groups:** 35-80 yrs **Study Design:** Repeated measures / longitudinal **N:** 1003 MI survivors **Statistical Analyses:** Mixed-effect models **Covariates:** City-specific confounders (age, sex, BMI); long-term time trend and apparent temperature; RH, time of day, day of week included if adjustment improved model fit **Season:** Long-term time trend **Dose-response Investigated?** Used p-splines to allow for nonparametric exposure-response functions **Statistical Package:** SAS v9.1	**Pollutant:** UFP (n/cm^3) **Averaging Time:** Hourly and 24 h (lag 0-4, mean of lags 0-4, mean of lags 0-1, mean of lags2-3, means of lags 0-3) **Mean (SD):** Presented by city only **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** Central monitoring sites in each city **Copollutant:** SO_2, O_3, NO; NO_2	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** % change in mean blood markers per increase in IQR of air pollutant. IL-6 Lag (IQR): % change in GM (95%CI); Lag 0 (11852): 1.88 (-0.16, 3.97); Lag 1 (11852): -0.67 (-2.56, 1.25); Lag 2 (11852): -2.12 (-4.03 to -0.17); 5-d avg (11003): -0.93 (-3.37, 1.56) Fibrinogen Lag (IQR): % change in AM (95%CI); Lag 0 (11852): 0.40 (-0.40, 1.19); Lag 1 (11852): 0.11 (-0.69, 0.91); Lag 2 (11852): 0.09 (-0.71, 0.90); 5-d avg (11003): 0.50 (-2.20, 3.20) CRP Lag (IQR): % change in GM (95%CI); Lag 0 (11852): 1.33 (-3.05, 5.90); Lag 1 (11852): -1.52 (-4.39, 1.45); Lag 2 (11852): -1.63 (-6.70, 3.71); 5-d avg (11003): -0.08 (-3.78, 3.75)
Reference: Pekkanen et al. (2002) **Period of Study:** Winter 1998 to 1999 **Location:** Helsinki, Finland	**Outcome**: ST Segment Depression (>0.1mV) **Age Groups: Study Design**: Panel of ULTRA Study participants **N**: 45 Subjects, n = 342 biweekly submaximal exercise tests, 72 exercise induced ST Segment Depressions **Statistical Analysis:** Logistic regression / GAM	**Pollutant:** Ultrafine $NC_{0.01\text{-}0.1}$ (n/cm^3) **Averaging Time:** 24 h **Median**: 14,890 **IQR**: 9830 **Monitoring Stations**: 1 **Copollutant:** NO_2, CO, $PM_{2.5}$, $PM_{10\text{-}2.5}$, PM1, ACP	**PM Increment:** IQR **Effect Estimate(s):** NC0.01-0.1: OR = 3.14 (1.56, 6.32), lag 2 **Notes**: The effect was strongest for ACP and $PM_{2.5}$, which in two pollutant models appeared independent. Increases in NO_2 and CO were also associated with increased risk of ST segment depression, but not with coarse particles.
Reference: Peters et al. (2005) **Period of Study:** February 1999–July 2001 **Location:** Augsburg, Germany	**Outcome:** Myocardial infarction **Study Design:** Case-crossover **N:** 691 myocardial infarction patients **Statistical Analysis:** Conditional logistic regression **Dose-response Investigated?**No	**Pollutant:** Ultrafine (TNC) (n/cm^3) **Averaging Time:** 1 h: Median = 10,001; IQR: 7919 24-h: Median = 10,934; IQR: 6276 **Copollutant:** NO_2, SO_2, CO	**PM Increment: Effect Estimate:** 2 h lag: OR = 0.95; 95% CI: 0.84, 1.06 24-h mean, 2-day lag: OR = 1.04; 95% CI: 0.90, 1.20 **Notes:** Examined triggering for MI at various lags before MI onset (up to 6 h before MI, up to 5 days before MI). No statistically significant increases in lagged ultrafine particle concentration were found.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ruckerl et al. (Ruckerl et al., 2007a) **Period of Study:** Oct 2000–Apr 2001 **Location:** Erfurt, Germany	**Outcome (ICD9 and ICD10):** Soluble CD40 ligand (sCD40L), platelets, leukocytes, erythrocytes, hemoglobin **Age Groups:** 50+ yrs **Study Design**: Panel (12 repeated measures at 2-wk intervals) **N:** 57 male subjects with coronary disease **Statistical Analyses:** Fixed effects linear regression models **Covariates:** Long-term time trend, weekday of the visit, temperature, RH, barometric pressure **Season:** Time trend as covariate **Dose-response Investigated?** No **Statistical Package:** SAS v8.2 and S-Plus v6.0	**Pollutant:** UFP **Averaging Time:** 24 h **Mean (SD):** 12,602 (6455) **Percentiles:** 25th: 7326 50th: 11,444 75th: 17,332 **Range (Min, Max):** 328, 4908 **Monitoring Stations:** 1 site **Copollutant:** AP $PM_{2.5}$ PM_{10} NO	**PM Increment:** IQR (10,005 ;; 5-d avg: 6,821) **Effect Estimate [Lower CI, Upper CI]:** **sCD40L, % change GM (pg/mL)** lag 0: 7.1 (0.1, 14.5); lag 1: 0.3 (-6.6, 8.6) lag 2: 0.6 (-5.9, 8.6); lag 3: -8.5 (-15.8, -0.5) 5-d mean: -0.7 (-7.6, 6.8) **Platelets, % change mean (10^3/µl)** lag 0: -1.8 (-3.4, -0.2); lag 1: -1.1 (-2.9, 0.6) lag 2: 1.0 (-2.9, 0.8); lag 3: -2.4(4.5, -0.3) 5-d mean: -2.2 (-4.0, -0.3) **Leukocytes, [10^3/µl]** lag 0: -2.4 (-4.5, -0.2); lag 1: -2.1 (-4.4, 0.2) lag 2: -0.2 (-2.4, 2.8); lag 3: -1.5 (-4.4, 1.4) 5-d mean: -1.6 (-4.1, 0.8)

E.1.2. Cardiovascular Emergency Department Visits and Hospital Admissions

Table E-5. Short-term exposure to PM_{10} and emergency department visits and hospital admissions for cardiovascular outcomes.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Andersen et al. (2008b) **Period of Study:** 5/2001 – 12/2004 **Location:** Copenhagen, Denmark	**Outcome (ICD-10):** CVD, including angina pectoris (I20), myocardial infarction (I21–22), other actue ischemic heart diseases (I24), chronic ischemic heart disease (I25), pulmonary embolism (I26), cardiac arrest (I46), cardiac arrhythmias (I48–48), and heart failure (I50). **Age Groups:** >65 yrs (CVD and RD), 5–18 years (asthma) **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson GAM **Covariates:** Temperature, dew-point temperature, long-term trend, seasonality, influenza, day of the week, public holidays. **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R statistical software (gam procedure, mgcv package) **Lags Considered:** Lag 0 -5 days, 4-day pollutant avg (lag 0 -3) for CVD.	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD; median; IQR; 99th percentile):** 24 (14; 21; 16–29; 72) **Monitoring Stations:** 1 **Copollutant (correlation):** NCtot: r = 0.39 NC100: r = 0.28 NCa12: r = 0.02 NCa23: r = -0.12 NCa57: r = 0.45 NC_{a212}: r = 0.63 $PM_{2.5}$: r = 0.80 CO: r = 0.37 NO_2: r = 0.35 NO_X: r = 0.32 NO_X curbside: r = 0.18 O_3: r = -0.21 **Other variables:** Temperature: r = 0.12 Relative humidity: r = 0.05	**PM Increment:** 13 µg/m³ (IQR) **Relative risk (RR) Estimate [CI]:** CVD hospital admissions (4-day avg, lag 0 -3), age 65+: One-pollutant model: 1.03 [1.01–1.05] Adj for NCtot: 1.04 [1.02–1.06] Adj for NC_{a212}: 1.05 [1.01–1.09] RD hospital admissions (5 day avg, lag 0 -4), age 65+: One-pollutant model: 1.06 [1.02–1.09] Adj for NCtot: 1.05 [1.01–1.10] Adj for NC_{a212}: 1.04 [0.98–1.11] Asthma hospital admissions (6-day avg lag 0–5), age 5 - 18: One-pollutant model: 1.02 [0.93–1.12] Adj for NCtot: 1.01 [0.91–1.12] Adj for NC_{a212}: 0.94 [0.81–1.09] Estimates for individual day lags reported only in figure form (see notes): **Notes:** Figure 2: Relative risks and 95% confidence intervals per IQR in single day concentration (0- to 5-day lag). Summary of Figure 2: CVD: Positive, marginally or statistically significant associations at Lag 0–Lag 2.
Reference: Andersen et al. (2007) **Period of Study:** 5/2002 – 12/2003 (components) **Location:** Copenhagen, Denmark	**Outcome (ICD10): CVD**, including angina pectoris (I20), myocardial infarction (I21 – 22), other actue ischemic heart diseases (I24), chronic ischaemic heart disease (I25), pulmonary embolism (I26), cardiac arrest (I46), cardiac arrhythmias (I48 – 48), and heart failure (I50). **Age Groups Analyzed:** Age >65 **Study Design:** Time series N: 2192 days, 9 Hospitals **Statistical Analyses:** Principal Component Analysis and Constrained Physical Receptor Model (COPREM), Poisson regression, GAM, **Covariates:** Season, day of the wk, public holidays, influenza epidemics and meterology **Season:** All year **Dose-response Investigated?** No **Statistical package:** R, gam/mgcv package **Lags Considered:** 0-6 days	**Pollutant:** Source specific PM_{10} components **Averaging Time:** 24-h **Mean (SD): Percentiles:** 25th: 16 50th (Median): NR 75th: 30 **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: Biomass; r = 0.53 Secondary; r = 0.73 Oil; r = 0.57 Crustal; r = 0.37 Sea salt; r = 0.04 Vehicle; r = 0.02 Notes: Correlations between source specific PM_{10} components presented in paper	**PM Increment:** IQR **RR Estimate** **Respiratory disease (age >65)** Single pollutant model : PM_{10} : 1.027 (1.013, 1.042), IQR=14 PM_{10} (other 5 sources): 1.045 (1.016, 1.074), IQR=13 Biomass : 1.040 (0.009, 1.072), IQR=5.4 Secondary : 1.050 (1.021, 1.081), IQR=6.1 Oil : 1.035 (1.006, 1.065), IQR=2.8 Crustal : 1.054 (1.028, 1.081), IQR=1.8 Sea salt : 0.98 (0.947, 1.017), IQR=2.2 Vehicle : 0.989 (0.949, 1.032), IQR=0.6 **Notes:** 2 pollutant model results for PM_{10} with source specific components and gases also presented in manuscript.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Anderson et al. (2003) **Period of Study:** 1992-1994 **Location:** London, United Kingdom	**Outcome:** All CVD **Age Groups:** 0-15, 15-64, 65-74, 75+ **Study Design:** Time series **N:** NR **Statistical Analyses:** NR **Covariates:** NR **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** NR **Monitoring Stations:** NR **Copollutant:** NR	**PM Increment:** 10th–90th percentile **% Change in Daily IHD Admissions by Age [CI]:** 0-15 yrs: NR 15-64 yrs: 2.6 [0.3,5] 65-74 yrs: 2.5 [0.1,4.9] 75+ yrs: 2.2 [0.2,4.6] **Notes:** RRs are presented in graph form showing little change with increasing age (PM increment of 10 µg/m^3). This article is primarily a systematic literature review of other studies.
Reference: Baccarelli et al. (2007a) **Period of Study:** Jan 1995–Aug 2005 **Location:** Lombardia region, Italy	**Outcome:** Fasting and postmethionine-load total homocysteine (tHcy) **Age Groups:** 11-84 yrs **Study Design:** Cross-sectional/Panel **N:** 1,213 participants **Statistical Analyses:** Generalized additive models **Covariates:** age, sex, BMI, smoking, alcohol, hormone use, temperature, day of the year, and long-term trends **Season:** Adjusted for long-term trends to account for season **Dose-response Investigated?** No **Statistical Package:** R software v2.2.1	**Pollutant:** PM_{10} (some TSP measures used to predict PM_{10}) **Averaging Time:** Hourly concentrations used to calculate 24-h moving averages and 7-day moving averages **Mean (SD):** NR **Percentiles:** 25th: 20.1 50th: 34.1 75th: 52.6 **Range (Min, Max):** Max: 390.0 **Monitoring Stations:** 53 sites **Copollutant:** CO, NO_2; SO_2; O_3	**PM Increment:** IQR **Effect Estimate [Lower CI, Upper CI]:** Estimates (%) per 32.5 µg/m^3 increase in 24-h moving avg of PM_{10} Homocysteine, fasting: 0.4 (-2.4, 3.3) Homocysteine, postmethionine-load: (-1.5, 3.7) Estimates (%) per 25.7m^3 increase in 7-day moving avg of PM_{10} Homocysteine, fasting: 1.0 (-1.9, 3.9) Homocysteine, postmethionine-load: 2.0 (-0.6, 4.7) Estimates of effect (%) on fasting homocysteine per IQR increase in 24-h PM_{10} levels Among smokers: 6.2 (0.0, 12.7) Among non-smokers: -1.6 (-5.5, 2.5) Estimates of effect (%) on postmethionine-load homocysteine per IQR increase in 24-h PM_{10} levels Among smokers: 6.0 (0.5, 11.8) Among non-smokers: -0.1 (-3.6, 3.5)
Reference: Ballester et al. (2006) **Period of Study:** 1995 - 1999 **Location:** 5 Spanish cities: Granada, Huelva, Madrid, Seville, Zaragoza	**Outcome (ICD-9):** All cardiovascular disease (390–459), including all heart diseases (410–414, 427, 428) **Age Groups:** All ages **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson GAMs **Covariates:** daily temperature, barometric pressure, and relative humidity; daily influenza incidence, day of the week, holidays, unusual events (ex. medical strikes), seasonal variation, trend **Dose-response Investigated:** No **Statistical Package:** S-Plus GAM function **Lags Considered:** lag 0 -3 days, lag 0-1 avg	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (10-90th percentile):** overall mean NR. City specific means Granada: 43.2 (24.8, 62.6) Huelva: 38.6 (23.1, 57.3) Madrid: 35.7 (21.4, 54.4) Seville: 41.9 (27.3, 57.6) Zaragoza: 32.8 (17.3, 50.3) **Monitoring Stations:** At least three stations per city (15 +) **Copollutant (correlation):** Summary of the correlation coefficients between each pair of pollutants within cities: BS: r = 0.48; TSP: N/A; NO_2: from r = 0.13 to r = 0.62 (median r = 0.40); SO_2: from r = 0.20 to r = 0.51 (median r = 0.46); CO: from r = 0.34 to r = 0.45 (median r = 0.37); O_3: from r = -0.07 to r = 0.16 (median r = 0.11)	**PM Increment:** 10 µg/m^3 **Relative risk [CI]:** Relative risks are expressed only in the form of figures (see notes). **Percentage change in risk [CI]:** All cardiovascular diseases (avg of lags 0 -1): 0.91% [0.35, 1.47] Heart disease (avg of lags 0 -1) 1.56% [0.82, 2.31] **Notes: Relative risks for the single pollutant models are expressed in Figure 2.** Figure 2: Time sequence of the combined assocation between PM_{10} and hospital admissions for all CVD (A) and heart disease (B). Summary of results: Significant, positive association of PM_{10} with both overall CVD and heart disease hospitalizations at Lag 0 and Lag 1. **Relative risks for two pollutant models are expressed in Figure 3:** Figure 3: Combined estimates of the association between hospital admissions for heart diseases and air pollutants (avg of lags 0–1; adjusted for CO, NO_2, O_3, or SO_2) Summary of results: Significant, positive association remains after adjusting for pollutants.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Bell et al. (2008b) **Period of Study:** 1995 - 2002 **Location:** Taipei, Taiwan	**Outcome (ICD-9):** Hospital admissions for ischemic heart disease (410 , 411, 414), cerebrovascular disease (430–437). **Age Groups:** All **Study Design:** Time series **N:** 6,909 hospital admissions for ischaemic heart diseases, 11,466 for cerebrovascular disease. **Statistical Analyses:** Poisson regression **Covariates:** Day of the week, time, apparent temperature, long-term trends, seasonality **Season:** All **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** lags 0-3 days, avg of lags 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (range; IQR):** 49.1 (12.7–215.5; 27.6) **Monitoring Stations:** Taipei area: 13 monitors Taipei City: 5 monitors Monitors with correlations of 0.75 + for PM_{10}: 12 monitors **Copollutant:** NR	**PM Increment:** 28 µg/m^3 (near IQR) **Percentage increase estimate [95% CI]: Ischemic heart disease:** Taipei area (13 monitors): L0: 1.91 (-1.25, 5.17); L1: 0.39 (-2.73, 3.61); L2: 1.80 (-1.33, 5.04); L3: 2.01 (-1.14, 5.26); L03: 2.91 (-1.52, 7.55) Taipei City (5 monitors): L0: 2.08 (-1.04, 5.30); L1: 0.43 (-2.64, 3.60); L2: 2.17 (-0.92, 5.36); L3: 2.16 (-0.94, 5.36); L03: 3.40 (-1.19, 8.20) Monitors with > = 0.75 between monitor correlations (12 monitors): L0: 1.82 (-1.29, 5.03) ; L1: 0.35 (-2.72, 3.52) ; L2: 1.93 (-1.15, 5.10); L3: 1.93 (-1.16, 5.12) ; L03: 2.86 (-1.63, 7.54) **Cerebrovascular disease:** Taipei area (13 monitors): L0: -1.41 (-3.80, 1.04); L1: -1.95 (4.31, 0.48); L2: 0.77 (-1.62, 3.23); L3: 2.64 (0.21, 5.12); L03: 0.01 (-3.33, 3.47) Taipei City (5 monitors): L0: -1.27 (-3.64, 1.16); L1: -2.13 (-4.47, 0.27); L2: 0.85 (-1.52, 3.28); L3: 2.52 (0.13, 4.97); L03: -0.07 (-3.53, 3.51) Monitors with > = 0.75 between monitor correlations (12 monitors): L0: -1.34 (-3.70, 1.07); L1: -1.98 (-4.31, 0.40) ; L2: 0.80 (-1.56, 3.22) ; L3: 2.61 (0.22, 5.05) ; L03: -0.02 (-3.40, 3.49)
Reference: Chan et al. (2008) **Period of Study:** 1995 - 2002 **Location:** Taipei Metropolitan area, Taiwan	**Outcome (ICD-9):** Emergency visits for ischaemic heart diseases (410–411, 414), cerebrovascular diseases (430–437), and COPD (493, 496) **Age Groups:** All **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson regression models **Covariates:** Year, month, day of week, temperature, dewpoint temperature, $PM_{2.5}$, NO_2 **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 8.0 **Lags Considered:** 0- to 7-day lags	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** High dust events: Pre-dust periods: 45.5 (17.6) Asian dust events: 122.7 (24.4) Low dust events: Pre-dust periods: 59.4 (31.0) Asian dust events: 61.1 (17.8) **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 25.4 µg/m^3 (IQR) **OR [95% CI]:** In environmental conditions without dust storms (results only shown for best-fitting model) Lag 3 days: 1.023 (1.003, 1.041)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Dominici et al. (2004b) **Period of Study:** 1986-1993 **Location:** 10 U.S. cities (Birmingham, Canton, Colorado Springs, Minneapolis/St. Paul, Seattle, Spokane, Chicago, Detroit, New Haven, Pittsburgh) and New York state	**Outcome:** Cardiovascular Diseases **Age Groups:** NR **Study Design:** Time series **N:** ≈758,000 hospitalizations **Statistical Analyses:** GAM (maximum likelihood estimate), Bayesian hierarchical model **Covariates:** Temperature, barometric pressure, relative humidity **Season:** NR **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** Avg of 0-1 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean:** Birmingham: 34.8 Canton: 28.4 Colorado Springs: 27.5 Minneapolis/St. Paul: 28.1 Seattle: 32.2 Spokane: 42.9 Chicago: 36.3 Detroit: 36.7 New Haven: 28.6 Pittsburgh: 36.0 New York: 28.8 **Monitoring Stations:** NR (data obtained from AIRS database) **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **Log RR Estimate [CI]:** Birmingham MLE: 0.28 [-0.28,0.84]; Bayes (combined): 0.55 [0.08,1.02]; Bayes (separate): 0.48 [-0.01,0.96] Canton MLE: 0.59 [-1.46,2.64]; Bayes (combined): 0.70 [0.00,1.39]; Bayes (separate): 0.67 [-0.13,1.47] Colorado Springs MLE: 0.99 [0.50,1.48]; Bayes (combined): 0.84 [0.49,1.19]; Bayes (separate): 0.85 [0.45,1.25] Minneapolis/St. Paul MLE: 0.47 [-1.51,2.45]; Bayes (combined): 0.70 [0.01,1.39]; Bayes (separate): 0.67 [-0.13,1.46] Seattle MLE: 0.63 [0.15,1.11]; Bayes (combined): 0.69 [0.33,1.04]; Bayes (separate): 0.66 [0.28,1.04] Spokane MLE: 0.32 [-0.60,1.24]; Bayes (combined): 0.63 [0.08,1.17]; Bayes (separate): 0.54 [-0.07,1.15]; Chicago MLE: 1.36 [0.26,2.47]; Bayes (combined): 0.87 [0.32,1.41]; Bayes (separate): 0.89 [0.18,1.60] Detroit MLE: 0.91 [0.48,1.35]; Bayes (combined): 0.82 [0.50,1.13]; Bayes (separate): 0.84 [0.47,1.21] New Haven MLE: 0.71 [0.10,1.33]; Bayes (combined): 0.73 [0.33,1.12]; Bayes (separate): 0.70 [0.23,1.17] Pittsburgh MLE: 0.14 [-0.64,0.93]; Bayes (combined): 0.54 [-0.07,1.15]; Bayes (separate): 0.47 [-0.13,1.06] New York Bayes (combined): 0.61 [-0.33,1.55] **Notes:** MLE = Maximum likelihood estimate; CVD mortality RRs were also provided in Table 4. The objective of this study was to develop a model to predict hospital admissions.
Reference: Fung et al., (2005) **Period of Study:** Nov 1, 1995–Dec 31, 2000 **Location:** London, Ontario	**Outcome (ICD-9):** Cardiovascular diseases (410-414, 427-428) **Age Groups:** <65 yrs, 65+ yrs **Study Design:** Time series **N:** 12,947 CVD admissions **Statistical Analyses:** GAM with locally weighted regression smoothers (LOESS) **Covariates:** Maximum and minimum temp, humidity, day of the week, seasonal cycles, secular trends **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** Current to 3-day mean	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 38.0 (5-248) SD = 23.5 **Monitoring Stations:** 4 **Copollutant (correlation):** NO_2: r = 0.30 SO_2: r = 0.24 CO: r = 0.21 O_3: r = 0.53 COH: r = 0.29	**PM Increment:** 26 µg/m^3 **% Change in Daily Admission [CI]:** Age <65 Current day mean: 2.6 [-2.3,7.7] 2-day mean: -1.2 [-7.2,5.1] 3-day mean: -3 [-9.6,4] Age 65+ Current day mean: 0.9 [-2.3,4.2] 2-day mean: -0.9 [-4.8,3.2] 3-day mean: -0.1 [-4.4,4.5]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hanigan et al (2008) **Period of Study:** 1996–2005 (April–November of each year) **Location:** Darwin, Australia	**Outcome:** Daily emergency hospital admissions for total cardiovascular (ICD-9: 390–459; ICD-10: I00–I99), ischemic heart disease (ICD-9: 410–414; ICD-10: I20–I25). **Age Groups:** All **Study Design:** Time series **N:** 8,279 hospital admissions **Statistical Analyses:** Poisson generalized linear models **Covariates:** Indigenous status, time in days, temperature, relative humidity, day of the week, influenza epidemics, change between ICD editions, holidays, yearly population **Season:** April–November (corresponding to the dry season) **Dose-response Investigated?** No **Statistical Package:** R version 2.3.1 **Lags Considered:** 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD; range):** 21.2 (8.2; 55.2) **Monitoring Stations:** N/A (see notes) **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **Percent change [95% CI]:** Overall CVD: Lag 0 (indigenous): -3.78 [-13.4, 6.91] Lag 0 (non-indigenous): -3.43 [-9.00, 2.49] All unstratified associations either negative or zero and not statistically significant. All other results of stratified analysis (by indigenous status) reported in a figure (see notes). **Notes:** Figure 3: Associations between hospitalizations for non-indigenous and indigenous people with estimated ambient PM_{10}. Summary: Confidence intervals were wide, but indigenous people generally had stronger associations with PM_{10} than non-indigenous people. Daily PM_{10} exposure levels were estimated for the population of the city from visibility data using a previousy validated models.
Reference: Henrotin et al. (2007) **Period of Study:** March 1994–December 2004 **Location:** Dijon, France	**Outcome:** Ischemic and hemorrhagic strokes **Age Groups:** All **Study Design:** Bi-directional case-crossover **N:** 1487 (ischemic) and 220 (hemorrhagic) stroke patients **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature, relative humidity, influenza epidemics, holidays **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** STATA software v. 8.2 **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 21.1 (2-103) SD = 11.3 **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **OR Estimate [CI]:** Ischemic stroke Same-day lag: 1.009 [0.930,1.094] 1-day lag: 1.011 [0.998,1.094] 2-day lag: 0.960 [0.889,1.036] 3-day lag: 0.990 [0.919,1.066] Hemorrhagic stroke Same-day lag: 0.901 [0.730,1.111] 1-day lag: 1.014 [0.828,1.241] 2-day lag: 1.100 [0.903,1.339] 3-day lag: 0.991 [0.881,1.212] **Notes:** Ischemic stroke ORs were also categorized into male and female, yielding similar results (none were significant for any lag days).
Reference: Issever et al. (2005) **Period of Study:** 1 Jan, 1997–31 Dec, 2001 **Location:** Istanbul, Turkey	**Outcome:** Acute coronary syndrome (ACS) **Age Groups:** All **Study Design:** Time series **N:** 2889 ACS admissions **Statistical Analyses:** Multiple stepwise regression, Pearson correlation **Covariates:** Humidity, temperature, pressure **Season:** NR **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean:** NR **Monitoring Stations:** 1 **Copollutant (correlation):** ACS: r = 0.37 (p = 0.003) ACS controlled for temp: r = 0.29 (p = 0.02)	**PM Increment:** NR **RR Estimate [CI]:** NR **Notes:** This study focused more on the seasonal change in acute coronary sydrome admissions.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Jalaludin et al. (2006) **Period of Study:** 1 Jan, 1997–31 Dec, 2001 **Location:** Sydney, Australia	**Outcome (ICD-9):** Cardiovascular disease (390-459), cardiac disease (390-429), ischemic heart disease (410-413) and cerebrovascular disease or stroke (430-438) **Age Groups:** 65+ yrs **Study Design:** Time series **N:** NR **Statistical Analyses:** GAM, GLM **Covariates:** Temperature, humidity **Season:** Warm (Nov-Apr) and cool (May-Oct) **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 16.8 (3.8-103.9) SD = 7.2 **Monitoring Stations:** 14 **Copollutant (correlation):** Warm BSP: r = 0.82; $PM_{2.5}$: r = 0.89; O_3: r = 0.59; NO_2: r = 0.44; CO: r = 0.31; SO_2: r = 0.37 Cool BSP: r = 0.75; $PM_{2.5}$: r = 0.88; O_3: r = 0.22; NO_2: r = 0.67; CO: r = 0.48; SO_2: r = 0.46 **Other variables:** Warm Temp: r = 0.36; Rel humidity: r = -0.25 Cool Temp: r = 0.13; Rel humidity: r = 0.05	**PM Increment:** 7.8 µg/m³ (IQR) **Percent Change Estimate [CI]:** All CVD Same-day lag: 0.72 [-0.14,1.60] ; Avg 0-1 day lag: 0.25 [-0.61,1.12] ; Cool (same-day lag): 1.34 [0.08,2.61] ; Warm (same-day lag): 0.33 [-0.83,1.50] Cardiac disease Same-day lag: 1.15 [0.14,2.18] ; Avg 0-1 day lag: 0.97 [-0.07,2.02] ; Cool (same-day lag): 1.35 [-0.16,2.89] ; Warm (same-day lag): 1.12 [-0.23,2.48] Ischemic heart disease Same-day lag: 0.59 [-0.95,2.17] ; Avg 0-1 day lag: 0.61 [-0.95,2.20] ; Cool (same-day lag): 0.33 [-2.00,2.72] ; Warm (same-day lag): 0.79 [-1.23,2.85] Stroke Same-day lag: -1.66 [-3.48,0.20] ; Avg 0-1 day lag: -2.05 [-3.88,-0.20] ; Cool (same-day lag): 0.46 [-2.17,3.17] ; Warm (same-day lag): -3.49 [-5.97,-0.95] **Notes:** All other lag-day ORs were provided, yet none were significant. Percent change in ED attendance was also reported graphically (Fig 1-5).
Reference: Johnston et al. (2007) **Period of Study:** 2000, 2004, 2005 (April–November of each year) **Location:** Darwin, Australia	**Outcome (ICD-10):** All cardiovascular conditions (I00–I99), including ischemic heart disease (I20–I25). **Age Groups:** All **Study Design:** Case-crossover **N:** 2466 emergency admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Weekly influenza rates, temperature, humidity, days with rainfall >5mm, public holidays, school holiday periods (for respiratory conditions only) **Season:** April–November (dry season) **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0–3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median (IQR, 10th–90th percentile, range):** 17.4 (13.6–22.3; 10.3–27.7; 1.1–70.0) **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 10 µg/m³ **OR Estimate [95% CI]: All respiratory conditions:** **Ischemic heart disease:** Lag 0: 0.82 [0.68–0.98]; Lag 0 (non-indigenous): 0.75 [0.61–0.93]; Lag 3 (indigenous): 1.71 [1.14–2.55] **Notes:** **Figure 5:** OR and 95% CI for hospital admissions for cardiovascular conditions. Summary: Negative associations in overall study population and in non-indigenous people. Positve associations in Indigenous people at Lag 1, Lag 2, and Lag 3. **Figure 6:** OR and 95% CI for hospital admissions for ischaemic heart disease. Summary: Negative associations in overall study population and non-indigenous people. Positive association in indigenous people.
Reference: Koken et al. (2003) **Period of Study:** July and August, 1993-1997 **Location:** Denver, Colorado	**Outcome (ICD-9):** Acute myocardial infarction (410.00-410.92), pulmonary heart disease (416.0-416.9), cardiac dysrhythmias (427.0-427.9), congestive heart failure (428.0) **Age Groups:** 65+ yrs **Study Design:** Time series **N:** 298 days **Statistical Analyses:** GLM, GEE **Covariates:** Maximum temp and dew point temp **Season:** NR **Dose-response Investigated:** Yes **Statistical Package:** SAS (PROC GENMOD) **Lags Considered:** 0-4 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 24.2 (7.0-51.6) SD = 6.25 **Monitoring Stations:** 3 **Copollutant (correlation):** NO_2: r = 0.56 SO_2: r = 0.36 O_3: r = 0.03 CO: r = 0.25 **Other variables:** Max temp: r = 0.38 Dew point temp: r = -0.24	**PM Increment:** 8.0 µg/m³ (IQR) **Percent Change Estimate [CI]:** No PM data reported

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lanki et al., (2006a) **Period of Study:** 1992-2000 **Location:** Augsburg, Barcelona, Helsinki, Rome, and Stockholm	**Outcome (ICD-9):** Acute myocardial infarction (410; ICD-10: I21, I22) **Age Groups:** 35+ yrs, <75 yrs, 75+ yrs **Study Design:** Time series **N:** 26,854 hospitalizations **Statistical Analyses:** GAM **Covariates:** Temperature, barometric pressure **Season:** Warm (April-September) and cold (October-March) **Dose-response Investigated:** No **Statistical Package:** R package mgcv 0.9-5 **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median:** Augsburg: 43.5 Barcelona: 57.4 Helsinki: 21.0 Rome: 48.5 Stockholm: 12.5 **Copollutant (correlation):** Augsburg PNC: r = 0.53; CO: r = 0.56; NO_2: r = 0.64; O_3: r = 0.43 Barcelona: PNC: r = 0.38; CO: r = 0.44; NO_2: r = 0.48; O_3: r = 0.01 Helsinki: PNC: r = 0.45; CO: r = 0.21; NO_2: r = 0.40; O_3: r = 0.40 Rome: PNC: r = 0.32; CO: r = 0.41; NO_2: r = 0.29; O_3: r = 0.59 Stockholm: PNC: r = 0.06; CO: r = 0.41; NO_2: r = 0.29; O_3: r = 0.59	**PM Increment:** 10 µg/m^3 **Pooled Rate Ratio [CI]:** All 5 cities (35+ yrs) Same-day lag: 1.003 [0.995,1.011] ; 1-day lag: 1.001 [0.990,1.011] ; 2-day lag: 1.002 [0.994,1.010] ; 3-day lag: 1.002 [0.991,1.013] 3 cities with hospital discharge register (35+ yrs) Same-day lag: 1.003 [0.994,1.012] ; 1-day lag: 0.997 [0.988,1.006] ; 2-day lag: 1.003 [0.995,1.012] ; 3-day lag: 1.003 [0.986,1.020] Warm season (35+ yrs) Same-day lag: 1.006 [0.990,1.022] ; 1-day lag: 1.000 [0.985,1.016] ; 2-day lag: 1.005 [0.990,1.020] ; 3-day lag: 1.010 [0.995,1.025] Cold season (35+ yrs) Same-day lag: 1.001 [0.991,1.012] ; 1-day lag: 0.998 [0.987,1.009] ; 2-day lag: 1.001 [0.991,1.012] ; 3-day lag: 0.991 [0.981,1.002] Age >75 Non-fatal Same-day lag: 1.012 [0.995,1.029] ; 1-day lag: 1.000 [0.983,1.017] ; 2-day lag: 0.999 [0.982,1.017] ; 3-day lag: 1.001 [0.984,1.018] ; Fatal Same-day lag: 1.009 [0.985,1.034] ; 1-day lag: 0.998 [0.974,1.023] ; 2-day lag: 1.003 [0.978,1.028] ; 3-day lag: 1.018 [0.975,1.063] **Notes:** Pooled rate ratios were also provided for groups <75 yielding similar results to the overall 3-city data.
Reference: Lee et al., (2003) **Period of Study:** 1 Dec, 1997–31 Dec, 1999 **Location:** Seoul, Korea	**Outcome (ICD-10):** Angina pectoris (I20), acute/subsequent myocardial infarction (I21-I23), other acute ischemic heart diseases (I24) **Age Groups:** All ages, 64+ yrs **Study Design:** Time series **N:** 822 days **Statistical Analyses:** GAM with LOESS, Pearson correlation **Covariates:** Temperature, relative humidity, day of the week **Season:** Summer (Jun-Aug) and winter **Dose-response Investigated:** Yes **Statistical Package:** NR **Lags Considered:** 0-6 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 64.0 (31.8) **Monitoring Stations:** 27 **Copollutant (correlation):** All year SO_2: r = 0.59; NO_2: r = 0.74; O_3: r = 0.11; CO: r = 0.60 Temp: r = -0.07; Humidity: r = 0.02 Summer SO_2: r = 0.61; NO_2: r = 0.73; O_3: r = 0.64; CO: r = 0.55 Temp: r = -0.01; Humidity: r = -0.11	**PM Increment:** 40.4 µg/m^3 (IQR) **RR Estimate [CI]: All year** All ages: 0.99 [0.96,1.01] 64+ yrs: 1.05 [1.01,1.10] **Summer** All ages: 1.03 [0.97,1.09] 64+ yrs: 1.09 [1.00,1.19] **Two-pollutant model** CO (1 ppm IQI): 1.04 [0.98,1.11] O_3 (21.7 ppb IQI): 1.07 [1.03,1.11] NO_2 (14.6 ppb IQI): 1.09 [1.02,1.16] SO_2 (4.4 ppb): 0.98 [0.94,1.03]
Reference: Larrieu et al. (2007) **Period of Study:** 1998 - 2003 **Location:** 8 French urban area: Bordeaux, Le Havre, Lille, Lyon, Marseille, Paris, Rouen, and Toulouse	**Outcome (ICD-10):** Hospital admissions for cardiovascular disease (I00–I99), cardiac disease (I00–I52), ischemic heart disease (I20–I25), and stroke (cerebrovascular disease: I60–64 and transient ischemic attack: G45–G46). **Age Groups:** All, and 65 + **Study Design:** Time series **N: Statistical Analyses:** generalized additive Poisson regression **Covariates:** Temperature, holidays, influenza epidemic periods, long-term trend, season, day of the week, **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R 2.2.1 **Lags Considered:** 0 -1 day lag (mean)	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean:** Bordeaux: 21.0 Le Havre: 21.7 Lille: 22.1 Lyon: 24.6 Marseille: 28.9 Paris: 23.1 Rouen: 21.2 Toulouse: 21.8 **Monitoring Stations:** 32 **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **ERR [95% CI]:** CVD: All ages: 0.7 [0.1, 1.2] 65+ years: 1.1 [0.5, 1.7] Cardiac diseases: All ages: 0.8 [0.2, 1.4] 65+ years: 1.5 [0.7, 2.2] Ischemic heart diseases: All ages: 1.9 [0.8, 3.0] 65+ years: 2.9 [1.5, 4.3] Strokes: All ages: 0.2 [-1.6, 1.9] 65+ years: 0.8 [-0.9, 2.5]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Le Tertre et al. (2002) **Period of Study:** 1990-1997 **Location:** Barcelona, Birmingham, London, Milan, the Netherlands, Paris, Rome, and Stockholm	**Outcome (ICD-9):** Cardiac diseases (390-429), ischemic heart disease (410-413), and stroke (430-438) **Age Groups:** <65 yrs, 65+ yrs **Study Design:** Time series **N:** NR **Statistical Analyses:** GAM **Covariates:** Long term trend, season, days of the week, holidays, influenza epidemics, temperature, and humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** S-Plus **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Barcelona: 55.7 (18.4) Birmingham: 24.8 (13.1) London: 28.4 (12.3) Milan: 51.5 (22.7) Netherlands: 39.5 (19.9) Paris: 22.7 (10.8) Rome: 52.5 (12.9) Stockholm: 15.5 (7.2) **Monitoring Stations:** 1-12 **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **Pooled Percent Increase [CI]:** Cardiac (all ages) Fixed: 0.5 [0.3,0.7]; Random: 0.5 [0.2,0.8] Cardiac (over 65) Fixed: 0.7 [0.4,1.0]; Random: 0.7 [0.4,1.0] IHD (<65) Fixed: 0.3 [-0.1,0.6] ; Random: 0.3 [-0.2,0.7] IHD (over 65) Fixed: 0.6 [0.3,0.8] ;Random: 0.8 [0.3,1.2] Stroke (over 65) Fixed: 0.0 [-0.3,0.3] ;Random: 0.0 [-0.3,0.3] Deaths: Cardiac: 0.5 [0.2,0.8] ;Cardiac (65+): 0.7 [0.4,1.0] IHD (65+): 0.8 [0.3,1.2] **Notes:** Estimated percentage increases are also provided by city for cardiac admissions and ischemic heart disease in Fig 1-3.
Reference: Mann et al. (2002) **Period of Study:** 1988-1995 **Location:** South Coast Air Basin, California	**Outcome (ICD-9):** Ischemic heart disease (410-414), secondary congestive heart failure (sCHF) (428), and secondary arrhythmia (sARR) (426, 427) **Age Groups:** All, 40-59 yrs, >60 yrs **Study Design:** Time series **N:** 54,863 IHD admissions **Statistical Analyses:** GAM **Covariates:** Temperature, day of the week, relative humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** S-Plus **Lags Considered:** 0-5 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 43.7 (0.22-251); SD = 27.7 **Monitoring Stations:** 20 **Copollutant (correlation):** Region 1: CO: r = 0.28; O_3: r = 0.20; NO_2: r = 0.36 Region 2: CO: r = 0.15; O_3: r = 0.57; NO_2: r = 0.53 Region 3: CO: r = 0.36; O_3: r = 0.30; NO_2: r = 0.46 Region 4: CO: r = 0.27; O_3: r = 0.33; NO_2: r = 0.50 Region 5: CO: r = 0.40; O_3: r = 0.43; NO_2: r = 0.53 Region 6: CO: r = 0.33; O_3: r = 0.20; NO_2: r = 0.42 Region 7: CO: r = 0.28; O_3: r = 0.48; NO_2: r = 0.60	**PM Increment:** 10 µg/m^3 **Percent Change in IHD Admissions [CI]:** Secondary ARR Same-day lag: 0.59 [-0.71,1.91] 1-day lag: 0.46 [-0.86,1.80] 2-day lag: -0.04 [-1.37,1.31] Secondary CHF Same-day lag: -0.62 [-1.77,0.55] 1-day lag: -0.45 [-1.60,0.71] 2-day lag: -0.36 [-1.52,0.82] No secondary diagnosis Same-day lag: -0.25 [-1.23,0.75] 1-day lag: 0.04 [-0.97,1.06] 2-day lag: 0.18 [-0.82,1.20] All IHD admissions: 0.19 [-0.576,0.955] MI admissions: -0.10 [-1.33,1.12] Other acute IHD admissions: 0.36 [-0.87,1.60]
Reference: Metzger et al. (2004) **Period of Study:** August 1993–August 2000 **Location:** Atlanta Metropolitan area (Georgia)	**Outcome (ICD-9):** Emergency visits for ischemic heart disease (410–414), cardiac dysrhythmias (427), cardiac arrest (427.5), congestive heart failure (428), peripheral vascular and cerebrovascular disease (433-437, 440, 443-444, 451–453), atherosclerosis (440), and stroke (436). **Age Groups:** All **Study Design:** Time series **N:** 4,407,535 emergency department visits **Statistical Analyses:** Poisson generalized linear modeling **Covariates:** Day of the week, hospital entry and exit indicator variables, federally observed holidays, temporal trends, temperature, dew point temperature **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 3-day moving avg, lags 0 -7	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median (10% - 90% range):** 26.3 (13.2, 44.7) **Monitoring Stations:** NR **Copollutant (correlation):** O_3: r = 0.59; NO_2: r = 0.49; CO: r = 0.47; SO_2: r = 0.20; $PM_{2.5}$: r = 0.84; $PM_{10-2.5}$: r = 0.59; UFP: r = -0.13; $PM_{2.5}$ water-sol; metals: r = 0.74; $PM_{2.5}$ sulfates: r = 0.74; $PM_{2.5}$ acidity: r = 0.68; $PM_{2.5}$ OC: r = 0.69; $PM_{2.5}$EC: r = 0.56; oxygenated hydrocarbon: r = 0.58 **Other variables:** Temperature: r = 0.58 Dew point: r = 0.44	**PM Increment:** 10 µg/m^3 (approximately 1 SD) **RR [95% CI]:** For 3-day moving avg: All CVD: 1.009 [0.998, 1.019] Dysrhythmia: 1.008 [0.989, 1.029] Congestive heart failure: 0.992 [0.968–1.016] Ischemic heart disease: 1.011 [0.992–1.030] Peripheral vascular and cerebrovascular disease: 1.020 [0.999–1.043] **Notes:** Results for Lags 0–7 expressed in figures Figure 1: RR (95% CI) for single-day lag models for the association of ER visits for CVD with daily ambient PM_{10}. Summary: Statistically significant association at Lag 0. Positive but not statistically significant association at Lag 1. Negative, statistically significant association at Lag 7, and negative associations at Lag 2 through Lag 6.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Middleton et al. (2008) **Period of Study:** 1995–1998, 2000 - 2004 **Location:** Nicosia, Cyprus	**Outcome:** Hospital admissions for all cardiovascular disease (ICD-10: I00–I52). **Age Groups:** All, also stratified by age (<15 vs. >15 years) **Study Design:** Time series **Statistical Analyses:** Generalized additive Poisson models **Covariates:** Seasonality, day of the week, long- and short-term trend, temperature, relative humidity **Dose-response Investigated:** No **Statistical Package:** STATA SE 9.0, R 2.2.0 **Lags Considered:** Lag 0 -2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD; median; 5% - 95%; range):** Cold: 57.6 (52.5; 50.8; 20.0–103.0; 5.0–1370.6) Warm: 53.4 (50.5; 30.7; 32.0–77.6; 18.4–933.5) **Monitoring Stations:** 2 **Copollutant:** NR	**PM Increment:** 10 µg/m^3, and across quartiles of increasing levels of PM_{10} **Percentage increase estimate [CI]: All age/sex groups (Lag 0):** All admissions: 0.85 (0.55, 1.15); Cardiovascular: 1.18 (-0.01, 2.37); **Nicosia residents (Lag 0):** Cardiovascular: 0.73 (-0.62, 2.09); **Males (Lag 0):** All admissions: 0.96 (0.54, 1.39); Cardiovascular: 1.27 (-0.15, 2.72); **Females (Lag 0):** All admissions: 0.74 (0.31, 1.18); Cardiovascular: 0.99 (-1.11, 3.14); **Aged <15 years (Lag 0):** All admissions: 0.47 (-0.13, 1.08); **Aged >15 years (Lag 0):** All admissions: 0.98 (0.63, 1.33);
Reference: Peel et al., (2007) **Period of Study:** 1 Jan, 1993–31 Aug, 2000 **Location:** Atlanta, GA	**Outcome (ICD-9):** Ischemic heart disease (410-414), dysrhythmia (427), congestive heart failure (428), peripheral vascular and cerebrovascular disease (433-437, 440, 443, 444, 451-453) **Age Groups:** All **Study Design:** Case-crossover **N:** 4,407,535 ED visits **Statistical Analyses:** Conditional logistic regression **Covariates:** Avg temp and dew point temp **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS v. 9.1 **Lags Considered:** 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Daily levels: 27.9 (12.3) Diff in case and control day avgs: 9.1 (7.5) **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **OR Estimate [CI]:** All CVD: 1.010 [1.000,1.020] IHD: 1.009 [0.991,1.027] Dysrhythmia: 1.011 [0.991, 1.031] Peripheral/Cerebrovascular disease: 1.017 [0.996,1.039] CHF: 1.001 [0.978,1.024] With comorbid hypertension IHD: 1.003 [0.973,1.034] Dysrhythmia: 1.037 [0.988,1.089] Peripheral/Cerebrovascular disease: 1.024 [0.990,1.060] CHF: 1.041 [0.999,1.084] No comorbid hypertension IHD: 1.013 [0.991,1.036] Dysrhythmia: 1.006 [0.985,1.028] Peripheral/Cerebrovascular disease: 1.013 [0.987,1.040] CHF: 0.982 [0.955,1.010] With comorbid diabetes IHD: 1.022 [0.979,1.067] Dysrhythmia: 1.049 [0.968,1.137] Peripheral/Cerebrovascular disease: 1.016 [0.965,1.069] CHF: 1.029 [0.982,1.078] No comorbid diabetes IHD: 1.006 [0.987,1.026] Dysrhythmia: 1.009 [0.989,1.029] Peripheral/Cerebrovascular disease: 1.018 [0.995,1.042] CHF: 0.992 [0.966,1.019] With comorbid COPD IHD: 0.981 [0.921,1.044] Dysrhythmia: 0.984 [0.889,1.088] Peripheral/Cerebrovascular disease: 1.086 [0.998,1.181] CHF: 1.010 [0.954,1.069] No comorbid COPD IHD: 1.012 [0.993,1.031] Dysrhythmia: 1.012 [0.992,1.032] Peripheral/Cerebrovascular disease: 1.013 [0.991,1.035] CHF: 0.999 [0.974,1.025]
Reference: Peters et al. (2005) **Period of Study:** February 1999–July 2001 **Location:** Augsburg, Germany	**Outcome:** Myocardial infarction **Study Design:** Case-crossover **N:** 691 myocardial infarction patients **Statistical Analysis:** Conditional logistic regression **Dose-response Investigated?**No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h: Median = 14.5 IQR: 9.1 24-h: Median = 14.9 IQR: 7.7 **Copollutant:** NO_2, SO_2, CO	**Effect Estimate:** 2-h lag: OR = 0.93; 95% CI: 0,83, 1.04 24-h mean, 2-day lag: OR = 1.18; 95% CI: 1.03, 1.34 **Notes:** Examined triggering for MI at various lags before MI onset (up to 6 h before MI, up to 5 days before MI). $PM_{2.5}$ levels 2 days before MI onset were associated with increased risk of MI, but not on the concurrent day, or lags 1, 3, 4, or 5. These findings are consistent with the prior Boston MI study for a 1- to 2-day lagged effect of $PM_{2.5}$.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Pope et al., (2006) **Period of Study:** 1994 - 2004 **Location:** Wasatch Front area, Utah	**Outcome:** Myocardial infarction or unstable angina (ICD codes not reported) **Age Groups:** All **Study Design:** Case-crossover **N:** 12,865 patients who underwent coronary arteriography **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and dewpoint temperature **Season:** NR **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0- to 3-day lag, 2- to 4-day lagged moving averages	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD; maximum):** Ogden: 28.5 (16.5; 163) SLC Hawthorne: 27.7 (17.4; 162) Provo/Orem, Lindom: 32.7 (21.1; 240) SLC AMC: 35.9 (20.4; 161) SLC North: 45.1 (25.1; 199) **Monitoring Stations:** 5 **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **Percent increase in risk [95% CI]:** Results summarized in figure (see notes). **Notes:** Figure 1: Percent increase in risk (and 95% CI) of acute coronary events associated with 10 µg/m^3 of PM_{10} for different lag structures. Summary of Figure 1: Positive, statistically significant or marginally significant associations between association seen for Lag 0, Lag 1; and 2-, 3-, and 4-day moving averages. Non-statistically significant associations
Reference: Pope et al. (2006) **Period of Study:** 1994 - 2004 **Location:** Wasatch Front, Utah	**Outcome:** Acute ischemic heart disease **Study Design:** Case-crossover study (time-stratified control selection) **N: Statistical Analysis:** Conditional logistic regression	**Pollutant:** $PM_{2.5}$ (FRM) **Averaging Time:** 24 h **Mean (SD):** Site 1: 10.1 Site 2: 10.8 Site 3: 11.3 **Monitoring Stations:** 3 **Copollutant:** PM_{10} (FRM) measured at 4 monitoring sites	**PM Increment:** 10 µg/m^3 **Effect Estimate:** For same-day increase in $PM_{2.5}$: OR = 1.045; 95% CI: 1.011, 1.080 **Notes:** Case-crossover study (time-stratified control selection) triggering of acute ischemic heart disease by ambient $PM_{2.5}$ concentrations on the same and previous 3 days. $PM_{2.5}$ measured at 3 sites and estimated for missing days. Effect estimates were larger for those with angiographically demonstrated coronary artery disease.
Reference: Tolbert et al. (2007) **Period of Study:** 1993 - 2004 **Location:** Atlanta Metropolitan area, Georgia	**Outcome (ICD-9):** Combined CVD group, including: Ischemic heart disease (410–414), cardiac dysrhythmias (427), congestive heart failure (428), and peripheral vascular and cardiovascular disease (433–437, 440, 443–445, and 451–453). **Age Groups:** All **Study Design:** Time series **N:** 10,234,490 ER visits (283,360 and 1,072,429 visits included in the CVD and RD groups, respectively) **Statistical Analyses:** Poisson generalized linear models **Covariates:** Long-term temporal trends, season (for RD outcome), temperature, dew point, days of week, federal holidays, hospital entry and exit **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 9.1 **Lags Considered:** 3-day moving avg(lag 0 -2)	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (median; IQR, range, 10th–90th percentiles):** 26.6 (24.8; 17.5–33.8; 0.5–98.4; 12.3–42.8) **Monitoring Stations:** NR **Copollutant (correlation):** O_3: r = 0.59 NO_2: r = 0.53 CO: r = 0.51 SO_2: r = 0.21 Coarse PM: r = 0.67 $PM_{2.5}$: r = 0.84 $PM_{2.5}$ SO4: r = 0.69 $PM_{2.5}$ EC: r = 0.61 $PM_{2.5}$ OC: r = 0.65 $PM_{2.5}$ TC: r = 0.67 $PM_{2.5}$ water-sol metals:r = 0.73 OHC: r = 0.53	**PM Increment:** 16.30 µg/m^3 (IQR) **Risk ratio [95% CI]:** Single pollutant models: CVD: 1.008 (0.997–1.020)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Tsai et al. (2003b) **Period of Study:** 1997-2000 **Location:** Kaohsiung, Taiwan	**Outcome (ICD-9):** Cerebrovascular diseases (430-438), subarachnoid hemorrhagic stroke (430), primary intracerebral hemorrhage (431-432), ischemic stroke (433-435), and others (436-438) **Age Groups:** All **Study Design:** Case-crossover **N:** 23,179 admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** Cumulative 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 78.82 (20.50-217.33) **Monitoring Stations:** 6 **Copollutant:** NR	**PM Increment:** 66.33 µg/m^3 (IQR) **OR Estimate [CI]: Two-pollutant model (all stroke admissions)** Primary intracerebral hemorrhage (PIH) Adj for SO_2: 1.55 [1.31,1.83] ; Adj for NO_2: 1.28 [1.01,1.61] ; Adj for CO: 1.45 [1.20,1.74] ; Adj for O_3: 1.56 [1.27,1.91] Ischemic stroke (IS) Adj for SO_2: 1.46 [1.32,1.61] ; Adj for NO_2: 1.16 [1.01,1.34] ; Adj for CO: 1.35 [1.21,1.51] ; Adj for O_3: 1.51 [1.34,1.71] **Single-pollutant model** Temp >20ºC PIH: 1.54 [1.31,1.81] ; IS: 1.46 [1.32,1.61] Temp <20ºC PIH: 0.82 [0.48,1.40] ; IS: 0.97 [0.65,1.44]
Reference: Ulirsch et al. (2007) **Period of Study:** November 1994–March 2000 **Location:** Pocatello, Idaho and Chubbuck, Idaho	**Outcome (ICD-9):** CVD (390-429). **Age Groups:** 65 + **Study Design:** Time series **N:** 39,347 admissions/visits **Statistical Analyses:** Log-linear generalized linear models **Covariates:** Time, temperature, relative humidity, influenza, day of the week **Season:** All, and separate analyses were performed for the all-age group for cool months (October–March) vs. warm months (April–September). **Dose-response Investigated:** No **Statistical Package:** S-plus version 6.1 **Lags Considered:** 0- to 4-day lags, and mean of days 0 -4	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (range; 10th - 90th percentiles):** 24.2 (3.0–183.0; 10.5–40.7) **Monitoring Stations:** 4 **Copollutant (correlation):** NO_2: r = 0.47 **Other variables:** Correlation for PM_{10} between monitors: r = 0.42–0.87	**PM Increment:** 50 µg/m^3 , and 24.3 µg/m^3 (mean increase in PM_{10}) **Mean percent of change (% change in the mean number of daily admissions and visits) [95% CI]:** **For 24.3** µg/m^3 **increase in** PM_{10}**:** All-age RD/CVD: 3.7 [1.3, 6.3] ; All-age CVD (Lag 0): -0.02 [-5.9, 6.3] ; All-age CVD (Lag 1): 1.9 [-4.1, 8.4] ; All-age CVD (Lag 2): -3.1 [-9.1, 3.4] ; All-age CVD (Lag 3): 0.5 [-5.6, 6.9] ; All-age CVD (Lag 4): -1.7 [-4.3, 0.9] ; Lag 0–4 days: -0.5 [-8.0, 7.6] **For 50** µg/m^3 **increase in** PM_{10} (single pollutant models, CIs not given): All-age respiratory disease: 8.4 ; All-age RD/CVD: 7.9 ; 18-64 years RD: 7.2 ; All-age CVD (Lag 3): 1.0 ; All-age CVD (Lag 4): -3.6 ; All-age CVD (Lag 0 -4): -1.1 **Notes:** Included urgent care visits as well as emergency department visits and hospital admissions.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2006) **Period of Study:** April, 1992 –March, 2002 **Location:** Edmonton, Canada	**Outcome (ICD-9):** Stroke (430-438), including ischemic stroke (434-436), hemorrhagic stroke (430,432), and transient ischemic attacks (TIA) (435). **Age Groups:** 65+ yrs **Study Design:** Case-crossover **N:** 12,422 visits **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and relative humidity **Season:** summer (Apr-Sep), winter (Oct-Mar) **Dose-response Investigated:** No **Statistical Package:** SAS (PHREG) **Lags Considered:** 0-, 1-, and 3-day	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** All year: 24.2 (14.8) Summer: 25.9 (16.4) Winter: 22.6 (12.9) **Monitoring Stations:** 3 **Copollutant (correlation):** All year SO_2: r = 0.19; NO_2: r = 0.34; CO: r = 0.30; O_3-mean: r = 0.07; O_3-max: r = 0.22; $PM_{2.5}$: r = 0.79 Summer SO_2: r = 0.18; NO_2: r = 0.57; CO: r = 0.38; O_3-mean: r = 0.20; O_3-max: r = 0.40; $PM_{2.5}$: r = 0.85 Winter SO_2: r = 0.27; NO_2: r = 0.48; CO: r = 0.53; O_3-mean: r = -0.26; O_3-max: r = -0.09; $PM_{2.5}$: r = 0.70	**PM Increment:** µg/m^3 (IQR) All year: 16.0 Summer: 17.5 Winter: 16.0 Adjusted OR Estimate [CI]: Acute ischemic stroke All year Same-day lag: 0.98 [0.94,1.03] ; 1-day lag: 1.00 [0.96,1.05] ; 3-day lag: 0.99 [.93,1.05] summer Same-day lag: 0.93 [0.87,1.00] ; 1-day lag: 1.01 [0.94,1.08] ; 3-day lag: 0.96 [0.88,1.04] Winter Same-day lag: 1.04 [0.97,1.11] ; 1-day lag: 1.00 [0.94,1.06] ; 3-day lag: 1.05 [0.95,1.15] Hemorrhagic stroke All year Same-day lag: 1.01 [0.90,1.12] ; 1-day lag: 1.03 [0.93,1.15] ; 3-day lag: 1.13 [0.98,1.30] summer Same-day lag: 1.02 [0.88,1.20] ; 1-day lag: 1.07 [0.91,1.26] ; 3-day lag: 1.20 [0.98,1.46] Winter Same-day lag: 1.05 [0.90,1.22] ; 1-day lag: 1.04 [0.91,1.19] ; 3-day lag: 1.11 [0.90,1.37] Transient cerebral ischemic attack All year Same-day lag: 0.96 [0.90,1.02] ; 1-day lag: 0.99 [0.94,1.05] ; 3-day lag: 0.94 [0.87,1.01] summer Same-day lag: 0.97 [0.89,1.09] ; 1-day lag: 0.99 [0.91,1.08] ; 3-day lag: 0.94 [0.84,1.04] Winter Same-day lag: 0.95 [0.87,1.04] ; 1-day lag: 0.99 [0.92,1.07] ; 3-day lag: 0.93 [0.83,1.05] **Notes:** Adjusted ORs are provided for an IQR increase in the 3-day mean in Fig 1-4 for single and two-pollutant models.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: von Klot et al. (2005) **Period of Study:** 1992-2001 **Location:** Augsburg, Germany; Barcelona, Spain; Helsinki, Finland; Rome, Italy; Stockholm, Sweden	**Outcome (ICD-9):** Acute myocardial infarction (410; ICD-10: I21-I22), angina pectoris (411, 413; ICD-10: I20, I24), dysrhythmia (427; ICD-10: I46.0, 46.9, I47-I49, R00.1, R00.8), heart failure (428; ICD-10: 150) **Age Groups:** 35+ yrs **Study Design:** Cohort **N:** 22,006 MI survivors **Statistical Analyses:** GAM, Spearman correlation **Covariates:** Temperature, dew point temp, avg barometric pressure, relative humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (5th–95th percentile):** Augsburg: 44.7 (16.8-81.4) Barcelona: 52.2 (25.3-89.2) Helsinki: 25.3 (9.5-57.6) Rome: 51.1 (23.3-89.4) Stockholm: 14.6 (6.4-30.0) **Monitoring Stations:** NR **Copollutant (correlation):** Augsburg PNC: r = 0.52 CO: r = 0.57 NO_2: r = 0.64 O_3: r = -0.32 Barcelona PNC: r = 0.29 CO: r = 0.39 NO_2: r = 0.36 O_3: r = -0.14 Helsinki PNC: r = 0.46 CO: r = 0.21 NO_2: r = 0.4; O_3: r = 0.02 Rome PNC: r = 0.3 CO: r = 0.31 NO_2: r = 0.48 O_3: r = -0.22 Stockholm PNC: r = 0.06 CO: r = 0.38 NO_2: r = 0.29 O_3: r = 0.15	**PM Increment:** 10 µg/m³ **Pooled RR Estimate [CI]:** All cardiac admissions: 1.021 [1.005,1.048] Myocardial infarction: 1.026 [0.995,1.058] Angina pectoris: 1.008 [0.986,1.032] **Notes:** Rate ratios for 0-3 day lags are provided in graphical form (Fig 1). Same-day levels were significantly associated with cardiac readmissions.
Reference: (2005c) et al., 2005 **Period of Study:** 1 Jan, 1987–30 Nov, 1999 **Location:** Pittsburgh, Pennsylvania	**Outcome (ICD-9):** Congestive heart failure (428.0-428.1) **Age Groups:** 65+ yrs **Study Design:** Case-crossover **N:** 55,019 patients **Statistical Analyses:** Conditional logistic regression, Pearson's pairwise correlation **Covariates:** Temperature, barometric pressure, dew point **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (5th–95th percentile):** 31.06 (8.89-70.49) SD = 20.10 **Monitoring Stations:** 17 **Copollutant (correlation):** CO: r = 0.57 NO_2: r = 0.64 O_3: r = 0.29 SO_2: r = 0.51	**PM Increment:** 24 µg/m³ (IQR) **Percent Increase [CI]:** Single-pollutant: 3.07 [1.59,4.57] Adj. for CO: -1.10 [-3.02,0.86] Adj. for NO_2: 0.52 [-1.46,2.53] Adj. for O_3: 2.80 [1.29,4.33] Adj. for SO_2: 2.18 [0.37,4.02] **Percent Increase (with 10 µg/m³ increment)** 1.27 [0.66,1.88]
Reference: Wellenius et al. (2005a) **Period of Study:** 1 Jan, 1986–30 Nov, 1999 **Location:** Birmingham, Chicago, Cleveland, Detroit, Minneapolis, New Haven, Pittsburgh, Salt Lake City, Seattle	**Outcome:** Ischemic stroke and hemorrhagic stroke **Age Groups:** 65+ yrs **Study Design:** Case-crossover (time-stratified) **N:** 115,503 hospital admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS (v.9) and R-statistical package **Lags Considered:** 0-2 lags	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 32.69 (19.75) **Monitoring Stations:** NR (data obtained from the US EPA) **Copollutant (correlation):** CO: r = 0.43 NO_2: r = 0.53 SO_2: r = 0.39 **Other variables:** Temp: r = 0.22	**PM Increment:** 22.96 µg/m³ (IQR) **Percent Increase [CI]:** Ischemic (same-day lag): 1.03 [0.04,2.04] Hemorrhagic: -0.58 [-5.48,4.58] **Notes:** Percent increase in rate for ischemic and hemorrhagic stroke are provided for each city in graphical form (Fig A and B).

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Wellenius et al. (2005b) **Period of Study:** 1 Jan, 1986–30 Nov, 1999 **Location:** Birmingham, Chicago, Cleveland, Detroit, Minneapolis, New Haven, Pittsburgh, Salt Lake City, Seattle	**Outcome (ICD-9):** Congestive heart failure (428) **Age Groups:** 65+ yrs **Study Design:** Case-crossover (time-stratified) **N:** 292,918 admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and barometric pressure **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS (v.9) and R-statistical package **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median:** Overall: 28.3 Birmingham: 33.0 Chicago: 31.5 Cleveland: 34.5 Detroit: 29.5 Minneapolis: 24.0 New Haven: 22. Seattle: 25.8 **Monitoring Stations:** NR (data obtained from the US EPA) **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **Percent Increase [CI]:** Same-day lag: 0.72 [0.35,1.10] p-value = 0.0002 **Notes:** City-specific percent increases are graphed in Fig 1 for same-day lag showing a significant association in Chicago, Detroit, Seattle, and the summary values. Percent increase in admission rate s are provided for lag 0-3 days in Fig 2 where same-day lag showed a significant association.
Reference: Yang et al. (2004b) **Period of Study:** 1997-2000 **Location:** Kaohsiung, Taiwan	**Outcome (ICD-9):** Cardiovascular diseases (410-429) **Age Groups:** All **Study Design:** Case-crossover **N:** 29,661 admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** Cumulative 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median (min-max):** 78.82 (20.50-217.33) **Monitoring Stations:** 6 **Copollutant:** NR	**PM Increment:** 66.33 µg/m^3 (IQR) **OR Estimate [CI]:** Temp >25ºC: 1.439 [1.316,1.573] Temp <25ºC: 1.568 [1.433,1.715] Adj for SO_2 Temp >25ºC: 1.460 [1.333,1.599] Temp <25ºC: 1.543 [1.404,1.696] Adj for NO_2 Temp >25ºC: 1.306 [1.154,1.478] Temp <25ºC: 0.912 [0.809,1.028] Adj for CO Temp >25ºC: 1.260 [1.144,1.388] Temp <25ºC: 1.259 [1.128,1.406] Adj for O_3 Temp >25ºC: 1.086 [0.967,1.220] Temp <25ºC: 1.703 [1.541,1.883]
Reference: Yang et al (2008) **Period of Study:** 1996 - 2004 **Location:** Taipei, Taiwan	**Outcome (ICD-9):** Congestive heart failure (428) **Age Groups:** All **Study Design:** Case-crossover **N:** 24,240 CHF hospital admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** temperature, humidity **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** Cumulative lag 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (median, range, IQR):** 49.47 (44.71, 14.42–234.91, 33.08–44.71) **Monitoring Stations:** 6 **Copollutant:** NR	**PM Increment:** 27.02 µg/m^3 (IQR) **OR [95% CI]:** Single pollutant models: >20 °C: 1.15 [1.10–1.21] <20 °C: 0.99 [0.93–1.05] Adjusted for SO_2: ≥ 20 °C: 1.23 [1.17–1.30] <20 °C: 0.96 [0.89–1.03] Adjusted for NO_2: ≥ 20 °C: 1.03 [0.97–1.10] <20 °C: 0.97 [0.90–1.04] Adjusted for CO: ≥ 20 °C: 1.09 [1.03–1.15] <20 °C: 0.96 [0.90–1.03] Adjusted for O_3: ≥ 20 °C: 1.10 [1.04–1.15] <20 °C: 1.00 [0.94–1.05]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Zanobetti and Schwartz (2002) **Period of Study:** 1988-1994 **Location:** Cook county (Chicago), Illinois; Wayne county (Detroit), Michigan; Allegheny county (Pittsburgh), Pennsylvania; and King county (Seattle), Washington	**Outcome (ICD-9):** Cardiovascular disease (390-429) with/without diabetes (250) **Age Groups:** 65-74 and 75+ yrs with diabetes, 65-74 and 75+ yrs without diabetes **Study Design:** Time series **N:** NR **Statistical Analyses:** GAM, meta-regression **Covariates:** Temperature, prior day's temperature, relative humidity, barometric pressure, day of the week **Season:** NR **Dose-response Investigated:** No	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median (25-75th percentile):** Chicago: 33 (23-46) Detroit: 32 (21-49) Pittsburgh: 30 (19-47) Seattle: 27 (18-39) **Monitoring Stations:** NR (obtained from USEPA Aerometric Information Retrieval System) **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **Percent Change [CI]:** All four cities <75 (w/ diabetes): 1.6 [1.2,2.0] 75+ (w/ diabetes): 2.0 [1.6,2.4] <75 (w/o diabetes): 0.9 [0.6,1.1] 75+ (w/o diabetes): 1.3 [1.0,1.5] Chicago <75 (w/ diabetes): 1.9 [1.1,2.7] 75+ (w/ diabetes): 2.0 [1.1,3.0] <75 (w/o diabetes): 0.7 [0.2,1.2] 75+ (w/o diabetes): 1.2 [0.8,1.7] Detroit <75 (w/ diabetes): 1.3 [0.5,2.2] 75+ (w/ diabetes): 2.1 [1.0,3.1] <75 (w/o diabetes): 1.2 [0.7,1.7] 75+ (w/o diabetes): 1.2 [0.7,1.6] Pittsburgh <75 (w/ diabetes): 1.8 [0.9,2.7] 75+ (w/ diabetes): 0.9 [-0.2,2.0] <75 (w/o diabetes): 0.6 [0.1,1.2] 75+ (w/o diabetes): 1.6 [1.2,2.1] Seattle <75 (w/ diabetes): 1.9 [0.1,3.7] 75+ (w/ diabetes): 2.7 [0.7,4.8] <75 (w/o diabetes): 0.8 [0.0,1.6] 75+ (w/o diabetes): 0.9 [0.2,1.6] **Notes:** Overall percent increases were also provided for each city, yielding similar results.
Reference: Zanobetti and Schwartz (2005) **Period of Study:** 1985-1999 **Location:** 21 U.S. cities (Birmingham, Alabama; Boulder, Colorado; Canton, Ohio; Chicago, Illinois; Cincinnati, Ohio; Cleveland, Ohio; Colorado Springs, Colorado; Detroit, Michigan; Honolulu, Hawaii; Houston, Texas; Minneapolis-St.Paul, Minnesota; Nashville, Tennessee; New Haven, Connecticut; Pittsburgh, Pennsylvania; Provo-Orem, Utah; Salt Lake City, Utah; Seattle, Washington; Steubenville, Ohio; Youngstown, Ohio)	**Outcome (ICD-9):** Myocardial infarction (410) **Age Groups:** >65 yrs **Study Design:** Case-crossover **N:** 302,453 admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature **Season:** NR **Dose-response Investigated:** Yes **Statistical Package:** SAS (PROC PHREG) **Lags Considered:** 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median:** Ranged from 15.5-34.1Avg across all cities = 27 **Monitoring Stations:** 1+ (data obtained from USEPA's Aerometric Information Retrieval System) **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **Percent Increase [CI]:** MI only: 0.65 [0.3,1] Previous COPD admission: 1.3 [-0.1,2.8] Secondary pneumonia diagnosis: 1.4 [-0.8,3.6] **Notes:** Figure 1 presents percent change in MI per lag day, showing same-day lag to be significant. Figure 2 shows percent change with/without other co-morbidities.

Table E-6. Short-term exposure to $PM_{10-2.5}$ and emergency department visits and hospital admissions for cardiovascular outcomes.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Host et al. (2008) **Period of Study:** 2000 - 2003 **Location:** Six French cities: Le Havre, Lille, Marseille, Paris, Rouen, and Toulouse	**Outcome (ICD-10):** Daily hospitalizations for all cardiovascular (I00–I99), cardiac (I00–I52), and ischemic heart diseases (I20–I25). **Age Groups:** For cardiovascular diseases: All ages, and restricted to ≥ 65 years **Study Design:** Time series **N:** NR (Total population of cities: approximately 10 million) **Statistical Analyses:** Poisson regression **Covariates:** Seasons, days of the week, holidays, influenza epidemics, pollen counts, temperature, and temporal trends **Dose-response Investigated:** No **Statistical Package:** MGCV package in R software (R 2.1.1) **Lags Considered:** Avg of 0-1 days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean** µg/m³ **(5th -95th percentile):** Le Havre: 7.3 (2.5–14.0) Lille: 7.9 (2.2–13.7) Marseille: 11.0 (4.5–21.0) Paris: 8.3 (3.2–15.9) Rouen: 7.0 (3.0–12.5) Toulouse: 7.7 (3.0–15.0) **Monitoring Stations:** 13 total: 1 in Toulouse 4 in Paris 2 each in other cities **Copollutant (correlation):** $PM_{2.5}$: Overall: r>0.6 Ranged between r = 0.28 and r = 0.73 across the six cities.	**PM Increment:** 10 µg/m³ , and an 18.8 µg/m³ increase (correspondding to an increase in pollutant levels between the lowest of the 5th percentiles and the highest of the 95th percentiles of the cities' distributions) **ERR (excess relative risk) Estimate [CI]:** For all cardiovascular diseases (10 µg/m³ increase): All ages: 0.5% [-1.2, 2.3]; ≥ 65 years: 1.0% [-1.0, 3.0] For all cardiovascular diseases (18 µg/m³ increase): All ages: 1.0% [-2.3, 4.3]; ≥ 65 years: 1.9% [-2.0, 5.9] For cardiac diseases (10 µg/m³ increase): All ages: 0.1% [-1.9, 2.1]; ≥ 65 years: 1.6% [-0.8, 4.1] For cardiac diseases (18.8 µg/m³ increase): All ages: 0.1% [-3.6, 4.0]; ≥ 65 years: 3.1% [-1.5, 7.9] For ischemic heart diseases (10 µg/m³ increase): All ages: 2.8% [-0.8, 6.6]; ≥ 65 years: 6.4% [1.6, 11.4] For ischemic heart diseases (18 µg/m³ increase): All ages: 5.4% [-1.5, 12.8]; ≥ 65 years: 12.4 [3.1, 22.6]
Reference: Metzger et al, (2004) **Period of Study:** August 1998–August 2000 **Location:** Atlanta Metropolitan area (Georgia)	**Outcome (ICD-9):** Emergency visits for ischemic heart disease (410–414), cardiac dysrhythmias (427), cardiac arrest (427.5), congestive heart failure (428), peripheral vascular and cerebrovascular disease (433-437, 440, 443-444, 451–453), atherosclerosis (440), and stroke (436). **Age Groups:** All **Study Design:** Time series **N:** 4,407,535 emergency department visits between 1993–2000 (data not reported for 1998 - 2000) **Statistical Analyses:** Poisson generalized linear modeling **Covariates:** Day of the week, hospital entry and exit indicator variables, federally observed holidays, temporal trends, temperature, dew point temperature **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 3-day moving avg, lags 0 -7	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Median** µg/m³ **(10% - 90% range):** 9.1 (4.4, 16.2) **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: r = 0.59; O_3: r = 0.35; NO_2: r = 0.46; CO: r = 0.32; SO_2: r = 0.21; $PM_{2.5}$: r = 0.43; UFP: r = 0.13; $PM_{2.5}$ water; soluble metals: r = 0.47; $PM_{2.5}$ sulfates: r = 0.26; $PM_{2.5}$ acidity: r = 0.23; $PM_{2.5}$ OC: r = 0.51; $PM_{2.5}$ EC: r = 0.48; $PM_{2.5}$ oxygenated hydrocarbon: r = 0.31 **Other variables:** Temperature: r = 0.20 Dew point: r = 0.00	**PM Increment:** 5 µg/m³ (approximately 1 SD) **RR [95% CI]:** For 3 day moving avg: All CVD: 1.012 [0.985, 1.040] Dysrhythmia: 1.021 [0.974, 1.070] Congestive heart failure: 1.020 [0.964–1.079] Ischemic heart disease: 0.994 [0.946–1.045] Peripheral vascular and cerebrovascular disease: 1.022 [0.972–1.074]] Results for Lags 0–7 expressed in figures (see notes). **Notes:** Figure 1: RR (95% CI) for single-day lag models for the association of ER visits for CVD with daily ambient $PM_{10-2.5}$. Summary of Figure 1 results: Positive association at Lag 0.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Peng et al. (2008) **Period of Study:** January 1, 1999–December 31, 2005 **Location:** 108 U.S. counties in the following states: Alabama, Arizona, California, Colorado, Connecticut, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin	**Outcome (ICD-9):** Emergency hospitalizations for: Cardiovascular disease, including heart failure (428), heart rhythm disturbances (426–427), cerebrovascular events (430–438), ischemic heart disease (410–414, 429), and peripheral vascular disease (440–448). **Age Groups:** 65 + years, 65–74, 75+ **Study Design:** Time series **N:** approximately 12 million Medicare enrollees (3.7 million CVD and 1.4 million RD admissions) **Statistical Analyses:** Two-stage Bayesian hierarchical models: Overdispersed Poisson models for county-specific data. Bayesian hierarchical models to obtain national avg estimate **Covariates:** Day of the week, age-specific intercept, temperature, dew point temperature, calendar time, indicator for age of 75 years or older. Some models were adjusted for $PM_{2.5}$. **Dose-response Investigated:** No **Statistical Package:** R version 2.6.2 **Lags Considered:** 0-2 days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean μg/m³ (IQR):** All counties assessed: 9.8 (6.9–15.0) Counties in Eastern US: 9.1 (6.6–13.1) Counties in Western US: 15.4 (10.3–21.8) **Monitoring Stations:** At least 1 pair of co-located monitors (physically located in the same place) for PM_{10} and $PM_{2.5}$ per county **Copollutant (correlation):** $PM_{2.5}$: r = 0.12 PM_{10}: r = 0.75 **Other variables:** Median within-county correlations between monitors: r = 0.60	**PM Increment:** 10 μg/m³ **Percentage change [95% CI]:** CVD: Lag 0 (unadjusted for $PM_{2.5}$): 0.36 [0.05, 0.68] Lag 0 (adjusted for $PM_{2.5}$): 0.25 [-0.11, 0.60] **Notes:** Effect estimates for $PM_{10-2.5}$ (0–2 day lags) are showing in Figures 2–5. Figure 2: Percentage change in emergency hospital admissions for CVD per 10 μg/m³ increase in PM (single pollutant model and model adjusted for $PM_{2.5}$ concentration) Figure 4: Percentage change in emergency hosptal admissions rate for CVD and RD per a 10 μg/m³ increase in $PM_{10-2.5}$ (0–2 day lags, Eastern vs. Western USA) Figure 5: County-specific log relative risks of emergency hospital admissions for CVD per 10 μg/m³ increase in $PM_{10-2.5}$ at Lag 0 (unadjusted for $PM_{2.5}$ and plotted vs percentage of urbanicity) No significant associations between $PM_{10-2.5}$ and cause-specific cardiovascular disease.
Reference: Tolbert et al. (2007) **Period of Study:** August 1998–December 2004 **Location:** Atlanta Metropolitan area, Georgia	**Outcome (ICD-9):** Combined CVD group, including: Ischemic heart disease (410–414), cardiac dysrhythmias (427), congestive heart failure (428), and peripheral vascular and cardiovascular disease (433–437, 440, 443–445, and 451–453) **Age Groups:** All **Study Design:** Time series **N:** NR for 1998–2004. For 1993–2004: 10,234,490 ER visits (283,360 visits). **Statistical Analyses:** Poisson generalized linear models **Covariates:** Long-term temporal trends, temperature, dew point, days of week, federal holidays, hospital entry and exit **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 9.1 **Lags Considered:** 3-day moving avg (lag 0-2)	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (median; IQR, range, 10th–90th percentiles):** 9.0 (8.2; 5.6–11.5; 0.5–50.3; 3.6–15.1) **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: r = 0.67 O_3: r = 0.36 NO_2: r = 0.48 CO: r = 0.38SO2: r = 0.16 $PM_{2.5}$: r = 0.47 $PM_{2.5}$ SO4: r = 0.32 $PM_{2.5}$ EC: r = 0.49 $PM_{2.5}$ OC: r = 0.49 $PM_{2.5}$ TC: r = 0.51 $PM_{2.5}$ water-sol metals: r = 0.50 OHC: r = 0.41	**PM Increment:** 5.89 μg/m³ (IQR) **Risk ratio [95% CI]:** CVD: 1.004 (0.990–1.019)

Table E-7. Short-term exposure to $PM_{2.5}$ (including PM components/sources) and emergency department visits and hospital admissions for cardiovascular outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Andersen et al. (2008b) **Period of Study:** May 2001 - December 2004 **Location:** Copenhagen, Denmark	**Outcome (ICD-10):** CVD, including angina pectoris (I20), myocardial infarction (I21–22), other actue ischemic heart diseases (I24), chronic ischaemic heart disease (I25), pulmonary embolism (I26), cardiac arrest (I46), cardiac arrhythmias (I48–48), and heart failure (I50). RD, including chronic bronchitis (J41–42), emphysema (J43), other chronic obstructive pulmonary disease (J44), asthma (J45), and status asthmaticus (J46). Pediatric hospital admissions for asthma (J45) and status asthmaticus (J46). **Age Groups: >** 65 yrs (CVD and RD), 5–18 years (asthma) **Study Design:** Time series **N (Specify units):** NR **Statistical Analyses:** Poisson GAM **Covariates:** Temperature, dew-point temperature, long-term trend, seasonality, influenza, day of the week, public holidays, school holidays (only for 5–18 year olds), pollen (only for pediatric asthma outcome) **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R statistical software (gam procedure, mgcv package) **Lags Considered:** Lag 0-5 days, 4-day pollutant avg (lag 0-3) for CVD, 5-day avg (lag 0-4) for RD, and a 6-day avg (lag 0-5) for asthma.	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean µg/m³ (SD; median; IQR; 99th percentile):** 10 (5; 9; 7–12; 28) **Monitoring Stations:** 1 **Copollutant (correlation):** NCtot: r = 0.40 NC100: r = 0.29 NCa12: r = 0.07 Nca23: r = -0.25 NCa57: r = 0.51 NC_{a212}: r = 0.82 PM_{10}: r = 0.80 CO: r = 0.46 NO_2: r = 0.42 Nox: r = 0.40 No_x curbside: r = 0.28 O_3: r = -0.20 **Other variables:** Temperature: r = -0.01 Relative humidity:r = 0.21	**PM Increment:** 5 µg/m³ (IQR) **Relative risk (RR) Estimate [CI]:** CVD hospital admissions (4 day avg, lag 0 -3), age 65+: One-pollutant model: 1.03 [1.01–1.06] Adj for NCtot: 1.03 [1.01–1.06] RD hospital admissions (5 day avg, lag 0 -4), age 65+: One-pollutant model: 1.00 [0.95–1.00] Adj for NCtot: 1.00 [0.95–1.06] Asthma hospital admissions (6 day avg lag 0–5), age 5 - 18: One-pollutant model: 1.15 [1.00–1.32] Adj for NCtot: 1.13 [0.98–1.32] Estimates for individual day lags reported only in figure form (see notes): **Notes:** Figure 2: Relative risks and 95% confidence intervals per IQR in single day concentration (0–5 day lag). Summary: CVD: Marginally significant association at Lag 0. RD: No statistically or marginally significant associations. Positive associatons at Lag 4–5.Asthma: Wide confidence intervals make interpretation dificult. Positive associations at Lag 1, 2, 3.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Bell et al. (2008b) **Period of Study:** 1995 - 2002 **Location:** Taipei, Taiwan	**Outcome (ICD-9):** Hospital admissions for ischemic heart disease (410, 411, 414), cerebrovascular disease (430–437). **Age Groups:** All **Study Design:** Time series **N (Specify units):** 6,909 hospital admissions for ischaemic heart diseases, 11,466 for cerebrovascular disease. **Statistical Analyses:** Poisson regression **Covariates:** Day of the week, time, apparent temperature, long-term trends, seasonality **Season:** All **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** lags 0-3 days, mean of lags 0-3	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean µg/m³ (range; IQR):** 31.6 (0.50–355.0; 20.2) **Monitoring Stations:** 2 **Copollutant (correlation):** NR	**PM Increment:** 20 µg/m³ (near IQR) **Percentage increase estimate [95% CI]:** Ischemic heart disease: L0: 3.48 (-0.39, 7.51) L1: 3.55 (-0.30, 7.56); L2: 3.32 (-0.50, 7.29) L3: 2.80 (-1.04, 6.79); L03: 8.38 (2.28, 14.84) Cerebrovascular disease: L0: -2.22 (-50.2, 0.67) L1: -1.30 (-4.08, 1.55); L2: 0.24 (-2.49, 3.040 L3: 1.21 (-1.41, 3.90); L03: -1.45 (-5.58, 2.87)
References: Bell et al. (2008a) **Period of Study:** 1999 - 2005 **Location:** 202 US counties	**Outcome (ICD-9):** Heart failure (428), heart rhythm disturbances (426–427), cerebrovascular events (430–438), ischemic heart disease (410–414, 429), peripheral vascular disease (440–449). **Age Groups:** 65+ **Study Design:** Time series **N (Specify units):** NR **Statistical Analyses:** Two-stage Bayesian hierarchical model to find national avg First stage: Poisson regression (county-specific) **Covariates:** day of the week, temperature, dew point temperature, temporal trends, indicator for persons 75+ years, population size **Season:** All, June–August (Summer), September–November (Fall), December–February (Winter), March–May (Spring) **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0–2 day lags	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean** (µg/m³): Descriptive information presented in Figure S2 (boxplots): **IQR:** 8.7 µg/m³ **Monitoring Stations:** NR **Copollutant (correlation):** NR	**PM Increment:** 10 µg/m³ **Percent increase [95% PI]: Cardiovascular admissions:** Lag 0 (all seasons): 0.80 [0.59–1.01] Lag 0 (winter, national): 1.49 [1.09–1.89] Lag 0 (winter, northeast): 2.01 [1.39–2.63] Lag 0 (winter, southeast): 1.06 [-0.07–2.21] Lag 0 (winter, northwest): 0.85 [-4.11–6.07] Lag 0 (winter, southwest): 0.76 [-0.25–1.79] Lag 0 (spring, national): 0.91 [0.47–1.35] Lag 0 (spring, northeast): 0.95 [0.32–1.58] Lag 0 (spring, southeast): 0.75 [-0.26–1.78] Lag 0 (spring, northwest): -0.07 [-12.40–13.98] Lag 0 (spring, southwest): 1.78 [-0.87–4.51] Lag 0 (summer, national): 0.18 [-0.23–0.58] Lag 0 (summer, northeast): 0.55 [0.08–1.02] Lag 0 (summer, southeast): -0.67 [-1.60–0.26] Lag 0 (summer, northwest): -1.55 [-15.22–14.31] Lag 0 (summer, southwest): -1.20 [-4.90–2.65] Lag 0 (autunmn, national): 0.68 [0.29–1.07] Lag 0 (autumn, northeast): 1.03 [0.48–1.58] Lag 0 (autumn, southeast): 0.17 [-0.72–1.07] Lag 0 (autumn, northwest): -0.67 [-6.96–6.05] Lag 0 (autumn, southwest): 0.30 [-0.98–1.59] Lag 1 (all seasons): 0.07 [-0.12–0.26]; Lag 1 (winter): 0.56 [0.16–0.96] Lag 1 (spring): -0.10 [-0.58–0.39]; Lag 1 (summer): -0.16 [-0.54–0.22] Lag 1 (autumn): 0.04 [-0.28–0.35] Lag2 (all seasons): [0.06 [-0.12–0.23] Lag 2 (winter): 0.27 [-0.12–0.65]; Lag 2 (spring): 0.19 [-0.23–0.60] Lag 2 (summer): -0.12 [-0.50–0.26]; Lag 2 (autumn): 0.02 [-0.30–0.34]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chan et al. (2008) **Period of Study:** 1995 - 2002 **Location:** Taipei Metropolitan area, Taiwan	**Outcome (ICD-9):** Emergency visits for ischaemic heart diseases (410–411, 414), cerebrovascular diseases (430-437), and COPD (493, 496) **Age Groups:** All **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson regression **Covariates:** Year, month, day of week, temperature, dewpoint temperature, PM_{10}, NO_2 **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 8.0 **Lags Considered:** 0- to 7-day lags	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean** μg/m³ **(SD):** NR **Monitoring Stations:** 1 **Copollutant (correlation):** NR	**PM Increment:** 19.7 μg/m³ (IQR) **OR [95% CI]:** In environmental conditions without dust storms (results only given for best-fitting model) Lag 6 days: 1.024 (1.004, 1.044)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Dominici et al. (2006) **Period of Study:** 1999 - 2002 **Location:** 204 US counties, located in: Alabama, Alaska, Arizona, Arkansas, California, Colorado, Connecticut, Delaware, District of Columbia, Florida, Georgia, Hawaii, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin	**Outcome (ICD-9:** Daily counts of hospital admissions for primary diagnosis of heart failure (428), heart rhythm disturbances (426–427), cerebrovascular events (430–438), ischemic heart disease (410–414, 429), peripheral vascular disease (440–448), chronic obstructive pulmonary disease (490–492), and respiratory tract infections (464–466, 480–487). **Age Groups:** >65 years **Study Design:** Time series **N:** 11.5 million Medicare enrollees **Statistical Analyses:** Bayesian 2-stage hierarchical models. First stage: Poisson regression (county-specific) Second stage: Bayesian hierarchical models, to produce a national avg estimate **Covariates:** Day of the week, seasonality, temperature, dew point temperature, long-term trends **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R statistical software version 2.2.0 **Lags Considered:** 0-2 days, avg of days 0-2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (μg/m³) (IQR):** 13.4 (11.3–15.2) **Monitoring Stations:** NR **Copollutant (correlation):** NR **Other variables:** Median of pairwise correlations among $PM_{2.5}$ monitors within the same county for 2000: r = 0.91 (IQR: 0.81-0.95)	**PM Increment:** 10 μg/m³ (Results in figures; see notes) **Percent increase in risk [95% PI]: Cerebrovascular disease (Lag 0):** Age 65+: 0.81 [0.30, 1.32]; Age 65-74: 0.91 [0.01, 1.82] Age 75+: 0.80 [0.21, 1.38] **Peripheral vascular disease (Lag 0):** Age 65+: 0.86 [-0.06, 1.79]; Age 65-74: 1.21 [-0.26, 2.67] Age 75+: 0.86 [-0.39, 2.11] **Ischemic heart disease (Lag 2):** Age 65+: 0.44 [0.02, 0.86]; Age 65-74: 0.37 [-0.22, 0.96] Age 75+: 0.52 [-0.01, 1.04] **Heart rhythm disturbances (Lag 0):** Age 65+: 0.57 [-0.01, 1.15]; Age 65-74: 0.46 [-0.63, 1.54] Age 75+: 0.72 [0.02, 1.42] **Heart failure (Lag 0):** Age 65+: 1.28 [0.78, 1.78] Age 65-74: 1.21 [0.35, 2.07] ; Age 75+: 1.36 [0.78, 1.94] **COPD (Lag 0):** Age 65 +: 0.91 [0.91, 1.64] Age 65–74: 0.42 [-0.64, 1.48]; Age 75+: 1.47 [0.54, 2.40] **Respiratory tract infection:** Age 65+: 0.92 [0.41, 1.43] Age 65–74: 0.93 [0.04, 1.82]; Age 75+: 0.92 [0.32, 1.53] **Annual reduction in admissions attributable to a 10 μg/m³ reduction in daily PM2.5 level (95% PI): Cerebrovascular disease:** Annual number of admissions: 226,641 Annual reduction in admissions: 1836 [680, 2992] **Peripheral vascular disease:** Annual number of admissions: 70,061 Annual reduction in admissions: 602 [-42, 1254] **Ischemic heart disease:** Annual number of admissions: 346,082 Annual reduction in admissions: 1523 [69, 2976] **Heart rhythm disturbances:** Annual number of admissions: 169,627 Annual reduction in admissions: 967 [-17, 1951] **Heart failure:** Annual number of admissions: 246,598 Annual reduction in admissions: 3156 [1923, 4389] **COPD:** Annual number of admissions: 108,812 Annual reduction in admissions: 990 [196, 1785] **Respiratory tract infections:** Annual number of admissions: 226,620 Annual reduction in admissions: 2085 [929, 3241] **Notes**: Figure 2: Point estimates and 95% posterior intervals of the % change in admissions rates per 10 μg/m³ (national avg relative rates) for single lag (0, 1, and 2 days) and distributed lag models for 0 to 2 days (total) for all outcomes. Summary: Positive significant or marginally significant associations between $PM_{2.5}$ and cerebrovascular disease at Lag 0; peripheral vascular disease at Lags 0 and 2; ischemic heart disease at Lag 2; heart rhythm disturbances at Lag 0; heart failure at Lag 0, Lag 2, and Lags 0 -2; COPD at Lag 0, Lag 1, and Lags 0–2; and respiratory tract infections at Lag 2 and Lags 0–2. Figure 3: Point estimates and 95% posterior intervals of the % change in admission rates per 10 μg/m³ (regional relative rates). Summary: For cardiovascular diseases, all estimates in the Midwestern, Northeastern, and Southern regions were positive, while estimates in the other regions (South, West, Central, Northwest) were close to 0. For respiratory disease, there were larger effects in the Central, Southeastern, Southern, and Western regions than in the other regions. Figure 4: Point estimates and 95% posterior intervals of the % change in admission per 10 μg/m³ (Eastern vs. Western regions): Summary: All estimates for cardiovascular outcomes were positive in the US Eastern region but not in the US Western region. The estimates for respiratory tract infections were larger in the Western region than in the Eastern region. The estimates for CCPD were positive in the both regions.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Host et al. (2008) **Period of Study:** 2000 - 2003 **Location:** Six French cities: Le Havre, Lille, Marseille, Paris, Rouen, and Toulouse	**Outcome (ICD-10):** Daily hospitalizations for all cardiovascular (I00–I99), cardiac (I00–I52), and ischemic heart diseases (I20–I25), all respiratory diseases (J00–J99), respiratory infections (J10–J22). **Age Groups:** For cardiovascular diseases:All ages, and restricted to ≥ 65 years. For all respiratory diseases: 0–14 years, 15–64 years, and ≥ 65 years. For respiratory infections: All ages **Study Design:** Time series **N:** NR (Total population of cities: approximately 10 million) **Statistical Analyses:** Poisson regression **Covariates:** Seasons, days of the week, holidays, influenza epidemics, pollen counts, temperature, and temporal trends **Season:** NR **Dose-response Investigated:** No **Statistical Package:** MGCV package in R software (R 2.1.1) **Lags Considered:** Avg of 0-1 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean** (5th -95th percentile): Le Havre: 13.8 (6.0–30.5) Lille: 15.9 (6.9–26.3) Marseille: 18.8 (8.0–33.0) Paris: 14.7 (6.5–28.8) Rouen: 14.4 (7.5–28.0) Toulouse: 13.8 (6.0–25.0) **Monitoring Stations:** 13 total: 1 in Toulouse 4 in Paris 2 each in other cities **Copollutant (correlation):** $PM_{10-2.5}$: Overall: r > 0.6 Ranged between r = 0.28 and r = 0.73 across the six cities.	**PM Increment:** 10 µg/m³ increase, and a 27 µg/m³ increase (corresponding to the difference between the lowest of the 5th percentiles and the highest of the 95th percentiles of the cities' distributions) **ERR (excess relative risk) Estimate [CI]:** For all cardiovascular diseases (10 µg/m³ increase): All ages: 0.9% [0.1, 1.8]; ≥ 65 years: 1.9% [0.9, 3.0] For all cardiovascular diseases (27 µg/m³ increase): All ages: 2.5% [0.2, 4.9]; ≥ 65 years: 5.3% [2.6, 8.2] For ischemic heart diseases (27 µg/m³ increase): All ages: 5.2% [-0.6, 11.3]; ≥ 65 years: 12.7% [6.3, 19.5] For cardiac diseases (10 µg/m³ increase): All ages: 0.9% [-0.1, 2.0]; ≥ 65 years: 2.4% [1.2, 3.7] For cardiac diseases (27 µg/m³ increase): All ages: 2.5% [-0.3, 5.4]; ≥ 65 years: 6.8% [3.3, 10.3] For ischemic heart diseases (10 µg/m³ increase): All ages: 1.9 % [-0.2, 4.0]; ≥ 65 years: 4.5% [2.3, 6.8] For all respiratory diseases (10 µg/m³ increase): 0–14 years: 0.4% [-1.2, 2.0] ; 15–64 years: 0.8% [-0.7, 2.3] ; ≥ 65 years: 0.5% [-2.0, 3.0] For all respiratory diseases (27 µg/m³ increase): 0–14 years: 1.1% [-3.1, 5.5]; 15–64 years: 2.2% [-1.8, 6.4] ; ≥ 65 years: 1.3% [-5.3, 8.2] For respiratory infections (10 µg/m³ increase): All ages: 2.5% [0.1, 4.8] For respiratory infections (27 µg/m³ increase): All ages: 7.0% [0.7, 13.6]
Reference: Jalaludin et al. (2006) **Period of Study:** 1 Jan, 1997–31 Dec, 2001 **Location:** Sydney, Australia	**Outcome (ICD-9):** Cardiovascular disease (390-459), cardiac disease (390-429), ischemic heart disease (410-413) and cerebrovascular disease or stroke (430-438) **Age Groups:** 65+ yrs **Study Design:** Time series **N:** NR **Statistical Analyses:** GAM, GLM **Covariates:** Temperature, humidity **Season:** Warm (Nov-Apr) and cool (May-Oct) **Dose-response Investigated:** No **Statistical Package:** S-Plus **Lags Considered:** 0-3 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 9.5 (2.4-82.1) SD = 5.1 **Monitoring Stations:** 14 **Copollutant (correlation):** Warm BSP: r = 0.93 PM_{10}: r = 0.89 O_3: r = 0.57 NO_2: r = 0.45 CO: r = 0.35 SO_2: r = 0.27 Cool BSP: r = 0.90 PM_{10}: r = 0.88 O_3: r = 0.05 NO_2: r = 0.68 CO: r = 0.60 SO_2: r = 0.46 **Other variables:** Warm Temp: r = 0.24 Rel humidity: r = -0.15 Cool Temp: r = -0.04 Rel humidity: r = 0.20	**PM Increment:** 4.8 µg/m³ (IQR) **Percent Change Estimate [CI]:** All CVD Same-day lag: 1.26 [0.56,1.96] Avg 0-1 day lag: 0.85 [0.18,1.52] Cool (same-day lag): 2.23 [0.98,3.50] Warm (same-day lag): 0.73 [-0.05,1.52] Cardiac disease Same-day lag: 1.55 [0.74,2.38] Avg 0-1 day lag: 1.33 [0.54,2.13] Cool (same-day lag): 2.37 [0.87,3.89] Warm (same-day lag): 1.13 [0.22,2.04] Ischemic heart disease Same-day lag: 1.17 [-0.08,2.44] Avg 0-1 day lag: 1.24 [0.04,2.45] Cool (same-day lag): 0.57 [-1.74,2.94] Warm (same-day lag): 1.31 [-0.04,2.68] Stroke Same-day lag: -0.89 [-2.41,0.65] Avg 0-1 day lag: -1.08 [-2.54,0.41] Cool (same-day lag): 1.45 [-1.17,4.15] Warm (same-day lag): -2.19 [-4.00,-0.36] **Notes:** All other lag-day ORs were provided, yet none were significant. Percent change in ED attendance was also reported graphically (Fig 1-5).

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lisabeth et al. (2008) **Period of Study:** 2001 - 2005 **Location:** Nueces County, Texas	**Outcome:** Ischemic stroke and transient ischemic attacks (ICD codes not reported). **Age Groups:** 45+ years **Study Design:** Time series **N:** 3,508 stroke/TIAs (2,350 strokes, and 1,158 TIAs) **Statistical Analyses:** Poisson regression **Covariates:** Temperature, day of week, temporal trends **Season:** All, but looked at potential effect modification by season (Summer: June–September; Non-summer: October-May) **Dose-response Investigated:** No **Statistical Package:** S-plus 7.0 **Lags Considered:** Lags 0–5 days, and averaged lag effect (0–5 days)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Median** μg/m^3 **(IQR):** 7.0 (4.8–10.0) **Monitoring Stations:** 6 **Copollutant (correlation):** NR	**PM Increment:** 5.1 μg/m^3 (IQR) **RR Estimate [CI]:** Lag 0: 1.03 (0.99, 1.07) Lag 1: 1.03 (1.00–1.07) All other lags and avg (lag 0–5) were not statistically or marginally significant. Adjusted for O_3: Lag 0: 1.03 (0.99, 1.07) Lag 1: 1.03 (0.99–1.06) All other lags and avg (lag 0–5) were not statistically or marginally significant. **Notes:** Figure 3: % change in stroke/TIA risk associated with an IQR increase in $PM_{2.5}$

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Metzger et al. (2004) **Period of Study:** August 1998–August 2000 **Location:** Atlanta Metropolitan area (Georgia)	**Outcome (ICD-9):** Emergency visits for ischemic heart disease (410–414), cardiac dysrhythmias (427), cardiac arrest (427.5), congestive heart failure (428), peripheral vascular and cerebrovascular disease (433-437, 440, 443-444, 451–453), atherosclerosis (440), and stroke (436). **Age Groups:** All **Study Design:** Time series **N:** 4,407,535 emergency department visits for 1993–2000 (data not reported for 1998-2000) **Statistical Analyses:**Poisson generalized linear modeling **Covariates:** Day of the week, hospital entry and exit indicator variables, federally observed holidays, temporal trends, temperature, dew point temperature **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 3-day moving avg, lags 0 -7	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Median µg/m³ (10%-90% range):** $PM_{2.5}$: 17.8 (8.9, 32.3) $PM_{2.5}$ water soluble metals: 0.021 (0.006–0.061) $PM_{2.5}$ acidity: 4.5 (1.9–1.07) $PM_{2.5}$ organic carbon: 0.010 (-0.001–0.045) $PM_{2.5}$ elemental carbon: 4.1 (2.2–7.1) **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: r = 0.84; O_3: r = 0.65; NO_2: r = 0.46; CO: r = 0.44; SO_2: r = 0.17; $PM_{10-2.5}$: r = .43; UFP: r = -0.16; $PM_{2.5}$ water-sol metals: r = 0.70; $PM_{2.5}$ sulfates: r = 0.77; $PM_{2.5}$ acidity: r = 0.58; $PM_{2.5}$ organic carbon: r = 0.51; $PM_{2.5}$ elemental carbon: r = 0.48; oxygenated hydrocarbon: r = 31 **Other variables:** Temperature: r = 0.20 Dew point: r = 0.00	**PM Increment:** Approximately 1 SD increase: $PM_{2.5}$: 10 µg/m³ $PM_{2.5}$ water-sol metals: 0.03 µg/m³ $PM_{2.5}$ sulfates: 5 µg/m³ $PM_{2.5}$ acidity: 0.02 µequ/m3 $PM_{2.5}$ organic carbon: 2 µg/m³ $PM_{2.5}$ elemental carbon: 1 µg/m³ **RR [95% CI]:** $PM_{2.5}$ (3-day moving avg): All CVD: 1.033 [1.010, 1.056] Dysrhythmia: 1.015 [0.976, 1.055] Congestive heart failure: 1.055 [1.006–1.105] Ischemic heart disease: 1.023 [0.983–1.064] Peripheral vascular and cerebrovascular disease: 1.050 [1.008–1.093] $PM_{2.5}$ water soluble metals (3-day moving avg): All CVD: 1.027[0.998, 1.056] Dysrhythmia: 1.031 [0.982, 1.082] Congestive heart failure: 1.040 [0.981–1.103] Ischemic heart disease: 1.000 [0.951–1.051] Peripheral vascular and cerebrovascular disease: 1.043 [0.991–1.098] $PM_{2.5}$ sulfates (3-day moving avg): All CVD: 1.003 [0.968, 1.039] Dysrhythmia: 0.986 [0.926, 1.048] Congestive heart failure: 1.009 [0.938–1.085] Ischemic heart disease: 0.997 [0.936–1.062] Peripheral vascular and cerebrovascular disease: 1.025 [0.964–1.090] $PM_{2.5}$ acidity (3-day moving avg): All CVD: 0.994 [0.966, 1.022] Dysrhythmia: 0.991 [0.942, 1.043] Congestive heart failure: 0.989 [0.930–1.052] Ischemic heart disease: 0.992 [0.944–1.043] Peripheral vascular and cerebrovascular disease: 1.004 [0.955–1.056] $PM_{2.5}$ organic carbon (3-day moving avg): All CVD: 1.026 [1.006, 1.046] Dysrhythmia: 1.008 [0.975, 1.044] Congestive heart failure: 1.048 [1.007–1.091] Ischemic heart disease: 1.028 [0.994–1.064] Peripheral vascular and cerebrovascular disease: 1.026 [0.990–1.062] hydrocarbons simultaneously. $PM_{2.5}$ organic carbon (3-day moving avg): All CVD: 1.020 [1.005, 1.036] Dysrhythmia: 1.011 [0.985, 1.037] Congestive heart failure: 1.035 [1.003–1.068] Ischemic heart disease: 1.019 [0.992–1.046] Peripheral vascular and cerebrovascular disease: 1.021 [0.994–1.049] Results for Lags 0–7 expressed in figures (see notes). **Notes:** Figure 1: RR (95% CI) for single-day lag models for the association of ER visits for CVD with daily ambient $PM_{2.5}$ and associated components. Summary of Figure 1 results: Statistically significant positive associations at Lag 0 and Lag 1 for $PM_{2.5}$, at Lag 0 for $PM_{2.5}$ water soluble metals (inverse association at Lag 7), at Lag 0, Lag 1, and Lag 3 for organic and elemental carbon (inverse association at Lag 7). Figure 2: RR (95%) of multipollutant models for the association of ER visits for CVD with daily ambient air quality measurements. Summary of Figure 2 results: Positive association after adjustment for NO_2, CO, and oxygenated hydrocarbons, but attentuated when adjusted for total carbon and null when adjusted for NO_2, CO, total carbon, and oxygenated

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Peng et al. (2008) **Period of Study:** January 1, 1999–December 31, 2005 **Location:** 108 U.S. counties in the following states: Alabama, Arizona, California, Colorado, Connecticut, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin	**Outcome (ICD-9):** Emergency hospitalizations for: Cardiovascular disease, including heart failure (428), heart rhythm disturbances (426–427), cerebrovascular events (430–438), ischemic heart disease (410–414, 429), and peripheral vascular disease (440–448). Respiratory disease, including COPD (490–492) and respiratory tract infections (464–466, 480-487) **Age Groups:** 65 + years, 65–74, ,75 + **Study Design:** Time series **N:** ~ 12 million Medicare enrollees (3.7 million CVD and 1.4 million RD admissions) **Statistical Analyses:** Two-stage Bayesian hierarchical models: Overdispersed Poisson models for county-specific data Bayesian hierarchical models to obtain national avg estimate **Covariates:** Day of the week, age-specific intercept, temperature, dew point temperature, calendar time, indicator for age of 75 years or older. Some models were adjusted for PM_{10}-2.5. **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R version 2.6.2 **Lags Considered:** 0-2 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean** μg/m^3 **(IQR):** All counties assessed: 13.5 (11.1–15.8) Counties in Eastern US: 13.8 (12.3–15.8) Counties in Western US: 11.1 (10.1–14.3) **Monitoring Stations:** At least 1 pair of co-located monitors (physically located in the same place) for PM_{10} and $PM_{2.5}$ per county **Other variables:** Median within-county correlations between monitors: r = 0.92	**PM Increment:** 10 μg/m^3 **Percentage change [95% CI]:** CVD and RD (unadjusted for PM_{10-25}): Lag 0: 0.71 [0.45, 0.96] Lag 2: 0.44 [0.06, 0.82] Most values NR (see note) **Notes:** Effect estimates for PM_{10}-2.5 (0–2 day lags) are showing in Figures 2–5. Figure 2: Percentage change in emergency hospital admissions for CVD per 10 μg/m^3 increase in $PM_{2.5}$ (single pollutant model and model adjusted for PM_{10}-2.5 concentration) Figure 3: Percentage change in emergency hospital admissions for RD per 10 μg/m^3 increase in $PM_{2.5}$ (single pollutant model and model adjusted for PM_{10}-2.5 concentration) No significant associations between $PM_{2.5}$ and cause-specific cardiovascular disease.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Peters et al. (2005) **Period of Study:** February 1999–July 31, 2001 **Location:** Germany: City of Augsburg, County Augsburg, and County Aichach-Friedlberg	**Outcome:** Transmural or nontransmural acute MI **Age Groups:** NR **Study Design:** Case-crossover and time series **N:** 851 MI survivors **Statistical Analyses:** Conditional logistic regression for case-crossover element. Poisson regression for time series element. **Covariates:** Case-crossover: Season, temperature, day of the week, time series: trend, season, influenza, weather, and day of the week **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS, version 8.2 Poisson: R, version 1.7.1 **Lags Considered:** Lags 0–6 h, 0–5 days Poisson: Single lagged days, 5-day, 15-day, 30-day, and 45-day moving averages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h and 24 h **Mean µg/m³ (range; IQR; median; IQR):** 1-h avg: 16.3 (-6.9–355.2; 10.7–19.8; 14.5) 24-h avg: 16.3 (6.1–58.5; 11.6–19.3; 14.9) **Monitoring Stations:** 1 **Copollutant (correlation):** 24-h avg: TNC: r = 0.37; TSP: r = 0.89; PM_{10}: r= 0.92; CO: r = 0.57; NO_2: r = 0.67; NO: r = 0.59; SO_2: r = 0.58; O_3: r = -0.24 1hr avg: TNC: r = 0.42; CO: r = 0.52; NO_2: r = 0.58; NO: r = 0.50; SO_2: r = 0.48; O_3: r = -0.35 **Other variables:** 24-h avg: Temperature: r = 0.05 1-h avg: Temperature: r = -0.01	**PM Increment:** 1-h avg: 9.1 µg/m³ (IQR); 24-h avg: 7.7 µg/m³ (IQR) **OR [95% CI]: Case-Crossover (control selection method (unidirectional with three control periods): 1-h averages:** Lag 0: 0.98 (0.88, 1.10); Lag 1: 0.97 (0.87, 1.09); Lag 2: 0.93 (0.83, 1.04); Lag 3: 0.98 (0.88, 1.09); Lag 4: 0.96 (0.86, 1.07); Lag 5: 0.94 (0.84, 1.05); Lag 6: 0.90 (0.80, 1.01). **24-h averages:** Lag 0: 0.95 (0.83, 1.080); Lag 1: 1.10 (0.96, 1.25); Lag 2: 1.18 (1.03, 1.34); Lag 3: 1.07 (0.94, 1.22); Lag 4: 0.94 (0.83, 1.07); Lag 5: 0.90 (0.79, 1.02) **Case-Crossover (control selection method: bidirectional with 16 control periods):** 24-h averages: Lag 0: 1.03 (0.94, 1.12); Lag 1: 1.07 (0.98, 1.16); Lag 2: 1.08 (0.99, 1.17); Lag 3: 1.01 (0.92, 1.10); Lag 4: 0.96 (0.88, 1.04); Lag 5: 0.93 (0.85, 1.02); Lag 0 -4 (IQR = 5.8): 1.03 (0.94, 1.14) **Unidirectional:** Model 1 (unadjusted): 1.175 (1.033, 1.337); Model 2 (adjusted for day of week using indicator variables): 1.179 (1.035, 1.343); Model 3 (adjusted for temperature-quadratic, linear air pressure): 1.170 (1.028, 1.333); Model 4 (adjusted for temperature-quadratic, linear air pressure, day of week): 1.176 (1.031, 1.341); Model 5 (temperature-quadratic, air pressure-quadratic, relative humidity-quadratic, day of week using indicator variables): 1.170 (1.026, 1.336); Model 6 (temperature–penalized spline, 4.4 df, linear air pressure, day of week using indicator variables): 1.175 (1.030, 1.340; Model 7 (temperature–penalized spline, 4.4 df, linear air pressure, relative humidity–penalized spline, 7.8 df, day of week using indicator variables: 1.177 (1.030, 1.344) **Bidirectional (16 control periods):** Model 1 (unadjusted): 1.077 (0.988, 1.174); Model 2 (adjusted for day of the week using indicator variables): 1.078 (0.988, 1.175); Model 3 (adjusted for temperature-quadratic, linear air pressure): 1.060 (0.970, 1.160); Model 4 (adjusted for temperature-quadratic, linear air pressure, day of the week): 1.060 (0.969, 1.160); Model 5 (temperature-quadratic, air pressure-quadratic, relative humidity-quadratic, day of the week using indicator variables): 1.065 (0.973, 1.166); Model 6 (temperature–penalized spline, 4.4 df, linear air pressure, day of the week using indicator variables): 1.068 (0.976, 1.168); Model 7 (temperature–penalized spline, 4.4 df, linear air pressure, relative humidity–penalized spline, 7.8 df, day of the week using indicator variables: 1.077 (0.983, 1.179) **Bidirectional (4 control periods):** Model 1 (unadjusted): NR Model 2 (adjusted for day of the week by design):1.049 (0.964, 1.141) Model 3 (adjusted for temperature-quadratic, linear air pressure):NR Model 4 (adjusted for temperature-quadratic, linear air pressure, day of the week): 1.032 (0.944, 1.128); Model 5 (temperature-quadratic, air pressure-quadratic, relative humidity-quadratic, day of the week by design): 1.033 (0.945, 1.130); Model 6 (temperature–penalized spline, 4.4 df, linear air pressure, day of the week by design): 1.036 (0.947, 1.132); Model 7 (temperature–penalized spline, 4.4 df, linear air pressure, relative humidity–penalized spline, 7.8 df, day of the week by design): 1.039 (0.950, 1.136) **Stratified:** Model 1 (unadjusted): NR; Model 2 (adjusted for day of week by design):1.059 (0.972, 1.154); Model 3 (adjusted for temperature-quadratic, linear air pressure): NR; Model 4 (adjusted for temperature-quadratic, linear air pressure, day of week): 1.047 (0.957, 1.145) Model 5 (temperature-quadratic, air pressure-quadratic, relative humidity-quadratic, day of week by design): 1.045 (0.954, 1.144); Model 6 (temperature–penalized spline, 4.4 df, linear air pressure, day of week by design): 1.054 (0.964, 1.153)Model 7 (temperature–penalized spline, 4.4 df, linear air pressure, relative humidity–penalized spline, 7.8 df, day of week by design): 1.056 (0.965, 1.156) **RR (95% CI): Time series (24 h avg):** Lag 0: 0.97 (0.89, 1.07); Lag 1: 1.04 (0.96, 1.13); Lag 2: 1.07 (0.98, 1.15); Lag 3: 1.03 (0.95, 1.11); Lag 4: 0.98 (0.90, 1.07); Lag 5: 0.98 (0.90, 1.06); Lag 0–4: 1.03 (0.94, 1.12); Lag 0–14: 1.03 (0.95, 1.13); Lag 0–29: 1.09 (1.01, 1.18); Lag 0–44: 1.08 (1.00, 1.17) **Time series (OR [95% CI]):** Model 1 (unadjusted): 1.059 (0.981, 1.142); Model 2 (adjusted for day of week using indicator variables): 1.056 (0.979, 1.140); Model 3 (adjusted for temperature-quadratic, linear air pressure): 1.062 (0.982, 1.148); Model 4 (adjusted for temperature-quadratic, linear air pressure, day of week): 1.059 (0.979, 1.146); Model 5 (temperature-quadratic, air pressure-quadratic, relative humidity-quadratic, day of week using indicator variables): 1.063 (0.981, 1.151); Model 6 (temperature–penalized spline, 4.4 df, linear air pressure, day of week using indicator variables): 1.065 (0.985, 1.153); Model 7 (temperature–penalized spline, 4.4 df, linear air pressure, relative humidity–penalized spline, 7.8 df, day of week using indicator variables: 1.069 (0.988, 1.157)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Pope et al. (2006) **Period of Study:** 1994-2004 **Location:** Wasatch Front area, Utah	**Outcome:** Myocardial infarction or unstable angina (ICD codes not reported) **Age Groups:** All, <65, 65+ **Study Design:** Case-crossover **N:** 12,865 patients who underwent coronary arteriography **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and dewpoint temperature **Season:** NR **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0- to 3-day lag, 2- to 4-day lagged moving averages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (μg/m³) (SD; maximum):** Ogden: 10.8 (10.6; 108) SLC Hawthorne: 11.3 (11.9; 94) Provo/Orem, Lindom: 10.1 (9.8; 82) **Monitoring Stations:** 3 **Copollutant (correlation):** NR	**PM Increment:** 10 μg/m³ **Percent increase in risk [95% CI]:** Same-day increase in $PM_{2.5}$ (Lag 0): Index MI and unstable angina: 4.81 [0.98–8.79] Subsequent MI: 3.23 [-3.87, 10.85] All acute coronary events: 4.46 [1.07–7.97] All acute coronary events excluding observations using imputed $PM_{2.5}$ data: 4.24 [0.33–8.31] Stable presentation: -2.57 [-5.39, 0.34] Remaining results summarized in figures (see notes). **Notes:** Figure 1: Percent increase in risk (and 95% CI) of acute coronary events associated with 10 μg/m³ of $PM_{2.5}$ for different lag structures. Summary of Figure 1: Positive, statistically significant association seen for Lag 0, Lag 1; and 2, 3, and 4 day moving averages. Positive but non-statistically significant associations seen for Lags 2 and 3. Figure 2: Percent increase in risk (and 95% CI) of acute coronary events associated with 10 μg/m³ of $PM_{2.5}$ stratified by various characteristics.
Reference: Sarnat et al. (2008) **Period of Study:** November 1998–December 2002 **Location:** Atlanta (Georgia) metropolitan area	**Outcome (ICD-9):** Cardiovascular disease ED visits:Ischemic heart disease (410–414), cardiac dysrhythmias (427), congestive heart failure (428), and peripheral vascular and cerebrovascular disease (433–437, 440, 443–444, 451-453) **Age Groups:** All **Study Design:** Time series **N:** >4.5 million emergency department visits **Statistical Analyses:** Poisson generalized linear models **Covariates:** Day of the week, holidays, hospital, long-term trends, temperature, dewpoint temperature **Season:** All, warm season (April 15–October 14), and cool season (October 15–April 14). **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0-day lag	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (μg/m³) (median; 10th-90th percentile):** Total $PM_{2.5}$: Cool season:15.8 (14.3; 7.5–25.5). Warm season: 18.2 (17.0; 9.1–29.0) $PM_{2.5}$ elemental carbon: Cool: 1.7 (1.4; 0.6–3.3). Warm: 1.4 (1.3; 0.6–2.5) $PM_{2.5}$ Zn (ng/m³): Cool: 15.7 (11.7; 4.6–30.2) Warm: 10.9 (8.5; 3.3–20.2) $PM_{2.5}$ K (ng/m³): Cool: 63.0 (53.9; 24.3–114.2) Warm: 52.7 (43.3; 23.2–93.5) $PM_{2.5}$ Si (ng/m³): Cool: 67.7 (54.1; 24.3–123.5). Warm: 110.9 (89.0; 32.9–186.3) $PM_{2.5}$ SO_4^{2-}: Cool: 3.4 (0.6; 1.5–5.8). Warm: 6.0 (5.2; 2.3–10.8) $PM_{2.5}$ NO_3-: Cool: 1.4 (1.2; 0.5–2.6). Warm: 0.7 (2.9; 0.3–1.2) $PM_{2.5}$ Se (ng/m³): Cool: 1.4 (1.1; 0.4–3.0). Warm: 1.2 (0.9; 0.4–2.7) $PM_{2.5}$ OC: Cool: 4.6 (3.9; 1.9–8.0) Warm: 4.0 (3.7; 2.1–6.4) **Monitoring Stations:** 1 **Copollutants:** NR	**PM Increment:** IQR (specific values not given) **Risk ratio [95% CI]: CVD (Lag 0):** All seasons: Total $PM_{2.5}$: 1.022 [1.007, 1.038] $PM_{2.5}$ elemental carbon: 1.02 [1.013–1.037] $PM_{2.5}$ zinc: 1.013 [1.005–1.022] $PM_{2.5}$ potassium: 1.030 [1.018–1.042] $PM_{2.5}$ silicon: 1.008 [1.00–1.016] $PM_{2.5}$ sulfate: 1.007 [0.994–1.019] $PM_{2.5}$ nitrate: 1.002 [0.990–1.014] $PM_{2.5}$ selenium: 1.002 [0.991–1.012] $PM_{2.5}$ organic carbon: 1.024 [1.013–1.035] Cool season: Total $PM_{2.5}$: 1.028 [1.012–1.044] $PM_{2.5}$ EC: 1.029 [1.015–1.044] $PM_{2.5}$ Zinc: 1.012 [1.002–1.022] $PM_{2.5}$ K: 1.037 [1.021–1.054] $PM_{2.5}$ Si: 1.022 [1.002–1.043] $PM_{2.5}$ sulfate: 1.014 [0.991–1.037] $PM_{2.5}$ nitrate: 1.006 [0.993–1.019] $PM_{2.5}$ Se: 1.012 [0.997–1.027] $PM_{2.5}$ organic carbon: 1.027 [1.013–1.040] Warm season: Total $PM_{2.5}$: 1.006 [0.990–1.022] $PM_{2.5}$ EC: 1.021 [1.000–1.043] $PM_{2.5}$ Zinc: 1.017 [1.002–1.033] $PM_{2.5}$ K: 1.024 [1.007–1.041] $PM_{2.5}$ Si: 1.005 [0.996–1.014] $PM_{2.5}$ sulfate: 1.001 [0.988–1.015] $PM_{2.5}$ nitrate: 1.000 [0.969–1.033] $PM_{2.5}$ Se: 0.996 [0.981–1.011] $PM_{2.5}$ organic carbon: 1.027 [1.004–1.051]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Sullivan et al. (2005a) **Period of Study:** 1988-1994 **Location:** King County, Washington	**Outcome:** Acute MI **Age Groups:** All, <50, 50–59, 70+ **Study Design:** Case-crossover **N:** 5793 cases of acute MI (5793 case days and 20,134 referent exposure days from these case individuals) **Statistical Analyses:** Conditional logistic regression **Covariates:** Relative humidity, temperature, season, day of week **Season:** All, and also conducted stratified analysis by season of event (heating season: November–February; nonheating season: March-October) **Dose-response Investigated:** No **Statistical Package:** SAS version 8.0 and SPSS version 10 **Lags Considered:** Lag 1 and Lag 2 for 24-h avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h, 2 h, 4 h, and 24 h **Summary of PM2.5 1 h before MI onset:** **Mean (μg/m³) (median; IQR, 90th percentile; range):** 12.8 (8.6; 5.3–15.9; 27.3; 2.0–147) **Monitoring Stations:** 3 **Copollutant (correlation):** 1-h avg: PM_{10}: r = 0.78 CO: r = 0.47 SO_2: r = 0.16	**PM Increment:** 10 μg/m³ **Odds ratio [95% CI]:** 1-h **Averaging Time:** 1.01 [0.98, 1.05] **2-h Averaging Time:** 1.01 [0.97, 1.05] **4-h Averaging Time:** 1.02 [0.98, 1.04] **24-h Averaging Time:** 1.02 [0.98, 1.07] Association between $PM_{2.5}$ (24 h) lagged 1 or 2 days non-significant (data not shown) Season (1-h avg): Heating: 1.01 [0.98–1.05]; Nonheating: 0.99 [0.91–1.09] Age (1-h avg): <50 years: 1.04 [0.95, 1.14]; 50–60 years: 0.99 [0.94, 1.05]; 70+ years: 1.03 [0.98, 1.08] Age (24-h avg): <50 years: 1.07 [0.98, 1.19]; 50–69 years: 0.99 [0.93, 1.06]; 70+ years: 1.04 [0.99, 1.11] Sex (1-h avg): Men: 1.02 [0.98, 1.06]; Women: 1.00 [0.95, 1.06] Sex (24-h avg): Men: 1.03 [0.99, 1.08]; Women: 1.00 [0.94, 1.07] Race (1-h avg): White: 1.01 [0.97, 1.04]; Nonwhite: 1.06 [0.97, 1.17] Race (24-h avg): White: 1.01 [0.97, 1.06]; Nonwhite: 1.10 [0.99, 1.23] Smoking status (1-h avg): Current: 0.99 [0.93, 1.06]; Nonsmoker: 1.03 [0.97, 1.08] Smoking status (24-h avg): Current: 0.99 [0.95, 1.14]; Nonsmoker: 1.03 [0.98, 1.09] Survivor of MI * (1-h avg): Yes:1.02 [0.98, 1.06]; No: 0.96 [0.86, 1.08] Survivor of MI * (24-h avg): Yes:1.03 [0.98, 1.07]; No: 0.97 [0.85, 1.10] Previous congestive heart failure (1 h avg): Yes: 1.06 [0.97, 1.16]; No: 1.00 [0.97, 1.04] Previous congestive heart failure (24-h avg): Yes:1.08 [0.97, 1.2]; No: 1.00 [0.97, 1.04] Previous MI (1-h avg): Yes: 1.03 [0.97, 1.1]; No: 1.01 [0.96, 1.06] Previous MI (24-h avg): Yes: 1.04 [0.97, 1.17]; No: 1.02 [0.98, 1.08] Hypertension (1-h avg): Yes:1.02 [0.97, 1.07]; No: 1.01 [0.96, 1.06] Hypertension (24-h avg): Yes: 1.02 [0.97, 1.07]; No: 1.02 [0.97, 1.08] Diabetes mellitus (1-h avg): Yes:1.06 [0.98, 1.14]; No: 1.01 [0.97, 1.05] Diabetes mellitus (24-h avg): Yes:1.04 [0.95, 1.14]; No: 1.01 [0.97, 1.06] *Compares those who survive hospitalization (yes) with those who died in hospital from complications of MI.
Reference: Symons et al. (2006) **Period of Study:** Apr–Dec, 2002 **Location:** Baltimore, Maryland	**Outcome:** Congestive heart failure **Age Groups:** All **Study Design:** Case-crossover **N:** 125 patients **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and humidity **Season:** NR **Dose-response Investigated:** Yes **Statistical Package:** SAS and S-Plus **Lags Considered:** 0-3 days (single and cumulative)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 8 & 24 h **Mean (min-max):** 8 h 17.0 (0.1-111.9) SD = 12.7 24 h 16.0 (3.5-69.2) SD = 10.0 **Monitoring Stations:** 8 **Copollutant (correlation):** NR	**PM Increment:** 9.2 μg/m³ (IQR) **RR Estimate [CI]:** 8 h (participant's onset period) Same-day lag: 0.87 [0.69,1.09] 1-day lag: 0.96 [0.78,1.18] 2-day lag: 1.09 [0.91,1.30] 3-day lag: 0.99 [0.79,1.23] Cumulative 1-day lag: 0.89 [0.67,1.16] Cumulative 2-day lag: 0.99 [0.74,1.33] Cumulative 3-day lag: 0.98 [0.70,1.36] 24 h avg Same-day lag: 0.81 [0.65,1.01] 1-day lag: 0.90 [0.74,1.11] 2-day lag: 0.85 [0.68,1.07] 3-day lag: 0.86 [0.70,1.05] Cumulative 1-day lag: 0.82 [0.64,1.04] Cumulative 2-day lag: 0.76 [0.57,1.01] Cumulative 3-day lag: 0.70 [0.51,0.97] **Notes:** β coefficients presented in Fig 5

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Tolbert et al. (2007) **Period of Study:** August 1998–December 2004 **Location:** Atlanta Metropolitan area, Georgia	**Outcome (ICD-9):** Combined CVD group, including: Ischemic heart disease (410–414), cardiac dysrhythmias (427), congestive heart failure (428), and peripheral vascular and cardiovascular disease (433–437, 440, 443–445, and 451–453) **Age Groups:** All **Study Design:** Time series **N:** NR for 1998–2004. For 1993–2004: 10,234,490 ER visits (283,360 and 1,072,429 visits included in the CVD and RD groups, respectively) **Statistical Analyses:** Poisson generalized linear models **Covariates:** long-term temporal trends, season (for RD outcome), temperature, dew point, days of week, federal holidays, hospital entry and exit **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 9.1 **Lags Considered:** 3-day moving avg(lag 0 -2)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (µg/m³) (median; IQR, range, 10th –90th percentiles):** $PM_{2.5}$: 17.1 (15.6; 11.0–21.9; 0.8–65.8; 7.9–28.8). $PM_{2.5}$ sulfate: 4.9 (3.9; 2.4–6.2; 0.5–21.9; 1.7–9.5). $PM_{2.5}$ organic carbon: 4.4 (3.8; 2.7–5.3; 0.4–25.9; 2.1–7.2). $PM_{2.5}$ elemental carbon: 1.6 (1.3; 0.9–2.0; 0.1–11.9; 0.6–3.0). $PM_{2.5}$ water-soluble metals: 0.030 (0.023; 0.014–0.039; 0.003–0.202; 0.009–0.059) **Monitoring Stations:** 1 **Copollutant (correlation):** Between $PM_{2.5}$ and:; PM_{10}: r = 0.84; O_3: r = 0.62; NO_2: r = 0.47; CO: r = 0.47; SO_2: r = 0.17; PM_{10}-2.5: r = 0.47; $PM_{2.5}$ SO4: r = 0.76; $PM_{2.5}$ EC: r = 0.65; $PM_{2.5}$ OC: r = 0.70; $PM_{2.5}$ TC: r = 0.71; $PM_{2.5}$ water-sol metals: r = 0.69; OHC: r = 0.50 Between $PM_{2.5}$ SO_4 and: PM_{10}: r = 0.69; O_3: r = 0.56; NO_2: r = 0.14; CO: r = 0.14; SO_2: r = 0.09; PM_{10}-2.5: r = 0.32; $PM_{2.5}$: r = 0.76; $PM_{2.5}$ EC: r = 0.32; $PM_{2.5}$ OC: r = 0.33; $PM_{2.5}$ TC: r = 0.34; $PM_{2.5}$ water-sol metals: r = 0.65; OHC: r = 0.47 Between $PM_{2.5}$ elemental carbon and:; PM_{10}: r = 0.61; O_3: r = 0.40; NO_2: r = 0.64; CO: r = 0.66; SO_2: r = 0.22; PM_{10}-2.5: r = 0.49; $PM_{2.5}$: r = 0.65$PM_{2.5}$; SO_4: r = 0.32; $PM_{2.5}$ OC: r = 0.82; $PM_{2.5}$ TC: r = 0.91; $PM_{2.5}$ water-sol metals: r = 0.52; OHC: r = 0.35 Between $PM_{2.5}$ organic carbon and:; PM_{10}: r = 0.65; O_3: r = 0.54; NO_2: r = 0.62; CO: r = 0.59; SO_2: r = 0.17; PM_{10}-2.5: r = 0.49; $PM_{2.5}$: r = 0.70; $PM_{2.5}$ SO_4: r = 0.33; $PM_{2.5}$ EC: r = 0.82; $PM_{2.5}$ TC: r = 0.98; $PM_{2.5}$ water-sol metals: r = 0.49; OHC: r = 0.37 Between $PM_{2.5}$ total carbon and:; PM_{10}: r = 0.67; O_3: r = 0.52; NO_2: r = 0.65; CO: r = 0.63; SO_2: r = 0.19; PM_{10}-2.5: r = 0.51; $PM_{2.5}$: r = 0.71; $PM_{2.5}$ SO4: r = 0.34; $PM_{2.5}$ EC: r = 0.91; $PM_{2.5}$ OC: r = 0.98; $PM_{2.5}$ water-sol metals: r = 0.52; OHC: r = 0.38 Between $PM_{2.5}$ water-soluble metals and: PM_{10}: r = 0.73; O_3: r = 0.43; NO_2: r = 0.32; CO: r = 0.35; SO_2: r = 0.06; PM_{10}-2.5: r = 0.50; $PM_{2.5}$: r = 0.69; $PM_{2.5}$ SO_4: r = 0.65; $PM_{2.5}$ EC: r = 0.52; $PM_{2.5}$ OC: r = 0.49; $PM_{2.5}$ TC: r = 0.52	**PM Increment:** $PM_{2.5}$: 10.96 µg/m³ (IQR) $PM_{2.5}$ sulfate: 3.82 µg/m³ (IQR) $PM_{2.5}$ total carbon: 3.63 µg/m³ (IQR) $PM_{2.5}$ organic carbon: 2.61 µg/m³ (IQR) $PM_{2.5}$ elemental carbon: 1.15 µg/m³ (IQR) $PM_{2.5}$ water-soluble metals: 0.03 µg/m³ (IQR) **Risk ratio [95% CI] (single pollutant models):** $PM_{2.5}$: CVD: 1.005 [0.993–1.017] $PM_{2.5}$ sulfate: CVD: 0.999 [0.987–1.011] $PM_{2.5}$ total carbon: CVD: 1.016 [1.005–1.026] $PM_{2.5}$ organic carbon: CVD: 1.015 [1.005–1.026] $PM_{2.5}$ elemental carbon: CVD: 1.015 [1.005–1.025] $PM_{2.5}$ water-soluble metals: CVD: 1.009 [0.997–1.021] **Notes:** Results of selected multi-pollutant models for cardiovascular disease are presented in Figure 1. Figure 1: $PM_{2.5}$ total carbon adjusted for CO, NO_2, or NO_2+CO Summary of results: $PM_{2.5}$ total carbon continued to have a positive, statistically significant association with CVD after adjustment for NO_2 but not after adjustmen

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2006) **Period of Study:** 1 Apr, 1992–31 Mar, 2002 **Location:** Edmonton, Canada	**Outcome (ICD-9):** Stroke (430-438), including ischemic stroke (434-436), hemorrhagic stroke (430,432), and transient ischemic attacks (TIA) (435). **Age Groups:** 65+ yrs **Study Design:** Case-crossover **N:** 12,422 visits **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and relative humidity **Season:** Summer (Apr-Sep), winter (Oct-Mar) **Dose-response Investigated:** No **Statistical Package:** SAS (PHREG) **Lags Considered:** 0, 1, and 3-day	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean** µg/m³ **(SD):** All year: 8.5 (6.2) Summer: 8.7 (7.1) Winter: 8.3 (5.2) **Monitoring Stations:** 3 **Copollutant (correlation):** All year SO_2: r = 0.22 NO_2: r = 0.41 CO: r = 0.43 O_3-mean: r = -0.07 O_3-max: r = 0.07 PM_{10}: r = 0.79 Summer SO_2: r = 0.20 NO_2: r = 0.52 CO: r = 0.42 O_3-mean: r = 0.11 O_3-max: r = 0.34 PM_{10}: r = 0.85 Winter SO_2: r = 0.28 NO_2: r = 0.57 CO: r = 0.71 O_3-mean: r = -0.45 O_3-max: r = -0.35 PM_{10}: r = 0.70	**PM Increment:** µg/m³ **(IQR)** All year: 6.3 Summer: 6.5 Winter: 6.0 Adjusted OR Estimate [CI]: Acute ischemic stroke All year: Same-day lag: 1.00 [0.96,1.04] 1-day lag: 1.00 [0.96,1.05]; 3-day lag: 1.01 [0.96,1.06] Summer: Same-day lag: 0.96 [0.90,1.03] 1-day lag: 1.01 [0.94,1.07]; 3-day lag: 0.98 [0.89 [1.07] Winter: Same-day lag: 1.04 [0.99,1.10] 1-day lag: 1.01 [0.96,1.07]; 3-day lag: 1.05 [0.98,1.13] Hemorrhagic stroke All year: Same-day lag: 0.99 [0.90,1.08] 1-day lag: 1.07 [0.98,1.16]; 3-day lag: 1.05 [0.93,1.19] Summer: Same-day lag: 0.99 [0.86,1.15] 1-day lag: 1.12 [0.97,1.30]; 3-day lag: 1.08 [0.88,1.31] Winter: Same-day lag: 1.04 [0.92,1.18] 1-day lag: 1.08 [0.97,1.20]; 3-day lag: 1.11 [0.94,1.31] Transient cerebral ischemic attack All year: Same-day lag: 0.98 [0.93,1.03] 1-day lag: 0.99 [0.95,1.04]; 3-day lag: 0.96 [0.90,1.03] Summer: Same-day lag: 1.00 [0.92,1.08] 1-day lag: 1.03 [0.95,1.12]; 3-day lag: 0.98 [0.88,1.09] Winter: Same-day lag: 0.97 [0.90,1.05] 1-day lag: 0.97 [0.91,1.04]; 3-day lag: 0.94 [0.86,1.03] **Notes:** Adjusted ORs are provided for an IQR increase in the 3-day mean in Fig 1-4 for single nd two-pollutant models.
Reference: Zanobetti and Schwartz (2006) **Period of Study:** 1995-1999 **Location:** Boston Metropolitan area	**Outcome (ICD-9):** Myocardial infarction (410) or pneumonia (480–487) **Age Groups:** 65 + years **Study Design:** Case-crossover **N:** 15,578 patients admitted for MI and 25,857 admitted for pneumonia **Statistical Analyses:** conditional logistic regression **Covariates:** temperature, day of the week. **Season:** All, and also tested for interaction by warm (April–September) vs. cold season **Dose-response Investigated:** No **Statistical Package:** SAS version 8.2 (PROC PHREG) **Lags Considered:** lag 0 , and mean of lags 0 -1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Median** (µg/m³) **(IQR; 5th-95th percentile):** 11.1 (7.23-16.14; 3.87–26.31) **Monitoring Stations:** 1 **Copollutant (correlation):** BC: r = 0.66 NO_2: r = 0.55 CO: r = 0.52 O_3: r = 0.20 PM non-traffic: r = 0.74	**PM Increment:** Difference between the 90th and 10th percentile for $PM_{2.5}$ Myocardial infarcation cohort (Lag 0): 17.17 µg/m³ Myocardial infarcation cohort (Lag 0-1): 16.32 µg/m³ Pneumonia cohort (Lag 0): 17.14 µg/m³ Pneumonia cohort (Lag 0): 16.32 µg/m³ **Percentage (%) increase in risk [95% CI]:** Myocardial infarction cohort: Lag 0: 8.50 (1.89–14.43) Lag 0–1: 8.65 (1.22–15.38) Pneumonia cohort: Lag 0: 6.48 (1.13–11.43) Lag 0–1: 5.56 (-0.45, 11.27) **Notes:** Assessed for effect modification by season. Results are reported in Figure 2. Summary of results: $PM_{2.5}$ is associated with pneumonia hospitalization in the cold season but not the hot season. $PM_{2.5}$ is associated with MI hospitalization in the hot season but not the cold season.

Table E-8. Short-term exposure to other PM size fractions and emergency department visits and hospital admissions for cardiovascular outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Andersen et al. (2008b) **Period of Study:** May 2001-December 2004 **Location:** Copenhagen, Denmark	**Outcome (ICD-10):** CVD, including angina pectoris (I20), myocardial infarction (I21–22), other actue ischemic heart diseases (I24), chronic ischaemic heart disease (I25), pulmonary embolism (I26), cardiac arrest (I46), cardiac arrhythmias (I48–48), and heart failure (I50). **Age Groups:** >65 yrs (CVD and RD), 5–18 years (asthma) **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson GAM **Covariates:** temperature, dew-point temperature, long-term trend, seasonality, influenza, day of the week, public holidays. **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R statistical software (gam procedure, mgcv package) **Lags Considered:** Lag 0 -5 days, 4-day pollutant avg (lag 0 -3) for CVD, 5-day avg (lag 0–4) for RD, and a 6-day avg (lag 0–5) for asthma.	**Pollutant:** Total number concentration of ultrafine and accumulation mode particles (NCtot) (particles/cm^3) **Averaging Time:** 24 h **Mean (SD; median; IQR; 99th percentile:** NCtot*: 8116 (3502; 7358; 5738–9645, 19,895) NCa12: 493 (315; 463; 308–650; 1463) Nca23: 2253 (1364; 2057; 1280–3066; 6096) NCa57: 5104 (2687; 4562; 3248–6274; 14,410) NC100: 6847 (2864; 6243; 4959–8218; 16189) NC_{a212}: 392 (441; 89; 246–584; 2248) *NC, number concentration; tot, total (all particles 6–700 in diameter); a12, size mode with mean diameter of 12 nm; a23, size mode with median diameter of 23 nm; a57, size mode with median diameter of 57 nm; a212; size mode with median diameter of 212 nm; NC100 = a12+a23+0.797*a57+0.084*a212. **Monitoring Stations:** 1 **Copollutant (correlation):** Correlation of NCtot with: PM_{10}: r = 0.39 $PM_{2.5}$: r = 0.40 NO_2: r = 0.68 NO_X: r = 0.66 NC100: r = 0.98 NCa12: r = 0.31 NCa23: r = 0.57 NCa57: r = 0.87 NC_{a212}: r = 0.29 CO: r = 0.54 NO_X curbside: r = 0.36 O_3: r = -0.12 **Other variables:** Temperature: r = -0.06 Relative humidity: r = -0.04	**PM Increment: IQR increase in pollutant level:** Nctot**:** 3907 particles/cm^3 (IQR) Nca12: 342 particles/cm^3 (IQR) Nca23: 1786 particles/cm^3 (IQR) Nca57: 3026 particles/cm^3 (IQR) NC100: 3259 particles/cm^3 (IQR) Nca212: 495 particles/cm^3 (IQR) **Relative risk (RR) Estimate [CI]:** CVD hospital admissions (4 day avg, lag 0 -3), age 65+ One-pollutant model (NCtot): 1.00 [0.99–1.02] Adj for PM_{10}: 0.98 [0.96–1.01] Adj for $PM_{2.5}$: 0.99 [0.95–1.03] Adj for CO: 0.99 [0.97–1.02] Adj for NO_2: 1.01 [0.98–1.03] Adj for O_3: 1.01 [0.96–1.06] One-pollutant model (NC100): 1.00 [0.98–1.02] One pollutant model (Nca12): 0.99 [.97–1.01] Adj for other size fractions: 0.99 [0.97–1.02] One pollutant model (Nca23): 0.99 [0.96–1.01] Adj for other size fractions: 0.99 [0.96–1.02] One pollutant model (NCa57): 1.01 [0.98–1.02] Adj for other size fractions: 0.99 [0.97–1.02] One pollutant model (Nca212): 1.02 [1.00–1.04] Adj for other size fractions: 1.02 [1.00–1.05] Adj for PM_{10}: 0.98 [0.95–1.01] **Notes:** Figure 2: Relative risks and 95% confidence intervals per IQR in single day concentration (0–5 day lag). Summary of Figure 2: CVD: Positive, marginally or statistically significant associations at Lag 2 (Nctot, Nca57, Nca212), Lag 3 (Nca212), and Lag 1 (Nca212).

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lanki et al. (2006a) **Period of Study:** 1992-2000 **Location:** Augsburg, Barcelona, Helsinki, Rome, and Stockholm	**Outcome (ICD-9):** Acute myocardial infarction (410; ICD-10: I21, I22) **Age Groups:** 35+ yrs, <75 yrs, 75+ yrs **Study Design:** Time series **N:** 26,854 hospitalizations **Statistical Analyses:** GAM **Covariates:** Temperature, barometric pressure **Season:** Warm (Apr-Sep) and cold (Oct-Mar) **Dose-response Investigated:** No **Statistical Package:** R package mgcv 0.9-5 **Lags Considered:** 0-3 days	**Pollutant:** UFP (PNC) **Averaging Time:** 24 h **Median particles/cm3:** Augsburg: 12,400 Barcelona: 76,300 Helsinki: 13,600 Rome: 46,000 Stockholm: 11,800 **Copollutant (correlation):** Augsburg PM_{10}: r = 0.53; CO: r = 0.63; NO_2: r = 0.65; O_3: r = 0.26 Barcelona: PM_{10}: r = 0.38; CO: r = 0.80; NO_2: r = 0.49; O_3: r = -0.35 Helsinki: PM_{10}: r = 0.45; CO: r = 0.48; NO_2: r = 0.82; O_3: r = 0.01 Rome: PM_{10}: r = 0.32; CO: r = 0.83; NO_2: r = 0.68; O_3: r = 0.03 Stockholm: PM_{10}: r = 0.06; CO: r = 0.56; NO_2: r = 0.83; O_3: r = -0.01	**PM Increment:** 10,000 particles/cm3 **Pooled Rate Ratio [CI]:** All 5 cities (35+ yrs) Same-day lag: 1.005 [0.996,1.015]; 1-day lag: 0.997 [0.982,1.012]; 2-day lag: 0.999 [0.990,1.008]; 3-day lag: 0.998 [0.979,1.017] 3 cities with hospital discharge register (35+ yrs) Same-day lag: 1.013 [1.000,1.026]; 1-day lag: 0.995 [0.953,1.039]; 2-day lag: 1.001 [0.989,1.014]; 3-day lag: 1.009 [0.974,1.046] Warm season (35+ yrs) Same-day lag: 1.009 [0.972,1.048]; 1-day lag: 1.023 [0.988,1.060]; 2-day lag: 1.050 [1.016,1.085]; 3-day lag: 1.022 [0.987,1.058] Cold season (35+ yrs) Same-day lag: 1.014 [1.001,1.028]; 1-day lag: 1.001 [0.956,1.048]; 2-day lag: 1.001 [0.989,1.014]; 3-day lag: 1.009 [0.971,1.049] Age >75Non-fatal Same-day lag: 1.032 [1.008,1.056]; 1-day lag: 1.009 [0.985,1.032]; 2-day lag: 0.989 [0.966,1.013]; 3-day lag: 1.009 [0.969,1.051] Fatal Same-day lag: 1.016 [0.978,1.055]; 1-day lag: 1.001 [0.966,1.038]; 2-day lag: 1.005 [0.969,1.041]; 3-day lag: 0.984 [0.948,1.021] **Notes:** Rate ratios for PNC are given for 0-5 lag days in graph form (Fig 1) for each city. Pooled rate ratios were also provided for groups <75 yielding similar results to the overall 3-city data.
Reference: Metzger et al. (2004) **Period of Study:** August 1998–August 2000 **Location:** Atlanta Metropolitan area (Georgia)	**Outcome (ICD-9):** Emergency visits for ischemic heart disease (410–414), cardiac dysrhythmias (427), cardiac arrest (427.5), congestive heart failure (428), peripheral vascular and cerebrovascular disease (433-437, 440, 443-444, 451–453), atherosclerosis (440), and stroke (436). **Age Groups:** All **Study Design:** Time series **N:** 4,407,535 emergency department visits between 1993–2000 (data not reported for 1998-2000) **Statistical Analyses:** Poisson generalized linear modeling **Covariates:** Day of the week, hospital entry and exit indicator variables, federally observed holidays, temporal trends, temperature, dew point temperature **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS **Lags Considered:** 3-day moving avg, lags 0-7	**Pollutant:** UFP (10–100 nm particle count) (no/cm^3) **Averaging Time:** 24 h **Median (10%-90% range):** 25,900 (11,500-74,600) **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: r = -0.13; O_3: r = -0.13; NO_2: r = 0.26; CO: r = 0.10; SO_2: r = 0.24; $PM_{2.5}$: r = -0.16; $PM_{2.5}$ water soluble metals: r = -0.27; $PM_{2.5}$ sulfates: r = -0.31; $PM_{2.5}$ acidity: r = -0.39; $PM_{2.5}$ organic carbon: r = 0.08; $PM_{2.5}$ elemental carbon: r = 0.08; $PM_{2.5}$ oxygenated hydrocarbon: r = 0.05 **Other variables:** Temperature: r = -0.33 Dew point: r = -0.41	**PM Increment:** 30,000 no/cm^3 (approximately 1 SD)3 **RR [95% CI]:** For 3 day moving avg: All CVD: 0.985 [0.965, 1.005] Dysrhythmia: 0.972 [0.937, 1.008] Congestive heart failure: 0.983 [0.943–1.025] Ischemic heart disease: 0.989 [0.953–1.026] Peripheral vascular and cerebrovascular disease: 0.998 [0.960–1.039] Results for Lags 0–7 expressed in figures (see notes). **Notes:** Figure 1: RR (95% CI) for single-day lag models for the association of ER visits for CVD with daily ambient UFP. Summary of Figure 1 results: Null or negative associations.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: von Klot et al. (2005) **Period of Study:** 1992-2001 **Location:** Augsburg, Germany; Barcelona, Spain; Helsinki, Finland; Rome, Italy; Stockholm, Sweden	**Outcome (ICD-9):** Acute myocardial infarction (410; ICD-10: I21-I22), angina pectoris (411, 413; ICD-10: I20, I24), dysrhythmia (427; ICD-10: I46.0, 46.9, I47-I49, R00.1, R00.8), heart failure (428; ICD-10: 150) **Age Groups:** 35+ yrs **Study Design:** Cohort **N:** 22,006 MI survivors **Statistical Analyses:** GAM, Spearman correlation **Covariates:** Temperature, dew point temp, avg barometric pressure, relative humidity **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R-software with "mgcv" package **Lags Considered:** 0-3 days	**Pollutant: UFP (**PNC) **Averaging Time:** 24 h **Mean particle/cm3 (5th–95th percentile):** Augsburg: Barcelona: Helsinki: Rome: Stockholm: **Monitoring Stations:** NR **Copollutant (correlation):** Augsburg PM_{10}: r = 0.52; CO: r = 0.63; NO_2: r = 0.64; O_3: r =-0.32Barcelona PM_{10}: r = 0.29; CO: r = 0.71; NO_2: r = 0.44; O_3: r =-0.55 Helsinki PM_{10}: r = 0.46; CO: r = 0.47; NO_2: r = 0.83; O_3: r =-0.16 Rome PM_{10}: r = 0.33; CO: r = 0.80; NO_2: r = 0.71; O_3: r =-0.47 Stockholm PM_{10}: r = 0.06; CO: r = 0.54; NO_2: r = 0.80; O_3: r =-0.17	**PM Increment:** 10,000 particles/cm3 **Pooled RR Estimate [CI]:** All cardiac admissions: 1.026 [1.005,1.048] Myocardial infarction: 1.039 [0.998.1.082] Angina pectoris: 1.020 [0.992,1.048]

E.2. Short-Term Exposure and Respiratory Outcomes

E.2.1. Panel Studies

Table E-9. Short-term exposure to PM_{10} and respiratory morbidity outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Aekplakorn, et al. (2003) **Period of Study:** 107 days, from October 1, 1997 to January 15, 1998 **Location:** Mae Mo district, Lampang Province, north Thailand	**Outcome:** Upper respiratory symptoms, lower respiratory symptoms, cough **Age Groups:** 6-14 years old **Study Design:** Logistic regression **N:** 98 asthmatic school children **Statistical Analyses:** GEE, stratified analysis, PROC GENMOD **Covariates:** Temperature and relative humidity **Season:** winter **Dose-response Investigated?** No **Statistical Package:** SAS v 8.1	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** Sob Pad station: 31.92 Sob Mo station: 16.99 Hua Fai station: 37.45 **Range (Min, Max):** Sob Pad: 6.63, 31.92 Sob Mo: 4.23, 33.64 Hua Fai: 6.98, 37.45 **Monitoring Stations**: 3 **Copollutant :** $PM_{2.5}$, SO_2	**PM Increment:** 10 µg/m^3 **Odds Ratios [Lower CI, Upper CI]; lag:** Asthmatics: URS: 1.03 (0.99, 1.07); lag 0 LRS: 1.04 (0.99, 1.09); lag 0 Cough: 1.04 (1.00, 1.07); lag 0 Non-Asthmatics: URS: 1.04 (0.99, 1.08); lag 0 LRS: 1.0 (0.93, 1.07); lag 0 Cough: 0.99 (0.94, 1.05); lag 0 PM_{10} + SO_2 Asthmatics: URS: 1.03 (0.99, 1.07); lag 0 LRS: 1.03 (0.98, 1.09); lag 0 Cough: 1.04 (1.00, 1.08); lag 0 Non-Asthmatics: URS: 1.04 (0.99, 1.08); lag 0 LRS: 1.0 (0.93, 1.07); lag 0 Cough: 0.99 (0.95, 1.05); lag 0

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Andersen et al. (2008a) **Period of Study:** Dec 12, 1998–Dec 19, 2004 **Location:** Copenhagen, Denmark	**Outcome:** Daily symptoms (prospective daily recording of symptoms via diary) **Age Groups:** 0-3 yrs **Study Design:** Panel study of children with genetic susceptibility to asthma (mothers had asthma) **N:** 205 children (living within a 15km radius of the central monitor during the first 3 yrs of life); born between Aug 2, 1998 and Dec 12, 2001 **Statistical Analyses:** logistic regression model (GEE) **Covariates:** temperature, season, gender, age, exposure to smoking, and paternal history of asthma **Effect modification:** gender, medication use, and paternal history of asthma **Statistical Package:** SAS v9.1 **Lag:** 0,1,2,3,4,2-4	**Pollutant:** PM_{10} **Mean:** 25.1 SD: 16.7 **Percentiles:** 25th: 15.7 75th: 30.2 IQR: 14.5 **Copollutant (correlation):** $PM_{2.5}$ (r = 0.79) Number concentration of ultrafine particles, UFP (r = 0.37) NO_2 (r = 0.43) NO_X (r = 0.40) CO (r = 0.45) O_3 (r = -0.32) Temp (r = 0.25)	**PM Increment:** IQR (14.5 µg/m^3) increase Odds Ratios (95%CI) for incident wheezing symptoms Age 0-1 L0: 1.05 (0.88, 1.25); L1: 1.00 (0.82, 1.22); L2: 1.01 (0.83, 1.23); L3: 1.20 (0.98, 1.46); L4: 1.23 (1.02, 1.48); L2-4: 1.21 (0.99, 1.48) Age 1-2 L0: 1.00 (0.86, 1.15); L1: 1.02 (0.87, 1.19); L2: 1.05 (0.93, 1.19); L3: 0.96 (0.84, 1.09); L4: 1.04 (0.90, 1.21); L2-4: 1.03 (0.88, 1.22) Age 2-3 L0: 0.87 (0.72, 1.06); L1: 0.95 (0.78, 1.15); L2: 0.99 (0.82, 1.17); L3: 1.03 (0.84, 1.25); L4: 0.89 (0.74, 1.09); L2-4: 0.94 (0.74, 1.19) Age 0-3 L0: 0.97 (0.87, 1.08); L1: 0.99 (0.89, 1.10); L2: 1.01 (0.92, 1.12); L3: 1.03 (0.93, 1.14); L4: 1.04 (0.94, 1.15); L2-4: 1.04 (0.92, 1.17) Two pollutant models (lag 2-4) 1-pollutant model: 1.21 (0.99, 1.48) 2-pollutant (adj for NO_2): 1.13 (0.88, 1.45) 2-pollutant (adj for NO_X): 1.16 (0.90, 1.48) 2-pollutant (adj for CO): 1.23 (0.96, 1.57) 110 children living within 5km radius from monitor (sensitivity analysis): Age 0-1: 1.32 (0.95, 1.82); Age 1-2: 1.20 (0.87, 1.67); Age 2-3: 0.78 (0.52, 1.16); Age 0-3: 1.11 (0.88, 1.39)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Boezen et al. (2005) **Period of Study:** Two consecutive winters (winter 1993-winter 1995) **Location:** rural (Meppel, Nunspeet) and urban (Amsterdam) areas in the Netherlands	**Outcome**: FEV_1, airway hyperresponsiveness (AHR), serum total IgE and daily data on lower respiratory symptoms (LRS), upper respiratory symptoms (URS), cough and morning and evening peak expiratory flow **Age Groups:** 50-70 years **Study Design:** Case-control study **N:** 327 patients **Statistical Analyses:** Logistic regression **Covariates:** daily minimum temperature, linear, quadratic and cubic time trend, weekend/holidays, and influenza incidence for the rural and urban areas and two winters separately **Season:** winter **Dose-response Investigated?** No **Lags Considered:** 0, 1, 2, and 5-day mean	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Winter 93/94 Urban: 41.5 Winter 93/94 Rural: 44.1 Winter 94/95 Urban: 31.1 Winter 94/95 Rural: 26.6 **Percentiles:** 50th(Median): Winter 93/94 Urban: 34.6 Winter 93/94 Rural: 30.4 Winter 94/95 Urban: 28.9 Winter 94/95 Rural: 23.7 **Range (Min, Max):** 93/94 Urban: (12.1-112.7) 93/94 Rural: (7.9-242.2) 94/95 Urban: (8.8-89.9) 94/95 Rural: (7.1-96.9) **Copollutant:** SO_2 NO_2 Black Smoke	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]: AHR-/IgE-** Upper Respiratory Symptoms Lag 0: OR = 0.99 (0.97-1.01); Lag 1: OR = 1.01 (0.99-1.03); Lag 2: OR = 1.00 (0.96-1.02); 5-day mean: OR = 1.00 (0.96-1.04) Cough Lag 0: OR = 1.00 (0.99-1.02); Lag 1: OR = 0.99 (0.98-1.01); Lag 2: OR = 1.00 (0.98-1.01); 5-day mean: OR = 0.98 (0.95-1.01) >10% fall in morning peak expiratory flow Lag 1: OR = 1.01 (0.98-1.04); Lag 2: OR = 0.97 (0.94-1.00); 5-day mean: OR = 0.97 (0.92-1.02) **AHR-/IgE+** Upper Respiratory Symptoms Lag 0: OR = 1.01 (0.99-1.03); Lag 1: OR = 1.02 (1.00-1.04); Lag 2: OR = 1.01 (0.99-1.03); 5-day mean: OR = 1.08 (1.04-1.11) Cough Lag 0: OR = 1.01 (0.99-1.03); Lag 1: OR = 0.99 (0.98-1.01); Lag 2: OR = 1.00 (0.98-1.02); 5-day mean: OR = 1.01 (0.97-1.05) >10% fall in morning peak expiratory flow Lag 1: OR = 0.99 (0.97-1.02); Lag 2: OR = 0.99 (0.97-1.02); 5-day mean: OR = 0.97 (0.93-1.01) **AHR+/IgE-** Upper Respiratory Symptoms Lag 0: OR = 0.99 (0.95-1.03); Lag 1: OR = 1.01 (0.97-1.05); Lag 2: OR = 0.99 (0.96-1.03); 5-day mean: OR = 0.98 (0.91-1.06) Cough Lag 0: OR = 1.00 (0.97-1.02); Lag 1: OR = 1.01 (0.98-1.03); Lag 2: OR = 0.99 (0.96-1.02); 5-day mean: OR = 1.02 (0.96-1.08) >10% fall in morning peak expiratory flow Lag 1: OR = 0.99 (0.95-1.03); Lag 2: OR = 0.99 (0.95-1.03); 5-day mean: OR = 0.99 (0.93-1.06) **AHR+/IgE+** Upper Respiratory Symptoms Lag 0: OR = 1.01 (0.98-1.04); Lag 1: OR = 1.03 (1.00-1.05); Lag 2: OR = 1.02 (0.99-1.05); 5-day mean: OR = 1.06 (1.00-1.11) Cough Lag 0: OR = 1.03 (1.01-1.06); Lag 1: OR = 1.00 (0.98-1.02); Lag 2: OR = 0.99 (0.97-1.01); 5-day mean: OR = 0.99 (0.95-1.04); Lag 2: OR = 0.99 (0.96-1.03); 5-day mean: OR = 0.99 (0.92-1.05) >10% fall in morning peak expiratory flow Lag 1: OR = 1.04 (1.00-1.07); Lag 2: OR = 1.03 (0.99-1.06); 5-day mean: OR = 1.05 (0.99-1.11)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Boezen et al. (1999) **Periods of Study:** 3 Winters (1992-1995) **Location:** Urban and rural areas of the Netherlands	**Outcome:** Respiratory symptoms Lower respiratory symptoms (wheeze, attacks of wheezing, shortness of breath) Upper respiratory symptoms (sore throat, runny or blocked nose) Bronchial hyperresponsiveness (BHR) **Study Design:** Time-series **Statistical Analyses:** Logistic regression (PROC model) **Age Groups**: 7-11	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Winter 1992-93 Urban: 54.8 Rural: 44.7 Winter 1993-94 Urban: 41.5 [3] Rural: 44.1 Winter 1994-95 Urban: 31.1 Rural: 26.6 **Range (Min, Max):** Winter 1992-93 Urban: (4.7, 145.6) Rural: (4.8, 103.8) Winter 1993-94 Urban: (12.1, 112.7) Rural: (7.9, 242.2) Winter 1994-95 Urban: (8.8, 89.9) Rural: (7.1, 96.9) **Copollutants:** BS SO_2 NO_2	**Increment:** 100 µg/m^3 **Odds Ratio (Lower CI, Upper CI); lag:** OR for respiratory symptoms and exposure to PM_{10} in children with BHR and high serum total IgE Lower Respiratory Symptoms 1.32 (1.07, 1.63); 0; 1.36 (1.13, 1.64); 1; 1.36 (1.13, 1.65); 2; 2.39 (1.71, 3.35); 0-5 avg. Upper Respiratory Symptoms 1.13 (0.97, 1.32); 0; 1.00 (0.87, 1.16); 1; 0.96 (0.84, 1.11); 2; 0.91 (0.70, 1.18); 0-5 avg >10% morning peak expiratory flow (PEF) decrease 1.10 (0.92, 1.33); 0; 1.08 (0.90, 1.28); 1; 1.03 (0.87, 1.23); 2; 1.10 (0.83, 1.46); 0-5 avg >10% evening peak expiratory flow (PEF) increase 1.37 (1.16, 1.63); 0; 1.09 (0.92, 1.29); 1; 1.16 (0.98. 1.36); 2; 1.35 (1.04, 1.77); 0-5 avg. OR for respiratory symptoms and exposure to PM_{10} in children without BHR and low serum total IgE Lower Respiratory Symptoms 1.08 (0.75, 1.57); 0; 1.04 (0.70, 1.53); 1; 0.98 (0.69, 1.39); 2; 1.15 (0.61, 2.15); 0-5 avg. Upper Respiratory Symptoms 1.12 (0.99, 1.28); 0; 1.01 (0.89, 1.15); 1; 1.01 (0.89, 1.15); 2; 0.93 (0.67, 1.28); 0-5 avg >10% morning PEF decrease 1.07 (0.93, 1.23); 0; 0.86 (0.75, 0.99); 1; 0.97 (0.85, 1.11); 2; 0.99 (0.79, 1.23); 0-5 avg >10% evening PEF decrease 1.13 (0.98, 1.30); 0; 1.05 (0.91, 1.21); 1; 0.99 (0.87, 1.14); 2; 0.94 (0.75, 1.17); 0-5 avg OR for respiratory symptoms and exposure to PM_{10} in children with BHR and low serum total IgE Lower Respiratory Symptoms 0.77 (0.48, 1.24); 0; 1.34 (0.94, 1.93); 1; 1.24 (0.86, 1.81); 2; 1.92 (0.84, 4.41); 0-5 avg Upper Respiratory Symptoms 1.13 (0.92, 1.40); 0; 0.98 (0.79, 1.22); 1; 0.97 (0.79, 1.20); 2; 0.83 (0.54, 1.25); 0-5 avg >10% morning PEF decrease 1.04 (0.78, 1.38); 0; 0.86 (0.66, 1.12); 1; 0.91 (0.71, 1.17); 2; 0.78 (0.51, 1.20); 0-5 avg >10% evening PEF decrease 1.07 (0.82, 1.41); 0; 0.98 (0.76, 1.26); 1; 0.93 (0.73, 1.19); 2; 0.83 (0.55, 1.26); 0-5 avg OR for respiratory symptoms and exposure to PM_{10} in children without BHR and high serum total IgE Lower Respiratory Symptoms 1.04 (0.80, 1.35); 0; 1.21 (0.98, 1.51); 1; 1.18 (0.96, 1.45); 2; 1.35 (0.89, 2.04); 0-5 avg Upper Respiratory Symptoms 1.01 (0.85, 1.20); 0; 0.95 (0.81, 1.12); 1; 0.93 (0.80, 1.09); 2; 0.93 (0.69, 1.25); 0-5 avg >10% morning PEF decrease 0.97 (0.80, 1.17); 0; 1.09 (0.91, 1.30); 1; 1.02 (0.85, 1.21); 2; 0.95 (0.71, 1.28); 0-5 avg >10% evening PEF decrease 1.02 (0.85, 1.22); 0; 1.06 (0.90, 1.25); 1; 1.08 (0.93, 1.27); 2; 1.04 (0.80, 1.34); 0-5 avg.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chattopadhyay et al. (2007) **Period of Study:** NR **Location:** Three different points in Kolkata, India: North, South, and Central	**Outcome:** pulmonary function tests (respiratory impairments) **Age Groups:** All ages **Study Design:** Cross-sectional **N:** 505 people studied for PFT; total population of Kolkata not given **Statistical Analyses:** Frequencies **Covariates:** Meteorologic data (i.e. temperature, wind direction, wind speed, and humidity) **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 8 h **Mean (SD):** North Kolkata: 535.9 Central Kolkata: 1114.5 South Kolkata: 909.2 **Monitoring Stations:** 1 **Copollutant:** PM<10-3.3 PM<3.3-0.4	**PM Increment:** NR **Respiratory impairments (SD):** North Kolkata Male (n = 137); Restrictive: 4 (2.92); Obstructive: 5 (3.64); Combined Res. And Obs.: 6 (4.37); Total: 15 (10.95); Female (n = 152); Restrictive: 3 (1.97); Obstructive: 5 (3.28); Combined Res. And Obs.: 0; Total: 8 (5.26); Total (n = 289); Restrictive: 7 (2.42); Obstructive: 10 (3.46); Combined Res. And Obs.: 6 (2.07); Total: 23 (7.96) Central Kolkata Male (n = 44); Restrictive: 6 (13.63); Obstructive: 1 (2.27); Combined Res. And Obs.: 1 (2.27); Total: 8 (18.18); Female (n = 50); Restrictive: 3 (6.00); Obstructive: 2 (4.00); Combined Res. And Obs.: 0 Total: 5 (10.00); Total (n = 94); Restrictive: 9 (9.57); Obstructive: 3 (3.19); Combined Res. And Obs.: 1 (1.06); Total: 13 (13.82) South Kolkata Male (n = 52); Restrictive: 1 (1.92); Obstructive: 2 (3.84); Combined Res. And Obs.: 3 (5.76); Total: 6 (11.53); Female (n = 70); Restrictive: 2 (2.85); Obstructive: 1 (1.42); Combined Res. And Obs.: 0; Total: 3 (4.28); Total (n = 122); Restrictive: 3 (2.45); Obstructive: 3 (2.45); Combined Res. And Obs.: 3 (2.45); Total: 9 (7.37)
Reference: Dales et al. (2006) **Period of Study:** 1/1/1986-12/31/2000 **Location:** 11 Canadian Cities: Calgary, Edmonton, Halifax, London, Hamilton, Ottawa, St. John, Toronto, Vancouver, Windsor, Winnipeg	**Health Outcome:** Respiratory Illness: Asphyxia (799); Respiratory failure (799.1); Dyspnea and respiratory abnormalities (786); Respiratory distress syndrome (769); Unspecified birth asphyxia in live-born infant (768.9); Other respiratory problems after birth (770.8); Pneumonia (486) **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Copollutants (correlation):** O_3: r = -0.29 to 0.41 NO_2: r = -0.26 to 0.69 SO_2: r = -0.09 to 0.61 CO: r = -0.13 to 0.71	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); Lag** In respiratory illness and exposure to PM_{10} in people of all ages PM_{10} alone: 2.13 (-0.50, 4.76) Multipollutant model PM_{10}: 1.45 (-1.90, 4.80) PM_{10}, O_3: 2.67 (0.98, 4.39) PM_{10}, NO_2: 2.48 (1.18, 3.80) PM_{10}, SO_2: 1.41 (0.35, 2.47) PM_{10}, CO: 1.30 (0.13, 2.49)
Reference: de Hartog et al. (2003) **Period of Study:** winter of 1998-1999 (in Amsterdam, from November 2, 1998 to June 18, 1999; in Erfurt, from October 12, 1998 to April 4, 1999; and in Helsinki, from November 2, 1998 to April 30, 1999.) **Location:** Amsterdam, the Netherlands; Erfurt, Germany; and Helsinki, Finland	**Outcome:** chest pain, chest pain at physical exertion, shortness of breath, feeling tired or weak, tripping or racing heart, cold hands or feet, cough, phlegm, being awakened by breathing problems, wheezing, and common cold or flu and fever **Age Groups:** ≥ 50 yrs **Study Design:** cohort **N:** 131 subjects with history of coronary heart disease **Statistical Analyses:** Logistic regression **Covariates:** ambient temperature, relative humidity, atmospheric pressure, incidence of influenza-like illness **Season:** Winter **Dose-response Investigated?** No **Statistical Package:** S-PLUS 2000 **Lags Considered:** 0, 1, 2, 3, and 5-day avg	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Amsterdam, the Netherlands: 36.5 Erfurt, Germany: 27.1 Helsinki, Finland: 19.6 **Range (Min, Max):** Amsterdam, the Netherlands: (13.6-112.0) Erfurt, Germany: (5.2-104.2) Helsinki, Finland: (6.4-67.4) **Monitoring Stations:** 1 **Copollutant:** $PM_{2.5}$; $NC_{0.01-0.1}$; CO; NO_2; SO_2	'There was a tendency toward positive associations between avoidance of activities and both particulate air pollution (PM_{10}) and gases, but none of the associations were statistically significant….In both incidence analyses and prevalence analyses, odds ratios for PM_{10} were generally similar to the corresponding odds ratios for $PM_{2.5}$, but were somewhat less significant.'
Reference: Delfino et al. (1998) **Period of Study:** August 1–October 30, 1995 **Location:** Alpine, CA	**Outcome:** asthma symptom severity **Age Groups:** 9-17 **Study Design:** Panel Study **N:** 24 non-smoking pediatric asthmatics **Statistical Analyses:** GEE **Covariates:** day of week, temperature, humidity, wind speed **Statistical Package:** SAS **Lags Considered:** 0-5, 0, 0-4	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 31 (8) 90th: 42 **Range (Min, Max):** 16, 54 **Copollutant (correlation):** O_3 (r = 0.32)	**PM Increment:** 42 µg/m^3 (90th percentile increase) Asthma symptoms: Everyone: 1.47 (0.90, 2.39) lag 0 Everyone: 1.73 (1.03, 2.89) lag 0-4 Less symptomatic: 2.47 (1.23-4.95) lag 0 Less symptomatic: 4.03 (1.22, 13.33) lag 0-4 More symptomatic: 1.50 (0.80, 2.80) lag 0 More symptomatic: 1.95 (1.12, 3.43) lag 0-4 PM_{10} + O_3 Asthma symptoms: 1.31 (0.84, 2.06) lag 0 1.65 (1.03, 2.66) lag 0-4 Less symptomatic: 2.08 (1.12-3.83) lag 0 Less symptomatic: 3.35 (1.06, 10.51) lag 0-4 More symptomatic: 1.40 (0.77, 2.53) lag 0 More symptomatic: 1.87 (1.11, 3.13) lag 0-4

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Delfino et al. (2002) **Period of Study:** March 1 through April 30, 1996 **Location:** Alpine, California (a semi-rural area)	**Outcome:** Asthma symptoms that interfere with daily activities **Age Groups:** 9-19 yrs **Study Design:** Daily panel study **N:** 22 asthmatic children **Statistical Analyses:** GEE **Covariates:** temperature, relative humidity, day-of-week trends, linear time trend across the 61 days, and upper or lower respiratory infection **Season:** "early spring season" of March through April **Dose-response Investigated?** Yes **Statistical Package:** SAS, version 8 **Lags Considered:** 0, 1, 2, 3, 4, 5, 3-day mov avg	**Pollutant:** PM_{10} **Averaging Time:** 1 h max **Mean (SD):** 38(15) **Percentiles:** 90th: 63 **Range (Min, Max):** (12-69) **Averaging Time:** 8 h max **Mean (SD):** 28(12) **Percentiles:** 90th: 46 **Range (Min, Max):** (8-57) **Averaging Time:** 24 h **Mean (SD):** 20(9) **Percentiles:** 90th: 32 **Range (Min, Max):** (7-42) **Copollutant (correlation):** 1 h max PM_{10}: 8 h max PM_{10}: r = 0.93 24 h PM_{10}: r = 0.84 1 h max O_3: r = 0.68 8 h max O_3: r = 0.95 1 h max NO_2: r = 0.49 8 h max NO_2: r = 0.55 8 h max PM_{10}: 1 h max PM_{10}: r = 0.93 24 h PM_{10}: r = 0.95 1 h max O_3: r = 0.72 8 h max O_3: r = 0.65 1 h max NO_2: r = 0.48 8 h max NO_2: r = 0.55 24 h PM_{10}: 1 h max PM_{10}: r = 0.84 8 h max PM_{10}: r = 0.95 1 h max O_3: r = 0.74 8 h max O_3: r = 0.71 1 h max NO_2: r = 0.37 8 h max NO_2: r = 0.44	**PM Increment:** 90th percentile increase **Effect Estimate [Lower CI, Upper CI]:** ORs for risk of asthma symptoms in those who report a respiratory infection compared to those who do not have a respiratory infection 1 h max PM_{10} lag 0: 4.88 (1.31-18.2) 8 h max PM_{10} lag 0: 6.78 (1.38-33.3) 24 h mean PM_{10} lag 0: 4.68 (0.71-30.7) 3-day mov avg 1 h max PM_{10}: 11.1 (1.10-112) 3-day mov avg 8 h max PM_{10}: 10.1 (1.42-72.0) 3-day mov avg 24 h PM_{10}: 2.67 (0.60-11.8) Effect modification by anti-inflammatory medication use on the relationship of asthma symptoms in children 1 h max PM_{10} lag 0: 1.41 (0.87-2.30) On medication: 0.96 (0.25-3.69) Not on medication: 1.92 (1.22-3.02) 8 h max PM_{10} lag 0: 1.19 (0.74-1.94) On medication: 0.75 (0.18-3.04) Not on medication: 1.68 (0.91-3.09) 24 h mean PM_{10} lag 0: 1.08 (0.73-1.61) On medication: 0.80 (0.24-2.69) Not on medication: 1.35 (0.82-2.22) 3-day mov avg 1 h max PM_{10}: 1.45 (0.76-2.76) On medication: 1.01 (0.14-7.02) Not on medication: 1.92 (0.99-3.71) 3-day mov avg 8 h max PM_{10}: 1.32 (0.76-2.29) On medication: 0.82 (0.17-3.94) Not on medication: 1.89 (1.10-3.24) 3-day mov avg 24 h PM_{10}: 1.22 (0.84-1.77) On medication: 0.75 (0.26-2.14) Not on medication: 1.75 (1.15-2.68) Dose-response results are found in Figure 2 and not quantitatively reported elsewhere.
Reference: Delfino et al. (2003) **Period of Study:** November 1999 to January 2000 **Location:** Huntington Park, Los Angeles	**Outcome:** Asthma severity scale; Peak Expiratory Flow Rate (PEF) **Age Groups:** Ages 10 to 15 **Study Design:** Longitudinal study **N:** 22 children **Statistical Analyses:** Regression analysis (GEE, GLM); multivariate regression models **Covariates:** Day of the week, Maximum Temperature, Respiratory Infections **Season:** Winter **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0, 1	**Pollutant:** PM_{10} **Mean (SD):** 59.9 (24.7) **Range (Min, Max):** 20-126 IQR: 37 90th: 86.0 **Monitoring Stations:** 1 **Copollutant (correlation):** 8-h max NO_2 = 0.38 8-h max O_3 = -0.16 8-h max CO = 0.50 8-h max SO_2 = 0.73	**PM Increment:** IQR 37.0 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI]; lag: Lag 0 Symptom Scores >1: 1.45 (1.11, 1.90) Symptom Scores >2: NR Lag 1 Symptom Scores >1: 1.07 (0.64, 1.77) Symptom Scores >2: NR

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Delfino et al. (2004) **Period of Study:** September–October 1999; April–June 2000 **Location:** Alpine, California	**Outcome:** FEV_1 **Age Groups**: 9-19 years old **Study Design:** Panel study **N:** 24 children **Statistical Analyses:** GLM; Akaike's information criterion and Bayesian information criterion **Covariates:** Day of week, Personal temperature and relative humidity, time of FEV_1 maneuver (morning, afternoon, or evening), Season (fall 1999 or spring 2000) As-needed medication use Presence or absence of upper or lower respiratory infections **Season:** Spring, Fall **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** Lag 0-4	**Pollutant:** PM_{10} **Averaging Time:** 4 h, 8 h, 12 h, 24-h **Personal Monitor** 1-h max personal PM last 24-h **Mean (SD):** 151.0 (12.03) 90th: 292.4 **Range (Min, Max):** (9.1, 996.8) Mean personal PM last 24-h **Mean (SD):** 37.9 (19.9) 90th: 65.1 **Range (Min, Max):** (3.9, 113.8) Central outdoor stationary-site PM 1-h Maximum TEOM PM_{10} last 24-h **Mean (SD):** 54.4 (13.8) 90th: 71.0 **Range (Min, Max):** (24.4, 95.4) Mean TEOM PM_{10} last 24-h **Mean (SD):** 29.7 (8.6) 90th: 40.9 **Range (Min, Max):** (12.9, 50.7) 24-h mean PM_{10} **Mean (SD):** 23.6 (9.1) 90th: 34.6 **Range (Min, Max):** (3.2, 48.0) **Copollutant (correlation):** 8-h max personal PM 8-h max O_3 = 0.03 8-h Max NO_2 = 0.26 24-h Mean Personal PM = 0.94 8-h Max TEOM PM_{10} = 0.38 24-h Mean TEOM PM_{10} = 0.40 24-h Central HI PM_{10} = 0.37 24-h Central HI $PM_{2.5}$ = 0.38 24-h Outdoor HI PM_{10} = 0.32 24-h Outdoor HI $PM_{2.5}$ = 0.39 24-h Indoor HI PM_{10} = 0.23 24-h Indoor HI $PM_{2.5}$ = 0.37 24-h mean personal PM 8-h max O_3 = 0.01 8-h Max NO_2 = 0.27 8-h Max Personal PM = 0.94 8-h Max TEOM PM_{10} = 0.36 24-h Mean TEOM PM_{10} = 0.39 24-h Central HI PM_{10} = 0.36 24-h Central HI $PM_{2.5}$ = 0.43 24-h Outdoor HI PM_{10} = 0.34 24-h Outdoor HI $PM_{2.5}$ = 0.44 24-h Indoor HI PM_{10} = 0.29 24-h Indoor HI $PM_{2.5}$ = 0.46 24-h Mean TEOM PM_{10} 8-h max O_3 = 0.41 8-h Max NO_2 = 0.58 8-h Max Personal PM = 0.40 24-h Mean Personal PM = 0.39 8-h Max TEOM PM_{10} = 0.92 24-h Central HI PM_{10} = 0.86 24-h Central HI $PM_{2.5}$ = 0.78 24-h Outdoor HI PM_{10} = 0.79 24-h Outdoor HI $PM_{2.5}$ = 0.78 24-h Indoor HI PM_{10} = 0.36 24-h Indoor HI $PM_{2.5}$ = 0.59	Results presented graphically: Percent predicted FEV_1 was inversely associated with personal exposure to fine particles. - Inverse associations of FEV_1 with stationary-site indoor, outdoor and central-site gravimetric $PM_{2.5}$ and PM_{10}, and with hourly TEOM PM_{10}

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Delfino et al. (2006) **Period of Study:** Region 1: August to Mid December 2003. Region 2: July through November 2004 **Location:** Region 1: Riverside, CA. Region 2: Whittier, CA	**Outcome:** Fractional Concentration of Nitric Oxide in exhaled air (FENO) **Age Groups:** 9 through 18 **Study Design:** Longitudinal Panel Study **N:** 45 children **Statistical Analyses:** Linear mixed-effects models; Two-stage hierarchical model; Empirical Variograms; Fourth-order polynomial distributed lag mixed-effects model **Covariates:** Personal temperature, Personal Rel. Humid., 10-day exposure run, Respiratory infections, Region of study, Sex, Cumulative daily use of as-needed B-agonist inhalers **Dose-response Investigated?** No **Lags Considered:** Lag 0, Lag 1, 2-day moving avg	**Pollutant:** PM_{10} Central Site **Averaging Time:** 24- h Riverside **Mean (SD):** 70.82 (29.36) 50th(Median): 65.96 **Range (Min, Max):** (30.75, 154.05) µg/m^3 Whittier **Mean (SD):** 35.73 (16.6) 50th(Median): 34.65 **Range (Min, Max):** (5.86, 105.46) µg/m^3 **Monitoring Stations:** 48 personal nephelometers, 2 central sites	**PM Increment:** IQR increase (Riverside: 28.41 µg/m^3, Whittier 21.87 µg/m^3) **Coefficient [Lower CI, Upper CI]; lag:** Lag = 2-day moving avg Stratified by Medication Use Not Taking Anti-Inflamm. Medication Central 0.76 (-1.54) Taking Anti-Inflamm. Medication Central 0.53 (-0.83, 1.90) Inhaled Corticosteroids Central 1.28 (-0.01, 2.58) Antileukotrienes +- inhaled corticosteroids Central -2.10 (-5.33, 1.12) **Notes:** Figure of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Figure of the Estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Figure of One- and two-pollutant models for change in FENO using 2-day Moving Averages personal and central-site pollutant measurements.
Reference: Desqueyroux et al. (2002) **Period of Study:** Nov 1995-Nov 1996 **Location:** Paris, France	**Outcome:** Asthma attacks **Age Groups:** Adults. **Study Design:** Panel study **N:** 60 moderate to severe adult asthmatics **Statistical Analyses:** Marginal logistic regression **Covariates:** FEV_1, smoking, allergy, oral steroid treatment, mean daily temperature, relative humidity, pollen counts, season, holiday period **Season:** winter, summer **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 1, 2, 3, 4, 5, 3-5	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Summer: 23 (9) Winter: 28 (14) **Range (Min, Max):** Summer: 6, 63 Winter: 9, 84 **Monitoring Stations**: 7 **Copollutant:** SO_2 , NO_2, O_3	**PM Increment:** 10 µg/m^3 **OR Estimate [Lower CI, Upper CI]; lag:** 0.87 [0.71, 1.06] lag 1; 0.93 [0.80, 1.08] lag 2; 1.11 [0.98, 1.26] lag 3; 1.17 [1.03, 1.33] lag 4; 1.16 [1.01, 1.34] lag 5; 1.21 [1.01, 1.34] lag 3-5 vs seasons alone: Winter: 1.41 [1.16, 1.71] lag 3-5 summer: 1.03 [0.72, 1.47] lag 3-5 vs link to explanatory factors: No link: [1.71 [1.20, 2.43] lag 3-5 Link: 1.27 [1.06, 1.52] lag 3-5 vs occurrence of infection: Without infection: 1.52 [1.16, 2.00] lag 3-5 With infection: 1.30 [1.03, 1.65] lag 3-5 vs baseline pulmonary function: FEV_1 >/ = 68% predicted: 1.38 [1.06, 1.79] lag 3-5 FEV <68% predicted: 1.45 [1.11, 1.90] lag 3-5 vs smoking habits: Nonsmokers: 1.53 [1.18, 1.98] lag 3-5 Current & ex-smokers: 1.18 [0.90, 1.54] lag 3-5 vs allergy: Non-allergic: 1.29 [0.94, 1.77] lag 3-5 Allergic: 1.49 [1.17, 1.90] lag 3-5 vs regular oral steroid treatment: No: 1.41 [1.15, 1.73] lag 3-5 Yes: 1.41 [0.88, 2.25] lag 3-5 Multipollutant model: PM_{10} + NO_2: 1.43 [1.16, 1.76] Lag 3-5 PM_{10} + SO_2: 1.51 [1.20, 1.90] Lag 3-5 PM_{10} + O_3: 1.09 [0.71, 1.67] Lag 3-5
Reference: Diette et al. (2007) **Period of Study:** 9/2001-12/2003 **Location:** East Baltimore, MD	**Outcome:** Asthma in the last 12 months (493.x) **Age Groups:** 2 to 6 years old **Study Design**: Prospective cohort **N:** 150 with asthma; 150 without asthma **Statistical Analyses:** Student's two-tailed t-test; Kruskal-Wallis test; Pearson's chi square; Fisher's exact test **Covariates:** Season of collection **Dose-response Investigated?** No **Statistical Package:** STATASE 8.0	**Pollutant:** PM_{10} **Averaging Time:** 72 50th(Median): 43.7 IQR: (29-70)	**Notes:** "Pollutant concentrations in the homes of asthmatic and control children who lived in the same home for their whole life were not different compared with those who had moved at least once."

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ebelt et al. (2005) **Period of Study:** summer of 1998 **Location:** Vancouver, Canada	**Outcome**: Adverse health effects: spirometry, systolic/diastolic blood pressure measurements, symptom questionnaires, arrhythmia, heart rate, and heart rate variability (from electrocardiogram) **Age Groups:** range from 54-86 yrs; mean age = 74 years **Study Design:** extended analysis of a repeated-measures panel study **N:** 16 persons with COPD **Statistical Analyses:** Earlier analysis expanded by developing mixed-effect regression models and by evaluating additional exposure indicators **Dose-response Investigated?** No **Statistical Package:** SAS V8	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Ambient PM_{10}: 17 (6); Exposure to ambient PM_{10}: 10.3 (4.6) **Range (Min, Max):** Ambient PM_{10}: (7-36); Exposure to ambient PM_{10}: (1.5-23.8) **Monitoring Stations**: 5 **Copollutant (correlation):** Ambient $PM_{10-2.5}$: r = 0.69 Ambient $PM_{2.5}$: r = 0.78 Exposure to Ambient PM_{10}: r = 0.71	**PM Increment:** Ambient PM_{10}: 7 (IQR) Exposure to ambient PM_{10}: 6.5 (IQR) **Notes:** Effect estimates are presented in Figure 2 and Electronic Appendix Table 1 (only available with electronic version of article) and not provided quantitatively elsewhere.
Reference: Fischer et al. (2007) **Period of Study:** 7 weeks (dates not specified) **Location:** Netherlands	**Outcome:** Respiratory Symptoms, Sore throat, Runny nose, Cold, Sick at home **Study Design:** Prospective cohort **Statistical Analyses:** Linear regression model (PROC mixed) **Age Groups:** 10-11	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 56 µg/m³ IQ (25th, 75th): (21, 187) **Copollutants:** BS NO_2 CO NO	**Increment:** 10 µg/m³ % Increase in eNO and PM_{10} and change in spirometric lung function; lag eNO and PM_{10} only 6.5 (0.9, 12.4); 1; 7.8 (-11.3, 31.0); 2 FVC 0.4 (-0.1, 0.9); 1; 0.6 (-1.0, 2.2); 2 FEV_1 -0.3 (-0.8, 0.2); 1; -2.1 (-4.0 to -0.2); 2 PEF -2.8 (-6.1, 0.2); 1; 7.1 (-4.9, 19.1); 2 MMEF -0.5 (-2.2, 1.2); 1; -2.5 (-8.4, 3.9); 2
Reference: Forsberg et al. (1998) **Period of Study:** 1/3/1994–3/27/1994 **Location:** Urban and rural areas of Umea, Sweden	**Outcome:** Respiratory Symptoms, Shortness of breath; Wheeze, Asthma attacks, Recent asthma, Dry cough, Doctor-diagnosed asthma, Recently treated for asthma, Early chest illness **Study Design:** Cross-sectional **Statistical Analyses:** Logistic linear regression **Age Groups:** 6-12	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Urban: 13.4 Rural: 11.5 **Range (Min, Max):** Urban: (0, 40.5) Rural: (1.6, 29.0) **Copollutants (correlation):** BS: r = 0.75 SO_2: r = 0.75 NO_2: r = 0.89	**Increment:** 10 µg/m³ OR between prevalence of acute respiratory symptoms and PM_{10} exposure for urban and rural children; lag **Urban children – Cough**: 1.031 (0.957, 1.112); 0; 0.997 (0.923, 1.077); 1; 1.018 (0.940, 1.103): 2; 1.094 (0.895, 1.338); 0-6 avg **Phlegm:** 0.998 (0.899, 1.108); 0; 1.035 (0.928, 1.154); 1; 1.121 (1.013, 1.240); 2; 1.043 (0.822, 1.324); 0-6 avg **Upper respiratory symptoms:** 1.004 (0.949, 1.063); 0; 0.975 (0.922, 1.031); 1; 0.951 (0.895, 1.010); 2; 0.849 (0.687, 1.050); 0-6 avg **Lower respiratory symptoms:** 0.984 (0.872, 1.110); 0; 0.919 (0.812, 1.039); 1; 0.894 (0.771, 1.036); 2 ; 0.800 (0.617, 1.038); 0-6 avg **Rural children- Cough**: 0.997 (0.900, 1.105); 0; 1.003 (0.906, 1.112); 1; 0.997 (0.891, 1.116); 2; 0.855 (0.655, 1.115); 0-6 avg **Phlegm:** 1.024 (0.880, 1.192); 0; 0.995 (0.853, 1.160); 1; 1.117 (0.956, 1.305); 2; 1.041 (0.742, 1.459); 0-6 avg **Upper respiratory symptoms:** 1.093 (0.989, 1.208); 0; 1.018 (0.918, 1.130); 1; 1.075 (0.962, 1.201); 2; 1.052 (0.786, 1.407); 0-6 avg **Lower respiratory symptoms:** 1.022 (0.855, 1.180); 0; 0.998 (0.855, 1.164); 1; 1.000 (0.830, 1.206); 2; 0.939 (0.703, 1.253); 0-6 avg OR between incidence of acute respiratory symptoms and PM_{10} exposure in urban and rural children; lag **Urban Children- Cough**: 1.114 (0.886, 1.401); 0; 0.891 (0.703, 1.130); 1; 0.766 (0.577, 1.017); 2; 0.817 (0.523, 1.276); 0-6 avg **Phlegm:** 0.954 (0.664, 1.371); 0; 1.056 (0.744, 1.501); 1; 1.416 (0.969, 2.069); 2; 0.808 (0.357, 1.827); 0-6 avg **Upper respiratory symptoms:** 1.155 (0.965, 1.383); 0; 0.788 (0.629, 0.986); 1; 0.886 (0.728, 1.077); 2; 0.770 (0.549, 1.081); 0-6 avg **Lower respiratory symptoms:** 1.060 (0.828, 1.356); 0; 0.763 (0.584, 0.996); 1; 0.652 (0.493, 0.863); 2; 0.519 (0.306, 0.882); 0-6 avg **Rural Children – Cough**: 1.052 (0.767, 1.444); 0;

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			0.753 (0.547, 1.038); 1; 0.840 (0.571, 1.235); 2; 0.800 (0.409, 1.565); 0-6 avg **Phlegm:** 1.051 (0.731, 1.509); 0; 1.010 (0.693, 1.472); 1; 0.998 (0.652, 1.528); 2; 0.797 (0.344, 1.847); 0-6 avg **Upper respiratory symptoms:** 1.044 (0.813, 1.341); 0; 0.810 (0.612, 1.072); 1; 0.800 (0.611, 1.048); 2; 0.714 (0.417, 1.220); 0-6 avg **Lower respiratory symptoms:** 1.079 (0.756, 1.539); 0; 0.888 (0.615, 1.281); 1; 0.715 (0.472, 1.083); 2; 0.822 (0.395, 1.711); 0-6 avg OR between prevalence of medication use and PM_{10} exposure in urban and rural children; lag **Bronchodilator use - Urban children:** 0.998 (0.951, 1.048); 0; 0.999 (0.952, 1.049); 1; 1.006 (0.953, 1.062); 2; 0.919 (0.775, 1.090); 0-6 avg **Rural children:** 0.970 (0.904, 1.040); 0; 0.959 (0.893, 1.030); 1; 1.008 (0.927, 1.095); 2; 1.087 (0.914, 1.292); 0-6 avg OR between incidence of medication use and PM_{10} exposure in urban and rural children; lag **Bronchodilator use - Urban children:** 1.498 (0.899, 2.498); 0; 1.049 (0.565, 1.947); 1; 1.148 (0.674, 1.954); 2; 1.787 (0.611, 5.227); 0-6 avg **Rural children:** 1.275 (0.702, 2.315); 0; 0.924 (0.437, 1.956); 1; 1.005 (0.522, 1.936); 2; 1.823 (0.534, 6.277); 0-6 avg
Reference: Goncalves et al. (2005) **Period of Study:** Dec-Mar 1992/93. Dec-Mar 1993/94 **Location:** Sao Paulo	**Outcome:** Respiratory morbidity/admissions **Age Groups:** Children <13 yrs **Study Design:** Time series **Statistical Analyses:** Principal component analysis **Covariates:** Daily mean temperature, daily mean water vapor density, solar radiation **Season:** summer **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** Lag 3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Copollutant:** SO_2 , O_3	**PCA coefficients:** PC1, PC2, PC3: **Summer 1992/1993**: PM_{10}: 0.69, 0.45, 0.13 Solar Radiation: -0.04, 0.94 to -0.12 Mean Temperature: 0.62, 0.44 to -0.47 Mean Water Vapor Density: 0.73 to -0.46 to -0.26 SO_2: 0.78 to -0.03, 0.33 O_3: 0.18, 0.63, 0.37 Respiratory Mortality: 0.05 to -0.02, 0.81 Variations explained by Principal Component: PC1: 0.29; PC2: 0.27; PC3: 0.17 **Summer 1993/1994:** PM_{10}: 0.38, 0.80 to -0.23 Solar Radiation: 0.02, 0.09 to -0.97 Mean Temperature: 0.71, 0.40 to -0.37 Mean Water Vapor Density: 0.88, 0.25, 0.09 SO_2: 0.01, 0.92, 0.00 O_3: 0.47 to -0.06 to -0.35 Respiratory Mortality: -0.73, 0.11, 0.08 Variations explained by Principal Component: PC1: 0.31; PC2: 0.25; PC3: 0.18 **Notes:** Association between respiratory morbidity and air pollution more likely during summer with smaller contrasts in synoptic weather condition (summer 1992/93) but respiratory morbidity more related to weather variables during summer with larger contrasts (summer 1993/94).
Reference: Gordian and Choudhury (2003) **Period of Study:** 1994-Dec 1996 **Location:** Anchorage, Alaska	**Outcome:** Asthma medication among school children **Age Groups:** Elementary school children (kindergarten-6th grade) **Study Design:** Time series **Statistical Analyses:** Time series regression model **Covariates:** Day of the week, month, time trend, temperature **Season:** All seasons **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 1, 2, 7, 14, 21, 28	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 36.11 (30.46) **Range (Min, Max):** 2.96, 210.0 **Monitoring Stations:** 1	**Model regression slope coefficient for** PM_{10} **(estimated SE); lag:** 7.25 (2.88); lag 21 RR: 1.075 (1.016, 1.138) **Notes:** PM_{10} coefficients for other lags were also statistically significant but not reported.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Harre et al. (1997) **Period of Study:** 6/1994–8/1994 **Location:** Christchurch, New Zealand	**Outcome:** Respiratory symptoms, Cough, Wheeze, Chest tightness, Shortness of breath, Change in sputum volume, Nose, throat, or eye irritation, PEFR **Study Design**: Prospective cohort **Statistical Analyses:** Poisson, log linear regression **Age Groups:** >55	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Copollutants:** CO SO_2 NO_2	**Increment:** 35.04 µg/m^3 Relative Risk (Lower CI, Upper CI); lag: Chest symptoms: 1.38 (1.07, 1.78); 1 Wheeze: 0.97 (0.75, 1.26); 1 Nebuliser Use: 0.71 (0.42, 1.18); 1 Inhaler Use: 0.94 (0.78, 1.13); 1
Reference: Hastings and Jardine (2002) **Period of Study:** 1997-1998 **Location:** Bosnia (US military camps)	**Outcome:** Weekly rates of upper respiratory disease (URD), reported by the medical treatment facility in each military camp **Age Groups:** US soldiers **Study Design:** Ecologic (at level of military camp) **N:** 5 camps **Statistical Analyses:** 1.Pearson correlations between weekly URD rates and weekly PM_{10} (avg and max); 2.Kruskal Wallace test to compare URD rates in the 4 exposure quartiles; 3. Mann Whitney test to compare dichotomized exposure groups (above and below 50th percentile) **Dose-response Investigated?** Yes **Lags Considered:** Weekly rates of URD disease were related to avg weekly PM levels in the same week	**Pollutant:** PM_{10} **Mean (SD):** PM_{10} avg: 75.5 PM_{10} max: 92.9 Percentiles: PM_{10} max: 25th: 58.57 50th: 74.55 75th: 107.56 PM_{10} avg: 25th: 42.19 50th: 64.17 75th: 81.75 **Range (Min, Max):** PM_{10} avg: 25.0, 338.7 PM_{10} max: 25.0, 338.7 **Monitoring Stations**: at least one in each of the 5 camps	**PM max Quartiles (combining all camps):** Q1: <58.7 µg/m^3; Q2: 60.1 to <75.54 µg/m^3; Q3: 78.56 to <107.56 µg/m^3; Q4: >107.56 µg/m^3 For dichotomous analysis cutoff = 74.55 µg/m^3 PM avg Quartiles (combining all camps): Q1: <42.19 µg/m^3; Q2: 42.19 to 64.17 µg/m^3; Q3: 64.17 to 81.75 µg/m^3; Q4: >81.75 µg/m^3 For dichotomous analysis cutoff = 64.17 µg/m^3 Pearson correlation coefficients between URD rate and PM category [p-value]: PM_{10} max: quartiles of PM*URD rates; All camps 0.203 [0.041]; Blue Factory camp 0.277 [0.095]; Comanche 0.165 [0.237]; Demi 0.639 [0.123]; McGovern 0.535 [0.177]; Tuzla Main 0.107 [0.327] PM_{10} max: dichotomous PM*URD rates: All camps 0.283 [0.007]; Blue Factory camp 0.038 [0.430]; Comanche 0.282 [0.107]; Demi 0.927 [0.012]; McGovern 0.853 [0.033]; Tuzla Main 0.155 [0.258] PM_{10} avg: quartiles of PM*URD rates: All camps 0.149 [0.101]; Blue Factory camp 0.301 [0.077]; Comanche 0.246 [0.141]; Demi 0.437 [0.231]; McGovern 0.853 [0.033]; Tuzla Main 0.182 [0.222] PM_{10} avg: dichotomous PM*URD rates: All camps 0.060 [0.305]; Blue Factory camp -0.075 [0.365]; Comanche 0.143 [0.268]; Demi N/A*; McGovern N/A*; Tuzla Main 0.123 [0.303] Kruskal Wallace p-value comparing URD rates across exposure quartiles: PM_{10} max All camps 0.047; Blue Factory camp 0.321; Comanche 0.556; Demi 0.165; McGovern 0.202; Tuzla Main 0.554 PM_{10} avg All camps 0.672; Blue Factory camp 0.809; Comanche 0.658; Demi 0.564; McGovern 0.157; Tuzla Main 0.891 Mann-Whitney p-value comparing URD rates between upper and lower 50th percentile of PM: PM_{10} max All camps 0.034; Blue Factory camp 0.173; Comanche 0.314; Demi 0.083; McGovern 0.401; Tuzla Main 0.481 PM_{10} avg All camps 0.824; Blue Factory camp 0.682; Comanche 0.508; Demi N/A*; McGovern N/A*; Tuzla Main 0.656 **Notes:** * there were no days that fell in the upper 50%ile for PM avg in these camps -Rates of URD by PM quartiles for each camp presented in figures. Authors state, "Generally the avg URD rate increased with quartile of maximum exposure…the trend was not as clear for quartiles of PM_{10} avg exposure"

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hong et al. (2007) **Period of Study:** March 23-May 3, 2004 **Location:** School on the Dukjeok Island near Incheon City, Korea	**Outcome:** Peak expiratory flow rate (PEFR) **Age Groups:** 3rd to 6th grade (mean age = 9.6 yrs) **Study Design:** panel study **N:** 43 schoolchildren **Statistical Analyses:** Mixed linear regression **Covariates:** age, sex, height, weight, asthma history, and passive smoking exposure at home **Dose-response Investigated?** No **Lags Considered:** 0, 1, 2, 3, 4, 5	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 35.30 (23.48) 50th (Median): 29.36 **Range (Min, Max):** (12.24-124.87) **PM Component:** Fe: mean = 0.208 (0.203) µg/m^3 Median = 0.112 **Range (Min, Max):** (0.061-0.806) Mn: mean = 0.008 (0.005) µg/m^3 Median = 0.007 **Range (Min, Max):** (0.000-0.019) Pb: mean = 0.051 (0.031) µg/m^3 Median = 0.051 **Range (Min, Max):** (0.011-0.155) Zn: mean = 0.021 (0.021) µg/m^3 Median = 0.013 **Range (Min, Max):** (0.006-0.112) Al: mean = 0.085 (0.100) µg/m^3 Median = 0.031 **Range (Min, Max):** (0.017-0.344) **Copollutant:** $PM_{2.5}$	**Effect Estimate:** Regression coefficients of morning and daily mean PEFR on PM_{10} and metal components using linear mixed-effects regression Lag 1 (PM_{10}) Morning PEFR Crude: ß = -0.00, p = 0.99 Adjusted: ß = -0.04, p = 0.37 Mean PEFR Crude: ß = 0.00, p = 0.93 Adjusted: ß = -0.05, p = 0.12; Lag 1 (logFe) Morning PEFR Crude: ß = -1.26, p = 0.31 Adjusted: ß = -3.24, p = 0.13 Mean PEFR Crude: ß = -1.20, p = 0.20 Adjusted: ß = -2.37, p = 0.15; Lag 1 (logMn) Morning PEFR Crude: ß = -4.40, p<0.01 Adjusted: ß = -9.82, p<0.01 Mean PEFR Crude: ß = -4.05, p<0.01 Adjusted: ß = -8.44, p<0.01; Lag 1 (logPb) Morning PEFR Crude: ß = -6.79, p<0.01 Adjusted: ß = -6.83, p<0.01 Mean PEFR Crude: ß = -6.23, p<0.01 Adjusted: ß = -6.37, p<0.01; Lag 1 (logZn) Morning PEFR Crude: ß = -0.55, p = 0.71 Adjusted: ß = -0.98, p = 0.59 Mean PEFR Crude: ß = 1.33, p = 0.24 Adjusted: ß = 1.53, p = 0.28; Lag1 (logAl) Morning PEFR Crude: ß = -0.58, p = 0.57 Adjusted: ß = -2.22, p = 0.25 Mean PEFR Crude: ß = -0.59, p = 0.45 Adjusted: ß = -1.48, p = 0.32 Regression coefficients of morning and daily mean PEFR on metal components of PM_{10} and GSTM1 and GSTT1 genotype using linear mixed-effects regression Lag 1 (logPb) Morning PEFR: ß = -7.26, p<0.01 Mean PEFR: ß = -6.43, p<0.01 GSTM1 Morning PEFR: ß = 21.19, p = 0.23 Mean PEFR: ß = 20.09, p = 0.25; Lag 1 (logMn) Morning PEFR: ß = -10.31, p<0.01 Mean PEFR: ß = -8.66, p<0.01 GSTM1 Morning PEFR: ß = 21.02, p = 0.23 Mean PEFR: ß = 19.84, p = 0.25; Lag 1 (logPb) Morning PEFR: ß = -7.26, p<0.01 Mean PEFR: ß = -6.43, p<0.01 GSTT1 Morning PEFR: ß = 2.07, p = 0.90 Mean PEFR: ß = -2.39, p<0.88; Lag 1 (logMn) Morning PEFR: ß = -10.32, p<0.01 Mean PEFR: ß = -8.67, p<0.01 GSTT1 Morning PEFR: ß = 2.02, p = 0.90 Mean PEFR: ß = 2.33, p = 0.88

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hwang et al. (2006) **Period of Study:** 2001 **Location:** Taiwan	**Outcome:** Allergic rhinitis **Study Design:** Cross-sectional **Statistical Analyses:** Two-stage hierarchical models **Age Groups:** 6-15	**Pollutant:** PM_{10} **Averaging Time:** 1-h avg **Mean (SD):** 55.58 (16.57) **Range (Min, Max):** (29.36, 99.58) **Copollutants (correlation):** CO: r = 0.27 NO_X: r = 0.34 O_3: r = 0.28 SO_2: r = 0.58	**Increment:** 10 µg/m^3 **Odds Ratio (Lower CI, Upper CI); lag:** PM_{10} alone: 1.00 (0.99, 1.02); NO_X, PM_{10}: 0.99 (0.97, 1.00); CO, PM_{10}: 1.00 (0.99, 1.01); O_3, PM_{10}: 1.00 (0.99, 1.02) Gender Male: 1.02 (0.99, 1.04); Female: 0.99 (0.97, 1.02) Parental atopy* Yes: 1.00 (0.98, 1.03); No: 1.01 (0.99, 1.03) Parental education <6 years: 1.05 (0.96, 1.14); 6-8 years: 1.03 (0.98, 1.07); 9-11 years: 1.00 (0.98, 1.03); 12+ years: 0.99 (0.97, 1.02) Environmental tobacco smoke Yes: 1.01 (0.99, 1.03); No: 1.00 (0.98, 1.03) Visible mold** Yes: 1.02 (0.99, 1.06); No: 1.00 (0.98, 1.02) * Parental atopy was a measure of genetic predisposition and was defined as the father or the mother of the index child ever having been diagnosed as having asthma, allergic rhinitis, or atopic eczema. ** Visible mold found in the home.
Reference: Islam et al. (2007) **Period of Study:** 2006 **Location:** 12 California communities	**Outcome:** Respiratory symptoms, Asthma **Study Design:** Longitudinal study **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** 7-9; 10-11; >11	**Pollutants:** PM_{10} **Averaging Time:** 24-h avg **Copollutants (correlation):** O_3; NO_2; EC; OC	The study doesn't present quantitative results on PM_{10}.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Jalaludin et al. (2004) **Period of Study:** 2/1/1994–12/31/1994 **Location:** Western and southwestern Sydney, Australia	**Outcome:** Respiratory symptoms, Wheeze, Dry cough, Wet cough **Study Design:** Longitudinal study **Statistical Analyses:** Logistic regression model (GEE) **Age Groups:** 9-11	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 22.8 (13.8) **IQ Range (25th,75th):** (12.00, 122.8) **Copollutants (correlation):** O_3: r = 0.13 NO_2: r = 0.26	**Increment:** 10 µg/m^3 **Odds Ratio (Lower CI, Upper CI); Lag** Wheeze 1.01 (0.99, 1.03); 0; 1.01 (0.97, 1.04); 1; 0.99 (0.96, 1.03); 2; 1.02 (0.98, 1.06); 0-2 avg; 1.04 (0.99, 1.10); 0-5 avg Dry Cough 1.00 (0.98, 1.03); 0; 1.00 (0.97, 1.03); 1; 1.00 (0.97, 1.02); 2; 1.00 (0.97, 1.03); 0-2 avg; 1.03 (0.98, 1.08); 0-5 avg Wet Cough 1.01 (0.99, 1.04); 0; 0.99 (0.97, 1.01); 1; 1.00 (0.97, 1.03); 2; 0.99 (0.96, 1.02); 0-2 avg; 0.99 (0.94, 1.04); 0-5 avg Inhaled B2-agonist Use 0.99 (0.98, 1.01); 0; 1.00 (0.98, 1.03); 1; 0.99 (0.97, 1.01); 2; 1.00 (0.97, 1.02); 0-2 avg; 1.02 (0.98, 1.06); 0-5 avg Inhaled Corticosteroid Use 1.00 (0.99, 1.01); 0; 1.00 (0.99, 1.02); 1; 1.00 (0.99, 1.02); 2; 1.00 (0.98, 1.02); 0-2 avg; 1.00 (0.97, 1.02); 0-5 avg Doctor Visit for Asthma 1.11 (1.04, 1.19); 0; 1.10 (1.02, 1.19); 1; 1.15 (1.06, 1.24); 2; 1.11 (1.03, 1.20); 0-2 avg; 1.14 (0.98, 1.31); 0-5 avg **OR for respiratory symptoms and PM_{10} exposure by different groups** All children Wheeze: 1.01 (0.99, 1.04); Dry Cough: 1.00 (0.97, 1.02); Wet Cough: 1.01 (0.98, 1.04); Inhaled B2-agonist Use: 1.00 (0.98, 1.02); Inhaled Corticosteroid Use: 0.99 (0.98, 1.01); Doctor Visit for asthma: 1.11 (1.03, 1.19) Group 1* Wheeze: 1.01 (0.98, 1.04); Dry Cough: 0.97 (0.94, 0.99); Wet Cough: 1.00 (0.97, 1.03); Inhaled B2-agonist use: 1.00 (0.98, 1.02); Inhaled Corticosteroid Use: 1.00 (0.98, 1.01); Doctor Visit for asthma: 1.09 (0.98, 1.21) Group 2** Wheeze: 1.01 (0.97, 1.05); Dry Cough: 1.02 (0.98, 1.06); Wet Cough: 1.01 (0.96, 1.06); Inhaled B2-agonist use: 0.99 (0.94, 1.05); Inhaled Corticosteroid Use: 0.99 (0.97, 1.01); Doctor Visit for asthma: 1.12 (1.02, 1.23) Group 3*** Wheeze: 1.08 (0.90, 1.31); Dry Cough: 1.01 (0.91, 1.11); Wet Cough: 1.02 (0.94, 1.11); Inhaled B2-agonist use: 0.98 (0.84, 1.11); Inhaled Corticosteroid Use: 1.27 (1.08, 1.49); Doctor Visit for asthma: NR *Group 1 consists of children with a history of wheeze in the past 12 months, positive histamine challenge, and doctor diagnosed asthma. **Group 2 consists of children with a history of wheeze in the past 12 months and doctor diagnosed asthma. ***Group 3 consists of children only with a history y of wheeze in the past 12 months.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Jansen, et al. (2005) **Period of Study:** 1987-2000 **Location:** Seattle, WA	**Outcome:** FENO: fractional exhaled nitrogen oxide, Spirometry, Blood pressure, SaO2: oxygen saturation, Pulse rate **Age Groups:** 60-86-years-old **Study Design:** short-term cross-sectional case series **N:** 16 subjects diagnosed with COPD, asthma, or both **Statistical Analyses:** linear mixed effects model with random intercepts **Covariates:** age, relative humidity, temperature, medication use **Season:** winter 2002-2003 **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** Fixed-site Monitor: 18.0 All Subjects (N = 16) Indoor, home: 11.93 Outdoor, home: 13.47 Personal: 23.34 Asthmatic Subjects (N = 7) Indoor, home: 12.54 Outdoor, home: 11.86 Personal: 26.88 COPD Subjects (N = 9) Indoor, home: 11.45 Outdoor, home: 14.76 Personal: 19.91 **Range (Min, Max):** Fixed-site Monitor 2.5, 51 **IQR:** All Subjects Indoor, home: 6.93 Outdoor, home: 9.53 Personal: 20.72 Asthmatic Subjects Indoor, home: 10.19 Outdoor, home: 8.77 Personal: 20.08 COPD Subjects Indoor, home: 4.56 Outdoor, home: 6.14 Personal: 19.94	**PM Increment:** 10 $\mu g/m^3$ Slope [95% CI]: dependence of FENO concentration [ppb] on PM_{10} Asthmatic Subjects Indoor, home: 3.81 [-0.86: 8.50] Outdoor, home: 5.87 [2.87: 8.88]* Personal: 0.66 [-0.56: 1.88] COPD Subjects Indoor, home: 2.19 [-3.48: 7.87] Outdoor, home: 4.45 [-1.11: 10.01] Personal: 0.17 [-1.61: 1.96] Results indicate that FENO may be a more sensitive biomarker of PM exposure than other traditional health endpoints.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Johnston, et al. (2006) **Period of Study:** 7 months. (April 7 through November 7, 2004) **Location:** Darwin, Australia	**Outcome:** Asthma symptoms **Age Groups:** all ages **Study Design:** Time-series **N:** 251 people (130 adults, 121 children **Statistical Analyses:** Logistic regression model **Covariates:** minimum air temperature, doctor visits for influenza and the prevalence of asthma symptoms and, the fungal spore count and both onset of asthma symptoms and commencement of reliever medication **Season:** "dry season"-specific months NR, note Southern Hemisphere **Dose-response Investigated?** No **Statistical Package:** STATA8 **Lags Considered:** 0-5 days	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** 20 (6.4) **Range (Min, Max):** 2.6-43.3 **PM Component:** Vegetation fire smoke (95%) and motor vehicle emissions (5%) **Monitoring Stations:** 1	**PM Increment:** 10 µg/m^3 **RR Estimate [Lower CI, Upper CI]** Symptoms attributable to asthma Overall-1.010 (0.98,1.04); Adults–1.027 (0.987,1.068); Children–0.930 (0.96, 1.060); Using preventer- 1.022 (0.985, 1.060) Became symptomatic Overall- 1.240 (1.106,1.39); Adults- 1.277 (1.084,1.504); Children- 1.247 (1.058,1.468); Using preventer–1.317 (1.124,1.543) Used Reliever Overall- 1.010 (0.99, 1.04); Adults- 1.026 (0.990, 1.063); Children- 1.006 (0.960,1.055); Using preventer–1.035 (1.004,1.060) Commenced Reliever Overall- 1.132 (0.99, 1.29); Adults- 1.199 (0.994, 1.446); Children- 1.093 (0.906,1.319); Using preventer–1.194 (0.996, 1.432) Commenced Oral Steroids Overall- 1.540 (1.01, 2.34); Adults- 1.752 (1.008, 3.045); Children- 1.292 (0.682, 2.448); Using preventer–1.430 (0.888, 2.304) Asthma Attack Overall- 1.030 (0.95, 1.12); Adults- 1.08 (0.976, 1.202); Children- 0.861 (0.710, 1.044); Using preventer–1.051 (0.939,1.175) Exercise induced asthma Overall- 0.980 (0.92, 1.05); Adults- 0.988 (0.902, 1.081); Children- 0.972 (0.844,1.119); Using preventer–1.026 (0.928,1.134) Saw a health professional for asthma Overall- 1.030 (0.85, 1.26); Adults- 1.064 (0.794, 1.424); Children- 0.998 (0.749,1.328); Using preventer–0.924 (0.731, 1.169) Missed school or work due to asthma Overall- 1.102 (0.941, 1.290); Adults- 1.135 (0.897, 1.435); Children- 1.073 (0.862,1.333); Using preventer–1.025 (0.857,1.228) Mean daily number of asthma symptoms Overall- 1.020 (1.001,1.031); Adults- 1.027 (1.005,1.049); Children- 1.016 (0.986,1.047); Using preventer–1.034 (1.011,1.058) Mean Daily number of applications of reliever Overall- 1.020 (1.00,1.030); Adults- 1.032 (1.008, 1.057); Children- 1.002 (0.969,1.034); Using preventer–1.022 (1.001,1.043)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Just et al. (2002) **Period of Study:** 4/1/1996–6/30/1996 **Location:** Paris, France	**Outcome:** Incident and prevalent episodes of asthma attacks, nocturnal cough, wheeze, symptoms of irritation, respiratory infections, supplementary use of β2-agonists, Z-transformed peak expiratory flow (PEF), daily PEF variability **Age Groups:** 7-15 years old **Study Design:** Cohort **N:** 82 children **Statistical Analyses:** Linear regression, logistic regression, GEE **Covariates:** Effects of time trend, day of the week, weather, pollen levels **Season:** Spring/summer **Lags Considered:** 0, 0-2 mean, 0-4 mean	**Pollutant:** PM_{10} **Averaging Time:** Daily **Mean (SD):** 23.5 (8.4) **Range (Min, Max):** 9.0, 44.0 **Monitoring Stations:** 5 **Copollutant (correlation):** BS: 0.59 SO_2: 0.70 NO_2: 0.54 O_3: 0.21 temp: 0.04 humid: -0.41	**PM Increment:** 10 µg/m^3 for binary responses data (results that use odds ratios [ORs]) **Incident episodes of** 1) Asthma a) lag 0: 1.06 (0.61, 1.83); b) 0-2 mean: 1.09 (0.48, 2.49); c) 0-4 mean: 1.07 (0.44, 2.65) 2) Nocturnal cough a) lag 0: 1.10 (0.88, 1.37); b) 0-2 mean: 1.03 (0.77, 1.37); c) 0-4 mean: 1.11 (0.86, 1.42) 3) Respiratory infections a) lag 0: 0.64 (0.35, 1.15); b) 0-2 mean: 0.74 (0.38, 1.43); c) 0-4 mean: 0.99 (0.58, 1.68) **Prevalent episodes of** 1) Asthma a) lag 0: 1.07 (0.72, 1.59); b) 0-2 mean: 1.18 (0.64, 2.17); c) 0-4 mean: 1.16 (0.63, 2.13) 2) Nocturnal cough a) lag 0: 1.05 (0.83, 1.34); b) 0-2 mean: 1.10 (0.81, 1.50); c) 0-4 mean: 1.09 (0.79, 1.52) 3) Respiratory infections a) lag 0: 1.17 (0.68, 2.03); b) 0-2 mean: 1.31 (0.51, 3.36); c) 0-4 mean: 1.71 (0.71, 4.12) 4) Eye irritation a) lag 0: 1.18 (1.01, 1.39); b) 0-2 mean: 1.28 (1.03, 1.59); c) 0-4 mean: 1.42 (1.12, 1.80) **Analysis restricted to days with no steroid use** **Incident episodes of** 1) Eye irritation a) lag 0: 1.07 (0.66, 1.71); b) 0-2 mean: 0.83 (0.45, 1.53); c) 0-4 mean: 0.92 (0.46, 1.83) 2) Throat irritation a) lag 0: 1.33 (0.66, 2.69); b) 0-2 mean: 1.28 (0.58, 2.80); c) 0-4 mean: 1.06 (0.38, 2.95) 3) Nose irritation a) lag 0: 0.74 (0.48, 1.13); b) 0-2 mean: 0.76 (0.42, 1.36); c) 0-4 mean: 0.96 (0.53, 1.73) **Prevalent episodes of** 1) Eye irritation a) lag 0: 1.20 (0.88, 1.65); b) 0-2 mean: 1.71 (0.97, 3.01); c) 0-4 mean: 1.97 (1.03, 3.76) 2) Throat irritation a) lag 0: 1.23 (0.83, 1.82); b) 0-2 mean: 1.08 (0.68, 1.73); c) 0-4 mean: 0.91 (0.47, 1.73) 3) Nose irritation a) lag 0: 1.20 (0.91, 1.58); b) 0-2 mean: 1.09 (0.78, 1.52); c) 0-4 mean: 1.09 (0.73, 1.61) **Notes:** The authors noted that incident or prevalent wheeze was not correlated with levels of any type of pollutant; also, they state no relationship was observed between PEF variables and levels of PM. The authors also note that in a multipollutant model assessing independent effects of PM and O_3 on prevalent episodes of eye irritation (mean 0-4), the PM parameter decreased and was not significant ($p = 0.19$).

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kulkarni et al (2006) **Period of Study:** 11/2002–12/2003 **Location:** Leicester, United Kingdom	**Outcome:** Lung function by spirometry: FVC, FEV_1, FEV_1: FVC, FEF_{25-75} **Age Groups:** 8-15 **Study Design:** Cross-sectional **N:** 114 children, 64 provided sputum for assessment of carbon content of macrophages. **Statistical Analyses:** Linear regressions, Spearman rank correlations. Mann-Whitney, Chi-square and unpaired t tests were used to compare results between asthmatic and non asthmatic children **Covariates:** BMI, sex, exercise, traffic PM_{10} **Dose-response Investigated?** Yes **Statistical Package:** SPSS	**Pollutant:** Primary PM_{10} ($\mu g/m^3$) concentration was modeled, and was considered a covariate for carbon content of macrophages. Carbon content of alveolar macrophages was the primary variable of interest. **Averaging Time:** 1 yr 50th(Median): Children without asthma, 1.21; Children with asthma, 1.81 **Range (Min, Max):** Children without asthma, 0.10, 2.17; Children with asthma, 0.17, 2.13 **PM Component:** Carbon content in alveolar macrophages **Monitoring Stations:** NR. **Copollutant (correlation):** vs carbon content in macrophages (increment, coefficient [range])–1.0 $\mu g/m^3$, 0.1 [0.01-0.18]	**PM Increment:** 1.0 $\mu g/m^3$ **% Change [Lower CI, Upper CI]:** Single pollutant model: FEV_1: -4.3 [-8.5, 0.2] p = 0.04; R^2 = 0.06 Single pollutant model: FVC: -1.2 [-5.6, 3.2] p = 0.59; R^2 = 0.005 Single pollutant model: FEF_{25-75}: -8.6 [-17.3, 0.1] p = 0.05; R^2 = 0.06 2 pollutant model with Macrophage Carbon: FEV_1: PM_{10} -2.9 [-6.9, 1.2]; p = 0.17 (FVC): PM_{10} 0.1 [-4.4, 4.6]; p = 0.96 FEF_{25-75}: PM_{10} -5.5 [-14.2, 3.1]; p = 0.21
Reference: Kuo, et al. (2002) **Period of Study:** 1-yr period (year not specified) **Location:** Central Taiwan	**Outcome:** Asthma (yes/no) **Age Groups:** 13-16 years **Study Design:** Cohort **N:** 12926 total children; 775 asthmatic children; 8 junior high schools **Statistical Analyses:** Pearson correlation coefficients; Logistic regression **Covariates:** Gender, age, residential area, level of parental education, number cigarettes smoked by family members, incense burning in the home, frequency of physical activities **Dose-response Investigated?** No **Statistical Package:** SAS 6.12 **Lags Considered:** Monthly averages at each school	**Pollutant:** PM_{10} **Averaging Time:** 1-h **Mean (SD):** School A: 59.7 School B: 65.3 School C: 84.3 School D: 59.2 School E: 75.3 School F: 60.2 School G: 54.1 School H: 69.0 **Monitoring Stations:** 8 (1 for each school)	**PM Increment:** Dichotomized annual avg: <65.9 $\mu g/m^3$ ≥ 65.9 $\mu g/m^3$ OR Estimate [Lower CI, Upper CI]; lag: Crude (outcome = asthma, yes/no) <65.9 $\mu g/m^3$: 1 (ref) ≥ 65.9 $\mu g/m^3$: 0.837 [NR] Adjusted (outcome = asthma, yes/no) <65.9 $\mu g/m^3$: 1 (ref) ≥ 65.9 $\mu g/m^3$: 0.947 [0.640, 1.401] **Notes:** asthma prevalence was highest in urban areas and lowest in rural areas Pearson correlation between annual PM levels at each school and asthma prevalence at each school: 0.214 [p>0.05]
Reference: Lagorio et al. (2006) **Period of Study:** 5/24/1999 to 6/24/1999 and 11/181999 to 12/22/1999 **Location:** Rome, Italy	**Outcome:** Lung function of subjects (FVC and FEV_1) with COPD, Asthma **Age Groups:** COPD 50 to 80 yrs Asthma 18 to 64 yrs **Study Design**: Time series **N:** COPD N = 11; Asthma N = 11 **Statistical Analyses:** Non-parametric Spearman correlation; GEE; **Covariates:** COPD and IHD: daily mean temperature, season variable (spring or winter), relative humidity, day of week; Asthma: season variable, temperature, humidity, and β-2-agonist use **Season:** Spring and winter **Dose-response Investigated?** Yes **Statistical Package:** STATA **Lags Considered:** 1–3 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** Overall: 42.8 (21.8) Spring: 36.9 (10.8) Winter: 49.0 (28.1) **Range (Min, Max):** (7.9, 123) **PM Component:** NR **Monitoring Stations**: Two fixed sites: (Villa Ada and Istituto superior di Sanita) **Copollutant (correlation):** NO_2 r = 0.45 O_3 r = -0.36 CO r = 0.55 SO_2 r = 0.21 $PM_{10-2.5}$ r = 0.61 $PM_{2.5}$ r = 0.93	**PM Increment:** 1 $\mu g/m^3$ They observed negative association between ambient PM_{10} and respiratory function (FVC and FEV_1) in the COPD panel. The effect on FVC was seen at lag 24 h, 48 h, and 72 h. The effect on FEV_1 was evident at lag 72 h. There was no statistically significant effect of PM_{10} on FVC and FEV_1 in the asthmatic and IHD panels. β **Coefficient (SE)** **COPD** FVC(%) 24 h -0.66 (0.30); 48-h -0.75 (0.35); 72-h -0.94 (0.47) FEV_1(%) 24 h -0.37 (0.27); 48-h -0.58 (0.31); 72-h -0.87 (0.43) **Asthma** FVC(%) 24 h -0.12 (0.24); 48-h -0.09 (0.29); 72-h -0.08 (0.36) FEV_1(%) 24 h -0.28 (0.28); 48-h -0.40 (0.34); 72-h -0.40 (0.43)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lee, et al. (2007b) **Period of Study:** 2000-2001 **Location:** South-Western Seoul Metropolitan area, Seoul, South Korea	**Outcome:** PEFR (peak expiratory flow rate), lower respiratory symptoms (cold, cough, wheeze) **Age Groups:** 61-89 years of age (77.8 mean age) **Study Design:** longitudinal panel survey **N:** 61 adults **Statistical Analyses:** Logistic regression model **Covariates:** Temperature (Celsius), relative humidity, age, season **Dose-response Investigated?** No **Statistical Package:** SAS 8.0 **Lags Considered:** 0-4 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD)**: 71.40 (30.69) Percentiles: 25th: 43.47 50th(Median): 74.92 75th: 87.54 **Range (Min, Max):** 26.23, 148.34 **Monitoring Stations:** 2	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]; lag:** PEFR (peak expiratory flow rate) -0.39 (-0.63 to -0.14); 1 day relative odds of a lower respiratory symptom (cold, cough, wheeze) 1.015 (0.900,1.144); 1 day
Reference: Lewis, et al (2005) **Period of Study:** winter 2001-spring 2002 **Location:** Detroit, Michigan, USA	**Outcome**: Poorer lung function (increased diurnal variability and decreased forced expiratory volume) **Age Groups:** 7-11 years old **Study Design:** longitudinal cohort study **N:** 86 children **Statistical Analyses:** descriptive statistics and bivariate analyses of exposures, multivariable regression models that included interaction terms between exposure measures and CS use or, alternatively, presence of a URI, multivariate analog of linear regression. **Covariates:** sex, home location, annual family income, presence of one or more smokers in household, race,season (entered as dummy variables), and parameters to account for intervention group effect. **Season: W**inter 2001 (February 10–23), spring 2001 (May 5–18), summer 2001 (July 14–27), fall 2001 (September 22–October 5), winter 2002 (January 18–31), and spring 2002 (May 18–31). **Dose-response Investigated?** No **Lags Considered:** 1 to 2 days; 3-5 days	**Pollutant:** PM_{10} **Averaging Time:** 2 weeks **Mean (SD):** Eastside 23.0 (13.5) Southwest 28.5 (16.1) **Range (Min, Max):** 2.9, 70.9 **PM Component:** ("likely" in southwest site) carbon and diesel emissions **Monitoring Stations:** 2 **Copollutant:** $PM_{2.5}$ 0.93 O_3 Daily mean 0.59 O_3 8-h peak 0.57	**PM Increment:** 19.1 µg/m^3 **Lung function among children reporting use of maintenance CSs** Diurnal variability FEV_1 Lag 1: 1.53 [-0.85, 3.90]; Lag 1: 2.94 [-1.07, 6.96] PM_{10} + O_3; Lag 2: 5.32 [0.32, 10.33]; Lag 2: 13.73 [8.23, 19.23] PM_{10} + O_3; Lag 3-5: 1.46 [-2.21,5.13]; Lag 3-5: 3.30 [0.58, 6.02] PM_{10} + O_3 Lowest daily value FEV_1 Lag 1: -0.28 [-2.34, 1.77]; Lag 1: -6.25 [-11.15 to -1.36] PM_{10} + O_3; Lag 2: -2.21 [-3.97 to -0.46]; Lag 2: -5.97 [-11.06 to -0.87] PM_{10} + O_3; Lag 3-5: -2.58 [-7.65, 2.49]; Lag 3-5: 1.98 [-0.38, 4.33] PM_{10} + O_3 **Lung function among children reporting presence of URI on day of lung function assessment** Diurnal variability FEV_1 Lag 1: 3.51 [-4.52,11.55]; Lag 1: 3.21 [-1.28,7.71] PM_{10} + O_3; Lag 2: 1.12 [-4.62, 6.86]; Lag 2: 5.40 [-0.82, 11.62] PM_{10} + O_3; Lag 3-5: 3.90 [0.34, 7.47]; Lag 3-5: 6.27 [0.07, 12.47] PM_{10} + O_3 Lowest daily value FEV_1 Lag 1: -2.72 [-9.47, 4.03]; Lag 1: -13.11 [-21.59 to -4.62] PM_{10} + O_3; Lag 2: 0.24 [-5.10, 4.63]; Lag 2: -3.32 [-6.83, 0.18] PM_{10} + O_3; Lag 3-5: -4.48 [-8.36, 0.60]; Lag 3-5: -3.17 [-5.82 to -0.51] PM_{10} + O_3

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Mar et al. (2004) **Period of Study:** 1997-1999 **Location:** Spokane, Washington	**Outcome:** Respiratory symptoms **Age Groups:** Adults: Ages 20-51 yrs; Children: Ages 7-12 yrs **Study Design:** Time-series **N:** 25 people **Statistical Analyses:** Logistic regression **Covariates:** Temperature, relative humidity, day of-the-wk **Statistical Package:** STATA 6 **Lags Considered:** 0-2 days	**Pollutant:** PM_{10} **Mean (SD):** 1997: 24.5 (18.5) 1998: 20.6 (12.3) 1999: 16.8 (8.0) **Monitoring Stations:** 1 station **Copollutant (correlation):** PM_{10} PM_1; r = 0.48 $PM_{2.5}$; r = 0.61 $PM_{10-2.5}$; r = 0.93	**PM Increment:** 10 µg/m^3 **OR Estimate [Lower CI, Upper CI]; lag:** **Adult Respiratory symptoms:** Wheeze: 1.01[0.93, 1.09]; lag 0; 0.98[0.91, 1.06]; lag 1; 0.99[0.92, 1.06]; lag 2 Breath: 1.02[0.96, 1.08]; lag 0 ; 1.01[0.97, 1.06]; lag 1; 1.02[0.97, 1.06]; lag 2 Cough: 0.96[0.88, 1.05]; lag 0 ; 0.97[0.90, 1.04]; lag 1; 0.98[0.92, 1.05]; lag 2 Sputum: 1.01[0.92, 1.12]; lag 0; 0.99[0.91, 1.08]; lag 1; 1.00[0.93, 1.08]; lag 2 Runny Nose: 0.98[0.93, 1.04]; lag 0; 0.97[0.93, 1.02]; lag 1;0.97[0.94, 1.01]; lag 2 Eye Irritation: 0.97[0.87, 1.08]; lag 0; 0.97[0.88, 1.06]; lag 1; 0.97[0.91, 1.04]; lag 2 Lower Symptoms: 0.96[0.91, 1.02]; lag 0; 0.95[0.89, 1.00]; lag 1; 0.95[0.90, 1.00]; lag 2 Any Symptoms: 0.97[0.93, 1.02]; lag 0; 0.96[0.91, 1.00]; lag 1; 0.95[0.91, 0.99]; lag 2 **Children Respiratory symptoms**: Wheeze: 0.92[0.71, 1.18]; lag 0; 0.89[0.64, 1.24]; lag 1; 0.95[0.69, 1.31]; lag 2 Breath: 1.04[0.95, 1.15]; lag 0; 1.04[0.95, 1.15]; lag 1; 1.06[0.95, 1.19]; lag 2 Cough: 1.09[1.02, 1.16]; lag 0; 1.08[1.02, 1.14]; lag 1; 1.10[1.02, 1.18]; lag 2 Sputum: 1.08[0.98, 1.17]; lag 0; 1.07[0.98, 1.17]; lag 1; 1.07[0.98, 1.16]; lag 2 Runny Nose: 1.08[1.00, 1.16]; lag 0; 1.08[1.02, 1.15]; lag 1; 1.08[1.02, 1.14]; lag 2 Eye Irritation: 1.06[0.74, 1.51]; lag 0; 0.94[0.70, 1.26]; lag 1; 0.99[0.88, 1.12]; lag 2 Lower Symptoms: 1.07[1.00, 1.14]; lag 0; 1.06[0.98, 1.15]; lag 1; 1.07[0.95, 1.19]; lag 2 Any Symptoms: 1.07[1.02, 1.11]; lag 0; 1.09[1.03, 1.15]; lag 1; 1.10[1.03, 1.17]; lag 2
Reference: Mar et al. (2005b) **Period of Study:** 1999-2001 **Location:** Seattle, Washington	**Outcome:** Pulmonary function (arterial oxygen saturation) and cardiac function (heart rate and blood pressure) **Study Design:** Time series **Statistical Analyses:** Linear logistic regression **Age Groups:** >57	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); Lag** Indoor Systolic: 0.92 (-0.95, 2.78); 0; Diastolic: 0.63 (-0.29, 1.56); 0 Outdoor Systolic: -0.10 (-1.37, 1.18); 0; Diastolic: -0.03 (-0.79, 0.73); 0 Nephelometer Systolic: 0.35 (-0.91, 1.61); 0; Diastolic: -0.12 (-0.91, 0.67); 0 **% Increase between heart rate and PM_{10} exposure for people >57** PM_{10} Indoor: 0.02 (-0.54, 0.58); 0; Outdoor: -0.48 (-1.03, 0.06); 0; Nephelometer: -0.31 (-0.76, 0.14); 0

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: McConnell et al. (2002) **Period of Study:** 1993-1998 **Location:** 12 communities in Southern California (grouped into either high and low pollution communities)	**Outcome:** Asthma (new diagnosis) **Age Groups:** 9-12 yrs, 12-13 yrs, 15-16 yrs **Study Design:** Cohort **N:** 3535 **Statistical Analyses:** Multivariate proportion hazard model **Covariates:** Sex, age, ethnic origin, BMI, child history of allergies and asthma history, SES, maternal smoking, time spent outside, history of wheezing, ownership of insurance (yes/no), number and type of sports played **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.1	**Pollutant:** PM_{10} **Averaging Time:** 4 yrs **Mean (SD):** Low pollution communities: 21.6 (3.8) **High pollution communities:** 43.3 (12.0) **Percentiles:** Low pollution communities: 50th(Median): 20.8 High pollution communities: 50th(Median): 43.3 **Range (Min, Max):** Low pollution communities: 16.62, 27.3 High pollution communities: 33.5, 66.9 **Monitoring Stations:** 12 **Copollutant (correlation):** $PM_{2.5}$: r = 0.96; NO_2: r = 0.65; O_3	**RR Estimate [Lower CI, Upper CI]; lag:** Low PM communities: 1.0 [ref] 0 sport; 1.5 [1.0, 2.2] 1 sport; 1.2 [0.7, 1.9] 2 sports; 1.7 [0.9, 3.2] >/ = 3 sports High PM communities: 1.0 [ref] 0 sport; 1.1 [0.7, 1.7] 1 sport; 0.9 [0.5, 1.7] 2 sports; 2.0 [1.1, 3.6] >/ = 3 sports **High vs Low PM_{10} communities: 0.8 (0.6, 1.0)** **Incidence–N (incidence) number of sports:** Low PM communities: 49 (0.023) 0; 54 (0.032) 1; 22 (0.024) 2; 13 (0.033) >/ = 3 High PM communities: 55 (0.021) 0; 36 (0.021) 1; 14 (0.018) 2; 16 (0.033) >/ = 3
Reference: McCreanor et al. (2007) **Period of Study:** 2003-2005 **Location:** London, England	**Outcome:** Decreased Lung Function **Age Groups:** Adults **Study Design:** Crossover study **N:** 60 adults **Statistical Analyses:** Linear regression **Covariates:** Temperature, relative humidity, age, sex, bod-mass index, and race or ethnic group	**Pollutant:** UFP **50th (Median):** Oxford St: 125 Hyde St: 72 **Range (Min, Max):** Oxford St: (62, 161) Hyde Park: (60, 100)	% changes in FEV and FVC are presented in figures 1-3. Results are not presented quantitatively in text or tables. The authors did not find any significant differences in respiratory symptoms between the two locations. Also, there were no significant differences in sputum eosinophili counts or eosinophil cationic protein levels.
Reference: Mortimer et al. (2008) **Period of Study:** 1989-2000 **Location:** Joaquin Valley, California	**Outcome:** Respiratory Symptoms, Decreased lung function **Study Design:** Time series **Statistical Analyses:** Deletion/Substitution/ Addition algorithm (GEE); Logistic linear regression **Age Groups:** 6-11	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Copollutants (correlation):** CO: r = 0.05; NO_2: r = 0.30; O_3: r = 0.39	**Increment:** NR β (SE): **FVC:** PM_{10} (age 0-3 yrs): 0.0121 (0.0037) FEV_1**:** PM_{10} (age 0-3 yrs): 0.0102 (0.0034) **PEF:** PM_{10} (Mother smoked during pregnancy): -0.0102 (0.0039)
Reference: Mortimer et al. (2002) **Period of Study:** June-August 1993 **Location:** Eight urban areas of the US: Bronx and East Harlem, NY; Baltimore, MD; Washington, DC; Detroit, MI; Cleveland, OH; Chicago, IL; and St. Louis, MO.	**Outcome:** peak expiratory flow rate (PEFR) and symptoms **Age Groups:** 4-9 yrs **Study Design:** Cohort study **N:** 846 children with a history of asthma **Statistical Analyses:** Mixed linear models and GEE **Covariates:** day of study, previous 12-h mean temperature, urban area, diary number, rain in the past 24 h **Season: S**ummer **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 1-5 avg, 1-4 avg, 0-4 avg, 0-3 avg	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 53 **Monitoring Stations:** NR **Copollutant (correlation):** 8-h avg ozone: r = 0.51	**PM Increment:** 20 μg/m^3 **Effect Estimate [Lower CI, Upper CI]:** (RR estimates are odds ratios for incidence of morning asthma symptoms using the avg of lag 1-2) 3 urban areas (DE, CL, CH) Single pollutant: OR = 1.26 (1.00-1.59) Ozone+PM_{10}: OR = 1.25 (0.97-1.61) Ozone+SO_2 +NO_2+PM_{10}: OR = 1.14 (0.80-1.48)
Reference: Moshammer and Neuberger (2003) **Period of Study:** 2000-2001 **Location:** Linz, Austria	**Outcome:** Lung Function: FVC, FEV_1, MEF_{25}, MEF_{50}, MEF_{75}, PEF, LQ Signal, PAS Signal **Age Groups:** Ages 7 to 10 **Study Design:** Case-crossover **N:** 161 children; 1898–2120 "half-h means" **Statistical Analyses:** Correlations Regression Analysis **Covariates:** Morning, Evening, Night **Season:** Spring, summer, Winter, Fall **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 8 h Daily Means **Mean (SD):** 23.13 (20.08) **Range (Min, Max):** (NR, 190.79) **Monitoring Stations:** 1 **Copollutant (correlation):** LQ = 0.751 PAS = 0.406	**Notes:** "Acute effects of 'active particle surface' as measured by diffusion charging were found on pulmonary function (FVC, FEV_1, MEF50) of elementary school children and on asthma-like symptoms of children who had been classified as sensitive."

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Moshammer et al. (2006) **Period of Study:** 2000-2001 **Location:** Linz, Austria	**Outcome:** Respiratory symptoms and decreased lung function **Age Groups:** Children ages 7-10 **Study Design:** Time-series **N:** 163 children **Statistical Analyses:** GEE model **Covariates:** Sex, age, height, weight **Dose-response Investigated?** NR **Statistical Package:** NR **Lags Considered:** 1	**Pollutant:** PM_{10} **Averaging Time:** 8-h **Mean (SD):** Maximum 24 h: 76.39 Annual avg: 19.06 **Percentiles:** 8-h mean 25th: 14.39 8-h mean 50th(Median): 24.85 8-h mean 75th: 38.82 **Monitoring Stations:** 1 station **Copollutant (correlation):** PM_1; r = 0.91; $PM_{2.5}$; r = 0.93; NO_2; r = 0.62	**PM Increment:** 10 µg/m^3 **% change in Lung Function per 10** µg/m^3 FEV: 0.11 FVC: 0.06 $FEV_{0.5}$: -0.19 $MEF_{75\%}$: -0.30 $MEF_{50\%}$: -0.36 $MEF_{25\%}$: 0.41 PEF: 0.22 **% change in Lung Function per IQR** FEV: -0.27 FVC: -0.07 $FEV_{0.5}$: -0.47 $MEF_{75\%}$: -0.74 $MEF_{50\%}$: -0.86 $MEF_{25\%}$: 0.98 PEF: -0.54
Reference: Neuberger et al. (2004) **Period of Study:** 6/1999-6/2000 **Location:** Austria (Vienna and a rural area near Linz)	**Outcome:** Questionnaire derived asthma score, and a 1-5 point respiratory health rating by parent **Age Groups:** 7-10 years **Study Design:** Cross-sectional survey **N:** about 2000 children **Statistical Analyses:** mixed models linear regression-used factor analysis to develop the "asthma score" **Covariates:** Pre-existing respiratory conditions, temperature, rainy days, # smokers in household, heavy traffic on residential street, gas stove or heating, molds, sex, age of child, allergies of child, asthma in other family members **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 4 week avg (preceding interview)	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Copollutant (correlation):** $PM_{2.5}$ (r = 0.94) in Vienna	**PM Increment:** 10 µg/m^3 **Change in mean associated unit increase in PM (p-value); lag** Respiratory Health score Vienna: 0.005 (p>0.05); lag 4 week avg Rural area: 0.008 (p>0.05); lag 4 week avg Asthma score Vienna: 0.006 (p>0.05); lag 4 week avg Rural area: -0.001 (p>0.05); lag 4 week avg
Reference: Neuberger et al. (2004) **Period of Study:** Sept 1999-March 2000 **Location:** Vienna, Austria	**Outcome:** Ratio measure: Time to peak tidal expiratory flow divided by total expiration time (i.e., tidal lung function, a surrogate for bronchial obstruction) **Age Groups:** 3.0-5.9 years (preschool children) **Study Design:** Longitudinal prospective cohort **N:** 56 children **Statistical Analyses:** mixed models linear regression, with autoregressive correlation structure **Covariates:** Age, sex, respiratory rate, phase angle, temperature, kindergarten, parental education, observer (also in sensitivity analyses: height, weight, cold/sneeze on same day, heating with fossil fuels, hair cotinine, number of tidal slopes used to measure tidal lung function) **Dose-response Investigated?** No **Statistical Package:** SAS 8.0 **Lags Considered:** 0	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Copollutant (correlation):** $PM_{2.5}$ (r = 0.94) in Vienna	**PM Increment:** Interquartile range (NR) Change in mean associated with an IQR increase in PM (p-value); lag -1.067 (0.241); lag 0

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Neuberger et al. (2004) **Period of Study:** Oct. 2000-May 2001 **Location:** Linz, Austria	**Outcome:** Forced oscillatory resistance (at zero Hz), FVC, FEV_1, MEF_{25}, MEF_{50}, MEF_{75}, PEF **Age Groups:** 7-10 years **Study Design:** Longitudinal prospective cohort **N:** 164 children **Statistical Analyses:** mixed models linear regression with autoregressive correlation structure **Covariates:** sex, time and individual **Season:** Oct-May **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-7	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Monitoring Stations:** 1	**PM Increment:** 1 µg/m^3 **Notes:** No significant associations between PM_{10} and the metrics of lung function were reported. The authors state they only reported significant associations, so results are assumed to be null.
Reference: Peacock, et al (2003) **Period of Study:** November 1, 1996 to 14 February 1997 **Location**: northern Kent, UK	**Outcome:** Reduced peak expiratory flow rate (PEFR) **Age Groups:** 7-13 years of age **Study Design:** Time-series **N:** 179 **Statistical Analyses:** GEE **Covariates:** Day of the week, 24-h mean outside temperature. **Season:** winter **Dose-response Investigated?** No **Statistical Package:** STATA **Lags Considered:** Same day, lag 1, lag 2, five day moving avg	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** Rural (nationally validated) 21.2 (11.3); Rural (locally validated) 18.7 (11.3); Urban 1 18.4 (9.8); Urban 2 22.7 (10.6) **Percentiles:** 10th Rural (nationally validated) 11.0; Rural (locally validated) 9.0; Urban 1 10.5; Urban 2 12.5 90th Rural (nationally validated) 33.0; Rural (locally validated) 32.5; Urban 1 32.0; Urban 2 36.0 **Range (Min, Max):** Rural (nationally validated) 7.0, 82.0; Rural (locally validated) 6.6, 87.9; Urban 1 4.7, 62.8; Urban 2 6.7, 63.7 **Monitoring Stations:** 3	**PM Increment:** 10 µg/m^3 Odds ratio [Lower CI, Upper CI]; lag: 1.037 [0.992, 1.084]; 5 days
Reference: Peacock et al. (2003) **Period of Study:** 11/1/1996–2/14/1997 **Location:** Southern England	**Outcome:** Respiratory Symptoms, Cough, Cold, Wheezing, Change in PEFR **Study Design:** Time-series **Statistical Analyses:** Multiple linear regression **Age Groups:** 7-11; 10-11; 12-13	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 21.2 (11.5) **Range (Min, Max):** (6.6, 87.9) **Copollutants:** NO_2 O_3 SO_2 SO_4^{2-}	**Increment:** 10 µg/m^3 **Odds Ratio (Lower CI, Upper CI); Lag** **Change in PEFR** Community -0.04 (-0.11, 0.03); 0; 0.03 (-0.04, 0.05); 1; -0.01 (-0.07, 0.05); 2; -0.10 (-0.25, 0.05); 0-4 avg Local -0.01 (-0.06, 0.03); 0; 0.04 (0.01, 0.08); 1; 0.01 (-0.04, 0.05); 2; 0.04 (-0.05, 0.13); 0-4 avg **20% decrease in PEFR** All children 1.012 (0.992, 1.031); 0; 1.016 (0.995, 1.036); 1; 1.013 (1.000, 1.025); 2; 1.037 (0.992, 1.084); 0-4 avg Wheezy Children Only 1.016 (0.986, 1.047); 0; 1.030 (1.001, 1.060); 1; 1.018 (0.995, 1.041); 2; 1.114 (1.057, 1.174); 0-4 avg

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Peled, et al (2005) **Period of Study:** 5-6 weeks between March-June 1999 and September-December 1999. **Location:** Ashdod, Ashkelon and Sderot, Israel	**Outcome:** Reduced peak expiratory flow (PEF) **Age Groups:** 7-10 years **Study Design:** Nested cohort study **N:** 285 **Statistical Analyses:** Time series analysis, generalized linear model, GEE, one-way ANOVA, generalized linear model **Covariates:** seasonal changes, meteorological conditions and personal physiological, clinical and socioeconomic measurements **Season:** spring, autumn **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** PM_{10} **Averaging Time:** daily Mean: Ashkelon: 67.1 Sderot: 52.9 Ashdod: 31.0 **PM Component:** Local industrial emissions, desert dust, vehicle emissions and emissions from two electric power plants **Monitoring Stations:** 6 **Copollutant:** $PM_{2.5}$	**PM Increment:** 1 µg/m^3 β **coefficient (SE) [95% CI]** Sderot: PM_{10} MAX: -0.34 (0.41) [-1.16, 0.46] PM_{10} MAX x sin(ω2 day): 0.84 (0.22) [0.405, 1.28] PM_{10} MAX x cos (ω1 day): -1.61 (0.41) [-2.43, 0.79] PM_{10} MAX x sin (ω1 day): 0.44 (0.120) [-0.68-0.21] In Sderot, an interaction between PM_{10} and the sequential day were significantly associated with PEF.
Reference: Pitard, et al (2004) **Period of Study:** 732 days (July 1998-June 2000) **Location:** City of Rouen, France	**Outcome:** Respiratory drug sales **Age Groups:** 0-14, 15-64, 65-74, over 75 years **Study Design:** Ecological time-series **N:** 106,592 **Statistical Analyses:** Generalized additive model **Covariates:** Days of the weeks, trend, seasonal variations, influenza epidemics, meteorological variables, holidays **Dose-response Investigated?** No **Statistical Package:** S-plus **Lags Considered:** 0 to 10 days	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** 16.7 (13.3) **Percentiles:** 25th: 8.00 50th(Median): 13.0 75th: 20 **Range (Min, Max):** 2.00, 126 **Monitoring Stations:** 2 **Copollutant (correlation):** SO_2 (0.39); NO_2 (0.61)	**PM Increment:** 10 µg/m^3 Percent increase in sales of anti-asthmatics and bronchodialators (Lower CI, Upper CI); lag: 6.2 (2.4, 10.1); lag 10 days Percent increase in sales of cough and cold preparation for children under 15 years of age (Lower CI, Upper CI); lag: 9.2 (5.9, 12.6); 10 days
Reference: Preutthipan et al. (2004) **Period of Study:** 31 days (school days) from January 14 to February 26, 1999 **Location:** Mae Pra Fatima School, central Bangkok, Thailand	**Outcome:** Decreases in peak expiratory flow rates (PEFR), respiratory symptoms including wheeze, shortness of breath, runny/stuffed nose, sneezing, cough, phlegm, and sore throat **Age Groups:** Third to ninth grade **Study Design:** Time- Series **N:** 133 children (93 asthmatics, 40 nonasthmatics) **Statistical Analyses:** For continuous data, an unpaired t-test or Mann-Whitney U test was used. For categorical data, the chi-square test or Fisher's exact test was used. One-way analysis of covariance (ANCOVA) was used to compare avg daily reported respiratory symptoms, diurnal PEFR variability, and the prevalence of PEFR decrements between groups of days. **Covariates:** Age, sex, weight, height, parents smoking, person smoking in home, daily number of household cigarettes, air-conditioned bedroom, fuel used for cooking (charcoal, gas), distance from home to main road **Dose-response Investigated?** No **Lags Considered:** Up to 5 days	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** 111.0 (39) **Range (Min, Max):** 46, 201 **Monitoring Stations:** 1 **Copollutant:** SO_2 CO O_3	**PM Increment:** Authors classified exposure according to High and Low PM_{10} days: High = >120 µg/m^3; Low = <120 µg/m^3 Daily reported respiratory symptoms and diurnal PEFR variablility as classified by concurrent days with high vs. low PM_{10} Mean % reporting (SEM) Asthmatics: High PM_{10}; Wheeze/shortness of breath = 21.3 (1.4); Runny/stuffed nose or sneezing = 42.3 (1.8); Cough = 59.9 (1.9); Phlegm = 60.5 (2.3); Sore throat = 23.7 (1.5); Any respiratory symptoms = 72.2 (3.2); Diurnal PEFR variability = 3.0 (0.4) Asthmatics: Low PM_{10} Wheeze/shortness of breath = 19.3 (1.3); Runny/stuffed nose or sneezing = 35.8 (1.6); Cough = 59.1 (1.6); Phlegm = 58.6 (2.0); Sore throat = 21.0 (1.4); Any respiratory symptoms = 63.8 (2.8); Diurnal PEFR variability = 2.8 (0.3) Nonasthmatics: High PM_{10} Wheeze/shortness of breath = 11.7 (1.4); Runny/stuffed nose or sneezing = 40.9; Cough = 50.4 (2.6); Phlegm = 50.2 (2.5); Sore throat = 27.1 (1.7); Any respiratory symptoms = 67.8 (3.7); Diurnal PEFR variability = 2.4 (0.4) Nonasthmatics: Low PM_{10} Wheeze/shortness of breath = 9.3 (1.2); Runny/stuffed nose or sneezing = 33.1 (2.2); Cough = 54.0 (2.2); Phlegm = 49.9 (2.2); Sore throat = 23.9 (1.5); Any respiratory symptoms = 56.4 (3.2); Diurnal PEFR variability = 2.1 (0.4) **Notes:** None of the daily reported respiratory symptoms had significant direct correlations with daily PM_{10} levels, according to the authors.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Rabinovitch et al. (2004) **Periods of Study:** 11/15/1999–3/15/2000 11/13/2000–3/23/2001 11/15/2001–3/22/2002 **Location:** Denver, Colorado	**Outcome:** Respiratory symptoms, Asthma symptoms (cough and wheeze), Upper respiratory symptoms **Study Design:** Time-series **Statistical Analyses:** Logistic linear regression **Age Groups:** 6-12	**Pollutants:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 28.1 (13.2) **Range (Min, Max):** (6.0, 102.0) **Copollutant:** CO NO_2 SO_2 O_3	**Increment:** 1 µg/m^3 β (SE) AM: -0.010 (0.008); PM: -0.011 (0.010) **Odds Ratio (Lower CI, Upper CI); Lag** 1.016 (0.911, 1.133); 0-3 avg. OR for respiratory symptoms and PM_{10} exposure for children age 6-12 Asthma exacerbation: 1.00 (0.75, 1.25); 0-3 avg Medication: 0.85 (0.75, 0.95); 0-3 avg Previous night's symptoms: 1.10 (1.00, 1.20); 0-3 avg Current day's symptoms: 1.00 (0.90, 1.10); 0-3 avg **% Increase (Lower CI, Upper CI); Lag** % Increase in FEV_1 or PEF and PM_{10} exposure for children age 6-12 AM FEV_1: -0.01 (-0.02, 0.01); 0-3 avg; PM FEV_1: -0.02 (-0.03, 0.02); 0-3 avg; AM PEF: -0.025 (-0.035, 0.02); 0-3 avg; PM PEF: 0.00 (-0.03, 0.03); 0-3 avg.
Reference: Rojas-Martinez et al. (2007) **Period of Study:** 1996-1999 **Location:** Mexico City, Mexico	**Outcome:** Lung function: FEV_1, FVC, $FEF_{25-75\%}$ **Age Groups:** Children 8 years old at time of cohort recruitment **Study Design:** school-based "dynamic" cohort study **N:** 3170 children; 14,545 observations **Statistical Analyses:** Three-level generalized linear mixed models with unstructured variance-covariance matrix **Covariates:** age, body mass index, height, height by age, weekday spent outdoors, environmental tobacco smoke, previous-day mean air pollutant concentration, time since first test **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-1 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h, 6-mo **Mean (SD):** 24-h averaging Tlalnepantla: 66.7 (35.6) Xalostoc: 96.7 (49.4) Merced: 79.3 (40.8) Pedregal: 53.4 (31.9) Cerro de la Estrella: 69.6 (35.3) 6-mo averaging **Mean:** 75.6 **Percentiles:** 6-mo averaging 25th: 55.8 50th(Median): 67.5 75th: 92.2 **Monitoring Stations:** 5 sites for PM_{10}, 10 for other pollutants **Copollutant:** O_3, NO_2	**PM Increment:** IQR; PM_{10}, 6-LC: 36.4 **GIRLS** **One-pollutant model** FVC: -39 [-47: -31]; FEV: -29 [-36: -21]; $FEF_{25-75\%}$: -17 [-36: 1]; FEV_1/FVC: 0.12 [0.07: 0.17] **Two-pollutant model** PM_{10}, 6-LC & O_3 FVC: -30 [-39: -22]; FEV: -24 [-31: -16]; $FEF_{25-75\%}$: -9 [-26: 9]; FEV_1/FVC: 0.10 [0.06: 0.15] PM_{10}, 6-LC & NO_2 FVC: -21 [-30: -13]; FEV: -17 [-25: -8]; $FEF_{25-75\%}$: -23 [-43: -4]; FEV_1/FVC: 0.07 [0.02: 0.13] **Multipollutant model** PM_{10}, 6-LC, O_3, & NO_2 FVC: -14 [-23: -5]; FEV: -11 [-20: -3]; $FEF_{25-75\%}$: -7 [-27: 12]; FEV_1/FVC: 0.08 [0.03: 0.13] **BOYS** **One-pollutant model** FVC: -33 [-41: -25]; FEV: -27 [-34: -19]; $FEF_{25-75\%}$: -18 [-34: -2]; FEV_1/FVC: 0.04 [-0.01: 0.09] **Two-pollutant model** PM_{10}, 6-LC & O_3 FVC: -28 [-36: -19]; FEV: -22 [-30: -15]; $FEF_{25-75\%}$: -10 [-27: 7]; FEV_1/FVC: 0.04 [-0.01: 0.09] PM_{10}, 6-LC & NO_2 FVC: -16 [-26: -7]; FEV: -19 [-27: -10]; $FEF_{25-75\%}$: -26 [-44: -9]; FEV_1/FVC: 0.005 [-0.06: 0.05] **Multipollutant model** PM_{10}, 6-LC, O_3, & NO_2 FVC: -12 [-22: -3]; FEV: -15 [-23: -6]; $FEF_{25-75\%}$: -12 [-30: 6]; FEV_1/FVC: -0.002 [-0.06: 0.05] Long-term exposure to O_3, PM_{10}, and NO_2 is associated with decrements in FVC and FEV_1 growth in Mexico City schoolchildren. In a multipollutant model, PM_{10} (-12%), O_3 (-9%), and NO_2 (-41%) each contribute independently and statistically significantly to diminished FVC growth. For FEV_1, however, the multipollutant model indicates that only PM_{10} (-15%) and NO_2 (-25%) each contribute independently and statistically significantly to diminished FEV_1 growth.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Sanchez-Carrillo et al. (2003) **Period of Study:** 1996-1997 **Location:** metropolitan Mexico City, Mexico	**Outcome:** Upper respiratory symptom indicator (wet cough, sore throat, hoarseness, nose dryness, and head cold); Lower respiratory symptom indicator (dry cough, lack of air, and chest sounds); and Ocular symptom indicator (eye irritation, eye itch, eye burning, teary eyes, red eyes, and eye infection) **Age Groups:** All ages **Study Design:** Cohort **N:** 151,418 interviews **Statistical Analyses:** Logistic regression models **Covariates:** sex, age, education, cigarette smoking, season, emergency episode mass media report, temperature, and relative humidity **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** 1	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Northeast: 132 (52) Northwest: 87 (46) Central: 85 (37) Southeast: 79 (35) Southwest: 55 (28) **Range (Min, Max):** Northeast: (34-269) Northwest: (10-275) Central: (9-319) Southeast: (14-225) Southwest: (12-264) **Monitoring Stations**: Up to 32 **Copollutant (correlation):** O_3: r = 0.067 O_3 8: 00-18: 00 h: r = 0.075 SO_2: r = 0.265 NO_2: r = 0.265	**Effect Estimate [Lower CI, Upper CI]:** **PM_{10} quartiles**10.04-52.62 (ref) 52.63-73.58 Upper respiratory indicator: 1.02 (0.99-1.06) Lower respiratory indicator: 1.04 (0.99-1.09) Ocular indicator: 0.99 (0.95-1.03) 73.59-101.91 Upper respiratory indicator: 1.07 (1.03-1.10) Lower respiratory indicator: 1.09 (1.04-1.14) Ocular indicator: 0.89 (0.86-0.92)101.92-318.80 Upper respiratory indicator: 0.93 (0.90-0.97) Lower respiratory indicator: 1.03 (0.98-1.08) Ocular indicator: 0.84 (0.81-0.87) **Northeast -** 2nd quartile Upper respiratory indicator: 0.354 (0.112-1.222) Lower respiratory indicator: 0.215 (0.040-1.160) Ocular indicator: 1.080 (0.915-1.274) 3rd quartile Upper respiratory indicator: 0.118 (0.039-0.356) Lower respiratory indicator: 0.126 (0.023-0.690) Ocular indicator: 1.228 (0.720-2.095) 4th quartile Upper respiratory indicator: 0.095 (0.034-0.267) Lower respiratory indicator: 0.119 (0.026-0.549) Ocular indicator: 0.878 (0.619-1.246) **Northwest -** 2nd quartile Upper respiratory indicator: 0.990 (0.898-1.090) Lower respiratory indicator: 1.246 (1.087-1.429) Ocular indicator: 1.218 (0.808-1.834) 3rd quartile Upper respiratory indicator: 1.133 (0.974-1.317) Lower respiratory indicator: 1.202 (1.044-1.385) Ocular indicator: 0.345 (0.125-0.951) 4th quartile Upper respiratory indicator: 1.019 (0.904-1.149) Lower respiratory indicator: 1.344 (1.137-1.589) Ocular indicator: 1.949 (1.416-2.683) **Central -** 2nd quartile Upper respiratory indicator: 1.088 (1.002-1.183) Lower respiratory indicator: 1.046 (0.930-1.176) Ocular indicator: 1.220 (1.115-1.335) 3rd quartile Upper respiratory indicator: 1.054 (0.977-1.137) Lower respiratory indicator: 1.055 (0.948-1.175) Ocular indicator: 1.049 (0.965-1.142) 4th quartile Upper respiratory indicator: 0.899 (0.826-0.979) Lower respiratory indicator: 0.952 (0.845-1.073) Ocular indicator: 0.875 (0.796-0.963) **Southeast -** 2nd quartile Upper respiratory indicator: 0.778 (0.575-1.052) Lower respiratory indicator: 1.047 (0.916-1.196) Ocular indicator: 0.460 (0.299-0.708) 3rd quartile Upper respiratory indicator: 1.297 (1.127-1.491) Lower respiratory indicator: 1.391 (1.131-1.711) Ocular indicator: 0.474 (0.314-0.715) 4th quartile Upper respiratory indicator: 0.893 (0.812-0.983) Lower respiratory indicator: 0.937 (0.818-1.073) Ocular indicator: 0.314 (0.182-0.542) **Southwest -** 2nd quartile Upper respiratory indicator: 0.987 (0.913-1.066) Lower respiratory indicator: 2.181 (1.177-4.040) Ocular indicator: 1.026 (0.928-1.135) 3rd quartile Upper respiratory indicator: 0.673 (0.673-1.886) Lower respiratory indicator: 0.899 (0.790-1.024) Ocular indicator: 1.017 (0.862-1.200) 4th quartile Upper respiratory indicator: 0.524 (0.524-1.787) Lower respiratory indicator: 4.346 (0.917-20.606) Ocular indicator: 0.187 (0.090-0.387)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Schildcrout et al. (2006) **Period of Study:** November 1993 to September 1995 **Location:** Albuquerque, New Mexico; Baltimore, Maryland; Boston, Massachusetts; Denver, Colorado; San Diego, California; Seattle, Washington; St. Louis, Missouri; Toronto, Ontario, Canada	**Outcome:** Asthma Symptoms, Rescue Inhaler Uses **Age Groups:** 5 to 12 year olds **Study Design:** Meta-analysis of CAMP **N:** 990 children **Statistical Analyses:** "Working independence covariance structure" Logistic Regression Poisson Regression "GEE Procedure" **Covariates:** Season, age, race-ethnicity, annual family income, day of the week **Dose-response Investigated?** **Statistical Package:** SAS 8.2; R **Lags Considered:** 0 day lag, 1 day lag, 2 day lag, 3-day moving sum	**Pollutant:** PM_{10} **Averaging Time:** 24-h averages Seattle: Daily Albuquerque: Daily Baltimore: 50% of study days measured Boston: 23% of study days measured Denver: 37% of study days measured San Diego: 24% of study days measured St. Louis: 19% of study days measured Toronto: 47% of study days measured **Percentiles:** 10th: 6.8-14.0 25th: 12.0-22.4 50th(Median): 17.7-32.4 75th: 26.2-42.7 90th: 32.5-53.9 **Monitoring Stations:** 1-12 **Copollutant (correlation):** NO_2 r = 0.26-0.64 SO_2 r = 0.31-0.65 O_3 r = 0.03-0.73 CO r = 0.24-0.88	**PM Increment:** 25 µg/m^3 **One-pollutant Model** Asthma Symptoms: 1.02 [0.94, 1.11]; 0; 1.01 [0.97, 1.06]; 1; 1.02 [0.98, 1.07]; 2; 1.01 [0.98, 1.05]; 3-day moving sum Rescue Inhaler Uses: [0.97, 1.05]; 0; [0.97, 1.05]; 1; 1.00 [0.97, 1.03]; 2; 1.01 [0.98, 1.03]; 3-day moving sum **Two-pollutant Model** Asthma Symptoms: CO-PM_{10} 1.08 [1.01, 1.15]; 0; 1.06 [0.99, 1.14]; 1; 1.08 [1.02, 1.14]; 2; 1.05 [1.01, 1.08]; 3-day moving sum NO_2-PM_{10} 1.06 [0.99, 1.13]; 0; 1.04 [0.97, 1.11]; 1; 1.08 [1.02, 1.15]; 2; 1.04 [1.00, 1.07]; 3-day moving sum SO_2-PM_{10} 1.05 [0.98, 1.13];0; 1.04 [0.96, 1.14]; 1; 1.05 [0.98, 1.12]; 2; 1.04 [0.99, 1.08]; 3-day moving sum Rescue Inhaler Uses: CO-PM_{10} 1.06 [0.99, 1.13]; 0; 1.05 [0.99, 1.11]; 1; 1.05 [1.01, 1.09]; 2; 1.03 [1.00, 1.07]; 3-day moving sum NO_2-PM_{10} 1.03 [0.97, 1.08]; 0; 1.03 [0.98, 1.08]; 1; 1.04 [1.00, 1.09]; 2; 1.02 [1.00, 1.05]; 3-day moving sum SO_2-PM_{10} 1.01 [0.95, 1.07]; 0; 1.02 [0.97, 1.07]; 1; 1.03 [0.98, 1.09]; 2; 1.02 [0.98, 1.05]; 3-day moving sum

Table E-10. Short-term exposure to $PM_{10-2.5}$ and respiratory morbidity outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Aekplakorn et al. (2003) **Period of Study:** 107 days, from October 1, 1997 to January 15, 1998 **Location:** Mae Mo district, Lampang Province, north Thailand	**Outcome:** Upper respiratory symptoms, lower respiratory symptoms, cough **Age Groups**: 6-14 years old **Study Design:** Logistic regression **N:** 98 asthmatic school children **Statistical Analyses:** Generalized Estimating Equations, stratified analysis, PROC GENMOD **Covariates:** Temperature and relative humidity **Season**: Winter **Dose-response Investigated?** No **Statistical Package**: SAS v 8.1	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** daily **Mean (SD):** NR **Range (Min, Max):** NR **Monitoring Stations**: 3 **Copollutant**: PM_{10}, SO_2	**PM Increment:** 10 µg/m^3 Odds Ratios [Lower CI, Upper CI] ; lag: Asthmatics: URS: 1.04 (0.93, 1.17); lag 0 LRS: 1.09 (0.95, 1.26) ; lag 0 Cough: 1.08 (0.96, 1.21) ; lag 0 Non-Asthmatics: URS: 1.05 (0.99, 1.19); lag 0 LRS: 0.90 (0.72, 1.11) ; lag 0 Cough: 0.95 (0.81, 1.11) ; lag 0

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Bourotte et al. (2007) **Period of Study:** 13 May 2002, 19 July 2002 **Location:** Sao Paolo, Brazil	**Outcome:** Peak expiratory flow (PEF) **Age Groups**: Avg age 39.8 +/- 12.3 **Study Design:** Cross-sectional **N:** 33 patients **Statistical Analyses:** Linear mixed-effects model **Covariates:** Gender, Age, BMI, Air Pollutants, Ambient temperature, Relative Humidity **Season:** Winter **Dose-response Investigated?** No **Statistical Package**: S-plus **Lags Considered:** 2 day lag, 3 day lag	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 21.7 (12.9) µg/m^3 **Range (Min, Max):** (4.13, 6.20) **Components:** Na^+ K^+ Mg^{2+} Ca^{2+} F_{inf} Cl- NO_3^- SO_4^{2-} **Monitoring Stations**: 1	**PM Increment:** NR Effect [Lower CI, Upper CI] ; lag: Morning PEF Na^+ concurrent day = -0.454 (-1.605, 0.697) Na^+ 2-day lag = -0.907 (-2.288, 0.474) Na^+ 3-day lag = -1.361 (-2.972, 0.251) K^+ concurrent day = 1.685 (-0.492, 3.862) K^+ 2-day lag = 1.838 (-1.272, 4.984) K^+ 3-day lag = 2.604 (-0.812, 6.025) Mg^{2+} concurrent day = 2.265* (-0.427, 4.956) Mg^{2+} 2-day lag = 1.271 (-1.869, 4.410) Mg^{2+} 3-day lag = 0.939 (-2.425, 4.303) Ca^{2+} concurrent day = 5.491* (2.558, 8.424) Ca^{2+} 2-day lag = 6.358* (2.251, 10.465) Ca^{2+} 3-day lag = 6.069 (1.962, 10.176) F_{inf} concurrent day = 1.572 (-0.792, 3.935) F_{inf} 2-day lag = 1.630 (-1.679, 4.939) F_{inf} 3-day lag = 2.736* (-1.754, 7.226) Cl^- concurrent day = -0.951 (-2.238, 0.336) Cl^- 2-day lag = -1.871 (-3.242 to -0.4997) Cl^- 3-day lag = -2.286* (-3.934 to -0.638)\ NO_3^- concurrent day = 4.195* (-0.063, 8.452) NO_3^- 2-day lag = 6.292* (2.034, 10.55) NO_3^- 3-day lag = 7.341* (3.083, 11.60) SO_4^{2-} concurrent day = 3.528 (-0.053, 7.110) SO_4^{2-} 2-day lag = 4.411* (0.829, 7.991)| SO_4^{2-} 3-day lag = 6.175* (2.593, 9.756) Evening PEF Na^+ concurrent day = -0.680 (-1.831, 0.471) Na^+ 2-day lag = -1.90 (-3.316 to -0.494) Na^+ 3-day lag = -2.336* (-3.878 to -0.794) K^+ concurrent day = 0.613 (-1.564, 2.790) K^+ 2-day lag = 0.613 (-2.497, 3.723) K^+ 3-day lag = 0.000 (-3.421, 3.421)| Mg^{2+} concurrent day = -0.718 (-3.522, 2.085) Mg^{2+} 2-day lag = -1.933 (-5.073, 1.206) Mg^{2+} 3-day lag = -3.591 (-7.056 to -0.126) Ca^{2+} concurrent day = 2.312* (-1.208, 5.832) Ca^{2+} 2-day lag = 2.023 (-2.084, 6.130) Ca^{2+} 3-day lag = 0.578 (-3.530, 4.685) F_{inf} concurrent day = -1.281 (-3.644, 1.083) F_{inf} 2-day lag = -2.503 (-5.930, 0.924) F_{inf} 3-day lag = -4.540 (-9.149, 0.068) Cl^- concurrent day = -0.317 (-1.604, 0.970) Cl^- 2-day lag = -1.268 (-2.556, 0.019) Cl^- 3-day lag = -1.902 (-3.589 to -0.216) NO_3^- concurrent day = 3.146 (-1.112, 7.404) NO_3^- 2-day lag = 3.146 (-1.112, 7.404) NO_3^- 3-day lag = 1.049 (-3.209, 5.306) SO_4^{2-} concurrent day = 1.764 (-1.817, 5.346) SO_4^{2-} 2-day lag = 2.646 (-0.935, 6.228) SO_4^{2-} 3-day lag = 1.764 (-1.817, 5.346)
Reference: Ebelt et al. (2005) Period of Study: Summer of 1998 Location: Vancouver, Canada	**Outcome:** Adverse health effects: spirometry, systolic/diastolic blood pressure measurements, symptom questionnaires, arrhythmia, heart rate, and heart rate variability (from electrocardiogram) **Age Groups**: range from 54-86 yrs; mean age= 74 years **Study Design:** extended analysis of a repeated-measures panel study **N:** 16 persons with COPD **Statistical Analyses:** Earlier analysis expanded by developing mixed-effect regression models and by evaluating additional exposure indicators **Dose-response Investigated?** No **Statistical Package**: SAS V8	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (SD):** Ambient PM_{10}-2.5: 5.6 (3.0) Exposure to ambient PM_{10}-2.5: 2.4 (1.7) **Range (Min, Max):** Ambient $PM_{10-2.5}$: (-1.2-11.9) Exposure to ambient $PM_{10-2.5}$: (-0.4-7.2) **Monitoring Stations**: 5 **Copollutant (correlation):** Ambient PM_{10}: r= 0.69 Ambient $PM_{2.5}$: r= 0.15 Nonsulfate Ambient $PM_{2.5}$: r= 0.14 Exposure to Ambient $PM_{10-2.5}$: r= 0.73	**PM Increment:** Ambient PM_{10}-2.5: 4.5 (IQR) Exposure to ambient PM_{10}-2.5: 2.4 (IQR) Notes: Effect estimates are presented in Figure 2 and Electronic Appendix Table 1 (only available with electronic version of article) and not provided quantitatively elsewhere.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lagorio et al.(2006) **Period of Study:** 5/24/1999 to 6/24/1999 and 11/181999 to 12/22/1999 **Location:** Rome, Italy	**Outcome:** Lung function of subjects (FVC and FEV_1) with COPD, Asthma **Age Groups**: COPD 50 to 80 yrs Asthma 18 to 64 yrs **Study Design:** Time series **N:** COPD N = 11; Asthma N = 11 **Statistical Analyses:** Non-parametric Spearman correlation; GEE; **Covariates:** COPD: daily mean temperature, season variable (spring or winter), relative humidity, day of week; Asthma: season variable, temperature, humidity, and -2-agonist use **Season:** Spring and winter **Dose-response Investigated?** Yes **Statistical Package**: STATA **Lags Considered:** 1–3 days	**PM Size:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (SD):** Overall: 15.6 (7.2) Spring:18.7 (7.4) Winter: 12.3 (5.4) **Range (Min, Max):** (3.4, 39.6) **PM Component:** Cd: 0.46±0.40 ng/m^3 Cr: 1.9±1.7 ng/m^3 Fe:283±167 ng/m^3 Ni:4.8±6.5 ng/m^3 Pb:30.6±19.0 ng/m^3 Pt:5.0±8.6 pg/m^3 V:1.8±1.4 ng/m^3 Zn:45.8±33.1 ng/m^3 **Monitoring Stations**: Two fixed sites:(Villa Ada and Istituto superior di Sanita) **Copollutant (correlation):** NO_2 r = 0.51 O_3 r = 0.31 CO r = -0.09 SO_2 r = -0.16 PM_{10} r = 0.61 $PM_{2.5}$ r = 0.34	**PM Increment:** 1 µg/m^3 They observed no statistically significant effect of $PM_{10-2.5}$ on FVC and FEV_1 on any of the panels (COPD, Asthma). β Coefficient (SE) COPD FVC(%) 24 h -1.32 (1.06) 48-h -1.46 (1.31) 72-h -1.38 (1.53) FEV_1(%) 24 h -0.59 (0.95) 48-h -1.01 (1.19) 72-h -0.90 (1.42) Asthma FVC(%) 24 h -0.17 (0.75) 48-h -0.36 (0.91) 72-h -0.24 (1.07) FEV_1(%) 24 h -0.67 (0.89) 48-h -1.19 (1.07) 72-h -0.51 (1.26)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Mar et al. (2004) **Period of Study:** 1997-1999 **Location:** Spokane, Washington	**Outcome:** Respiratory symptoms **Age Groups**: Adults: Ages 20-51 yrs; Children: Ages 7-12 yrs **Study Design:** Time-series **N:** 25 people **Statistical Analyses:** Logistic regression **Covariates:** Temperature, relative humidity, day of-the-wk **Statistical Package**: STATA 6 **Lags Considered:** 0-2 days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h **Monitoring Stations**: 1 station **Copollutant (correlation):** PM1; r = 0.16 $PM_{2.5}$; r = 0.28 PM_{10}; r = 0.93	**PM Increment:** 10 µg/m^3 OR Estimate [Lower CI, Upper CI] ; lag: Adult Respiratory symptoms: **Wheeze:** 1.01[0.92, 1.10]; lag 0; 0.97[0.89, 1.07]; lag 1 0.99[0.90, 1.09]; lag 2 **Breath:** 1.03[0.95, 1.12]; lag 0; 1.02[0.95, 1.10]; lag 1 1.03[0.95, 1.09]; lag 2 **Cough:** 0.99[0.92, 1.06]; lag 0; 0.99[0.93, 1.05]; lag 1 1.00[0.95, 1.06]; lag 2 **Sputum:** 1.04[0.96, 1.13]; lag 0; 1.01[0.94, 1.08]; lag 1 1.02[0.95, 1.08]; lag 2 **Runny Nose:** 0.98[0.91, 1.04]; lag 0; 0.97[0.91, 1.03]; lag 1 0.98[0.93, 1.03]; lag 2 **Eye Irritation:** 0.97[0.87, 1.08]; lag 0; 0.98[0.89, 1.07]; lag 1 0.99[0.93, 1.05]; lag 2 **Lower Symptoms:** 0.97[0.91, 1.03]; lag 0; 0.95[0.89, 1.01]; lag 1 0.96[0.91, 1.01]; lag 2 **Any Symptoms:** 0.90[0.76, 1.06]; lag 0; 0.96[0.91, 1.02]; lag 1 0.96[0.91, 1.01]; lag 2 **Children Respiratory symptoms: Wheeze:** 1.12[0.98, 1.28]; lag 0; 0.98[0.78, 1.24]; lag 1 1.08[0.88, 1.33]; lag 2 **Breath:** 1.03[0.93, 1.13]; lag 0; 1.05[0.97, 1.14]; lag 1 1.08[1.00, 1.17]; lag 2 **Cough:** 1.07[0.96, 1.20]; lag 0; 1.06[1.02, 1.10]; lag 1 1.10[1.02, 1.18]; lag 2 **Sputum:** 1.13[1.00, 1.28]; lag 0; 1.10[0.99, 1.22]; lag 1 1.10[0.99, 1.23]; lag 2 **Runny Nose:** 1.13[1.06, 1.20]; lag 0; 1.1[1.07, 1.15]; lag 1 1.11[1.06, 1.17]; lag 2 **Eye Irritation:** 1.12[0.73, 1.73]; lag 0; 0.99[0.74, 1.32]; lag 1 1.06[0.84, 1.34]; lag 2 **Lower Symptoms:** 1.04[0.93, 1.17]; lag 0; 1.05[0.95, 1.15]; lag 1 1.06[0.94, 1.20]; lag 2 Any Symptoms: 1.05[0.95, 1.16]; lag 0 1.07[1.00, 1.15]; lag 1 1.10[1.03, 1.18]; lag 2

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Tang et al. (2007) **Period of Study:** Dec 2003 to Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups**: 6–12 years **Study Design:** Panel study **N:** 30 children **Statistical Analyses:** Linear mixed-effect models were used to estimate the effect of PM exposure on PEFR **Covariates:** Gender, age, BMI, history of respiratory or atopic disease in family, SHS, acute asthmatic exacerbation in past 12 months, ambient temperature and relative humidity, presence of indoor pollutants, and presence of outdoor pollutants, **Dose-response Investigated?** yes **Statistical Package**: S-Plus 2000 **Lags Considered:** 0-2	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 1 h **Mean (SD):** Personal: 17.8 (19.6) Ambient: 17.0 (10.6) **Range (Min, Max):** Personal: 0.3–195.7 Ambient: 0.1–80.2 **Monitoring Stations**: 1	**PM Increment:** 15.9 µg/m^3 RR Estimate [Lower CI, Upper CI] ; lag: Change in morning PEFR: -20.55 (-45.83, 4.73) lag 0 -39.05 (-104.16 , 26.06) lag 1 -39.56 (-79.56, 0.44) lag 2 -37.15 (-105.01, 30.7) 2-day mean -35.47 (-27.32, 56.38) 3-day mean Change in evening PEFR: -1.68 (-19.13, 15.78) lag 0 1.59 (-14.32, 17.5) lag 1 0.86 (-30.84, 32.57) lag 2 5.97 (-15.57, 27.5) 2-day mean 29.75 (-1.69, 61.18) 3-day mean
Reference: Trenga et al., (2006) **Period of Study:** 1999-2002 **Location:** Seattle, WA	**Outcome**: Lung function: FEV_1, PEF, MMEF (maximal midexpiratory flow; assessed only for children) **Age Groups**: Adults (56-89-years-old) healthy & with COPD; asthmatic children 6-13-years-old **Study Design:** adult and pediatric panel study over three years with 1 monitoring period ("session") per year **N:** 57 adults (33 healthy, 24 with COPD) = 692 subject-days = 207 study-days; 17 asthmatic children = 319 subject-days = 98 study-days **Statistical Analyses:** mixed effects, longitudinal regression models, with the effects of pollutant decomposed into each subject's a) overall mean; b) difference between their session-specific mean and overall mean; c) difference between their daily values and session-specific mean **Covariates:** gender, age, ventral site temperature and relative humidity, CO, NO_2 **Season:** NR **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** 0-1 days	**Pollutant:** $PM_{10-2.5}$ (coarse) **Averaging Time:** 24-h **Percentiles:** Subject-specific exposure PM_{10}-$PM_{2.5}$ Outdoor 25th: 3.3 50th (Median): 4.7 75th: 6.9 Adults Outdoor 25th: 3.3 50th (Median): 5.0 75th: 7.1 **Range (Min, Max):** Subject-specific exposure Children Outdoor (0.0, 25.3) Adults Outdoor (0.0, 25.7) **Monitoring Stations**: 2; also subject-specific local outdoors (i.e., at each home), indoor, and personal **Copollutant (correlation):** CO NO_2 $PM_{2.5}$	**PM Increment:** 10 µg/m^3 **Adult** Outdoor Home PM_{10}-$PM_{2.5}$ FEV_1 Overall: Lag 0 -27.9 [-87.5:31.8]; Lag 1 47.1 [-5.1:99.4] No-COPD: Lag 0 -49.2 [-22.3:23.9]; Lag 1 74.3 [6.8:141.8] COPD: Lag 0 7.3 [-84.7:99.4]; Lag 1 11.5 [-65.4:88.3] PEF Overall: Lag 0 5.3 [-5.1:15.7]; Lag 1 -2.5 [-11.6:6.5] No-COPD: Lag 0 5.1 [-7.7:17.8]; Lag 1 -5.8 [-17.5:5.9] COPD: Lag 0 5.7 [-10.3:21.6]; Lag 1 1.7 [-11.5:14.9] **Pediatric** FEV_1 Outdoor Home PM_{10}-$PM_{2.5}$ Overall Lag 0 -7.43 [-69.41:54.55]; Lag 1 -25.61 [-88.16:36.94] No Anti-inflam. Medication Lag 0 -63.87 [-199.58:71.84]; Lag 1 -96.48 [-232.48:39.52] Anti-inflam. Medication Lag 0 6.57 [-96.90:110.04]; Lag 1 -8.63 [-217.39:200.14] PEF Outdoor Home PM_{10}-$PM_{2.5}$ Overall Lag 0 4.53 [-6.60:15.67]; Lag 1 -3.35 [-14.31:7.62] No Anti-inflam. Medication Lag 0 2.05 [-22.36:26.45]; Lag 1 -6.56 [-30.90:17.78] Anti-inflam. Medication Lag 0 5.15 [-7.90:18.19]; Lag 1 -2.58 [-15.35:10.19] MMEF Outdoor Home PM_{10}-$PM_{2.5}$ Overall Lag 0 -0.01 [-7.29:7.28]; Lag 1 -2.07 [-9.25:5.12] No Anti-inflam. Medication Lag 0-7.14 [-23.16:8.87]; Lag 1 -14.39 [-30.11:1.32] Anti-inflam. Medication Lag 0 1.76 [-6.78:10.30]; Lag 1 0.89 [-7.56:9.33]

Table E-11. Short-term exposure to $PM_{2.5}$ (including components/sources) and respiratory morbidity outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Adamkiewicz et al. (2004) **Period of Study:** August–December 2000 **Location:** Steubenville, Ohio	**Outcome:** FENO **Age Groups**: ranged 53.5-90.6 years **Study Design:** prospective cohort **N:** total of 294 breaths from 29 subjects **Statistical Analyses:** Fixed effect models, ANOVA, GLM procedure **Covariates:** Subject, week of study, day of the week, h of the day, ambient barometric pressure, temperature, and relative humidity **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** Hourly lags, 0-48 h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h **Mean (SD):** 19.5 **Percentiles:** 25th: 7.6 75th: 25.5 **Range (Min, Max):** NR, 105.8 **Monitoring Stations**: 1 **Averaging Time:** 24 h **Mean (SD):** 19.7 **Percentiles:** 25th: 9.7 75th: 27.4 **Range (Min, Max):** NR, 57.8 **Monitoring Stations**: 1 **Copollutant (correlation):** Ambient NO; Indoor NO; NO_2; O_3; SO_2	**PM Increment:** 17.9 µg/m^3 Effect Estimate [Lower CI, Upper CI]: 1-h Single pollutant models:0.36 (0.58-2.14) **PM Increment:** 17.7 Effect Estimate [Lower CI, Upper CI]: 24 h moving avg: 1.45 (0.33-2.57) Multipollutant models for $PM_{2.5}$, ambient NO and room NO and estimated change in FENO (ppb) for an IQR in pollutant measure Model 1 1.95 (0.47-3.43) Model 2 1.38 (0.26-2.51) Model 4 1.97 (0.48-3.46) Notes: Association of FENO with $PM_{2.5}$ at different lags presented in Figure 1 are not presented quantitatively elsewhere.
Reference: Adar et al. (2007) **Period of Study:** March-June 2002 **Location:** St. Louis, MO	**Outcome:** FENO **Age Groups**: 60+ **Study Design:** Panel Study **N:** 44 non-smoking seniors **Statistical Analyses:** mixed models containing random subject effects **Covariates:** Day of week, trip type, FENO collection device, current illness, use of vitamins, antihistamines, statins, steroids, and asthma medications, temperature, pollen, mold, NO concentration in testing room **Statistical Package**: SAS **Lags Considered:** 0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Pretrip: 14.8; Post-trip: 16.5 **Percentiles:** 25th (pretrip): 11.2 75th (pretrip): 20.1 25th (post-trip): 11.7 75th (post-trip): 21.6 **Monitoring Stations**: 1 **Copollutant (correlation):** BC; CO; NO_2; SO_2 ; O_3	**PM Increment:** 9.8 µg/m^3 Effect Estimate [Lower CI, Upper CI]: Pre-trip % change: 21.9 (6.7, 39.4) Post-trip % change: -4.7 (-17.1, 9.6)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Aekplakorn et al (2003) **Period of Study:** 107 days, from October 1, 1997 to January 15, 1998 **Location:** Mae Mo district, Lampang Province, north Thailand	**Outcome:** Upper respiratory symptoms, lower respiratory symptoms, cough **Age Groups**: 6-14 years old **Study Design:** Logistic regression **N:** 98 asthmatic school children **Statistical Analyses:** Generalized Estimating Equations, stratified analysis, PROC GENMOD **Covariates:** Temperature and relative humidity **Season:** Winter **Dose-response Investigated?** No **Statistical Package**: SAS v 8.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD):** Sob Pad station: 24.77 Sob Mo station: 24.89 Hua Fai station: 26.27 **Range (Min, Max):** Sob Pad: 4.52, 24.77 Sob Mo: 3.13, 24.89 Hua Fai: 3.67, 26.27 **Monitoring Stations**: 3 **Copollutant:** PM_{10} SO_2	**PM Increment:** 10 µg/m^3 **Odds Ratios [Lower CI, Upper CI] ; lag:** Asthmatics: URS: 1.04 (0.99, 1.09); lag 0 LRS: 1.05 (0.98, 1.2) ; lag 0 Cough: 1.05 (0.99, 1.10) ; lag 0 Non-Asthmatics: URS: 1.03 (0.96, 1.09); lag 0 LRS: 1.02 (0.93, 1.10) ; lag 0 Cough: 1.00 (0.93, 1.07) ; lag 0 PM_{10} + SO_2 Asthmatics: URS: 1.04 (0.99, 1.10); lag 0 LRS: 1.05 (0.98, 1.10) ; lag 0 Cough: 1.05 (0.99, 1.11) ; lag 0 Non-Asthmatics: URS: 1.03 (0.97, 1.09); lag 0 LRS: 1.02 (0.93, 1.11) ; lag 0 Cough: 1.00 (0.93, 1.07) ; lag 0
Reference: Allen et al .(2008) **Period of Study:** 1999-2002 (additional PM composition data collected Dec 2000 and May 2001) **Location:** Seattle, USA	**Outcome:** daily changes in exhaled nitric oxide (FENO) and 4 lung function measures, midexpiratory flow (MEF), peak expiratory flow (PEF), forced expiratory volume in one second (FEV_1), and forced vital capacity (FVC) Age Groups: 6-13 yrs **Study Design:** Panel study **N:** 19 children with asthma **Statistical Analyses:** linear mixed effects model with random intercept to test for within participant associations **Covariates:** Tmperature, relative humidity, BMI, age, and, in the case of FENO, ambient NO measured at a centrally located monitoring site; models also included a term for within-participant, within-session effects, and a term for participant between-session effects **Effect modification**: Dcided a priori to include interaction term for $PM_{2.5}$ exposure and inhaled corticosteroids	**Pollutant:** $PM_{2.5}$ **Mean (SD):** 11.23 (6.48) **Range (Min, Max):** 2.76-40.38 25th: 6.38 75th: 14.73 **Copollutant (correlation):** Ambient LAC* r=0.83 Ambient LG**r=0.84 Personal $PM_{2.5}$: r=0.34 Personal LAC: r=0.54 Ambient-generated $PM_{2.5}$: r=0.87 Nonambient-generated $PM_{2.5}$: r=-0.06 * LAC Light-absorbing carbon ** LG: Leroglucosan (a marker of woodsmoke)	Health effect estimates presented in graphic form (Fig 1). Summary from text is as follows: Personal LAC, personal $PM_{2.5}$, and ambient-generated $PM_{2.5}$ were associated with ($p<0.05$) and ambient $PM_{2.5}$ was marginally associated ($p=0.09$) with increased FENO. Neither of the ambient combustion markers (LAC, LG) nor nonambient-generated $PM_{2.5}$ was associated with FENO changes. All of the ambient concentrations were associated with decrements in PEF and MEF while ambient-generated $PM_{2.5}$ was marginally associated ($p<0.10$). Only ambient LG was associated with a decrease in FEV_1 and there were no associations between exposure metrics and FVC.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Barraza-Villarreal et al.(2008) **Period of Study:** 6/2003–6/2005 **Location:** Mexico City	**Outcome:** Respiratory Symptoms, Coughing, Wheezing, Airway inflammation, Asthma **Study Design:** Prospective cohort **Statistical Analyses:** Bivarate analysis **Age Groups**: 6-14	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Maximum 8-h avg **Mean (SD) unit:** 28.9 (2.8) **Range (Min, Max):** (4.2, 102.8) **Copollutants (correlation):** O_3 NO_2	**Increment:** 17.5 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** **Athmatic children** Inflammatory Marker: FENO: 1.08 (1.01, 1.16); 0; IL-8: 1.08 (0.98, 1.19); 0; ph_EBC: -0.03 (-0.09, 0.03); 0 Lung Function: FEV_1: -16.0 (-31.0 to -0.13); 0-4 avg FVC: -23.0 (-42.0 to -5.21); 0-4 avg FEV_{25-75}: -11.0 (-42.0, 20.3); 0-4 avg **Nonasthmatic children** Inflammatory Marker: FENO: 0.89 (0.78, 1.01); 0; IL-8: 1.16 (1.00, 1.36); 0; ph_EBC: -0.05 (-0.14, 0.04); 0 Lung Function: FEV_1: -21.0 (-42.3, 0.38); 0-4 avg FVC: -29.0 (-52.8 to -4.35); 0-4 avg FEV_{25-75}: -20.0 (-69.0, 29.0); 0-4 avg **All children age 6-14** Respiratory Symptom: Cough: 1.11 (1.06, 1.17); Wheezing: 1.06 (0.99, 1.13)
Reference: Bennett et al. (2007) **Period of Study:** 1992-2005 **Location:** Melbourne, Australia	**Outcome:** Adverse respiratory symptoms (wheeze, shortness of breath on waking, cough in the morning, phlegm in the morning, cough with phlegm in the morning, asthma attack) **Age Groups**: All ages with a mean of 37.2 yrs **Study Design:** cohort study **N:** 1446 persons **Statistical Analyses:** Logistic regression models **Covariates:** Age, gender, current smoking status, medication use (ß2-agonist and inhaled steroid), atopy **Dose-response Investigated?** No **Statistical Package**: STATA statistical software, version 9 (Statcorp, 2005)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 6.8 **Range (Min, Max):** (1.8-73.3) **Monitoring Stations**: 1	**PM Increment:** 1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Within-person (longitudinal effects) Wheeze: OR=1.08 (0.79-1.48) SOB on waking: OR=1.34 (0.84-2.16) Cough in the morning: OR=0.74 (0.47-1.15) Phlegm in the morning: OR=1.55 (0.95-2.53) Cough w/ phlegm morning: OR=1.28 (0.70-2.33) Asthma attack: OR=0.91 (0.55-1.49) Between-person (cross-sectional) effects Wheeze: OR=1.32 (0.82-2.10) SOB on waking: OR=1.29 (0.46-3.60) Cough in the morning: OR=0.21 (0.07-0.62) Phlegm in the morning: OR=0.49 (0.16-1.44) Cough w/ phlegm morning: OR=0.28 (0.08-0.97) Asthma attack: OR=0.52 (0.17-1.59)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Bourotte et al. (2007) **Period of Study:** 13 May 2002-19 July 2002 **Location:** Sao Paolo, Brazil	Outcome: Peak expiratory flow (PEF) **Age Groups**: Avg age 39.8 +/- 12.3 **Study Design:** Cross-sectional **N:** 33 patients **Statistical Analyses:** Linear mixed-effects model **Covariates:** Gender, Age, BMI, Air Pollutants, Ambient temperature, Relative Humidity **Season:** Winter **Dose-response Investigated?** No **Statistical Package**: S-plus **Lags Considered:** 2 day lag, 3 day lag	**Pollutant:** $PM_{2.5}$ (Fine) **Averaging Time:** 24 h **Mean (SD):** 11.9 (5.12) **Range (Min, Max):** (2.82, 26.6) **Components:** K^+ Mg^{2+} Ca^{2+} F_{inf} Cl- NO_3- SO_4^{2-} **Monitoring Stations**: 1	**PM Increment:** NR **Effect [Lower CI, Upper CI] ; lag:** **Morning PEF** Na^+ concurrent day = -0.409 (-2.485, 1.667) Na^+ 2-day lag = -0.818 (-4.139, 2.503) Na^+ 3-day lag = -0.205 (-4.356, 3.974) K^+ concurrent day = -0.211 (-2.778, 2.357) K^+ 2-day lag = -0.843 (-4.695, 3.008) K^+ 3-day lag = 0.843 (-4.292, 5.978) Mg^{2+} concurrent day = -1.750 (-5.302, 1.802) Mg^{2+} 2-day lag = -5.016 (-10.79, 0.762) Mg^{2+} 3-day lag = -3.850 (-10.15, 2.449) Ca^{2+} concurrent day = 3.192* (-0.599, 6.943) Ca^{2+} 2-day lag = 5.880 (1.105, 10.65) Ca^{2+} 3-day lag = 7.560* (2.103, 13.02) F_{inf} concurrent day = 2.218* (-0.033, 4.470) F_{inf} 2-day lag = 3.697* (1.446, 5.949) F_{inf} 3-day lag =4.067* (1.065, 7.069) Cl^- concurrent day = -1.010 (-3.469, 1.450) Cl^- 2-day lag = -1.615 (-5.714, 2.483) Cl^- 3-day lag = -1.615 (-6.534, 3.303) NO_3^- concurrent day = 3.144 (0.409, 5.878) NO_3^- 2-day lag = 3.593 (0.858, 6.328) NO_3^- 3-day lag = 4.491 (1.756, 7.226) SO_4^{2-}concurrent day = 2.210 (-0.032, 4.272) SO_4^{2-} 2-day lag = 3.180 (1.028, 5.332) SO_4^{2-} 3-day lag = 3.180 (1.028, 5.332) **Evening PEF** Na^+ concurrent day = -1.636 (-3.712, 0.440) Na^+ 2-day lag = -0.205 (-3.256, 3.117) Na^+ 3-day lag = -1.023 (-5.174, 3.129) K^+ concurrent day = -1.897 (-4.465, 0.670) K^+ 2-day lag = -1.686 (-5.966, 2.592) K^+ 3-day lag = -1.054 (-6.189, 4.081) Mg^{2+} concurrent day = -2.753 (-6.400, 0.894) Mg^{2+} 2-day lag = -2.567 (-8.534, 3.401) Mg^{2+} 3-day lag = -4.876 (-11.36, 1.612) Ca^{2+} concurrent day = 2.184 (-1.567, 5.935) Ca^{2+} 2-day lag = 5.040 (0.265, 9.815) Ca^{2+} 3-day lag = 5.040 (-0.417, 10.50) F_{inf} concurrent day = 1.479 (-0.773, 3.730) F_{inf} 2-day lag = 1.819 (-0.403, 4.100) F_{inf} 3-day lag = 2.958 (-0.044, 5.960) Cl^- concurrent day = -0.404 (-2.863, 2.055) Cl^- 2-day lag = 0.000 (-4.099, 4.099) Cl^- 3-day lag =0.202 (-4.716, 5.120) NO^{3-} concurrent day = 1.796 (-0.939, 4.531) NO^{3-} 2-day lag = 2.695 (-0.040, 5.430) NO^{3-} 3-day lag = 3.144 (0.409, 5.878) SO_4^{2-} concurrent day = 2.120 (-0.032, 4.272) SO_4^{2-} 2-day lag = 2.120 (-0.032, 4.272) SO_4^{2-} 3-day lag =2.120 (-0.032, 4.272)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: de Hartog et al. (2003) **Period of Study:** winter of 1998-1999 (in Amsterdam, from November 2, 1998 to June 18, 1999; in Erfurt, from October 12, 1998 to April 4, 1999; and in Helsinki, from November 2, 1998 to April 30, 1999.) **Location:** Amsterdam, the Netherlands; Erfurt, Germany; and Helsinki, Finland	**Outcome:** chest pain, chest pain at physical exertion, shortness of breath, feeling tired or weak, tripping or racing heart, cold hands or feet, cough, phlegm, being awakened by breathing problems, wheezing, and common cold or flu and fever **Age Groups**: ≥ 50 yrs **Study Design: C**ohort **N:** 131 subjects with history of coronary heart disease **Statistical Analyses:** Logistic regression **Covariates:** Ambient temperature, relative humidity, atmospheric pressure, incidence of influenza-like illness **Season**: Winter **Dose-response Investigated?** No **Statistical Package**: S-PLUS 2000 **Lags Considered:** 0, 1, 2, 3, and 5-day avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Amsterdam, the Netherlands: 20.0 Erfurt, Germany: 23.4 Helsinki, Finland: 12.8 **Range (Min, Max):** Amsterdam, the Netherlands: (3.8-82.2) Erfurt, Germany: (4.5-118.1) Helsinki, Finland: (3.1-39.8) Unit (i.e. µg/m³): µg/m³ **Monitoring Stations**: 1 **Copollutant:** PM_{10} $NC_{0.01-0.1}$ CO NO_2 SO_2	**PM Increment:** 10 µg/m³ **Effect Estimate [Lower CI, Upper CI]:** Association of air pollution and incidence of symptoms in three panels of elderly subjects **Lag 0** Chest pain w/ physical exertion: 1.04 (0.96-1.13) Shortness of breath: 1.04 (0.96-1.12) Awakened, breathing problems: NA Avoidance of activities: 1.04 (0.96-1.14) Phlegm: 1.03 (0.93-1.13) **Lag 1** Chest pain w/ physical exertion: 1.01 (0.93-1.09) Shortness of breath: 1.06 (0.99-1.14) Awakened, breathing problems: 1.09 (1.00-1.20) Avoidance of activities: 1.03 (0.95-1.12) Phlegm: 1.10 (1.01-1.19) **Lag 2** Chest pain w/ physical exertion: 0.98 (0.90-1.05) Shortness of breath: 1.05 (0.98-1.12) Awakened, breathing problems: 1.04 (0.95-1.14) Avoidance of activities: 1.05 (0.97-1.14) Phlegm: 1.08 (1.00-1.18) **Lag 3** Chest pain w/ physical exertion: 1.00 (0.93-1.08) Shortness of breath: 1.08 (1.01-1.15) Awakened, breathing problems: 0.99 (0.91-1.08) Avoidance of activities: 1.06 (0.98-1.14) Phlegm: 1.10 (1.01-1.19) **5-day** Chest pain w/ physical exertion: 1.02 (0.91-1.13) Shortness of breath: 1.12 (1.02-1.24) Awakened, breathing problems: 1.03 (0.90-1.18) Avoidance of activities: OR= 1.09 (0.97-1.22) Phlegm: OR= 1.16 (1.03-1.32)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Delfino et al. (2004) **Period of Study:** September–October 1999; April–June 2000 **Location:** Alpine, California	**Outcome**: FEV_1 **Age Groups**: 9-19 years old **Study Design:** Panel study **N:** 24 children **Statistical Analyses:** GLM; Akaike's information criterion and Bayesian information criterion **Covariates:** Day of week, Personal temperature and relative humidity, time of FEV_1 maneuver (morning, afternoon, or evening), Season (fall 1999 or spring 2000), As-needed medication use, Presence or absence of upper or lower respiratory infections **Season:** Spring, Fall **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** 0-4	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg 1-h max personal PM last 24 h **Mean (SD):**151.0 (12.03) 90th: 292.4 **Range (Min, Max):** (9.1, 996.8) Mean personal PM last 24 h **Mean (SD):** 37.9 (19.9) 90th: 65.1 **Range (Min, Max):** 3.9, 113.8 Home stationary-site PM 24-h Mean indoor $PM_{2.5}$ **Mean (SD):** 12.1 (5.4) 90th: 20.2 **Range (Min, Max):** 2.8, 35.3 24-h Mean outdoor $PM_{2.5}$ **Mean (SD):** 11.0 (5.4) 90th: 18.4 **Range (Min, Max):** 1.8, 31.0 Central outdoor stationary-site PM 24-h Mean $PM_{2.5}$ **Mean (SD):** 10.3 (5.6) 90th: 18.4 **Range (Min, Max):** 1.7, 29.1 **Copollutant (correlation):** 24-h Central HI $PM_{2.5}$ 8-h max O_3 = 0.24 8-h Max NO_2 = 0.73 8-h Max Personal PM = 0.38 24-h Mean Personal PM = 0.43 8-h Max TEOM PM_{10} = 0.71 24-h Mean TEOM PM_{10} = 0.78 24-h Central HI PM_{10} = 0.90 24-h Outdoor HI $PM_{2.5}$ = 0.89 24-h Outdoor HI PM_{10} = 0.72 24-h Indoor HI PM_{10} = 0.40 24-h Indoor HI $PM_{2.5}$ = 0.73	Results presented graphically;-Percent predicted FEV_1 was inversely associated with personal exposure to fine particles. - Inverse associations of FEV_1 with stationary-site indoor, outdoor and central-site gravimetric $PM_{2.5}$ and PM_{10}, and with hourly TEOM PM_{10}

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Delfino et al. (2006) **Period of Study:** Region 1: August to Mid December 2003. Region 2: July through November 2004 **Location:** Region 1: Riverside, CA. Region 2: Whittier, CA	**Outcome:** Fractional Concentration of Nitric Oxide in exhaled air (FENO) **Age Groups**: 9 through 18 **Study Design:** Longitudinal Panel Study **N:** 45 children; Riverside children; 32 Whittier children **Statistical Analyses:** Linear mixed-effects models; Two-stage hierarchical model ; Empirical Variograms; Fourth-order polynomial distributed lag mixed-effects model **Covariates:** Personal temperature, Personal Rel. Humid., 10-day exposure run, Respiratory infections, Region of study, Sex, Cumulative daily use of as-needed B-agonist inhalers **Dose-response Investigated?** No **Lags Considered:** 0, 1, 2, MA day	**Pollutant**: $PM_{2.5}$ **Personal Exposure Averaging Time:** 24 h **Riverside** **Mean (SD):** 32.78 (21.84) 50th(Median): 28.14 **Range (Min, Max):** 7.27, 98.43 **Whittier** **Mean (SD):** 36.2 (25.46) 50th(Median): 29.07 **Range (Min, Max):** 7.55, 197.05 **Personal Exposure Averaging Time:** 1 h **Riverside** **Mean (SD):** 97.94 (70.29) 50th(Median): 83.7 **Range (Min, Max):** 14.9, 431.8 **Whittier** **Mean (SD):** 93.63 (75.19) 50th(Median): 71.95 **Range (Min, Max):** 5.8, 572.9 **Personal Exposure Averaging Time:** 8 h **Riverside** **Mean (SD):**47.21 (30.9) 50th(Median): 38.5 **Range (Min, Max):** 8.9, 132.1 **Whittier** **Mean (SD):** 51.75 (36.88) 50th(Median): 40.15 **Range (Min, Max):** 8.7, 254.1 **Central Site Averaging Time:** 24 h **Riverside** **Mean (SD):**36.63 (23.46) 50th(Median): 29.26 **Range (Min, Max):** (9.52, 87.22) **Whittier** **Mean (SD):** 18 (12.14) 50th(Median): 16.3 **Range (Min, Max):** 2.7, 77.09 **Monitoring Stations**: 48 personal nephelometers; 2 central sites **Copollutant (correlation):** **Personal** 24-h personal $PM_{2.5}$ 1.00 24-h personal EC 0.18 24-h personal OC 0.15 24-h personal NO_2 0.33 24-h central $PM_{2.5}$ 0.64 24-h central EC 0.12 24-h central OC 0.21 24-h central NO_2 0.22 **Central** 24-h personal $PM_{2.5}$ 0.64 24-h personal EC 0.00 24-h personal OC -0.11 24-h personal NO_2 0.12 24-h central $PM_{2.5}$ 1.00 24-h central EC 0.55 24-h central OC 0.66 24-h central NO_2 0.25	**PM Increment:** IQR increase (Riverside: 28.41 µg/m^3, Whittier 21.87 µg/m^3) **Coefficient [Lower CI, Upper CI] ; lag:** Mixed-model estimates of the association between personal and central-site air pollutant exposure and FENO Lag 0 Personal 0.42 (-0.15, 0.99) Central 0.03 (-0.68, 0.74) Lag 1 Personal 0.51 (-0.10, 1.12) Central 0.44 (-0.28, 1.16) 2-day MA Personal 1.01 (0.14, 1.88) Central 0.52 (-0.43, 1.47) Stratified by Medication Use Lag = 2-day moving avg Not Taking Anti-Inflamm. Medication Personal 1.11 (-1.39, 3.60) Central 0.44 (-1.65, 2.53) Taking Anti-Inflamm. Medication Personal 1.01 (0.19, 1.84) Central 0.55 (-0.47, 1.57) Inhaled Corticosteroids Personal 1.58 (0.72, 2.43) Central 1.16 (0.11, 2.20) Antileukotrienes +- inhaled corticosteroids Personal -0.89 (-2.73, 0.95) Central -0.75 (-2.83, 1.32) Notes: Figure of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Figure of the Estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Figure of One- and two-pollutant models for change in FENO using 2-day Moving Averages personal and central-site pollutant measurements.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Delfino et al. (2006) **Period of Study:** Region 1: August to Mid December 2003. Region 2: July through November 2004 **Location:** Region 1: Riverside, CA. Region 2: Whittier, CA	**Outcome:** Fractional Concentration of Nitric Oxide in exhaled air (FENO) **Age Groups**: 9 through 18 **Study Design:** Longitudinal Panel Study **N:** 45 children **Statistical Analyses:** Linear mixed-effects models; Two-stage hierarchical model; Empirical Variograms; Fourth-order polynomial distributed lag mixed-effects model **Covariates:** Personal temperature, personal rel. humid., 10-day exposure run, respiratory infections, region of study, sex, cumulative daily use of as-needed B-agonist inhalers **Dose-response Investigated?** No **Lags Considered:** Lag 0, Lag 1, 2-day moving avg	**Pollutant:** $PM_{2.5}$ **PM Component:** Elemental carbon **Personal Exposure** **Averaging Time:** 24 h Riverside **Mean (SD):** 0.42 (0.69) 50th(Median): 0.34 µg/m^3 **Range (Min, Max):** 0.01, 6.94 Whittier **Mean (SD):** 0.78 (1.42) 50th(Median): 0.47 **Range (Min, Max):** 0, 17.2 **Central Site** **Averaging Time:** 24 h Riverside **Mean (SD):** 1.61 (0.78) 50th(Median): 1.35 **Range (Min, Max):** 0.52, 3.64 Whittier **Mean (SD):** 0.71 (0.43) 50th(Median): 0.63 **Range (Min, Max):** 0.14, 2.95 **Monitoring Stations**: 48 personal nephelometers, 2 central sites **Copollutant (correlation):** **Personal** 24-h personal $PM_{2.5}$ 0.18 24-h personal EC 1.00 24-h personal OC 0.41 24-h personal NO_2 0.0.21 24-h central $PM_{2.5}$ 0.00 24-h central EC 0.04 24-h central OC -0.01 24-h central NO_2 0.23 **Central** 24-h personal $PM_{2.5}$ 0.12 24-h personal EC 0.04 24-h personal OC 0.03 24-h personal NO_2 0.19 24-h central $PM_{2.5}$ 0.55 24-h central EC 1.00 24-h central OC 0.87 24-h central NO_2 0.70	**PM Increment:** IQR increase (Riverside: 28.41 µg/m^3, Whittier 21.87 µg/m^3) Coefficient [Lower CI, Upper CI] ; lag: Mixed-model estimates of the association between personal and central-site air pollutant exposure and FENO Lag 0 Personal 0.29 (0.10, 0.48) Central 0.10 (-0.65, 0.85) Lag 1 Personal -0.01 (-0.23, 0.21) Central 0.99 (0.27, 1.71) 2-day MA Personal 0.72 (0.32, 1.12) Central 1.38 (0.15, 2.61) Stratified by Medication Use Lag = 2-day moving avg Not Taking Anti-Inflamm. Medication Personal 0.84 (0.08, 1.60) Central 1.02 (-2.55, 4.60) Taking Anti-Inflamm. Medication Personal 0.71 (0.28, 1.15) Central 1.42 (0.25, 2.60) Inhaled Corticosteroids Personal 0.67 (0.28, 1.07) Central 1.28 (0.07, 2.49) Antileukotrienes +- inhaled corticosteroids Personal 0.03 (-3.29, 3.35) Central 1.15 (-1.58, 3.88) Notes: Figure of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Figure of the Estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Figure of One- and two-pollutant models for change in FENO using 2-day Moving Averages personal and central-site pollutant measurements.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Delfino et al. (2006) **Period of Study:** Region 1: August to Mid December 2003. Region 2: July through November 2004 **Location:** Region 1: Riverside, CA. Region 2: Whittier, CA	**Outcome:** Fractional Concentration of Nitric Oxide in exhaled air (FENO) **Age Groups**: 9 through 18 **Study Design:** Longitudinal Panel Study **N:** 45 children **Statistical Analyses:** Linear mixed-effects models; Two-stage hierarchical model; Empirical Variograms; Fourth-order polynomial distributed lag mixed-effects model **Covariates:** Personal temperature, personal rel. humid., 10-day exposure run, respiratory infections, region of study, sex, cumulative daily use of as-needed B-agonist inhalers **Dose-response Investigated?** No **Lags Considered:** Lag 0, Lag 1, 2-day moving avg	**Pollutant:** $PM_{2.5}$ **PM Component:** Organic carbon **Personal Exposure** **Averaging Time:** 24 h Riverside **Mean (SD):** 5.63 (2.59) 50th(Median): 4.98 **Range (Min, Max):** 1.94, 12.38 Whittier **Mean (SD):** 6.81 (3.45) 50th(Median): 6.43 **Range (Min, Max):** 2.18, 31.5 **Central Site** **Averaging Time:** 24 h Riverside **Mean (SD):** 6.88 (1.86) **Percentiles:** 50th Median: 6.07 **Range (Min, Max):** 4.11, 11.62 Whittier **Mean (SD):** 3.93 (1.49) 50th(Median): 3.76 **Range (Min, Max):** 1.64, 8.82 **Monitoring Stations**: 48 personal nephelometers, 2 central sites **Copollutant (correlation):** **Personal** 24-h personal $PM_{2.5}$ 0.15 24-h personal EC 0.41 24-h personal OC 1.00 24-h personal NO_2 0.20 24-h central $PM_{2.5}$ -0.11 24-h central EC 0.03 24-h central OC -0.02 24-h central NO_2 0.21 **Central** 24-h personal $PM_{2.5}$ 0.21 24-h personal EC -0.01 24-h personal OC -0.02 24-h personal NO_2 0.17 24-h central $PM_{2.5}$ 0.66 24-h central EC 0.87 24-h central OC 1.00 24-h central NO_2 0.62	**PM Increment:** IQR increase (Riverside: 28.41 µg/m³, Whittier 21.87 µg/m³) Mixed-model estimates of the association between personal and central-site air pollutant exposure and FENO Lag 0 Personal 0.51 (-0.28, 1.30) Central 0.93 (-0.20, 2.06) Lag 1 Personal 0.13 (-0.77, 1.03) Central0.51 (-0.64, 1.66) 2-day MA Personal 0.94 (-0.47, 2.35) Central 1.6 (-0.17, 3.37) Stratified by Medication Use Lag = 2-day moving avg. Not Taking Anti-Inflamm. Medication Personal 0.88 (-1.62, 3.38) Central 0.36 (-4.07, 4.79) Taking Anti-Inflamm. Medication Personal 0.87 (-0.79, 2.53) Central 2.05 (0.24, 3.86) Inhaled Corticosteroids Personal 2.47 (0.30, 4.64) Central 1.96 (0.14, 3.78) Antileukotrienes +- inhaled corticosteroids Personal 0.52 (-1.99, 3.02) Central 1.29 (-2.58, 5.15) Notes: Figure of Estimated lag effect of hourly personal $PM_{2.5}$ on FENO. Figure of the Estimated lag effect of hourly personal $PM_{2.5}$ on FENO by use of medications. Figure of One- and two-pollutant models for change in FENO using 2-day Moving Averages personal and central-site pollutant measurements

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: DeMeo et al. (2004) **Period of Study:** July through August, 1999 **Location:** Boston, MA	**Outcome:** Oxygen Saturation **Age Groups**: 60.4 to 89.2 years **Study Design:** Cross-sectional study **N:** 28 adult participants **Statistical Analyses:** GLM, Natural Spline Smoothing, Regression Analysis, Random-effects model **Covariates:** Mean temperature, Dew point temperature, Barometric pressure, Medication use **Season**: Summer **Dose-response Investigated?** No **Statistical Package**: S-PLUS, SAS **Lags Considered:** Hourly lags between 2 and 7 h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 6 h, 12 h, 24 h, 48 h	**PM Increment:** IQR (13.42 µg/m^3) increase 6 h: 13.42 µg/m^3; 12 h: 10.81 µg/m^3; 24 h: 10.26µg/m^3; 48: 10.57 µg/m^3 Overall: 0.172% (-0.313, 0.031) decrease 6-h: -0.769% (-1.21 to -0.327) decrease B-blocker users: -0.062% (-0.248, 0.123) Rest: 6 h: -0.173 (-0.345 to -0.001) 12 h: -0.160 (-0.308 to -0.012) 24 h: -0.169 (-0.316 to -0.022) 48 h: -0.153 (-0.304, 0.002) Exercise: 6 h: -0.005 (-0.215, 0.205) 12 h: -0.014 (-0.196, 0.168) 24 h: 0.001 (-0.180, 0.182) 48 h: -0.011 (-0.196, 0.174) Postexercise Rest: 6 h: -0.173 (-0.332 to -0.014) 12 h: -0.128 (-0.266, 0.010) 4 h: -0.113 (-0.250, 0.023) 48 h: -0.157 (-295 to -0.019) Paced breathing: 6 h: -0.142 (-0.292, 0.007) 12 h: -0.139 (-0.269 to -0.010) 24 h: -0.121 (-0.248, 0.007) 48 h: -0.082 (0.211, 0.047) Summary over protocol 6 h: -0.131 (-0.247 to -0.015) 12 h: -0.120 (-0.221, 0.020) 24 h: -0.112 (-0.212 to -0.013) **Notes:** Figure of the Variation in Oxygen Saturation during the first rest period versus individual hourly lag measurements for $PM_{2.5}$
Reference: Diette et al. (2007) **Period of Study:** 9/2001-12/2003 **Location:** East Baltimore, MD	**Outcome:** Asthma in the last 12 months (493.x) **Age Groups**: 2 to 6 years old **Study Design:** Prospective cohort **N:** 150 with asthma; 150 without asthma **Statistical Analyses:** Student's two-tailed t-test; Kruskal-Wallis test; Pearson's chi square; Fisher's exact test; **Covariates:** Season of collection **Dose-response Investigated?** No **Statistical Package**: STATASE 8.0	**Pollutant**:$PM_{2.5}$ **Averaging Time:** 72-h Avg 50th(Median): 28.7 IQR: (18-51)	% Homes above NAAQS of 65 µg/m^3 for $PM_{2.5}$: With Asthma 14.1% Without Asthma 16.8% **Notes:**"Pollutant concentrations in the homes of asthmatic and control children who lived in the same home for their whole life were not different compared with those who had moved at least once."

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Dubowsky et al (2006) **Period of Study:** 3/2002-6/2002 **Location:** St. Louis, Missouri	**Outcome:** Chronic inflammation, Diabetes, Obesity, Hypertension, Cardiac Risk **Study Design:** Prospective Cohort **Statistical Analyses:** Poisson, LOESS **Age Groups**: ≥ 60	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD) unit:** 16 (6.0) **Range (Min, Max):** 6.5, 28 **Copollutants:** BC CO NO_2 SO_2 O_3	**Increment:** 5.4 µg/m^3 **% Increase (Lower CI, Upper CI); Lag** % increase in inflammatory response and exposure to $PM_{2.5}$ in people ≥ 60 Inflammatory Marker: IL-6: -8 (-16, 8); 1: -6 (-10, 5); 2: -5 (-11, 6); 3: -3 (-9, 6); 4: -4 (-12, 10); 5: -5 (-13, 8); 6: -6 (-14, 9); 7 CRP: -2 (-22, 15); 1: 3 (-8, 17); 2: 4 (-9, 20); 3: 9 (-4, 27); 4: 11 (-5, 35); 5: 8 (-9, 29); 6: 5 (-12, 26); 7 WBC: 0 (-2, 4); 1: 1 (-1, 2); 2: 2 (-1, 3); 3: 1 (-2, 5); 4: 3 (-1, 10); 5: 5 (0, 12); 6: 8 (0, 14); 7 % Increase in inflammatory responses and exposure to ambient $PM_{2.5}$ concentrations in people ≥ 60 Inflammatory Marker: CRP All conditions*: 14 (-5.4, 37); 0-5 avg 3 conditions met*: 81 (21, 172); 0-5 avg 2 conditions met*: 11 (-7.3, 33); 0-5 avg IL-6 All conditions*: -2.1 (-13, 11); 0-5 avg 3 conditions met*: 23 (-5.3, 59); 0-5 avg 2 conditions met*: -3.1 (-14, 9.7); 0-5 avg WBC All conditions*: 3.4 (-1.8, 8.9); 0-5 avg 3 conditions met*: 0.4 (-8.8, 11); 0-5 avg 2 conditions met*: 3.6 (-1.7, 9.1); 0-5 avg * All conditions met means model is adjusted for sex, obesity, diabetes, smoking history, ambient and microenvironmental apparent temperature, mold, pollen, trip, h, and vitamins. Three conditions met means model is adjusted for three of the variables. Two conditions met means model is adjusted for two of the variables.
Reference: Ebelt et al. (2005) **Period of Study:** S ummer of 1998 **Location:** Vancouver, Canada	**Outcome:** Adverse health effects: spirometry, systolic/diastolic blood pressure measurements, symptom questionnaires, arrhythmia, heart rate, and heart rate variability (from electrocardiogram) **Age Groups**: range from 54-86 yrs; mean age= 74 years **Study Design:** extended analysis of a repeated-measures panel study **N:** 16 persons with COPD **Statistical Analyses:** Earlier analysis expanded by developing mixed-effect regression models and by evaluating additional exposure indicators **Dose-response Investigated?** No **Statistical Package**: SAS V8	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Ambient $PM_{2.5}$: 11.4 (4.6) Exposure to ambient $PM_{2.5}$: 7.9 (3.7) Nonsulfate ambient $PM_{2.5}$: 9.3 (3.7) Exposure to nonsulfate ambient $PM_{2.5}$: 6.5 (3.0) Total exposure to $PM_{2.5}$: 18.5 (14.9) Exposure to nonambient $PM_{2.5}$: 10.6 (14.5) **Range (Min, Max):** Ambient $PM_{2.5}$: (4.2-28.7) Exposure to ambient $PM_{2.5}$: (0.9-21.3) Nonsulfate ambient $PM_{2.5}$: (3.3-23.3) Exposure to nonsulfate ambient $PM_{2.5}$: (0.7-16.9) Total exposure to $PM_{2.5}$: (2.2-90.9) Exposure to nonambient $PM_{2.5}$: (-2.6-85.0) **Monitoring Stations**: 5 **Copollutant (correlation):** Ambient PM_{10}: r= 0.78 Ambient $PM_{10-2.5}$: r= 0.15 Ambient Sulfate- 0.82 Nonsufate Ambient $PM_{2.5}$: r= 0.98	**PM Increment:** Ambient $PM_{2.5}$: 5.8 (IQR) Exposure to ambient $PM_{2.5}$: 4.4 (IQR) Nonsulfate ambient $PM_{2.5}$: 4.2 (IQR) Exposure to nonsulfate ambient $PM_{2.5}$: 3.4 (IQR) Total exposure to $PM_{2.5}$: 10.1 (IQR) Exposure to nonambient $PM_{2.5}$: 8.9 (IQR) **Notes:** Effect estimates are presented in Figure 2 and Electronic Appendix Table 1 (only available with electronic version of article) and not provided quantitatively elsewhere.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ebelt et al. (2005) **Period of Study:** Summer of 1998 **Location:** Vancouver, Canada	**Outcome:** Adverse health effects: spirometry, systolic/diastolic blood pressure measurements, symptom questionnaires, arrhythmia, heart rate, and heart rate variability (from electrocardiogram) **Age Groups**: Range from 54-86 yrs; mean age= 74 years **Study Design:** extended analysis of a repeated-measures panel study **N:** 16 persons with COPD **Statistical Analyses:** Earlier analysis expanded by developing mixed-effect regression models and by evaluating additional exposure indicators **Dose-response Investigated?** No **Statistical Package**: SAS V8	**Pollutant:** Sulfate (SO_4) **Averaging Time:** 24 h **Mean (SD):** Ambient Sulfate: 2.0 (1.1) Exposure to Ambient Sulfate: 0.2 (4.7) **Range (Min, Max):** Ambient Sulfate: (0.4-5.4) Exposure to ambient Sulfate: (0.2-4.7) **Monitoring Stations**: 5 **Copollutant (correlation):** Ambient $PM_{2.5}$: r= 0.82 Nonsulfate Ambient $PM_{2.5}$: r= 0.74 Exposure to Ambient Sulfate: r= 0.82	**PM Increment:** Ambient Sulfate: 1.5 (IQR) Exposure to Ambient Sulfate: 0.9 (IQR) Notes: Effect estimates are presented in Figure 2 and Electronic Appendix Table 1 (only available with electronic version of article) and not provided quantitatively elsewhere.
Reference: Ferdinands et al. (2008) **Period of Study:** 8/16/2004–8/31/2004 **Location:** Atlanta, Georgia	**Outcome:** Respiratory Symptoms, airway inflammation **Study Design:** Prospective cohort **Statistical Analyses:** Pearson Correlation Analysis **Age Groups**: 14-18	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD) unit:** 27.2 (11.9) **Range (Min, Max):** 21.7, 34.7 **Copollutants (correlation):** O_3: r= 0.8-0.9	The study presents results qualitatively not quantitatively.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Gent et al. (2003) **Period of Study:** April 1 through September 30, 2001 **Location:** Connecticut; Springfield, MA	**Outcome:** Respiratory symptoms including: Wheeze, persistent cough, chest tightness, shortness of breath **Age Groups**: Infants **Study Design:** 1-year prospective cohort study **N:** 1002 infants; 17160 observations **Statistical Analyses:** Logistic regression analysis; General estimating equations; Tests for linear trend; Test for goodness of fit; Hosmer-Lemeshow statistic for regression **Covariates:** Temperature **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** 1-day lag	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 13.1 (7.9) **Percentiles**: 20th: 6.9 40th: 9.0 50th(Median): 10.3 60th: 12.1 80th: 19.0 **Range (Min, Max):** 3.7, 44.2 **Monitoring Stations**: 4 sites **Copollutant (correlation):** Temperature: 0.58	**PM Increment:** 12 µg/m³ same day ; 19 µg/m³ previous day **Model 5 (same day)** **Wheeze** <6.9 = 1.00; 6.9–8.9 = 0.95 (0.83, 1.10); 9.0–12.0 = 1.04 (0.89, 1.20); 12.1–18.9 = 1.05 (0.92, 1.20); ≥ 19.0 = 0.93 (0.78, 1.11) **Persistent Cough** <6.9 =1.00; 6.9–8.9 = 0.95 (0.87, 1.04); 9.0–12.0 = 0.96 (0.87, 1.06); 12.1–18.9 = 1.00 (0.91, 1.09); ≥ 19.0 = 0.95 (0.83, 1.09) **Chest Tightness** <6.9 = 1.00; 6.9–8.9 = 1.01 (0.86, 1.19); 9.0–12.0 = 1.06 (0.89, 1.26); 12.1–18.9 = 1.24 (1.06, 1.45); ≥ 19.0 = 1.05 (0.84, 1.33) **Shortness of Breath** <6.9 = 1.00; 6.9–8.9 = 1.01 (0.87, 1.17); 9.0–12.0 = 1.03 (0.87, 1.22); 12.1–18.9 = 1.07 (0.91, 1.25); ≥ 19.0 = 1.03 (0.83, 1.28) **Bronchodilator** <6.9 = 1.00; 6.9–8.9 = 1.04 (0.99, 1.09); 9.0–12.0 = 1.02 (0.96, 1.08); 12.1–18.9 = 1.04 (0.99, 1.09); ≥ 19.0 = 1.02 (0.97, 1.08) **Model 6 (previous day)** **Wheeze** <6.9 = 1.00; 6.9–8.9 = 1.06 (0.95, 1.20); 9.0–12.0 = 1.09 (0.94, 1.28); 12.1–18.9 = 1.03 (0.89, 1.19); ≥ 19.0 = 1.14 (0.97, 1.34) **Persistent Cough** <6.9 =1.00; 6.9–8.9 = 1.04 (0.94, 1.14); 9.0–12.0 = 1.05 (0.94, 1.17); 12.1–18.9 = 1.03 (0.94, 1.14); ≥ 19.0 = 1.12 (1.02 1.24) **Chest Tightness** <6.9 = 1.00; 6.9–8.9 = 1.03 (0.87, 1.23); 9.0–12.0 = 1.04 (0.85, 1.27); 12.1–18.9 = 1.00 (0.84, 1.19); ≥ 19.0 = 1.21 (1.00, 1.46); **Shortness of Breath** <6.9 = 1.00; 6.9–8.9 = 1.00 (0.84, 1.19); 9.0–12.0 = 1.09 (0.90, 1.31); 12.1–18.9 = 1.09 (0.90, 1.31); ≥ 19.0 = 1.26 (1.02, 1.54) **Bronchodilator** <6.9 = 1.00; 6.9–8.9 = 0.98 (0.94, 1.03); 9.0–12.0 = 0.99 (0.95, 1.03); 12.1–18.9 = 0.97 (0.94, 1.01); ≥ 19.0 = 0.99 (0.95, 1.04) **$PM_{2.5}$ + O_3: Medication Users: Same-day** **Wheeze** <6.9 = 1.00; 6.9–8.9 = 0.89 (0.75, 1.29); 9.0–12.0 = 1.02 (0.87, 1.19); 12.1–18.9 = 0.94 (0.77, 1.15); ≥ 19.0 = 0.83 (0.65, 1.06) **Persistent Cough** <6.9 = 1.00; 6.9–8.9 = 0.95 (0.84, 1.06); 9.0–12.0 = 0.97 (0.86, 1.10); 12.1–18.9 = 0.94 (0.77, 1.15); ≥ 19.0 = 0.83 (0.65, 1.06) **Chest Tightness** <6.9 = 1.00; 6.9–8.9 = 0.90 (0.74, 1.09); 9.0–12.0 = 0.97 (0.79, 1.18); 12.1–18.9 = 0.97 (0.76, 1.25); ≥ 19.0 = 0.76 (0.54, 1.05) **Shortness of Breath** <6.9 = 1.00; 6.9–8.9 = 0.95 (0.80, 1.12); 9.0–12.0 = 1.00 (0.82, 1.21); 12.1–18.9 = 0.90 (0.73, 1.12); ≥ 19.0 = 0.87 (0.65, 1.17) **Bronchodilator** <6.9 = 1.00; 6.9–8.9 = 1.03 (0.98, 1.08); 9.0–12.0 = 1.01 (0.96, 1.07); 12.1–18.9 = 1.02 (0.95, 1.08); ≥ 19.0 = 0.99 (0.91, 1.07) **Previous Day** **Wheeze** <6.9 = 1.00; 6.9–8.9 = 1.03 (0.89, 1.18); 9.0–12.0 = 1.05 (0.88, 1.24); 12.1–18.9 = 0.98 (0.82, 1.17); ≥ 19.0 = 1.05 (0.85, 1.29) **Persistent Cough** <6.9 = 1.00; 6.9–8.9 = 0.99 (0.89, 1.11); 9.0–12.0 = 0.98 (0.86, 1.10); 12.1–18.9 = 0.95 (0.83, 1.10); ≥ 19.0 = 1.00 (0.88, 1.15) **Chest Tightness** <6.9 = 1.00; 6.9–8.9 = 0.89 (0.72, 1.10); 9.0–12.0 = 0.90 (0.70, 1.16); 12.1–18.9 = 0.81 (0.63, 1.03); ≥ 19.0 = 0.91 (0.71, 1.17) **Shortness of Breath** <6.9 = 1.00; 6.9–8.9 = 0.96 (0.78, 1.18); 9.0–12.0 = 1.00 (0.81, 1.25); 12.1–18.9 = 0.96 (0.74, 1.24); ≥ 19.0 = 1.20 (0.94, 1.52) **Bronchodilator** <6.9 = 1.00; 6.9–8.9 = 0.99 (0.94, 1.04); 9.0–12.0 = 0.97 (0.93, 1.02); 12.1–18.9 = 0.96 (0.91, 1.02); ≥ 19.0 = 0.97 (0.89, 1.04) **$PM_{2.5}$ + O_3: Non-users: Same-day** **Wheeze** <6.9 = 1.00; 6.9–8.9 = 0.92 (0.72, 1.17); 9.0–12.0 = 1.08 (0.85, 1.36); 12.1–18.9 = 0.94 (0.73, 1.22); ≥ 19.0 = 1.15 (0.75, 1.75) **Persistent Cough** <6.9 = 1.00; 6.9–8.9 = 0.96 (0.83, 1.12); 9.0–12.0 = 1.02 (0.89, 1.18); 12.1–18.9 = 0.93 (0.78, 1.12); ≥ 19.0 = 1.07 (0.85, 1.34) **Chest Tightness** <6.9 = 1.00; 6.9–8.9 = 0.84 (0.54, 1.31); 9.0–12.0 = 1.09 (0.74, 1.61); 12.1–18.9 = 0.78 (0.47, 1.30); ≥ 19.0 = 0.71 (0.36, 1.39) **Shortness of Breath** <6.9 = 1.00; 6.9–8.9 = 0.61 (0.39, 0.95); 9.0–12.0 = 1.13 (0.85, 1.50); 12.1–18.9 = 0.72 (0.42, 1.23); ≥ 19.0 = 1.17 (0.72, 1.90); **Bronchodilator Use:** <6.9 = 1.00; 6.9–8.9 = 0.95 (0.78, 1.15); 9.0–12.0 = 0.95 (0.78, 1.16); 12.1–18.9 = 0.85 (0.69, 1.06); ≥ 19.0 = 0.99 (0.76, 1.30) **Previous-day** **Wheeze** <6.9 = 1.00; 6.9–8.9 = 1.01 (0.78, 1.31); 9.0–12.0 = 1.15 (0.88, 1.51); 12.1–18.9 = 1.08 (0.78, 1.51); ≥ 19.0 = 1.18 (0.71, 1.97) **Persistent Cough** <6.9 = 1.00; 6.9–8.9 = 1.07 (0.94, 1.22); 9.0–12.0 = 1.13 (0.97, 1.32); 12.1–18.9 = 1.03 (0.87, 1.22); ≥ 19.0 = 1.14 (0.88, 1.46) **Chest Tightness** <6.9 = 1.00; 6.9–8.9 = 1.44 (0.90, 2.30); 9.0–12.0 = 1.50 (0.97, 2.33); 12.1–18.9 = 1.56 (0.91, 2.66); ≥ 19.0 = 1.76 (0.83, 3.73) **Shortness of Breath** <6.9 = 1.00; 6.9–8.9 = 0.99 (0.75, 1.30); 9.0–12.0 = 1.30 (0.88, 1.91); 12.1–18.9 = 0.84 (0.57, 1.24); ≥ 19.0 = 1.48 (0.94, 2.34) **Bronchodilator Use** <6.9 = 1.00; 6.9–8.9 = 1.05 (0.85, 1.34); 9.0–12.0 = 1.28 (1.01, 1.62); 12.1–18.9 = 1.05 (0.80, 1.37); ≥ 19.0 = 1.19 (0.83, 1.71) **Notes:** Line graphs of daily levels of ozone and $PM_{2.5}$ and daily temperature with daily prevalence of respiratory symptoms for users of asthma maintenance medication

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Girardot et al. (2006) **Period of Study:** 10 August 2002-16 October 2002; 17 June 2003-27 August 2003 **Location:** Charlies Bunion Trail (portion of Appalachia Trail)	**Outcome:** Pulmonary function/spirometry–FVC, FEV_1, PEF, FVC/FEV_1, FEF_{25-75} **Age Groups**: 18-82 yrs **Study Design:** Cohort **N:** 354 hikers **Statistical Analyses:** Multiple linear regression **Covariates:** Age, h hiked, mean temperature, sex, smoking status, history of asthma or wheeze symptoms, carriage of backpack, whether reaching summit or not **Season:** Fall 2002, Summer 2003 **Dose-response Investigated?** No **Statistical Package**: SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean:** Trail: 13.9 +/- 8.2 Estimated personal: 15.0 +/- 7.4 **Range (Min, Max):** Trail: 1.6 , 38.4 Estimated personal: 0.21, 41.9 **Copollutant (correlation):** O_3 (r=0.67, for estimated personal exposure)	**PM Increment:** 1 µg/m^3 % Change +/- CI ; p value Univariate: FVC: 0.023 +/ 0.035 ; 0.51 FEV_1: 0.015 +/- 0.029 ; 0.607 PEF: 0.185 +/- 0.091 ; 0.043 FVC/FEV_1: 0.003 +/- 0.023 ; 0.905 $FEF_{25-75\%}$: 0.052 +/- 0.093 ; 0.578 Adjusted: FVC: 0.007 +/ 0.040 ; 0.966 FEV_1: 0.003 +/- 0.033 ; 0.937 PEF: 0.258 +/- 0.103 ; 0.013 FVC/FEV_1:- 0.011 +/- 0.027 ; 0.676 $FEF_{25-75\%}$:- 0.041 +/- 0.109 ; 0.707 Spirometry result for each quintile +/- CI **Quintile 1 (6.0 µg/m^3):** FVC (L): Prehike: 4.32 +/- 0.13; Posthike: 4.33 +/- 0.12 FEV_1 (L): Prehike: 3.39 +/- 0.10; Posthike: 3.40 +/- 0.10 FEV_1/FVC (%): Prehike: 78.66 +/- 0.86; Posthike: 78.63 +/- 0.81 $FEF_{25-75\%}$ (L/sec): Prehike: 3.27 +/- 0.14; Posthike: 3.26 +/- 0.14 PEF (L/sec): Prehike: 7.91 +/ 0.22; Posthike: 7.58 +/- 0.22 **Quintile 2 (10.4 µg/m^3):** FVC (L): Prehike: 4.30 +/- 0.11; Posthike: 4.30 +/- 0.11 FEV_1 (L): Prehike: 3.42 +/- 0.09; Posthike: 3.43 +/- 0.09 FEV_1/FVC (%): Prehike: 79.37 +/- 0.71; Posthike: 79.55 +/- 0.69 $FEF_{25-75\%}$ (L/sec): Prehike: 3.39 +/- 0.14; Posthike: 3.38 +/- 0.14 PEF (L/sec): Prehike: 8.37 +/ 0.23; Posthike: 8.26 +/- 0.25 **Quintile 3 (14.8 µg/m^3):** FVC (L): Prehike: 4.34 +/- 0.12; Posthike: 4.33 +/- 0.12 FEV_1 (L): Prehike: 3.42 +/- 0.10; Posthike: 3.40 +/- 0.09 FEV_1/FVC (%): Prehike: 79.20 +/- 0.81; Posthike: 78.83 +/- 0.80 $FEF_{25-75\%}$ (L/sec): Prehike: 3.19 +/- 0.13; Posthike: 3.21 +/- 0.13 PEF (L/sec): Prehike: 8.12 +/ 0.25; Posthike: 7.89 +/- 0.25 **Quintile 4 (17.9 µg/m^3):** FVC (L): Prehike: 4.23 +/- 0.11; Posthike: 4.23 +/- 0.11 FEV_1 (L): Prehike: 3.36 +/- 0.10; Posthike: 3.36 +/- 0.10 FEV_1/FVC (%): Prehike: 79.18 +/- 0.81; Posthike: 79.26 +/- 0.79 $FEF_{25-75\%}$ (L/sec): Prehike: 3.34 +/- 0.15; Posthike: 3.30 +/- 0.15 PEF (L/sec): Prehike: 7.75 +/ 0.25; Posthike: 7.73 +/- 0.26 **Quintile 5 (25.6 µg/m^3):** FVC (L): Prehike: 4.15 +/- 0.11; Posthike: 4.18 +/- 0.12 FEV_1 (L): Prehike: 3.31 +/- 0.09; Posthike: 3.33 +/- 0.10 FEV_1/FVC (%): Prehike: 79.73 +/- 0.66; Posthike: 79.55 +/- 0.64 $FEF_{25-75\%}$ (L/sec): Prehike: 3.22 +/- 0.14; Posthike: 3.24 +/- 0.14 PEF (L/sec): Prehike: 7.72 +/ 0.22; Posthike: 7.77 +/- 0.23 **Overall (15.0 µg/m^3):** FVC (L): Prehike: 4.27 +/- 0.05; Posthike: 4.27 +/- 0.05 FEV_1 (L): Prehike: 3.38 +/- 0.04; Posthike: 3.38 +/- 0.04 FEV_1/FVC (%): Prehike: 79.2 +/- 0.34; Posthike: 79.2 +/- 0.33 $FEF_{25-75\%}$ (L/sec): Prehike: 3.28 +/- 0.06; Posthike: 3.28 +/- 0.06 PEF (L/sec): Prehike: 7.97 +/ 0.11; Posthike: 7.97 +/- 0.11

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hertz-Picciotta et al. (2007) **Period of Study:** 1994-2003 **Location:** Teplice and Prachatice, Czech Republic	**Outcome:** Lower respiratory illness–croup (J05, J04), acute bronchitis (J20), acute bronchiolitis (J21) **Age Groups**: Neonates followed for 2 to 4.5 yrs **Study Design:** Cohort **N:** 1133 children **Statistical Analyses:** Generalized linear longitudinal models **Covariates:** District, mother's age, mother's education, mother or adult smoke, child's sex, season, day of the week, fuel for heating and/or cooking, breastfeeding category, number of other children, temperature **Season:** Winter, spring, summer and fall **Dose-response Investigated?** No **Statistical Package**: SUDAAN version 8 **Lags Considered:** 1-3, 1-7, 1-14, 1-30, 1-45	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** PAH: 22.3 (SD–16 for 3-day avg and 11 for 45-day avg)	**PM Increment:** 25 µg/m^3 **RR Estimate [Lower CI, Upper CI] ; lag:** Birth–23 months: 1.30 [1.08, 1.58] lag 1-30 2–4.5 yrs: 1.23 [0.94, 1.62] lag 1-30 RR Estimate for categories of exposure [Lower CI, Upper CI] ; lag: Crude RR: Birth–23 months: > 50 µg/m^3: 2.26 [1.81, 2.82] lag 1-30 25-50 µg/m^3: 1.48 [1.32, 1.65] lag 1-30 < 25 µg/m^3: Reference 2–4.5 yrs: > 50 µg/m^3: 3.66 [2.07, 6.48] lag 1-30 25-50 µg/m^3: 1.60 [1.41, 1.82] lag 1-30 < 25 µg/m^3: Reference
Reference: Hertz-Picciotta et al. (2007) **Period of Study:** 1994-2003 **Location:** Teplice and Prachatice, Czech Republic	**Outcome:** Lower respiratory illness–croup (J05, J04), acute bronchitis (J20), acute bronchiolitis (J21) **Age Groups**: Neonates followed for 2 to 4.5 yrs **Study Design:** Cohort **N:** 1133 children **Statistical Analyses:** Generalized linear longitudinal models **Covariates:** District, mother's age, mother's education, mother or adult smoke, child's sex, season, day of the week, fuel for heating and/or cooking, breastfeeding category, number of other children, temperature **Dose-response Investigated?** No **Statistical Package**: SUDAAN version 8 **Lags Considered:** 1-3, 1-7, 1-14, 1-30, 1-45	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** PAH: 52.5 ng/m3 (SD–57 ng/m3 for 3-day avg and 46 ng/m3 for 45-day avg)	**PAH Increment:** 100 ng/m3 **RR Estimate [Lower CI, Upper CI] ; lag:** Birth–23 months: 1.29 [1.07, 1.54] lag 1-30 2–4.5 yrs: 1.56 [1.22, 2.00] lag 1-30 RR Estimate for categories of exposure [Lower CI, Upper CI] ; lag: Crude RR: Birth–23 months: > 100 ng/m3: 2.52 [2.22, 2.87] lag 1-30 40-100 ng/m3: 1.87 [1.65, 2.13] lag 1-30 < 40 ng/m3: Reference 2–4.5 yrs: > 100 ng/m3: 2.26 [1.93, 2.65] lag 1-30 40-100 ng/m3: 1.40 [1.20, 1.64] lag 1-30 < 40 ng/m3: Reference

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hogervorst, et al (2006) **Period of Study:** 2002 **Location:** Maastricht, the Netherlands (six schools selected)	**Outcome**: Decreased lung function **Age Groups**: 8-13 years old **Study Design:** Multivariate linear regression (enter method) analysis **N:** 342 children **Statistical Analyses:** ANOVA, chi square **Covariates:** Independent variables: Age, height, gender, smoking at home by parents, pets, use of ventilation hoods during cooking, presence of unvented geysers, tapestry in the home, indoor/outdoor time, education level of parents. Dependent variables:lung function indices **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily **Mean (SD):** 19.0 (3.2) **Monitoring Stations**: 6 **Copollutant:** PM_{10} Total Suspended Particles (TSP)	**PM Increment:** 10 µg/m^3 **RR Estimate [Lower CI, Upper CI] ; lag:** FEV: 3.62 [0.50,7.63]; lag NR FVC: 1.80 [-2.10, 5.80]; lag NR FEF: 5.93 [-2.34, 14.89]; lag NR
Reference: Hong et al. (2007) **Period of Study:** March 23-May3, 2004 **Location:** School on the Dukjeok Island near Incheon City, Korea	**Outcome:** Peak expiratory flow rate (PEFR) **Age Groups**: 3rd to 6th grade (mean age=9.6 yrs) **Study Design: P**anel study **N:** 43 schoolchildren **Statistical Analyses:** Mixed linear regression **Covariates:** age, sex, height, weight, asthma history, and passive smoking exposure at home **Dose-response Investigated?** No **Lags Considered:** 0, 1, 2, 3, 4, 5	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 20.27 (8.23) 50th(Median): 22.07 **Range (Min, Max):** 5.94-36.28 **Copollutant:** PM_{10} Components of PM_{10} (Fe,Mn, Pb, Zn, Al)	**Effect Estimate:** Regression coefficients of morning and daily mean PEFR on $PM_{2.5}$ Lag 1 ($PM_{2.5}$) Morning PEFR Crude: ß= -0.14, p=0.12 Adjusted: ß= -0.54, p,0.01 Mean PEFR Crude: ß= -0.15, p=0.02 Adjusted: ß= -0.54, p,0.01 Regression coefficients of morning and daily mean PEFR on $PM_{2.5}$ and GSTM1 and GSTT1 genotype using linear mixed-effects regression Lag 1 ($PM_{2.5}$) Morning PEFR: ß= -0.57, p<0.01 Mean PEFR: ß= -0.56, p<0.01 GSTM1 Morning PEFR: ß= 20.04, p=0.25 Mean PEFR: ß= 18.75, p=0.28 GSTT1 Morning PEFR: ß= 2.31, p=0.89 Mean PEFR: ß= 1.75, p=0.91
Reference: Islam et al, (2007) **Period of Study:** 2006 **Location:** 12 California communities	**Outcome:** Respiratory symptoms, Asthma **Study Design:** Longitudinal study **Statistical Analyses:** Cox proportional hazards regression **Age Groups**: 7-9; 10-11; > 11	**Pollutants**: $PM_{2.5}$ **Averaging Time:** 24-h avg **Copollutants:** O_3; NO_2; EC; OC	The study doesn't presents quantitative results for $PM_{2.5}$.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Jansen, et al. (2005) **Period of Study:** 1987-2000 **Location:** Seattle, WA	**Outcome:** FENO: fractional exhaled nitrogen oxide, Spirometry, Blood pressure, SaO2: oxygen saturation, Pulse rate **Age Groups**: 60-86-years-old **Study Design:** Short-term cross-sectional case series **N:** 16 subjects diagnosed with COPD, asthma, or both **Statistical Analyses:** Linear mixed effects model with random intercepts **Covariates:** Age, relative humidity, temperature, medication use **Season: W**inter 2002-2003 **Dose-response Investigated?** No **Statistical Package**: STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** **Fixed-Site Monitor:** 14.0 All Subjects (N=16) Indoor, home: 7.29 Outdoor, home: 10.47 Asthmatic Subjects (N=7) Indoor, home: 7.25 Outdoor, home: 8.99 COPD Subjects (N=9) Indoor, home: 7.33 Outdoor, home: 11.66 **Range (Min, Max):** **Fixed-Site Monitor:** 1.3, 44 IQR All Subjects Indoor, home: 4.05 Outdoor, home: 8.87 Asthmatic Subjects Indoor, home: 5.72 Outdoor, home: 7.55 COPD Subjects Indoor, home: (3.18 Outdoor, home: 6.71	**PM Increment:** $PM_{2.5}$: 10 $\mu g/m^3$ Slope [95% CI]: dependence of FENO concentration [ppb] on $PM_{2.5}$ **Asthmatic Subjects** Indoor, home: 3.69 [-0.74:8.12] Outdoor, home: 4.23 [1.33:7.13]* **Copd Subjects** Indoor, home: -0.35 [-7.45:6.75] Outdoor, home: 3.83 [-1.84:9.49] Results indicate that FENO may be a more sensitive biomarker of PM exposure than other traditional health endpoints.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Janssen et al. (2003) **Period of Study:** 4/1997–7/1998 **Location:** Netherlands–24 schools	**Outcome:** Symptoms of asthma and allergic disease (asthma, conjunctivitis, hay fever, itchy rash, eczema, phlegm, bronchitis), skin prick test (SPT) reaction to allergens, lung function (forced vital capacity [FVC], forced expiratory volume in one second [FEV_1], and positive test for fall in $FEV_1 \geq 15\%$ after inhalation of maximal 23 mL hypertonic saline [BHR = bronchial hyper-responsiveness]) **Age Groups**: 7-12 years old **Study Design:** Cohort **N:** 24 schools (see notes) **Statistical Analyses:** Multilevel model **Covariates:** Age, sex, non-Dutch nationality, cooking on gas, current parental smoking, current pet possession, parental education level, number of persons in the household, presence of an unvented water heater in kitchen, questionnaire not filled out by the mother, presence of mold stains in kitchen or living room or bedroom, parental respiratory symptoms, distance of home to motorway, cough or cold at time of lung function measurement, bronchitis or severe cold or flu in 3 weeks preceding measurement, season **Dose-response Investigated?** No **Statistical Package**: MLwiN	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean (SD):** 20.5 µg/m³ (2.2) **Percentiles:** 25th: 18.6 50th (Median): 20.4 75th: 22.1 **Range (Min, Max):** 17.3, 24.4	**PM Increment:** 'Difference between the maximum and the minimum of the exposure indicator' (3.5 µg/m³) **RR Estimate [Lower CI, Upper CI] ; lag:** Current wheeze 1.51 (0.90, 2.53) Asthma ever 1.03 (0.59, 1.82) Current conjunctivitis 2.08 (1.17, 3.71) Hay fever ever 2.28 (1.13, 4.57) Current itchy rash 1.63 (0.91, 2.89) Ezcema ever 1.31 (0.94, 1.83) Current phlegm 1.53 (0.74, 3.19) Current bronchitis 1.71 (0.84, 3.50) Elevated total ige 1.45 (0.74, 2.84) Any allergen (spt reactivity) 1.33 (0.83, 2.11) Indoor allergens (spt reactivity) 1.17 (0.70, 1.94) Outdoor allergens (spt reactivity) 1.90 (1.06, 3.40) FVC < 85% predicted 0.54 (0.29, 1.00) FEV_1 < 85% predicted 0.88 (0.37, 2.09) BHR 0.93 (0.51, 1.68) **Notes:** Figure 1 of the article illustrates the association between exposures, including $PM_{2.5}$, and various respiratory symptoms among children with and without a positive SPT and positive BHR. In general, the association between $PM_{2.5}$ and respiratory symptoms were higher for children with a positive SPT or BHR, except for the outcome of current phelgm. This effect appeared to be the strongest for children with a positive BHR, particularly for current wheeze and current bronchitis. The authors also reported separate analyses for children with SPT reactivity for indoor and outdoor allergens, but did not report any clear differences between the two groups. The authors did report, in the text, that the OR of $PM_{2.5}$ exposure for children sensitized for outdoor allergens was 7.64 for current itchy rash ($p < 0.05$).

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Johnston, et al. (2006) **Period of Study:** 7 months (April 7 through November 7, 2004) **Location:** Darwin, Australia	**Outcome:** Asthma symptoms **Age Groups**: All Ages **Study Design:** Time-series **N: 2**51 people (130 adults, 121 children **Statistical Analyses:** Logistic regression model **Covariates:** Minimum air temperature, doctor visits for influenza and the prevalence of asthma symptoms and, the fungal spore count and both onset of asthma symptoms and commencement of reliever medication **Season:** "Dry season"-note Southern Hemisphere **Dose-response Investigated?** No **Statistical Package**: STATA8 **Lags Considered:** 0-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time: D**aily **Mean (SD):** 11.1 (5.4) **Range (Min, Max):** 2.2, 36.5 **PM Component:** Vegetation fire smoke (95%) and motor vehicle emissions (5%) **Monitoring Stations**: 1	**PM Increment:** 5 µg/m^3 **RR Estimate [Lower CI, Upper CI] ; lag:** **Symptoms attributable to asthma** Overall: 1.000 (0.98,1.01) Adults: 1.000 (0.976,1.026) Children: 1.008 (0.980, 1.037) Using preventer: 1.013 (0.990, 1.037) **Became symptomatic** Overall: 1.150 (1.07,1.23) Adults: 1.165 (1.058,1.284) Children: 1.148 (1.042,1.264) Using preventer: 1.181 (1.076,1.296) **Used Reliever** Overall: 1.000 (0.98,1.02) Adults: 1.007 (0.980, 1.035) Children: 1.002 (0.972,1.034) Using preventer: 1.020 (1.000,1.042) **Commenced Reliever** Overall: 1.120 (1.03,1.210) Adults: 1.141 (1.021, 1.275) Children: 1.112 (0.994,1.243) Using preventer: 1.129 (1.013,1.257) **Commenced Oral Steroids** Overall: 1.310 (1.03,1.66) Adults: 1.601 (1.192, 2.150) Children: 0.995 (0.625,1.459) Using preventer: 1.350 (1.040,1.752) **Asthma Attack** Overall: 0.980 (0.94,1.04) Adults: 1.026 (0.962, 1.095) Children: 0.832 (0.731, 0.946) Using preventer: 1.002 (0.934,1.075) **Exercise induced asthma** Overall: 0.990 (0.95,1.03) Adults: 0.998 (0.943, 1.056) Children: 0.982 (0.899,1.071) Using preventer: 1.002 (0.942,1.067) **Saw a health professional for asthma** Overall: 1.030 (0.91,1.16) Adults: 1.079 (0.899, 1.296) Children: 1.003 (0.841,1.195) Using preventer: 0.980 (0.847,1.133) **Missed school or work due to asthma** Overall: 1.025 (0.9284,1.131) Adults: 1.077 (0.923, 1.247) Children: 1.000 (0.873,1.458) Using preventer: 1.005 (0.897,1.124) **Mean daily number of asthma symptoms** Overall: 1.003 (0.99,1.01) Adults: 0.998 (0.984, 1.012) Children: 1.004 (0.985,1.023) Using preventer: 1.013 (0.999,1.028) **Mean Daily number of applications of reliever** Overall: 1.002 (0.993,1.010) Adults: 1.001 (0.986, 1.016) Children: 1.000 (0.980,1.021) Using preventer: 1.005 (0.994,1.017)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Koenig et al. (2003) **Period of Study:** Winter 2000-2001, Spring 2001 **Location:** Seattle, WA	**Outcome:** Exhaled NO (eNO) **Age Groups**: 6-13 years old **Study Design:** Cohort **N** (Specify units): 19 children **Statistical Analyses:** Linear mixed-effects regression **Covariates:** Medication use, ambient NO reading for specific individual on specific day of session, mean ambient NO for subject during session, mean ambient NO for subject during all sessions Season: Winter, Spring **Dose-response Investigated?** No **Statistical Package**: STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 10 consecutive days **Mean (SD):** Outdoor: 13.3 (1.4) Indoor: 11.1 (4.9) Personal: 13.4 (3.2) Central-site: 10.1 (5.7) **Range (Min, Max):** Outdoor: Max: 40.4 Indoor: Max: 36.3 Personal: Max: 49.4 Central-site: NR **Monitoring Stations**: Outdoor: NR Indoor: NR Personal: NR Central-site: 3 **Copollutant (correlation):** Outdoor PM-central-site NO: 0.50 For NO values < 100 ppb, outdoor PM-central-site NO: 0.04	**PM Increment:** 10 µg/m^3 Results presented as change in eNO (95% CI) Among ICS* nonuser Personal monitor 4.48 (1.02, 7.93) Outdoor monitor 4.28 (1.38, 7.17) Indoor monitor 4.21 (1.02, 7.41) Central site 3.82 (1.22, 6.43) Among ICS* user Personal monitor -0.09 (-2.39, 2.21) Outdoor monitor 0.74 (-2.28, 3.76) Indoor monitor -1.11 (-5.08, 2.87) Central site 1.28 (-1.23, 3.79) * ICS: Inhaled corticosteroid
Reference: Koenig et al. (2003) **Period of Study:** Winter 2000-2001, spring 2001 **Location:** Seattle, WA	**Outcome**: Increased exhaled nitric oxide (eNO) **Age Groups**: 6–13 years of age **Study Design:** Combined recursive and predictive model **N:** 19 children with asthma **Statistical Analyses:** Linear mixed effects model **Covariates:** Residence type, air cleaner, avg outdoor temperature, avg daily rainfall **Season:** Winter, Spring **Dose-response Investigated?** No **Statistical Package**: STATA 7.0 for health analyses, SAS 8.0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily **Mean:** Home indoor 9.5 Home outdoor 11.1 Recursive model Eag: 7.0 Recursive model Eig: 2.1 Predictive model Eag: 6.0 Predictive model Eig: 4.0 Combined model Eag: 6.4 Combined model Eig: 3.2 **25th:** Home indoor 5.7 Home outdoor 6.3 Recursive model Eag: 4.2 Recursive model Eig: 0.0 Predictive model Eag: 3.4 Predictive model Eig: 0.9 Combined model Eag: 3.7 Combined model Eig: 0.5 **50th(Median):** Home indoor 7.6 Home outdoor 9.5 Recursive model Eag: 5.9 Recursive model Eig: 1.2 Predictive model Eag: 5.0 Predictive model Eig: 2.2 Combined model Eag: 5.5 Combined model Eig: 1.7 **75th**: Home indoor 10.8 Home outdoor 14.6 Recursive model Eag: 9.2 Recursive model Eig: 2.3 Predictive model Eag: 7.5 Predictive model Eig: 4.9 Combined model Eag: 7.8 Combined model Eig: 4.2 **Range (Min, Max):** Home indoor 2.3, 36.3 Home outdoor 2.8, 40.4 Recursive Eag: 1.8,22.6 Recursive Eig: 0.0,17.2 Predictive Eag: 1.3,22.6 Predictive Eig: 0.0,33.0 Combined Eag: 1.3,22.6 Combined Eig: 0.0,33.0 **Monitoring Stations**: 19 personal environmental monitors	**PM Increment:** 10-µg/m3 RR Estimate [Lower CI, Upper CI] ; lag: Eag= ambient-generated personal exposure Eig= indoor-generated personal exposure eNO= exhaled nitric oxide Recursive model with 8 children, Eag was marginally associated with increases in eNO [5.6 ppb [–0.6,11.9]. Eig was not associated with eNO (–0.19 ppb). For those combined estimates, only Eag was significantly associated with an increase in eNO: Eag: 5.0 ppb [0.3, 9.7] Eig: 3.3 ppb [1.1, 7.7] Notes: Effects were seen only in children who were not using corticosteroid therapy

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kongtip et al. (2006) **Period of Study:** September 1–October 31, 2004 **Location:** Dindang district, Bangkok metropolitan, Thailand	**Outcome:** respiratory and other Outcomes reported **Age Groups**: Age range 15 to 55 yrs **Study Design:** panel study **N:** 77 street vendors **Statistical Analyses:** Binary logistic regression **Covariates:** Gender, age, type of fuel used, working duration (months) **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 70.94 **Percentiles:** 50th(Median): 72.05 **Range (Min, Max):** 23.20-120.00 **Monitoring Stations**: 1 **Copollutant (correlation):** SO_2 NO_2 O_3 VOCs CO	**PM Increment:** 1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** **Model 1** Headache: 1.011 (0.999-1.022) Nose congestion: 1.006 (0.997-1.015) Sore throat: 1.000 (0.991-1.008) Cold: 1.006 (0.995-1.017) Cough: 0.989 (0.980-0.998) Phlegm: 0.998 (0.992-1.003) Chest tightness: 0.995 (0.955-1.036) Fever: 1.008 (0.993-1.024) Eye irritation: 1.022 (1.011-1.033) Dizziness: 1.027 (1.013-1.041) Weakness: 0.996 (0.983-1.008) Upper respiratory symptom: 1.001 (0.994-1.008) Lower respiratory symptom: 0.997 (0.992-1.002) **Model 2** Headache: 1.004 (0.996-1.013) Nose congestion: 1.003 (0.996-1.010) Sore throat: 0.995 (0.989-1.001) Cold: 0.996 (0.988-1.004) Cough: 0.990 (0.983-0.996) Phlegm: 0.995 (0.991-0.999) Chest tightness: 0.997 (0.970-1.025) Fever: 1.010 (0.998-1.022) Eye irritation: 1.019 (1.010-1.028) Dizziness: 1.020 (1.009-1.032) Weakness: 1.003 (0.994-1.012) Upper respiratory symptom: 0.995 (0.990-1.000) Lower respiratory symptom: 0.995 (0.991-0.999)
Reference: Lagorio et al. (2006) **Period of Study:** 5/24/1999 to 6/24/1999 and 11/181999 to 12/22/1999 **Location:** Rome, Italy	**Outcome:** Lung function (FVC and FEV_1) of subjects with COPD, Asthma **Age Groups**: COPD 50 to 80 yrs; Asthma 18 to 64 yrs **Study Design:** Time series **N:** COPD = 11; Asthma = 11 **Statistical Analyses:** Non-parametric Spearman correlation; GEE; **Covariates:** COPD and IHD: daily mean temperature, season variable (spring or winter), relative humidity, day of week; Asthma: season variable, temperature, humidity, and β-2-agonist use **Season**: Spring and Winter **Dose-response Investigated?** Yes **Statistical Package**: STATA **Lags Considered:** 1–3 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Overall: 27.2 (19.4) Spring:18.2 (5.0) Winter: 36.7 (24.1) **Range (Min, Max):** 4.5, 100 **PM Component:** Cd: 0.46±0.40 ng/m^3 Cr: 1.9±1.7 ng/m^3 Fe:283±167 ng/m^3 Ni:4.8±6.5 ng/m^3 Pb:30.6±19.0 ng/m^3 Pt:5.0±8.6 pg/m^3 V:1.8±1.4 ng/m^3 Zn:45.8±33.1 ng/m^3 **Monitoring Stations**: 2 fixed sites:(Villa Ada and Istituto superior di Sanita) **Copollutant (correlation):** NO_2 r = 0.43; O_3 r = -0.51; CO r = 0.67; SO_2 r = 0.34; $PM_{10-2.5}$ r = 0.34; PM_{10} r = 0.93	**PM Increment:** 1 µg/m^3 They observed negative association between ambient $PM_{2.5}$ and respiratory function (FVC and FEV_1) in the COPD panel. The effect on FVC was seen at lag 24 h, 48 h, and 72 h. The effect on FEV_1 was evident at lag 72 h. There was no statistically significant effect of $PM_{2.5}$ on FVC and FEV_1 in the asthmatic and IHD panels. β **Coefficient (SE)** **COPD** FVC(%) 24 h -0.80 (0.36); 48-h -0.89 (0.41); 72-h -1.10 (0.55) FEV_1(%) 24 h -0.47 (0.33); 48-h -0.69 (0.37); 72-h -1.06 (0.50) **Asthma** FVC(%) 24 h -0.14 (0.29); 48-h -0.07 (0.33); 72-h -0.06 (0.39) FEV_1(%) 24 h -0.30 (0.34); 48-h -0.36 (0.39); 72-h -0.40 (0.46)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lee et al. (2007b) **Period of Study:** 2000-2001 **Location:** South-Western Seoul Metropolitan area, Seoul, South Korea	**Outcome:** PEFR (peak expiratory flow rate), lower respiratory symptoms (cold, cough, wheeze) **Age Groups**: 61-89 years of age (77.8 mean age) **Study Design:** longitudinal panel survey **N:** 61 adults **Statistical Analyses:** SAS MIXED, logistic regression model **Covariates:** Temperature (Celcius), relative humidity, age, **Dose-response Investigated?** No **Statistical Package**: SAS 8.0 **Lags Considered:** 0-4 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 51.15 (19.94) **Percentiles:** 25th: 33.00 50th(Median): 53.20 75th: 87.54 **Range (Min, Max):** 17.94, 92.71 **Monitoring Stations**: 2	**PM Increment:** 10 μg/m^3 **Effect Estimate [Lower CI, Upper CI] ; lag:** PEFR (peak expiratory flow rate) -0.54 (-0.89,-0.19); 1 day relative odds of a lower respiratory symptom (cold, cough, wheeze) 0.976 (0.849,1.121) ; 1 day
Reference: Lewis et al. (2005) **Period of Study:** winter 2001-spring 2002 **Location:** Detroit, Michigan, USA	**Outcome:** Poorer lung function (increased diurnal variability and decreased forced expiratory volume) **Age Groups**: 7-11 years old **Study Design:** Longitudinal cohort study **N:** 86 children **Statistical Analyses:** Descriptive statistics and bivariate analyses of exposures, multivariable regression multivariate analog of linear regression. **Covariates:** Sex, home location, annual family income, presence of one or more smokers in household, race, season (entered as dummy variables), and parameters to account for intervention group effect. **Season:** Winter 2001 (February 10–23), Spring 2001 (May 5–18), Summer 2001 (July 14–27), Fall 2001 (September 22–October 5), Winter 2002 (January 18–31), and Spring 2002 (May 18–31)]. **Dose-response Investigated?** No **Lags Considered:** 1 to 2 days, 3-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2 weeks **Mean (SD):** Eastside 15.7 (10.6) Southwest 17.5 (12.2) **Range (Min, Max):** 1.0, 56.1 **Monitoring Stations**: 2 **Copollutant (correlation):** PM_{10} 0.93 O_3 Daily mean 0.57 O_3 8-h peak 0.53	**PM Increment:** 12.5 μg/m^3 **RR Estimate [Lower CI, Upper CI] ; lag:** Lung function among children reporting use of maintenance CSs **Diurnal variability** FEV_1 Lag 1: 1.61 [-0.5,3.72] Lag 1: 0.99 [-5.64, 7.62] $PM_{2.5}$ + O_3 Lag 2: 2.96 [-1.74,7.66] Lag 2: 4.62 [-4.31, 13.54] $PM_{2.5}$ + O_3 Lag 3-5: 1.37 [-1.49,4.22] Lag 3-5: 2.70 [1.0, 4.40] $PM_{2.5}$ + O_3 **Lowest daily value** FEV_1 Lag 1: -2.23 [-6.99,2.53] Lag 1: 3.36 [-3.92, 10.63] $PM_{2.5}$ + O_3 Lag 2: -0.21 [-4.09,3.68] Lag 2: 0.88 [-8.69, 10.46] $PM_{2.5}$ + O_3 Lag 3-5: -0.76 [-5.00, 3.49] Lag 3-5: -2.78 [-4.87 to -0.70] $PM_{2.5}$ + O_3 Lung function among children reporting presence of URI on day of lung function assessment **Diurnal variability** FEV_1 Lag 1: 4.08 [-1.78, 9.94] Lag 1: 3.99 [-2.76, 10.74] $PM_{2.5}$ + O_3 Lag 2: 7.62 [-0.49, 15.73] Lag 2: 4.10 [-1.41, 9.60] $PM_{2.5}$ + O_3 Lag 3-5: 1.47 [-7.73, 10.67] Lag 3-5: 3.81 [-1.83, 9.45] $PM_{2.5}$ + O_3 **Lowest daily value** FEV_1 Lag 1: -1.21 [5.62,3.21] Lag 1: -0.74 [-4.14, 2.65] $PM_{2.5}$ + O_3 Lag 2: -0.10 [4.36,4.16] Lag 2: -1.67 [-5.09, 1.75] $PM_{2.5}$ + O_3 Lag 3-5: -2.88 [-5.46 to -0.30] Lag 3-5: -2.78 [-4.79 to -0.77] $PM_{2.5}$ + O_3

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Mar et al. (2004) **Period of Study:** 1997-1999 **Location:** Spokane, Washington	**Outcome:** Respiratory Symptoms **Age Groups**: Adults: Ages 20-51 yrs; Children: Ages 7-12 yrs **N:** 25 people **Statistical Analyses:** Logistic regression **Covariates:** Temperature, relative humidity, day of-the-wk **Statistical Package**: STATA 6 **Lags Considered:** 0-2 days	**Pollutant:** $PM_{2.5}$ **Mean (SD):** 1997: 11.0 (5.9) 1998: 10.3 (5.4) 1999: 8.1 (3.8) Unit (i.e. µg/m³): **Monitoring Stations**: 1 station **Copollutant (correlation):** $PM_{2.5}$ PM_1; r = 0.92 PM_{10}; r = 0.61 $PM_{10-2.5}$; r = 0.28	**PM Increment:** 10 µg/m³ **OR Estimate [Lower CI, Upper CI] ; lag:** **Adult Respiratory symptoms: Wheeze:** 1.04[0.86, 1.26]; lag 0 ; 1.00[0.83, 1.19]; lag 1; 0.99[0.84, 1.17]; lag 2 **Breath:** 0.97[0.87, 1.08]; lag 0 ; 0.98[0.87, 1.10]; lag 1; 0.95[0.80, 1.13]; lag 2 **Cough**: 0.86[0.62, 1.21]; lag 0; 0.87[0.63, 1.20]; lag 1; 0.89[0.66, 1.20]; lag 2 **Sputum**: 0.94[0.63, 1.41]; lag 0; 0.90[0.62, 1.31]; lag 1; 0.92[0.66, 1.27]; lag 2 **Runny Nose:** 0.98[0.83, 1.15]; lag 0; 0.95[0.82, 1.10]; lag 1; 0.93[0.80, 1.08]; lag 2 **Eye Irritation:** 0.91[0.70, 1.20]; lag 0; 0.89[0.70, 1.13]; lag 1; 0.86[0.68, 1.08]; lag 2 **Lower Symptoms:** 0.91[0.73, 1.13]; lag 0; 0.89[0.72, 1.10]; lag 1; 0.89[0.72, 1.10]; lag 2 **Any Symptoms:** 0.92[0.80, 1.07]; lag 0; 0.89[0.76, 1.04]; lag 1; 0.89[0.75, 1.05]; lag 2 **Children Respiratory symptoms: Wheeze:** 0.55[0.26, 1.19]; lag 0; 0.53[0.18, 1.58]; lag 1; 0.55[0.19, 1.64]; lag 2 **Breath:** 1.13[0.86, 1.48]; lag 0; 1.12[0.86, 1.44]; lag 1; 1.10[0.82, 1.48]; lag 2 **Cough:** 1.17[0.98, 1.40]; lag 0; 1.21[1.00, 1.47]; lag 1; 1.18[0.99, 1.42]; lag 2 **Sputum:** 1.06[0.92, 1.22]; lag 0; 1.10[0.91, 1.34]; lag 1; 1.09[0.92, 1.30]; lag 2 **Runny Nose**: 1.09[0.85, 1.39]; lag 0; 1.12[0.89, 1.41]; lag 1; 1.16[0.94, 1.42]; lag 2 **Eye Irritation:** 0.93[0.53, 1.64]; lag 0; 0.75[0.45, 1.27]; lag 1; 0.77[0.65, 0.91]; lag 2 **Lower Symptoms:** 1.18[1.00, 1.38]; lag 0; 1.21[1.00, 1.46]; lag 1; 1.17[0.96, 1.43]; lag 2 **Any Symptoms:** 1.17[1.03, 1.34]; lag 0; 1.22[1.04, 1.43]; lag 1; 1.23[1.07, 1.42]; lag 2
Reference: Mar et al. (2005b) **Period of Study:** 1999-2001 **Location:** Seattle, Washington	**Outcome:** Pulmonary function (arterial oxygen saturation) and cardiac function (heart rate and blood pressure) **Study Design:** Time series **Statistical Analyses:** Linear logistic regression **Age Groups**: > 57	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg	**Increment:** 10 µg/m³ **% Increase (Lower CI, Upper CI); Lag** **Personal:** Systolic: 0.37 (-0.93, 1.67); 0; Diastolic: -0.20 (-0.85, 0.46); 0 **Indoor:** Systolic: 0.92 (-2.04, 3.87); 0; Diastolic: 0.38 (-1.43, 2.20); 0 **Outdoor:** Systolic: -0.81 (-2.34, 0.73); 0; Diastolic: -0.46 (-1.49, 0.57); 0 % Increase between heart rate and $PM_{2.5}$ exposure for people > 57 **$PM_{2.5}$:** Personal: 0.44 (0.04, 0.84); 0; Indoor: 0.22 (-0.71, 1.16); 0; Outdoor: -0.75 (-1.42 to -0.07); 0
Reference: Mar et al. (2005a) **Period of Study:** 1999-2002 **Location:** Seattle, Washington	**Outcome**: Respiratory Symptoms **Age Groups**: 6-13 years **Study Design:** Time-Series **N:** 19 children **Statistical Analyses:** Polynomial distributed lag model, Poisson regression **Covariates:** Age, ambient NO levels, temperature, relative humidity, modification of use of inhaled corticosteroids **Season:** Winter, Spring **Dose-response Investigated?** No **Statistical Package**: STATA **Lags Considered:** 0-8 h	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** Results presented in Figure 1. **Monitoring Stations**: 3 Stations	**PM Increment:** 10 µg/m³ **Change in FE(NO) (exhaled NO concentration) with air pollution [Lower CI, Upper CI] ; lag:** Medication use: No meds: 6.99[3.43, 10.55]; lag 1-h Meds: -0.18[-3.33, 2.97]; lag 1-h No meds: 6.30[2.64, 9.97]; lag 4-h Meds: -0.77[-4.58, 3.04]; lag 4-h No meds: 0.46[-1.18, 2.11]; lag 8-h Meds: 0.40[-1.94, 2.74]; lag 8-h

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: McConnell et al. (2002) **Period of Study:** 1993-1998 **Location:** 12 communities in Southern California (grouped into either high and low pollution communities)	**Outcome:** Asthma (new diagnosis) **Age Groups**: 9-12 yrs, 12-13 yrs, 15-16 yrs **Study Design:** Cohort **N:** 3535 **Statistical Analyses:** Multivariate proportion hazard model **Covariates:** Sex, age, ethnic origin, BMI, child history of allergies and asthma history, SES, maternal smoking, time spent outside, history of wheezing, ownership of insurance (yes/no), number and type of sports played **Dose-response Investigated?** Yes **Statistical Package**: SAS 8.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 4 yrs **Mean (SD):** Low pollution communities: 7.6 (1.0) High pollution communities: 21.4 (6.0) **Percentiles:** Low pollution communities: 50th(Median): 7.7 High pollution communities: 50th(Median): 21.8 **Range (Min, Max):** Low pollution communities: 6.1, 8.6 High pollution communities: 13.5, 30.7 **Monitoring Stations**: 12 **Copollutant (correlation):** PM_{10}: r = 0.96; NO_2; O_3	**RR Estimate [Lower CI, Upper CI] ; lag:** **Low PM communities:** 1.0 [ref] 0 sport; 1.5 [1.0, 2.2] 1 sport; 1.2 [0.7, 1.9] 2 sports; 1.7 [0.9, 3.2] ≥3 sports **High PM communities:** 1.0 [ref] 0 sport; 1.1 [0.7, 1.7] 1 sport; 0.9 [0.5, 1.7] 2 sports; 2.0 [1.1, 3.6] ≥ 3 sports **High vs Low $PM_{2.5}$ communities: 0.8 (0.6, 1.0)** **Incidence–N (incidence) number of sports:** **Low PM communities:** 49 (0.023) 0; 54 (0.032) 1; 22 (0.024) 2; 13 (0.033) ≥3 **High PM communities:** 55 (0.021) 0; 36 (0.021) 1; 14 (0.018) 2; 16 (0.033)≥ 3
Reference: McCreanor et al. (2007) **Period of Study:** 2003-2005 **Location:** London, England	**Outcome**: Decreased Lung Function **Age Groups**: Adults **Study Design:** Crossover study **N:** 60 adults **Statistical Analyses:** Linear regression **Covariates:** Temperature, relative humidity, age, sex, bod-mass index, and race or ethnic group	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h **Mean (SD):** NR 50th(Median): Oxford St: 28.3 Hyde Park: 11.9 **Range (Min, Max):** Oxford St: (13.9, 76.1) Hyde Park: (3, 55.9)	% changes in FEV and FVC are presented in figures 1-3. Results are not presented quantitatively in text or tables. The authors did not find any significant differences in respiratory symptoms between the two locations. Also, there were no significant differences in sputum eosinophili counts or eosinophil cationic protein levels.
Reference: Moshammer and Neuberger (2003) **Period of Study:** 2000-2001 **Location:** Linz, Austria	**Outcome:** Lung Function: FVC, FEV_1, MEF_{25}, MEF_{50}, MEF_{75}, PEF, LQ Signal, PAS Signal **Age Groups**: Ages 7 to 10 **Study Design:** Case-crossover **N:** 161 children; 1898–2120 "half-h means" **Statistical Analyses:** Correlations; Regression Analysis **Covariates:** Morning, evening, night **Season:** Spring, Summer, Winter, Fall **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 8 h means & Daily Means **Mean (SD):** 14.61 (10.83) **Range (Min, Max):** (NR, 119.92) **Monitoring Stations**: 1 **Copollutant (correlation):** LQ = 0.751 PAS = 0.354	**Notes:** "Acute effects of 'active particle surface' as measured by diffusion charging were found on pulmonary function (FVC, FEV_1, MEF50) of elementary school children and on asthma-like symptoms of children who had been classified as sensitive."

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Moshammer et al. (2006) **Period of Study:** 2000-2001 **Location:** Linz, Austria	**Outcome**: Respiratory symptoms and decreased lung function **Age Groups**: Children ages 7-10 **Study Design:** Time-series **N:** 163 children **Statistical Analyses:** Generalized estimating equations model **Covariates:** Sex, age, height, weight **Dose-response Investigated?** NR **Statistical Package**: NR **Lags Considered:** 1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 8 h **Mean (SD):** Maximum 24 h: 76.39 Annual avg: 19.06 **Percentiles:** 8-h mean 25th: 8.64 8-h mean 50th(Median): 15.70 8-h mean 75th: 25.82 **Monitoring Stations**: 1 station **Copollutant (correlation):** PM_1; r = 0.95; PM_{10}; r = 0.93; NO_2; r = 0.54	**PM Increment:** 10 µg/m^3 **% change in Lung Function per 10** µg/m^3 FEV: 0.23; FVC: 0.08; $FEV_{0.5}$: 0.33; $MEF_{75\%}$: -0.49; $MEF_{50\%}$: -0.58; $MEF_{25\%}$: -0.83; PEF: 0.41 **% change in Lung Function per IQR** FEV: -0.59; FVC: -0.2; $FEV_{0.5}$: 0.85; $MEF_{75\%}$: -1.25; $MEF_{50\%}$: -1.48; MEF_{25}%: -2.14; PEF: -1.06 **Multiple pollutant model** FEV: 0.10; FVC: 0.21; $FEV_{0.5}$: 0.06; $MEF_{75\%}$: -0.15; MEF50%: 0.04; MEF25%: -0.21; PEF: -0.18 **% change in Lung Function per IQR** FEV: 0.27; FVC: 0.54; $FEV_{0.5}$: 0.15; $MEF_{75\%}$: -0.39; $MEF_{50\%}$: 0.11; $MEF_{25\%}$: 0.54; PEF: 0.015: -0.47
Reference: Murata et al. (2007) **Period of Study:** Nov 2nd- 12th 2004 **Location:** Tokyo, Japan	**Outcome:** Exhaled nitric oxide levels, (eNO), a marker of airway inflammation **Age Groups**: 5-10 years **Study Design:** Cohort/Panel study **N:** 19 schoolchildren* **Statistical Analyses:** Linear regression **Covariates:** None **Season:** November (fall) **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** Lag h 1-24, 8-h moving avg, 7-h moving avg, 6-h moving avg, 24-h moving avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Hourly, 24-h **Mean (SD):** 39.0 (16.9) (daily mean) **Range (Min, Max):** 10, 120 (range of hourly values) **Monitoring Stations**: 1, on the street where the children lived	**PM Increment:** IQR 110 µg/m^3 **Mean [Lower CI, Upper CI] ; lag:** 0.145 [0.62, 0.228] ppb eNO; 8 h moving avg **Notes:** Associations for lag h 1-24 presented in figures. Authors state "Individual hourly lag models showed a consistent association between the eNO value and $PM_{2.5}$ for exposure in the previous 24 h" "The trend on the graphs strongly suggest that fluctuations in eNO were affected by changes in air pollutants over at least the previous 8-h period" $PM_{2.5}$, black carbon, and NOx were all highly correlated (shown in figures), so effects are difficult to separate Pollutant concentrations peaked in the morning and evening h during traffic peaks
Reference: Neuberger et al. (2004) **Period of Study:** 6/1999-6/2000 **Location:** Austria (Vienna and a rural area near Linz)	**Outcome:** Questionnaire derived asthma score, and a 1-5 point respiratory health rating by parent **Age Groups**: 7-10 years **Study Design:** Cross-sectional survey **N:** about 2000 children **Statistical Analyses:** mixed models linear regression-used factor analysis to develop the "asthma score" **Covariates:** Pre-existing respiratory conditions, temperature, rainy days, # smokers in household, heavy traffic on residential street, gas stove or heating, molds, sex, age of child, allergies of child, asthma in other family members **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 4 week avg (preceding interview)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Copollutant (correlation):** PM_{10} (r=0.94) in Vienna	**PM Increment:** 10 µg/m^3 **Change in mean associated unit increase in PM (p-value); lag** Respiratory Health score Vienna: 0.016 (p>0.05); lag 4 week avg Rural area: 0.022 (p<0.05); lag 4 week avg Asthma score Vienna: 0.006 (p>0.05); lag 4 week avg Rural area: 0.004 (p>0.05); lag 4 week avg

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Neuberger et al. (2004) **Period of Study:** Sept 1999-March 2000 **Location:** Vienna, Austria	**Outcome:** Ratio measure: Time to peak tidal expiratory flow divided by total expiration time (i.e., tidal lung function, a surrogate for bronchial obstruction) **Age Groups**: 3.0-5.9 years (preschool children) **Study Design:** Longitudinal prospective cohort **N:** 56 children **Statistical Analyses:** mixed models linear regression, with autoregressive correlation structure **Covariates:** Age, sex, respiratory rate, phase angle, temperature, kindergarten, parental education, observer (also in sensitivity analyses: height, weight, cold/sneeze on same day, heating with fossil fuels, hair cotinine, number of tidal slopes used to measure tidal lung function) **Dose-response Investigated?** No **Statistical Package**: SAS 8.0 **Lags Considered:** Lag 0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **PM Component: T**otal carbon Elemental carbon Organic Carbon **Copollutant (correlation):** PM_{10} (r=0.94) in Vienna	**PM Increment:** Interquartile range (NR) **Change in mean associated with an IQR increase in PM (p-value); lag** $PM_{2.5}$ mass: -0.987 (0.091); lag 0 Total carbon: -0.815 (0.041); lag 0 Elemental carbon: -0.657 (0.126); lag 0 Organic carbon: -0.942 (0.025); lag 0
Reference: Neuberger et al. (2004) **Period of Study:** Oct. 2000-May 2001 **Location:** Linz, Austria	**Outcome**: Forced oscillatory resistance (at zero Hz), FVC, FEV_1, MEF_{25}, MEF_{50}, MEF_{75}, PEF **Age Groups**: 7-10 years **Study Design:** Longitudinal prospective cohort **N:** 164 children **Statistical Analyses:** **M**ixed models linear regression with autoregressive correlation structure **Covariates:** Sex, time and individual **Season:** October–May **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** Lag 0-7	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Monitoring Stations**: 1	**PM Increment:** 1 $\mu g/m^3$ **Notes:** Authors report increased oscillatory resistance significantly associated with $PM_{2.5}$ (lag 0)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: O'Connor et al. (2008) **Period of Study:** August 1998–July 2001 **Location:** Boston, the Bronx, Chicago, Dallas, New York, Seattle, Tucson	**Outcome:** Pulmonary function and respiratory symptoms **Age Groups**: 5-12 years **Study Design:** Inner-City Asthma Study (ICAS)–Panel/cohort study **N:** 861 children **Statistical Analyses:** Mixed effects models **Lags Considered:** Lag 0-6, 0-4	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** 14 **Range (Min, Max):** 5-35 (estimated from figure) **Copollutant (correlation):** NO_2 (r=0.59) SO_2 (r=0.37) CO (r=0.44) O_3 (r=-0.02)	**PM Increment:** 13.2 µg/m^3 90th-10th percentile **Change in pulmonary function; lag** FEV_1: -1.47 (-2.00 to -0.94); lag 0-4 PEFR: -1.10 (-1.65 to -0.56); lag 0-4 **$PM_{2.5}$+O_3+NO_2** FEV_1: -0.73 (-1.33 to -0.12); lag 0-4 PEFR: -0.25 (-0.88, 0.38); lag 0-4 **Risk of Respiratory Symptoms; lag** Wheeze: 0.98 (0.88, 1.09); lag 0-4 Nighttime asthma: 1.11 (0.94, 1.30); lag 0-4 Slow play: 1.01 (0.89, 1.15); lag 0-4 Missed school: 1.33 (1.06, 1.66); lag 0-4 **$PM_{2.5}$+O_3+NO_2** Wheeze: 0.92 (0.81, 1.05); lag 0-4 Nighttime asthma: 1.03 (0.86, 1.23); lag 0-4 Slow play: 0.92 (0.79, 1.06); lag 0-4 Missed school: 1.13 (0.87, 1.45); lag 0-4
Reference: Peacock et al. (2003) **Period of Study:** November 1, 1996 to 14 February 1997 **Location:** northern Kent, UK	**Outcome:** Reduced peak expiratory flow rate (PEFR) **Age Groups**: 7-13 years of age **Study Design:** Time Series **N:** 179 **Statistical Analyses:** generalized estimating equations **Covariates:** Day of the week, 24-h mean outside temperature. **Season:** Winter **Dose-response Investigated?** No **Statistical Package**: STATA **Lags Considered:** Same day, lag 1, lag 2, five day moving avg	**Pollutant:** Sulfate (SO_4^{2-}) **Averaging Time:** Daily avg **Mean (SD):** Urban 2 24 h avg: 1.3 (1.1) **Percentiles:** 10th: Urban 2 0.5 90th: Urban 2 2.4 **Range (Min, Max):** Urban 2 0.3, 6.7 Unit (i.e. µg/m^3): µg/m^3 **Monitoring Stations**: 3	Sulfate (SO_4^{2-}) Increment: 1.3 µg/m^3 **Odds ratio [Lower CI, Upper CI] ; lag:** 1.090 [0.898, 1.322] ; 5 days
Reference: Peled, et al. (2005) **Period of Study:** 5-6 weeks between March-June 1999 and September-December 1999. **Location:** Ashdod, Ashkelon and Sderot, Israel	**Outcome:** Reduced peak expiratory flow (PEF) **Age Groups**: 7-10 years **Study Design:** Nested cohort study **N:** 285 **Statistical Analyses:** Time series anaylsis; Generalized linear model, generalized estimating equations, one-way ANOVA, generalized linear model **Covariates:** Seasonal changes, meteorological conditions and personal physiological, clinical and socioeconomic measurements **Season:** Spring, Autumn **Dose-response Investigated?** No **Statistical Package**: STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily **Mean:** Ashkelon: 24.0 Sderot: 29.2 Ashdod: 23.9 **PM Component:** Local industrial emissions, desert dust, vehicle emissions and emissions from two electric power plants **Monitoring Stations**: 6 **Copollutant:** PM_{10}	**PM Increment:** 1 µg/m^3 β coefficient (SE) [95% CI] Ashkelon: $PM_{2.5}$ MAX: -0.144 (0.12) [-0.38-0.09] Ashdod: $PM_{2.5}$ MAX: -2.74 (0.61) [-3.95-1.53] $PM_{2.5}$ MAX x TMAX: 0.11 (0.02) [0.06-0.16] In Ashdod, $PM_{2.5}$ and an interaction between $PM_{2.5}$ and temperature were significantly associated.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Penttinen et al. (2006) **Period of Study:** 11/1996–4/1997 **Location:** Helsinki, Finland	**Outcome:** Decreased lung function and respiratory symptoms **Age Groups**: Adults, mean age 53 years **Study Design:** Time Series **N:** 78 people **Statistical Analyses:** Generalized least squares autoregressive model **Covariates:** Temperature, relative humidity, day of study, day of study squared, binary dummy variable for weekends **Season:** Winter, Spring **Dose-response Investigated?** NR **Statistical Package**: SAS version 6 **Lags Considered:** 0-3	**Pollutant:** $PM_{2.5}$ **PM Component:** Soil, heavy fuel oil, sea salt **Averaging Time:** 24 h **Percentiles: 25th:** Long range transport: 2.44 Local combustion: 1.75 Soil: 0.14 Heavy fuel oil: -0.13 Sea Salt: 0.22 Unidentifiable: -1.41 All sources: 6.47 **50th(Median):** Long range transport: 4.15 Local combustion: 2.41 Soil: 0.64 Heavy fuel oil: 0.10 Sea Salt: 0.27 Unidentifiable: 0.02 All sources: 8.37 **75th:** Long range transport: 7.33 Local combustion: 3.05 Soil: 1.46 Heavy fuel oil: 0.52 Sea Salt: 0.42 Unidentifiable: 0.74 All sources: 11.15 **Range (Min, Max):** Long range transport: (-0.89, 28.31) Local combustion: (0.83, 6.51) Soil: (-1.13, 6.43) Heavy fuel oil: (-0.67, 4.74) Sea Salt: (0.09, 0.98) Unidentifiable: (-4.40, 4.77) All sources: (4.11, 33.53) **Monitoring Stations**: 1 site	**PM Increment:** 1.3 µg/m^3 **$PM_{2.5}$, long range: PEF Morning:** 0.37[-0.59, 1.34]; lag 0; -1.04[-1.88 to -0.19]; lag 1; -0.82[-1.81, 0.16]; lag 2; 0.22[-0.64, 1.08]; lag 3; -0.24[-1.12, 0.64]; 5 day mean. **PEF Afternoon:** 0.20[-0.67, 1.06]; lag 0; -0.20[-1.24, 0.83]; lag 1; -0.30[-1.14, 0.53]; lag 2; 0.45[-0.57, 1.47]; lag 3; 0.03[-0.79, 0.85]; 5 day mean. **PEF Evening:** -0.33[-1.30, 0.64]; lag 0; -0.29[-1.13, 0.55]; lag 1; -0.41[-1.46, 0.64]; lag 2; 0.39[-0.47, 1.24]; lag 3; 0.07[-0.81, 0.95]; 5 day mean **$PM_{2.5}$, local combustion: PEF Morning:** -0.73[-1.69, 0.23]; lag 0; -0.46[-1.24, 0.32]; lag 1; -0.43[-1.49, 0.63]; lag 2; 0.34[-0.47, 1.15]; lag 3; -0.25[-1.03, 0.53]; 5 day mean. **PEF Afternoon:** -0.21[-1.07, 0.65]; lag 0; -0.81 [-1.77, 0.16]; lag 1; -0.83[-1.74, 0.09]; lag 2; 0.20[-0.80, 1.20]; lag 3; -0.87[-1.63 to -0.12]; 5 day mean. **PEF Evening:** -0.51[-1.48, 0.45]; lag 0; -1.16[-1.93 to -0.39]; lag 1; 0.23[-1.35, 0.90]; lag 2; 0.56[-0.21, 1.32]; lag 3; -1.14[-1.95 to -0.33]; 5 day mean **$PM_{2.5}$, soil: PEF Morning:** 0.81[0.05, 1.57]; lag 0; 0.03 [-0.65, 0.71]; lag 1; 0.50[-0.34, 1.35]; lag 2; -0.07[-0.74, 0.61]; lag 3; 0.39[-0.46, 1.23]; 5 day mean. **PEF Afternoon:** 1.05[0.38, 1.72]; lag 0; 0.40[-0.38, 1.19]; lag 1; 0.66 [0.03, 1.30]; lag 2; -0.36[-1.12, 0.41]; lag 3; 0.55 [-0.21, 1.32]; 5 day mean. **PEF Evening:** 1.08[0.33, 1.84]; lag 0; 1.00[0.31, 1.68]; lag 1; 0.33[-0.56, 1.22]; lag 2; -0.84 [-1.53 to -0.15]; lag 3 0.90[0.08, 1.73]; 5 day mean **$PM_{2.5}$, oil: PEF Morning:** -0.22[-1.00, 0.56]; lag 0; -0.20[-1.24, 0.84]; lag 1; 0.66[-0.68, 2.00]; lag 2; 0.57 [-0.18, 1.32]; lag 3; 0.10[-0.61, 0.81]; 5 day mean. **PEF Afternoon:** -0.04[-0.75, 0.67]; lag 0; 0.29[-0.98, 1.55]; lag 1; 0.08 [-1.13, 1.28]; lag 2; 0.62[-0.31, 1.54]; lag 3; 0.07 [-0.64, 0.78]; 5 day mean. **PEF Evening:** 0.57[-0.23, 1.37]; lag 0; 0.12[-0.92, 1.15]; lag 1; -0.97[-2.39, 0.45]; lag 2; 0.40[-0.31, 1.12]; lag 3; 0.43[-0.33, 1.19]; 5 day mean **$PM_{2.5}$, salt: PEF Morning:** 0.76[-0.13, 1.65]; lag 0; 0.43 [-0.30, 1.16]; lag 1; 0.13[-0.75, 1.02]; lag 2; 0.38[-0.47, 1.23]; lag 3; 0.95[-0.18, 2.09]; 5 day mean. **PEF Afternoon:** 0.62[-0.18, 1.41]; lag 0; 0.80[-0.08, 1.69]; lag 1; 0.14[-0.62, 0.90]; lag 2; 0.16[-0.83, 1.15]; lag 3; 0.88 [-0.18, 1.94]; 5 day mean. **PEF Evening:** 1.09[0.19, 1.98]; lag 0; 0.63[-0.10, 1.35]; lag 1; 0.32[-0.62, 1.26]; lag 2; -0.31[-1.16, 0.54]; lag 3; 0.88[-0.27, 2.02]; 5 day mean **$PM_{2.5}$, unidentified: PEF Morning:** 0.38[-0.67, 1.43]; lag 0; 0.09[-0.83, 1.00]; lag 1; 0.22[-0.82, 1.26]; lag 2; 0.78 [-0.10, 1.66]; lag 3; 0.78[-0.14, 1.69]; 5 day mean. **PEF Afternoon:** 0.02[-0.92, 0.96]; lag 0; 0.65[-0.48, 1.77]; lag 1; 0.17[-0.71, 1.05]; lag 2; 0.69[-0.36, 1.75]; lag 3; 0.17 [-0.72, 1.06]; 5 day mean. **PEF Evening:** -0.11[-1.17, 0.95]; lag 0; 0.19[-0.72, 1.10]; lag 1; 0.86[-0.25, 1.96]; lag 2; 0.15[-0.70, 1.01]; lag 3; -0.19[-1.15, 0.77]; 5 day mean **$PM_{2.5}$, local combustion**: **PEF morning:** Cu: -0.25 [-1.25, 0.75]; Zn: -0.45[-1.19, 0.29]; Mn: 0.13[-0.83, 1.08]; Fe: 0.08[-0.70, 0.85]. **PEF afternoon:** Cu: -0.37[-1.29, 0.55]; Zn: -0.19[-0.87, 0.50]; Mn: -0.48[-1.37, 0.42]; Fe: 0.29[-0.45, 1.04]. **PEF evening:** Cu: -0.48[-1.47, 0.52]; Zn: -0.17[-0.92, 0.57]; Mn: 0.51[-0.44, 1.47]; Fe: 0.34[-0.46, 1.14] **$PM_{2.5}$, long range: PEF morning:** S: 0.11[-0.886, 1.07]; K: -0.10[-1.00, 0.80]; Pb: -0.62[-1.37, 0.13]; Br: -0.40 [-1.40, 0.60]. **PEF afternoon:** S: -0.05[-0.92, 0.81]; K: 0.26[-0.56, 1.07]; Pb: -0.12[-0.84, 0.60]; Br: 0.15[-0.81, 1.12]. **PEF evening:** S: 0.08[-0.86, 1.02]; K: 0.18[-0.70, 1.07];Pb: -0.20[-0.97, 0.58]; Br: 0.35[-0.71, 1.40] **$PM_{2.5}$, soil:PEF morning:** Si: 0.27[-0.43, 0.97]; Al: 0.17 [-0.72, 1.05]; Ca: 0.13[-1.08, 1.35]. **PEF afternoon:** Si: 0.39[-0.24, 1.01]; Al: 0.49[-0.29, 1.27]; Ca: 0.15[-0.92, 1.22] **PEF evening"** Si: 0.60[-0.06, 1.26]; Al: 0.76[-0.08, 1.60]; Ca: 0.90[-0.22, 2.03] **$PM_{2.5}$, Oil combustion: PEF morning:** V: -0.01[-0.87, 0.86]; Ni: -0.09[-1.08, 0.90]. **PEF afternoon:** V: -0.48[-1.32, 0.35]; Ni: 0.26[-0.72, 1.23]. **PEF evening:** V: 0.02[-00.88, 0.92]; Ni: 0.50[-0.55, 1.55] **$PM_{2.5}$, Sea salt: PEF morning:** Na: 0.92[-0.34, 2.17]; Cl: 0.93[0.08, 1.79]; **PEF afternoon:** Na: 0.96[-0.24, 2.16]; Cl: 0.57[-0.22, 1.36] **PEF evening** Na: 0.87[-0.40, 2.15]; Cl: 0.65[-0.19, 1.49]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Pino et al. (2004) **Period of Study:** 4/1995–10/1996 **Location:** Santiago, Chile	**Outcome:** Respiratory Symptoms, Wheezing bronchitis **Study Design:** Time-series **Statistical Analyses:** Bayesian hierarchical analysis, cubic spline **Age Groups**: 4 months–2 years old	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD) unit:** 52.0 (31.6) **Range (5th, 95th):** 17.0, 114.0 **Copollutants (correlation):** SO_2: r= 0.73 NO_2: r= 0.85	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** % increase in wheezing bronchitis and $PM_{2.5}$ exposure for infants 4 months to 2 years old 4.75 (1.25, 8.25); 1 3.85 (0.45, 7.75); 2 2.25 (-1.00, 6.00); 3 1.75 (-2.20, 5.75); 4 4.00 (0.25, 8.00); 5 5.00 (1.00, 8.50); 6 7.00 (3.50, 11.00); 7 8.10 (4.00, 11.25); 8 9.00 (6.00, 12.00); 9 8.75 (5.75, 12.00); 10 1.50 (-3.50, 4.75); 11 0.25 (-3.75, 4.25); 12 0.00 (-4.00, 4.00); 13 1.00 (-3.50, 4.50); 14 1.50 (-3.50, 4.50); 15 OR for wheezing bronchitis and $PM_{2.5}$ exposure in infants 4 months to 2 years old according to family history of asthma Yes to family history of asthma 1.09 (1.00, 1.19); 1 1.10 (1.02, 1.20); 2 1.11 (1.02, 1.22); 3 No to family history of asthma 1.04 (1.00, 1.08); 1 1.02 (0.98, 1.06); 2 1.01 (0.96, 1.05); 3

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Rabinovitch et al., (2006) **Period of Study:** 2001-2003 (two winters 2001-2002 and 2002-2003) **Location:** Denver, CO	**Outcome**: Bronchodilator doser activations (daily) and urinary leukotriene E4 (daily) **Age Groups**: Children 6-13 years old **Study Design: S**chool-based cohort study **N:** 73 children **Statistical Analyses:** Doser activation: Poisson regression with GEE with AR1 working covariance; Urinary leukotriene E4: linear mixed model with spatial exponential covariance **Covariates:** Temperature, pressure, humidity, time trend, Friday indicator, upper respirtory infection (URI), height (leukotriene E4 only). **Season:** Winter **Dose-response Investigated?** NR **Statistical Package**: SAS **Lags Considered:** 0-2 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Morning (midnight to 11:00 AM) mean Morning (midnight to 11:00 AM) maximum 24-h mean **Mean (SD):** 24-h mean, TEOM **Year 1, N: 55 days** 6.5 (3.2) **Year 2, N: 128 days** 8.2 (3.7) 24-h mean, FRM **Year 1, N: 55 days:**11.8 (7.2) **Year 2, N: 122 days:** 11.2 (5.5) Morning mean, TEOM **Year 1, N: 71 days:** 7.4 (4.7) **Year 2, N: 127 days:** 9.1 (5.0) Morning maximum, TEOM **Year 1, N: 71 days:** 15.5 (9.5) **Year 2, N: 127 days:** 18.4 (9.6) **Percentiles**: 24-h mean, TEOM **Year 1** 25th: 4.4; 50th(Median): 6.2; 75th: 7.9 **Year 2** 25th: 55; 50th(Median): 7.3; 75th: 9.9 24-h mean, FRM **Year 1** 25th: 7.8; 50th(Median): 10.1; 75th: 14.1 **Year 2** 25th: 7.5; 50th(Median): 9.3; 75th: 13.3 Morning mean, TEOM **Year 1** 25th: 4.0; 50th(Median): 5.9; 75th: 9.6 **Year 2** 25th: 5.2; 50th (Median): 8.5; 75th: 11.6 Morning maximum, TEOM **Year 1** 25th: 8; 50th (Median): 13; 75th: 20 **Year 2** 25th: 11; 50th (Median): 16; 75th: 23 **Range (Min, Max):** 24-h mean, TEOM Year 1 (2.1, 23.7) Year 2 (1.7, 20.5) 24-h mean, FRM Year 1 (4.3, 53.5) Year 2 (3.4, 26.3) Morning mean, TEOM Year 1 (1.4, 22.7) Year 2 (1.6, 30.2) Morning maximum, TEOM Year 1 (4, 42) Year 2 (4, 46) **Monitoring Stations**: 2 (1 TEOM and 1 Federal Reference Monitor [FRM])	**PM Increment:** IQR (over current and previous day) **Doser Activation** **Morning avg $PM_{2.5}$ TEOM** Year 1: Pct Increase: 3.0 [-0.5:6.6] p = 0.10 Year 2: Pct Increase: 2.7 [1.1:4.4] p = 0.006 Aggregated years: 2.2 [0.7:3.6] p = 0.005 **Morning max $PM_{2.5}$ TEOM** Year 1 Pct Increase: 4.0 [0.5:7.6] p = 0.02 Year 2 Pct Increase: 2.3 [0.7:4.0] p = 0.009 Aggregated years 2.6 [0.9:4.2] p= 0.002 **24-h $PM_{2.5}$** **TEOM** Lag 0: 0.4 [-0.7:1.6] p-value = 0.45 Lag 1: 0.9 [-0.7:2.4] p-value = 0.27 Lag 2: -0.4 [-1.7:0.9] p-value = 0.59 Lag 0-2 Avg: 0.6 [-1.0:2.2] p-value = 0.43 **FRM** Lag 0: 0.2 [-1.2:1.6] p-value = 0.81 Lag 1: 0.9 [-0.9:2.6] p-value = 0.31 Lag 2: -0.2 [-2.2:1.8] p-value = 0.88 Lag 0-2 Avg: 1.2 [-0.6:2.9] p-value = 0.20 **Morning avg $PM_{2.5}$** **TEOM** URI not adjusted Mild/Moderate Asthmatics: 1.5 [-0.5:3.4] p = 0.14 Severe Asthmatics: 3.7 [1.6:5.8] p- = 0.0006 Difference between severity groups, p = 0.12 Aggregated severity group: 2.2 [0.7:3.6] p= 0.005 URI adjusted Mild/Moderate Asthmatics: 1.0 [-1.9:3.9]p= 0.50 Severe Asthmatics: 6.0 [1.8:10.1] p = 0.006 Difference between severity groups, p = 0.08 Aggregated severity groups: 2.7 [-0.1:5.4] p= 0.06 **Morning maximum $PM_{2.5}$** **TEOM** URI not adjusted Mild/Moderate Asthmatics: 1.9 [-0.2:4.1] p= 0.07 Severe Asthmatics: 3.9 [1.1:6.8] p = 0.006 Difference between severity groups, p = 0.29 Aggregated severity groups: 2.6 [0.9:4.2] p= 0.002 URI adjusted Mild/Moderate Asthmatics: 1.6 [-2.2:5.4] p = 0.41 Severe Asthmatics: 8.1 [2.9:13.4] p = 0.003 Difference between severity groups, p = 0.03 Aggregated severity groups: 3.8 [0.2:7.4] p = 0.04 **Leukotriene E4** **24-h $PM_{2.5}$** **TEOM** Lag 0: 3.3 [-0.7:7.2] p = 0.09 Lag 1: -1.6[-5.7:2.5] p = 0.40 Lag 2: 1.1 [-2.8:5.1] p= 0.64 Lag 0-2 Avg: 2.3 [-4.0:8.6] p = 0.45 **FRM** Lag 0: 2.7 [1.1:6.5] p = 0.12 Lag 1: -0.8 [-4.9:3.3] p = 0.65 Lag 2: -0.8 [-4.9:3.3] p = 0.71 Lag 0-2 Avg: 2.6 [-2.3:7.5] p = 0.27 **Leukotriene E4** **Morning avg $PM_{2.5}$ TEOM** Height 25%ile: 8.9 [3.0:14.7] p= 0.004 Height 50%ile: 5.9 [1.4:10.4] p = 0.01 Height 75%ile: 1.9 [-3.4:7.3] p = 0.47 Model w/o Height × Pollutant: 5.6 [1.0:10.2] p = 0.02 **Morning maximum $PM_{2.5}$** **TEOM** Height 25%ile: 8.3 [3.4:13.2] p = 0.001 Height 50%ile: 6.1 [2.1:10.2] p= 0.004 Height 75%ile: 3.2 [-2.0:8.4] p= 0.23 Model w/o Height × Pollutant: 6.2 [1.9:10.5] p = 0.006

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Rabinovitch et al. (2004) **Periods of Study:** 11/15/1999–3/15/2000 11/13/2000–3/23/2001 11/15/2001–3/22/2002 **Location:** Denver, Colorado	**Outcome:** Respiratory symptoms, Asthma symptoms (cough and wheeze), Upper respiratory symptoms **Study Design:** Time-series **Statistical Analyses:** Logistic linear regression, PROC Mixed, PROC Genmod **Age Groups**: 6-12	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 10.8 (7.1) **Range (Min, Max):** (1.8, 53.5) **Copollutant (correlation):** CO; NO_2; SO_2 ; O_3	**PM Increment:** 1 µg/m^3 β (SE) AM: -0.003 (0.009) PM: 0.004 (0.011) Odds Ratio (Lower CI, Upper CI); Lag 0.971 (0.843, 1.118); 0-3 avg.
Reference: Ranzi et al. (2004) **Period of Study:** February-May 1999 **Location:** Emilia-Romagmna, Italy (urban-industrial and rural area)	**Outcome:** respiratory symptoms, PEF measurements, drug consumption and daily activity **Age Groups**: Children, mean age=(7.2-7.9 yrs) **Study Design:** Panel study **N:** 120 children **Statistical Analyses:** Ecological analysis and Panel analysis **Covariates:** Temperature, humidity, gender, medicinal use, symptomatic status of previous day **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 0-3 mov avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Urban= 53.07 Rural= 29.11 **Monitoring Stations**: 3 **Copollutant (correlation):** TSP: r=0.613 daily air pollution concentrations: r= 0.658	**PM Increment:** 10 µg/m^3 Effect Estimate: Urban-industrial panel Cough and Phlegm: RR=1.0044 (1.0011-1.0077)
Reference: Rodriguez et al. (2007) **Period of Study:** 1996-2003 **Location:** Perth, Australia	**Outcome:** Body temperature, cough, runny/blocked nose, wheeze/rattle chest (daily) **Age Groups**: Children 0-5 years old **Study Design:** hospital-based cohort study **N:** 198-263 children **Statistical Analyses:** Logistic regression with GEE and AR (order not specified) working covariance **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** 0-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1-h and 24-h **Mean (SD):** 1-h averaging, 20.767 24-h averaging, 8.534 **Range (Min, Max):** 1-h averaging (0.012:93.433) 24-h averaging (0.004:39.404) **Monitoring Stations**: 10 total, usually 3-5 sites for each pollutant **Copollutant (correlation):** O_3 NO+ CO	**PM Increment:** NR [Lower CI, Upper CI] ; lag: NR LAG: 0 day **$PM_{2.5}$, 1-h** Body temperature: 1.004 [0.998:1.011] Cough: 1.006 [1.000:1.012] Wheeze/rattle chest: 1.004 [0.998:1.010] Runny/blocked nose: 0.997 [0.983:1.010] **$PM_{2.5}$, 24-h** Body temperature: 1.005 [0.986:1.024] Cough: 1.019 [0.999:1.040] Wheeze/rattle chest: 0.990 [0.969:1.012] Runny/blocked nose: 0.968 [0.926:1.013] LAG: 5 days **$PM_{2.5}$, 1-h** Body temperature: 1.005 [0.999:1.040] Cough: 1.003 [0.995:1.010] Wheeze/rattle chest: 1.005 [0.998:10.12] Runny/blocked nose: 1.015 [1.000:1.030] **$PM_{2.5}$, 24-h** Body temperature: 1.020 [0.998:1.011] Cough: 1.006 [0.984:1.011] Wheeze/rattle chest: 1.018 [0.997:1.040] Runny/blocked nose: 1.039 [0.990:1.089] LAG: 0-5 days **$PM_{2.5}$, 1-h** Body temperature: 1.000 [0.998:1.002] Cough: 1.001 [0.999:1.003] Wheeze/rattle chest: 1.002 [1.000:1.004] Runny/blocked nose: 1.001 [0.997:1.006] **$PM_{2.5}$, 24-h** Body temperature: 1.000 [0.994:1.005] Cough: 1.004 [0.997:1.011] Wheeze/rattle chest: 1.001 [0.995:1.007] Runny/blocked nose: 0.998 [0.985:1.011]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Silkoff et al. (2005) **Period of Study:** Winter 1999-2000, Winter 2000-2001 **Location:** Denver, CO	**Outcome:** Lung function: FEV_1, PEF **Age Groups**: Adults (>40 years-old) with COPD, as well as >10 pack-years tobacco use, FEV_1 < 70%, FEV_1/FVC < 60%, and no other lung disease **Study Design:** COPD patient panel study (2 independent panels; one for each winter) **N:** 34 subjects (16 1st winter, 18 second winter) **Statistical Analyses:** mixed effects mdels with first-order, autoregressive, moving avg variance-covariance; binary outcomes (rescue medication use, total symptom score) assessed using Poisson regression with GEE and first-order, auto-regressive variance-covariance **Covariates:** temperature, relative humidity, barometric pressure; analysis run separately for each winter **Season:** Winter **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** 0-2 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** Winter 1999-2000: 9.0 (5.2) Winter 2000-2001: 14.3 (9.6) **Percentiles:** Winter 1999-2000 25th 5.4 50th(Median): 7.7 75th: 11.3 Winter 2000-2001 25th 7.6 50th(Median): 11.7 75th: 17.2 **Range (Min, Max):** Winter 1999-2000 (1.8, 36.6) Winter 2000-2001 (3.4, 59.6) **Monitoring Stations**: multiple sites **Copollutant (correlation):** CO NO_2 PM_{10}	**PM Increment:** SD Winter 1999-2000: 5.2 Winter 2000-2001: 9.6 Model results reported graphically only. No quantitative results reported. Direction of slope (+/-) and statistical significance (SIG: yes; NS: no) inferred from graphs. Among subjects with severe COPD observed in Winter 1999-2000, statistically significant, but marginal, improvements in PEF associated with morning lag 0 $PM_{2.5}$. There were no statistically significant associations between rescue medication use and symptom score with PM.
Reference: Sivacoumar et al. (2006a) **Period of Study:** 4/1998–5/1998 9/1998–10/1998 **Location:**Pammal, India	**Outcome:** Respiratory symptoms, Decreased pulmonary function **Study Design:** Case-control **Statistical Analyses:** Poisson **Age Groups**: > 18	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg	The study does not present quantitative results of association.
Reference: Slaughter et al. (2003) **Period of Study:** 1994 **Location:** Seattle, WA	**Outcome:** Asthma attacks, asthma severity, medication use **Age Groups**: 5.1 to 13.1 years old **Study Design:** Cross-sectional study **N:** 133 children **Statistical Analyses:** Ordinal Logistic Regression Poisson Modeling **Covariates:** Temperature, Day of the Week, Seasonality **Dose-response Investigated?** No **Statistical Package**: STATA **Lags Considered:** 1, 2, 3 day lag	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily Averages 25th: 5.0 50th(Median): 7.3 [3] 75th: 11.3 **Monitoring Stations**: 3 **Copollutant (correlation):** PM_{10} = 0.75 CO = 0.82	**PM Increment:** 10 µg/m^3 increase RR Estimate [Lower CI, Upper CI] ; lag: Inhaler use: 1-day lag: 1.04 (0.98, 1.10) OR Estimate [Lower CI, Upper CI] ; lag: Asthma Attack: 1-day lag: 1.20 (1.05, 1.37) Previous day: 1.13 (1.03, 1.23) Medication Use Nontransition model: Previous Day: 1.08 (1.01, 1.15) **Notes**:Figures of estimated odds ratios for having a more serious asthma attack for short-term, within-subject increases in $PM_{2.5}$, PM_{10}, and CO. Transition models additionally control for the previous day's severity. Figures of estimated relative risks for having inhaler use for short-term, within-subject increases in $PM_{2.5}$, PM_{10}, and CO. Transition models additionally control for the previous day's severity.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Strand et al (2006) **Period of Study:** 2002-2004 **Location:** Denver, Colorado, United States	**Outcome:** Reduced forced expiratory volume (FEV_1) **Age Groups**: 6-12 years old **Study Design:** Mixed model analysis (using the default retricted maximum likelihood (REML) estimators) **N:** 50 children **Statistical Analyses:** least squares regression, SAS "Output Delivery System" (ODS) **Season:** Autumn and Winter **Dose-response Investigated?** Yes **Statistical Package**: SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD):** Outdoor: 12.699 (6.426) Indoor: 8.148 (4.348) Sulfate/$PM_{2.5}$/outdoor: 0.079 (0.067) Sulfate/$PM_{2.5}$/indoor: 0.074 (0.060) **Range (Min, Max):** Mean Personal: (0, 3.035) Outdoor: (0, 6.303) Indoor: (0, 2.759) PM Component: elemental carbon, sulfate, nitrate and ETS. **Monitoring Stations**: 2 fixed monitors and up to 10 personal monitors on a given day. **Copollutant (correlation):** Sulfate (0.63)	**PM Increment:** 10 μg/m^3 Effects Estimate: Using the estimated slope for the validation study model [Lower CI, Upper CI] ; lag: 2.2 percent decrease in FEV_1 per 10 μg/m^3 increase in ambient $PM_{2.5}$ [0.0, 4.3 decrease]; 1 day
Reference: Tang et al. (2007) **Period of Study:** Dec 2003 to Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups**: 6–12 years **Study Design:** Panel study **N:** 30 children **Statistical Analyses:** Linear mixed-effect models were used to estimate the effect of PM exposure on PEFR **Covariates:** Gender, age, BMI, history of respiratory or atopic disease in family, SHS, acute asthmatic exacerbation in past 12 months, ambient temp and relative humidity, presence of indoor pollutants, and presence of outdoor pollutants, **Dose-response Investigated?** yes **Statistical Package**: S-Plus 2000 **Lags Considered:** 0-2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 h **Mean (SD):** Personal: 27.8 (25.3) **Range (Min, Max):** Personal: 1.4–263.4 **Monitoring Stations**: 1	**PM Increment:** 24.5 μg/m^3 RR Estimate [Lower CI, Upper CI] ; lag: Change in morning PEFR: -6.00 (-29.85, 17.85) lag 0 -12.52 (-77.93, 52.9) lag 1 -24.87 (-71.49, 21.74) lag 2 -45.67 (-117.09, 25.74) 2-day mean -5.69 (-105.96, 94.59) 3-day mean Change in evening PEFR: 0.50 (-18.82, 19.82) lag 0 16.66 (-7.59, 40.9) lag 1 11.60 (-11.1, 34.31) lag 2 39.97 (7.1, 72.85) 2-day mean -3.32 (-66.14, 59.5) 3-day mean

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Timonen et al. (2004) **Period of Study:** Oct 1998 to April 1999 **Location:** Amsterdam, Netherlands; Erfurt, Germany; Helsinki, Finland	**Outcome:** Urinary concentration of Clara cell protein CC16 of subjects with coronary heart disease **Age Groups**: 50+ **Study Design:** Longitudinal cohort study (panel) **N:** 37 (Amsterdam); 47 (Erfurt); 47 (Helsinki) **Statistical Analyses:** The response of interest was log transformed, createnine adjusted CC16. Mixed-effect model was used to investigate the association between CC16 and air pollutants. **Covariates:** Subjects, long term time trend, temperature (lags 0-3), relative humidity (lags 0-3), barometric pressure (lags 0-3), and weekday of visit. **Dose-response Investigated?** yes **Statistical Package**: S-Plus and SAS **Lags Considered:** 0-3	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Amsterdam: 20.0 µg/m^3 Erfurt: 23.1 µg/m^3 Helsinki: 12.7 µg/m^3 **Range (Min, Max):** Amsterdam: 3.8–82.2 Erfurt: 4.5–118.1 Helsinki: 3.1–39.8 **Monitoring Stations**: 3 **Copollutant (correlation):** Spearman Correlation: $NC_{0.01-0.1}$: Amsterdam -0.15 Erfurt 0.62 Helsinki 0.14 $NC_{0.1-1.0}$: Amsterdam 0.80 Erfurt 0.84 Helsinki 0.80 NO_2: Amsterdam 0.49 Erfurt 0.82 Helsinki 0.35 CO: Amsterdam 0.58 Erfurt 0.77 Helsinki 0.40	**PM Increment:** 10 µg/m^3 RR Estimate [Lower CI, Upper CI] ; lag: Pooled estimate ; 2.8 (-1.1–6.7) lag 0 2.9 (-0.6–6.5) lag 1 5.0 (-2.4–12.4) lag 2 1.6 (-4.7–7.9) lag 3 9.7 (-6.0–25.4) 5-day mean CC16 was not associated to $PM_{2.5}$ in the pooled analysis but CC16 was significantly associated to $PM_{2.5}$ in Helsinki: 23.3 (6.3–40.3) lag 0 6.4 (-8.2–21.1) lag 1 20.2 (6.9–33.5) lag 2 17.6 (4.3–30.9) lag 3 38.8 (15.8–61.8) 5-day mean

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Trenga et al. (2006) **Period of Study:** 1999-2002 **Location:** Seattle, WA	**Outcome:** Lung function: FEV_1, PEF, MMEF (maximal midexpiratory flow; assessed only for children) **Age Groups**: Adults (56-89-years-old) healthy & with COPD; asthmatic children 6-13-years-old **Study Design:** adult and pediatric panel study over three years with 1 monitoring period ("session") per year **N:** 57 adults (33 healthy, 24 with COPD) = 692 subject-days = 207 study-days; 17 asthmatic children = 319 subject-days = 98 study-days **Statistical Analyses:** mixed effects, longitudinal regression models, with the effects of pollutant decomposed into each subject's a) overall mean; b) difference between their session-specific mean and overall mean; c) difference between their daily values and session-specific mean **Covariates:** gender, age, ventral site temperature and relative humidity, CO, NO_2 **Dose-response Investigated?** No **Statistical Package**: SAS **Lags Considered:** 0-1 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h Percentiles: Children, Personal 25^{th}: 8.1 50th(Median): 11.3 75th: 16.3 Indoor 25th: 5.7 50th(Median): 7.5 75th: 10.2 Local outdoor 25th: 6.4 50th(Median): 9.6 75th: 14.8 Adults, Personal 25th: 5.9 50th(Median): 8.5 75th: 12.4 Indoor 25th: 5.1 50th(Median): 7.6 75th: 10.8 Local outdoor 25th: 6 50th(Median): 8.6 75th: 13.1 **Range (Min, Max):** Children, Personal 1.0, 49.4 Indoor (2.2, 36.3) Local outdoor (2.8, 40.4) Adults, Personal 1.3, 66.6 Indoor(1.6, 65.3) Local outdoor (0.0, 41.5) **Monitoring Stations**: 2; also subject-specific local outdoors (i.e., at each home), indoor, and personal **Copollutant (correlation):** CO NO_2 $PM_{2.5}$ $PM_{10-2.5}$ (coarse)	**PM Increment:** 10 µg/m³ **ADULT Personal $PM_{2.5}$ - FEV_1** Overall: Lag 0 -6.0 [-29.1:17.2]; Lag 1 12.0 [-12.9:36.9] No-COPD: Lag 0 -4.6 [-31.0:21.9]; Lag 1 19.3 [-8.2:46.7] COPD: Lag 0 -10.2 [-55.8:35.4]; Lag 1 -19.0 [-74.1:36.2] PEF: Lag 0 1.5 [-2.2:5.2]; Lag 1 2.1 [-1.9:6.1] No-COPD: Lag 0 3.4 [-0.9:7.6]; Lag 1 1.9 [-2.5:6.3] COPD: Lag 0 -4.3 [-11.5:3.0]; Lag 1 2.6 [-6.3:11.5] **Indoor $PM_{2.5}$ - FEV_1** Overall: Lag 0 -12.8 [-44.5:19.0]; Lag 1 19.4 [-11.3:50.1] No-COPD: Lag 0 -15.8 [-50.0:18.4]; Lag 1 28.4 [-4.6:61.3] COPD: Lag 0 2.6 [-71.7:76.8]; Lag 1 -29.7 [-102.9:43.5] **PEF** Overall: Lag 0 -0.5 [-5.6:4.6]; Lag 1 2.3 [-3.3:7.8] No-COPD: Lag 0 0.1 [-5.4:5.6]; Lag 1 2.5 [-3.5:8.4] COPD: Lag 0 -3.2 [-15.1:8.7]; Lag 1 1.1 [-12.0:14.3] **Outdoor Home $PM_{2.5}$ - FEV_1** Overall: Lag 0 -1.4 [-35.6:32.7]; Lag 1 -2.4 [-37.6:32.7]. No-COPD: Lag 0 1.5 [-36.1:39.2]; Lag 1 10.7 [-26.9:48.4] COPD: Lag 0 -8.9 [-62.2:44.4]; Lag 1 -45.2 [-102.6:12.1] **PEF** Overall: Lag 0 2.3 [-3.3:7.9]; Lag 1 0.4 [-5.6:6.4] No-COPD: Lag 0 4.0 [-2.2:10.1]; Lag 1 2.0 [-4.4:8.4] COPD: Lag 0 -1.8 [-10.6:6.9]; Lag 1 -4.8 [-14.6:4.9] **Central Sites $PM_{2.5}$ - FEV_1** Overall: Lag 0 -35.5 [-70.0:-1.0]; Lag 1 -40.4 [-71.1:-9.6]. No-COPD: Lag 0 -32.6 [-69.5:4.3]; Lag 1 -29.0 [-62.5:4.5] COPD: Lag 0 -43.6 [-95.0:7.8]; Lag 1 -70.8 [-118.4:23.1] **PEF** Overall: Lag 0 1.5 [-4.2:7.1]; Lag 1 -2.3 [-7.4:2.9] No-COPD: Lag 0 2.5 [-3.5:8.6]; Lag 1 -0.5 [-6.1:5.0] COPD: Lag 0 -1.5 [-9.9:6.9]; Lag 1 -7.1 [-15.0:0.9] **PEDIATRIC FEV_1 Personal $PM_{2.5}$** Overall: Lag 0 -13.08 [-38.26:12.10]; Lag 1 -16.12 [-42.61:10.37]. No Anti-inflam. Medication: Lag 0 -41.73 [-94.31:10.84]; Lag 1 -30.99 [-82.17:20.19]. Anti-inflam. Medication: Lag 0 -4.61 [-34.49:25.28]; Lag 1 -10.87 [-45.01:23.27] **Indoor $PM_{2.5}$** Overall: Lag 0 -45.90 [-89.92:1.88]; Lag 1 -64.78 [-111.27:18.28] No Anti-inflam. Medication: Lag 0 -75.92 [-145.16:6.67]; Lag 1 -65.08 [-136.98:6.82]. Anti-inflam. Medication: Lag 0 -28.50 [-94.72:37.71]; Lag 1 -64.60 -147.23:18.04] **Outdoor Home $PM_{2.5}$** Overall: Lag 0 -13.11 [-57.41:31.19]; Lag 1 -9.37 [-54.73:36.00]. No Anti-inflam. Medication: Lag 0 -24.42 [-81.22:32.38]; Lag 1 16.52 [-45.76:78.80]. Anti-inflam. Medication: Lag 0 -3.59 [-75.88:68.70]; Lag 1-26.76 [-89.53:36.01] **Central Sites $PM_{2.5}$.** Overall: Lag 0 -12.32 [-53.21:28.56]; Lag 1 5.75 [-33.27:44.76]. No Anti-inflam. Medication: Lag 0 -33.59 [-89.99:22.82]; Lag 1 31.30 [-29.91:92.51]Anti-inflam. Medication: Lag 0 -2.13 [-71.99:67.73]; Lag 1 -3.53 [-67.32:60.27] **PEF: Personal $PM_{2.5}$** Overall: Lag 0 0.31 [-4.02:4.64]; Lag 1 -2.19 [-6.49:2.12] No Anti-inflam. Medication: Lag 0 0.22 [-8.85:9.29]; Lag 1 -10.48 [-18.68:2.28] Anti-inflam. Medication: Lag 0 0.34 [-4.67:5.35]; Lag 1 0.74 [-4.21:5.69] **Indoor $PM_{2.5}$** Overall: Lag 0 -8.68 [-16.64:-0.72]; Lag 1 -9.22 [-17.51:-0.93] No Anti-inflam. Medication: Lag 0 -13.34 [-25.90:-0.79]; Lag 1 -17.13 [-29.86:4.41]. Anti-inflam. Medication: Lag 0 -5.98 [-15.85:3.89]; Lag 1 -4.19 [-14.59:6.20] **Outdoor Home $PM_{2.5}$** Overall: Lag 0 -6.27 [-14.07:1.53]; Lag 1 -5.64 [-13.73:2.44]. No Anti-inflam. Medication: Lag 0 -7.52 [-17.56:2.51]; Lag 1 -6.92 [-18.03:4.19]. Anti-inflam. Medication: Lag 0 -5.22 [-14.77:4.34]; Lag 1 -4.78 [-14.42:4.86] **Central Sites $PM_{2.5}$** Overall: Lag 0 -5.62 [-12.86:1.62]; Lag 1 -2.45 [-9.34:4.43]. No Anti-inflam. Medication: Lag 0 -6.32 [-16.31:3.68]; Lag 1 -0.83 [-11.60:9.95] Anti-inflam. Medication: Lag 0 -5.29 [-13.42:2.85]; Lag 1 -3.04 [-10.76:4.67] **MMEF - Personal $PM_{2.5}$** Overall: Lag 0 -0.99 [-3.96:1.98]; Lag 1 -1.08 [-4.05:1.88]. No Anti-inflam. Medication: Lag 0 -3.32 [-9.52:2.88]; Lag 1 -2.49 [-8.23:3.25]. Anti-inflam. Medication: Lag 0 -0.31 [-3.77:3.16]; Lag 1 -0.59 [-4.06:2.89] **Indoor $PM_{2.5}$** Overall**:** Lag 0 -3.29 [-8.52:1.94]**;** Lag 1 -11.08 [-16.26:5.90]**.** No Anti-inflam. Medication: Lag 0-12.65 [-20.74:-4.56]' Lag 1 -13.84 [-21.82:5.85]/. Anti-inflam. Medication: Lag 0 2.14 [-4.17:8.45]; Lag 1 -9.33 [-15.89:-2.78] **Outdoor Home $PM_{2.5}$** Overall**:** Lag 0 -4.13 [-9.28:1.01]**;** Lag 1 -0.73 [-6.02:4.56] No Anti-inflam. Medication: Lag 0 -8.23 [-14.77:1.69]; Lag 1 -1.19 [-8.45:6.07] Anti-inflam. Medication: Lag 0 -0.68 [-6.87:5.50]; Lag 1 -0.42 [-6.72:5.87] **Central Sites $PM_{2.5}$.** Overall**:** Lag 0 -2.10 [-6.99:2.79]; Lag 1 -0.12 [-4.67:4.42] No Anti-inflam. Medication: Lag 0 -8.21 [-14.79:1.62]; Lag 1 -0.22 [-7.34:6.90] Anti-inflam. Medication: Lag 0 0.82 [-4.48:6.12]. Lag 1 -0.09 [-5.19:5.01]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ward et al. (2002) **Period of Study:** 1997 (two 8-week periods) **Location:** Birmingham and Sandwell, UK	**Outcome:** Change in PEF (peak expiratory flow), self reported respiratory symptoms (same day cough, illness, short of breath, waking up at night with cough or wheeze, wheeze) **Age Groups**: 9 year olds **Study Design:** Time–series panel study **N:** 162 children from 5 schools **Statistical Analyses:** Linear regression (PEF), Logistic regression (respiratory symptoms) **Covariates:** Trend, temperature, schoolday (yes/no) **Season:** Winter (Jan 13-Mar 10) Summer (May 19- July 14) **Dose-response Investigated?** No **Statistical Package**: Nr **Lags Considered:** Lag 0, lag 1, lag 2, lag 3, 7-day moving avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** Winter: 12.7 µg/m^3 Summer: 12.3 µg/m^3 **Range (Min, Max):** Winter: 4, 37 Summer: 5, 28 PM Component: Total mass **Monitoring Stations**: 5 stations near the 5 schools **Copollutant (correlation):** Winter: PM_{10}(r=0.93) NO_2 (r=0.88) O_3 (r=-0.83) Summer: HNO3 (r=0.81)	**PM Increment:** Winter: 12.3 µg/m^3; Summer: 6.3 µg/m^3 Mean (PEF l/min) [Lower CI, Upper CI] ; lag: **Winter morning:** 0.80 [-1.97, 3.67]; lag0; 0.62 [-2.22, 3.54]; lag 1 -0.86 [-4.32, 2.47]; lag 2; -2.47 [-5.30, 0.36]; lag 3 -4.07 [-10.60, 2.42]; 7-day mean **Winter afternoon:** 0.95 [-2.22, 4.23]; lag0; -0.99 [-4.69, 2.72]; lag 1 -1.60 [-5.18, 2.01]; lag 2; -3.45 [-6.53 to -0.25]; lag 3 1.00 [-11.47, 13.56]; 7-day mean **Summer morning:** -1.49 [-3.65, 0.67]; lag 0; 0.21 [-2.12, 2.55]; lag1 2.50 [0.28, 4.72]; lag2; 3.41 [1.40, 5.44]; lag3 3.90 [-2.53, 10.33]; 7-day mean **Summer afternoon:** -0.49 [-2.43, 1.45]; lag 0; -0.78 [-2.72, 1.16]; lag 1 0.57 [-1.35, 2.49]; lag 2; 0.16 [-1.85, 2.17]; lag 3 -0.08 [-5.43, 5.27]; 7-day mean **Winter morning in atopy/recent wheezing subgroup:** –0.072 [–0.527, 0.383]; lag 0; –0.271 [–0.701, 0.159]; lag 1 0.127 [–0.354, 0.608]; lag 2; 0.055 [–0.391, 0.501]; lag 3 **Winter morning in no atopy or recent wheezing subgroup:** 0.126 [–0.413 , 0.666]; lag 0; 0.193 [–0.340 , 0.728] lag 1 –0.170 [–0.788 , 0.447];lag2 ; –0.314 [–0.846 , 0.216]; lag 3 **Winter morning in subgroup with parental atopy/recent wheezing:** 0.187 [–0.008 , 0.382]; lag 0; –0.006 [–0.207 , 0.195]; lag 1 –0.011 [–0.226 , 0.204]; lag 2; –0.037 [–0.228 , 0.154]; lag 3 **Winter morning in subgroup without parental atopy/recent wheezing:** 0.026 [–0.341 , 0.395]; lag 0; 0.068 [–0.307 , 0.444]; lag 1 –0.099 [–0.535 , 0.335]; lag 2; –0.252 [–0.615 , 0.110]; lag 3 **RR Estimate [Lower CI, Upper CI] ; lag:** **Cough:** **Winter:** 0.98 [0.80, 1.18]; lag 0; 0.95 [0.77, 1.17]; lag 1; 1.02 [0.83, 1.24]; lag 2; 1.01 [0.83, 1.23]; lag 3; 1.31 [0.82, 2.09]; 7-day mean **Summer:** 1.13 [1.04, 1.22]; lag 0; 1.04 [0.94, 1.13]; lag 1; 0.94 [0.87, 1.02]; lag 2; 0.89 [0.82, 0.96]; lag 3; 0.81 [0.62, 1.06]; 7 day mean **Illness:** **Winter:** 1.17 [1.05, 1.32]; lag 0; 1.07 [0.95, 1.23]; lag 1; 1.16 [1.01, 1.35]; lag 2; 1.01 [0.90, 1.16]; lag 3; 1.57 [1.15, 2.13]; 7-day mean **Summer:** 1.02 [0.91, 1.13]; lag 0; 1.00 [0.89, 1.13]; lag 1; 0.96 [0.85, 1.07]; lag 2; 0.97 [0.86, 1.09]; lag 3; 0.68 [0.41, 1.13]; 7-day mean **Shortness of breath:** **Winter:** 1.07 [0.94, 1.24]; lag 0; 0.98 [0.84, 1.13]; lag 1; 0.96 [0.82, 1.13]; lag2; 0.91 [0.79, 1.07]; lag 3; 0.82 [0.58, 1.18]; 7-day mean **Summer:**1.04 [0.90, 1.20]; lag 0; 1.08 [0.93, 1.25]; lag 1 0.97 [0.84, 1.13]; lag 2; 0.93 [0.81, 1.08]; lag 3 1.16 [0.76, 1.77]; 7-day mean **Wake at night with cough/wheeze:** **Winter:** 1.10 [0.96, 1.26]; lag 0; 1.05 [0.90, 1.22]; lag 1 0.98 [0.83, 1.13];lag 2; 0.94 [0.81, 1.09];lag 3 0.93 [0.66, 1.32]; 7-day mean **Summer:** 0.93 [0.78, 1.10]; lag 0; 0.81 [0.67, 0.98]; lag 1 0.91 [0.77, 1.09]; lag 2; 0.97 [0.83, 1.13]; lag 3 1.04 [0.57, 1.90]; 7-day mean **Wheeze:** **Winter:** 0.98 [0.83, 1.16]; lag 0; 0.90 [0.75, 1.05]; lag 1; 1.00 [0.83, 1.20]; lag 2; 1.13 [0.95, 1.35]; lag 3; 1.02 [0.68, 1.57];7-day mean **Summer:** 1.02 [0.88, 1.19] ; lag 0**;** 0.98 [0.84, 1.16] ; lag 1**;** 0.87 [0.74, 1.02] ; lag 2**;** 0.85 [0.72, 0.99]; lag 3**;** 0.96 [0.51, 1.81]; 7-day mean

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ward et al. (2002) **Period of Study:** 1997 (two 8-week periods) **Location:** Birmingham and Sandwell, UK	Outcome:Change in PEF (peak expiratory flow), self reported respiratory symptoms (same day cough, illness, short of breath, waking up at night with cough or wheeze, wheeze) **Age Groups**: 9 year olds **Study Design:** Time–series panel study **N:** 162 children from 5 schools **Statistical Analyses:** Linear regression (PEF), Logistic regression (respiratory symptoms) **Covariates:** Trend, temperature, schoolday (yes/no) **Season:** Winter (Jan 13-Mar 10) Summer (May 19- July 14) **Dose-response Investigated?** No **Statistical Package**: Nr **Lags Considered:** Lag 0, lag 1, lag 2, lag 3, 7-day moving avg	**Pollutant:** Sulfate **Averaging Time:** 24-h **Mean (SD):** Winter: 2.4 µg/m^3 Summer: 3.8 µg/m^3 **Range (Min, Max):** Winter: 0.8, 14.9 Summer: 1.1, 7.8 PM Component: SO_4 **Monitoring Stations**: 2 stations	**PM Increment:** Winter: 4.8 µg/m^3; Summer: 3.1 µg/m^3 Mean (PEF l/min) [Lower CI, Upper CI] ; la **Winter morning:** -1.75 [-4.00, 0.50]; lag0; -0.91 [-3.44, 1.62]; lag 1 -0.62 [-3.16, 1.91]; lag 2; -1.82 [-4.27, 0.64]; lag 3 -3.22 [-8.03, 1.58]; 7-day mean **Winter afternoon:** 0.99 [-1.58, 3.55]; lag0**;** 0.79 [-2.42, 4.00]; lag 1 -1.89 [-4.99, 1.21]; lag 2; -1.73 [-4.69, 1.23]; lag 3 -1.96 [-13.35, 9.42]; 7-day mean **Summer morning:** -0.72 [-3.27, 1.82]; lag 0; -1.69 [-4.28, 0.90]; lag1 1.35 [-1.27, 3.97]; lag2; 3.38 [1.03, 5.72]; lag3 2.98 [-4.17, 10.13]; 7-day mean **Summer afternoon:** -0.32 [-2.81, 2.17]; lag 0; 0.84 [-1.63, 3.30]; lag 1 -0.08 [-2.61, 2.44]; lag 2; -0.25 [-2.69, 2.19]; lag 3 -2.20 [-9.51, 5.12]; 7-day mean **Winter morning in atopy/recent wheezing subgroup:** 0.200 [–0.755, 1.156]; lag 0; –0.219 [–1.318, 0.881]; lag 1 –0.431 [–1.526, 0.664];lag 2; 1.200 [0.095, 2.305]; lag 3 **Winter morning in no atopy or recent wheezing subgroup:** –0.613 [–1.714, 0.488]; lag 0; –0.174 [–1.423, 1.075]; lag 1 0.006 [–1.243, 1.253]; lag 2; –1.080 [–2.308, 0.148]; lag 3 **Winter morning in subgroup with parental atopy/recent wheezing:** 0.457 [0.003 , 0.910]; lag 0**;** 0.078 [–0.503, 0.660]; lag 1 –0.102 [–0.656, 0.452]; lag 2; 0.002 [–0.609, 0.613]; lag 3 **Winter morning in subgroup without parental atopy/recent wheezing:** –0.622 [–1.379, 0.136]; lag 0; –0.272 [–1.147, 0.602]; lag 1 –0.138 [–1.005, 0.728]; lag 2; –0.496 [–1.359, 0.367]; lag 3 **RR Estimate [Lower CI, Upper CI] ; lag:** **Cough:** **Winter:** 1.01 [0.84, 1.20]; lag 0**;** 1.02 [0.85, 1.24]; lag 1 0.99 [0.82, 1.20]; lag 2; 0.86 [0.71, 1.05]; lag 3 0.78 [0.53, 1.14]; 7-day mean **Summer:** 1.08 [0.98, 1.20]; lag 0**;** 1.03 [0.93, 1.15]; lag 1 0.97 [0.88, 1.07]; lag 2' 0.90 [0.82, 0.99]; lag 3 0.73 [0.54, 0.97]; 7 day mean **Illness:** **Winter:** 1.06 [0.96, 1.17]; lag 0**;** 1.15 [1.03, 1.28]; lag 1 1.14 [1.00, 1.28]; lag 2; 1.04 [0.92, 1.18]; lag 3 1.30 [1.00, 1.66]; 7-day mean **Summer:** 0.98 [0.86, 1.11]; lag 0; 0.97 [0.84, 1.12]; lag 1 1.01 [0.88, 1.16]; lag 2' 0.95 [0.84, 1.09]; lag 3 0.72 [0.46, 1.12]; 7-day mean **Shortness of breath:** **Winter:** 0.96 [0.85, 1.07]; lag 0: 0.98 [0.86, 1.12]; lag 1 0.94 [0.82, 1.07]; lag2; 0.93 [0.81, 1.08]; lag 3 0.80 [0.59, 1.07]; 7-day mean **Summer:** 0.95 [0.80, 1.14]; lag 0**;** 1.07 [0.89, 1.28]; lag 1 1.04 [0.87, 1.24]; lag 2; 0.94 [0.80, 1.12]; lag 3 |0.58 [0.33, 1.04]; 7-day mean **Wake at night with cough/wheeze:** **Winter:** 0.97 [0.87, 1.08]; lag 0; 1.01 [0.89, 1.15]; lag 1 1.00 [0.88, 1.14];lag 2; 0.93 [0.82, 1.07];lag 3 0.79 [0.59, 1.05]; 7-day mean **Summer:** 0.95 [0.78, 1.16] ; lag 0; 0.81 [0.67, 0.99] ; lag 1 0.93 [0.76, 1.13] ; lag 2; 0.87 [0.72, 1.05]; lag 3 0.77 [0.41, 1.48]; 7-day mean **Wheeze:** **Winter:** 1.00 [0.87, 1.15]; lag 0; 0.96 [0.82, 1.13]; lag 1 0.88 [0.75, 1.04]; lag 2; 1.12 [0.95, 1.32]; lag 3 0.83 [0.58, 1.20];7-day mean **Summer:** 0.97 [0.80, 1.17] ; lag 0**;** .09 [0.89, 1.32] ; lag 1 1.00 [0.82, 1.22] ; lag 2; 0.81 [0.69, 0.97]; lag 3 1.30 [0.68, 2.50]; 7-day mean

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ward et al. (2002) **Period of Study:** 1997 (two 8-week periods) **Location:** Birmingham and Sandwell, UK	**Outcome:** Change in PEF (peak expiratory flow), self reported respiratory symptoms (same day cough, illness, short of breath, waking up at night with cough or wheeze, wheeze) **Age Groups**: 9 year olds **Study Design:** Time–series panel study **N:** 162 children from 5 schools **Statistical Analyses:** Linear regression (PEF), Logistic regression (respiratory symptoms) **Covariates:** Trend, temperature, schoolday (yes/no) **Season:** Winter (Jan 13-Mar 10) Summer (May 19- July 14) **Dose-response Investigated?** No **Statistical Package**: Nr **Lags Considered:** Lag 0, lag 1, lag 2, lag 3, 7-day moving avg	**Pollutant:** NO_3 **Averaging Time:** 24-h **Mean (SD):** Winter: 3.6 µg/m^3 Summer: 3.5 µg/m^3 **Range (Min, Max):** Winter: 0.1, 29.9 Summer: 0.7, 13.2 **Monitoring Stations**: 2 stations	**PM Increment:** Winter: 6.7 µg/m^3; Summer: 3.7 µg/m^3 Mean (PEF l/min) [Lower CI, Upper CI] ; lag: **Winter morning:** -2.08 [-4.02 to -0.15]; lag0; -0.64 [-2.87, 1.59]; lag 1 0.71 [-1.69, 3.11]; lag 2; -1.38 [-3.61, 0.84]; lag 3 -0.92 [-5.32, 3.47]; 7-day mean **Winter afternoon:** 0.24 [-1.89, 2.38]; lag0; -0.72 [-3.87, 2.43]; lag 1 -1.37 [-5.11, 2.38]; lag 2; -2.54 [-5.74, 0.66]; lag 3 0.21 [-7.67, 8.11]; 7-day mean **Summer morning:** -0.80 [-2.74, 1.15]; lag 0; 0.68 [-1.31, 2.67]; lag1 1.42 [-0.73, 3.58]; lag2; 2.54 [0.48, 4.59]; lag3 1.74 [-2.66, 6.13]; 7-day mean **Summer afternoon:** -0.72 [-2.47, 1.03]; lag 0; -0.59 [-2.36, 1.18]; lag 1 -0.33 [-2.11, 1.45]; lag 2; 0.66 [-1.26, 2.58]; lag 3 0.47 [-3.36, 4.29]; 7-day mean **Winter morning in atopy/recent wheezing subgroup:** –0.036 [–0.627 , 0.555]; lag 0; 0.142 [–0.573 , 0.857]; lag 1; 0.000 [–0.760, 0.759]; lag 2; 0.689 [–0.061, 1.439]; lag 3 **Winter morning in no atopy or recent wheezing subgroup**: –0.434 [–1.116, 0.248]; lag 0; –0.201 [–1.002 , 0.600]; lag 1 0.154 [–0.703 , 1.010]; lag 2; –0.605 [–1.422 , 0.210]; lag 3 **Winter morning in subgroup with parental atopy/recent wheezing**: 0.228 [–0.054, 0.511]; lag 0; 0.476 [0.060, 0.892]; lag 1 0.196 [–0.202, 0.594]; lag 2; 0.083 [–0.321, 0.487]; lag 3 **Winter morning in subgroup without parental atopy/recent wheezing:** –0.482 [–0.952, –0.012]; lag 0; –0.276 [–0.846, 0.294]; lag 1 0.078 [–0.520, 0.675]; lag 2; –0.298 [–0.864, 0.268]; lag 3 **RR Estimate [Lower CI, Upper CI] ; lag:** **Cough: Winter:** 0.92 [0.80, 1.07]; lag 0; 0.91 [0.77, 1.07]; lag 1 0.99 [0.83, 1.17]; lag 2; 0.87 [0.73, 1.03]; lag 3 0.71 [0.52, 0.97]; 7-day mean **Summer:** 1.05 [0.97, 1.13]; lag 0; 1.01 [0.93, 1.10]; lag 1 0.95 [0.88, 1.03]; lag 2; 0.89 [0.83, 0.96]; lag 3 0.81 [0.68, 0.97]; 7 day mean **Illness: Winter:** 1.05 [0.97, 1.14]; lag 0; 1.11 [1.01, 1.22]; lag 1; 1.13 [1.01, 1.26]; lag 2 1.13 [1.04, 1.26]; lag 3; 1.13 [0.92, 1.38]; 7-day mean **Summer:** 0.97 [0.87, 1.09]; lag 0; 0.98 [0.87, 1.10]; lag 1 0.95 [0.85, 1.06]; lag 2; 0.94 [0.85, 1.05]; lag 3 0.74 [0.54, 1.03]; 7-day mean **Shortness of breath: Winter:** 0.99 [0.90, 1.10]; lag 0; 1.01 [0.90, 1.13]; lag 1 0.93 [0.82, 1.05]; lag2; 0.98 [0.86, 1.13]; lag 3 0.85 [0.67, 1.08]; 7-day mean **Summer:** 1.04 [0.90, 1.18]; lag 0; 1.12 [0.98, 1.28]; lag 1 1.04 [0.90, 1.20]; lag 2; 0.90 [0.79, 1.03]; lag 3 1.06 [0.78, 1.43]; 7-day mean **Wake at night with cough/wheeze: Winter:** 0.98 [0.89, 1.08]; lag 0; 1.05 [0.94, 1.16]; lag 1 0.99 [0.88, 1.12];lag 2; 0.99 [0.87, 1.12];lag 3 0.84 [0.67, 1.05]; 7-day mean **Summer:** 0.94 [0.80, 1.09] ; lag 0; 0.86 [0.72, 1.01] ; lag 1 0.94 [0.79, 1.11] ; lag 2; 0.92 [0.79, 1.07]; lag 3 0.95 [0.62, 1.47]; 7-day mean **Wheeze: Winter:** 0.98 [0.87, 1.10] ; lag 0; 1.00 [0.87, 1.14] ; lag 1 0.89 [0.77, 1.03] ; lag 2; 1.11 [0.95, 1.30]; lag 3 0.80 [0.61, 1.07]; 7-day mean **Summer:** 1.01 [0.87, 1.17] ; lag 0; 0.96 [0.83, 1.11] ; lag 1 0.95 [0.82, 1.10] ; lag 2; 0.87 [0.75, 1.01]; lag 3 1.04 [0.67, 1.60]; 7-day mean

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ward et al. (2002) **Period of Study:** 1997 (two 8-week periods) **Location:** Birmingham and Sandwell, UK	**Outcome:**Change in PEF (peak expiratory flow), self reported respiratory symptoms (same day cough, illness, short of breath, waking up at night with cough or wheeze, wheeze) **Age Groups:** 9 year olds **Study Design:** Time–series panel study **N:**162 children from 5 schools **Statistical Analyses:** Linear regression (PEF), Logistic regression (respiratory symptoms) **Covariates:** Trend, temperature, schoolday (yes/no) **Season:** Winter (Jan 13-Mar 10) Summer (May 19- July 14) **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** Lag 0, lag 1, lag 2, lag 3, 7-day moving avg	**Pollutant:** HNO_3 (µg/m³) **Averaging Time:** 24-h **Mean (SD):** Winter: 0.5 µg/m³ Summer: 1.1 µg/m³ **Range (Min, Max):** Winter: 0.2, 2.2 Summer: 0.4, 3.8 **Monitoring Stations**: 2 stations **Copollutant (correlation):** Summer: $PM_{2.5}$ (r=0.81) PM_{10} (r=0.77) NO_2 (r=0.65)	**PM Increment:** Winter: 0.4 µg/m³ Summer: 1.3 µg/m³ **Mean (PEF l/min) [Lower CI, Upper CI] ; lag:** Winter morning: -1.16 [-2.67, 0.36]; lag0; -1.07 [-2.50, 0.37]; lag 1 -0.21 [-1.77, 1.35]; lag 2; -1.03 [-2.51, 0.44]; lag 3 -1.78 [-5.45, 1.89]; 7-day mean **Winter afternoon**: -0.35 [-1.94, 1.24]; lag0; 0.87 [-0.57, 2.31]; lag 1 0.41 [-1.13, 1.96]; lag 2; -0.87 [-2.36, 0.62]; lag 3 -0.27 [-6.87, 6.34]; 7-day mean **Summer morning**: -1.09 [-3.26, 1.07]; lag 0; 0.53 [-1.74, 2.81]; lag1 0.72 [-1.62, 3.06]; lag2; 2.26 [0.08, 4.43]; lag3 -0.59 [-7.31, 6.14]; 7-day mean **Summer afternoon**: -0.08 [-2.14, 1.97]; lag 0; -0.72 [-2.84, 1.40]; lag 1 0.36 [-1.77, 2.49]; lag 2; -1.92 [-4.01, 0.17]; lag 3 -4.67 [-10.29, 0.96]; 7-day mean **Winter morning in atopy/recent wheezing subgroup**: 3.506 [–4.273, 11.285]; lag 0 –0.445 [–8.083, 7.192]; lag 1 –7.616 [–14.989, –0.242]; lag 2 3.240 [–4.568, 11.048]; lag3 **Winter morning in no atopy or recent wheezing subgroup:** –5.964 [–15.195, 3.266]; lag 0 –3.866 [–12.741, 5.010]; lag 1 2.588 [–6.644, 11.819]; lag 2 –5.384 [–14.498, 3.730]; lag3 **Winter morning in subgroup with parental atopy/recent wheezing:** 1.005 [–2.115, 4.124]; lag 0 –2.266 [–5.135, 0.603]; lag 1 –1.835 [–4.775, 1.105]; lag 2 –0.442 [–3.366, 2.481]; lag3 **Winter morning in subgroup without parental atopy/recent wheezing:** –4.324 [–10.556, 1.907]; lag 0 –2.982 [–8.869, 2.904]; lag 1 –0.157 [–6.499, 6.183]; lag 2 –3.445 [–9.496, 2.607]; lag 3 **RR Estimate [Lower CI, Upper CI]; lag: Cough: Winter:** 1.04 [0.93, 1.16]; lag 0; 1.05 [0.95, 1.16]; lag 1 1.05 [0.94, 1.16]; lag 2; 0.90 [0.81, 1.00]; lag 3 1.14 [0.84, 1.54]; 7-day mean **Summer:** 1.09 [1.00, 1.19]; lag 0; 1.01 [0.92, 1.11]; lag 1 0.94 [0.86, 1.03]; lag 2; 0.89 [0.82, 0.97]; lag 3 0.66 [0.49, 0.88]; 7 day mean **Illness: Winter:** 0.97 [0.91, 1.04]; lag 0; 0.96 [0.90, 1.03]; lag 1 1.01 [0.94, 1.07]; lag 2; 1.02 [0.95, 1.09]; lag 3 1.09 [0.90, 1.32]; 7-day mean **Summer**: 0.92 [0.83, 1.04]; lag 0 0.98 [0.86, 1.12]; lag 1; 0.95 [0.83, 1.08]; lag 2 1.04 [0.92, 1.18]; lag 3; 0.79 [0.46, 1.34]; 7-day mean **Shortness of breath: Winter**: 0.97 [0.90, 1.05]; lag 0; 0.90 [0.83, 0.97]; lag 1 0.91 [0.84, 0.98]; lag2; 1.00 [0.92, 1.08]; lag 3 0.79 [0.63, 0.99]; 7-day mean **Summer**:1.04 [0.90, 1.21]; lag 0 1.01 [0.86, 1.18]; lag 1; 0.99 [0.85, 1.15]; lag 2 0.95 [0.82, 1.10]; lag 3; 1.02 [0.65, 1.61]; 7-day mean **Wake at night with cough/wheeze: Winter**: 0.96 [0.89, 1.04]; lag 0; 0.90 [0.83, 0.97]; lag 1 0.90 [0.84, 0.98];lag 2; 1.02 [0.94, 1.10];lag 3 0.78 [0.63, 0.96]; 7-day mean **Summer**: 0.83 [0.69, 0.99] ; lag 0 0.76 [0.62, 0.92] ; lag 1; 0.94 [0.78, 1.12] ; lag 2 0.89 [0.75, 1.05]; lag 3; 0.93 [0.50, 1.73]; 7-day mean **Wheeze: Winter**: 1.00 [0.91, 1.10] ; lag 0 0.98 [0.89, 1.07] ; lag 1; 0.89 [0.82, 0.98] ; lag 2 0.97 [0.88, 1.07]; lag 3; 0.76 [0.58, 0.99]; 7-day mean **Summer**: 0.93 [0.80, 1.09] ; lag 0 0.87 [0.74, 1.02] ; lag 1; 0.87 [0.73, 1.04] ; lag 2 0.70 [0.60, 0.82]; lag 3; 0.71 [0.43, 1.20]; 7-day mean

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ward et al. (2002) **Period of Study:** 1997 (two 8-week periods) **Location:** Birmingham and Sandwell, UK	**Outcome:** Change in PEF (peak expiratory flow), self reported respiratory symptoms (same day cough, illness, short of breath, waking up at night with cough or wheeze, wheeze) **Age Groups**: 9 year olds **Study Design:** Time–series panel study **N:** 162 children from 5 schools **Statistical Analyses:** Linear regression (PEF), Logistic regression (respiratory symptoms) **Covariates:** Trend, temperature, schoolday (yes/no) **Season:** Winter (Jan 13-Mar 10) Summer (May 19- July 14) **Dose-response Investigated?** No **Statistical Package**: Nr **Lags Considered:** Lag 0, lag 1, lag 2, lag 3, 7-day moving avg	**Pollutant:** Cl-, HCl, NH3, NH4 **Averaging Time:** 24-h **Mean (SD):** Cl- Winter: 3.0 Summer: 0.8 HCl Winter: 0.3 Summer: 0.3 NH_3 Winter: 5.6 Summer: 4.2 NH_4 Winter: 2.0 Summer: 2.5 **Range (Min, Max):** Cl- Winter: 0.9, 7.3 Summer: 0.3, 5.1 HCl Winter: 0.0, 1.7 Summer: 0.0, 1.0 NH3 Winter: 0.9, 23.8 Summer: 0.6, 8.8 NH4 Winter: 0.2, 15.5 Summer: 0.5, 7.1 **Monitoring Stations**: 2 stations	Authors do not present quantitative results for these particle species: "Results for incident symptoms and the acid and anion species HCl, Cl-, NH4, and NH3 are not shown for brevity. No pattern in the nature of the pollutants or the lag of greatest measured effect were noted and, in particular, there were no consistent responses to ozone or particles as PM_{10} of $PM_{2.5}$."

Table E-12. Short-term exposure to other PM size fractions and respiratory morbidity outcomes

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Andersen et al. (2008a) **Period of Study:** Dec 12, 1998–Dec 19, 2004 **Location:** Copenhagen, Denmark	**Outcome:** Daily symptoms Age Groups: 0-3 yrs **Study Design:** Panel study of children with genetic susceptibility to asthma (mothers had asthma) **N:** 205 children (living within a 15km radius of the central monitor during the first 3 yrs of life); born between Aug 2, 1998 and Dec 12, 2001 **Statistical Analyses:** logistic regression model (GEE) **Covariates:** temperature, season, gender, age, exposure to smoking, and paternal history of asthma **Effect modification:** gender, medication use, and paternal history of asthma **Statistical Package:** SAS v9.1 **Lag:** Lag0, Lag1, Lag2, Lag3, Lag4, Lag2-4 (3-day mean)	**Pollutant:** UFP Mean: 8092 SD: 3470 Percentile 25th: 5706 75th: 9825 IQR: 4119 **Units:** particles/cm^3 **Copollutant (correlation):** Number concentration of ultrafine particles, UFP PM_{10} (r=0.37) $PM_{2.5}$ (r=0.40) NO_2 (r=0.67) NO_X (r=0.65) CO (r=0.52) O_3 (r=-0.12) Temp (r=-0.06)	**PM Increment:** IQR (4119 particles/cm3) increase Odds Ratios (95%CI) Associations between incident wheezing symptoms Age 0-1 L0: 0.71 (0.44, 1.16) L1: 0.88 (0.56, 1.38) L2: 1.60 (0.92, 2.67) L3: 1.07 (0.67, 1.73) L4: 1.50 (0.89, 2.54) L2-4: 1.92 (0.98, 3.76) Age 1-2 L0: 0.82 (0.62, 1.09) L1: 0.92 (0.70, 1.21) L2: 0.88 (0.67, 1.16) L3: 0.79 (0.59, 1.06) L4: 0.99 (0.76, 1.29) L2-4: 0.83 (0.58, 1.17) Age 2-3 L0: 1.00 (0.67, 1.49) L1: 0.93 (0.68, 1.26) L2: 1.03 (0.73, 1.44) L3: 0.89 (0.63, 1.27) L4: 0.62 (0.44, 0.89) L2-4: 0.72 (0.49, 1.04) Age 0-3 L0: 0.85 (0.68, 1.05) L1: 0.91 (0.75, 1.10) L2: 1.00 (0.81, 1.24) L3: 0.84 (0.70, 1.02) L4: 0.88 (0.73, 1.05) L2-4: 0.85 (0.68, 1.07) Two pollutant models 1-pollutant model: 1.92 (0.98, 3.76) 2-pollutant (adj for PM_{10}): 1.86 (0.88, 4.14) 2-pollutant (adj for NO_2): 1.82 (0.62, 5.34) 2-pollutant (adj for NOX): 2.04 (0.68, 6.16) 2-pollutant (adj for CO): 1.67 (0.69, 4.02) 110 children living within 5km radius from monitor (sensitivity analysis) Age 0-1: 2.46 (1.04, 5.84) Age 1-2: 1.09 (0.61, 1.94) Age 2-3: 0.40 (0.21, 0.76) Age 0-3: 0.92 (0.63, 1.34)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chattopadhyay et al. (2007) **Period of Study:** NR **Location:** Three different points in Kolkata, India: North, South, and Central	**Outcome:** pulmonary function tests (respiratory impairments) **Age Groups**: All ages **Study Design:** Cross-sectional **N:** 505 people studied for PFT; total population of Kolkata not given **Statistical Analyses:** Frequencies **Covariates:** Meteorologic data (i.e. temperature, wind direction, wind speed, and humidity) **Dose-response Investigated?** No	**Pollutant:** $PM_{<3.3-0.4}$ **Averaging Time:** 8 h **Mean (SD):** North Kolkata: 266.1 Central Kolkata: 435.3 South Kolkata: 449.1 Unit (i.e. µg/m³): µg/m³ **Monitoring Stations**: 1 **Copollutant (correlation):** PM_{10} $PM_{<10-3.3}$	**PM Increment:** NR Respiratory impairments (SD): North Kolkata Male (n=137) Restrictive: 4 (2.92) Obstructive: 5 (3.64) Combined Res. And Obs.: 6 (4.37) Total: 15 (10.95) Female (n=152) Restrictive: 3 (1.97) Obstructive: 5 (3.28) Combined Res. And Obs.: 0 Total: 8 (5.26) Total (n=289) Restrictive: 7 (2.42) Obstructive: 10 (3.46) Combined Res. And Obs.: 6 (2.07) Total: 23 (7.96) Central Kolkata Male (n=44) Restrictive: 6 (13.63) Obstructive: 1 (2.27) Combined Res. And Obs.: 1 (2.27) Total: 8 (18.18) Female (n=50) Restrictive: 3 (6.00) Obstructive: 2 (4.00) Combined Res. And Obs.: 0 Total: 5 (10.00) Total (n=94) Restrictive: 9 (9.57) Obstructive: 3 (3.19) Combined Res. And Obs.: 1 (1.06) Total: 13 (13.82) South Kolkata Male (n=52) Restrictive: 1 (1.92) Obstructive: 2 (3.84) Combined Res. And Obs.: 3 (5.76) Total: 6 (11.53) Female (n=70) Restrictive: 2 (2.85) Obstructive: 1 (1.42) Combined Res. And Obs.: 0 Total: 3 (4.28) Total (n=122) Restrictive: 3 (2.45) Obstructive: 3 (2.45) Combined Res. And Obs.: 3 (2.45) Total: 9 (7.37)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chattopadhyay et al. (2007) **Period of Study:** NR **Location:** Three different points in Kolkata, India: North, South, and Central	**Outcome:** pulmonary function tests (respiratory impairments) **Age Groups**: All ages **Study Design:** Cross-sectional **N:** 505 people studied for PFT; total population of Kolkata not given **Statistical Analyses:** Frequencies **Covariates:** Meteorologic data (i.e. temperature, wind direction, wind speed, and humidity) **Dose-response Investigated?** No	**Pollutant:** $PM_{<10-3.3}$ **Averaging Time:** 8 h **Mean (SD):** North Kolkata: 269.8 Central Kolkata: 679.2 South Kolkata: 460.1 Unit (i.e. µg/m³): µg/m³ **Monitoring Stations**: 1 **Copollutant (correlation):** PM_{10} $PM_{<3.3-0.}$	**PM Increment:** NR Respiratory impairments (SD): North Kolkata Male (n=137) Restrictive: 4 (2.92) Obstructive: 5 (3.64) Combined Res. And Obs.: 6 (4.37) Total: 15 (10.95) Female (n=152) Restrictive: 3 (1.97) Obstructive: 5 (3.28) Combined Res. And Obs.: 0 Total: 8 (5.26) Total (n=289) Restrictive: 7 (2.42) Obstructive: 10 (3.46) Combined Res. And Obs.: 6 (2.07) Total: 23 (7.96) Central Kolkata Male (n=44) Restrictive: 6 (13.63) Obstructive: 1 (2.27) Combined Res. And Obs.: 1 (2.27) Total: 8 (18.18) Female (n=50) Restrictive: 3 (6.00) Obstructive: 2 (4.00) Combined Res. And Obs.: 0 Total: 5 (10.00) Total (n=94) Restrictive: 9 (9.57) Obstructive: 3 (3.19) Combined Res. And Obs.: 1 (1.06) Total: 13 (13.82) South Kolkata Male (n=52) Restrictive: 1 (1.92) Obstructive: 2 (3.84) Combined Res. And Obs.: 3 (5.76) Total: 6 (11.53) Female (n=70) Restrictive: 2 (2.85) Obstructive: 1 (1.42) Combined Res. And Obs.: 0 Total: 3 (4.28) Total (n=122) Restrictive: 3 (2.45) Obstructive: 3 (2.45) Combined Res. And Obs.: 3 (2.45) Total: 9 (7.37)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: de Hartog et al. (2003) **Period of Study:** winter of 1998-1999 (in Amsterdam, from November 2, 1998 to June 18, 1999; in Erfurt, from October 12, 1998 to April 4, 1999; and in Helsinki, from November 2, 1998 to April 30, 1999.) **Location:** Amsterdam, the Netherlands; Erfurt, Germany; and Helsinki, Finland	**Outcome:** chest pain, chest pain at physical exertion, shortness of breath, feeling tired or weak, tripping or racing heart, cold hands or feet, cough, phlegm, being awakened by breathing problems, wheezing, and common cold or flu and fever **Age Groups**: ≥ 50 yrs **Study Design:** cohort **N:** 131 subjects with history of coronary heart disease **Statistical Analyses:** Logistic regression **Covariates:** ambient temperature, relative humidity, atmospheric pressure, incidence of influenza-like illness **Season:** Winter **Dose-response Investigated?** No **Statistical Package**: S-PLUS 2000 **Lags Considered:** 0, 1, 2, 3, and 5-day avg	**Pollutant:** Number concentration ($NC_{0.01-0.1}$) [ultrafine particles] **Averaging Time:** 24 h **Mean (SD):** Amsterdam, the Netherlands: 17,309 Erfurt, Germany: 21,228 Helsinki, Finland: 17,078 **Range (Min, Max):** Amsterdam, the Netherlands: (5,699-37,195) Erfurt, Germany: (3,867-96,678) Helsinki, Finland: (2,305-50,306) **Monitoring Stations**: 1 **Copollutant (correlation):** $NC_{0.01-0.1-CPC}$ r= 0.91 for all centers PM_{10} $PM_{2.5}$ CO NO_2 SO_2	**PM Increment:** 10,000 particles/cm3 Effect Estimate [Lower CI, Upper CI]: association of air pollution and incidence of symptoms in three panels of elderly subjects Lag 0 Chest pain w/ physical exertion: 0.98 (0.87-1.11) Shortness of breath: 0.97 (0.88-1.07) Being awakened by breathing problems: NA Avoidance of activities: 1.12 (0.98-1.28) Phlegm: 0.98 (0.84-1.14) Lag 1 Chest pain w/ physical exertion: 0.94 (0.84-1.05) Shortness of breath: 0.87 (0.79-0.97) Awakened, breathing problems: 0.92 (0.80-1.06) Avoidance of activities: 1.01 (0.88-1.16) Phlegm: 0.92 (0.79-1.08) Lag 2 Chest pain w/ physical exertion: 0.92 (0.82-1.03) Shortness of breath: 0.99 (0.89-1.09) Awakened, breathing problems: 1.01 (0.88-1.16) Avoidance of activities: 1.11 (0.96-1.27) Phlegm: 1.06 (0.92-1.23) Lag 3 Chest pain w/ physical exertion: 0.99 (0.89-1.11) Shortness of breath: 1.09 (0.99-1.21) Awakened, breathing problems: 1.14 (1.01-1.30) Avoidance of activities: 1.06 (0.92-1.21) Phlegm: 1.07 (0.93-1.24) 5-day Chest pain w/ physical exertion: 0.93 (0.77-1.12) Shortness of breath: 0.93 (0.77-1.13) Awakened, breathing problems: 1.18 (0.92-1.52) Avoidance of activities: 1.17 (0.91-1.49) Phlegm: 1.08 (0.82-1.41)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Mar et al. (2004) **Period of Study:** 1997-1999 **Location:** Spokane, Washington	**Outcome:** Respiratory **Age Groups**: Adults: Ages 20-51 yrs; Children: Ages 7-12 yrs **Study Design:** Time-Series **N:** 25 people **Statistical Analyses:** Logistic regression **Covariates:** Temperature, relative humidity, day of-the-wk **Statistical Package**: STATA 6 **Lags Considered:** 0-2 days	**Pollutant:** PM_{10} **Mean (SD):** 1997: 9.8 (5.3) 1998: 9.2 (4.7) 1999: 6.9 (3.7) **Monitoring Stations**: 1 station **Copollutant (correlation):** PM_1: $PM_{2.5}$; r = 0.92 PM_{10}; r = 0.48 $PM_{10-2.5}$; r = 0.16	**PM Increment:** 10 µg/m^3 **Adult Respiratory symptoms:** **Wheeze:** 1.01[0.79, 1.28]; lag 0; 0.96[0.77, 1.19]; lag 1 0.97[0.80, 1.17]; lag 2 **Breath:** 0.95[0.84, 1.08]; lag 0 ; 0.99[0.87, 1.13]; lag 1 0.92[0.76, 1.11]; lag 2 **Cough:** 0.86[0.62, 1.21]; lag 0 ; 0.87[0.63, 1.20]; lag 1 0.92[0.67, 1.25]; lag 2 **Sputum:** 0.94[0.67, 1.46]; lag 0 ; 0.90[0.67, 1.32]; lag 1 0.92[0.72, 1.34]; lag 2 **Runny Nose:** 1.00[0.86, 1.16]; lag 0 ; 0.96[0.83, 1.11]; lag 1 0.94[0.82, 1.09]; lag 2 **Eye Irritation:** 0.89[0.61, 1.28]; lag 0 ; 0.86[0.68, 1.09]; lag 1 0.81[0.60, 1.09]; lag 2 **Lower Symptoms:** 0.92[0.75, 1.14]; lag 0 ; 0.90[0.73, 1.11]; lag 1 0.92[0.75, 1.11]; lag 2 **Any Symptoms:** 0.94[0.80, 1.10]; lag 0 ; 0.90[0.77, 1.05]; lag 1 0.91[0.77, 1.07]; lag 2 **Children Respiratory symptoms:** **Wheeze:** 0.51[0.14, 1.83]; lag 0 ; 0.42[0.10, 1.75]; lag 1 0.40[0.12, 1.32]; lag 2 **Breath:** 1.08[0.80, 1.45]; lag 0 ; 1.08[0.81, 1.44]; lag 1 1.05[0.77, 1.43]; lag 2 **Cough:** 1.20[1.00, 1.44]; lag 0 ; 1.24[0.99, 1.56]; lag 1 1.21[1.02, 1.43]; lag 2 **Sputum:** 1.10[0.94, 1.29]; lag 0 ; 1.14[0.92, 1.40]; lag 1 1.11[0.93, 1.33]; lag 2 **Runny Nose:** 1.10[0.84, 1.42]; lag 0 ; 1.14[0.91, 1.42]; lag 1 1.14[0.91, 1.42]; lag 2 **Eye Irritation:** 0.91[0.56, 1.47]; lag 0 ; 0.85[0.52, 1.40]; lag 1 0.72[0.62, 0.84]; lag 2 **Lower Symptoms:** 1.21[1.01, 1.44]; lag 0 ; 1.25[1.01, 1.55]; lag 1 1.19[0.98, 1.44]; lag 2 **Any Symptoms:** 1.18[1.04, 1.33]; lag 0 ; 1.24[1.08, 1.43]; lag 1 1.24[1.08, 1.43]; lag 2
Reference: McCreanor et al. (2007) **Period of Study:** 2003-2005 **Location:** London, England	**Outcome:** Decreased Lung Function **Age Groups:** Adults **Study Design:** Crossover study **N:** 60 adults **Statistical Analyses:** Linear regression **Covariates:** Temperature, relative humidity, age, sex, bod-mass index, and race or ethnic group	**Pollutant:** UFP **50th (Median):** Oxford St: 125 Hyde St: 72 **Range (Min, Max):** Oxford St: (62, 161) Hyde Park: (60, 100)	% changes in FEV and FVC are presented in figures 1-3. Results are not presented quantitatively in text or tables. The authors did not find any significant differences in respiratory symptoms between the two locations. Also, there were no significant differences in sputum eosinophili counts or eosinophil cationic protein levels.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Moshammer and Neuberger (2003) **Period of Study:** 2000-2001 **Location:** Linz, Austria	**Outcome:** Lung Function: FVC, FEV_1, MEF_{25}, MEF_{50}, MEF_{75}, PEF, LQ Signal, PAS Signal **Age Groups**: Ages 7 to 10 **Study Design:** Case-crossover **N:** 161 children; 1898–2120 "half-h means" **Statistical Analyses:** Correlations Regression Analysis **Covariates:** Morning, Evening, Night **Season:** Spring, Summer, Winter, Fall **Dose-response Investigated?** No	**Pollutant:** PM_1 **Averaging Time:** 8 h means & Daily Means **Mean (SD):**10.79 (9.31) **Range (Min, Max):** (NR, 98.90) **Monitoring Stations**: 1 **Copollutant (correlation):** LQ = 0.660 PAS = 0.276	Notes: "Acute effects of 'active particle surface' as measured by diffusion charging were found on pulmonary function (FVC, FEV_1, MEF_{50}) of elementary school children and on asthma-like symptoms of children who had been classified as sensitive."
Reference: Moshammer et al. (2006) **Period of Study:** 2000-2001 **Location:** Linz, Austria	**Outcome:** Respiratory symptoms and decreased lung function **Age Groups:** Children ages 7-10 **Study Design:** Time-series **N:** 163 children **Statistical Analyses:** GEE model **Covariates:** Sex, age, height, weight **Dose-response Investigated?** NR **Statistical Package:** NR **Lags Considered:** 1	**Pollutant:** $PM_{1.0}$ **Averaging Time:** 8-h **Mean (SD):** Maximum 24 h: 58.20 Annual avg: 15.03 Percentiles: 8-h mean 25th: 6.90 8-h mean 50th(Median): 12.30 8-h mean 75th: 17.82 **Monitoring Stations**: 1 station **Copollutant (correlation):** $PM_{2.5}$; r = 0.95 PM_{10}; r = 0.91 NO_2; r = 0.53	**PM Increment:** 10 µg/m³ **% change in Lung Function per 10** µg/m³ FEV: 0.38 FVC: 0.14 $FEV_{0.5}$: -0.50 MEF75%: -0.85 MEF50%: -0.82 MEF25%: -1.17 PEF: -0.63 **% change in Lung Function per IQR** FEV: 0.41 FVC: 0.15 $FEV_{0.5}$: 0.54 MEF75%: -0.93 MEF50%: -0.89 MEF25%: -1.27 PEF: -0.68
Reference: Moshammer and Neuberger (2003) **Period of Study:** 2000-2001 **Location:** Linz, Austria	**Outcome:** Lung Function: FVC, FEV_1, MEF25, MEF50, MEF75, PEF, LQ Signal, PAS Signal **Age Groups**: Ages 7 to 10 **Study Design:** Case-crossover **N:** 161 children; 1898–2120 "half-h means" **Statistical Analyses:** Correlations Regression Analysis **Covariates:** Morning, Evening, Night **Season:** Spring, Summer, Winter, Fall **Dose-response Investigated?** No	**Pollutant:** CPC (condensed particle count) **Averaging Time:** 8 h means & Daily Means **Mean (SD):** 25024 (16937) **Range (Min, Max):** (20, 140972) **Monitoring Stations**: 1 **Copollutant (correlation):** LQ = 0.673 PAS = 0.472	Notes: "Acute effects of 'active particle surface' as measured by diffusion charging were found on pulmonary function (FVC, FEV_1, MEF_{50}) of elementary school children and on asthma-like symptoms of children who had been classified as sensitive."
Reference: Neuberger et al. (2004) **Period of Study:** 6/1999-6/2000 **Location:** Austria (Vienna and a rural area near Linz)	**Outcome**: Questionnaire derived asthma score, and a 1-5 point respiratory health rating by parent **Age Groups**: 7-10 years **Study Design:** Cross-sectional survey **N:** about 2000 children **Statistical Analyses:** mixed models linear regression -used factor analysis to develop the "asthma score" **Covariates:** Pre-existing respiratory conditions, temperature, rainy days, # smokers in household, heavy traffic on residential street, gas stove or heating, molds, sex, age of child, allergies of child, asthma in other family members **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 4 week avg (preceding interview)	**Pollutant:** PM_1 **Averaging Time:** 24 h	**PM Increment:** 10 µg/m³ Change in mean associated unit increase in PM (p-value); lag Respiratory Health score Vienna: 0.008 (p>0.05); lag 4 week avg Rural area: 0.027 (p<0.05); lag 4 week avg Asthma score Vienna: 0.008 (p>0.05); lag 4 week avg Rural area: -0.002 (p>0.05); lag 4 week avg

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Neuberger et al. (2004) **Period of Study:** Sept 1999-March 2000 **Location:** Vienna, Austria	**Outcome:** Ratio measure: Time to peak tidal expiratory flow divided by total expiration time (i.e., tidal lung function, a surrogate for bronchial obstruction) **Age Groups**: 3.0-5.9 years (preschool children) **Study Design:** Longitudinal prospective cohort **N:** 56 children **Statistical Analyses:** mixed models linear regression, with autoregressive correlation structure **Covariates:** Age, sex, respiratory rate, phase angle, temperature, kindergarten, parental education, observer (also in sensitivity analyses: height, weight, cold/sneeze on same day, heating with fossil fuels, hair cotinine, number of tidal slopes used to measure tidal lung function) **Dose-response Investigated?** No **Statistical Package**: SAS 8.0 **Lags Considered:** Lag 0	**Pollutant:** PM_1 **Averaging Time:** 24 h	**PM Increment:** Interquartile range (NR) Change in mean associated with an IQR increase in PM (p-value); lag -1.059 (0.060); lag 0
Reference: Neuberger et al. (2004) **Period of Study:** Oct. 2000-May 2001 **Location:** Linz, Austria	Outcome:Forced oscillatory resistance (at zero Hz), FVC, FEV_1, MEF25, MEF50, MEF75, PEF **Age Groups**: 7-10 years **Study Design:** Longitudinal prospective cohort **N:** 164 children **Statistical Analyses:** mixed models linear regression with autoregressive correlation structure **Covariates:** sex, time and individual Season: Oct-May **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** Lag 0-7	**Pollutant:** PM_1 **Averaging Time:** 24 h **Monitoring Stations**: 1	**PM Increment:** 1 μg/m^3 Change in mean volume flow (1/s) (standard error); lag FVC -0.00139 (0.000283); lag 7 FEV_1 -0.00139 (0.000249); lag 7 PEF -0.00321 (0.001007); lag 7 MEF75 -0.00407 (0.000946); lag 7 MEF25 -0.00102 (0.000471); lag 7 Notes: Results for change in oscillatory resistance presented in figure: authors report significant associations with PM1 (lag 0) and PM1 (lag 3). Though quantitative results were not presented.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Sakai et al. (2004) **Period of Study:** November 14, 1999-March 28, 2001 **Location:** Diesel-powered ship from Tokyo, Japan to Showa Station on Ongul Island, Antarctica for 366 days (from February 1, 2000) and then heading back to Japan on February 1, 2001	**Outcome:** circulating leukocyte counts and serum inflammatory cytokine levels **Age Groups**: 24-57 yrs, mean=36.1 ± 4.7 yrs **Study Design:** cohort **N:** 39 members of 41st Japanese Antarctic Research Expedition (JARE-41) **Statistical Analyses:** ANOVA **Covariates:** Smoking history, occupational pollutant exposure **Dose-response Investigated?** No **Statistical Package**: SPSS 11.5J	**Pollutant:** $PM_{2.0-0.3}$ **Averaging Time:** 24-h Unit (i.e. μg/m^3): particles/L PM Component: organic and inorganic substances, including microorganisms **Copollutant (correlation):** $PM_{5.0-2.0}$ $PM_{10-5.0}$	Effect Estimate: Multiple regression analysis between inhaled factors in Antarctica Total leukocyte Cigarette smoking= 0.211, p<0.001 Support staff= 0.139, p=0.024 Total PM= 0.168, p=0.004 Segmented PMN Cigarette smoking= 0.015, p=0.805 Support staff= 0.097, p=0.119 Total PM= 0.272, p<0.001 Band-formed PMN Cigarette smoking= 0.035, p=0.543 Support staff= 0.010, p=0.864 Total PM= 0.470, p<0.001 Monocyte Cigarette smoking= 0.081, p=0.187 Support staff= -0.019, p=0.759 Total PM= 0.328, p<0.001 G-CSF Cigarette smoking= 0.131, p<0.038 Support staff= 0.176, p=0.005 Total PM= 0.078, p=0.186 IL-6 Cigarette smoking= 0.182, p=0.004 Support staff= 0.076, p=0.228 Total PM= 0.158, p=0.008
Reference: Sakai et al. (2004) **Period of Study:** November 14, 1999-March 28, 2001 **Location:** Diesel-powered ship from Tokyo, Japan to Showa Station on Ongul Island, Antarctica for 366 days (from February 1, 2000) and then heading back to Japan on February 1, 2001	**Outcome:** circulating leukocyte counts and serum inflammatory cytokine levels **Age Groups**: 24-57 yrs, mean=36.1 ± 4.7 yrs **Study Design:** cohort **N:** 39 members of 41st Japanese Antarctic Research Expedition (JARE-41) **Statistical Analyses:** ANOVA **Covariates:** Smoking history, occupational pollutant exposure **Dose-response Investigated?** No **Statistical Package**: SPSS 11.5J	**Pollutant:** $PM_{5.0-2.0}$ **Averaging Time:** 24 h Unit (i.e. μg/m^3): particles/L PM Component: organic and inorganic substances, including microorganisms **Copollutant (correlation):** $PM_{2.0-0.3}$ $PM_{10-5.0}$	Effect Estimate: Multiple regression analysis between inhaled factors in Antarctica Total leukocyte Cigarette smoking= 0.211, p<0.001 Support staff= 0.139, p=0.024 Total PM= 0.168, p=0.004 Segmented PMN Cigarette smoking= 0.015, p=0.805 Support staff= 0.097, p=0.119 Total PM= 0.272, p<0.001 Band-formed PMN Cigarette smoking= 0.035, p=0.543 Support staff= 0.010, p=0.864 Total PM= 0.470, p<0.001 Monocyte Cigarette smoking= 0.081, p=0.187 Support staff= -0.019, p=0.759 Total PM= 0.328, p<0.001 G-CSF Cigarette smoking= 0.131, p<0.038 Support staff= 0.176, p=0.005 Total PM= 0.078, p=0.186 IL-6 Cigarette smoking= 0.182, p=0.004 Support staff= 0.076, p=0.228 Total PM= 0.158, p=0.008

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Sakai et al. (2004) **Period of Study:** November 14, 1999-March 28, 2001 **Location:** Diesel-powered ship from Tokyo, Japan to Showa Station on Ongul Island, Antarctica for 366 days (from February 1, 2000) and then heading back to Japan on February 1, 2001	**Outcome:** circulating leukocyte counts and serum inflammatory cytokine levels **Age Groups**: 24-57 yrs, mean=36.1 ± 4.7 yrs **Study Design:** cohort **N:** 39 members of 41st Japanese Antarctic Research Expedition (JARE-41) **Statistical Analyses:** ANOVA **Covariates:** Smoking history, occupational pollutant exposure **Dose-response Investigated?** No **Statistical Package**: SPSS 11.5J	**Pollutant:** $PM_{10-5.0}$ **Averaging Time:** 24-h Unit (i.e. µg/m^3): particles/L **Monitoring Stations**: NR **Copollutant (correlation):** $PM_{2.0-0.3}$ $PM_{10-5.0}$	Effect Estimate: Multiple regression analysis between inhaled factors in Antarctica Total leukocyte Cigarette smoking= 0.211, p<0.001 Support staff= 0.139, p=0.024 Total PM= 0.168, p=0.004 Segmented PMN Cigarette smoking= 0.015, p=0.805 Support staff= 0.097, p=0.119 Total PM= 0.272, p<0.001 Band-formed PMN Cigarette smoking= 0.035, p=0.543 Support staff= 0.010, p=0.864 Total PM= 0.470, p<0.001 Monocyte Cigarette smoking= 0.081, p=0.187 Support staff= -0.019, p=0.759 Total PM= 0.328, p<0.001 G-CSF Cigarette smoking= 0.131, p<0.038 Support staff= 0.176, p=0.005 Total PM= 0.078, p=0.186 IL-6 Cigarette smoking= 0.182, p=0.004 Support staff= 0.076, p=0.228 Total PM= 0.158, p=0.008
Reference: Tang et al. (2007) **Period of Study:** Dec 2003 to Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups**: 6–12 years **Study Design:** Panel study **N:** 30 children **Statistical Analyses:** Linear mixed-effect models were used to estimate the effect of PM exposure on PEFR **Covariates:** Gender, age, BMI, history of respiratory or atopic disease in family, SHS, acute asthmatic exacerbation in past 12 months, ambient temp and relative humidity, presence of indoor pollutants, and presence of outdoor pollutants, **Dose-response Investigated?** yes **Statistical Package**: S-Plus 2000 **Lags Considered:** 0-2	**Pollutant:** $PM_{2.5-1}$ **Averaging Time:** 1 h **Mean (SD):** Personal: 6.2 (4.8) **Range (Min, Max):** Personal: 0.3–86.8 **Monitoring Stations**: 1	No quantitative effects reported.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Tang et al. (2007) **Period of Study:** Dec 2003 to Feb 2005 **Location:** Sin-Chung City, Taipei County, Taiwan	**Outcome:** Peak expiratory flow rate (PEFR) of asthmatic children **Age Groups**: 6–12 years **Study Design:** Panel study **N:** 30 children **Statistical Analyses:** Linear mixed-effect models were used to estimate the effect of PM exposure on PEFR **Covariates:** Gender, age, BMI, history of respiratory or atopic disease in family, SHS, acute asthmatic exacerbation in past 12 months, ambient temp and relative humidity, presence of indoor pollutants, and presence of outdoor pollutants, **Dose-response Investigated?** yes **Statistical Package**: S-Plus 2000 **Lags Considered:** 0-2	**Pollutant:** PM_1 **Averaging Time:** 1 h **Mean (SD):** Personal: 34.0 (28.9) Ambient: 31.4 (18.8) **Range (Min, Max):** Personal: 1.8–284.6 Ambient: 0.1–128.4 Unit (i.e. µg/m³): µg/m³ **Monitoring Stations**: 1	**PM Increment:** 27.6 µg/m³ RR Estimate [Lower CI, Upper CI] ; lag: Change in morning PEFR: -6.44 (-30.18, 17.29) lag 0 -12.26 (-77.6 , 53.09) lag 1 -4.38 (-54.79, 46.03) lag 2 -44.06 (-113.79, 25.67) 2-day mean -6.01 (-101.48, 89.46) 3-day mean Change in evening PEFR: 1.17 (-17.79, 20.13) lag 0 -4.98 (-27.77, 17.81) lag 1 11.30 (-11.55, 34.16) lag 2 41.74 (11.36, 72.13) 2-day mean 28.21 (-19.08, 75.5) 3-day mean
Reference: Timonen et al. (2004) **Period of Study:** Oct 1998 to April 1999 **Location:** Amsterdam, Netherlands; Erfurt, Germany; Helsinki, Finland	**Outcome:** Urinary concentration of Clara cell protein CC16 of subjects with coronary heart disease **Age Groups**: 50+ **Study Design:** Longitudinal cohort study (panel) **N:** N=37 (Amsterdam) N=47 (Erfurt) N=47 (Helsinki) **Statistical Analyses:** The response of interest was log transformed, creatinine adjusted CC16. Mixed-effect model was used to investigate the association between CC16 and air pollutants. **Covariates:** Subjects, long term time trend, temperature (lags 0-3), relative humidity (lags 0-3), barometric pressure (lags 0-3), and weekday of visit. **Dose-response Investigated?** yes **Statistical Package**: S-Plus and SAS **Lags Considered:** 0-3	**Pollutant:** $NC_{0.01-0.1}$ **Averaging Time:** 24 h **Mean (SD):** Amsterdam: 17338 /cm3 Erfurt: 21124 /cm3 Helsinki: 17041 /cm3 **Range (Min, Max):** Amsterdam: 5699-37195 Erfurt: 3867-96678 Helsinki: 2305-50306 Unit (i.e. µg/m³): 1/cm3 **Monitoring Stations**: 3 $PM_{2.5}$: Amsterdam -0.15 Erfurt 0.62 Helsinki 0.14 NO_2: Amsterdam 0.49 Erfurt 0.82 Helsinki 0.72 CO: Amsterdam 0.22 Erfurt 0.72 Helsinki 0.35	**PM Increment:** 10,000 /cm³ RR Estimate [Lower CI, Upper CI] ; lag: Pooled estimate ; 1.7 (-4.4–7.8) lag 0 -1.8 (-8.3–4.6) lag 1 1.5 (-5.6–8.6) lag 2 2.3 (-4.8–9.3) lag 3 1.8 (-9.4–13.0) 5-day mean There was no association between $NC_{0.01-0.1}$ and CC16 in the pooled analysis.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Timonen et al. (2004) **Period of Study:** Oct 1998 to April 1999 **Location:** Amsterdam, Netherlands; Erfurt, Germany; Helsinki, Finland	**Outcome:** Urinary concentration of Clara cell protein CC16 of subjects with coronary heart disease **Age Groups**: 50+ **Study Design:** Longitudinal cohort study (panel) **N:** N=37 (Amsterdam) N=47 (Erfurt) N=47 (Helsinki) **Statistical Analyses:** The response of interest was log transformed, creatinine adjusted CC16. Mixed-effect model was used to investigate the association between CC16 and air pollutants. **Covariates:** Subjects, long term time trend, temperature (lags 0-3), relative humidity (lags 0-3), barometric pressure (lags 0-3), and weekday of visit. **Dose-response Investigated?** yes **Statistical Package**: S-Plus and SAS **Lags Considered:** 0-3	**Pollutant:** $NC_{10-0.1}$ **Averaging Time:** 24 h **Mean (SD):** Amsterdam: 2131 /cm3 Erfurt: 1829 /cm3 Helsinki: 1390 /cm3 **Range (Min, Max):** Amsterdam: 413-6413 Erfurt: 303-6848 Helsinki: 344-3782 Unit (i.e. µg/m^3): 1/cm3 **Monitoring Stations**: 3 **Copollutant (correlation):** Spearman Correlation: $NC_{0.1-0.01}$: Amsterdam 0.16 Erfurt 0.67 Helsinki 0.53 $PM_{2.5}$: Amsterdam 0.80 Erfurt 0.84 Helsinki 0.80 NO_2: Amsterdam 0.67 Erfurt 0.82 Helsinki 0.72 CO: Amsterdam 0.60 Erfurt 0.78 Helsinki 0.51	**PM Increment:** 1000 /cm3 RR Estimate [Lower CI, Upper CI] ; lag: Pooled estimate ; 4.3 (-1.4–10.0) lag 0 5.1 (-0.6–10.7) lag 1 4.5 (-0.5–9.6) lag 2 1.6 (-3.5–6.7) lag 3 13.1 (-4.3–30.5) 5-day mean CC16 was not associated to NC 0.1-1.0 in the pooled analysis but CC16 was significantly associated to NC 0.1-1.0 in Helsinki: 15.5 (0.001–30.9) lag 0 10.8 (-4.2–25.8) lag 1 10.5 9-4.1–25.1) lag 2 17.4 (3.4–31.4) lag 3 43.2 (17.4–69.0) 5-day mean
Reference: von Klot et al. (2002) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2- agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean=59.0 yrs and range =37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression models **Covariates:** seasonal variation in medication use or symptom prevalences, meteorological factors (relative humidity, temperature), weekend, Christmas holidays Season: winter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	**Pollutant:** $MC_{0.5-0.1}$ **Averaging Time:** 10 min intervals **Mean (SD):** 24.8 Percentiles: 25th: 11.4 50th(Median): 19.6 75th: 33.1 **Range (Min, Max):** (2.4-108.3) **Copollutant (correlation):** $PM_{10-2.5}$: r= 0.51 $NC_{0.1-0.01}$: r= 0.45 $NC_{0.5-0.1}$: r= 0.95 $NC_{2.5-0.5}$: r= 0.92 $MC_{2.5-0.01}$: r= 1.00 PM_{10}: r= 0.91 NO_2: r= 0.69 CO: r= 0.66 SO_2: r= 0.60	NC Increment: 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2-agonist use and MC0.1-0.5 Same day, IQR= 21, OR= 0.98 (0.92-1.04) 5-day mean, IQR= 21 OR= 1.11 (1.02-1.20) 14-day mean IQR= 17, OR= 1.01 (0.93-1.10) Association between the prevalence of inhaled corticosteroid use and MC0.1-0.5 Same day, IQR= 2, OR= 1.09 (1.02-1.17) 5-day mean IQR= 21, OR= 1.28 (1.18-1.39) 14-day mean, IQR= 17, OR= 1.49 (1.38-1.61) Association between the prevalence of wheezing and MC0.1-0.5 Same day, IQR= 21, OR= 1.01 (0.94-1.08) 5-day mean, IQR= 21, OR= 1.08 (0.99-1.17) 14-day mean, IQR= 17, OR= 1.05 (0.96-1.15)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: von Klot et al. (2002) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2- agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean=59.0 yrs and range =37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression models **Covariates:**seasonal variation in medication use or symptom prevalences, meteorological factors (relative humidity, temperature), weekend, Christmas holidays **Season:** Winter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	**Pollutant:** $MC_{2.5-0.01}$ **Averaging Time:** 10 min intervals **Mean (SD):** 30.3 Percentiles: 25th: 13.5 50th(Median): 24.6 75th: 41.3 **Range (Min, Max):** (3.6-133.8) **Copollutant (correlation):** $PM_{10-2.5}$: r= 0.52 $NC_{0.5-0.1}$: r= 0.45 $NC_{2.5-0.5}$: r= 0.94 $MC_{0.5-0.1}$: r= 1.00 $NC_{0.1-0.01}$: r= 0.45 PM_{10}: r= 0.94 NO_2: r= 0.68 CO: r= 0.65 SO_2: r= 0.62	NC Increment: 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2-agonist use and MC0.01-2.5 Same day, IQR= 28, OR= 0.96 (0.90-1.04) 5-day mean, IQR= 26 , OR= 1.10 (1.01-1.20) 14-day mean, IQR= 20, OR= 1.03 (0.95-1.12)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: von Klot et al. (2002) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2- agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean=59.0 yrs and range =37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression models **Covariates:**seasonal variation in medication use or symptom prevalences, meteorological factors (relative humidity, temperature), weekend, Christmas holidays Season: winter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	**Pollutant:** $NC_{0.1-0.01}$ **Averaging Time:** 10 min intervals **Mean (SD):** 17300 /cm3 Percentiles: 25th: 9286 50th(Median): 16940 75th: 24484 **Range (Min, Max):** (3272-46195) Unit (i.e. µg/m^3): 1/cm3 **Copollutant (correlation):** $PM_{10-2.5}$: r= 0.41 $NC_{0.5-0.1}$: r= 0.55 $NC_{2.5-0.5}$: r= 0.34 $MC_{0.5-0.1}$: r= 0.45 $MC_{2.5-0.01}$: r= 0.45 PM_{10}: r= 0.51 NO_2: r= 0.66 CO: r= 0.66 SO_2: r= 0.36	NC Increment: 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2-agonist use and NC0.01-0.1 Same day, IQR= 15000, OR= 0.97 (0.90-1.04) 5-day mean, IQR= 10000, OR= 1.11 (1.01-1.21) 14-day mean, IQR= 7700, OR= 1.08 (0.96-1.21) Association between two pollutants, jointly in one model, and the Outcomes Inhaled short-acting ß2- agonist use $NC_{0.1-0.01}$ OR= 1.07 (0.97-1.18) $MC_{0.5-0.1}$: OR= 1.07 (0.98-1.18) Inhaled corticosteroid use $NC_{0.1-0.01}$ OR= 1.01 (0.87-1.18) $MC_{0.5-0.1}$: OR= 1.53 (1.39-1.69) Wheezing $NC_{0.1-0.01}$ OR= 1.12 (1.01-1.24) $MC_{0.5-0.1}$: OR= 1.02 (0.92-1.12) Association between the prevalence of inhaled corticosteroid use and NC0.01-0.1 Same day, IQR= 15000, OR= 1.07 (1.00-1.15) 5-day mean, IQR= 10000, OR= 1.22 (1.12-1.33) 14-day mean, IQR= 7700, OR= 1.45 (1.29-1.63) Association between the prevalence of wheezing and $NC_{0.1-0.01}$ Same day, IQR= 15000, OR= 0.94 (0.86-1.01) 5-day mean, IQR= 10000, OR= 1.13 (1.03-1.24) 14-day mean, IQR= 7700, OR= 1.27 (1.13-1.43) Association between the prevalence of respiratory symptoms and $NC_{0.1-0.01}$ Attack of shortness of breath and wheezing Same day, IQR= 15000, OR= 1.01 (0.91-1.12) 5-day mean, IQR= 10000, OR= 1.08 (0.96-1.21) 14-day mean, IQR= 7700, OR= 1.26 (1.08-1.48) Walking up with breathing problems Same day, IQR= 15000, OR= 1.04 (0.96-1.13) 5-day mean, IQR= 10000, OR= 1.09 (0.99-1.19) 14-day mean, IQR= 7700, OR= 1.26 (1.13-1.41) Shortness of breath Same day, IQR= 15000, OR= 0.98 (0.90-1.06) 5-day mean, IQR= 10000, OR= 1.09 (0.99-1.19) 14-day mean, IQR= 7700, OR= 1.24 (1.11-1.40) Phlegm Same day, IQR= 15000, OR= 1.01 (0.94-1.09) 5-day mean, IQR= 10000, OR= 1.11 (1.02-1.21) 14-day mean, IQR= 7700, OR= 1.11 (0.99-1.25) Cough Same day, IQR= 15000, OR= 1.07 (0.98-1.16) 5-day mean, IQR= 10000, OR= 1.17 (1.07-1.28) 14-day mean, IQR= 7700, OR= 1.20 (1.06-1.35)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: von Klot et al. (2002) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2- agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean=59.0 yrs and range =37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression models **Covariates:**seasonal variation in medication use or symptom prevalences, meteorological factors (relative humidity, temperature), weekend, Christmas holidays Season: winter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	**Pollutant:** $NC_{0.5-0.1}$ **Averaging Time:** 10 min intervals **Mean (SD):** 2005 /cm^3 Percentiles: 25th: 958 50th(Median): 1610 75th: 2767 **Range (Min, Max):** (291-6700) Unit (i.e. µg/m^3): 1/cm^3 **Copollutant (correlation):** $PM_{10-2.5}$: r= 0.50 $NC_{0.1-0.01}$: r= 0.55 $NC_{2.5-0.5}$: r= 0.76 $MC_{0.5-0.1}$: r= 0.95 $MC_{2.5-0.01}$: r= 0.93 PM_{10}: r= 0.85 NO_2: r= 0.75 CO: r= 0.79 SO_2: r= 0.51	NC Increment: 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2-agonist use and $NC_{0.5-0.1}$ Same day, IQR= 1800, OR= 0.99 (0.92-1.05) 5-day mean, IQR= 1500, OR= 1.10 (1.03-1.19) 14-day mean, IQR= 1450, OR= 0.95 (0.86-1.05) Association between the prevalence of inhaled corticosteroid use and $NC_{0.5-0.1}$ Same day, IQR= 1800, OR= 1.06 (0.99-1.14) 5-day mean, IQR= 1500, OR= 1.23 (1.14-1.32) 14-day mean, IQR= 1450, OR= 1.51 (1.37-1.67) Association between the prevalence of wheezing and $NC_{0.5-0.1}$ Same day, IQR= 1800, OR= 1.00 (0.93-1.07) 5-day mean, IQR= 1500, OR= 1.08 (1.00-1.17) 14-day mean, IQR= 1450, OR= 1.11 (1.00-1.24)
Reference: von Klot et al. (2002) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2- agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean=59.0 yrs and range =37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression models **Covariates:**seasonal variation in medication use or symptom prevalences, meteorological factors (relative humidity, temperature), weekend, Christmas holidays Season: winter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	**Pollutant:** $NC_{2.5-0.5}$ **Averaging Time:** 10 min intervals **Mean (SD):** 21.4 /cm3 Percentiles: 25th: 5.6 50th(Median): 13.0 75th: 31.6 **Range (Min, Max):** (0.9-127.6) Unit (i.e. µg/m^3): 1/cm3 **Copollutant (correlation):** $PM_{10-2.5}$: r= 0.48 $NC_{0.1-0.01}$: r= 0.34 $NC_{0.5-0.1}$: r= 0.76 $MC_{0.5-0.1}$: r= 0.92 $MC_{2.5-0.01}$: r= 0.94 PM_{10}: r= 0.88 NO_2: r= 0.54 CO: r= 0.46 SO_2: r= 0.66	NC Increment: 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2-agonist use and $NC_{2.5-0.5}$ Same day, IQR= 26, OR= 0.99 (0.93-1.05) 5-day mean, IQR= 22, OR= 1.09 (1.01-1.17) 14-day mean, IQR= 17, OR= 1.08 (1.02-1.15) Association between the prevalence of inhaled corticosteroid use and $NC_{2.5-0.5}$ Same day, IQR= 26, OR= 1.13 (1.06-1.21) 5-day mean, IQR= 22, OR= 1.28 (1.19-1.37) 14-day mean, IQR= 17, OR= 1.44 (1.36-1.53) Association between the prevalence of wheezing and $NC_{2.5-0.5}$ Same day, IQR= 26, OR= 1.03 (0.95-1.10) 5-day mean, IQR= 22, OR= 1.05 (0.97-1.13) 14-day mean, IQR= 17, OR= 1.03 (0.96-1.10)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: von Klot et al. (2002) **Period of Study:** September 1996 to March 1997 (winter) **Location:** Erfurt, Germany	**Outcome:** Asthma symptoms (wheezing, shortness of breath at rest, waking up with breathing problems, or coughing without having a cold) and Asthma medication (inhaled short-acting ß2- agonists, inhaled long-acting ß2- agonists, inhaled corticosteroids, cromolyn sodium, theophylline, oral corticosteroids, and N-acetylcysteine) **Age Groups**: Adults, mean=59.0 yrs and range =37-77 yrs **Study Design:** panel study **N:** 53 adult asthmatics **Statistical Analyses:** Logistic regression models **Covariates:**seasonal variation in medication use or symptom prevalences, meteorological factors (relative humidity, temperature), weekend, Christmas holidays **Season**: winter **Dose-response Investigated?** No **Statistical Package**: NR **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, mov avg calculated from same day and preceding days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (SD):** 10.3 Percentiles: 25th: 2.9 50th(Median): 6.9 75th: 14.6 **Range (Min, Max):** (-8.7-64.3) **Copollutant (correlation):** $NC_{0.1-0.01}$: r= 0.41 $NC_{0.5-0.1}$: r= 0.50 $NC_{2.5-0.5}$: r= 0.48 $MC_{0.5-0.1}$: r= 0.51 $MC_{2.5-0.01}$: r= 0.52 PM_{10}: r= 0.67 NO_2: r= 0.45 CO: r= 0.42 SO_2: r= 0.28	**PM Increment:** 1 IQR Effect Estimate [Lower CI, Upper CI]: Association between the prevalence of inhaled ß2-agonist use and $PM_{10-2.5}$ Same day, IQR= 12, OR= 1.01 (0.95-1.06) 5-day mean, IQR= 11, OR= 1.01 (0.94-1.09) 14-day mean, IQR= 6.7, OR= 0.92 (0.86-1.00) Association between the prevalence of inhaled corticosteroid use and $PM_{10-2.5}$ Same day, IQR= 12, OR= 1.03 (0.98-1.08) 5-day mean, IQR= 11, OR= 1.12 (1.04-1.20) 14-day mean, IQR= 6.7, OR= 1.27 (1.18-1.37) Association between the prevalence of wheezing and $PM_{10-2.5}$ Same day, IQR= 12, OR= 0.97 (0.91-1.02) 5-day mean, IQR= 11, OR= 1.06 (0.98-1.15) 14-day mean, IQR= 6.7, OR= 1.05 (0.96-1.15)

E.2.2. Respiratory Emergency Department Visits and Hospital Admissions

Table E-13. Short-term exposure to PM_{10} and emergency department visits and hospital admissions for respiratory outcomes.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Andersen et al. 2008 (2008b) **Period of Study:** May 2001 - December 2004 **Location:** Copenhagen, Denmark	Hospital Admissions/ED visits **Outcome (ICD-10):** **RD**, including chronic bronchitis (J41 – 42), emphysema (J43), other chronic obstructive pulmonary disease (J44), asthma (J45), and status asthmaticus (J46). **Pediatric hospital admissions for asthma** (J45) and status asthmaticus (J46). **Age Groups Analyzed:** >65 yrs (RD combined), 5 – 18 years (asthma) **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson GAM **Covariates:** temperature, dew-point temperature, long-term trend, seasonality, influenza, day of the week, public holidays, school holidays (only for 5 – 18 year olds), pollen (only for pediatric asthma outcome) **Season:** NR **Dose-response Investigated:** No **Statistical package:** R statistical software (gam procedure, mgcv package) **Lags Considered:** Lag 0 -5 days, 5-day average (lag 0 – 4) for RD, and a 6-day average (lag 0 – 5) for asthma.	**Pollutant:** PM_{10} ($\mu g/m^3$) **Averaging Time:** 24 h **Mean (SD; median; IQR; 99th percentile:** 24 (14; 21; 16 – 29; 72) **Monitoring Stations:** 1 **Copollutant (correlation):** NCtot: r = 0.39; NC100: r = 0.28; NCa12: r = 0.02; Nca23: r = -0.12; NCa57: r = 0.45; Nca212: r = 0.63; $PM_{2.5}$: r = 0.80; CO: r = 0.37; NO_2: r = 0.35; NO_X: r = 0.32; NO_Xkerbside: r = 0.18; O_3: r = -0.21 **Other variables:** Temperature: r = 0.12 Relative humidity: r = 0.05	**PM Increment:** 13 $\mu g/m^3$ [3] (IQR) **Relative risk (RR) Estimate [CI] :** **RD hospital admissions (5 day average, lag 0 -4), age 65+:** One-pollutant model: 1.06 [1.02 – 1.09] Adj for NCtot: 1.05 [1.01 – 1.10] Adj for NCa212: 1.04 [0.98 – 1.11] **Asthma hospital admissions (6 day avg lag 0 – 5), age 5 - 18 :** One-pollutant model: 1.02 [0.93 – 1.12] Adj for NCtot: 1.01 [0.91 – 1.12] Adj for NCa212: 0.94 [0.81 – 1.09] Estimates for individual day lags reported only in figure form (see notes): **Notes :** Figure 2: Relative risks and 95% confidence intervals per IQR in single day concentration (0 – 5 day lag). Summary of Figure 2: RD: Positive, statistically or marginally significant associatons at Lag 2 – 5. Asthma: Wide confidence intervals make interpretation dificult. Positive associations at Lag 1, 2, 3, and 5.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Andersen et al. (2007) **Period of Study:** 1/99-12-04 **Location:** Copenhagen, Denmark	**Outcome (ICD10):** Respiratory diseases: Chronic bronchitis (J41-42), emphysema ((J43), other COPD (J44), asthma (J45), status asthmaticus (J46) **Age Groups:** Age >65, Ages 5-18 **Study Design:** Time series **N:** 2192 days, 9 Hospitals **Statistical Analyses:** Principal Component Analysis and Constrained Physical Receptor Model (COPREM), Poisson regression, GAM, **Covariates:** Season, day of the wk, public holidays, influenza epidemics, grass pollen, school holidays, and meterology **Season:** All year **Dose-response Investigated?** No **Statistical Package:** R, gam/mgcv package **Lags Considered:** 0-6 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h Avg **Mean (SD):** 25 (14) μg/m^3 Percentiles: 25th: 16 50th(Median): NR 75th: 30 **Monitoring Stations**: 1 station Notes: **Copollutant (correlation):** PM_{10}: CO; r = 0.45 NO_2; r = 0.42 PM_{10}: Biomass; r = 0.53 Secondary; r = 0.73 Oil; r = 0.57 Crustal; r = 0.37 Sea salt; r = 0.04 Vehicle; r = 0.02 Notes: ASV	**PM Increment:**14 μg/m^3 RR Estimate Respiratory disease (age >65) Single pollutant model: 1.037 [1.014, 1.060], 5 d ma 2-pollutant model: PM_{10} w/ CO: 1.035[1.006, 1.065], 5 d ma PM_{10} w/ NO_2: 1.032[1.007, 1.059], 5 d ma Asthma (age 5-18) Single pollutant model: 1.077 [1.004-1.155] 6 d ma Two-pollutant model: 1.077[0.989, 1.172]; 6 d ma 1.032[1.007, 1.059]; 6 d ma
Reference: Anderson et al. (2007) **Period of Study:** 1/99-12-04 **Location:** Copenhagen, Denmark	Hospital Admission **Outcome (ICD10):** Respiratory diseases: Chronic bronchitis (J41-42), emphysema ((J43), other COPD (J44), asthma (J45), status asthmaticus (J46) **Age Groups:** Age >65, Ages 5-18 **Study Design:** Time series **N:** 2192 days, 9 Hospitals **Statistical Analyses:** Principal Component Analysis and Constrained Physical Receptor Model (COPREM), Poisson regression, GAM, **Covariates:** Season, day of the wk, public holidays, influenza epidemics, grass pollen, school holidays, and meterology **Season:** All year **Dose-response Investigated?** No **Statistical Package:** R, gam/mgcv package **Lags Considered:** 0-6 days	**Pollutant:** Source specific PM_{10} components **Averaging Time:** 24-h Avg **Mean (SD): Percentiles:** 25th: 16 50th(Median): NR 75th: 30 **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: Biomass; r = 0.53 Secondary; r = 0.73 Oil; r = 0.57 Crustal; r = 0.37 Sea salt; r = 0.04 Vehicle; r = 0.02 Notes: Correlations between source specific PM_{10} components presented in paper	**PM Increment:** 14 μg/m^3 **RR Estimate** **Respiratory disease (age >65)** Single pollutant model: PM_{10} (other 5 sources): 1.045 [1.016, 1.074] Biomass: 1.04 [1.009, 1.072] Secondary: 1.05 1.021, 1.081] Oil: 1.035[1.006, 1.065] Crustal: 1.054 [1.028, 1.081] Sea salt: 0.98 [0.947, 1.017] Vehicle: 0.989 [0.949, 1.032] Asthma (age 5-18) Single pollutant model: PM_{10} (other 5 sources): 1.004 [0.866, 1.164] Biomass: 0.979 [0.848, 1.131] Secondary: 0.936 [0.815, 1.075] Oil: 1.004 [0.862, 1.17] Crustal: 0.942 [0.8, 1.108] Sea salt: 0.93 [0.793, 1.091] Vehicle: 1.203 [0.983, 1.473] **Notes:** 2 pollutant model results for source specific components also presented in manuscript.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Atkinson et al. (2004) **Period of Study:** 1992 - 1996 **Location:** 8 European cities: Barcelona, Spain; Birmingham, UK; London, UK; Milan, Italy; The Netherlands; Paris, France; Rome; Italy; and Stockholm, Sweden	**Outcome:** Daily counts of hospital admissions for asthma (ICD-9: 493), COPD and asthma (ICD-9: 490–496), and all respiratory disease (ICD-9: 460–419). **Age Groups:** 0–14 years, 15–64 years, and 65 + years **Study Design:** Time series **N:** NR **Statistical Analyses:** "Regression models"–type not specified. **Covariates:** season, temperature, humidity, holiday periods, influenza episodes, and air pollution measure **Season:** NR **Dose-response Investigated:** No **Statistical Package:** GAM with strict convergence criteria **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median (SD; median; minimum - maximum):** Barcelona: 53.3 (17.1, 131.7) Birmingham: 21.5 (6.5, 115) London: 24.9 (7.8, 80.4) Netherlands: 33.4 (11.3, 130.8) Stockholm: 13.6 (4.3, 43.3) **Copollutant (correlation):** Barcelona: SO_2: r = 0.34; O_3: r = 0.03; NO_2: r = 0.48 Birmingham: SO_2: r = 0.77; O_3: r = -0.28; NO_2: r = 0.68 London: SO_2: r = 0.72; O_3: r = 0.00; NO_2: r = 0.70 Milan: SO_2: r = 0.64; O_3: r = -0.25; NO_2: r = 0.72 Netherlands: SO_2: r = 0.67; O_3: r = -0.01; NO_2: r = 0.64 Paris: SO_2: r = 0.63; O_3: r = -0.11; NO_2: r = 0.44 Rome: SO_2: r = 0.15; O_3: r = 0.12; NO_2: r = 0.32 Stockholm: SO_2: r = 0.36; O_3: r = 0.40; NO_2: r = 0.30 **Other variables:** Barcelona: Temperature: r = -0.02 Humidity: r = 0.11 Birmingham: Temperature: r = -0.13 Humidity: r = 0.11 London: Temperature: r = 0.2 Humidity: r = 0.04 Milan: Temperature: r = -0.21 Humidity: r = 0.17 Netherlands: Temperature: r = -0.07 Humidity: r = -0.08 Paris: Temperature: r = -0.17 Humidity: r = 0.12 Rome: Temperature: r = 0.21 Humidity: r = 0.03 Stockholm: Temperature: r = 0.06 Humidity: r = -0.13	**PM Increment:** 10 µg/m^3 **Percentage increase estimate [95% CI]:** Asthma (0–14 years old): 1.5 (0.1, 2.8) Asthma (15–64 yr):1.0 (0.3, 1.8) COPD + asthma (65 + years): 1.0 (0.6, 1.4) All respiratory diseases (65 + yr): 1.0 (0.7, 1.3) **Notes:** This is a reanalysis of a 2001 study using a reduction in the criterion for model convergence and an increase in the number of iterations allowed for this convergence criterion to be met.
Reference: Bedeschi et al. (2007) **Period of Study:** 1/3/2001–31/3/2002 **Location:** Reggio Emilia, Italy	ER visits (pediatric) **Outcome(s):** All RD (symptoms): asthma; asthma-like disorders; other (upper and lower respiratory illness, sinusitis, bronchitis, pneumonia) **Age Groups:** <15 yrs **Study Design:** time series **N:** 1051 ER visits **Statistical Analyses:** GAM. penalized splines **Covariates:** Temperature (current and lagged), humidity, precipitation, weekday, festivity day, flu, pollen concentrations; Stratified on Italian/ foreign born **Season:** all seasons **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** 0-5 days **Notes:** Children with more than 5 ER visits due to influenza were not enrolled	**Pollutant:** PM_{10} **Averaging Time:** 24 H Avg **Mean (SD):** 51.2(30.6) µg/m^3 50th(Median): 44.7 **Range (Min, Max):** (5, 196.8) **Monitoring Stations:** 6 **Copollutant (correlation):** Tsp: r = 0.89 SO_2: r = 0.57 NO_2: r = 0.57 Co: r = 0.61 O_3: r = -0.52	**PM Increment:** 10 µg/m^3 % Change, Lag All Children 3%(0.4, 5.7%) Lag 3 Italian Children Only 2.9%(0.0, 5.9%) Lag3 Foreign Children 4.3%(-0.5, 9.4%) Lag 4 Notes: Results For All Lags Presented In Figure, Significant Results Highlighted In The Text.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Bell et al. (2008b) **Period of Study:** 1995 - 2002 **Location:** Taipei, Taiwan	**Outcome (ICD-9):** Asthma (493), and pneumonia (486). **Age Groups:** All **Study Design:** Time series **N:** 19,966 for pneumonia and 10,231 for ashtma. **Statistical Analyses:** Poisson regression **Covariates:** Day of the week, time, apparent temperature, long-term trends, seasonality **Season:** All **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** lags 0-3 days, avg of lags 0-3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (range; IQR):** 49.1 (12.7–215.5; 27.6) **Monitoring Stations:** Taipei area: 13 monitors Taipei City: 5 monitors Monitors with correlations of 0.75 + for PM_{10}: 12 monitors **Copollutant:** NR	**PM Increment:** 28 µg/m^3 (near IQR) **Percentage increase estimate [95% CI]: Asthma:** Taipei area (13 monitors): L0: 2.63 (-0.01, 5.35); L1: 1.79 (-0.82, 4.48); L2: 2.20 (-0.41, 4.88); L3: 1.87 (-0.71, 4.50); L03: 4.48 (0.71, 8.38) Taipei City (5 monitors): L0: 2.53 (-0.09, 5.21); L1: 1.60 (-0.98, 4.25); L2: 2.21 (-0.37, 4.86); L3: 2.08 (-0.47, 4.69); L03: 4.68 (0.78, 8.73) Monitors with > = 0.75 between monitor correlations (12 monitors): L0: 2.56 (-0.04, 5.23); L1: 1.63 (-0.95, 4.26) ; L2: 1.97 (-0.59, 4.60); L3: 2.78 (-0.75, 4.37) ; L03: 4.27 (0.47, 8.22) **Pneumonia:** Taipei area (13 monitors): L0: 0.75 (-1.80, 3.36); L1: 0.16 (-2.36, 2.74); L2: 0.47 (-2.04, 3.03); L3: -0.70 (-3.19, 1.85); L03: 0.31 (-3.22, 3.97) Taipei City (5 monitors): L0: 0.88 (-1.64, 3.46); L1: 0.50 (-1.98, 3.05); L2: 0.59 (-1.88, 3.13); L3: -0.72 (-3.18, 1.79); L03: 0.83 (-2.83, 4.62) Monitors with > = 0.75 between monitor correlations (12 monitors): L0: 0.86 (-1.67, 3.41) ; L1: 0.12 (-2.35, 2.65) ; L2: 0.52 (-1.94, 3.05) ; L3: -0.53 (-2.98, 1.97) ; L03: 0.65 (-2.93, 4.36)
Reference: Bennett et al. (2006) **Period of Study:** Jan 1997-Dec 1999 **Location:** Greater Vancouver, British Columbia, Canada	Hospital Admissions **Outcome:** primary code was "respiratory": All RD **Age Groups:** all **Study Design:** time series **N:** 34,990 respiratory hospitalizations. **Statistical Analyses:** Chi-squared tests **Covariates:** age, sex, postcode, admissions, discharges, diagnoses **Season:** all **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** up to 2 weeks **Notes:** patients in long term care, day surgery patients and rehabilitation cases excluded	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Monitoring Stations:** 8 **Notes:** During Event Hourly PM_{10} Levels In Excess Of 100 µg/m^3 Observed. Daily Averages Several Times Greater Than Normal. Copollutant**:** NR	**Notes:** No statistically significant results observed. time series graphically presented **Notes:** naturally derived PM from a Gobi desert dust event in 1998
Reference: Chardon et al (2007) **Period of Study:** 2000-2003 **Location:** Greater Paris Area, France	Doctors house calls **Outcome (ICPC2):** Asthma (R96), Upper respiratory disease (URD R07, R21, R29, R75, R76, R02) , Lower respiratory disease (LRD, R05, R78) **Age Groups:** all **Study Design:** Time series **N:** 8027 for asthma; 52928 for LRD; 74845 for URD **Statistical Analyses:** Quasi-Poisson, GAM, parametric penalized spline smoothers. **Covariates:** Lagged and current temperature, humidity, long term trends, seasonality, pollen counts, influenza epidemic, days of the week, holidays, bank holidays **Season:** All **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 H Avg **Mean (SD):** 23.0(9.87) µg/m^3 **Percentiles:** 25th: 16.2 50th(Median): 21.0 75th: 27.7 **Range (Min, Max):** (6.3,97.3) **Monitoring Stations:** 7-9 **Copollutant (correlation):** $PM_{2.5}$: r = 0.95 NO_2: r = 0.68	**PM Increment:** 10 µg/m^3 % Change, Lag 0-3 D Avg Urd 2.9 (0.8, 5.1) Lrd 3.1(0.9,5.4) Asthma 2.5(-1.7, 6.8) % Change, Lag 0-15 D Avg Lrd 8.7(5.0,12.5) Urd 4.9(1.1,9.0) % Change, Lag 0-15 D Avg, Controlled For 0-15 D Lag Weather Lrd 10.5(6.7,14.4) Urd 6.3(2.4, 10.3) **Notes:** Additional Results For Lrd At Other Lags Given In A Figure

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Fung et al. (2006) **Period of Study:** 6/1/95–3/31/99 **Location:** Vancouver, Canada	Hospital Admission/ED **Outcome:** Respriatory diseases (460-519) **Age Groups:** Age >65 **Study Design:** Time series **N:** 26,275 individuals admitted **Statistical Analyses:** Poisson regression (spline 12 knots), case-crossover (controls +/7 d days from case date), Dewanji and Moolgavkar (DM) method **Covariates:** Long-term trends, day-of-the-week effect, weather **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SPlus, R **Lags Considered:** 0-7 d	**Pollutant:** PM_{10} **Averaging Time:** 24-h Avg **Mean (SD):** 13.31(6.13) µg/m^3 **Range (Min, Max):** (3.77, 52.17) **Monitoring Stations:** NR **Copollutant (correlation):** PM_{10}: $PM_{2.5}$; r = 0.80 $PM_{10-2.5}$; r = -0.11 Co; r = 0.46 Coh; r = 0.61 O_3; r = -0.08 NO_2; r = 0.54 SO_2; r = 0.61	**PM Increment::** 7.9 µg/m^3 Rr Estimate (65+ Years) Dm Method: 1.014[0.998,1.029]; Lag 0 1.016[0.998,1.034]; 3 D Avg 0.988[0.970, 1.006]; 5 D Avg 0.983[0.963, 1.004]; 7 D Avg Time Series: 1.016[0.999, 1.033]; Lag 0 1.015[0.996, 1.035]; 3 D Avg 1.009[0.987, 1.032]; 5 D Avg 1.009[0.983, 1.036]; 7 D Avg Case-Crossover: 1.017[0.998, 1.036]; Lag 0 1.015[0.993, 1.037]; 3 D Avg 1.008[0.984, 1.033]; 5 D Avg 1.003[0.976, 1.031]; 7 D Avg
Reference: Hajat et al. (2002) **Period of Study:** 1/1992-12-1994 **Location:** London, England	Family Practice consultations **Outcome:** Upper Resp Disease (excluding allergic rhinitis) (460-3), (465), (470-5), (478) **Age Groups:** 0-14, 15-64, >65 yrs **Study Design:** Time series **N:** 268,718-295,740 registered patients **Statistical Analyses:** Poisson regression, GAM, LOESS smoothers, default convergence criteria **Covariates:** long term trends, pollen counts, flu, meteorological variables **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SPLUS **Lags Considered:** 2-3	**Pollutant:** PM_{10} **Averaging Time:** 24-H **Mean (SD):** 28.5 (13.7) µg/m^3 Percentiles: 10th: 15.8 90th: 46.5 **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** All Year: 18 Warm Season: 15 Cold Season: 20 **% Change, Single Pollutant Models:** All Year: Ages 0-14: 2.0[-0.2, 4.2] Lag 3; Ages 15-64: 5.7[2.9, 8.6] Lag 2; Ages >65: 10.2[5.3, 15.3] Lag 2 Warm Season: Ages 0-14: 1.1[-2.4, 4.8] Lag 3; Ages 15-64: 6.0[2.7, 9.4] Lag 2; Ages >65: 0.1[-7.7, 8.5] Lag 2 Cold Season: Ages 0-14: 2.7[-0.1, 5.5] Lag 3; Ages 15-64: 3.6[1.0, 6.4] Lag 2; Ages >65: 18.9[11.7, 26.7] Lag 2 **% Change, 2 Pollutant Models:** 0-14 Yrs PM_{10} w/ NO_2: 3.8[1.6, 6.1]; PM_{10} w/ O_3: 1.8[-0.4, 3.9]; PM_{10} w/ SO_2: 2.0[-0.6, 4.6] 15-65 Yrs PM_{10} w/ NO_2: 2.8[0.7, 4.9]; PM_{10} w/ O_3: 4.8[2.6, 7.0]; PM_{10} w/ SO_2: 4.8[2.2, 7.5] >65 Yrs PM_{10} w/ NO_2: 4.6[0.5, 8.8]; PM_{10} w/ O_3: 10.7[5.7, 16.0]; PM_{10} w/ SO_2: 10.6[4.5, 17.1]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hanigan et al (2008) **Period of Study:** 1996–2005 (April–November of each year) **Location:** Darwin, Australia	**Outcome: T**otal respiratory (ICD-9: 460–519; ICD-10: J00–J99), asthma (ICD-9: 493; ICD-10: J45–J47), COPD (ICD-9: 490–492, 494–496; ICD-10: J40–J44, J47, J67), and respiratory infections (ICD-9: 461–466, 480–487, 514; ICD-10: J00–J22). **Age Groups:** All **Study Design:** Time series **N:** 8,279 hospital admissions **Statistical Analyses:** Poisson generalized linear models **Covariates:** Indigenous status, time in days, temperature, relative humidity, day of the week, influenza epidemics, change between ICD editions, holidays, yearly population **Season:** April–November (corresponding to the dry season) **Dose-response Investigated?** No **Statistical Package:** R version 2.3.1 **Lags Considered:** Lag 0 -3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD; range):** 21.2 (8.2; 55.2) **Monitoring Stations:** N/A (see notes) **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **Percent change [95% CI]:** Overall respiratory disease: Lag 0: 4.81 [-1.04, 11.01] Lag 0 (indigenous people): 9.40 [1.04, 18.46] Lag 0 (non-indigenous people): 3.14 [-2.99, 9.66] In unstratified analyses, the subgroups of respiratory infections, asthma, and COPD all had positive associations with PM_{10} Lag 0. Asthma: Lag 1 (indigenous people): 16.27 [-3.55; 40.17] Lag 1 (non-indigenous people): 8.54 [-5.60, 24.80] Respiratory infections: Lag 3 (indigenous people): 15.02 [3.73, 27.54] Lag 3 (non-indigenous people); 0.67 [-7.55, 9.61]
Reference: Hwang and Chan (2002) **Period of Study:** 1998 **Location:** Taiwan	Clinic visits **Outcome:** LRI 466, 480-486 (acute bronchitis, acute bronchiolitis, pneumonia) **Age Groups:** 0-14 yrs, 15-64, 65+ yrs **Study Design:** Cluster analysis of small study areas **N:** 50 communities **Statistical Analyses:** GLM to model temporal patterns, hierarchical model to obtain estimates across 50 communities **Covariates:** day of week, temperature, dew point, summer/Winter **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** 0-2	**Pollutant:** PM_{10} **Averaging Time:** 24 H **Mean (SD):** 58.9 µg/m^3 (14.0) **Range (Min, Max):** 33.3, 83.1 µg/m^3 **PM Component:** **Monitoring Stations:** 59 **Notes:** Number Of Stations Estimated From Figure. **Copollutant:** NR	**PM Increment:** 10% Increase In PM_{10} (5.9 µg/m^3) Percent Change: 0-14 0.5% (-0.1, 0.8] Lag0 [-0.3, 0.3] Lag1 0.3 [0.0, 0.6] Lag2 15-64 0.6 [0.2, 0.9] Lag0 0.2 [-0.1, 0.5] Lag1 0.3 [0.0, 0.6] Lag2 65+ 0.8 [0.4, 1.1] Lag0 0.3 [-0.1, 0.6] Lag1 0.5 [0.1, 0.8] Lag2 All Ages 0.5 [0.2, N0.8] Lag0 [-0.3, 0.3] Lag1 0.3 [0.0, 0.6] Lag2

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Jaffe et al. (2003) **Period of Study:** 7/1/91–6/30/96 **Location:** Cincinnati, Cleveland, Columbus, Ohio	ED visits **Outcome (ICD10):** Asthma (493) **Age Groups:** Age 5-34 years **Study Design:** Time-series **N:** 4,416 recipients **Statistical Analyses:** Poisson regression, GAM **Covariates:** City, day of week, wk, yr, minimum temperature, dispersion parameter **Season:** June-August only **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24-H **Mean (SD):** Cincinnati: 43.0(16.4) Cleveland: 60.8(28.4) Columbus: 37.4(16.3) **Range (Min, Max):** Cincinnati: (16,90) Cleveland: (12,183) Columbus: (7,87) **Monitoring Stations:** 3 **Copollutant (correlation):** Cincinnati: PM_{10} O_3; r = 0.42; NO_2; r = 0.36; SO_2; r = 0.31 Cleveland: PM_{10} O_3; r = 0.42; NO_2; r = 0.34; SO_2; r = 0.29 Columbus: PM_{10} O_3; r = 0.51; NO_2; r = Na; SO_2; r = 0.42	**PM Increment:** 50 µg/m^3 **% Change** Asthma Cincinnati: -22%[-49,-19] Lag 3 Cleveland: 12%[0,27] Lag 2 Columbus: 32%[-6,-85] Lag 3 **Ar Estimate [Lower Ci, Upper Ci]; Lag:** Asthma Cincinnati: PM_{10}: Nr Cleveland: PM_{10}: 1.32 Columbus: PM_{10}: 3.62 **Notes:** dose response was investigated by assessing the relationship between odds of ed visit by quintile of PM_{10}. Results are displayed in figure. "no consistent effects for all three cities were observed for PM_{10}." Rate ratios were also reported for each city.
Reference: Johnston et al. (2007) **Period of Study:** 2000, 2004, 2005 (April–November of each year) **Location:** Darwin, Australia	**Outcome (ICD-10):** All respiratory conditions (J00–J99), including asthma (J45–46), COPD (J40–J44), and respiratory infections (J00–J22). **Age Groups:** All **Study Design:** Case-crossover **N:** 2466 emergency admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Weekly influenza rates, temperature, humidity, days with rainfall >5mm, public holidays, school holiday periods (for respiratory conditions only) **Season:** April–November (dry season) **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0–3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median (IQR, 10th–90th percentile, range):** 17.4 (13.6–22.3; 10.3–27.7; 1.1–70.0) **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **OR Estimate [95% CI]:** **All respiratory conditions:** Lag 0: 1.08 [0.98–1.18]; Lag 0 (indigenous): 1.17 [0.98–1.40] **COPD:** Lag 0: 1.21 [1.0–1.47]; Lag 0 (indigenous): 1.98 [1.10–3.59] **Asthma:** Lag 0: 1.14 [0.90–1.44] **Asthma + COPD:** Lag 0: 1.19 [1.03–1.38] **Notes:** All other results expressed in Figures. **Figure 1:** Adjusted OR and 95% CI for hospital admissions for all respiratory conditions per 10 µg/m^3 rise in PM_{10} for the same day and lags up to 3 days, overall and stratified by indigenous status. Summary: Marginally significant positive association at Lag 0 in overall study population. Larger marginally significant positive association among indigenous people. **Figure 2:** OR and 95% CI for hospital admissions for COPD. Summary: Marginally significant positive associations at Lag 0 and Lag 1 in overall study population and among non-indigenous people. Large, statistically significant positive association at Lag 0 for indigenous people, with smaller, non-significant positive associations at Lag 1 and Lag 2. **Figure 3:** OR and 95% CI for hospital admissions for asthma. Summary: Positive, non-significant (sometime marginally significant) associations at Lag 0, Lag 2, and Lag 3 for overall population and indigenous status strata. **Figure 4:** OR and 95% CI for hospital admissions for respiratory infections. Summary: Negative associations at Lag 2 and Lag 3 in all population strata.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kim et al. (2007B) **Period of Study:** 2002 **Location:** Seoul, Korea	Ed Visits **Outcome (ICD10):** Asthma (J45), (J46) **Age Groups:** All Ages **Study Design:** Cass-Crossover **N:** 92,535 Visits **Statistical Analyses:** Conditional Logistic Regressioin, Relative Effect Modification (Rem) **Covariates:** Time Trend, Season, Daily Mean Temperature, Relative Humidity, Air Pressure. Sep As Modifier Of Air Pollution Asthma Visit Association. **Season:** All Year **Dose-response Investigated?** No **Statistical Package:** Nr **Lags Considered:** 0-2 Days	**Pollutant:** PM_{10} **Averaging Time:** 8-H **Mean (SD):** Daily Concentration: 67.6 (39.0) µg/m³ Relevant Exposure Term (Difference Between Concentration On Event Day And Mean Of Concentrations On Control Days): 26.0 (19.7) **Percentiles:** 50th(Median): Daily Concentration: 61.9 Relevant Exposure Term: 21.6 **Range (Min, Max):** Daily Concentration: (4.9, 302.0) Relevant Exposure Term: (0.0, 143.1) **Monitoring Stations:** 3 **Copollutant:** Nr	**PM Increment:** 47.4 µg/m³ **Rr Estimate For Asthma (Stratified By Sep):** Individual Level Sep: Quintile 1–1.06[1.02, 1.09] Quintile 2–1.07[1.04, 1.10] Quintile 3–1.06[1.03, 1.10] Quintile 4–1.03[0.99, 1.07] Quintile 5–1.10[1.05, 1.14] Regional Level Sep: Quintile 1–1.04[0.99, 1.10] Quintile 2–1.03[1.00, 1.07] Quintile 3–1.05[1.03, 1.08] Quintile 4–1.06[1.02, 1.10] Quintile 5–1.09[1.06, 1.13] Total-1.06[1.04, 1.08], 3 D Ma **Notes:** Relative Effect Modification (Rem) Estimates Presented In Paper.
Reference: Ko et al. (2007b) **Period Of Study:** 1/2000-12/2004 **Location:** Hong Kong, China	Ed Visits **Outcome (ICD-9):** COPD: chronic bronchitis (491), emphysema (492), chronic airway obstruction (496) **Age Groups:** All Ages **Study Design:** Time Series **N:** 15 hospitals, 119,225 admissions **Statistical Analyses:** Poisson regression, gam with stringent convergence criteria, aphea2 protocol. **Covariates: t**ime trend, season, temperature, humidity, other cyclical factors, day, day of wk, holidays **Season:** All year, interactions with season tested **Dose-response Investigated?** No **Statistical Package:** Splus 4.0 **Lags Considered:** 0-5 days	**Pollutant:** PM_{10} **Averaging Time:** 24-H **Mean (SD):** 50.1(23.9) µg/m³ **Percentiles**: 25th: 31.9 50th(Median): 44.5 75th: 64.1 **Range (Min, Max):** (13.6, 172.2) **Monitoring Stations:** 14 Stations **Copollutant (correlation):** PM_{10}: SO_2; r = 0.436 NO_2; r = 0.229 O_3; r = 0.421 $PM_{2.5}$; r = 0.952	**PM Increment:** 10 µg/m³ **Rr Estimate** COPD: 1.003[1.000, 1.005]; Lag 0 1.005[1.002, 1.007]; Lag 1 1.010[1.007, 1.012]; Lag 2 1.011[1.008, 1.013]; Lag 3 1.008[1.006, 1.011]; Lag 4 1.007[1.004, 1.009]; Lag 5 1.005[1.002, 1.008]; Lag 0-1 1.011[1.008, 1.014]; Lag 0-2 1.016[1.013, 1.019]; Lag 0-3 1.020[1.017, 1.024]; Lag 0-4 1.024[1.021, 1.028]; Lag 0-5
Reference: Ko et al. (2007) **Period of Study:** 1/2000-12/2005 **Location:** Hong Kong, China	Hospital Admission **Outcome (ICD-9):** Asthma (493) **Age Groups:** All, 0-14, 15-56, 65+ **Study Design:** Time series **N:** 69,716 admissions, 15 hospitals **Statistical Analyses:** Poisson regression, with GAM with stringent convergence criteria. **Covariates:** Time trend, season, temperature, humidity, other cyclical factors **Season:** All year, evaluated effect of season in analysis **Dose-response Investigated?** No **Statistical Package:** SPLUS 4.0 **Lags Considered:** 0-5 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 52.5(27.1) µg/m³ **Percentiles: 25th:** 30.9 **50th(Median):** 47.1 **75th:** 68.8 **Range (Min, Max):** (13.4, 198.9) **Monitoring Stations:** 14 stations **Copollutant (correlation):** PM_{10}: SO_2; r = 0.436 NO_2; r = 0.761 O_3; r = 0.600 $PM_{2.5}$; r = 0.956	**PM Increment:** 10.0 µg/m³ **RR Estimate:** Asthma (Single-pollutant model): 1.006[1.003, 1.010]; lag 0 1.005[1.002, 1.009]; lag 1 1.005[1.002, 1.009]; lag 2 1.008[1.005, 1.012]; lag 3 1.006[1.002, 1.009]; lag 4 1.006[0.999, 1.006]; lag 5 1.008[1.004, 1.012];lag 0-1 1.012[1.008, 1.016]; lag 0-2 1.015[1.011, 1.019]; lag 0-3 1.018[1.013, 1.022]; lag 0-4 1.019[1.015, 1.024]; lag 0-5 Asthma by age group 0-14: 1.023[1.015, 1.031]; lag 0-5 14-65: 1.014[1.006, 1.022]; lag 0-5 >65: 1.015[1.009, 1.022]; lag 0-4 Asthma–Effect of seasoN: 1.148[1.051, 1.245] lag 0-5

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lee et al. (2002) **Period of Study:** 12/1/1997-12/31/1999 **Location:** Seoul, Korea	Hospital Admissions **Outcome (ICD10):** Asthma, J45, J46, **Age Groups:** Children <15 years **Study Design:** Time-Series **N:** 822 d, 6,436 admissions **Statistical Analyses:** Poisson regression, GAM, LOESS smoothers. **Covariates:** Days of the week, temperature, humidity **Season:** All **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-5, 0-1 moving averages for 1-2, 2-3, and 3-4 days	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 64.0 (31.8) µg/m^3 **Percentiles: 25th:** 40.5 µg/m^3 **50th(Median):** 59.1 µg/m^3 **75th:** 80.9 µg/m^3 **Range (Min, Max):** NR **Monitoring Stations:** 27 **Notes: Copollutant (correlation):** PM_{10}-SO_2: 0.585 PM_{10}-NO_2: 0.738 PM_{10}-O_3: 0.106 PM_{10}-CO: 0.598	**PM Increment:** IQR: 40.4 µg/m^3 **RR Estimate:** Single **Pollutant:** 1.07 (1.04, 1.11) lag 1 Two pollutant models: +SO_2: 1.05 (1.01, 1.09) lag 1 +NO_2: 1.03 (0.99, 1.07) lag 1 +O_3: 1.06 (1.03, 1.10) lag 1 +CO: 1.04 (1.00, 1.08) lag 1 Three pollutant models: +O_3 + CO: 1.02 (0.98, 1.06), lag 1 Four pollutant models: +O_3 + CO +SO_2: 1.02 (0.98, 1.06), lag 1 Five pollutant model: 1.016 (0.975, 1.059) lag 1 **Notes:** Investigated the association between outdoor air pollution and asthma attacks in children <15 yrs.
Reference: Lee et al. (2006) **Period of Study:** 1/1997-12/2002 **Location:** Hong Kong, China	**Hospital Admission** **Outcome:** Asthma (493) **Age Groups:** <18 years **Study Design**: Time series **N:** 26,663 asthma admissions for asthma and 5821 admissions for influenza **Statistical Analyses:** Poisson regression, GAM **Covariates:** Temperature, atmospheric pressure, relative humidity **Season:** All **Dose-response Investigated?**No **Statistical Package:** SAS 8.02 **Lags Considered:** 0-5 **Notes:** Controls were admissions for influenza ICD9 487	**Pollutant:** PM_{10} **Averaging Time:** 24-hs **Mean (SD):** 56.1 (24.2) **Percentiles: 25th:** 37.3 50th(Median): 51.1 75th: 70.7 **Monitoring Stations:** 10 **Notes: Copollutant (correlation):** PM_{10}-$PM_{2.5}$: 0.90 PM_{10}-SO_2: 0.39 PM_{10}-NO_2: 0.80 PM_{10}-O_3: 0.60	**PM Increment:** IQr = 33.4 **Percent Increase:** Single pollutant model: 4.97 [2.96, 7.03], lag 0 5.71 [3.78, 7.68], lag 1 6.40 [4.51, 8.32], lag 2 7.25 [5.38, 9.16], lag 3 7.45 [5.58, 9.35], lag 4 5.96 [4.11, 7.85], lag 5 Multipollutant model (SO_2, CO, NO_2, O_3) 3.67 [1.52,5.86] lag4
Reference: Linares et al. (2006) **Period of Study:** Jan 1995-Dec 2000 **Location:** Madrid, Spain	**Outcome:** Respiratory system diseases 460-519, bronchitis 460-496, pneumonia 480-487 **Age Groups:** <10 years **Study Design:** Time series **N:** ~15,000 admissions, 2192 days **Statistical Analyses:** Poisson regression, dummy variables to adjust for season and weather **Covariates:** Temperature, difference in barometric pressure, relative humidity, pollen counts, influenza epidemics **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** S-Plus 2000 **Lags Considered:** 0-13	**Pollutant:** PM_{10} **Averaging Time:** 24-hs **Mean (SD):** 33.4 µg/m^3, (13.7) **Range (Min, Max):** 6, 109 µg/m^3 **Monitoring Stations:** 24 **Notes: Copollutant (correlation):** PM_{10}-SO_2: 0.532 PM_{10}-O_3: -0.289 PM_{10}-NO_X: 0.721 PM_{10}-NO_2: 0.711	**PM Increment:** 10 µg/m^3 **RR Estimate** Bronchitis 1.09 [1.01, 1.16] lag 2 **AR% Estimate** Bronchitis 7.9 [CI NR] lag2 **Notes:** Only statistically significant relative and attributable risks were presented by the authors. The authors conducted multivariate modeling using a linear term to represent PM_{10}. They also report an apparent estimated PM_{10} effect threshold of 60 µg/m^3, based on examination of a scatterplot of respiratory emergency hospital admissions and PM_{10} levels.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Martins et al. (2002) **Period of Study:** May 1996-Sep 1998 **Location:** Sao Paulo, Brazil	Hospital Admission/ED: ER visits **Outcome (ICD10):** Chronic lower respiratory disease (CLRD) (40-47); includes chronic bronchitis, emphysema, other COPDs, asthma, bronchiectasia **Age Groups:** >64 years **Study Design:** Time series **N:** 712 for CLRD; 1 hospital **Statistical Analyses:** Poisson regression GAM, LOESS smoothers, no mention of stringent criteria **Covariates:** Day of week, time minimum temperature, relative humidity **Season:** All **Statistical Package:** S-Plus **Lags Considered:** 2-7 3 d ma	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** 60.0 µg/m3, (26.3) **Range (Min, Max):** 22.8. 186.5 µg/m3 **Unit (i.e. µg/m3):** µg/m3 **PM Component:** None **Monitoring Stations:** 12 **Notes: Copollutant (correlation):** PM_{10}-CO: 0.73 PM_{10}- NO_2: 0.83 PM_{10}-SO_2: 0.72 PM_{10}-O_3: 0.35	**PM Increment:** 1 µg/m3 Regression Coefficients (SE): 0.0024 (0.0023), 6 d ma **Notes:** % Increase (SD) for ER visits per 2435 µg/m3 (IQR) PM_{10} (lag 6 d ma) presented graphically in text.
Reference: Medina-Ramon et al (2006) **Period of Study:** 1986-99 **Location:** 36 US Cities	**Outcome:** 490-496, except 493 (COPD), 480-487 (Pneumonia) **Age Groups:** 65 + (US Medicare beneficiaries) **Study Design:** Case crossover **N:** 578,006 COPD admissions; 1,384,813 Pneumonia admissions **Statistical Analyses:** Conditional logistic regression, Meta-analysis using REML random effects models **Covariates:** Mean and variance of daily summer apparent temperature index, % 65+ living in poverty,% households with central air-conditioning mortality rate for emphysema among 65+(surrogate for smoking history), % PM_{10} from traffic **Season:** Warm(May –Sep)and Cold(Oct-Apr) **Dose-response Investigated?** No **Statistical Package:** SAS; STATA **Lags Considered:** 0-1 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h avg **Mean (SD):** 30.4 µg/m3 (5.1) **Monitoring Stations:** at least one per city **Notes:** PM_{10} measurements made every 2, 3 or 6 days depending on the city. **Copollutant:** none considered	**PM Increment:** 10 µg/m3 **% change [Lower CI, Upper CI]; lag:** COPD warm season 0.81(0.22,1.41) at lag 0 1.47(0.93,2.01) at lag 1 COPD cold season 0.06(-0.40,0.51) at lag 0 0.10(-0.30,0.49) at lag 1 Pneumonia warm season 0.84 (0.50,1.19) at lag 0 0.79 (0.45,1.13) at lag 1 Pneumonia cold season 0.30 (0.07,0.53) at lag 0 0.14 (-0.17,0.45) at lag 1
Reference: Meng et al., (2007) **Period of Study:** Nov 2000–Sep 2001 **Location:** Los Angeles and San Diego counties, California	**Outcome:** Poorly controlled asthma defined as (1) daily or weekly asthma symptoms or (2) at least 1 ED visit or hospitalization due to asthma over the past 12 months **Age Groups:** >18 yrs **Study Design:** Time series **N:** 1609 asthma patients **Statistical Analyses:** Logistic regression **Covariates:** Age, sex, race/ethnicity, poverty level, insurance status, smoking behavior, employment, asthma medication use, and county **Season:** NR **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (25-75th percentile):** NR **Monitoring Stations:** NR **Copollutant (correlation):** $PM_{2.5}$: r = 0.84 O_3: r = -0.72 NO_2: r = 0.83 CO: r = 0.42 Other variables: Traffic: r = 0.14	**PM Increment:** 10 µg/m3 **OR Estimate [CI]:** All Adults: 1.08 [0.82,1.43] 18-64 yrs: 1.14 [0.84,1.55] 65+: 0.84 [0.41,1.73] Men: 0.72 [0.42,1.21] Women: 1.38 [0.99,1.94] Exposure above 44.01 µg/m3 (annual concentration) All Adults: 1.56 [0.96,2.52] 18-64 yrs: 1.40 [0.81,2.41] 65+: 2.23 [0.60,8.27] Men: 0.80 [0.27,2.41] Women: 2.06 [1.17,3.61] **Notes:** This study focused more on the relation between poorly controlled asthma and traffic density.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Middleton et al. (2008) **Period of Study:** 1995–1998, 2000 - 2004 **Location:** Nicosia, Cyprus	**Outcome: R**espiratory disease (ICD-10: J00–J99). **Age Groups:** All, also stratified by age (<15 vs. >15 years) **Study Design:** Time series **Statistical Analyses:** Generalized additive Poisson models **Covariates:** Seasonality, day of the week, long- and short-term trend, temperature, relative humidity **Dose-response Investigated:** No **Statistical Package:** STATA SE 9.0, R 2.2.0 **Lags Considered:** Lag 0 -2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD; median; 5% - 95%; range):** Cold: 57.6 (52.5; 50.8; 20.0–103.0; 5.0–1370.6) Warm: 53.4 (50.5; 30.7; 32.0–77.6; 18.4–933.5) **Monitoring Stations:** 2 **Copollutant:** NR	**PM Increment:** 10 µg/m^3, and across quartiles of increasing levels of PM_{10} **Percentage increase estimate [CI]: All age/sex groups (Lag 0):** Respiratory (cold months): -0.33 (-1.47, 0.82); Respiratory (warm months): 1.42 (-0.42, 3.31); CVD + RD: 0.56 (-0.21, 1.34) **Nicosia residents (Lag 0):** Respiratory (all): 0.25 (-0.84, 1.36); Respiratory (cold months): -0.22 (-1.45, 1.02); Respiratory (warm months): 1.80 (-0.22, 3.85); CVD + RD: 0.38 (-0.47, 1.23) **Males (Lag 0):** Cardiovascular: 1.27 (-0.15, 2.72); Respiratory (all): -0.06 (-1.37, 1.26); Respiratory (cold months): -0.16 (-1.76, 1.46); Respiratory (warm months): 1.10 (-1.47, 3.74); CVD + RD: 0.63 (-0.34, 1.62) **Females (Lag 0):** Respiratory (all): 0.39 (-1.21, 2.02); Respiratory (cold months): -0.26 (-2.18, 1.70); Respiratory (warm months): 3.27 (-0.00, 6.65); CVD + RD: 0.59 (-0.68, 1.87) **Aged <15 years (Lag 0):** Respiratory (all): -0.35 (-1.77, 1.08); Respiratory (cold months): -0.31 (-2.02, 1.42); Respiratory (warm months): -0.59 (-3.53, 2.45) **Aged >15 years (Lag 0):** Respiratory (all): 0.59 (-0.87, 2.07); Respiratory (cold months): 0.02 (-1.76, 1.83); Respiratory (warm months): 3.89 (1.05, 6.80)
Reference: Oftedal et al. (2003) **Period of Study:** 1995-2000 **Location:** Drammen, Norway	**Hospital** Admissions **Outcome:** All Respiratory (460-517) **Age Groups:** All **Study Design:** Time-series **N:** ~4,458 admissions **Statistical Analyses:** Poisson regression, GAM w/ stringent convergence criteria **Covariates:** Temperature, humidity, influenza epidemics, summer and Christmas vacation **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** S-Plus **Lags Considered:** 2-3	**Pollutant:** PM_{10} **Averaging Time:** 24-hs **Mean (SD):** 16.8 µg/m^3, (10.2) 1994-1997 16.5 µg/m^3, (10.3) 1998-2000 16.6 , µg/m^3 (10.2) total period **PM Component:** Benzene, formaldehyde, toluene **Monitoring Stations:** NR **Notes: Copollutant (correlation):** Correlation between pollutants ranged from -0.47–0.78 with the exception of the VOCs studied **Notes:** Benzene, formaldehyde and toluene also evaluated	**PM Increment:** IQr = 11.04 **RR Estimate** 1.035 [0.990, 1.083] 1994-1997 0.992 [0.948, 1.037] 1998-2000 1.021 [0.990, 1.053] 1994-2000 2 Pollutant Model PM_{10} w/ benzene: 1.01 (0.978, 1.043)
Reference: Peel et al. (2005) **Period of Study:** Jan 1993-Aug 2000 **Location:** Atlanta, Georgia	ED visits **Outcome:** Asthma (493, 786.09); COPD (491, 492, 496); URI (460-466, 477); Pneumonia (480-486) **Age Groups:** All ages. Secondary analyses conducted by age group: 0-1, 2-18, >18 **Study Design:** Time series **N:** 31 hospitals **Statistical Analyses:** Poisson GEE for URI, asthma and all RD; Poisson GLM for pneumonia and COPD) **Covariates:** Avg temperature and dew point, pollen counts **Season:** All (secondary analyses of warm season) **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.3, S-Plus 2000 **Lags Considered:** 0-7 d , 3 d ma, 0-13 d unconstrained distributed lag	**Pollutant:** PM_{10} **Averaging Time:** 24 h avg **Mean (SD):** 27.9 (12.3) µg/m^3 **Percentiles: 10th:** 13.2 **90th:** 44.7 **Monitoring Stations:** "Several" **Copollutant (correlation):** 8 h O_3: r = 0.59 1 h NO_2: r = 0.49 1 h CO: r = 0.47 1 h SO_2: r = 0.20 24-h $PM_{2.5}$: 0.84 24 h $PM_{10-2.5}$: r = 0.59 24 h UF: r = -0.13 Components: r ranged from 0.42-0.74	**PM Increment:** PM_{10}: 10 µg/m^3 RR Estimate [Lower CI, Upper CI] All Respiratory Outcomes: 1.013 (1.004–1.021), 3 d ma URI: 1.014 (1.004–1.025) , 3 d ma 1.073 (1.048–1.099) , 14-day dist. lag Asthma: 1.009 (0.996–1.022), 3 d ma 1.099 (1.065–1.135), 14-day dist. lag: Pediatric Asthma 2–18yrs): 1.016 (0.998 –1.034) Pneumonia: 1.011 (0.996–1.027) , 3 d ma 1.087 (1.044–1.132), 14-day dist. lag COPD: 1.018 (0.994–1.043), 3 d ma 1.092 (1.023–1.165), 14-day dist. lag Notes: RRs obtained using AQS 1993-2000, AQS 1998-2000 and ARIES data compared. Infant (0-1 y) and pediatric (2-18 y) asthma was associated more strongly with PM_{10}, PM2.5 and OC than adult asthma.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Sinclair and Tolsma (2004) **Period of Study:** 25 Months **Location:** Atlanta, Georgia	**Outpatient Visits** **Outcome:** Asthma (493); URI (460, 461, 462, 463, 464, 465, 466, 477); LRI (466.1, 480, 481, 482, 483, 484, 485, 486). **Age Groups:** < = 18 y, 18+ y (asthma); All ages (URI//LRI) **Study Design:** Times series **N:** 25 months; 260,000 to 275,000 health plan members (August 1998–August 2000) **Statistical Analyses:** Poisson GLM **Covariates:** Season, Day of week, Federal Holidays, Study Months **Season:** NR **Dose-response Investigated?:** No **Statistical Package:** SAS **Lags Considered:** Three 3 d moving averages (0-2, 2-5, 6-8)	**Pollutant:** PM_{10} **Averaging Time:** 24 h avg **Mean (SD):** PM_{10} mass– 29.03 µg/m^3 (11.61) **Monitoring Stations:** 1 **Notes: Copollutant:** NR	**PM Increment:** 11.61 (1 SD) **RR Estimate [Lower CI, Upper CI]; lag:** Child Asthma: 1.049 (S), lag 3-5 d LRI: 1.074 (S), 3-5 d lag **Notes:** Numerical findings for significant results only presented in manuscript. Results for all lags presented graphically for each outcome (asthma, URI, and LRI).
Reference: Slaughter et al. (2005) **Period of Study:** January 1995 through June 2001 **Location:** Spokane, WA	Hospital Admissions and ED visits **Outcome:** All respiratory (460-519); Asthma (493); COPD (491,492, 494,496); Pneumonia (480-487); Acute URI not including colds and sinusitis (464, 466, 490) **Age Groups:** All, 15+ years for COPD **Study Design:** Time series **N:** 2373 visit records **Statistical Analyses:** Poisson regression, GLM with natural splines. For comparison also used GAM with smoothing splines and default convergence criteria. **Covariates:** Season, temperature, relative humidity, day of week **Season:** All **Dose-response Investigated?:** No **Statistical Package:** SAS, SPLUS **Lags Considered:** 1 -3 d	**Pollutant:** PM_{10} **Averaging Time:** 24 h avg **Range (90% of concentrations):** 7.9-41.9 µg/m^3 **Monitoring Stations:** 1 **Notes: Copollutant (correlation):** PM_{10} PM_1 r = 0.50 $PM_{2.5}$ r = 0.62 $PM_{10-2.5}$ r = 0.94 CO r = 0.32 Temperature r = 0.11	**PM Increment:** 25 µg/m^3 **RR Estimate [Lower CI, Upper CI]; lag:** **ER visits --** PM_{10} All Respiratory Lag 1: 1.01 [0.99, 1.04] ; Lag 2: 1.01 [0.98, 1.03] ; Lag 3: 1.02 [0.99, 1.04] Acute Asthma Lag 1: 1.03 [0.98, 1.07] ; Lag 2: 1.01 [0.96, 1.05] ; Lag 3: 1.00 [0.95, 1.04] COPD (adult) Lag 1: 1.00 [0.93, 1.07] ; Lag 2: 0.99 [0.92, 1.06] ; Lag 3: 1.02 [0.95, 1.08] **Hospital Admissions --** PM_{10} All Respiratory Lag 1: 0.99 [0.95, 1.02] ; Lag 2: 0.99 [0.96, 1.02] ; Lag 3: 1.00 [0.97, 1.03] Asthma Lag 1: 1.03 [0.95, 1.12] ; Lag 2: 1.01 [0.94, 1.10] ; Lag 3: 1.00 [0.92, 1.09] COPD (adult) Lag 1: 0.98 [0.90, 1.07] ; Lag 2: 1.03 [0.96, 1.11] ; Lag 3: 1.02 [0.94, 1.09]
Reference: Sun et al. (2006) **Period of Study:** January 1, 2004 to December 31, 2004 **Location:** Taichung, Taiwan (Central Taiwan)	ED visits **Outcome:** Asthma (493.xx) **Age Groups:** <55, <16, 16-55 yrs **Study Design:** Cross-sectional **N:** NR; All diagnoses for all patients at 4 medical centers **Statistical Analyses:** Pearson's correlations, multiple correlation coefficients from regression analyses. **Covariates:** Only copollutants considered **Dose-response Investigated?** No **Statistical Package:** SPSS **Lags Considered:** None	**Pollutant:** PM_{10} **Averaging Time:** Monthly avg for 2004 **Mean (SD):** ~ 60.3 µg/m^3 (NR) (estimated from figure)* **Range (Min, Max):** (~35, 80) **Monitoring Stations:** 11 **Copollutant:** NR	Children ED Visits r = 0.626 P = 0.015 Adult ED Visits r = 0.384 P = 0.109

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Tolbert et al. (2007) **Period of Study:** 1993 - 2004 **Location:** Atlanta Metropolitan area, Georgia	**Outcome (ICD-9):** Combined RD group, including: Asthma (493, 786.07, 786.09), COPD (491, 492, 496), URI (460–465, 460.0, 477), pneumonia (480–486), and bronchiolitis (466.1, 466.11, and 466.19)) **Age Groups:** All **Study Design:** Time series **N:** 10,234,490 ER visits (1,072,429 visits in the RD group) **Statistical Analyses:** Poisson generalized linear models **Covariates:** Long-term temporal trends, season (for RD outcome), temperature, dew point, days of week, federal holidays, hospital entry and exit **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 9.1 **Lags Considered:** 3-day moving avg(lag 0 -2)	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (median; IQR, range, 10th–90th percentiles):** 26.6 (24.8; 17.5–33.8; 0.5–98.4; 12.3–42.8) **Monitoring Stations:** NR **Copollutant (correlation):** O_3: r = 0.59 NO_2: r = 0.53 CO: r = 0.51 SO_2: r = 0.21 Coarse PM: r = 0.67 $PM_{2.5}$: r = 0.84 $PM_{2.5}$ SO4: r = 0.69 $PM_{2.5}$ EC: r = 0.61 $PM_{2.5}$ OC: r = 0.65 $PM_{2.5}$ TC: r = 0.67 $PM_{2.5}$ water-sol metals: r = 0.73 OHC: r = 0.53	**PM Increment:** 16.30 µg/m^3 (IQR) **Risk ratio [95% CI]:** RD: 1.015 (1.006–1.024) **Notes:** Results of selected multi-pollutant models for respiratory disease are presented in Figure 2. Figure 2: PM_{10} adjusted for CO, O_3, NO_2, or NO_2/O_3 (non-winter months only) Summary of results: PM_{10} remained predictive of RD in non-winter months after adjustment for pollutants.
Reference: Tsai et al. (2006) **Period of Study:** 1996 to 2003 **Location:** Kaohsiung City, Taiwan	**Outcome:** Asthma (493) **Age Groups:** All (universal health care covers >96% of the population) **Study Design:** Case crossover **N:** 17,682 admissions; 63 hospitals **Statistical Analyses:** Conditional Logistic Regression **Covariates:** Temperature, humidity **Season:** Warm and cool seasons **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2 d cumulative	**Pollutant:** PM_{10} **Averaging Time:** 24 h avg **Mean (SD):** 76.62 µg/m^3 (NR) **Percentiles:** 25th: 41.73 50th(Median): 74.40 75th: 104.01 **Range (Min, Max):** (16.70, 232.00) **Monitoring Stations:** 6 **Copollutant:** NR	**PM Increment:** 62.28 µg/m^3 **OR Estimate [Lower CI, Upper CI]; lag:** **Single-pollutant model, 0-2 d cumulative lag** ≥ 25°C: 1.302 [1.155, 1.467] ; <25°C: 1.556 [1.398, 1.371] **Two-pollutant models, 0-2 d cumulative lag** PM_{10} w/ SO_2 ≥ 25°C: 1.305 [1.156, 1.473] ; <25°C: 1.540 [1.374, 1.727] PM_{10} w/ O_3 ≥ 25°C: 0.985 [0.842, 1.152] ; <25°C: 1.581 [1.402, 1.783] PM_{10} w/ NO_2 ≥ 25°C: 1.237 [1.052, 1.455] ; <25°C: 1.009 [0.875, 1.163] PM_{10} w/ CO ≥ 25°C: 1.156 [1.012, 1.320] ; <25°C: 1.300 [1.134, 1.490]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ulirsch et al. (2007) **Period of Study:** 11/1994 to 3/2000 **Location:** Pocatello, Idaho; Chubbuck, Idaho	**Outcome:** Respiratory Disease (460-499, 509-519); Reactive Airway Disease (786.09) **Age Groups:** All age groups **Study Design:** Time series **N:** 39,347 visits (TS1); 29,513 visits (TS2) **Statistical Analyses:** Poisson regression, GLM. Sensitivity Analyses **Covariates:** Time, Temperature, Relative Humidity Influenza **Season:** Warm/Cool **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0 to 4 day lags **Notes:** Time series (TS) 1 includes HA, ED and urgent care visits. TS 2 includes family practice data available after 1997	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (SD):** TS1: 24.2 µg/m³ (NR) 10th: 10.5 90th: 40.7 TS2: 23.2 10th: 10.0 90th: 37.4 **Range (Min, Max):** TS1: (3.0, 183.0) TS2: (3.0, 183.0) **Monitoring Stations:** 4 **Notes: Copollutant (correlation):** PM_{10} w/ NO_2: r = 0.47. PM_{10} with other copollutants weakly correlated.	**PM Increment:** Single Pollutant Models, TS1: 24.4 µg/m³ Single Pollutant Models: TS2: 23.2 µg/m³ Multipollutant Models: TS1/TS2: 50 µg/m³ **Mean Percentage Change, lag 0** TS 1: Single Pollutant All-age (all year): 4.0 [1.4,6.7] ; 18-64: 3.4 [0.2, 6.7] ; 0-17: 4.3 [-0.1, 8.9] ; 65+: 5.6 [-1.4, 13.1] ; 0-17/65+: 5.5 [1.4, 9.6] All age (Cool season): 4.3 [1.3, 7.5] All age (Warm season): 6.7 [-0.8, 14.8] TS2: Single Pollutant All-age: 3.3 [0.3, 6.3] ; 18-64: 3.3 [-0.4, 7.0] ; 0-17: 5.0 [0.1, 10.1] ; 65+: 6.9 [-0.4, 14.7] Multipollutant (PM_{10} + SO_2) All-age (all year): TS1 10.8; TS2 17.5 ; 18-64: TS1 8.0; TS2 9.1 ; 0-17: TS1 10.8; TS2 32.7 65+: TS1 8.7; TS2 31.3 0-17/65+: TS1 14.2; TS2 25.3 All age (Cool season) TS1 11.9 Multipollutant (PM_{10} + NO_2) All-age (all year) TS1: TS2 16.3 18-64: TS1 9.3; TS2 17.3 0-17: TS1 4.6; TS2 18.7 65+: TS1 12.4; TS2 32.7 0-17/65+: TS1 9.5; 32.7 All age (Cool season): TS1 11.1; TS2 16.8 **Notes:** Results from multipollutant model with PM_{10}, SO_2 and NO_2 also available.
Reference: Ulirsch et al. (2007) **Period of Study:** November 1994–March 2000 **Location:** Pocatello, Idaho and Chubbuck, Idaho	**Outcome (ICD-9):** Respiratory disease (460–519 and 786.09 [reactive airway disease]; excluding 500–500.8 for lung diseases due to external causes), and CVD (390 - 429). **Age Groups:** All, 0–17 (RD only), 65 + , 18–64 (RD only) **Study Design:** Time series **N:** 39,347 admissions/visits **Statistical Analyses:** Log-linear generalized linear models **Covariates:** Time, temperature, relative humidity, influenza, day of the week **Season:** All, and separate analyses were performed for the all-age group for cool months (October–March) vs. warm months (April–September). **Dose-response Investigated:** No **Statistical Package:** S-plus version 6.1 **Lags Considered:** 0- to 4-day lags, and mean of days 0 -4	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (range; 10th - 90th percentiles):** 24.2 (3.0–183.0; 10.5–40.7) **Monitoring Stations:** 4 **Copollutant (correlation):** NO_2: r = 0.47 **Other variables:** Correlation for PM_{10} between monitors: r = 0.42–0.87	**PM Increment:** 50 µg/m³ , and 24.3 µg/m³ (mean increase in PM_{10}) **Mean percent of change (% change in the mean number of daily admissions and visits) [95% CI]:** **For 24.3** µg/m³ **increase in** PM_{10}**:** All-age respiratory disease (all year): 4.0 [1.4, 6.7] All-age RD/CVD: 3.7 [1.3, 6.3] ; 18-64 years RD: 3.4 [0.2, 6.7] ; 0-17 years RD: 4.3 [-0.1, 8.9] ; 65+ years RD: 5.6 [-1.4, 13.1] ; 65+ years RD/CVD: 2.9 [-2.9, 8.7] ; 0-17/65+years RD: 5.5 [1.4, 9.6] ; All-age RD (cool season): 4.3 [1.3, 7.5] ; All-age RD (warm season): 6.7 [-0.8, 14.8] ; All-age CVD (Lag 0): -0.02 [-5.9, 6.3] ; All-age CVD (Lag 1): 1.9 [-4.1, 8.4] ; All-age CVD (Lag 2): -3.1 [-9.1, 3.4] ; All-age CVD (Lag 3): 0.5 [-5.6, 6.9] ; All-age CVD (Lag 4): -1.7 [-4.3, 0.9] ; Lag 0–4 days: -0.5 [-8.0, 7.6] **For 50** µg/m³ **increase in** PM_{10} (single pollutant models, CIs not given): All-age respiratory disease: 8.4 ; All-age RD/CVD: 7.9 ; 18-64 years RD: 7.2 ; 0-17 years RD: 9.1 ; 65+ years RD: 12.0 ; 65+ years RD/CVD: 6.1 ; 0-17/65+years RD: 11.6 ; All-age RD (cool season): 9.1 ; All-age RD (warm season): 14.3 ; All-age CVD (Lag 0): -0.05 ; All-age CVD (Lag 1): 4.0 ; All-age CVD (Lag 2): -6.2 ; All-age CVD (Lag 3): 1.0 ; All-age CVD (Lag 4): -3.6 ; All-age CVD (Lag 0 -4): -1.1 **For 50** µg/m³ **increase in** PM_{10} (multi-pollutant models, CIs not given): Adjusted for SO_2 (for respiratory disease): All-age (all year): 10.8 ; 18-64: 8.0 ; 0-17: 10.8 ; 65+: 8.7 ; 0-17/65+: 14.2 ; All-age (cool season): 11.9 ; Adjusted for NO_2 (for respiratory disease): All-age (all year): 10.5 ; 18-64: 9.3 ; 0-17: 4.6 ; 65+: 12.4; 0-17/65+: 9.5 ; All-age (cool season): 11.1 Adjusted for SO_2 and NO_2 (for respiratory disease): All-age (all year): 11.3 ; 18-64: 9.0 ; 0-17: 6.2 ; 65+: 12.0 ; 0-17/65+: 10.3All-age (cool season): 11.0 **Notes:** Included urgent care visits as well as emergency department visits and hospital admissions.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Vigotti (Vigotti et al., 2007) **Period of Study:** 1/2000–12/2000 **Location:** Pisa, Italy	**ED Visits** **Outcome:** Asthmatic attack (493), dry cough (468), acute bronchitis (466) **Age Groups:** <10 y; 65+ **Study Design:** Time series **N:** 966 Emergency room visits **Statistical Analyses:** Poisson regression, GAM, LOESS smoothers, stringent criteria **Covariates:** temperature, humidity, relative humidity, day of study, rainfall, influenza, day of-the-wk, holidays, time trend **Season:** All year **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-5 d	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 35.4 (15.8) µg/m³ **Percentiles:** 25th: NR 50th(Median): 31.6 75th: NR **Range (Min, Max):** (9.5, 100.1) **Monitoring Stations:** 2 **Copollutant (correlation):** PM_{10}: NO_2; r = 0.58 CO; r = 0.70	**PM Increment:** 10 µg/m³ **RR Estimate [Lower CI, Upper CI]; lag:** <10 y: 10%[2.3, 18.2]; lag 1 65+: 8.5% [1.5, 16.1]; lag 2
Reference: Yang et al. (2007) **Period of Study:** 1996-2003 **Location:** Taipei, Taiwan	**Hospital Admission/ED:** **Outcome:** Asthma (493) **Age Groups:** All ages **Study Design:** Case-crossover **N:** 25,602 asthma hospital admissions **Statistical Analyses:** NR **Covariates:** Temperature, humidity, day of-the-wk, seasonality, long term trends **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (SD):** 48.99 µg/m³ **Percentiles: 25th:** 32.64 **50th(Median):** 44.13 **75th:** 59.05 **Range (Min, Max):** (14.44, 234.91) **PM Component:** NR **Monitoring Stations:** 6 Stations **Notes: Copollutant:** NR	**PM Increment:** 26.41 µg/m³ **OR Estimate [Lower CI, Upper CI]; lag:** **Asthma** Single-Pollutant Model: Temperature >25° C: 1.046[0.971, 1.128] Temperature <25° C: 1.048[1.011, 1.251] Two-Pollutant Model: Adjusted for SO_2: >25° C-1.006[0.920, 1.099] ; <25° C–1.088[1.040, 1.138] Adjusted for NO_2: >25° C–0.800[0.717, 0.892] ; <25° C–0.982[0.937, 1.029] Adjusted for CO: >25° C–0.920[0.844, 1.002] ; <25° C–1.029[0.984, 1.076] Adjusted for O_3: >25° C–1.038[0.950, 1.134] ; <25° C–1.042[1.004, 1.081] **AR Estimate [Lower CI, Upper CI]; lag:** NR **Notes: Other Outcomes Assessed?** NR **Other Exposures Assessed?** SO_2, NO_2, CO, O_3
Reference: Yang et al. (2007) **Period of Study:** 1996-2003 **Location:** Taipei, Taiwan	Hospital Admission **Outcome:** COPD (490-192), (494), (496) **Age Groups:** All ages **Study Design:** Case-crossover **N:** 46,491COPD admissions, 47 hospitals **Statistical Analyses:** Conditional logistic regression **Covariates:** Weather, day of-the-wk, seasonality, long term trends **Season: Warm/Cool** **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2 cumulative	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 48.99 µg/m³ **25th:** 32.64 **50th(Median):** 44.13 **75th:** 59.05 **Range (Min, Max):** (14.44, 48.99) **Monitoring Stations:** 6 Stations **Notes: Copollutant:** NR	**PM Increment:** 26.41 µg/m³ **OR Estimate [Lower CI, Upper CI];** Single-Pollutant Model (0-2 d cum lag): Temperature >20° C: 1.133[1.098, 1.168] Temperature <20° C: 1.035[0.994, 1.077] Two-Pollutant Model: PM_{10} w/ SO_2: >20° C–1.180[1.139, 1.223] ; <20° C–1.004[0.954, 1.057] PM_{10} w/ NO_2: >20° C–1.013[0.973, 1.055] ; <20° C–1.074[1.022, 1.129] PM_{10} w/ CO: >20° C–1.061[1.023, 1.100] ; <20° C–1.067[1.016, 1.120] PM_{10} w/ O_3: >20° C–1.097[1.062, 1.133] ; <20° C–1.036[0.996, 1.079]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Yang et al. (2007) **Period of Study:** 1996-2003 **Location:** Taipei, Taiwan	Hospital Admission **Outcome:** Asthma (493) **Age Groups:** All ages **Study Design:** Case-crossover **N:** 25,602 admissions, 47 hospitals **Statistical Analyses:** Conditional logistic regression **Covariates:** Weather, day of-the-wk, seasonality, long term trends **Season: Warm/Cool** **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2 cumulative	**Pollutant:** 10 µg/m^3 **Averaging Time:** 24 h **Mean (SD):** 48.99 µg/m^3 25th: 32.64 50th(Median): 44.13 75th: 59.05 **Range (Min, Max):** (14.44, 48.99) **Monitoring Stations:** 6 Stations **Notes: Copollutant:** NR	**PM Increment:** 26.41 µg/m^3 **OR Estimate [Lower CI, Upper CI];** Single-Pollutant Model (0-2 d cum lag): Temperature >20° C: 1.046[0.971, 1.128] Temperature <20° C: 1.048[1.011, 1.087] Two-Pollutant Model: PM_{10} w/ SO_2: >20° C–1.006[0.920, 1.099] ; <20° C–1.088[1.040, 1.138] PM_{10} w/ NO_2: >20° C–0.800[0.717, 0.892] ; <20° C–0.982[0.937, 1.029] PM_{10} w/ CO: >20° C–0.920[0.844, 1.002] ; <20° C–1.029[0.984, 1.076] PM_{10} w/ O_3: >20° C–1.038[0.95, 1.134] ; <20° C–1.042[1.004, 1.081]
Reference: Xirasagar et al. (2006) **Period of Study:** 1998–2001 **Location:** Taiwan	**Hospital Admission/ED:** **Outcome:** Asthma or Asthmatic Bronchitis (493) **Age Groups:** Less than 2 years old, 2~5 years old, 6~14 years old **Study Design: N:** N = 27, 275 pediatric hospitalizations **Statistical Analyses:** ARIMA Modeling Spearman's Correlations **Covariates: S**eason, ambient temp., rel. humidity, atmospheric pressure, rainfall, h of sunshine **Season:** Spring: February to April; summer: May to July; Autumn: August to October; Winter: November to January **Dose-response Investigated?** No **Statistical Package:** EViews 4 **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** Monthly Means **Mean (SD):** 24.4 µg/m^3 (NR) **Percentiles:** NR **Range (Min, Max):** NR **PM Component:** NR **Monitoring Stations:** 44 air quality monitoring banks. 23 weather observatories **Notes: Copollutant (correlation):** Less than 2 years old: r = 0.315 2~5 years old: r = 0.589 6~14 years old: r = 0.493	**PM Increment:** NR **RR Estimate [Lower CI, Upper CI]; lag:** NR **AR Estimate [Lower CI, Upper CI]; lag:** NR **Notes:** Plot of monthly asthma admission rates per 100,000 population by age group Plot of mean monthly concentration trends of criteria air pollutants Mean monthly trends of climatic factors **Other Outcomes Assessed?** NR **Other Exposures Assessed?** Seasonality
Reference: Barnett et al. (2005) **Period of Study:** 1998-2001 **Location:** 5 Australian cities (Brisbane, Canberra, Melbourne, Perth, and Sydney) and 2 New Zealand cities (Auckland, Christchurch)	**Outcome (ICD: NR):** All respiratory admissions (including asthma, pneumonia, and acute bronchitis) **Age Groups:** Children aged <1 year, 1-4 years, and 5-14 years **Study Design:** Matched case-crossover **N:** ~2.4 million children <15 years old **Statistical Analyses:** Random effects meta-analysis **Covariates:** Temperature, current minus previous day's temperature, relative humidity, pressure, extremes of hot and cold, day of the week, public holiday, and day after public holiday **Season:** Warm (Nov-Apr) and Cool (May-Oct) **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24-hs **Mean (min-max):** Auckland (A): 18.8 (3.2-101.4) Brisbane (B): 16.5 (3.8-50.2) Canberra (Ca): NR Christchurch (Ch): 20.6 (1.3-156.3) Melbourne (M): 16.6 (3.1-71.1) Perth (P): 16.5 (4.4-68.9) Sydney (S): 16.6 (3.7-104.7) **Monitoring Stations:** 1-11 per city **Copollutant:** NR	**PM Increment:** 7.5 µg/m^3 (IQR) **Percent Increase Estimate [CI]:** Respiratory Admissions: Single Pollutant Model 1-4 yrs (B,Ch,M,P,S): 1.7 [0.5,2.9] 5-14 yrs (B,Ch,M,P,S): 1.9 [0.1,3.8] Matched Multipollutant Model 1-4 yrs with 24-h $PM_{2.5}$ (B,M,P,S): 5.5 [-0.2,11.5] 1-4 yrs with 1-h SO_2 (B,Ch,S): 3.2 [0.3,6.1] 1-4 yrs with 1-h NO_2 (B,Ch,M,P,S): 0.0 [-2.1,2.1] 1-4 yrs with temp (B,Ch,M,P,S): 2.3 [0.6,3.9] 5-14 yrs with 24-h NO_2 (B,Ch,M,P,S): 1.2 [-1.8,4.4] 5-14 yrs with temp (B,Ch,M,P,S): 3.6 [1.4,5.8]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chen et al. (2006a) **Period of Study:** Jan 1998–Dec 2001 **Location:** Taiwan	**Outcome (ICD-9: 493):** Asthma or asthmatic bronchitis **Age Groups:** 0-4; 5-14; 15-44; 45-64; >65 years of age **Study Design:** Time series **N:** 126,671 hospitalizations **Statistical Analyses:** Auto-Regressive Integrated Moving Avg (ARIMA); Spearman rank correlations **Covariates:** Ambient temp, relative humidity, atmospheric pressure, rainfall, and h of sunshine **Season:** Feb-Apr (spring), May-Jul (summer), Aug-Oct (autumn), Nov-Jan (winter) **Dose-response Investigated?** No **Statistical Package:** SPSS **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 1 month **Mean (min-max):** 24.43 (16.08-34.48) SD: 4.79 **Monitoring Stations:** 55 **Copollutant:** NR	**PM Increment:** N/A **Correlations:** Seasonal variations in adult asthma admissions significantly correlated with PM_{10} levels (r = 0.293, p = 0.0453) **Notes:** Fig 3 shows seasonal patterns of PM_{10}
Reference: Chen et al. (2006b) **Period of Study:** Jul 1, 1997–Dec 31, 2000 **Location:** Brisbane, Australia	**Outcome (ICD-9: 460-519; ICD-10: J00-99):** Respiratory disease excluding influenza (ICD-9: 487 or ICD-10: J11-11) **Age Groups:** NR **Study Design:** Time series **N:** 42,268 cases **Statistical Analyses:** GLM, multivariate negative binomial model **Covariates:** Daily avg max and min temp, relative humidity, rainfall, wind direction, seasonality, day of the week, holidays, long-term trends, and influenza **Season:** Bushfire and non-bushfire periods **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** same day, 1-, 3-, and 5-day	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** Overall: 16.08 (4.90-60.60) Bushfire period: 18.28 (7.50-60.60) Non-bushfire period: 14.91 (4.90-58.10) **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** N/A **RR Estimate [CI]:** Overall Same Day Lag: <15: referent 15-20: 1.11 [1.05,1.15] >20: 1.16 [1.10,1.23] p-value <0.01 Overall 1-Day Lag: <15: referent 15-20: 1.10 [1.05,1.15] >20: 1.14 [1.08,1.20] p-value <0.01 **Notes:** Author also reported 3- and 5-day lag RR for overall, bushfire, and non-bushfire categories, finding similar results
Reference: Cheng et al. (2007) **Period of Study:** 1996-2004 **Location:** Kaohsiung, Taiwan	**Outcome (ICD-9: 480-486):** Pneumonia **Age Groups:** NR **Study Design:** Case-crossover **N:** 82,587 pneumonia hospital admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** Temperature and humidity on the same day **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** Cumulative lag period up to 2 previous days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 77.01 (16.7-232) Percentiles: 25%: 42.12 50%: 75.27 75%: 104.65 **Monitoring Stations:** 6 **Copollutant:** NR	**PM Increment:** 62.53 µg/m^3 (IQR) **OR Estimate [CI]:** Single Pollutant Model: Temp>25ºC: 1.21 [1.15,1.28] Temp<25ºC: 1.57 [1.50,1.65] Two-Pollutant Model: Temp>25ºC Adj. for SO_2: 1.21 [1.14,1.28] Adj. for NO_2: 1.15 [1.07,1.24] Adj. for CO: 1.10 [1.03,1.17] Adj. for O_3: 0.96 [0.89,1.03] Temp<25ºC Adj. for SO_2: 1.56 [1.48,1.65] Adj. for NO_2: 1.09 [1.02,1.16] Adj. for CO: 1.30 [1.22,1.39] Adj. for O_3: 1.56 [1.48,1.65]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chimonas and Gessner (2007) **Period of Study:** January 1, 1999–June 30, 2003 **Location:** Anchorage, Alaska	**Outcome (ICD-9):** Asthma (493.0-493.9); Lower respiratory illness-LRI (466.1, 466.0, 480-487, 490, 510-511); Inhaled quick-relief medication; Steroid medication **Age Groups:** <20 years old **Study Design:** Time series **N:** 42,667 admissions **Statistical Analyses:** GEE for multivariable modeling **Covariates:** Season, serial correlation, year, weekend, temperature, precipitation, and wind speed **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SPSS (dataset), SAS (analysis) **Lags Considered:** 1 day and 1 week	**Pollutant:** PM_{10} **Averaging Time:** 24-hs and 1 week **Mean (min-max):** Daily: 27.6 (2-421) Weekly: 25.3 (5.0-116.0) **Monitoring Stations:** NR **Copollutant:** Daily $PM_{2.5}$ ρ = 0.25 (p<0.01) Weekly $PM_{2.5}$ ρ = 0.08 (p = 0.21)	**PM Increment:** 10 µg/m³ **RR Estimate [CI]:** Same Day Outpatient Asthma: 1.006 [1.001,1.013] Outpatient LRI: 1.001 [0.987,1.015] Inpatient Asthma: 1.003 [0.922,1.091] Inpatient LRI: 1.015 [0.978,1.053] Inhaled Steroid Prescriptions: 1.006 [0.996,1.011] Quick-relief Medication: 1.018 [1.006,1.030] Weekly (median increase) Outpatient Asthma: 1.021 [1.004,1.038] Outpatient LRI: 1.013 [0.978,1.049] Inpatient Asthma: 1.023 [0.948,1.104] Inpatient LRI: 1.025 [0.981,1.072] Inhaled Steroid Prescriptions: 0.989 [0.969,1.010] Quick-relief Medication: 1.057 [1.037,1.077]
Reference: Farhat et al. (2005) **Period of Study:** Aug 1996–Aug 1997 **Location:** São Paulo, Brazil	Hospital Admissions and Emergency Room Visits **Outcome (ICD-9):** Lower respiratory tract diseases (466, 480-519) including pneumonia or bronchopneumonia (480-486), asthma (493), bronchiolitis (466) **Age Groups:** <13 yrs **Study Design:** Time series **N:** 43,635 **Statistical Analyses:** GAM, Poisson regression, Pearson correlation **Covariates:** Time, temperature, humidity, weekday **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0-7 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 62.6 (25.5-186.3) SD = 26.6 IQr = 30 N = 396 **Monitoring Stations:** 13 **Copollutant (correlation):** SO_2: r = 0.69 NO_2: r = 0.83 O_3: r = 0.35 CO: r = 0.72 (all p<0.05) **Additional correlations:** Rel humidity: r = -0.55 Min temp: r = -0.44 (both p<0.05)	**PM Increment:** 30 µg/m³ (IQR) **RR Estimate [CI]:** Lower respiratory tract disease 5-day moving avg Copollutant model: NO_2: 2.1 [-7.1,11.3]; SO_2: 16.5 [10.5,22.6]; O_3:10.1 [5.0,15.2]; CO: 14.1 [8.1,20.2]; Multipollutant model: 5.2 [-4.6,15.1] Pneumonia or bronchopneumonia 6-day moving avg Copollutant model: NO_2: 14.8 [-3.8,33.4]; SO_2: 14.8 [-0.3,30.0];O_3:16.2 [1.0,31.3] CO: 17.6 [0.4,34.8]; Multipollutant model: 5.23 [-16.2,26.6] Asthma or bronchiolitis 2-day moving avg Copollutant model: NO_2: -11.04 [-50.0,28.0]; SO_2: 15.8 [-7.8,39.3]; O_3:11.7 [-10.4, 33.9]; CO: 12.4 [-14.8,39.7]; Multipollutant model: -15.5 [-61.2,30.2]
Reference: Galán et al. (2003) **Period of Study:** 1995-1998 **Location:** Madrid, Spain	Hospital Admissions **Outcome (ICD):** Asthma (493) **Age Groups:** all ages **Study Design:** Time series **N:** 555,153 at-risk **Statistical Analyses:** GAM, autoregressive Poisson regression **Covariates:** temperature, relative humidity, pollen, year, day of the week, public holiday **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0, 1, 2, 3, and 4-day	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 32.1 (11.2-108.6) SD = 12.1 **Monitoring Stations:** 13 **Copollutant (correlation):** SO_2: r = 0.581; NO_2: r = 0.717; O_3: r = -0.188; **Other variables:** *O.europaea*: r = -0.066 *Plantago sp.*: r = -0.202 Poaceae: r = -0.132 Urticaceae: r = -0.104 Temp: r = -0.122 Humidity: r = 0.119	**PM Increment:** 10 µg/m³ **RR Estimate [CI]:** Single-pollutant Current-day lag: 1.011 (0.980-1.042) 1-day lag: 1.006 (0.976-1.037) 2-day lag: 1.008 (0.978-1.038) 3-day lag: 1.039 (1.010-1.068) 4-day lag: 1.027 (0.999-1.056) Adjustment for pollen (PM_{10} 3-day lag) *O. europaea*: 1.041 (1.011-1.071) *Plantago sp.*: 1.046 (1.017-1.076) Poaceae: 1.043 (1.015-1.073) Urticaceae: 1.038 (1.009-1.068) All four: 1.045 (1.016-1.074)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chen et al. (2005) **Period of Study:** Jun 1, 1995–Mar 31, 1999 **Location:** Vancouver area, BC	Hospital Admissions **Outcome (ICD-9):** Acute respiratory infections (460-466), upper respiratory tract infections (470-478), pneumonia and influenza (480-487), COPD and allied conditions (490-496), other respiratory diseases (500-519) **Age Groups:** >65 yrs **Study Design:** Time series **N:** 12,869 **Statistical Analyses:** GLM **Covariates:** Temp and relative humidity **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 1, 2, 3, 4, 5, 6, and 7-day avg	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 13.3 (3.8-52.2) SD = 6.1 **Monitoring Stations:** 13 **Copollutant (correlation):** $PM_{2.5}$: r = 0.83 $PM_{10-2.5}$: r = 0.83 COH: r = 0.61 CO: r = 0.46 O_3: r = -0.07 NO_2: r = 0.54 SO_2: r = 0.60 Other variables: Mean temp: r = 0.34 Rel humidity: r = -0.30	**PM Increment:** 7.9 µg/m³ (IQR) **RR Estimate [CI]:** Adj for weather conditions Overall admission 1-day avg: 1.04 [1.01,1.07] 2-day avg: 1.05 [1.02,1.09] 3-day avg: 1.05 [1.01,1.10] Adj for weather conditions and copollutants Overall admission 1-day avg: 1.03 [0.99,1.09] 2-day avg: 1.05 [1.00,1.11] 3-day avg: 1.05 [0.99,1.10] **Notes:** RR's were also provided for lags 4-7 in Table 3, yielding similar results
Reference: Erbas et al. (2005) **Period of Study:** Jan 2000–Dec 2001 **Location:** Melbourne, Australia	Hospital Admissions **Outcome (ICD-10):** Asthma (J45, J46) **Age Groups:** 1-15 yrs **Study Design:** Time series **N:** 8955 asthma cases **Statistical Analyses:** GAM, GEE (if autocorrelation was present in residuals) **Covariates:** Temp and humidity **Season:** NR **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0, 1, 2 days	**Pollutant:** PM_{10} **Averaging Time:** 1 h **Mean (SD):** Western: 2.99 (2.11) 10th centile: 13.67 90th centile: 48.00 Inner Melbourne: 4.54 (2.65) 10th centile: 15.63 90th centile: 59.73 South/Southeastern: 1.13 (1.18) 10th centile: 12.00 90th centile: 36.05 Eastern: 3.61 (2.39) 10th centile: 16.00 90th centile: 51.05 Combined: 30.07 (10.55-112.33) SD = 15.27 10th centile: 16.00 90th centile: 50.51 **Monitoring Stations:** Data obtained from an air quality simulation model (TAPM) by CSIRO Atmospheric Research **Copollutant:** NR	**PM Increment:** Increase from 10th to 90th centile **RR Estimate [CI]:** Same day lag Western: NR Inner Melbourne: 1.17 [1.05,1.31] South/Southeastern: 1.14 [0.95,1.33] Eastern: 1.09 [1.01,1.18] **Notes:** All other lags NR
Reference: Kuo et al. (2002) **Period of Study:** 1 yr **Location:** central Taiwan	Hospital Admissions **Outcome:** Asthma **Age Groups:** 13-16 yrs **Study Design:** Cohort **N:** 12,926 **Statistical Analyses:** Multiple logistic regression, Pearson correlation **Covariates:** Sex, age, residential area, level of parents' education, number of cigarettes smoked by smokers in the family, incense burning, frequency of physical activity **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 1 h **Mean (min-max):** NR Range: (54.1-84.3) **Monitoring Stations:** 8 **Copollutant:** Values NR **Notes:** Author states that a positive correlation was found between NO_2 and PM_{10}	**PM Increment:** NR **OR Estimate:** PM_{10} <65.9 µg/m³–referent PM_{10} >65.9 µg/m³ Crude OR: 0.837 Adj OR: 0.947 95% CI: (0.640,1.401)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Langley-Turnbaugh et al. (2005) **Period of Study:** 2000-2001 **Location:** Portland, Bridgeton, and Presque Isle, Maine	Hospital Admissions **Outcome (ICD-9):** Asthma (493xx) **Age Groups:** 0-18 yrs, 19+ yrs **Study Design:** Time series **N:** NR **Statistical Analyses:** NR **Covariates:** NR **Season:** Winter, spring, summer, fall **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** NR **Notes:** Hospital admissions were used to determine seasonality of asthma admissions so that PM components from those time periods could be analyzed	**Pollutant:** PM_{10} **Averaging Time:** NR **Mean (min-max):** NR **Monitoring Stations:** NR **Copollutant:** NR	**PM Increment:** NR **RR Estimate [CI]:** NR **Notes:** Portland filters contained more PM in the winter (Jan) and Bridgeton filters contained more PM in the spring (May); study analyzed metal components of PM_{10} (Mn, Cu, Pb, As, V, Ni, Al) Clinical data shows a strong peak in fall and weaker peaks in Jan and May for asthma admissions
Reference: Lin et al. (2005) **Period of Study:** 1998-2001 **Location:** Toronto, North York, East York, Etobicoke, Scarborough, and York (Canada)	Hospital Admissions **Outcome (ICD-9):** Respiratory infections including laryngitis, tracheitis, bronchitis, bronchiolitis, pneumonia, and influenza (464, 466, 480-487) **Age Groups:** 0-14 yrs **Study Design:** Bidirectional case-crossover **N:** 6782 respiratory infection hospitalizations **Statistical Analyses:** Conditional logistic regression (Cox proportional hazards model) **Covariates:** Daily mean temp and dew point temp **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS 8.2 PHREG procedure **Lags Considered:** 1-7 day averages	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 20.41 (4.00-73.00) SD = 10.14 **Monitoring Stations:** 4 **Copollutant (correlation):** $PM_{2.5}$: r = 0.87 $PM_{10-2.5}$: r = 0.76 CO: r = 0.10 SO_2: r = 0.48 NO_2: r = 0.54 O_3: r = 0.54	**PM Increment:** 12.5 µg/m^3 **OR Estimate [CI]:** Adjusted for weather 4 day avg: 1.22 [1.10,1.34] 6 day avg: 1.25 [1.11,1.40] Adj for weather and other gaseous pollutants 4 day avg: 1.14 [0.99,1.32] 6 day avg: 1.20 [1.01,1.42] **Notes:** OR's were also categorized into "Boys" and "Girls," yielding similar results

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lin et al. (2002) **Period of Study:** Jan 1, 1981–Dec 31, 1993 **Locaton:** Toronto	Hospital Admissions **Outcome (ICD-9):** Asthma (493) **Age Groups:** 6-12 yrs **Study Design:** Uni- and bi-directional case-crossover (UCC, BCC) and time-series (TS) **N:** 7,319 asthma admissions **Statistical Analyses:** Conditional logistic regression, GAM **Covariates:** Maximum and minimum temp, avg relative humidity **Season:** Apr-Sep, Oct-Mar **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1-7 day averages	**Pollutant:** PM_{10} **Averaging Time:** 6 days (predicted daily values) **Mean (min-max):** 30.16 (3.03-116.20) SD = 13.61 **Monitoring Stations:** 1 **Copollutant (correlation):** $PM_{2.5}$: r = 0.87 $PM_{10-2.5}$: r = 0.83 CO: r = 0.38 SO_2: r = 0.44 NO_2: r = 0.52 O_3: r = 0.44	**PM Increment:** 14.8 µg/m^3 **RR Estimate [CI]:** Adj for weather and gaseous pollutants BCC 5 day avg: 0.99 [0.90,1.09] BCC 6 day avg: 1.01 [0.90,1.12] TS 5 day avg: 1.03 [0.95,1.11] TS 6 day avg: 1.02 [0.94,1.11] Boys–adj for weather UCC 1 day avg: 1.10 [1.04,1.17] UCC 2 day avg: 1.10 [1.02,1.17] BCC 1 day avg: 1.04 [0.98,1.09] BCC 2 day avg: 1.01 [0.95,1.08] TS 1 day avg: 1.03 [0.99,1.07] TS 2 day avg: 1.01 [0.96,1.05] Girls–adj for weather UCC 1 day avg: 1.07 [0.99,1.16] UCC 2 day avg: 1.15 [1.04,1.26] BCC 1 day avg: 0.99 [0.92,1.06] BCC 2 day avg: 1.03 [0.95,1.12] TS 1 day avg: 0.99 [0.94,1.04] TS 2 day avg: 1.02 [0.96,1.08] **Notes:** The author also provides RR using UCC, BCC, and TS analysis for female and male groups for days 3-7, yielding similar results
Reference: Masjedi et al. (2003) **Period of Study:** Sep 1997–Feb 1998 **Location:** Tehran, Iran	Hospital Admissions **Outcome (ICD-9):** Acute asthma and COPD exacerbations (ICD: NR) **Age Groups:** NR **Study Design:** Time series **N:** 355 patients **Statistical Analyses:** Multiple stepwise regression, autoregression method (time series), Pearson correlation **Covariates:** NR **Season:** NR **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 3, 7, and 10 day mean	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 108.41 (14.5-506.60) SD = 59.55 **Monitoring Stations:** 3 **Copollutant:** NR	**PM Increment:** NR **Results:** Time-series analysis Asthma: β = 0.002; p = 0.32 COPD: β = 0.004; p = 0.02 Total Acute Resp Conditions: β = 0.006; p = 0.27 Correlation of 3-day mean Asthma: r = -0.21; β = -0.16; p = 0.08 Correlation of weekly mean Asthma: r = -0.27; β = -0.008; p = 0.12 Correlation of 10-day mean Asthma: r = -0.38; β = -0.066; p = 0.089
Reference: McGowan et al. (2002) **Period of Study:** Jun 1988–Dec 1998 **Location:** Christchurch, New Zealand	Hospital Admissions **Outcome (ICD-9):** Pneumonia (480-487), acute respiratory infections (460-466), chronic lung diseases (491-492, 494-496), asthma (493) **Age Groups:** <15 yrs, 15-64, 65+ **Study Design:** Time series **N:** 20,938 admissions **Statistical Analyses:** GAM with log link, Linear Regression Model **Covariates:** Wind speed, relative humidity, temperature **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-PLUS **Lags Considered:** 0-6 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 25.17 (0-283) SD = 25.49 **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 14.8 µg/m^3 (IQR) **% Increase [CI]:** Respiratory Admissions (2-day lag) 0-14 yrs: 3.62 [2.34,4.90]; 15-64 yrs: 3.39 [1.85,4.93]; 65+ yrs: 2.86 [1.23,4.49]; All ages: 3.37 [2.34,4.40] Overall Acute respiratory infections: 4.53 [2.82,6.24]; Pneumonia/influenza: 5.32 [3.46,7.18]; Chronic lung diseases: 3.95 [2.15,5.75]; Asthma: 1.86 [0.48,.3.24] Total Respiratory Admissions Same day lag: 2.52 [1.49,3.55]; 1-day lag: 2.56 [1.53,3.59]; 2-day lag: 3.37 [2.34,4.40]; 3-day lag: 3.09 [2.06,4.12]; 4-day lag: 3.13 [2.10,4.16]; 5-day lag: 3.21 [2.18,4.24]; 6-day lag: 3.09 [2.06,4.12]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Vegni and Ros (2004) **Period of Study:** Sep 1, 2001–Sep 31, 2002 **Location:** Milan area, Italy	Hospital Admissions **Outcome (ICD-9):** Respiratory, non-infectious admissions (ICD: NR) **Age Groups:** NR **Study Design:** Time series **N:** 9881 admissions **Statistical Analyses:** Poisson regression **Covariates:** Temperature, wind velocity, relative humidity, week day, holidays **Season:** Spring, summer, autumn, winter **Dose-response Investigated?** No **Statistical Package:** STATA v. 5 **Lags Considered:** 0, 1, and 2-day	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (5th-95th percentile):** Overall: 41.5 (13-98) SD = 28.2 Spring: 29.0 (10-51) SD = 12.6 summer: 24.8 (10-40) SD = 9.9 Autumn: 51.8 (21-114) SD = 27.1 Winter: 64.1 (20-135) SD = 35.7 **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** Increase from 5th–95th percentile Spring: 85 µg/m^3 summer: 30 µg/m^3 Autumn: 93 µg/m^3 Winter: 115 µg/m^3 **RR Estimate [CI]:** Overall: 1.10 [0.83,1.46] Adjusted: 0.97 [0.67,1.41] **Notes:** 1-day and 2-day lags show similar results, with no association between PM_{10} and daily hospital admissions
Reference: Yang et al. (2004c) **Period of Study:** Jun 1, 1995–Mar 31, 1999 **Location:** Vancouver area, Briti	Hospital Admissions **Outcome (ICD-9):** Respiratory diseases (460-519), pneumonia only (480-486), asthma only (493) **Age Groups:** 0-3 yrs **Study Design:** Case control, bidirectional case-crossover (BCC), and time series (TS) **N:** 1610 cases **Statistical Analyses:** Chi-square test, Logistic regression, GAM (time-series), GLM with parametric natural cubic splines **Covariates:** Gender, socioeconomic status, weekday, season, study year, influenza epidemic month **Season:** Spring, summer, fall, winter **Dose-response Investigated?** No **Statistical Package:** SAS (Case control and BCC), S-Plus (TS) **Lags Considered:** 0-7 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 13.3 (3.8-52.2) SD = 6.1 **Monitoring Stations:** NR (data obtained from Greater Vancouver Regional District Air Quality Dept) **Copollutant (correlation):** $PM_{2.5}$: r = 0.83 $PM_{10-2.5}$: r = 0.83 CO: r = 0.46 O_3: r = -0.08 NO_2: r = 0.54 SO_2: r = 0.61	**PM Increment:** 7.9 µg/m^3 (IQR) **OR Estimate [CI]:** Values NR **Notes:** Author states that ORs for PM_{10} increased with lag time up to 3 days for both single and multiple-pollutant models.
Reference: Fung al. (2005) **Period of Study:** Nov 1, 1995–Dec 31, 2000 **Location:** London, Ontario	Hospital Admissions **Outcome (ICD-9):** Asthma (493) and all other respiratory diseases (460-519) **Age Groups:** <65 yrs 65+ yrs **Study Design:** Time series **N:** 5574 respiratory admissions **Statistical Analyses:** GAM with locally weighted regression smoothers (LOESS) **Covariates:** Maximum and minimum temp, humidity, day of the week, seasonal cycles, secular trends **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** Current to 3-day mean	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 38.0 (5-248) SD = 23.5 **Monitoring Stations:** 4 **Copollutant (correlation):** NO_2: r = 0.30 SO_2: r = 0.24 CO: r = 0.21 O_3: r = 0.53 COH: r = 0.29	**PM Increment:** 26 µg/m^3 **% Change in Daily Admission [CI]:** Age <65 Current day mean: -0.9 [-6.8,5.4] 2-day mean: -1.3 [-8.5,6.6] 3-day mean: 1.9 [-6.5,11] Age 65+ Current day mean: 3.3 [-1.7,8.6] 2-day mean: 5 [-1.5,11.9] 3-day mean: 1.2 [-6.1,9.1]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Neuberger et al. (2004) **Period of Study:** 1999-2000 (1 yr period) **Location:** Vienna and Lower Austria	Hospital Admissions **Outcome (ICD-9):** Bronchitis, emphysema, asthma, bronchiectasis, extrinsic allergic alveolitis, and chronic airway obstruction (490-496) **Age Groups:** 3.0-5.9 yrs; 7-10 yrs; 65+ **Study Design:** Time series **N:** 366 days (admissions NR) **Statistical Analyses:** GAM **Covariates:** SO_2, NO, NO_2, O_3, temperature, humidity, and day of the week **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus 2000 **Lags Considered:** 0-14 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Maximum daily mean:** Vienna: 105 Rural area: NR **Monitoring Stations:** NR **Copollutant:** NR	**PM Increment:** 10 µg/m^3 **Log Relative Rate Estimate (p-value):** Vienna Male: 2 day lag = 4.217 (0.030) Association with tidal lung functioN: β = -1.067 (p-value = 0.241) **Notes:** Effect parameters with significant coefficients for respiratory health included: male sex, allergy, asthma in family, and traffic for Vienna and age, allergy, asthma in family, and passive smoking for the rural area. Effect parameters with significant coefficients for log asthma score were allergy, asthma in family, and rain for Vienna and allergy, asthma in family, and passive smoking for the rural area.
Reference: Jalaludin et al. (2004) **Period of Study:** Feb 1–Dec 31, 1994 **Location:** Sydney, Australia	Doctor Visits **Outcome (ICD- NR):** Respiratory symptoms (wheeze, dry cough, and wet cough), asthma medication use, and doctor visits for asthma **Age Groups:** Primary school children **Study Design:** Longitudinal cohort study **N:** 125 children **Statistical Analyses:** GEE logistic regression models **Covariates:** Temperature, humidity, daily pollen count, daily alternaria count, number of h spend outdoors, season **Season:** Autumn (Feb-Apr), winter (May-Aug), spring/summer (Sep-Dec) **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 22.8 (13.8) **Monitoring Stations:** 4 **Copollutant (correlation):** O_3: r = 0.13 NO_2: r = 0.26 Other variables: Temp: r = 0.04 Humidity: r = -0.29 Total pollen: r = 0.04 Alternaria: r = 0.04	**PM Increment:** IQR (µg/m^3) Same day: 12.0 1-day lag: 12.02 2-day lag: 12.25 2-day avg: 11.15 5-day avg: 10.23 **OR Estimate [CI]:** Doctor Visits for Asthma Same day: 1.11 [1.04,1.19] 1-day lag: 1.10 [1.02,1.19] 2-day lag: 1.15 [1.06,1.24] 2-day avg: 1.11 [1.03,1.20] 5-day avg: 1.14 [0.98,1.31] **Prevalence of Doctor Visits for Asthma:** Quartile 1: 0.50 (mean PM = 12.4) Quartile 2: 0.38 (mean PM = 17.2) Quartile 3: 0.65 (mean PM = 23.0) Quartile 4: 0.63 (mean PM = 38.3) **Notes:** ORs and prevalence are also provided for wheeze, dry cough, wet cough, inhaled β2-agonist use, and inhaled corticosteroid use. None were statistically significant.
Reference: Anderson et al. (2003) **Period of Study:** 1992-1994 **Location:** London, United Kingdom	**Outcome:** Lower respiratory disease (LRD), COPD, and asthma **Age Groups:** 0-15, 15-64, 65-74, 75+ **Study Design:** Time series **N:** NR **Statistical Analyses:** NR **Covariates:** NR **Season:** NR **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** NR **Monitoring Stations:** NR **Copollutant:** NR	**PM Increment:** 10th–90th percentile **% Change in Daily LRD admissions [CI]:** 0-15: 5.6 [2.3,9] 15-64: 3.9 [0.5,7.5] 65-74: 3.1 [-1,7.4] 75+: 2.1 [-1.7,6.1] **Notes:** RRs are presented in graph form showing a decline in hospital admissions with increasing age (PM increment of 10 µg/m^3). This article is primarily a systematic literature review of other studies.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Arena et al. (2006) **Period of Study:** 1995-2000 **Location:** Allegheny County, Pennsylvania	**Outcome (ICD-9):** Respiratory (460-519) and cardiac (390-459) outcomes combined **Age Groups:** 65+ **Study Design:** Time series **N:** 253,151 hospital admissions **Statistical Analyses:** GAM **Covariates:** Daily temperature, humidity, day of the week, time **Season:** Spring, summer, Fall, Winter **Dose-response Investigated?** Yes **Statistical Package:** S-Plus **Lags Considered:** 0-5 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 27.9 (4.8-102.4) SD = 15.5 **Monitoring Stations:** 8 **Copollutant (correlation):** NR Other variables: Daily admissions: r = -0.031 Temp: r = 0.501* Humidity: r = -0.211* *p<0.0001	**PM Increment:** NR **Lag Model Coefficients:** Ranged from 0.000652-0.000551, same-day to 5-day models **Notes:** Monthly mean PM was graphed in Fig 2 with higher values in the summer and fall. All coefficients are listed for the distributed and unconstrained lag models in tables 3 and 4.
Reference: Bakonyi et al. 2004 **Period of Study:** Jan 1, 1999–Dec 31, 2000 **Location:** Curitiba, State of Parana, Brazil	Hospital Admissions **Outcome (ICD-9):** Respiratory disease (460-519) **Age Groups:** 0-14 yrs **Study Design:** Time series **N:** 81,229 **Statistical Analyses:** GAM using nonparametric smoothing functions (loess) **Covariates:** day of the week, temperature, relative humidity **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus and SPSS **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 90.39 (20.00-245.00) SD = 37.37 **Monitoring Stations:** 1 **Copollutant (correlation):** NO_2: r = 0.53 O_3: r = 0.23 Other variables: Min temp: r = -0.35 Rel humidity: r = -0.36 Respiratory disease: r = 0.29	**PM Increment:** NR **RR Estimate [CI]:** Same-day avg: 1.0008 [1.0004,1.0012] 2-day avg: 1.0011 [1.0006,1.0016] 3-day avg: 1.0012 [1.0007,1.0017] **Notes:** Figure 2 showed a percent increase ~11 for the 3-day moving avg (90.39 μg/m^3 unit increase).
Reference: Ren et al. (2006) **Period of Study:** Jan 1, 1996–Dec 31, 2001 **Location:** Brisbane, Australia	Hospital Admissions **Outcome (ICD-9):** Respiratory diseases (460-519) excluding influenza (487.0-487.8) **Age Groups:** NR **Study Design:** Time series **N:** NR **Statistical Analyses:** GAM **Covariates:** Day of week, relative humidity, influenza outbreaks **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus **Lags Considered:** 0, 1, and 2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 15.84 (2.5-60) **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** NR **Coefficient Estimates:** Respiratory Hospital Admissions Same day: -0.004296 1-day lag: -0.002474 2-day lag: -0.004229 *all statistically significant Respiratory Emergency Visits Same day: -0.000887 1-day lag: -0.004209 2-day lag: -0.003440 **Notes:** Relative risks were provided in graphical form (Fig 3)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Wong et al., (2002) **Period of Study:** 1995-1997 (Hong Kong) and 1992-1994 (London) **Location:** Hong Kong and London	Hospital Admissions **Outcome (ICD- NR):** Asthma (493) for ages 15-64 and respiratory disease (460-519) for ages 65+ **Age Groups:** 15-64, 65+ **Study Design:** Time series **N:** NR **Statistical Analyses:** Poisson regression, GAM **Covariates:** Temperature, humidity, and influenza **Season:** Warm (Apr-Sep) and cool (Oct-Mar) **Dose-response Investigated?** Yes **Statistical Package:** S-Plus **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** Hong Kong: 51.8 (14.1-163.8) SD = 25.0 London: 28.5 (6.8-99.8) SD = 13.7 **Monitoring Stations:** NR **Copollutant (correlation):** Hong Kong NO_2: r = 0.82; SO_2: r = 0.30; O_3: r = 0.54 London NO_2: r = 0.68; SO_2: r = 0.64; O_3: r = 0.17 **Other variables:** Hong Kong Temp: r = -0.42 Humidity: r = -0.53 London Temp: r = 0.02 Humidity: r = -0.05	**PM Increment:** 10 µg/m^3 **ER Estimate [CI]:** Single-pollutant excess risk (mean lag 0-1 day) Asthma–Hong Kong: -1.1 [-2.4,0.1] Asthma–London: 1.4 [-0.1,3.0] Respiratory Disease–Hong Kong: 1.0 [0.5,1.5] Respiratory Disease–London: 0.4 [-0.3,1.2] Warm season Asthma–Hong Kong: -1.0 [-2.8, 0.8] Asthma–London: 0.6 [-1.9,3.1] Respiratory Disease–Hong Kong: 0.8 [0.1,1.4] Respiratory Disease–London: 1.8 [0.5,3.1] Cool season Asthma–Hong Kong: -1.2 [-2.8,0.4] Asthma–London: 1.6 [-0.3,3.6] Respiratory Disease–Hong Kong: 1.2 [0.6,1.9] Respiratory Disease–London: -0.5 [-1.5,0.5] **Notes:** RRs are shown graphically in Fig 1 and 2. Exposure response curves are provided in Fig 5 of the article
Reference: Wong et al. (2006) **Period of Study:** 2000-2002 **Location:** Hong Kong (8 districts)	General Practitioner Visits **Outcome (ICPC-2):** Respiratory diseases/symptoms: upper respiratory tract infections (URTI), lower respiratory infections, influenza, asthma, COPD, allergic rhinitis, cough, and other respiratory diseases **Age Groups:** All ages **Study Design:** Time series **N:** 269,579 visits **Statistical Analyses:** GAM, Poisson regression **Covariates:** Season, day of the week, climate **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0-3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** Ranged from 43.4-56.9 (dependent on location) **Monitoring Stations:** 1 per district **Copollutant (correlation):** $PM_{2.5}$: r = 0.94 O_3: r = 0.40 SO_2: r = 0.28	**PM Increment:** 10 µg/m^3 **RR Estimate [CI]:** Overall URTI 1.020 [1.016,1.025] Overall Non-UTRI 1.025 [1.018,1.032] **Notes:** RRs are also reported for each individual general practitioner yielding similar results
Reference: Nascimento et al. (2006) **Period of Study:** May 1, 2000–Dec 31, 2001 **Location:** São Jose dos Campos, Brazil	Hospital Admissions **Outcome (ICD-10):** Pneumonia (J12-J18) **Age Groups:** 0-10 yrs **Study Design:** Time series **N:** 1265 admissions **Statistical Analyses:** GAM, Poisson regression **Covariates:** Temperature, humidity **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus, SPSS **Lags Considered:** 0-7 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (min-max):** 40.2 (3.4-196.6) SD = 26.9 **Monitoring Stations:** 2 **Copollutant (correlation):** SO_2: r = 0.30 O_3: r = 0.09 Other variables: Admissions: r = 0.21 Temp: r = -0.14 **Notes:** All p<0.05	**PM Increment:** 24.7 µg/m^3 **Regression coefficients (SE):** Same day: -0.00053 (0.00125) 1-day lag: 0.00029 (0.00057) 2-day lag: 0.00089 (0.00069) 3-day lag: 0.00122 (0.00053)* 4-day lag: 0.00126 (0.00055)* 5-day lag: 0.00098 (0.00071) 6-day lag: 0.00035 (0.00056) 7-day lag: -0.00067 (0.00123) *p<0.05 **Notes:** Percent increase over all lag days is displayed in Fig 2

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hapcioglu et al. (2006) **Period of Study:** Jan 1, 1997–Dec 31, 2001 **Location:** Istanbul, Turkey	Hospital Admissions **Outcome (ICD-9):** COPD (ICD: NR) **Age Groups:** NR **Study Design:** Time series **N:** 1586 patients **Statistical Analyses:** Multiple stepwise regression, Pearson correlation **Covariates:** Humidity, temperature, and pressure **Season:** summer, autumn, winter, spring **Dose-response Investigated?** No **Statistical Package:** SPSS **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** 1 month **Mean (SD):** NR **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** NR **Correlation with COPD:** r = 0.28; p = 0.03 Adj for temp: r = 0.16; p = 0.23 **Notes:** RRs only provided for season, not PM
Reference: Luginaah, et al. 2005 **Period of Study:** Apr 1995-Dec 2000 **Location:** Windsor, Ontario, Canada	**Hospital Admission/ED:** admission **Outcome:** All respiratory: 460-519 **Age Groups: A**ll, 0-14, 15-64, and >65 **Study Design:** Times-series, bi-directional case-crossover **N:** 4214 admissions **Statistical Analyses:** Poisson regression, GAM w/ stringent convergence criteria or natural splines, conditional logistic regression **Covariates:** Age, sex Maximum & minimum temperature, change in barometric pressure from previous day **Season:** All **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 1-3	**Pollutant:** PM_{10} **Averaging Time:** 24 h maximum **Mean (SD):** 50.6 ,(35.5) **Range (Min, Max):** 9, 349 **Monitoring Stations:** 4 **Notes: Copollutant (correlation):** PM_{10}-NO_2: 0.33 PM_{10}-SO_2: 0.22 PM_{10}-CO: 0.21 PM_{10}-O_3: 0.33	**PM Increment:** Interquartile range (75th-25th) 31 µg/m^3 **RR Estimates (Time Series)** **All Age Groups Females** 0.996 [0.950, 1.044], lag 1 1.015 [0.963, 1.069], lag 2 1.022 [0.968, 1.078], lag 3 **All Age Groups Males** 1.008 [0.965, 1.054], lag 1 1.036 [0.986, 1.089], lag 2 1.027 [0.974, 1.083], lag 3 **RR Estimates (Case Crossover)** **All Age Groups Females** 1.034 [0.974, 1.098], lag 1 1.045 [0.972, 1.124], lag 2 1.054 [0.970, 1.145], lag 3 **All Age Groups Males** 0.997 [0.942, 1.056], lag 1 1.022 [0.953, 1.097], lag 2 1.008 [0.930, 1.092], lag 3 **Notes:** Results, stratified by age group available in manuscript.
Reference: Bell et al, 2008 **Period of Study:** 1995 - 2002 **Location:** Taipei, Taiwan	Hospital Admissions **Outcome (ICD-9):** asthma (493), and pneumonia (486). **Age Groups Analyzed:** All **Study Design:** Time series **N** 19,966 for pneumonia, and 10,231 for asthma **Statistical Analyses:** Poisson regression **Covariates:** day of the week, time, apparent temperature, long-term trends, seasonality **Season:** All **Dose-response Investigated:** No **Statistical package:** NR **Lags Considered:** lags 0 – 3 days, average of lags 0 - 3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (range; IQR):** 49.1 (12.7 – 215.5; 27.6) **Monitoring Stations:** Taipei area: 13 monitors Taipei City: 5 monitors Monitors with correlations of 0.75 + for PM_{10}: 12 monitors **Copollutant (correlation):** NR	**PM Increment:** 28 µg/m^3 (near IQR) **Percentage increase estimate [95% CI] : Asthma:** **Taipei area (13 monitors):** L0: 2.63 (-0.01, 5.35); L1: 1.79 (-0.82, 4.48); L2: 2.20 (-0.41, 4.88); L3: 1.87 (-0.71, 4.50); L03: 4.48 (0.71, 8.38) **Taipei City (5 monitors):** L0: 2.53 (-0.09, 5.21); L1: 1.60 (-0.98, 4.25); L2: 2.21 (-0.37, 4.86); L3: 2.08 (-0.47, 4.69); L03: 4.68 (0.78, 8.73) **Monitors with > = 0.75 between monitor correlations (12 monitors):** L0: 2.56 (-0.04, 5.23); L1: 1.63 (-0.95, 4.26) ; L2: 1.97 (-0.59, 4.60); L3: 2.78 (-0.75, 4.37) ; L03: 4.27 (0.47, 8.22) **Pneumonia: Taipei area (13 monitors):** L0: 0.75 (-1.80, 3.36); L1: 0.16 (-2.36, 2.74); L2: 0.47 (-2.04, 3.03); L3: -0.70 (-3.19, 1.85); L03: 0.31 (-3.22, 3.97) **Taipei City (5 monitors):** L0: 0.88 (-1.64, 3.46); L1: 0.50 (-1.98, 3.05); L2: 0.59 (-1.88, 3.13); L3: -0.72 (-3.18, 1.79); L03: 0.83 (-2.83, 4.62) **Monitors with > = 0.75 between monitor correlations (12 monitors):** L0: 0.86 (-1.67, 3.41) ; L1: 0.12 (-2.35, 2.65) ; L2: 0.52 (-1.94, 3.05) ; L3: -0.53 (-2.98, 1.97) ; L03: 0.65 (-2.93, 4.36)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hanigan et al, 2008 **Period of Study:** 1996 – 2005 (April – November of each year) **Location:** Darwin, Australia	Hospital Admissions/ED visits **Outcome (ICD-9 or ICD-10):** Daily emergency hospital admissions for total respiratory (ICD-9: 460 – 519; ICD-10: J00 – J99), asthma (ICD-9: 493; ICD-10: J45 – J47), COPD (ICD-9: 490 – 492, 494 – 496; ICD-10: J40 – J44, J47, J67), and respiratory infections (ICD-9: 461 – 466, 480 – 487, 514; ICD-10: J00 – J22). **Age Groups Analyzed:** All **Study Design:** Time series **N:** 8,279 hospital admissions **Statistical Analyses:** Poisson generalized linear models **Covariates:** indigenous status, time in days, temperature, relative humidity, day of the week, influenza epidemics, change between ICD editions, holidays, yearly population **Season:** April – November (corresponding to the dry season) **Dose-response Investigated?** No **Statistical package:** R version 2.3.1 **Lags Considered:** lag 0 -3	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD; range):** 21.2 (8.2; 55.2) **Monitoring Stations:** N/A (see notes) **Copollutant (correlation):** NR Other variables:	**PM Increment:** 10 µg/m^3 **Percent change [95% CI] :** Overall respiratory disease: Lag 0: 4.81 [-1.04, 11.01] Lag 0 (indigenous people): 9.40 [1.04, 18.46] Lag 0 (non-indigenous people): 3.14 [-2.99, 9.66] In unstratified analyses, the subgroups of respiratory infections, asthma, and COPD all had positive associations with PM_{10} Lag 0. Asthma: Lag 1 (indigenous people): 16.27 [-3.55; 40.17] Lag 1 (non-indigenous people): 8.54 [-5.60, 24.80] Respiratory infections: Lag 3 (indigenous people): 15.02 [3.73, 27.54] Lag 3 (non-indigenous people); 0.67 [-7.55, 9.61] **Notes :** **Figure 3:** Associations between hospitalizations for non-indigenous and indigenous people with estimated ambient PM_{10}. **Summary of Figure 3:** Confidence intervals were wide, but indigenous people generally had stronger associations with PM_{10} than non-indigenous people. Daily PM_{10} exposure levels were estimated for the population of the city from visibility data using a previousy validated models.
Reference: Johnston et al 2007 **Period of Study:** 2000, 2004, 2005 (April – November of each year) **Location:** Darwin, Australia	Hospital Admissions/ED visits **Outcome (ICD-10):** All respiratory conditions (J00 – J99), including asthma (J45 – 46), COPD (J40 – J44), and respiratory infections (J00 – J22). **Age Groups Analyzed:** All **Study Design:** Case-crossover **N:** 2466 emergency admissions **Statistical Analyses:** Conditional logistic regression **Covariates:** weekly influenza rates, temperature, humidity, days with rainfall >5mm, public holidays, school holiday periods (for respiratory conditions only) **Season:** April – November (dry season) **Dose-response Investigated?** No **Statistical package:** NR **Lags Considered:** 0 – 3 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Median (IQR, 10th – 90th percentile, range):** 17.4 (13.6 – 22.3; 10.3 – 27.7; 1.1 – 70.0) **Monitoring Stations:** 1 **Copollutant (correlation):** NR	**PM Increment:** 10 µg/m^3 **OR Estimate [95% CI] : All respiratory conditions:** Lag 0: 1.08 [0.98 – 1.18]; Lag 0 (indigenous): 1.17 [0.98 – 1.40] **COPD:** Lag 0: 1.21 [1.0 – 1.47]; Lag 0 (indigenous): 1.98 [1.10 – 3.59] **Asthma:** Lag 0: 1.14 [0.90 – 1.44] **Asthma + COPD:** Lag 0: 1.19 [1.03 – 1.38] **Notes : Figure 1:** Adjusted OR and 95% CI for hospital admissions for all respiratory conditions per 10 µg/m^3 rise in PM_{10} for the same day and lags up to 3 days, overall and stratified by indigenous status. **Summary of Figure 1 results:** Marginally significant positive association at Lag 0 in overall study population. Larger marginally significant positive association among indigenous people. **Figure 2**: OR and 95% CI for hospital admissions for COPD. **Summary of Figure 2 results:** Marginally significant positive associations at Lag 0 and Lag 1 in overall study population and among non-indigenous people. Large, statistically significant positive association at Lag 0 for indigenous people, with smaller, non-significant positive associations at Lag 1 and Lag2. **Figure 3:** OR and 95% CI for hospital admissions for asthma. **Summary of Figure 3 results:** Positive, non-significant (sometime marginally significant) associations at Lag 0, Lag 2, and Lag 3 for overall population and indigenous status strata. **Figure 4:** OR and 95% CI for hospital admissions for respiratory infections. **Summary of Figure 4 results:** Negative associations at Lag 2 and Lag 3 in all population strata.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Middleton et al. (2008) **Period of Study:** 1995 – 1998, 2000 - 2004 **Location:** Nicosia, Cyprus	Hospital Admissions/ED visits **Outcome:** Hospital admissions for all respiratory disease (ICD-10: J00 – J99). **Age Groups Analyzed:** All, also stratified by age (<15 vs. >15 years) **Study Design:** Time series **N: Statistical Analyses:** generalized additive Poisson models **Covariates: seasonality, day of the week, long- and short-term trend, temperature, relative humidity** **Season:** NR **Dose-response Investigated:** No **Statistical package:** STATA SE 9.0, and the MGCV package in the R software (R 2.2.0) **Lags Considered:** lag 0 -2 days	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD; median; 5% - 95%; range):** **Cold:** 57.6 (52.5; 50.8; 20.0 – 103.0; 5.0 – 1370.6) **Warm:** 53.4 (50.5; 30.7; 32.0 – 77.6; 18.4 – 933.5) **Monitoring Stations:** 2 **Copollutant (correlation):** NR Other variables:	**PM Increment:** 10 µg/m^3 , and across quartiles of increasing levels of PM_{10} **Percentage increase estimate [CI] : All age/sex groups (Lag 0):** All admissions: 0.85 (0.55, 1.15); Respiratory (all): 0.10 (-0.91, 1.11); Respiratory (cold months): -0.33 (-1.47, 0.82); Respiratory (warm months): 1.42 (-0.42, 3.31); CVD + RD: 0.56 (-0.21, 1.34) **Nicosia residents (Lag 0):** Respiratory (all): 0.25 (-0.84, 1.36); Respiratory (cold months): -0.22 (-1.45, 1.02); Respiratory (warm months): 1.80 (-0.22, 3.85); CVD + RD: 0.38 (-0.47, 1.23) **Males (Lag 0):** All admissions: 0.96 (0.54, 1.39); Respiratory (all): -0.06 (-1.37, 1.26); Respiratory (cold months): -0.16 (-1.76, 1.46); Respiratory (warm months): 1.10 (-1.47, 3.74); CVD + RD: 0.63 (-0.34, 1.62) **Females (Lag 0):** All admissions: 0.74 (0.31, 1.18); Respiratory (all): 0.39 (-1.21, 2.02); Respiratory (cold months): -0.26 (-2.18, 1.70); Respiratory (warm months): 3.27 (-0.00, 6.65); CVD + RD: 0.59 (-0.68, 1.87) **Aged <15 years (Lag 0):** All admissions: 0.47 (-0.13, 1.08); Respiratory (all): -0.35 (-1.77, 1.08); Respiratory (cold months): -0.31 (-2.02, 1.42); Respiratory (warm months): -0.59 (-3.53, 2.45) **Aged >15 years (Lag 0):** All admissions: 0.98 (0.63, 1.33); Respiratory (all): 0.59 (-0.87, 2.07); Respiratory (cold months): 0.02 (-1.76, 1.83); Respiratory (warm months): 3.89 (1.05, 6.80)
Reference: Tolbert et al. (2007) **Period of Study:** 1993 - 2004 **Location:** Atlanta Metropolitan area, Georgia	Hospital Admissions/ED visits **Outcome (ICD-9):** **Combined RD group, including:** Asthma (493, 786.07, 786.09), COPD (491, 492, 496), URI (460 – 465, 460.0, 477), pneumonia (480 – 486), and bronchiolitis (466.1, 466.11, and 466.19)) **Age Groups Analyzed:** All **Study Design:** Time series **N:** 10,234,490 ER visits (283,360 and 1,072,429 visits included in the CVD and RD groups, respectively) **Statistical Analyses:** Poisson generalized linear models **Covariates:** long-term temporal trends, season (for RD outcome), temperature, dew point, days of week, federal holidays, hospital entry and exit **Season:** All **Dose-response Investigated:** No **Statistical package:** SAS version 9.1 **Lags Considered:** 3-day moving average(lag 0 -2)	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (median; IQR, range, 10th – 90th percentiles):** 26.6 (24.8; 17.5 – 33.8; 0.5 – 98.4; 12.3 – 42.8) **Monitoring Stations:** NR **Copollutant (correlation):** O_3: r = 0.59 NO_2: r = 0.53 CO: r = 0.51 SO_2: r = 0.21 Coarse PM: r = 0.67 $PM_{2.5}$: r = 0.84 $PM_{2.5}$ SO4: r = 0.69 $PM_{2.5}$ EC: r = 0.61 $PM_{2.5}$ OC: r = 0.65 $PM_{2.5}$ TC: r = 0.67 $PM_{2.5}$ water-sol metals: r = 0.73 OHC: r = 0.53	**PM Increment:** 16.30 µg/m^3 (IQR) **Risk ratio [95% CI] :** **Single pollutant models:** RD: 1.015 (1.006 – 1.024) **Notes :** Results of selected multi-pollutant models for respiratory disease are presented in Figure 2. **Figure 2:** PM_{10} adjusted for CO, O_3, NO_2, or NO_2/O_3 (non-winter months only) **Summary of results:** PM_{10} remained predictive of RD in non-winter months after adjustment for pollutants.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ulirsch et al. (2007) **Period of Study:** November 1994 – March 2000 **Location:** Pocatello, Idaho and Chubbuck, Idaho	Hospital Admissions/ED visits **Outcome (ICD-9):** Respiratory disease (460 – 519 and 786.09 [reactive airway disease]; excluding 500 – 500.8 for lung diseases due to external causes), and CVD (390 - 429). **Age Groups Analyzed:** All, 0 – 17 (RD only), 65 + , 18 – 64 (RD only) **Study Design:** Time series **N:** 39,347 admissions/visits **Statistical Analyses:** Log-linear generalized linear models **Covariates:** Time, temperature, relative humidity, influenza, day of the week **Season:** All, and separate analyses were performed for the all-age group for cool months (October – March) vs. warm months (April – September). **Dose-response Investigated:** No **Statistical package:** S-plus version 6.1 **Lags Considered:** 0 – 4 day lags, and mean of days 0 -4	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (range; 10th - 90th percentiles):** 24.2 (3.0 – 183.0; 10.5 – 40.7) **Monitoring Stations:** 4**Copollutant (correlation):** NO_2: r = 0.47 **Other variables:** Correlation for PM_{10} between monitors: r = 0.42 – 0.87	**PM Increment:** 50 µg/m³ , and 24.3 µg/m³ (mean increase in PM_{10}) **Mean percent of change (% change in the mean number of daily admissions and visits) [95% CI] :** **For 24.3 µg**/m³ **increase in** PM_{10}**:** All-age respiratory disease (all year): 4.0 [1.4, 6.7] All-age RD/CVD: 3.7 [1.3, 6.3] ; 18-64 years RD: 3.4 [0.2, 6.7] ; 0-17 years RD: 4.3 [-0.1, 8.9] ; 65+ years RD: 5.6 [-1.4, 13.1] ; 65+ years RD/CVD: 2.9 [-2.9, 8.7] ; 0-17/65+years RD: 5.5 [1.4, 9.6] ; All-age RD (cool season): 4.3 [1.3, 7.5] ; All-age RD (warm season): 6.7 [-0.8, 14.8] ; All-age CVD (Lag 0): -0.02 [-5.9, 6.3] ; **For 50 µg**/m³ **increase in** PM_{10} **(single pollutant models, CIs not given):** All-age respiratory disease: 8.4 ; All-age RD/CVD: 7.9 ; 18-64 years RD: 7.2 ; 0-17 years RD: 9.1 ; 65+ years RD: 12.0 ; 65+ years RD/CVD: 6.1 ; 0-17/65+years RD: 11.6 ; All-age RD (cool season): 9.1 ; All-age RD (warm season): 14.3 ; **For 50 µg**/m³ **increase in** PM_{10} **(multi-pollutant models, CIs not given):** Adjusted for SO_2 (for respiratory disease): All-age (all year): 10.8 ; 18-64: 8.0 ; 0-17: 10.8 ; 65+: 8.7 ; 0-17/65+: 14.2 ; All-age (cool season): 11.9 ; Adjusted for NO_2 (for respiratory disease): All-age (all year): 10.5 ; 18-64: 9.3 ; 0-17: 4.6 ; 65+: 12.4 ; 0-17/65+: 9.5 ; All-age (cool season): 11.1 Adjusted for SO_2 and NO_2 (for respiratory disease): All-age (all year): 11.3 ; 18-64: 9.0 ; 0-17: 6.2 ; 65+: 12.0 ; 0-17/65+: 10.3All-age (cool season): 11.0 **Notes :** Included urgent care visits as well as emergency department visits and hospital admissions.

Table E-14. Short-term exposure to $PM_{10-2.5}$ and emergency department visits and hospital admissions for respiratory outcomes.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Fung et al. (2006) **Period of Study:** 6/1/95–3/31/99 **Location:** Vancouver, Canada	**Hospital Admission/ED:** Hospital Admission **Outcome:** Respiratory diseases (460-519) **Age Groups:** Age >65 **Study Design:** Time series **N:** 26,275 individuals admitted **Statistical Analyses:** Poisson regression (spline 12 knots), case-crossover (controls +/7 d days from case date), Dewanji and Moolgavkar (DM) method **Covariates:** Long-term trends, day-of-the-week effect, weather **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SPlus, R **Lags Considered:** 0-7 d	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h Avg **Mean (SD)** 5.6(3.88) **Range (Min, Max):** (-2.9, 27.07) **Monitoring Stations:** NR **Notes: Copollutant (correlation):** $PM_{10-2.5}$ PM_{10}; r = 0.83 $PM_{2.5}$; r = 0.34 CO; r = 0.51 CoH; r = 0.61 O_3; r = -0.11 NO_2; r = 0.52 SO_2; r = 0.57	**PM Increment::** 4.3 µg/m³ **RR Estimate** (65+ years) DM method: 1.011[0.998,1.024]; lag 0 1.016[1.0,1.032]; 3 d avg 1.020[1.001,1.039]; 5 d avg 1.020[0.998,1.042]; 7 d avg Time series: 1.0168[1.003, 1.031]; lag 0 1.020[1.003, 1.037]; 3 d avg 1.019[0.999, 1.039]; 5 d avg 1.018[0.994, 1.042]; 7 d avg Case-crossover: 1.019[1.003, 1.034]; lag 0 1.019[1.009, 1.038]; 3 d avg 1.020[0.999, 1.042]; 5 d avg 1.018[0.994, 1.043]; 7 d avg

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Host et al. (2007) **Period of Study:** 2000 - 2003 **Location:** Six French cities: Le Havre, Lille, Marseille, Paris, Rouen, and Toulouse	**Outcome (ICD-10):** Daily hospitalizations for all respiratory diseases (J00–J99), respiratory infections (J10–J22). **Age Groups:** For all respiratory diseases: 0–14 years, 15–64 years, and ≥ 65 years For respiratory infections: All ages **Study Design:** Time series **N:** NR (Total population of cities: approximately 10 million) **Statistical Analyses:** Poisson regression **Covariates:** Seasons, days of the week, holidays, influenza epidemics, pollen counts, temperature, and temporal trends **Dose-response Investigated:** No **Statistical Package:** MGCV package in R software (R 2.1.1) **Lags Considered:** Avg of 0-1 days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (5th -95th percentile):** Le Havre: 7.3 (2.5–14.0) Lille: 7.9 (2.2–13.7) Marseille: 11.0 (4.5–21.0) Paris: 8.3 (3.2–15.9) Rouen: 7.0 (3.0–12.5) Toulouse: 7.7 (3.0–15.0) **Monitoring Stations:** 13 total: 1 in Toulouse 4 in Paris 2 each in other cities **Copollutant (correlation):** $PM_{2.5}$: Overall: r>0.6 Ranged between r = 0.28 and r = 0.73 across the six cities.	**PM Increment:** 10 μg/m³ , and an 18.8 μg/m³ increase (corresponding to an increase in pollutant levels between the lowest of the 5th percentiles and the highest of the 95th percentiles of the cities' distributions) **ERR (excess relative risk) Estimate [CI]:** For all respiratory diseases (10 μg/m³ increase): 0–14 years: 6.2% [0.4, 12.3] ; 15–64 years: 2.6% [-0.5, 5.8] ; ≥ 65 years: 1.9% [-1.9, 5.9] For all respiratory diseases (18.8 μg/m³ increase): 0–14 years: 12.0 [0.8, 24.3] ; 15–64 years: 5.0 [-0.9, 11.1] ; ≥ 65 years: 3.7 [-3.6, 11.4] For respiratory infections (10 μg/m³): All ages: 4.4% [0.9, 8.0] For respiratory infections (18 μg/m³): All ages: 8.4% [1.7, 15.5]
Reference: Peel et al. (2005) **Period of Study:** Jan 1993-Aug 2000 **Location:** Atlanta, Georgia	ED visits **Outcome:** Asthma (493, 786.09); COPD (491, 492, 496); URI (460-466, 477); Pneumonia (480-486) **Age Groups:** All ages. Secondary analyses conducted by age group: 0-1, 2-18, >18 **Study Design:** Time series **N:** 31 hospitals **Statistical Analyses:** Poisson GEE for URI, asthma and all RD; Poisson GLM for pneumonia and COPD) **Covariates:** Avg temperature and dew point, pollen counts **Season:** All (secondary analyses of warm season) **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.3; S-Plus 2000 **Lags Considered:** 0-7 d , 3 d ma, 0-13 d unconstrained distributed lag	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h avg **Mean (SD):** 9.7 (4.7) Percentiles: 10th: 4.4 90th: 16.2 **Monitoring Stations:** "Several" **Copollutant (correlation):** 24 h PM_{10}: r = 0.59 8 h O_3: r = 0.35 1 h NO_2: r = 0.46 1 h CO: r = 0.32 1 h SO_2: r = 0.21 24 h $PM_{2.5}$: r = 0.43 Components: r ranged from 0.23-0.51	**PM Increment:** 5 RR Estimate [Lower CI, Upper CI] All Respiratory Outcomes: 1.003 [0.982, 1.025]; URI: 1.013 [0.987, 1.039]; Asthma: 0.998 [0.987, 1.039]; Pneumonia: 0.975 [0.940, 1.011]; COPD: 0.948 [0.897, 1.003]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Peng et al. (2008) **Period of Study:** January 1, 1999–December 31, 2005 **Location:** 108 U.S. counties in the following states: Alabama, Arizona, California, Colorado, Connecticut, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin	**Outcome (ICD-9):** Emergency hospitalizations for respiratory disease, including COPD (490–492) and respiratory tract infections (464–466, 480 - 487) **Age Groups:** 65 + years, 65–74, ,75 + **Study Design:** Time series **N:** approximately 12 million Medicare enrollees (1.4 million RD admissions) **Statistical Analyses:** Two-stage Bayesian hierarchical models: Overdispersed Poisson models for county-specific data. Bayesian hierarchical models to obtain national avg estimate **Covariates:** Day of the week, age-specific intercept, temperature, dew point temperature, calendar time, indicator for age of 75 years or older. Some models were adjusted for $PM_{2.5}$. **Dose-response Investigated:** No **Statistical Package:** R version 2.6.2 **Lags Considered:** 0-2 days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (IQR):** All counties assessed: 9.8 (6.9–15.0) Counties in Eastern US: 9.1 (6.6–13.1) Counties in Western US: 15.4 (10.3–21.8) **Monitoring Stations:** At least 1 pair of co-located monitors (physically located in the same place) for PM_{10} and $PM_{2.5}$ per county **Copollutant (correlation):** $PM_{2.5}$: r = 0.12 PM_{10}: r = 0.75 **Other variables:** Median within-county correlations between monitors: r = 0.60	**PM Increment:** 10 µg/m^3 **Percentage change [95% CI]:** Respiratory disease (RD): Lag 0 (unadjusted for $PM_{2.5}$): 0.33 [-0.21, 0.86] Lag 0 (adjusted for $PM_{2.5}$): 0.26 [-0.32, 0.84] Most values NR (see note) **Notes:** Figure 3: Percentage change in emergency hospital admissions for RD per 10 µg/m^3 increase in PM (single pollutant model and model adjusted for $PM_{2.5}$ concentration) Figure 4: Percentage change in emergency hospital admissions rate for CVD and RD per a 10 µg/m^3 increase in $PM_{10-2.5}$ (0–2 day lags, Eastern vs. Western USA)
Reference: Sinclair and Tolsma (2004) **Period of Study:** 25 Months **Location:** Atlanta, Georgia	Outpatient Visits **Outcome:** Asthma (493); URI (460, 461, 462, 463, 464, 465, 466, 477); LRI (466.1, 480, 481, 482, 483, 484, 485, 486). **Age Groups:** < = 18 y, 18+ y (asthma); All ages (URI//LRI) **Study Design:** Times series **N:** 25 months; 260,000 to 275,000 health plan members (August 1998–August 2000) **Statistical Analyses:** Poisson GLM **Covariates:** Season, Day of week, Federal Holidays, Study Months **Season:** NR **Dose-response Investigated?:** No **Statistical Package:** SAS **Lags Considered:** Three 3 d moving averages (0-2, 2-5, 6-8)	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h avg **Mean (SD):** PM coarse mass ((2.5-10 µm))–9.67 µg/m^3 (4.74) **Monitoring Stations:** 1 **Copollutant (correlation):** NR	**PM Increment:** 4.74 (1 SD) **RR Estimate [Lower CI, Upper CI]; lag:** Child Asthma: Coarse PM = 1.053 (S); 3-5 days lag URI: Course PM = 1.021 (S); 3-5 days lag LRI: Coarse PM = 1.07 (S); 3-5 days lag **Notes:** Numerical findings for significant results only presented in manuscript. Results for all lags presented graphically for each outcome (asthma, URI, and LRI).

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Slaughter et al. (2005) **Period of Study:** January 1995 through June 2001 **Location:** Spokane, WA Notes	Hospital Admissions and ED visits **Outcome:** All respiratory (460-519); Asthma (493); COPD (491,492, 494,496); Pneumonia (480-487); Acute URI not including colds and sinusitis (464, 466, 490) **Age Groups:** All, 15+ years for COPD **Study Design:** Time series **N:** 2373 visit records **Statistical Analyses:** Poisson regression, GLM with natural splines. For comparison also used GAM with smoothing splines and default convergence criteria. **Covariates:** Season, temperature, relative humidity, day of week **Season:** All **Dose-response Investigated?**: No **Statistical Package:** SAS, SPLUS **Lags Considered:** 1 -3 d	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h avg **Range (90% of Concentrations):** Reported for $PM_{2.5}$ and PM_{10} only **Monitoring Stations:** 1 **Copollutant (correlation):** $PM_{10-2.5}$ PM1 r = 0.19 $PM_{2.5}$ r = 0.31 PM_{10} r = 0.94 CO r = 0.32 Temperature r = 0.11	**PM Increment:** 25 µg/m^3 RR Estimate [Lower CI, Upper CI]; lag: ER visits: $PM_{10-2.5}$ All Respiratory Lag 1: 1.01 [0.98, 1.04] Lag 2: 1.01 [0.98, 1.04] Lag 3: 1.02 [0.99, 1.05] Acute Asthma Lag 1: 1.03 [0.98, 1.08] Lag 2: 1.01 [0.96, 1.07] Lag 3: 0.99 [0.94, 1.05] COPD (adult) Lag 1: 1.01 [0.93, 1.09] Lag 2: 0.98 [0.90, 1.06] Lag 3: 1.02 [0.95, 1.10]
Reference: Chen et al. (2005) **Period of Study:** Jun 1, 1995–Mar 31, 1999 **Location:** Vancouver area, BC	Hospital Admissions **Outcome (ICD-9):** Acute respiratory infections (460-466), upper respiratory tract infections (470-478), pneumonia and influenza (480-487), COPD and allied conditions (490-496), other respiratory diseases (500-519) **Age Groups:** >65 yrs **Study Design:** Time series **N:** 12,869 **Statistical Analyses:** GLM **Covariates:** Temp and relative humidity **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 1, 2, 3, 4, 5, 6, and 7-day avg	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 5.6 (0.1-24.6) SD = 3.6 **Monitoring Stations:** 13 **Copollutant (correlation):** $PM_{2.5}$: r = 0.38 PM_{10}: r = 0.83 COH: r = 0.63 CO: r = 0.53 O_3: r = -0.13 NO_2: r = 0.54 SO_2: r = 0.57 Other variables: Mean temp: r = 0.13 Rel humidity: r = -0.27	**PM Increment:** 4.2 µg/m^3 RR Estimate [CI]: Adj for weather conditions Overall admission 1-day avg: 1.03 [1.00,1.06] 2-day avg: 1.05 [1.02,1.08] 3-day avg: 1.06 [1.02,1.09] Adj for weather conditions and copollutants Overall admission 1-day avg: 1.02 [0.98,1.06] 2-day avg: 1.05 [1.01,1.10] 3-day avg: 1.06 [1.02,1.11] Notes: RR's were also provided for lags 4-7 in Table 3, yielding similar results
Reference: Lin et al. (2005) **Period of Study:** 1998-2001 **Location:** Toronto, North York, East York, Etobicoke, Scarborough, and York (Canada)	Hospital Admissions **Outcome (ICD-9):** Respiratory infections including laryngitis, tracheitis, bronchitis, bronchiolitis, pneumonia, and influenza (464, 466, 480-487) **Age Groups:** 0-14 yrs **Study Design:** Bidirectional case-crossover **N:** 6782 respiratory infection hospitalizations **Statistical Analyses:** Conditional logistic regression (Cox proportional hazards model) **Covariates:** Daily mean temp and dew point temp **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS 8.2 PHREG procedure **Lags Considered:** 1-7 day averages	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 10.86 (0-45.00) SD = 5.37 **Monitoring Stations:** 4 **Copollutant (correlation):** $PM_{2.5}$: r = 0.33 PM_{10}: r = 0.76 CO: r = 0.06 SO_2: r = 0.29 NO_2: r = 0.40 O_3: r = 0.30	**PM Increment:** 6.5 µg/m^3 OR Estimate [CI]: Adjusted for weather 4 day avg: 1.16 [1.07,1.26] 6 day avg: 1.21 [1.10,1.32] Adj for weather and other gaseous pollutants 4 day avg: 1.13 [1.03,1.23] 6 day avg: 1.17 [1.06,1.29] Notes: OR's were also categorized into "Boys" and "Girls," yielding similar results

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lin et al. (2002) **Period of Study:** Jan 1, 1981–Dec 31, 1993 **Location:** Toronto	Hospital Admissions **Outcome (ICD-9)**: Asthma (493) **Age Groups:** 6-12 yrs **Study Design:** Uni- and bi-directional case-crossover (UCC, BCC) and time-series (TS) **N:** 7,319 asthma admissions **Statistical Analyses:** Conditional logistic regression, GAM **Covariates:** Maximum and minimum temp, avg relative humidity **Season:** Apr-Sep, Oct-Mar **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1-7 day averages	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 6 days (predicted daily values) **Mean (min-max):** 12.17 (0-68.00) SD = 7.55 **Monitoring Stations:** 1 **Copollutant (correlation):** $PM_{2.5}$: r = 0.44 PM_{10}: r = 0.83 CO: r = 0.17 SO_2: r = 0.28 NO_2: r = 0.38 O_3: r = 0.56	**PM Increment:** 8.4 µg/m^3 **RR Estimate [CI]:** Adj for weather and gaseous pollutants BCC 5 day avg: 1.14 [1.01,1.28] BCC 6 day avg: 1.17 [1.03,1.33] TS 5 day avg: 1.14 [1.05,1.23] TS 6 day avg: 1.15 [1.06,1.25] Boys–adj for weather UCC 1 day avg: 1.08 [1.01,1.16] UCC 2 day avg: 1.08 [0.99,1.17] BCC 1 day avg: 1.06 [1.00,1.14] BCC 2 day avg: 1.06 [0.98,1.14] TS 1 day avg: 1.08 [1.03,1.12] TS 2 day avg: 1.07 [1.01,1.13] Girls–adj for weather UCC 1 day avg: 1.07 [0.97,1.18] UCC 2 day avg: 1.16 [1.03,1.31] BCC 1 day avg: 0.98 [0.90,1.07] BCC 2 day avg: 1.05 [0.94,1.16] TS 1 day avg: 1.00 [0.94,1.06] TS 2 day avg: 1.05 [0.98,1.13] **Notes:** The author also provides RR using UCC, BCC, and TS analysis for female and male groups for days 3-7, yielding similar results
Reference: Yang et al., (2004c) **Period of Study:** Jun 1, 1995–Mar 31, 1999 **Location:** Vancouver area, British Columbia	Hospital Admissions **Outcome (ICD-9):** Respiratory diseases (460-519), pneumonia only (480-486), asthma only (493) **Age Groups:** 0-3 yrs **Study Design:** Case control, bidirectional case-crossover (BCC), and time series (TS) **N:** 1610 cases **Statistical Analyses:** Chi-square test, Logistic regression, GAM (time-series), GLM with parametric natural cubic splines **Covariates:** Gender, socioeconomic status, weekday, season, study year, influenza epidemic month **Season:** Spring, summer, fall, winter **Dose-response Investigated?** No **Statistical Package:** SAS (Case control and BCC), S-Plus (TS) **Lags Considered:** 0-7 days	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 5.6 (0-24.6) SD = 3.6 **Monitoring Stations:** NR (data obtained from Greater Vancouver Regional District Air Quality Dept) **Copollutant (correlation):** $PM_{2.5}$: r = 0.39 PM_{10}: r = 0.83 CO: r = 0.33 O_3: r = -0.16 NO_2: r = 0.37 SO_2: r = 0.54	**PM Increment:** 4.2 µg/m^3 (IQR) OR Estimate [CI]: 3-day lag 1.12 [0.98,1.28] Adj for gaseous pollutants: 1.22 [1.02,1.48] Notes: Author states that ORs for $PM_{10-2.5}$ increased with lag time up to 3 days for both single and multiple-pollutant models. More adjusted ORs and RRs are provided in Fig 1.

Table E-15. Short-term exposure to $PM_{2.5}$ (including PM components/sources) and emergency department visits and hospital admissions for respiratory outcomes.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Andersen et al. (2008b) **Period of Study:** May 2001 - December 2004 **Location:** Copenhagen, Denmark	**Outcome (ICD-10):** RD, including chronic bronchitis (J41–42), emphysema (J43), other chronic obstructive pulmonary disease (J44), asthma (J45), and status asthmaticus (J46). Pediatric hospital admissions for asthma (J45) and status asthmaticus (J46). **Age Groups: >** 5–18 years (asthma) **Study Design:** Time series **N (Specify units):** NR **Statistical Analyses:** Poisson GAM **Covariates:** Temperature, dew-point temperature, long-term trend, seasonality, influenza, day of the week, public holidays, school holidays (only for 5–18 year olds), pollen (only for pediatric asthma outcome) **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R statistical software (gam procedure, mgcv package) **Lags Considered:** Lag 0-5 days, 4-day pollutant avg (lag 0-3) for CVD, 5-day avg (lag 0-4) for RD, and a 6-day avg (lag 0-5) for asthma.	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean µg/m3 (SD; median; IQR; 99th percentile):** 10 (5; 9; 7–12; 28) **Monitoring Stations:** 1 **Copollutant (correlation):** NCtot: r = 0.40 NC100: r = 0.29 NCa12: r = 0.07 Nca23: r = -0.25 NCa57: r = 0.51 NC_{a212}: r = 0.82 PM_{10}: r = 0.80 CO: r = 0.46 NO_2: r = 0.42 Nox: r = 0.40 No_x curbside: r = 0.28 O_3: r = -0.20 **Other variables:** Temperature: r = -0.01 Relative humidity:r = 0.21	**PM Increment:** 5 µg/m³ (IQR) **Relative risk (RR) Estimate [CI]:** RD hospital admissions (5 day avg, lag 0 -4), age 65+: One-pollutant model: 1.00 [0.95–1.00] Adj for NCtot: 1.00 [0.95–1.06] Asthma hospital admissions (6 day avg lag 0–5), age 5 - 18: One-pollutant model: 1.15 [1.00–1.32] Adj for NCtot: 1.13 [0.98–1.32] Estimates for individual day lags reported only in figure form (see notes): **Notes:** RD: No statistically or marginally significant associations. Positive associatons at Lag 4–5.Asthma: Wide confidence intervals make interpretation dificult. Positive associations at Lag 1, 2, 3.
Reference: Babin et. al. (2007) **Period of Study:** 10/2001-9/2004 **Location:** Washington, DC	ED Visit/Admissions **Outcome:** Asthma–493 **Age Groups:** 1-17 years,1-4, 5-12, 13-17 **Study Design:** Time-series **N:** NR **Statistical Analyses:** Poisson regression, spline w/ 12 knots to adjust for long term trend **Covariates:** Temperature, mold, pollen, seasonal trends, **Season:** All **Dose-response Investigated?**No **Statistical Package:** STATA **Lags Considered:** 0-4	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-hs **Mean:** "low, never reached code red" **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** 3 **Copollutant (correlation):** NR	**PM Increment:** 1 µg/m³ %Change ED Visits Ages 5-12: -0.2 (-0.6,0.2), lag 0 % Change ED Admissions: Ages 5-12: -0.4 (-1.6,0.8), lag 0 Ages 1-17: 0.2 (-0.6,1.1), lag 0 AR Estimate [Lower CI, Upper CI]; lag: NR Notes: No significant interactions between PM and ozone or other covariates were observed.
Reference: Bell et al. (2008b) **Period of Study:** 1995 - 2002 **Location:** Taipei, Taiwan	**Outcome (ICD-9):** Hospital admissions for asthma (493), and pneumonia (486). **Age Groups:** All **Study Design:** Time series **N (Specify units):** 19,966 hospital admissions for pneumonia, and 10,231 for asthma **Statistical Analyses:** Poisson regression **Covariates:** Day of the week, time, apparent temperature, long-term trends, seasonality **Season:** All **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** lags 0-3 days, mean of lags 0-3	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (range; IQR):** 31.6 (0.50–355.0; 20.2) **Monitoring Stations:** 2 **Copollutant (correlation):** NR	**PM Increment:** 20 µg/m³ (near IQR) **Percentage increase estimate [95% CI]:** Asthma: L0: 0.46 (-2.41, 3.42) L1: -1.36 (-4.33, 1.71); L2: -0.83 (-3.67, 2.10) L3: -0.78 (-3.63, 2.16); L03: -1.75 (-6.21, 2.92) Pneumonia: L0: 0.06 (-2.74, 2.94) L1: 0.34 (-2.446, 3.20); L2: -0.59 (-3.38, 2.29) L3: -0.44 (-3.22, 2.41); L03: -0.61 (-4.87, 3.85)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
References: Bell et al. (2008a) **Period of Study:** 1999 - 2005 **Location:** 202 US counties	**Outcome (ICD-9):** COPD (490–492), respiratory tract infections (464 - 466, 480 - 487) **Age Groups:** 65+ **Study Design:** Time series **N (Specify units):** NR **Statistical Analyses:** Two-stage Bayesian hierarchical model to find national avg First stage: Poisson regression (county-specific) **Covariates:** day of the week, temperature, dew point temperature, temporal trends, indicator for persons 75+ years, population size **Season:** All, June–August (Summer), September–November (Fall), December–February (Winter), March–May (Spring) **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0–2 day lags	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean** (µg/m^3): Descriptive information presented in Figure S2 (boxplots): IQR: 8.7 µg/m^3 **Monitoring Stations:** NR **Copollutant (correlation):** NR	**PM Increment:** 10 µg/m^3 **Percent increase [95% PI]: Respiratory admissions:** Lag 0 (all seasons): 0.22 [-0.12–0.56] Lag 0 (winter, national): 1.05 [0.29–1.82] Lag 0 (winter, northeast): 1.76 [0.60–2.93] Lag 0 (winter, southeast): 0.59 [-1.35–2.58] Lag 0 (winter, northwest): -0.07 [-6.74–7.08] Lag 0 (winter, southwest): 0.03 [-1.25–1.34] Lag 0 (spring, national): 0.31 [-0.47–1.11] Lag 0 (spring, northeast): 0.34 [-0.66–1.34] Lag 0 (spring, southeast): -0.06 [-1.77–1.68] Lag 0 (spring, northwest): -8.52 [-25.62–12.51] Lag 0 (spring, southwest): 1.87 [-2.00–5.90] Lag 0 (summer, national): -0.62 [-1.33–0.09] Lag 0 (summer, northeast): -0.8 [-1.65–0.07] Lag 0 (summer, southeast): -0.15 [-1.88–1.61] Lag 0 (summer, northwest): 0.25 [-21.46–27.96] Lag 0 (summer, southwest): 0.64 [-5.38–7.04] Lag 0 (autumn, national): 0.02 [-0.63–0.67] Lag 0 (autumn, northeast): -0.01 [-0.87–0.85] Lag 0 (autumn, southeast): -0.58 [-2.06–0.91] Lag 0 (autumn, northwest): -1.38 [-11.84–10.32] Lag 0 (autumn, southwest): 1.77 [-0.73–4.33] Lag 1 (all seasons): 0.05 [-0.29–0.39] Lag 1 (winter): 0.50 [-0.27–1.27] Lag 1 (spring): -0.24 [-1.01–0.53] Lag 1 (summer): 0.28 [-0.39–0.95] Lag 1 (autumn): 0.15 [-0.49–0.79] Lag 2 (all seasons): 0.41 [0.09–0.74] Lag 2 (winter, national): 0.72 [0.01–1.43] Lag 2 (winter, northeast): 0.79 [-0.21–1.80] Lag 2 (winter, southeast): 0.4 [-1.45, 2.27] Lag 2 (winter, northwest): -0.06 [-6.52–6.85] Lag 2 (winter, southwest): 1.2 [-0.10–2.52] Lag 2 (spring, national): 0.35 [-0.29–0.99] Lag 2 (spring, northeast): 0.04 [-0.88–0.97] Lag 2 (spring, southeast): 0.75 [-0.82–2.34] Lag 2 (spring, northwest): 2.29 [-14.26–22.03] Lag 2 (spring, southwest): 1.05 [-2.18–4.39] Lag 2 (summer, national): 0.57 [-0.07–1.23] Lag 2 (summer, northeast): 0.77 [-0.01–1.56] |Lag 2 (summer, southeast): -0.52 [-2.07–1.06] Lag 2 (summer, northwest): 0.74 [-18.73–24.86] Lag 2 (summer, southwest): 2.41 [-2.61–7.69] Lag 2 (autumn, national): 0.39 [-0.22–1.01] Lag 2 (autumn, northeast): 0.12 [-0.82–1.07] Lag 2 (autumn, southeast): 0.14 [-1.29–1.59] Lag 2 (autumn, northwest): -0.74 [-10.08–9.58] Lag 2 (autumn, southwest): 0.97[-1.36–3.36]
Reference: Chardon et al. (2007) **Period of Study:** 2000-2003 **Location:** Greater Paris Area, France	Doctors house calls **Outcome (ICPC2):** Asthma (R96), Upper respiratory disease (URD R07, R21, R29, R75, R76, R02) , Lower respiratory disease (LRD, R05, R78) **Age Groups:** all **Study Design:** Time series **N:** 8027 for asthma; 52928 for LRD; 74845 for URD **Statistical Analyses:** Quasi-Poisson, GAM, parametric penalized spline smoothers. **Covariates:** Lagged and current temperature, humidity, long term trends, seasonality, pollen counts, influenza epidemic, days of the week, holidays, bank holidays **Season:** All **Dose-response Investigated?** No **Statistical Package:** R **Lags Considered:** 0-3 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** mean of the daily means **Mean (SD):** 14.7(7.34) µg/m^3 **Percentiles**: 25th: 9.5 50th(Median): 12.9 75th: 18.2 **Range (Min, Max):** (3, 69.6) **Monitoring Stations**:1- 4 **Copollutant:** PM_{10}: r = 0.95 NO_2: r = 0.68	**PM Increment:** 10 µg/m^3 % Change, lag 0-3 d avg URD 6.0 (3.1, 9.1) LRD 5.8 (2.8, 8.9) Asthma 4.4 (-1.3, 10.4)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Dominici et al. (2006) **Period of Study:** 1999 - 2002 **Location:** 204 US counties, located in: Alabama, Alaska, Arizona, Arkansas, California, Colorado, Connecticut, Delaware, District of Columbia, Florida, Georgia, Hawaii, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin	**Outcome (ICD-9:** Daily counts of hospital admissions for primary diagnosis of chronic obstructive pulmonary disease (490–492), and respiratory tract infections (464–466, 480–487). **Age Groups:** >65 years **Study Design:** Time series **N (Specify units):** 11.5 million Medicare enrollees **Statistical Analyses:** Bayesian 2-stage hierarchical models. First stage: Poisson regression (county-specific) Second stage: Bayesian hierarchical models, to produce a national avg estimate **Covariates:** Day of the week, seasonality, temperature, dew point temperature, long-term trends **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R statistical software version 2.2.0 **Lags Considered:** 0-2 days**,** avg of days 0-2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (**µg/m^3 **) (IQR):** 13.4 (11.3–15.2) **Monitoring Stations:** NR **Copollutant (correlation):** NR **Other variables:** Median of pairwise correlations among $PM_{2.5}$ monitors within the same county for 2000: r = 0.91 (IQR: 0.81-0.95)	**PM Increment:** 10 µg/m^3 (Results in figures; see notes) **Percent increase in risk [95% PI]: COPD (Lag 0):** Age 65 +: 0.91 [0.18, 1.64] Age 65–74: 0.42 [-0.64, 1.48]; Age 75+: 1.47 [0.54, 2.40] **Respiratory tract infection:** Age 65+: 0.92 [0.41, 1.43] Age 65–74: 0.93 [0.04, 1.82]; Age 75+: 0.92 [0.32, 1.53] **Annual reduction in admissions attributable to a 10 µg/m^3 reduction in daily PM2.5 level (95% PI):** **Cerebrovascular disease:** Annual number of admissions: 226,641 Annual reduction in admissions: 1836 [680, 2992] **COPD:** Annual number of admissions: 108,812 Annual reduction in admissions: 990 [196, 1785] **Respiratory tract infections:** Annual number of admissions: 226,620 Annual reduction in admissions: 2085 [929, 3241]
Reference: El-Zein et al. (2007) **Period of Study:** 2000-2004 **Location:** Beirut, Lebanon	ED Admissions **Outcome:** Acute respiratory symptoms: asthma, URTI, pneumonia, bronchitis **Age Groups:** <17 **Study Design:** Ecological (natural experiment comparing admissions before and after ban on diesel fuel) **N:** 5 hospitals, 7573 admissions Oct-Feb, 4303 admissions Oct-Dec **Statistical Analyses:** t-test, Poisson regression **Covariates:** Month of Year, temperature, humidity, orthogonalized rainfall **Season:** Oct-Dec (excluding flu season) and Oct-Feb **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1-2 years before the ban compared to 1-2 years after the ban	**Pollutant:** PM from diesel **Range (Min, Max):** NR **PM Component:** NR **Monitoring Stations:** 1 **Notes:** Did not look at specific exposure data; looked at outcome with respect to a timeline that plotted admissions before and after a ban on diesel fuel. **Copollutant:** NR	**PM Increment:** NA β (p-value): **2 years pre-ban vs. 2 years post-ban** Oct to Feb All Resp: 0.128 (0.32) ; Asthma: -0.176 (0.16) ; Bronchitis: 0.505 (0.02) ; Pneumonia: 0.287 (0.17) ; URTI: -0.265 (0.41) Oct to Dec All Resp: -0.022 (0.87) ; Asthma: -0.21 (0.07) ; Bronchitis: 0.2 (0.35) ; Pneumonia: -0.065 (0.78) ; URTI: -0.628 (0.05) **2 years pre-ban vs. 1 year post-ban** Oct–Feb All Resp: -0.093 (0.45) ; Asthma: -0.208 (0.05) ; Bronchitis: 0.286 (0.32) ; Pneumonia: -0.07 (0.76) ; URTI: -0.715 (0.11) Oct to Dec All Resp: -0.147 (0.02) ; Asthma: -0.147 (0.00) ; Bronchitis: -0.011 (0.96) ; Pneumonia: -0.214 (0.15) ; URTI: -0.885 (0.06) **1 years pre-ban vs. 1 year post-ban** Oct–Feb All Resp: -0.165 (0.04) ; Asthma: -0.212 (0.09) ; Bronchitis: 0.059 (0.85) ; Pneumonia: -0.034 (0.84) ; URTI: -1.023 (0.00) Oct to Dec All Resp: -0.17 (0.00) ; Asthma: -0.131 (0.00) ; Bronchitis: -0.145 (0.001) ; Pneumonia: -0.168 (0.12) ; URTI: -1.036 (0.00)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Fung et al. (2006) **Period of Study:** 6/1/95–3/31/99 **Location:** Vancouver, Canada	Hospital Admission/ED: Hospital Admission **Outcome:** Respriatory diseases (460-519) **Age Groups:** Age >65 **Study Design:** Time series, case crossover **N:** 26,275 individuals admitted **Statistical Analyses:** Poisson regression (spline 12 knots), case-crossover (controls +/7 d days from case date), Dewanji and Moolgavkar (DM) method **Covariates:** Long-term trends, day-of-the-week effect, weather **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SPlus, R **Lags Considered:** 0-7 d	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h Avg **Mean (SD):** 7.72(3.61) **Range (Min, Max):** (2, 32) **Monitoring Stations:** NR **Copollutant (correlation):** $PM_{2.5}$: PM_{10}; r = 0.80 $PM_{10-2.5}$; r = 0.34 CO; r = 0.23 CoH; r = 0.38 O_3; r = -0.03 NO_2; r = 0.36 SO_2; r = 0.42	**PM Increment::** 4 µg/m^3 **RR Estimate (65+ years)** DM method: 1.007[0.994, 1.020]; Current 1.007[0.990,1.023]; 3 day 0.995[0.979,1.012]; 5 day 0.995[0.971,1.020]; 7 day Time series: 1.003[0.989, 1.018]; Current 1.000[0.982, 1.018]; 3 day 0.993[0.972, 1.014]; 5 day 0.995[0.971, 1.020]; 7 day Case-crossover: 1.002[0.986, 1.019]; Current 1.001[0.981, 1.021]; 3 day 0.988[0.966, 1.011]; 5 day 0.984[0.959, 1.010]; 7 day
Reference: Hinwood et al. (2006) **Period of Study:** 1/1992-12/1998 **Location:** Perth, Australia	Hospital Admission **Outcome (ICD-9):** COPD (490-496.99, except asthma), pneumonia /influenza (480-489.99), asthma **Age Groups:** All ages **Study Design:** Time stratified case-crossover **N:** NR **Statistical Analyses:** Conditional logistic regression **Covariates:** Time trend, season, temperature, humidity, day of wk, holidays **Season:** All year **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0-3 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h Avg **Mean (SD):** 9.2 (4.3) **Percentiles:** 10th: 5.0 90th: 14.5 **Monitoring Stations:** 13 **Notes: Copollutant:** NR	**Increment:** 1 µg/m^3 **Notes:** Odds ratio for $PM_{2.5}$ and all respiratory, COPD, pneumonia and asthma. Authors found an elevation in the odds ratio for lags 2 and 3 reaaching significance in all age groups for lag 3. For each increase of 1 µg/m^3, the number of hospitalizations increases 0.2% for respiratory disease, 0.5% for pneumonia and 0.3% for asthna. $PM_{2.5}$ concentrations were also significantly associated with asthma for those aged under 15 years with an estimated 0.5% increase in hospitalizations.
Reference: Host et al. (2007) **Period of Study:** 2000 - 2003 **Location:** Six French cities: Le Havre, Lille, Marseille, Paris, Rouen, and Toulouse	**Outcome (ICD-10):** Daily hospitalizations for all respiratory diseases (J00–J99), respiratory infections (J10–J22). **Age Groups:** For all respiratory diseases: 0–14 years, 15–64 years, and ≥ 65 years. For respiratory infections: All ages **Study Design:** Time series **N:** NR (Total population of cities: approximately 10 million) **Statistical Analyses:** Poisson regression **Covariates:** Seasons, days of the week, holidays, influenza epidemics, pollen counts, temperature, and temporal trends **Season:** NR **Dose-response Investigated:** No **Statistical Package:** MGCV package in R software (R 2.1.1) **Lags Considered:** Avg of 0-1 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean** (5th -95th percentile): Le Havre: 13.8 (6.0–30.5) Lille: 15.9 (6.9–26.3) Marseille: 18.8 (8.0–33.0) Paris: 14.7 (6.5–28.8) Rouen: 14.4 (7.5–28.0) Toulouse: 13.8 (6.0–25.0) **Monitoring Stations:** 13 total: 1 in Toulouse 4 in Paris 2 each in other cities **Copollutant (correlation):** $PM_{10-2.5}$: Overall: r > 0.6 Ranged between r = 0.28 and r = 0.73 across the six cities.	**PM Increment:** 10 µg/m^3 increase, and a 27 µg/m^3 increase (corresponding to the difference between the lowest of the 5th percentiles and the highest of the 95th percentiles of the cities' distributions) **ERR (excess relative risk) Estimate [CI]:** For all respiratory diseases (27 µg/m^3 increase): 0–14 years: 1.1% [-3.1, 5.5]; 15–64 years: 2.2% [-1.8, 6.4] ; ≥ 65 years: 1.3% [-5.3, 8.2] For respiratory infections (10 µg/m^3 increase): All ages: 2.5% [0.1, 4.8] For respiratory infections (27 µg/m^3 increase): All ages: 7.0% [0.7, 13.6]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ko et al. (2007b) **Period of Study:** 1/2000-12/2004 **Location:** Hong Kong, China	ED Visits **Outcome (ICD-9):** COPD: Chronic bronchitis (491), Emphysema (492), Chronic airway obstruction (496) **Age Groups:** All ages **Study Design:** Time series **N:** 15 hospitals, 119,225 admissions **Statistical Analyses:** Poisson regression, GAM with stringent convergence criteria, APHEA2 protocol. **Covariates:** Time trend, season, temperature, humidity, other cyclical factors, day, day of wk, holidays **Season:** All year, interactions with season tested **Dose-response Investigated?** No **Statistical Package:** SPLUS 4.0 **Lags Considered:** 0-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** 35.7 (20.6) **Percentiles:** 25th: 19.4 50th(Median): 31.7 75th: 46.7 **Range (Min, Max):** (6.0, 163.2) **Monitoring Stations:** 14 **Copollutant (correlation):** $PM_{2.5}$: PM_{10}; r = 0.952 NO_2; r = 0.441 O_3; r = 0.394 SO_2; r = 0.282	**PM Increment:** PM_{10} **RR Estimate** COPD 1.002[0.998, 1.001]; lag 0 1.003[0.999, 1.007]; lag 1 1.011[1.007, 1.014]; lag 2 1.013[1.010, 1.017]; lag 3 1.011[1.008, 1.015]; lag 4 1.009[1.006, 1.013]; lag 5 1.004[0.999, 1.008]lag 0-1 1.010[1.006, 1.015]lag 0-2 1.018[1.013, 1.022]lag 0-3 1.024[1.019, 1.029]lag 0-4 1.031[1.026, 1.036]lag 0-5 4-Pollutant model: 1.014[1.007, 1.022]; lag 0-5 3-Pollutant model: 1.011[1.004, 1.017]; lag 0-5
Reference: Ko et al. (2007a) **Period of Study:** 1/2000-12/2005 **Location:** Hong Kong, China	Hospital Admission **Outcome (ICD-9):** Asthma (493) **Age Groups:** All, 0-14, 15-56, 65+ **Study Design:** Time series **N:** 69,716 admissions, 15 hospitals **Statistical Analyses:** Poisson regression, with GAM with stringent convergence criteria. **Covariates:** Time trend, season, temperature, humidity, other cyclical factors **Season:** All year, evaluated effect of season in analysis **Dose-response Investigated?** No **Statistical Package:** SPLUS 4.0 **Lags Considered:** 0-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** 36.4 (21.1) **Percentiles**: 25th: 20.0 50th(Median): 32.5 75th: 47.7 **Range (Min, Max):** (6, 163) **Monitoring Stations:** 14 **Copollutant (correlation):** $PM_{2.5}$: PM_{10}; r = 0.956 NO_2; r = 0.774 O_3; r = 0.585 SO_2; r = 0.482	**PM Increment:** 10.0 µg/m^3 **RR Estimate** Asthma (Single-pollutant model): 1.008[1.004, 1.013]; lag 0 ; 1.004[1.000, 1.009]; lag 1 ; 1.004[1.000, 1.009]; lag 2 ; 1.009[1.005, 1.014]; lag 3 ; 1.006[1.001, 1.011]; lag 4 ; 1.002[0.998, 1.007]; lag 5 ; 1.009[1.004, 1.014]; lag 0-1 ; 1.012[1.007, 1.018]; lag 0-2 ; 1.017[1.011, 1.022]; lag 0-3 ; 1.020[1.014, 1.026]; lag 0-4 ; 1.021[1.015, 1.028]; lag 0-5 Asthma in Age 0-14: 1.024[1.013, 1.034]; lag 0-5 14-65: 1.018[1.008, 1.029]; lag 0-5 >65: 1.021[1.012, 1.030]; lag 0-4 Asthma–Cold Season: 1.139[1.043, 1.244] lag 0-5
Reference: Lee et al. (2006) **Period of Study:** 1/1997-12/2002 **Location:** Hong Kong, China	Hospital Admission **Outcome:** Asthma (493) **Age Groups:** <18 years **Study Design:** Time series **N:** 26,663 asthma admissions for asthma and 5821 admissions for influenza **Statistical Analyses:** Poisson regression, GAM **Covariates:** Temperature, atmospheric pressure, relative humidity **Season:** All **Dose-response Investigated?** No **Statistical Package:** SAS 8.02 **Lags Considered:** 0-5 **Notes:** Controls were admissions for influenza ICD9 487	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-hs **Mean (SD):** 45.3 µg/m^3, (16.2) **Percentiles:** 25th: 33.4 50th(Median): 43.0 75th: 54.0 **Range (Min, Max):** NR **Monitoring Stations:** 10 **Copollutant (correlation):** $PM_{2.5}$-PM_{10}: 0.89 $PM_{2.5}$-SO_2: 0.48 $PM_{2.5}$-NO_2: 0.74 $PM_{2.5}$-O_3: 0.47	**PM Increment:** IQr = 20.6 µg/m^3 Percent increase: Single pollutant model: 5.10 [2.95, 7.30], lag 0 5.00 [2.88, 7.16], lag 1 5.48 [2.75, 6.95], lag 2 4.83 [2.78, 6.93], lag 3 6.59 [4.51, 8.72], lag 4 5.24 [3.18, 7.34], lag 5 Multipollutant model (SO_2, NO_2, CO, O_3) 3.24 [0.93, 5.60], lag 4

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Peel et al. **Period of Study:** Jan 1993-Aug 2000 **Location:** Atlanta, Georgia	ED visits **Outcome:** Asthma (493, 786.09); COPD (491, 492, 496); URI (460-466, 477); Pneumonia (480-486) **Age Groups:** All ages. Secondary analyses conducted by age group: 0-1, 2-18, >18 **Study Design:** Time series **N:** 31 hospitals **Statistical Analyses:** Poisson GEE for URI, asthma and all RD; Poisson GLM for pneumonia and COPD) **Covariates:** Avg temperature and dew point, pollen counts **Season:** All (secondary analyses of warm season) **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.3; S-Plus 2000 **Lags Considered:** 0-7 d , 3 d ma, 0-13 d unconstrained distributed lag	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h avg **Mean (SD):** 19.2 (8.9) **Percentiles:** 10th: 8.9; 90th: 32.3 **Components:** Water soluble metals, OC, EC, sulfate **Monitoring Stations:** "Several" **Notes:** $PM_{2.5}$ acidity and 24-h oxygenated hydrocarbons included in analyses **Copollutant (correlation):** PM_{10}: r = 0.84 O_3: r = 0.65 NO_2: r = 0.46 CO: r = 0.44 SO_2: r = 0.17 $PM_{10-2.5}$: r = 0.43 UF: r = -0.16 $PM_{2.5}$ components: r = 0.40 to 0.77	**PM Increment:** 10 µg/m^3 **RR Estimate [Lower CI, Upper CI], (3 d ma-0, 1, 2 d lag):** 24-h $PM_{2.5}$ All Respiratory Outcomes: 1.016 (0.997–1.035) URI: 1.018 (0.995–1.041) Asthma: 1.005 (0.977–1.033) Pneumonia: 1.011 (0.981–1.042) COPD: 1.015 (0.969–1.063) **Notes:** Infant (0-1 y) and pediatric (2-18 y) asthma was associated more strongly with PM_{10}, $PM_{2.5}$ and OC than adult asthma.
Reference: Peel et al. **Period of Study:** Jan 1993-Aug 2000 **Location:** Atlanta, Georgia	ED visits **Outcome:** Asthma (493, 786.09); COPD (491, 492, 496); URI (460-466, 477); Pneumonia (480-486) **Age Groups:** All ages. Secondary analyses conducted by age group: 0-1, 2-18, >18 **Study Design:** Time series **N:** 31 hospitals **Statistical Analyses:** Poisson GEE for URI, asthma and all RD; Poisson GLM for pneumonia and COPD) **Covariates:** Avg temperature and dew point, pollen counts **Season:** All (secondary analyses of warm season) **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.3; S-Plus 2000 **Lags Considered:** 0-7 d , 3 d ma, 0-13 d unconstrained distributed lag	**Pollutant:** $PM_{2.5}$ Components **Averaging Time:** 24 h avg Water soluble metals: **Mean (SD):**-0.28 (0.025); 10th-0.006; 90th–0.061 Sulfate: Mean (SD)-5.5 (3.7); 10th-1.9; 90th-10.7 OC: Mean (SD)–4.5 (2.2); 10th: 2.2; 90th: 7.1 EC: Mean (SD) -2.0 (1.4); 10th- 0.8; 90th- 3.7 **Monitoring Stations:** "Several" **Notes:** $PM_{2.5}$ acidity and 24-h oxygenated hydrocarbons included in analyses **Copollutant (correlation):** $PM_{2.5}$ components: r = 0.40 to 0.77	**PM Increment:** $PM_{2.5}$ 10 g/ m3 $PM_{2.5}$ Water-soluble metals 0.03 g/m^3 $PM_{2.5}$ Sulfate: 5 g/m $PM_{2.5}$ Acidity: 0.02 equ/ m3 $PM_{2.5}$ OC: 2 g/m $PM_{2.5}$ EC: ,1 g/m^3 RR Estimate [Lower CI, Upper CI] 3 d ma Water-soluble metals: All Respiratory Outcomes: 1.005 (0.981–1.031); URI: 1.010 (0.980–1.040); Asthma: 1.007 (0.973–1.043); Pneumonia: 0.997 (0.958–1.039); COPD: 0.971 (0.913–1.032) 24-h $PM_{2.5}$ Sulfate: All Respiratory Outcomes: 0.998 (0.968–1.028); URI: 1.001 (0.965–1.039); Asthma: 0.991 (0.949–1.035); Pneumonia: 1.013 (0.959–1.069); COPD: 1.004 (0.929–1.085) 24-h $PM_{2.5}$ Acidity: All Respiratory Outcomes: 1.005 (0.977–1.033) ; URI: 1.012 (0.979–1.045) ; Asthma: 0.986 (0.948–1.025) ; Pneumonia: 1.010 (0.964–1.059) ; COPD: 0.997 (0.936–1.061) 24-h $PM_{2.5}$ OC: All Respiratory Outcomes: 1.011 (0.997–1.025) URI: 1.011 (0.995–1.028); Asthma: 1.000 (0.978–1.023); Pneumonia: 1.028 (1.004–1.053); COPD: 0.996 (0.959–1.035) 24-h $PM_{2.5}$ EC: All Respiratory Outcomes: 0.999 (0.987–1.011); URI: 0.999 (0.985–1.013); Asthma: 0.993 (0.976–1.011); Pneumonia: 1.006 (0.987–1.026); COPD: 0.981 (0.952–1.012) **Notes:** Single day lag results (0-15 d) for asthma and URI presented graphically. Infant (0-1 y) and pediatric (2-18 y) asthma was associated more strongly with PM_{10}, $PM_{2.5}$ and OC than adult asthma.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Peng et al. (2008) **Period of Study:** January 1, 1999–December 31, 2005 **Location:** 108 U.S. counties in the following states: Alabama, Arizona, California, Colorado, Connecticut, District of Columbia, Florida, Georgia, Idaho, Illinois, Indiana, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Nevada, New Hampshire, New Jersey, New Mexico, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Washington, West Virginia, Wisconsin	**Outcome (ICD-9):** Emergency hospitalizations for: Respiratory disease, including COPD (490–492) and respiratory tract infections (464–466, 480-487) **Age Groups:** 65 + years, 65–74, ,75 + **Study Design:** Time series **N:** ~ 12 million Medicare enrollees (3.7 million CVD and 1.4 million RD admissions) **Statistical Analyses:** Two-stage Bayesian hierarchical models: Overdispersed Poisson models for county-specific data Bayesian hierarchical models to obtain national avg estimate **Covariates:** Day of the week, age-specific intercept, temperature, dew point temperature, calendar time, indicator for age of 75 years or older. Some models were adjusted for PM_{10}-2.5. **Season:** NR **Dose-response Investigated:** No **Statistical Package:** R version 2.6.2 **Lags Considered:** 0-2 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean** µg/m³ **(IQR):** All counties assessed: 13.5 (11.1–15.8) Counties in Eastern US: 13.8 (12.3–15.8) Counties in Western US: 11.1 (10.1–14.3) **Monitoring Stations:** At least 1 pair of co-located monitors (physically located in the same place) for PM_{10} and $PM_{2.5}$ per county **Other variables:** Median within-county correlations between monitors: r = 0.92	**PM Increment:** 10 µg/m³ **Percentage change [95% CI]:** Most values NR (see note) **Notes:** Effect estimates for PM_{10}-2.5 (0–2 day lags) are showing in Figures 2–5. Figure 3: Percentage change in emergency hospital admissions for RD per 10 µg/m³ increase in $PM_{2.5}$ (single pollutant model and model adjusted for PM_{10}-2.5 concentration)
Reference: Sarnat et al. (2008) **Period of Study:** November 1998–December 2002 **Location:** Atlanta (Georgia) metropolitan area	**Outcome (ICD-9):** Respiratory disease ED visits: asthma (493, 786.09), COPD (491, 492, 496), upper respiratory infection (460–466, 477), and pneumonia (480–486). **Age Groups:** All **Study Design:** Time series **N:** >4.5 million emergency department visits **Statistical Analyses:** Poisson generalized linear models **Covariates:** Day of the week, holidays, hospital, long-term trends, temperature, dewpoint temperature **Season:** All, warm season (April 15–October 14), and cool season (October 15–April 14). **Dose-response Investigated:** No **Statistical Package:** NR **Lags Considered:** 0-day lag	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (median; 10th-90th percentile):** Total $PM_{2.5}$: Cool season:15.8 (14.3; 7.5–25.5) Warm season: 18.2 (17.0; 9.1–29.0) $PM_{2.5}$ elemental carbon: Cool: 1.7 (1.4; 0.6–3.3) Warm: 1.4 (1.3; 0.6–2.5) $PM_{2.5}$ Zn (ng/m3): Cool: 15.7 (11.7; 4.6–30.2) Warm: 10.9 (8.5; 3.3–20.2) $PM_{2.5}$ K (ng/m3): Cool: 63.0 (53.9; 24.3–114.2) Warm: 52.7 (43.3; 23.2–93.5) $PM_{2.5}$ Si (ng/m3): Cool: 67.7 (54.1; 24.3–123.5) Warm: 110.9 (89.0; 32.9–186.3) $PM_{2.5}$ SO4(2-): Cool: 3.4 (0.6; 1.5–5.8) Warm: 6.0 (5.2; 2.3–10.8) $PM_{2.5}$ NO3-: Cool: 1.4 (1.2; 0.5–2.6) Warm: 0.7 (2.9; 0.3–1.2) $PM_{2.5}$ Se (ng/m3): Cool: 1.4 (1.1; 0.4–3.0) Warm: 1.2 (0.9; 0.4–2.7) $PM_{2.5}$ OC: Cool: 4.6 (3.9; 1.9–8.0) Warm: 4.0 (3.7; 2.1–6.4) **Monitoring Stations:** 1	**PM Increment:** IQR (specific values not given) **Risk ratio [95% CI]: RD (Lag 0):** All seasons: Total $PM_{2.5}$: 1.005 [0.996, 1.015] $PM_{2.5}$ elemental carbon: 0.996 [0.988–1.003] $PM_{2.5}$ zinc: 0.997 [0.991–1.002] $PM_{2.5}$ potassium: 1.002 [0.994–1.010] $PM_{2.5}$ silicon: 0.996 [0.990–1.003] $PM_{2.5}$ sulfate: 1.020 [1.010–1.030] $PM_{2.5}$ nitrate: 0.999 [0.991–1.006] $PM_{2.5}$ selenium: 0.998 [0.991–1.005] $PM_{2.5}$ organic carbon: 0.997 [0.990–1.005] Cool season: Total $PM_{2.5}$: 0.996 [0.978–1.015] $PM_{2.5}$ EC: 0.995 [0.982–1.008] $PM_{2.5}$ Zinc: 0.991 [0.982–1.001] $PM_{2.5}$ K: 0.998 [0.984–1.013] $PM_{2.5}$ Si: 0.986 [0.967–1.005] $PM_{2.5}$ sulfate: 0.982 [0.958–1.006] $PM_{2.5}$ nitrate: 0.992 [0.980–1.003] $PM_{2.5}$ Se: 0.999 [0.986–1.013] $PM_{2.5}$ organic carbon: 0.996 [0.984–1.008] Warm season: Total $PM_{2.5}$: 1.025 [1.012–1.039] $PM_{2.5}$ EC: 1.018 [1.003–1.032] $PM_{2.5}$ Zinc: 1.010 [0.999–1.021] $PM_{2.5}$ K: 1.011 [0.999–1.022] $PM_{2.5}$ Si: 1.000 [0.994–1.007] $PM_{2.5}$ sulfate: 1.018 [1.009–1.028] $PM_{2.5}$ nitrate: 1.018 [0.996–1.040] $PM_{2.5}$ Se: 1.001 [0.990–1.011] $PM_{2.5}$ organic carbon: 1.026 [1.010–1.041]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Sinclair and Tolsma (2004) **Period of Study:** 25 Months **Location:** Atlanta, Georgia	Outpatient Visits **Outcome:** Asthma (493); URI (460, 461, 462, 463, 464, 465, 466, 477); LRI (466.1, 480, 481, 482, 483, 484, 485, 486). **Age Groups:** < = 18 y, 18+ y (asthma); All ages (URI//LRI) **Study Design:** Times series **N:** 25 months; 260,000 to 275,000 health plan members (August 1998–August 2000) **Statistical Analyses:** Poisson GLM **Covariates:** Season, Day of week, Federal Holidays, Study Months **Season:** NR **Dose-response Investigated?**: No **Statistical Package:** SAS **Lags Considered:** Three 3 d moving averages (0-2, 2-5, 6-8)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h avg **Mean (SD):** $PM_{2.5}$–17.62 (9.32) **PM Component:** Sulfate; Acidity; EC; OC; Water-soluble metals **Monitoring Stations:** 1 **Copollutant:** NR	**PM Increment:** 9.32 (1 SD) RR Estimate [Lower CI, Upper CI]; lag: Adult Asthma: $PM_{2.5}$ = 0.906 [S]; 3-5 days lag URI: $PM_{2.5}$ = 0.965 [S]; 6-8 days lag **Notes:** Numerical findings for significant results only presented in manuscript. Results for all lags presented graphically for each outcome (asthma, URI, and LRI).
Reference: Sinclair and Tolsma (2004) **Period of Study:** 25 Months **Location:** Atlanta, Georgia	Outpatient Visits **Outcome:** Asthma (493); URI (460, 461, 462, 463, 464, 465, 466, 477); LRI (466.1, 480, 481, 482, 483, 484, 485, 486). **Age Groups:** < = 18 y, 18+ y (asthma); All ages (URI//LRI) **Study Design:** Times series **N:** 25 months; 260,000 to 275,000 health plan members (August 1998–August 2000) **Statistical Analyses:** Poisson GLM **Covariates:** Season, Day of week, Federal Holidays, Study Months **Season:** NR **Dose-response Investigated?**: No **Statistical Package:** SAS **Lags Considered:** Three 3 d moving averages (0-2, 2-5, 6-8)	**Pollutant:** $PM_{2.5}$ Components **Averaging Time:** 24 h avg **Mean (SD):** Sulfate 5.52 (3.5); Acidity 0.02 (0.02); EC 2 (1.38); OC 4.49 (2.2); Water-soluble metals 0.03 (0.03) **Monitoring Stations**: 1 **Copollutant:** NR	**PM Increment:** NR RR Estimate [Lower CI, Upper CI]; lag: Child Asthma EC: 1.046 (S), 3-5 d lag OC: 1.046 (S), 3-5 d lag URI: Sulfate: 0.976 (S), 6-8 d lag LRI: $PM_{2.5}$ acidity: 1.13 (S), lag 0-2 d EC: 1.079 (S), lag 3-5 d OC: 1.05 (S), lag 3-5 d Water-soluble metals: 1.062 (S), lag 3-5 d **Notes:** Numerical findings for significant results only presented in manuscript. Results for all lags presented graphically for each outcome (asthma, URI, and LRI).
Reference: Tolbert et al. (2007) **Period of Study:** August 1998–December 2004 **Location:** Atlanta Metropolitan area, Georgia	**Outcome (ICD-9):** Combined RD group, including: Asthma (493, 786.07, 786.09), COPD (491, 492, 496), URI (460–465, 460.0, 477), pneumonia (480–486), and bronchiolitis (466.1, 466.11, and 466.19)) **Age Groups:** All **Study Design:** Time series **N (Specify units):** NR for 1998–2004. For 1993–2004: 10,234,490 ER visits (283,360 and 1,072,429 visits included in the CVD and RD groups, respectively) **Statistical Analyses:** Poisson generalized linear models **Covariates:** long-term temporal trends, season (for RD outcome), temperature, dew point, days of week, federal holidays, hospital entry and exit **Season:** All **Dose-response Investigated:** No **Statistical Package:** SAS version 9.1 **Lags Considered:** 3-day moving avg(lag 0 -2)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (median; IQR, range, 10th–90th percentiles):** $PM_{2.5}$: 17.1 (15.6; 11.0–21.9; 0.8–65.8; 7.9–28.8); $PM_{2.5}$ sulfate: 4.9 (3.9; 2.4–6.2; 0.5–21.9; 1.7–9.5); $PM_{2.5}$ organic carbon: 4.4 (3.8; 2.7–5.3; 0.4–25.9; 2.1–7.2); $PM_{2.5}$ elemental carbon: 1.6 (1.3; 0.9–2.0; 0.1–11.9; 0.6–3.0); $PM_{2.5}$ water-soluble metals: 0.030 (0.023; 0.014–0.039; 0.003–0.202; 0.009–0.059) **Monitoring Stations:** 1 **Copollutant (correlation):** Between $PM_{2.5}$ and: PM_{10}: r = 0.84; O_3: r = 0.62; NO_2: r = 0.47; CO: r = 0.47; SO_2: r = 0.17; PM_{10}-2.5: r = 0.47; $PM_{2.5}$ SO4: r = 0.76; $PM_{2.5}$ EC: r = 0.65; $PM_{2.5}$ OC: r = 0.70; $PM_{2.5}$ TC: r = 0.71; $PM_{2.5}$ water-sol metals: r = 0.69; OHC: r = 0.50; Between $PM_{2.5}$ SO4 and: PM_{10}: r = 0.69; O_3: r = 0.56;	**PM Increment:** $PM_{2.5}$: 10.96 µg/m³ (IQR) $PM_{2.5}$ sulfate: 3.82 µg/m³ (IQR) $PM_{2.5}$ total carbon: 3.63 µg/m³ (IQR) $PM_{2.5}$ organic carbon: 2.61 µg/m³ (IQR) $PM_{2.5}$ elemental carbon: 1.15 µg/m³ (IQR) $PM_{2.5}$ water-soluble metals: 0.03 µg/m³ (IQR) **Risk ratio [95% CI] (single pollutant models):** $PM_{2.5}$: RD: 1.005 [0.995–1.015] $PM_{2.5}$ sulfate: RD: 1.007 [0.996–1.018] $PM_{2.5}$ total carbon: RD: 1.001 [0.993–1.008] $PM_{2.5}$ organic carbon: RD: 1.003 [0.995–1.011] $PM_{2.5}$ elemental carbon: RD: 0.996 [0.989–1.004] $PM_{2.5}$ water-soluble metals: RD: 1.005 [0.995–1.015]

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)

		NO_2: r = 0.14; CO: r = 0.14;
		SO_2: r = 0.09;
		PM_{10}-2.5: r = 0.32;
		$PM_{2.5}$: r = 0.76;
		$PM_{2.5}$ EC: r = 0.32;
		$PM_{2.5}$ OC: r = 0.33;
		$PM_{2.5}$ TC: r = 0.34;
		$PM_{2.5}$ water-sol metals:
		r = 0.65; OHC: r = 0.47
		Between $PM_{2.5}$ elemental carbon and: PM_{10}: r = 0.61;
		O_3: r = 0.40; NO_2: r = 0.64;
		CO: r = 0.66; SO_2: r = 0.22;
		PM_{10}-2.5: r = 0.49;
		$PM_{2.5}$: r = 0.65;
		$PM_{2.5}$ SO4: r = 0.32;
		$PM_{2.5}$ OC: r = 0.82;
		$PM_{2.5}$ TC: r = 0.91;
		$PM_{2.5}$ water soluble metals:
		r = 0.52; OHC: r = 0.35;
		Between $PM_{2.5}$ organic carbon and: PM_{10}: r = 0.65;
		O_3: r = 0.54; NO_2: r = 0.62;
		CO: r = 0.59; SO_2: r = 0.17;
		PM_{10}-2.5: r = 0.49;
		$PM_{2.5}$: r = 0.70;
		$PM_{2.5}$ SO4: r = 0.33;
		$PM_{2.5}$ EC: r = 0.82;
		$PM_{2.5}$ TC: r = 0.98;
		$PM_{2.5}$ water-sol metals:
		r = 0.49; OHC: r = 0.37;
		Between $PM_{2.5}$ total carbon and: PM_{10}: r = 0.67;
		O_3: r = 0.52; NO_2: r = 0.65;
		CO: r = 0.63; SO_2: r = 0.19;
		PM_{10}-2.5: r = 0.51;
		$PM_{2.5}$: r = 0.71;
		$PM_{2.5}$ SO4: r = 0.34;
		$PM_{2.5}$ EC: r = 0.91;
		$PM_{2.5}$ OC: r = 0.98;
		$PM_{2.5}$ water-sol metals:
		r = 0.52; OHC: r = 0.38;
		Between $PM_{2.5}$ water-soluble metals and: PM_{10}: r = 0.73;
		O_3: r = 0.43; NO_2: r = 0.32;
		CO: r = 0.35; SO_2: r = 0.06;
		PM_{10}-2.5: r = 0.50;
		$PM_{2.5}$: r = 0.69;
		$PM_{2.5}$ SO4: r = 0.65;
		$PM_{2.5}$ EC: r = 0.52;
		$PM_{2.5}$ OC: r = 0.49;
		$PM_{2.5}$ TC: r = 0.52	

Reference: Zanobetti and Schwartz (2006) **Period of Study:** 1995-1999 **Location:** Boston, MA	Hospital Admission/ED: **Outcome:** Pneumonia (480-487) **Age Groups:** >65 y **Study Design:** Case-crossover, time stratified **N:** 24,857 for Pneumonia **Statistical Analyses:** Condition logistic regression **Covariates:** Season, long term trend, day of-the-wk, mean temperature, relative humidity, barometric pressure, extinction coefficient **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-1 **Notes:** Also looked at MI cohort	**Pollutant:** PM non-traffic **Averaging Time:** 24 h **Percentiles (pneumonia cohort):** 5th: -7.3 25th: -3.28 μg/m^3 50th(Median): -0.88 75th: 1.92 95th: 12.11 **PM Component:** BC **Monitoring Stations:** 4-5 monitors **Copollutant (correlation):** PM non-traffic: $PM_{2.5}$; r = 0.74 CO; r = -0.01 NO_2; r = 0.14 O_3; r = -0.47 BC; r = -0.01	**PM Increment:** PM non-traffic lag 0: 13.44 μg/m^3 PM non-traffic lag 0-1 avg: 10.28 μg/m^3 % change in Pneumonia: PM non-traffic -0.57 [-7.51, 6.36]; lag 0 PM non-traffic -0.94 [-7.20, 5.32]; mean lag 1

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Barnett et al. (2005) **Period of Study:** 1998-2001 **Location:** 5 Australian cities (Brisbane, Canberra, Melbourne, Perth, and Sydney) and 2 New Zealand cities (Auckland, Christchurch)	**Outcome (ICD: NR):** All respiratory admissions (including asthma, pneumonia, and acute bronchitis) **Age Groups:** Children aged <1 year, 1-4 years, and 5-14 years **Study Design**: Matched case-crossover **N:** ~2.4 million children <15 years old **Statistical Analyses:** Random effects meta-analysis **Covariates:** Temperature, current minus previous day's temperature, relative humidity, pressure, extremes of hot and cold, day of the week, public holiday, and day after public holiday **Season:** Warm (Nov-Apr) and Cool (May-Oct) **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-hs **Mean (min-max):** Auckland (A): 11.0 (2.1-37.6) Brisbane (B): 9.7 (3.2-122.8) Canberra (Ca): NR Christchurch (Ch): NR Melbourne (M): 8.9 (2.8-43.3) Perth (P): 8.1 (1.7-29.3) Sydney (S): 9.4 (2.4-82.1) **Monitoring Stations:** 1-3 per city **Copollutant:** NR	**PM Increment:** 3.8 μg/m^3 (IQR) Percent Increase Estimate [CI]: Pneumonia & Acute Bronchitis: Single Pollutant Model <1 yr (B,M,P,S): 1.7 [0.0,3.4] 1-4 yrs (B,M,P,S): 2.4 [0.1,4.7] Matched Multipollutant Model 1-4 yrs with 1-h SO_2 (B,S): 1.9 [-1.7,5.6] 1-4 yrs with temp (B,M,P,S): 2.3 [-0.4,5.1] Respiratory Admissions: Single Pollutant Model <1 yr (B,M,P,S): 2.4 [1.0,3.8] 1-4 yrs (B,M,P,S): 1.7 [0.7,2.7] Matched Pollutant Model <1 yr with 1-h SO_2 (B,S): 3.1 [0.5,5.7] <1 yr with temp (B,M,P,S): 1.8 [0.2,3.4] 1-4 yrs with PM_{10} (B,M,P,S): 2.9 [0.2,5.6] 1-4 yrs with 1-h SO_2 (B,S): 1.3 [-1.8,4.4] 1-4 yrs with 1-h NO_2 (B,M,P,S): -1.5 [-3.2,0.2] 1-4 yrs with temp (B,M,P,S,): 1.5 [-0.2,3.1]
Reference: Chimonas and Gessner (2007) **Period of Study:** January 1, 1999–June 30, 2003 **Location:** Anchorage, Alaska	**Outcome (ICD-9):** Asthma (493.0-493.9); Lower respiratory illness-LRI (466.1, 466.0, 480-487, 490, 510-511); Inhaled quick-relief medication; Steroid medication **Age Groups:** <20 years old **Study Design:** Time series **N:** 42,667 admissions **Statistical Analyses:** GEE for multivariable modeling **Covariates:** Season, serial correlation, year, weekend, temperature, precipitation, and wind speed **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SPSS (dataset), SAS (analysis) **Lags Considered:** 1 day and 1 week	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-hs and 1 week **Mean (min-max):** Daily: 6.1 (0.5-69.8) Weekly: 5.8 (1.8-45.0) **Monitoring Stations:** NR **Copollutant:** N/A	**PM Increment:** 5 μg/m^3 RR Estimate [CI]: Same Day Outpatient Asthma: 0.992 [0.964,1.024] Outpatient LRI: 0.952 [0.907,1.001] Inpatient Asthma: 0.936 [0.798,1.098] Inpatient LRI: 0.919 [0.823,1.027] Inhaled Steroid Prescriptions: 0.988 [0.902,1.083] Quick-relief Medication: 0.962 [0.901,1.028] Weekly (median increase) Outpatient Asthma: 0.983 [0.935,1.038] Outpatient LRI: 0.969 [0.874,1.075] Inpatient Asthma: 0.754 [0.513.1.109] Inpatient LRI: 0.943 [0.715,1.245] Inhaled Steroid Prescriptions: 1.018 [0.883,1.175] Quick-relief Medication: 0.978 [0.882,1.087]
Reference: Dominici et al. (2006) **Period of Study:** 1999-2002 **Location:** U.S. (mainland)	Outcome (ICD-9): Respiratory tract infections (464-466, 480-487) and Chronic Obstructive Pulmonary Disease (490-492) **Age Groups:** All >65 yrs; 65-74 yrs; >75 yrs **Study Design:** Time series **N:** 11.5 million at-risk **Statistical Analyses:** Bayesian 2-stage hierarchical models (day-to-day variation), Poisson regression (county-specific RRs) **Covariates:** Calendar time (seasonality and year), temperature, dew point **Season:** NR **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 0, 1, 2 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily or every 3 days (depending on county) **Mean:** 13.4 (IQR: 11.3-15.2) **Monitoring Stations:** NR (used data from Air Quality System database) **Copollutant:** NR	**PM Increment:** 10 μg/m^3 **Percentage Change in Hospital Admission Rates [PI]:** COPD–Same day All >65: 0.91 [0.18,1.64] 65-74 yrs: 0.42 [-0.64,1.48] >75: 1.47 [0.54,2.40] Respiratory Tract Infections–2-day lag All >65: 0.92 [0.41,1.43] 65-74 yrs: 0.93 [0.04,1.82] >75: 0.92 [0.32,1.53] **Notes:** Other lag data shown in Fig 2-4

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lin et al. (2002) **Period of Study:** Jan 1, 1981–Dec 31, 1993 **Location:** Toronto	Hospital Admissions **Outcome (ICD-9):** Asthma (493) **Age Groups:** 6-12 yrs **Study Design:** Uni- and bi-directional case-crossover (UCC, BCC) and time-series (TS) **N:** 7,319 asthma admissions **Statistical Analyses:** Conditional logistic regression, GAM **Covariates:** Maximum and minimum temp, avg relative humidity **Season:** Apr-Sep, Oct-Mar **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1-7 day averages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 6 days (predicted daily values) **Mean (min-max):** 17.99 (1.22-89.59) SD = 8.49 **Monitoring Stations:** 1 **Copollutant (correlation):** PM_{10}: r = 0.87 $PM_{10-2.5}$: r = 0.44 CO: r = 0.45 SO_2: r = 0.46 NO_2: r = 0.50 O_3: r = 0.21	**PM Increment:** 9.3 µg/m³ **RR Estimate [CI]:** Adj for weather and gaseous pollutants BCC 5 day avg: 0.94 [0.85,1.03] BCC 6 day avg: 0.92 [0.83,1.02] TS 5 day avg: 0.96 [0.90,1.02] TS 6 day avg: 0.94 [0.88,1.01] Boys–adj for weather UCC 1 day avg: 1.09 [1.04,1.15] UCC 2 day avg: 1.09 [1.02,1.16] BCC 1 day avg: 1.01 [0.97,1.06] BCC 2 day avg: 0.99 [0.93,1.05] TS 1 day avg: 1.00 [0.97,1.04] TS 2 day avg: 0.98 [0.94,1.02] Girls–adj for weather UCC 1 day avg: 1.06 [0.99,1.14] UCC 2 day avg: 1.11 [1.02,1.21] BCC 1 day avg: 0.99 [0.93,1.06] BCC 2 day avg: 1.02 [0.94,1.09] TS 1 day avg: 0.99 [0.95,1.04] TS 2 day avg: 1.00 [0.95,1.06] **Notes:** The author also provides RR using UCC, BCC, and TS analysis for female and male groups for days 3-7, yielding similar results
Reference: Slaughter et al. (2005) **Period of Study:** January 1995 through June 2001 **Location:** Spokane, WA	Hospital Admissions and ED visits **Outcome:** All respiratory (460-519); Asthma (493); COPD (491,492, 494,496); Pneumonia (480-487); Acute URI not including colds and sinusitis (464, 466, 490) **Age Groups:** All, 15+ years for COPD **Study Design:** Time series **N:** 2373 visit records **Statistical Analyses:** Poisson regression, GLM with natural splines. For comparison also used GAM with smoothing splines and default convergence criteria. **Covariates:** Season, temperature, relative humidity, day of week **Season:** All **Dose-response Investigated?:** No **Statistical Package:** SAS, SPLUS **Lags Considered:** 1 -3 d	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h avg **Range (90% of Concentrations):** 4.2-20.2 µg/m³ **Monitoring Stations:** One **Notes: Copollutant (correlation):** $PM_{2.5}$ PM_1 r = 0.95 PM_{10} r = 0.62 $PM_{10-2.5}$ r = 0.31 CO r = 0.62 Temperature r = 0.21	**PM Increment:** 10 µg/m³ **RR Estimate [Lower CI, Upper CI]; lag:** **ER visits:** $PM_{2.5}$ All Respiratory Lag 1: 1.01 [0.98, 1.04] ; Lag 2: 1.02 [0.99, 1.04] ; Lag 3: 1.02 [0.99, 1.05] Acute Asthma Lag 1: 1.03 [0.98, 1.09] ; Lag 2: 1.00 [0.95, 1.05] ; Lag 3: 1.01 [0.96, 1.06] COPD (adult) Lag 1: 0.96 [0.89, 1.04] ; Lag 2: 1.01 [0.93, 1.09] ; Lag 3: 1.00 [0.93, 1.08] **Hospital Admissions:** $PM_{2.5}$ All Respiratory Lag 1: 0.98 [0.94, 1.01] ; Lag 2: 0.99 [0.96, 1.03] ; Lag 3: 1.01 [0.98, 1.05] Asthma Lag 1: 1.01 [0.91, 1.11] ; Lag 2: 1.03 [0.94, 1.13] ; Lag 3: 1.02 [0.93, 1.13] COPD (adult) Lag 1: 0.99 [0.91, 1.08] ; Lag 2: 1.06 [0.98, 1.16] ; Lag 3: 1.03 [0.94, 1.12]
Reference: Yang Q et al. (2004c) **Period of Study:** Jun 1, 1995–Mar 31, 1999 **Location:** Vancouver area, British Columbia	Hospital Admissions **Outcome (ICD-9):** Respiratory diseases (460-519), pneumonia only (480-486), asthma only (493) **Age Groups:** 0-3 yrs **Study Design:** Case control, bidirectional case-crossover (BCC), and time series (TS) **N:** 1610 cases **Statistical Analyses:** Chi-square test, Logistic regression, GAM (time-series), GLM with parametric natural cubic splines **Covariates:** Gender, socioeconomic status, weekday, season, study year, influenza epidemic month **Season:** Spring, summer, fall, winter **Dose-response Investigated?** No **Statistical Package:** SAS (Case control and BCC), S-Plus (TS) **Lags Considered:** 0-7 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 7.7 (2.0-32.0) SD = 3.7 **Monitoring Stations:** NR (data obtained from Greater Vancouver Regional District Air Quality Dept) **Copollutant (correlation):** PM_{10}: r = 0.83 $PM_{10-2.5}$: r = 0.39 CO: r = 0.24 O_3: r = -0.03 NO_2: r = 0.37 SO_2: r = 0.43	**PM Increment:** 4.0 µg/m³ (IQR) OR Estimate [CI]: Values NR **Notes:** Author states that no significant association was found between $PM_{2.5}$ and respiratory disease hospitalizations.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Zhong et al. (2006) **Period of Study:** Apr–Oct 2002 **Location:** Cincinnati, Ohio	Hospital Admissions **Outcome (ICD-9):** Asthma (493-493.91) **Age Groups:** 1-18 yrs **Study Design:** Time series **N:** 1254 admissions **Statistical Analyses:** Poisson multiple regression, GAM **Covariates:** Season, temperature, humidity, ozone, day of the week **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** 1-5 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Apr: 12.4 (3.8) May: 13.6 (5.8) Jun: 21.6 (9.9) Jul: 25.8 (11.9) Aug: 20.3 (8.7) Sep: 19.5 (11.1) Oct: 12.8 (6.4) **Monitoring Stations:** NR (data obtained from the National Virtual Data System) **Copollutant (correlation):** NR **Notes:** Author states all pairwise correlations were insignificant	**PM Increment:** NR RR Estimate [CI]: NR **Notes:** This study focused primarily on aeroallergens and asthma visits
Reference: Wong et al. (2006) **Period of Study:** 2000-2002 **Location:** Hong Kong (8 districts)	General Practitioner Visits **Outcome (ICPC-2):** Respiratory diseases/symptoms: upper respiratory tract infections (URTI), lower respiratory infections, influenza, asthma, COPD, allergic rhinitis, cough, and other respiratory diseases **Age Groups:** All ages **Study Design:** Time series **N:** 269,579 visits **Statistical Analyses:** GAM, Poisson regression **Covariates:** Season, day of the week, climate **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 0-3 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 35.7 (9-120) SD = 16.7 **Monitoring Stations:** 1 per district **Copollutant (correlation):** PM_{10}: r = 0.94	**PM Increment:** 10 µg/m^3 RR Estimate [CI]: Overall URTI 1.021 [1.010,1.032] **Notes:** RRs are also reported for each individual general practitioner yielding similar results
Reference: Neuberger et al. (2004) **Period of Study:** 1999-2000 (1 yr period) **Location:** Vienna and Lower Austria	Hospital Admissions **Outcome (ICD-9):** Bronchitis, emphysema, asthma, bronchiectasis, extrinsic allergic alveolitis, and chronic airway obstruction (490-496) **Age Groups:** 3.0-5.9 yrs; 7-10 yrs; 65+ **Study Design:** Time series **N:** 366 days (admissions NR) **Statistical Analyses:** GAM **Covariates:** SO_2, NO, NO_2, O_3, temperature, humidity, and day of the week **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus 2000 **Lags Considered:** 0-14 days	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Maximum daily mean:** Vienna: 96.4 Rural area: 48.0 **Monitoring Stations:** NR **Copollutant (correlation):** NR	**PM Increment:** 10 µg/m^3 Log Relative Rate Estimate (p-value): Vienna Male: 2 day lag = 5.467 (0.019) Female: 3 day lag = 5.596 (0.009) Rural Male: 10 day lag = 9.893 (0.012) Female: 11 day lag = 10.529 (0.011) Association with tidal lung functioN: β = -0.987 (p-value = 0.091) **Notes:** Effect parameters with significant coefficients for respiratory health included: male sex, allergy, asthma in family, and traffic for Vienna and age, allergy, asthma in family, passive smoking, and PM fraction for the rural area. Effect parameters with significant coefficients for log asthma score were allergy, asthma in family, and rain for Vienna and allergy, asthma in family, and passive smoking for the rural area. Cross-correlation coefficients are provided in Fig 1.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lin et al. (2005) **Period of Study:** 1998-2001 **Location:** Toronto, North York, East York, Etobicoke, Scarborough, and York (Canada)	Hospital Admissions **Outcome (ICD-9):** Respiratory infections including laryngitis, tracheitis, bronchitis, bronchiolitis, pneumonia, and influenza (464, 466, 480-487) **Age Groups:** 0-14 yrs **Study Design**: Bidirectional case-crossover **N:** 6782 respiratory infection hospitalizations **Statistical Analyses:** Conditional logistic regression (Cox proportional hazards model) **Covariates:** Daily mean temp and dew point temp **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS 8.2 PHREG procedure **Lags Considered:** 1-7 day averages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 9.59 (0.25-50.50) SD = 7.06 **Monitoring Stations:** 4 **Copollutant (correlation):** $PM_{10-2.5}$: r = 0.33 PM_{10}: r = 0.87 CO: r = 0.10 SO_2: r = 0.47 NO_2: r = 0.48 O_3: r = 0.56	**PM Increment:** 7.8 µg/m^3 OR Estimate [CI]: Adjusted for weather 4 day avg: 1.11 [1.02,1.22] 6 day avg: 1.11 [1.00,1.24] Adj for weather and other gaseous pollutants 4 day avg: 0.94 [0.81,1.08] 6 day avg: 0.90 [0.76,1.07] **Notes:** OR's were also categorized into "Boys" and "Girls," yielding similar results
Reference: Letz and Quinn (2005) **Period of Study:** Oct 1, 2001–Aug 24, 2002 **Location:** San Antonio, Texas	Emergency Dept Visits **Outcome (ICD-9):** Asthma or reactive airway disease (493.0-493.9), wheezing (786.07), dyspnea (786.01-786.9), shortness of breath (786.05), bronchitis (490-496), or cough (786.2) **Age Groups:** NR (basic air force trainees) **Study Design:** Historic (retrospective) cohort **N:** 149 ED visits **Statistical Analyses:** Pearson correlation **Covariates:** NR **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SPSS **Lags Considered:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h AQI **AQI Range (min-max):** (4-109) **Monitoring Stations:** Data obtained from the Texas Commission on Environmental Quality **Copollutant (correlation):** NR	**PM Increment:** NR Correlation with Outcomes: Same-day All visits: r = 0.082 Proven asthmatic events: r = -0.042 3-day All visits: r = 0.097 Proven asthmatic events: r = 0.011
Reference: Chen et al. (2005) **Period of Study:** Jun 1, 1995–Mar 31, 1999 **Location:** Vancouver area, BC	Hospital Admissions **Outcome (ICD-9):** Acute respiratory infections (460-466), upper respiratory tract infections (470-478), pneumonia and influenza (480-487), COPD and allied conditions (490-496), other respiratory diseases (500-519) **Age Groups:** >65 yrs **Study Design:** Time series **N:** 12,869 **Statistical Analyses:** GLM **Covariates:** Temp and relative humidity **Season:** NR **Dose-response Investigated?** No **Statistical Package:** S-Plus **Lags Considered:** 1, 2, 3, 4, 5, 6, and 7-day avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (min-max):** 7.7 (2.0-32.0) SD = 3.7 **Monitoring Stations:** 13 **Copollutant (correlation):** PM_{10}: r = 0.83 $PM_{10-2.5}$: r = 0.38 COH: r = 0.39 CO: r = 0.23 O_3: r = -0.01 NO_2: r = 0.36 SO_2: r = 0.42 **Other variables:** Mean temp: r = 0.41 Rel humidity: r = -0.23	**PM Increment:** 4.0 µg/m^3 (IQR) **RR Estimate [CI]:** Adj for weather conditions Overall admission 1-day avg: 1.02 [0.99,1.05] 2-day avg: 1.02 [0.99,1.06] 3-day avg: 1.02 [0.98,1.05] Adj for weather conditions and copollutants Overall admission 1-day avg: 1.01 [0.98,1.06] 2-day avg: 1.01 [0.98,1.05] 3-day avg: 1.00 [0.96,1.04] **Notes:** RR's were also provided for lags 4-7 in Table 3, yielding similar results

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Magas et al. (2007) **Period of Study:** 2001-2003 **Location:** Oklahoma City Metro area, Oklahoma and Cleveland counties	Hospital Admission/ED: Admissions **Outcome:** Asthma 493.01-493.99 **Age Groups:** <15 yrs **Study Design:** Time series **N:** 1,270 admissions **Statistical Analyses:** Negative binomial regression **Covariates:** Temperature, humidity, pollen count, mold **Season:** All **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** 1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h avg **Mean (SD):** NR **Range (Min, Max):** NR **Monitoring Stations:** 10 **Copollutant (correlation):** NR	**Notes:** Coefficient for $PM_{2.5}$ was not significant and thus not reported.
Reference: Zanobetti and Schwartz (2006) **Period of Study:** 1995-1999 **Location:** Boston, MA	**Outcome:** Pneumonia (480-487) **Age Groups:** >65 y **Study Design:** Case-crossover, time stratified **N:** 24,857 for Pneumonia **Statistical Analyses:** Condition logistic regression **Covariates:** Season, long term trend, day of-the-wk, mean temperature, relative humidity, barometric pressure, extinction coefficient **Season:** All year **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** 0-1 **Notes:** Also looked at MI cohort	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Percentiles** (pneumonia cohort)**:** 25th: 7.23 µg/m^3 50th(Median): 11.10 75th: 16.14 **PM Component:** Black Carbon (BC), PM non-traffic **Monitoring Stations:** 4-5 monitors **Copollutant (correlation):** $PM_{2.5}$: CO; r = 0.52 NO_2; r = 0.55 O_3; r = 0.20 BC; r = 0.66 PM non-traffic; r = 0.74	**PM Increment:** $PM_{2.5}$ lag 0: 17.17 µg/m^3 $PM_{2.5}$ lag 0-1 avg: 16.32 µg/m^3 % change in Pneumonia: 6.48[1.13, 11.43]; lag 0 5.56[-0.45, 11.27]; mean lag 1
Reference: Erbas et al. (2005) **Period of Study:** Jul 1, 1989–Dec 31, 1992 **Location:** Melbourne, Australia	**Outcome (ICD):** COPD (490-492, 494, 496); Asthma (493) **Age Groups:** NR **Study Design:** Time series **N:** NR **Statistical Analyses:** GLM, GAM, Parameter Driven Poisson Regression, Transitional Regression, Seasonal-Trend decomposition based on Loess smoothing for seasonal adjustment **Covariates:** Secular trends, seasonality, relative humidity, dry bulb temp, dew point temp **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus, SAS **Lags Considered:** 0-5 days	**Pollutant:** $PM_{0.1-1}$ (API) **Averaging Time:** 24-hs **Mean (min-max):** NR **Monitoring Stations:** 9 **Copollutant (correlation):** NR	**PM Increment:** Increase from the 10th-90th percentile (value NR) **RR Estimate [CI]:** COPD GAM: 0.95 [0.91,1.00] GLM, PDM, TRM: NR Asthma NR **Notes:** This study was used to demonstrate that conclusions are highly dependent on the type of model used

Table E-16. Short-term exposure to other PM size fractions and emergency department visits and hospital admissions for respiratory outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Andersen et al. (2008b) **Period of Study:** 2001-2004 **Location:** Copenhagen, Denmark	**Outcome (ICD10):** Respiratory disease (J41-46); Asthma (J45, 46) **Age Groups:** 5-18 and >65 **Study Design:** Time-series **N:** 1327 days; ~1.5 million people at-risk **Statistical Analyses:** Poisson regression, GAM. **Covariates:** influenza epidemics, pollen, temperature, dew point, day-of-week, holiday, season. **Season:** All **Dose-response Investigated?** No **Statistical Package:** R with *gam* and *mgcv* packages. **Lags Considered:** 0-5	**Pollutant:** Number concentration (NC) of ultrafine & accumulation mode particles **Averaging Time:** 24-h **Mean particles/cm^3 (SD):** NCtot (total): 8116 (3502) 25th: 4959; 50th: 6243; 75th: 8218; 99th: 16189; IQR: 3259 NC100 (<100 nm): 6847 (2864) 25th: 5738; 50th (Median): 7358; 75th: 9645; 99th: 19895; IQR: 3907 Mean particles/cm^3 for four size modes (median diameter (nm) noted): NCa12: 493(315) NCa23: 2253 (1364) NCa57: 5104 (2687) NCa212: 6847 (2864) **Monitoring Stations:** 3 (Background, rural Background, urban Curbside, urban) **Notes:** NC exposure data available for n = 578 days. Information on distribution of 4 size modes provided in the paper. **Copollutant (correlation):** NCtot and PM_{10}: r = 0.39 NCtot and $PM_{2.5}$: r = 0.40 NCtot and NO_2: r = 0.68 PM_{10} and $PM_{2.5}$: r = 0.8 "Low or no" correlations between 4 size modes NCa212 and $PM_{2.5}$: r = 0.8 NCa212 and PM_{10}: r = 0.63 NCa57 and NO_2: r = 0.57 **Notes:** selected correlations reported in text, all correlations in annex to the manuscript	**PM Increment:** Based on the IQR, specific to metric (see below). **RR Estimate:** Single pollutant results, Asthma, (5-18 yr.s), lag 0-5: $PM_{2.5}$: 1.15 [1, 1.32], IQr = 5 NCtot: 1.07 [0.98, 1.17], IQr = 3907 NC100: 1.06 [0.97, 1.16], IQr = 3259 NCa12: 1.08 [0.99, 1.18], IQr = 342 NCa212: 1.08 [1, 1.17], IQr = 495 NCa23: 1.09 [0.98, 1.21], IQr = 1786 NCa57: 1.02 [0.94, 1.12], IQr = 3026 2-pollutant results: NCa212 w/ PM_{10}: 1.1 [0.96, 1.13], IQr = 495 NCtot w/ PM_{10}: 1.03 [0.92, 1.15] NCtot w/ $PM_{2.5}$: 1.04 [0.85, 1.28] All RD, (>65 yr.s), lag 0-4, single pollutant results: $PM_{2.5}$: 1 [0.95, 1.05] NCtot: 1.04 [1, 1.07] IQr = 3907 NC100: 1.03 [0.99, 1.07], IQr = 3259 NC12: 1.01 [0.98, 1.05], IQr = 342 NC212: 1.04 [1.01, 1.08], IQr = 495 NCa23: 0.99 [0.94, 1.03], IQr = 1786 NCa57: 1.04 [1, 1.08], IQr = 3026 2-pollutant results: NCa212 w/ PM_{10}: 1.01 [0.96, 1.07], IQr = 495 NCtot w/ $PM_{2.5}$: 0.97 [0.89, 1.05] NCtot w/ PM_{10}: 1 [0.96, 1.05] **Notes:** Multipollutant model results also included for models with 4 size modes.
Reference: Michaud et al. (2004) **Period of Study:** Jan 1997-May 2001 **Location:** Hilo, Hawaii	ED visits **Outcome:** Asthma/COPD (490-496); Respiratory Irritation (506-508) **Age Groups:** All **Study Design:** Time-series **N:** 1,561 ER visits **Statistical Analyses:** Multiple linear regression **Covariates:** Hourly temperature, minimum daily temperature, minimum daily temperature, humidity, year, month, day of the week **Season:** all **Dose-response Investigated?** No **Statistical Package:** STATA 6.0; SAS **Lags Considered:** Previous night, 1,2,3	**Pollutant:** PM_1 **Averaging Time:** 24 h avg **Mean (SD):** 1.91 (2.95) µg/m^3 **Range (Min, Max):** 0.0, 56.6 µg/m^3 **Monitoring Stations:** 2 **Notes: Copollutant (correlation):** NR	**PM Increment:** 10 µg/m^3 **RR Estimate [Lower CI, Upper CI]; lag:** Asthma, COPD (499-496): Adjusted for day, month & year: 1.11 (0.92, 1.34), 00: 00-6: 00AM 1.14 (1.03, 1.26), lag 1 1.06 (0.83, 0.94), lag 2 0.91 (0.06, 1.05), lag 3 Asthma (493, 495): Adjusted for day, month & year: 1.03 (0.90, 1.42), 00: 00-6: 00AM 1.02 (0.94, 1.21), lag 1 1.02 (0.99, 1.23), lag 2 0.97 (0.69, 1.15), lag 3 Bronchitis (490, 491): Adjusted for day, month & year: 1.02 (0.82, 1.41), 00: 00-6: 00AM 1.07 (1.18, 1.49), lag 1 0.97 (0.60, 1.34), lag 2 0.93 (0.43, 1.18), lag 3 **Notes:** Crude and estimates adjusted for month and year only also presented. **Notes:** Volcanic fog = vog

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Peel et al. (2005) **Period of Study:** Jan 1993-Aug 2000 **Location:** Atlanta, Georgia	**Hospital Admission/ED:** ED visits **Outcome:** Asthma 493, 786.09; COPD 491, 492, 496; URI 460-466, 477; Pneumonia 480-486 **Age Groups:** All ages. Secondary analyses conducted by age group: Infants 0-1 yrs; Pediatric asthma 2-18 yrs; Adults >18 yrs **Study Design:** Case-control All respiratory disease vs. finger wounds **N:** 31 hospitals; ED visits NR **Statistical Analyses:** Poisson generalized linear models; General linear models **Covariates:** Avg temperature and dew point, pollen counts **Season:** All **Dose-response Investigated?** yes **Statistical Package:** SAS 8.3; S-Plus 2000 **Lags Considered:** 0-7 days and 14 day distributed lag	**Pollutant:** UF (10-100nm) **Averaging Time:** 24 h avg **Mean (SD):** 3800 (40700) **Percentiles:** 10th: 11500 90th: 74600 **PM Component:** Oxygenated hydrocarbons (OH), sulfate, acidity, elemental carbon (EC), organic carbon (OC), water-soluble transition metals **Monitoring Stations:** "Several" **Copollutant (correlation):** PM_{10}: r = -0.13 O_3: r = -0.13 NO_2: r = 0.26 CO: r = 0.10 SO_2: r = 0.24 $PM_{2.5}$: r = -0.16 $PM_{10-2.5}$: r = 0.13	**Increment:** 30,000 #/cm^3 All Respiratory Disease 0.984 [0.968-1.000] URI 0.986 [0.966, 1.006] Asthma 0.999 [0.977, 1.021] Pneumonia 0.997 [0.953, 1.002] COPD 0.982 [0.942, 1.022]
Reference: Sinclair and Tolsma (2004) **Period of Study:** 25 Months **Location:** Atlanta, Georgia	**Outpatient Visits** **Outcome:** Asthma (493); URI (460, 461, 462, 463, 464, 465, 466, 477); LRI (466.1, 480, 481, 482, 483, 484, 485, 486). **Age Groups:** < = 18 y, 18+ y (asthma); All ages (URI//LRI) **Study Design:** Times series **N:** 25 months; 260,000 to 275,000 health plan members (August 1998–August 2000) **Statistical Analyses:** Poisson GLM **Covariates:** Season, Day of week, Federal Holidays, Study Months **Season:** NR **Dose-response Investigated?:** No **Statistical Package:** SAS **Lags Considered:** Three 3 d moving averages (0-2, 2-5, 6-8)	**Pollutant:** UF (PM_{10-100} nm) **Averaging Time:** 24 h avg **Mean (SD):** PM_{10-100} nm area ($\mu m^2/cm^3$)– 249.33 (244.09) **Monitoring Stations:** 1 **Copollutant (correlation):** NR	**PM Increment:** NR **RR Estimate [Lower CI, Upper CI]; lag:** Adult Asthma: Ultrafine PM area = 1.223 (S); 3-5 days lag URI: Ultrafine PM: = 1.041 (S); 0-2 days lag LRI: Ultrafine PM area = 1.099 (S); 6-8 days lag **Notes:** Numerical findings for significant results only presented in manuscript. Results for all lags presented graphically for each outcome (asthma, URI, and LRI).
Reference: Slaughter et al. (2005) **Period of Study:** January 1995-June 2001 **Location:** Spokane, WA	Hospital Admissions and ED visits **Outcome:** All respiratory (460-519); Asthma (493); COPD (491,492, 494,496); Pneumonia (480-487); Acute URI not including colds and sinusitis (464, 466, 490) **Age Groups:** All, 15+ years for COPD **Study Design:** Time series **N:** 2373 visit records **Statistical Analyses:** Poisson regression, GLM with natural splines. For comparison also used GAM with smoothing splines and default convergence criteria. **Covariates:** Season, temperature, relative humidity, day of week **Season:** All **Dose-response Investigated?:** No **Statistical Package:** SAS, SPLUS **Lags Considered:** 1 -3 d	**Pollutant:** PM_1 **Averaging Time:** 24 h avg **Range (90% of concentrations):** 3.3-17.6 $\mu g/m^3$ **Monitoring Stations:** One **Copollutant (correlation):** PM_1 $PM_{2.5}$ r = 0.95 PM_{10} r = 0.50 $PM_{10-2.5}$ r = 0.19 CO r = 0.63	**PM Increment:** 10 $\mu g/m^3$ **RR Estimate [Lower CI, Upper CI]; lag:** ED visits: PM_1 All Respiratory Lag 1: 1.01 [0.98, 1.04] Lag 2: 1.02 [0.99, 1.06] Lag 3: 1.02 [0.99,1.06] Acute Asthma Lag 1: 1.03 [0.97, 1.09] Lag 2: 0.99 [0.93, 1.05] Lag 3: 1.02 [0.96, 1.08] COPD (adult) Lag 1: 0.96 [0.87, 1.05] Lag 2: 1.02 [0.93, 1.12] Lag 3: 0.99 [0.90, 1.09]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Neuberger et al. (2004) **Period of Study:** 1999-2000 (1 yr period) **Location:** Vienna and Lower Austria	**Outcome (ICD-9):** Bronchitis, emphysema, asthma, bronchiectasis, extrinsic allergic alveolitis, and chronic airway obstruction (490-496) **Age Groups:** 3.0-5.9 yrs; 7-10 yrs; 65+ **Study Design:** Time series **N:** 366 days (admissions NR) **Statistical Analyses:** GAM **Covariates:** SO_2, NO, NO_2, O_3, temperature, humidity, and day of the week **Season:** NR **Dose-response Investigated?** Yes **Statistical Package:** S-Plus 2000 **Lags Considered:** 0-14 days	**Pollutant:** PM_1 **Averaging Time:** 24 h **Mean** μg/m^3 **(SD):** NR **Monitoring Stations:** NR **Copollutant (correlation):** NR	**PM Increment:** NR **Effect parameters (Vienna children):** Respiratory Health Male sex = 0.098 Allergy = 0.238 Asthma in family = 0.190 Traffic = 0.112 Log Asthma Score Allergy = 0.210 Asthma in family = 0.112 Rain = 0.257 *only significant coefficients are presented Association with tidal lung functioN: β = -1.059 (p-value = 0.060) **Notes:** No significant associations between PM and respiratory mortality were found for either sex. Data is also provided for children in the rural area where age, allergy, asthma in family, passive smoking, and PM fraction had significant coefficients.
Reference: Bartzokas et al. (2004) **Period of Study:** Jun 1, 1992–May 31, 2000 **Location:** Athens, Greece	**Outcome:** Respiratory and cardiovascular diseases (combined) **Age Groups:** NR **Study Design:** Time series **N:** 1554 patients **Statistical Analyses:** Simple linear regression and linear stepwise regression, Pearson correlation **Covariates:** Temperature, atmospheric pressure, relative humidity, wind speed **Season:** Warm (May-Sep) and cold (Nov-Mar) **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** NR	**Pollutant:** $PM_{4.5}$ (black smoke) **Averaging Time:** 10-day moving avg **Mean** μg/m^3 **(SD):** NR **Monitoring Stations:** 1 **Copollutant (correlation):** N	**PM Increment:** NR **Correlation with Number of Admissions:** Entire year Original: r = 0.18 Smoothed: r = 0.31 Warm period Original: r = 0.19 Smoothed: r = 0.30 Cold period Original: r = 0.18 Smoothed: r = 0.34 *All above values are statistically significant

E.3. Short-Term Exposure and Mortality

Table E-17. Short-term exposure to PM_{10} and mortality

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Aga et al. (2003) **Period of Study:** ~5 yrs for most cities, during the 1990s **Location:** 28 European cities (APHEA2)	**Outcome:** Non-Accidental Mortality (<800) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages >65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** (15, 66) **Copollutant:** BS **Note:** PM_{10} only measured in 21 cities.	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** All ages Fixed effects: 0.71% (0.60,0.83); 0-1 Random effects: 0.67% (0.47,0.87); 0-1 >65 Fixed effects: 0.79% (0.66,0.92); 0-1 Random effects: 0.74% (0.52,0.95); 0-1 Models with effect modifiers (>65) 24-h NO_2: 25th Percentile: 0.30% (0.07,0.53) 75th Percentile: 0.97% (0.82,1.11) 24-h temperature: 25th Percentile: 0.44% (0.25,0.64) 75th Percentile: 0.91% (0.77,1.05) 24-h relative humidity: 25th Percentile: 0.98% (0.82,1.14) 75th Percentile: 0.52% (0.33,0.71) Age standardized annual mortality rate: 25th Percentile: 0.93% (0.77,1.09) 75th Percentile: 0.61% (0.43,0.79) Proportion individuals >65 25th Percentile: 0.67% (0.50,0.83) 75th Percentile: 0.85% (0.71,0.99) Northwest/Central East: 25th Percentile: 0.81% (0.63,0.98) 75th Percentile: 0.26% (-0.05,0.57) Northwest/South: 25th Percentile: 0.81% (0.63,0.98) 75th Percentile: 1.04% (0.81,1.27)
Reference: Analitis et al. (2006) **Period of Study:** NR **Location:** 29 European cities (APHEA2)	**Outcome:** Mortality: Cardiovascular diseases (390-459) Respiratory diseases (460-519) **Study Design:** Time-series **Statistical Analyses:** 2-stage hierarchical modeling **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD)** unit: Range: 9–64 µg/m^3 **Range (Min, Max):** NR **Copollutant:** BS **Note:** PM_{10} only measured in 21 cities.	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Cardiovascular: Fixed effects: 0.64% (0.47, 0.80); 0-1 Random effects: 0.76% (0.47, 1.05); 0-1 0.90% (0.57, 1.23); 0-5 Respiratory: Fixed effects: 0.58% (0.21, 0.95); 0-1 Random effects: 0.71% (0.22, 1.20); 0-1 1.24% (0.49, 1.99); 0-5

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ballester et al. (2002) **Period of Study:** 1990–1996 **Location:** 13 Spanish cities	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular diseases (390-459) Respiratory diseases (460-519) **Study Design:** Ecological time series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD): Huelva:** 42.5 (15) Madrid: 37.8 (17.7) Sevilla: 45.1 (14) **Range (Min, Max):** NR **Copollutant:** BS TSP SO_2 **Note:** PM_{10} only measured in 3 cities.	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** Non-accidental: Random effects: 1.006 (0.998, 1.015); 0-1 Fixed Effects: 1.005 (1.001, 1.010); 0-1 PM_{10}+SO_2: 1.013 (1.006, 1.020); 0-1 Cardiovascular: 1.012 (1.005, 1.018); 0-1 PM_{10}+SO_2: Random effects: 1.024 (1.001, 1.048); 0-1 Fixed effects: 1.021 (1.007, 1.035); 0-1 Respiratory: 1.013 (1.001, 1.026); 0-1 PM_{10}+SO_2: 1.003 (0.983, 1.023); 0-1
Reference: Bateson and Schwartz (2004) **Period of Study:** 1988–1991 **Location:** Cook County, Illinois	**Outcome:** Mortality: Heart Disease (390-429) Respiratory (460-519) **Study Design:** Bi-directional case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** ≥ 65 Study population: 65,180 elderly residents with history of hospitalization for heart or lung disease	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SE) unit:** 37.6 (15.5) µg/m^3 **Range (Min, Max):** (3.7, 128) **Copollutant:** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** All-cause: 1.14% (0.44, 1.85); 0-1 Modification of Effect by Prior Diagnosis Myocardial Infarction: 1.98% (-0.25, 4.26); 0-1 Diabetes: 1.49% (-0.06, 3.07); 0-1 Congestive heart failure: 1.28% (-0.06, 2.64); 0-1 COPD: 0.58% (-0.82, 2.00); 0-1 Conduction Disorders: 0.64% (-0.61, 1.90); 0-1 All other heart or lung diseases: 0.74% (-0.29, 1.79); 0-1 All-cause Men 65: 2.0% (0.3, 3.8); 0-1 75: 1.5% (-0.2, 3.1); 0-1 85: 0.9% (-0.7, 2.5); 0-1 95: 0.3% (-1.3, 1.9); 0-1 All: 1.3% (0.4, 2.3); 0-1 Women 65: 0.1% (-1.6, 1.9); 0-1 75: 0.7% (-1.1, 2.4); 0-1 85: 1.2% (-0.5, 3.0); 0-1 95: 1.8% (0.03, 3.6); 0-1 All: 1.0% (0.1, 1.9); 0-1 Total 65: 1.1% (-0.12, 2.3); 0-1 75: 1.1% (-0.1, 2.3); 0-1 85: 1.2% (-0.0, 2.4); 0-1 95: 1.2% (0.0, 2.4); 0-1 All: 1.1% (0.4, 1.9); 0-1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Bellini et al. (2007) **Period of Study:** 1996–2002 **Location:** 15 Italian cities	**Outcome:** Mortality All-cause (non-accidental) (<800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Meta-analysis **Statistical Analyses:** Poisson GLM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** SO_2 NO_2 CO O_3	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** All-cause: 0.31% (-0.19, 0.74); 0-1 Winter: 0.08%; 0-1 summer: 1.95%; 0-1 PM_{10}+O_3: 0.30%; 0-1 PM_{10}+NO_2: 0.08%; 0-1 Respiratory: 0.54% (-0.91, 1.74); 0-1 Winter: 0.27%; 0-1 summer: 3.61%; 0-1 PM_{10}+O_3: 0.55%; 0-1 PM_{10}+NO_2: 0.19%; 0-1 Cardiovascular: 0.54% (0.02, 1.02); 0-1 Winter: 0.20%; 0-1 summer: 2.79%; 0-1 PM_{10}+O_3: 0.57%; 0-1 PM_{10}+NO_2: 0.39%; 0-1
Reference: Burnett et al. (2004) **Period of Study:** 1981–1999 **Location:** 12 Canadian cities	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Time-series **Statistical Analyses:** 1. Poisson, natural splines 2. Random effects regression model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** $PM_{2.5}$**:** 12.8 $PM_{10\text{-}2.5}$: 11.4 **Range (Min, Max):** NR **Copollutant (correlation):** NO_2 O_3; SO_2; CO **Note:** PM_{10} measurement calculated as the sum of $PM_{2.5}$ and $PM_{10\text{-}2.5}$ measurements.	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** 1981–1999 PM_{10}: 0.57% (0.05, 0.89); 1 PM_{10}+NO_2: 0.07% (-0.44, 0.58); 1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Cakmak et al. (2007) **Period of Study:** 1/1997–12/2003 **Location:** Chile–7 cities	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular diseases (390-459) Respiratory diseases (460-519) **Study Design:** Time-series **Statistical Analyses:** Poisson; Random effects regression model **Age Groups:** All age ≤ 64 65–74 75–84 ≥ 85	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 84.9 **Range (Min, Max):** NR **Copollutant (correlation):** O_3: r = -0.16 to 0.13 SO_2: r = 0.37 to 0.77 CO: r = 0.49 to 0.82 **Note:** Correlations are between pollutants for seven monitoring stations.	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Non-accidental: 0.97% (-1.09, 2.76); 0 1.31% (-1.56, 3.68); 0-5 PM_{10}+O_3+SO_2+CO: 0.80% (-0.87, 2.28); 0 ≤ 64: 0.52% (-0.55, 1.51); 0 0.49% (-0.51, 1.43); 0-5 65-75: 1.07% (-1.23, 3.03); 0 1.31% (-1.57, 3.69); 0-5 75-84: 1.41% (-1.71, 3.94); 0 1.93% (-2.57, 5.30); 0-5 ≥ 85: 1.56% (-1.94, 4.34); 0 2.14% (-2.97, 5.85); 0-5 April-September: 1.03% (-1.17, 2.93); 0 1.37% (-1.64, 3.82); 0-5 October-March: 0.07% (-0.07, 0.21); 0 0.15% (-0.15, 0.44); 0-5 Cardiovascular: 1.14% (-1.31, 3.21); 0 1.49% (-1.82, 4.14); 0-5 Respiratory: 2.03% (-2.75, 5.56); 0 3.11% (-5.25, 8.25); 0-5
Reference: Chen et al. (2008) **Period of Study:** 2001–2004 **Location:** Shanghai, China	**Outcome** (ICD9: 2001; ICD10: 2002-2004): Mortality: Non-accidental causes (ICD9 <800; ICD10 A00-R99) Cardiovascular (ICD9 390-459; ICD10 I00-I99) Respiratory (ICD9 460-519; ICD10 J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 102.0 **Range (Min, Max):** (14.0-566.8) **Copollutant (correlation):** SO_2; r = 0.64 NO_2; r = 0.71	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Non-accidental Single **Pollutant:** 0.26% (0.14, 0.37) PM_{10}+SO_2: 0.08% (-0.07, 0.22) PM_{10}+NO_2: 0.01% (-0.14, 0.17) PM_{10}+SO_2+NO_2: 0.00% (-0.16, 0.16) Cardiovascular mortality Single **Pollutant:** 0.27% (0.10, 0.44) PM_{10}+SO_2: 0.12% (-0.10, 0.34) PM_{10}+NO_2: 0.01% (-0.22, 0.25) PM_{10}+SO_2+NO_2: 0.01% (-0.23, 0.25) Respiratory mortality Single **Pollutant:** 0.27% (-0.01, 0.56) PM_{10}+SO_2: -0.04% (-0.41, 0.33) PM_{10}+NO_2: -0.05% (-0.45, 0.34) PM_{10}+SO_2+NO_2: -0.10% (-0.50, 0.30)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Daniels et al. (2004) **Period of Study:** 1987–1994 **Location:** 20 Largest U.S. cities	**Outcome:** Mortality: Total (Non-accidental) mortality Cardiovascular-Respiratory (390-448); (480-486, 487, 490-496, 507) Other-cause mortality **Study Design:** Time-series **Statistical Analyses:** City-Specific Estimates: Poisson GLM, natural cubic splines; Combined Estimates: 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Los Angeles: 46.0 New York: 28.8 Chicago: 35.6 Dallas-Ft. Worth: 23.8 Houston: 30.0 San Diego: 33.6 Santa Ana-Anaheim: 37.4 Phoenix: 39.7 Detroit: 40.9 Miami: 25.7 Philadelphia: 35.4 Minneapolis: 26.9 Seattle: 25.3 San Jose: 30.4 Cleveland: 45.1 San Bernardino: 37.0 Pittsburgh: 31.6 Oakland: 26.3 Atlanta: 34.4 San Antonio: 23.8	**Increment:** 10 μg/m^3 **% Increase (Lower CI, Upper CI); lag:** Total (non-accidental): 0.17% (0.03, 0.30); 0 0.20% (0.07, 0.33); 1 0.28% (0.16, 0.41); 0-1 avg Cardiovascular-Respiratory: 0.17% (-0.01, 0.35); 0 0.27% (0.09, 0.44); 1 0.30% (0.18, 0.51); 0-1 avg Other-cause: 0.17% (-0.03, 0.37); 0 0.12% (-0.07, 0.31); 1 0.20% (0.01, 0.38); 0-1 avg Threshold Models: Total Mortality Threshold = 15 μg/m^3 0.30% (0.17, 0.42); 0-1 avg Threshold = 0 μg/m^3 0.28% (0.16, 0.41); 0-1 avg Threshold = 20 μg/m^3 0.30% (0.16, 0.43); 0-1 avg
Reference: De Leon et al. (2003) **Period of Study:** 1/1985–12/1994 **Location:** New York, New York	**Outcome:** Mortality: Circulatory (390-459) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages <75 >75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 33.27 μg/m^3 IQR (25th, 75th): (22.67, 40.83) **Copollutant (correlation):** O_3 CO SO_2 NO_2	**Increment:** 18.16 μg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** All Ages Cancer: 1.014 (1.000, 1.029); 0-1 -w/out respiratory: 1.011 (0.996, 1.026); 0-1 -w/ respiratory: 1.051 (0.998, 1.107); 0-1 Circulatory: 1.025 (1.014, 1.035); 0-1 -w/out respiratory: 1.022 (1.012, 1.033); 0-1 -w/ respiratory: 1.054 (1.022, 1.086); 0-1 <75 Cancer: 1.003 (0.985, 1.021); 0-1 -w/out respiratory: 1.002 (0.983, 1.022); 0-1 -w/ respiratory: 1.009 (0.943, 1.078); 0-1 Circulatory: 1.027 (1.012, 1.043); 0-1 -w/out respiratory: 1.027 (1.011, 1.043); 0-1 -w/ respiratory: 1.033 (0.980, 1.089); 0-1 >75 Cancer: 1.033 (1.009, 1.058); 0-1 -w/out respiratory: 1.025 (1.000, 1.050); 0-1 -w/ respiratory: 1.129 (1.041, 1.225); 0-1 -w/out pneumonia: 1.026 (1.002, 1.050); 0-1 -w/ pneumonia: 1.183 (1.058, 1.323); 0-1 -w/out COPD: 1.032 (1.008, 1.057); 0-1 -w/ COPD: 1.008 (0.849, 1.197); 0-1 Circulatory: 1.025 (1.012, 1.038); 0-1 -w/out respiratory: 1.022 (1.008, 1.035); 0-1 -w/ respiratory: 1.066 (1.027, 1.106); 0-1 -w/out pneumonia: 1.023 (1.010, 1.036); 0-1 -w/ pneumonia: 1.078 (1.018, 1.141); 0-1 -w/out COPD: 1.025 (1.012, 1.038); 0-1 -w/ COPD: 1.058 (0.991, 1.130); 0-1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Dominici et al. (2003) **Period of Study:** 1987–1994 **Location:** 88 U.S. cities	**Outcome:** Mortality: All-cause (non-accidental) (<800) Cardiac (390-448) Respiratory (490-496) Influenza (487) Pneumonia (480-486, 507) Other causes **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** <65; 65-74; ≥ 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Cardio-respiratory 0.31% (0.15, 0.50); 1 All-cause 0.22% (0.10, 0.38); 1 Other causes 0.13% (-0.05, 0.29); 1
Reference: Dominici et al. (2004a) **Period of Study:** 1987–1994 **Location:** 90 U.S. cities (NMMAPS)	**Outcome:** Mortality: Total (non-accidental) **Study Design:** Time-series **Statistical Analyses:** Poisson. GAM, GLM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** α = 3 0.2% (0.05, 0.35)
Reference: Dominici et al. (2004b) **Period of Study:** 1986-1993 **Location:** 10 U.S. cities	**Outcome:** Mortality: Total (non-accidental) **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Birmingham 34.8 Canton 28.4 Colorado Springs 27.5 Minneapolis/St. Paul 28.1 Seattle 32.2 Spokane 42.9 Chicago 36.3 Detroit 36.7 New Haven 28.6 Pittsburgh 36.0 New York: 28.8	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Combined analysis: 0.26% (-0.37, 0.65); 0-1 Separate analysis: 0.28% (-0.12, 0.63); 0-1 Notes: A separate analysis assumes the mortality data does not provide any information on the log relative rates of mortality.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Dominici et al. (2007b) **Period of Study:** PM_{10}: 1987–2000 $PM_{2.5}$: 1999–2000 **Location:** 100 U.S. counties (NMMAPS)	**Outcome:** Mortality: All-cause (non-accidental) Cardiorespiratory Other-cause **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** PM_{10} All-cause: East: 1987-1994: 0.29% (0.12, 0.46); 1 1995-2000: 0.13% (-0.19, 0.44); 1 1987-2000: 0.25% (0.11, 0.39); 1 West: 1987-1994: 0.12% (-0.07, 0.30); 1 1995-2000: 0.18% (-0.07, 0.44); 1 1987-2000: 0.12% (-0.02, 0.26); 1 National: 1987-1994: 0.21% (0.10, 0.32); 1 1995-2000: 0.18% (0.00, 0.35); 1 1987-2000: 0.19% (0.10, 0.28); 1 Cardiorespiratory: East: 1987-1994: 0.39% (0.16, 0.63); 1 1995-2000: 0.30% (-0.13, 0.73); 1 1987-2000: 0.34% (0.15, 0.54); 1 West: 1987-1994: 0.17% (-0.07, 0.40); 1 1995-2000: 0.13% (-0.23, 0.50); 1 1987-2000: 0.14% (-0.05, 0.33); 1 National: 1987-1994: 0.28% (0.14, 0.43); 1 1995-2000: 0.21% (-0.03, 0.44); 1 1987-2000: 0.24% (0.13, 0.36); 1 Other-cause: East: 1987-1994: 0.21% (-0.03, 0.44); 1 1995-2000: 0.00% (-0.49, 0.50); 1 1987-2000: 0.15% (-0.09, 0.39); 1 West: 1987-1994: 0.09% (-0.21, 0.38); 1 1995-2000: 0.23% (-0.15, 0.62); 1 1987-2000: 0.17% (-0.07, 0.41); 1 National: 1987-1994: 0.15% (-0.02, 0.32); 1 1995-2000: 0.17% (-0.07, 0.41); 1 1987-2000: 0.15% (0.00, 0.29); 1
Reference: Dominici et al. (2007a) **Period of Study:** 2000–2005 **Location:** 72 U.S. counties representing 69 communities	**Outcome:** Total mortality **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	The study does not provide results quantitatively. **Note:** The study investigated whether county-specific short-term effects of PM_{10} on mortality are modified by long-term county-specific nickel or vanadium $PM_{2.5}$ concentrations.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Fischer et al. (2003) **Period of Study:** 1986–1994 **Location:** The Netherlands	**Outcome:** Mortality: Non-accidental (<800) Pneumonia (480-486) COPD (490-496) Cardiovascular (390-448) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** <45 45-64 65-74 ≥ 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** 34 **Range (Min, Max):** (10, 278) **Copollutant:** BS O_3 NO_2 SO_2 CO	**Increment:** 80 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** Cardiovascular <45: 0.906 (0.728, 1.128); 0-6 45-64: 1.023 (0.945, 1.106); 0-6 65-74: 1.002 (0.945, 1.062); 0-6 ≥ 75: 1.016 (0.981, 1.052); 0-6 COPD <45: 1.153 (0.587, 2.268); 0-6 45-64: 1.139 (0.841, 1.541); 0-6 65-74: 1.166 (0.991, 1.372); 0-6 ≥ 75: 1.066 (0.965, 1.178); 0-6 Pneumonia <45: 1.427 (0.806, 2.525); 0-6 45-64: 1.712 (1.042, 2.815); 0-6 65-74: 1.240 (0.879, 1.748); 0-6 ≥ 75: 1.123 (1.011, 1.247); 0-6
Reference: Fischer et al. (2004) **Period of Study:** 6/2003–8/2003 **Location:** The Netherlands	**Outcome:** Total mortality **Study Design:** NR **Statistical Analyses:** NR **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** Weekly avg **Mean (SD):** 2000: 31 2002: 33 2003: 35 IQR (25th, 75th): NR **Copollutant:** O_3	The study does not present quantitative results. Notes: The study estimates the number of deaths attributable to PM_{10} during the summers of 2000, 2002, and 2003.
Reference: Forastiere et al. (2005) **Period of Study:** 1998-2000 **Location:** Rome, Italy	**Outcome:** Mortality: Ischemic heart disease (410-414) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** >35	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 52.1 (22.2) IQR (25th, 75th): (36.0, 65.7) **Copollutant (correlation):** PNC: r = 0.38 CO: r = 0.34 NO_2: r = 0.45 SO_2: r = 0.23 O_3: r = 0.13	**Increment:** 29.7 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** 4.8% (0.1, 9.8); 0 4.9% (0.0, 10.1); 1 3.8% (-1.0, 8.9); 2 2.8% (-2.0, 7.7); 3 6.1% (0.6, 11.9); 0-1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Forastiere et al. (2007) **Period of Study:** 1998–2001 **Location:** Rome, Italy	**Outcome:** Mortality: Natural (<800) Malignant neoplasms (140-208) Diabetes mellitus (250) Hypertensive disease (401-405) Previous acute myocardial infarction (410, 412) Other ischemic heart diseases (411, 413-414) Conduction disorders (426) Dysrhythmia (427) Heart failure (428) Cerebrovascular disease (430-438) Peripherical artery disease (440-448) COPD (490-496) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** >35	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean Range (SD) unit:** 51.0 (21.0) µg/m^3 IQR (25th, 75th): (36.1, 63.0) **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Non-accidental: 1.1% (0.7, 1.6); 0-1 Low income: 1.9%; 0-1 Low SES: 1.4%; 0-1 High income: 0.0%; 0-1 High SES: 0.1%; 0-1 Low PM Area: 0.9% (-0.4, 2.1); 0-1 High PM Area: 1.47% (0.4, 2.5); 0-1
Reference: Forastiere et al. (2008) **Period of Study:** 1997–2004 **Location:** 9 Italian cities	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** >35	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean Range (SD) unit:** 35.1 to 71.5 Range (5th, 95th): Lowest 5th: 14.3 Highest 95th: 147.0 **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Total: 0.60% (0.31, 0.89); 0-1 Age 35-64: -0.20% (-0.77, 0.37); 0-1 65-74: 0.51% (0.05, 0.98); 0-1 75-84: 0.59%(0.20, 0.97); 0-1 ≥ 85: 0.97% (0.53, 1.42); 0-1 ≥ 65: 0.75% (0.42, 1.09) Sex Men: 0.72% (0.37, 1.07); 0-1 Women: 0.83% (0.33, 1.33); 0-1 Median income (by census block) Low (<20th percentile): 0.80% (-0.02, 1.62); 0-1 Mid-low (20th-50th percentile): 0.68% (0.25, 1.12); 0-1 Mid-high (51st-80th percentile): 0.85% (0.40, 1.30); 0-1 High (>80th percentile): 0.30% (-0.25, 0.86); 0-1 Location of death Out-of-hospital: 0.71% (0.32, 1.11); 0-1 Discharged 2-28 d before death: 1.34% (0.49, 2.20); 0-1 In-hospital: 0.65% (0.33, 0.97); 0-1 Nursing home: -0.04% (-1.02, 0.95); 0-1
Reference: Goldberg et al. (2003) **Period of Study:** 1984–1993 **Location:** Montreal, Quebec, Canada	**Outcome:** Mortality: Congestive Heart Failure (428) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** PM_{10}: 32.2 (17.6) IQR (25th, 75th): PM_{10}: (19.7, 41.1) **Copollutant (correlation):** $PM_{2.5}$, TSP, Sulfate, CoH, SO_2, NO_2, CO, O_3	This study does not present results quantitatively for PM_{10}

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Goldberg et al. (2003) **Period of Study:** 1984–1993 **Location:** Montreal, Quebec, Canada	**Outcome:** Mortality: Diabetes (250) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural spline **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** PM_{10}: 32.2 (17.6) μg/m^3 IQR (25th, 75th): PM_{10}: (19.7, 41.1) **Copollutant (correlation):** $PM_{2.5}$, Sulfate, CoH, SO_2, NO_2, CO, O_3	This study does not present results quantitatively for PM_{10}
Reference: Kan and Chen (2003) **Period of Study:** 1/2000–12/2001 **Location:** Shanghai, China	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular (390-459) COPD (490-496) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages <65 65-75 >75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 91.14 (51.85) **Range (Min, Max):** (17.0, 385.0) **Copollutant (correlation):** SO_2: r = 0.71 NO_2: r = 0.73	**Increment:** 10 μg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** Non-accidental All ages: 1.003 (1.001, 1.005); 0 <65: 1.001 (0.997, 1.005); 0 65-75: 1.005 (1.001, 1.008); 0 >75: 1.003 (1.001, 1.006); 0 Cardiovascular All ages: 1.003 (1.000, 1.006); 0 <65: 1.002 (0.994, 1.010); 0 65-75: 1.003 (0.998, 1.008); 0 >75: 1.003 (1.000, 1.006); 0 COPD All ages: 1.005 (0.999, 1.011); 0 <65: 1.004 (0.981, 1.027); 0 65-75: 0.996 (0.986, 1.007); 0 >75: 1.006 (1.000, 1.012); 0 Multipollutant models SO_2: 1.001 (0.998, 1.003); 0 NO_2: 1.001 (0.998, 1.003); 0 SO_2+NO_2: 1.000 (0.997, 1.003); 0
Reference: Kan and Chen (2003) **Period of Study:** 1/2000–12/2001 **Location:** Shanghai, China	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular (390-459) COPD (490-496) **Study Design:** Case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 91.14 (51.85) IQR (25th, 75th): (54, 114) **Copollutant (correlation):** SO_2: r = 0.71 NO_2: r = 0.73	**Increment:** 10 μg/m^3 **Odds Ratio (Lower CI, Upper CI); lag:** Non-accidental: Bidirectional referent days: 7 d: 1.000 (0.9988, 1.002); 0-1 ma 7 and 14 d: 1.002 (1.000, 1.004); 0-1 ma 7, 14, and 21 d: 1.003 (1.001, 1.005); 0-1 ma Unidirectional referent days: 7 d: 1.015 (1.012, 1.018); 0-1 ma 7 and 14 d: 1.017 (1.015, 1.019); 0-1 ma 7, 14, and 21 d: 1.019 (1.012, 1.021); 0-1 ma Bidirectional referent days (7, 14, and 21 d): Cardiovascular: 1.004 (1.001, 1.007); 0-1 ma COPD: 1.006 (0.999, 1.013); 0-1 ma Non-accidental: PM_{10}+SO_2: 0.997 (0.994, 1.025); 0-1 ma PM_{10}+NO_2: 0.997 (0.994, 1.025); 0-1 ma PM_{10}+SO_2+NO_2: 0.995 (0.992, 1.025); 0-1 ma

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kan et al. (2005) **Period of Study:** 4/25/2003–5/31/2003 **Location:** Beijing, China	**Outcome:** Mortality: Severe acute respiratory syndrome (SARS) **Study Design:** Time-series **Statistical Analyses:** Poisson, GAM, smoothing spline **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 149.1 (8.1) **Range (Min, Max):** (34, 246) **Copollutant:** SO_2 NO_2	**Increment:** 10 µg/m^3 Relative Risk (Lower CI, Upper CI); lag: 0.99 (0.96 to 1.03); 0 1.00 (0.97 to 1.04); 1 1.02 (0.98 to 1.06); 2 1.04 (0.99 to 1.09); 3 1.06 (1.00 to 1.11); 4 1.06 (1.00 to 1.12); 5 1.05 (0.98 to 1.12); 6
Reference: Kan et al. (2007a) **Period of Study:** 3/2004–12/2005 **Location:** Shanghai, China	**Outcome (ICD10):** Mortality: Total (non-accidental) (A00-R99) Cardiovascular (I00-I99) Respiratory (J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, penalized splines **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 107.9 (2.39) µg/m^3 **Range (Min, Max):** (22.0, 403.0) **Copollutant (correlation):** PM_{10} $PM_{2.5}$: r = 0.84 $PM_{10-2.5}$: r = 0.88 O_3: r = 0.21	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** PM_{10} Total: 0.16% (0.02, 0.30); 0-1 Cardiovascular: 0.31% (0.10, 0.53); 0-1 Respiratory: 0.33% (-0.08, 0.75); 0-1
Reference: Kan et al. (2008) **Period of Study:** 1/2001–12/2004 **Location:** Shanghai, China	**Outcome:** Mortality: Total (non-accidental) (A00-R99) Cardiovascular (I00-I99) Respiratory (J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages; 0-4 5-44 45-64 ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Warm season: 87.4 (1.8) Cool season: 116.7 (2.8) Entire period: 102.0 (1.7) **Range (Min, Max):** NR **Copollutant (correlation):** SO_2 NO_2 O_3	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Non-accidental Warm season: 0.21 (0.09, 0.3); 0-1 Cool season: 0.26 (0.22, 0.30); 0-1 Entire period: 0.25 (0.14, 0.37); 0-1 Female: 0.33 (0.18, 0.48); 0-1 Male: 0.17 (0.03, 0.32); 0-1 5-44: 0.04 (-0.52, 0.59); 0-1 45-64: 0.17 (-0.11, 0.45); 0-1 ≥ 65: 0.26 (0.15, 0.38); 0-1 Cardiovascular Warm season: 0.22 (-0.14, 0.58); 0-1 Cool season: 0.25 (0.05, 0.45); 0-1 Entire period: 0.27 (0.10, 0.44); 0-1 Respiratory Warm season: -0.28 (-0.93, 0.38); 0-1 Cool season: 0.58 (0.25, 0.92); 0-1 Entire period: 0.27 (-0.01, 0.56); 0-1 Stratified by Educational Attainment Nonaccidental: Low: 0.33 (0.19, 0.47); 0-1 High: 0.18 (0.01, 0.36); 0-1 Cardiovascular: Low: 0.30 (0.10, 0.51); 0-1 High: 0.23 (-0.03, 0.50); 0-1 Respiratory: Low: 0.36 (0.00, 0.72); 0-1 High: 0.02 (-0.43, 0.47); 0-1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Keatinge and Donaldson (2006) **Period of Study:** 1991–2002 **Location:** London, England	**Outcome:** Mortality: Total (non-accidental) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** O_3 SO_2	**Increment:** 10 µg/m^3 **Mortality per 106 (Lower CI, Upper CI); lag:** PM_{10}+Temp: 2.1 (0.9, 3.3); 0-2 avg PM_{10}+Temp+Acclim: 1.6 (0.4, 2.8); 0-2 avg PM_{10}+Temp+Acclim+Acclim x T: 1.5 (0.3, 2.6); 0-2 avg PM_{10}+Temp+Acclim+Acclim x T+Sun: 1.4 (0.2, 2.5); 0-2 avg PM_{10}+Temp+Acclim+Acclim x T+Sun+Wind: 0.8 (-0.4, 1.9); 0-2 avg PM_{10}+Temp+Acclim+Acclim x T+Sun+Wind+Abs. Humid.: 0.8 (-0.3, 1.9); 0-2 avg PM_{10}+Temp+Acclim+Acclim x T+Sun+Wind+Abs. Humid.+ Rain: 0.9 (-0.3, 2.0); 0-2 avg PM_{10}+Temp+Abs. Humid.: 1.9 (0.7, 3.1); 0-2 avg
Reference: Kettunen et al. (2007) **Period of Study:** 1998–2004 **Location:** Helsinki, Finland	**Outcome (ICD10):** Mortality: Stroke (I60-I61, I63-I64) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, penalized thin-plate splines **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg Median (SD) unit: Cold Season: 16.3 Warm Season: 16.5 **Range (Min, Max):** Cold Season: (3.1, 136.7) Warm Season: (3.3, 67.4) **Copollutant:** $PM_{2.5}$; $PM_{10\text{-}2.5}$; UFP; O_3; CO; NO_2	**Increment:** Cold Season: 13.8 µg/m^3 Warm Season: 9.8 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Cold Season -0.56% (-3.32, 2.29); 0 -0.93% (-3.55, 1.75); 1 -1.68% (-4.30, 1.00); 2 -1.53% (-4.14, 1.14); 3 Warm Season 10.89% (0.95, 21.81); 0 8.56% (-0.88, 18.90); 1 2.06% (-6.76, 11.71); 2 -2.89% (-11.32, 6.34); 3

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kim et al. (2003) **Period of Study:** 1/1995–12/1999 **Location:** Seoul, Korea	**Outcome (ICD10):** Mortality: Non-accidental (all except S01-S99, T01-T98) Cardiovascular (I00-I52) Respiratory (J00-J98) Cerebrovascular (I60-I69) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 69.19 (10.36) IQR (25th, 75th): (44.82, 87.95) **Copollutant (corelation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** All cause: 2.8% (1.8, 3.7); 0 2.8% (1.9, 3.7); 1 1.4% (0.5, 2.3); 2 3.7% (2.1, 5.4); distributed lag (6-day) Respiratory: 8.3% (4.3, 12.5); 0 6.4% (2.7, 10.2); 1 6.5% (2.7, 10.4); 2 13.9% (6.8, 21.5); distributed lag (6-day) Pneumonia: 11.6% (4.2, 19.6); 0 9.0% (2.1, 16.3); 1 7.7% (0.8, 15.2); 2 17.1% (4.1, 31.7); distributed lag (6-day) COPD: 4.2% (-1.2, 10.0); 0 3.5% (-1.5, 8.9); 1 1.4% (-3.7, 6.8); 2 12.2% (2.5, 22.9); distributed lag (6-day) Cardiovascular: 2.0% (-0.9, 5.0); 0 3.3% (0.6, 6.2); 1 2.9% (0.1, 5.8); 2 4.4% (-0.6, 9.6); distributed lag (6-day) Myocardial infarction: 2.6% (-2.3, 7.8); 0 5.8% (1.0, 10.7); 1 5.5% (0.7, 10.6); 2 4.9% (-3.4, 13.9); distributed lag (6-day) Cerebrovascular: 3.2% (0.8, 5.5); 0 3.1% (0.9, 5.3); 1 2.4% (0.1, 4.6); 2 6.3% (2.3, 10.5); distributed lag (6-day) Ischemic stroke: -0.6% (-5.6, 4.7); 0 0.6% (-4.2, 5.7); 1 -0.1% (-4.9, 5.1); 2 10.3% (1.0, 20.4); distributed lag (6-day)
Reference: Kim et al. (2004) **Period of Study:** 1/1997–12/2001 **Location:** Seoul, Korea	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 68.23 (36.36) µg/m^3 IQR (25th, 75th): (42.56, 84.67) **Copollutant (correlation):** NR	**Increment:** 42.11 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** 1.021 (1.009, 1.035)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Le Tertre et al. (2005) **Period of Study:** NR **Location:** 21 European cities (APHEA-2)	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Time-series **Statistical Analyses:** Empirical Bayes **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** NO_2	**Increment:** 1.0 µg/m^3 **β coefficient (SE); lag:** Athens: 0.001311 (0.0003) Barcelona: 0.000575 (0.0002) Basel: 0.000462 (0.0005) Birmingham: 0.000305 (0.0003) Budapest: -0.000248 (0.0005) Cracow: 0.000155 (0.0004) Erfurt: -0.000465 (0.0004) Geneva: -0.000059 (0.0005) Helsinki: 0.000389 (0.0004) London: 0.000591 (0.0002) Lyon: 0.001554 (0.0005) Madrid: 0.000372 (0.0003) Milan: 0.000901 (0.0002) Paris: 0.000411 (0.0003) Prague: 0.000097 (0.0002) Rome: (0.001333 (0.0003) Stockholm: 0.000479 (0.0009) Tel Aviv: 0.000522 (0.0003) Teplice: 0.000876 (0.0004) Torino: 0.000938 (0.0002) Zurich: 0.000365 (0.0004) Toulouse: NR (NR) Overall: 0.00055 (0.000098)
Reference: Lee et al. (2007a) **Period of Study:** 1/2000–12/2004 **Location:** Seoul, Korea	**Outcome (ICD10):** Mortality: Non-accidental (A00-R99) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** w/ Asian dust days: 70.00 (47.80) w/o Asian dust days: 65.77 (33.60) Asian dust days only: 188.49 (142.85) **Copollutant:** CO; NO_2; SO_2; O_3	**Increment:** 41.49 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Model with Asian Dust Days 0.7% (0.2, 1.3); 1-3 Model without Asian dust days 1.0% (0.2, 1.8); 1-3
Reference: Lee and Shaddick (2007) **Period of Study:** 1/1/1993 –12/31/1997 **Location:** Cleveland, Ohio; Detroit, Michigan; Minneapolis, Minnesota; Pittsburgh, Pennsylvania	**Outcome (ICD10):** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** 1. Bayesian, penalized spline 2. Likelihood, penalized spline **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** Constant model Cleveland: 1.0049; 1 Detroit: 1.0046; 1 Minneapolis: 1.0052; 1 Pittsburgh: 1.0045; 1
Reference: Martins et al. (2004) **Period of Study:** 1/1997–12/1999 **Location:** São Paulo, Brazil	**Outcome (ICD10):** Mortality: Respiratory (J00-J99) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural cubic splines **Age Groups:** ≥ 60	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Cerqueira Cesar: 42.5(22.9) Santa Amaro: 49.6(32.1) Central: 52.1(23.5) Penha: 40.4(23.8) Santana: 72.6(24.5) Sao Miguel Paulista: 68.6(31.0) **Range (Min, Max):** NR	The study does not present quantitative results.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Nawrot et al. (2007) **Period of Study:** 1/1997–12/2003 **Location:** Flanders, Belgium	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Time-series **Statistical Analyses:** Main analysis: Segmented regression models Sensitivity analysis: Poisson GAM, LOESS **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** Winter: 43.3(0.88) Spring: 39.5(0.88) summer: 37.7(0.91) Fall: 37.2(0.88) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** Main analysis: NR Sensitivity analysis: 10 μg/m^3 **% Increase (Lower CI, Upper CI); lag:** Highest season-specific PM_{10} quartile versus the lowest season-specific PM_{10} quartile summer: 7.8% (6.1, 9.6) Spring: 6.3% (4.7, 7.8) Autumn: 2.2% (0.58, 3.8) Winter: 1.4% (0.06, 2.9) Warm months (June, July, August): 7.9% (6.2, 9.6) Cold months (December, January, February): 1.5% (0.22, 3.3) Intermediate months (March, April, May, September, October, November): 4.2% (2.9, 5.6) Warmer Periods (April–September) Non-accidental: 1.5% (1.1, 2.0); 0 Respiratory: 2.0% (0.6, 3.7); 0 Cardiovascular: 1.8% (1.1, 2.4); 0

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: O'Neill et al. (2004) **Period of Study:** 1996–1998; 1994–7/1995 **Location:** Mexico City, Mexico	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Poisson, natural cubic spline **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Range**: Hi-Vol: 46.3–164.0 TEOM: 48.2–107.5 Predicted: 30.2–162.4 Impactor: 58.4 **Range (Min, Max):** Xalostoc Hi-Vol: (40.0, 335.0) TEOM: (16.5, 291.2) Predicted: (60.6, 320.0) Tlalnepantla Hi-Vol: (25.0, 264.0) TEOM: (10.4, 275.9) Predicted: (17.7, 175.0) Merced Hi-Vol: (17.0, 266.0) TEOM: (9.4, 318.7) Predicted: (12.3, 160.8) Cerro de la Estrella Hi-Vol: (15.0, 292.0) TEOM: (13.7, 268.3) Predicted: (11.2, 154.4) Pedregal (1996-1998) Hi-Vol: (5.0, 226.0) TEOM: (7.8, 264.4) Predicted: (-0.5, 86.3) Pedregal (1994-1995) Hi-Vol: (24.0, 114.0) TEOM: (8.7, 152.5) Impactor: (15.0, 154.0) Predicted: (3.9, 75.9)	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** TEOM 0.04% (-0.12, 0.20); 0 -0.02% (-0.18, 0.13); 1 -0.01% (-0.27, 0.25); 2 -0.03% (-0.19, 0.13); 3 -0.03% (-0.19, 0.13); 4 -0.05% (-0.21, 0.11); 5 0.05% (-0.25, 0.35); 0-5 Predicted -0.05% (-0.29, 0.19); 0 0.09% (-0.16, 0.34); 1 -0.12% (-0.43, 0.20); 2 -0.02% (-0.26, 0.21); 3 -0.14% (-0.37, 0.09); 4 -0.05% (-0.28, 0.18); 5 0.00% (-0.39, 0.38); 0-5 Sierra-Anderson High Volume Air Sampler 0.02% (-0.29, 0.32); 0 0.13% (-0.27, 0.54); 1 0.21% (-0.10, 0.52); 2 0.53% (0.07, 0.99); 3 0.11% (-0.20, 0.41); 4 0.38% (0.07, 0.70); 5 GAM: 2 LOESS terms, default convergence 1.68% (0.45, 2.93); 0 -0.36% (-1.56, 0.86); 1 -0.21% (-1.40, 1.00); 2 -0.18% (-1.40, 1.05); 3 1.31% (0.08, 2.55); 4 1.49% (0.25, 2.73); 5 1.77% (-0.26, 3.83); 0-5 Parametric: cubic splines 5 df 1.45% (0.09, 2.83); 0 -0.71% (-2.06, 0.67); 1 -0.59% (-1.95, 0.79); 2 -0.70% (-2.09, 0.71); 3 0.92% (-0.46, 2.32); 4 1.17% (-0.19, 2.55); 5 1.17% (-1.54, 3.95); 0-5 10 df 1.60% (0.20, 3.02); 0 -0.80% (-2.18, 0.60); 1 -0.73% (-2.11, 0.68); 2 -1.05% (-2.49, 0.40); 3 0.64% (-0.79, 2.10); 4 1.05% (-0.36, 2.48); 5 0.51% (-2.60, 3.71); 0-5 2 df 1.79% (0.48, 3.11); 0 -0.09% (-1.38, 1.22); 1 0.10% (-1.18, 1.40); 2 0.20% (-1.10, 1.52); 3 1.60% (0.30, 2.91); 4 1.72% (0.43, 3.04); 5 1.90% (-0.36, 4.21); 0-5

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: O'Neill et al. (2005b) **Period of Study:** 1996–1998; 1996-1999 **Location:** Mexico City and Monterrey, Mexico	**Outcome:** Mortality: Non-accidental Cardiovascular (390-460) Respiratory (460-520) Other-causes **Study Design:** Time-series **Statistical Analyses:** Poisson, natural cubic splines **Age Groups:** All ages, 0-15, ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Mexico City: 75.8 (31.4) Monterrey: 50.0 (23.5) **Range (Min, Max):** Mexico City: (18.0, 233.9) Monterrey: (6.2, 230.8) **Copollutant:** O_3	The study focuses on the temperature–mortality relationship and only includes PM_{10} as a covariate in models.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: O'Neill et al. (2008) **Period of Study:** 1998–2002 **Location:** Mexico City, Mexico; Santiago, Chile; São Paulo, Brazil	**Outcome (ICD10):** Mortality: Non-accidental Cardiovascular (I <800) Respiratory (J100-118, 120,-189, 209-499, 690-700) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural cubic splines **Age Groups:** >22 >65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Mexico City: 53.8 (24.9) Santiago: 78.7 (33.0) São Paulo: 48.9 (21.9) **Range (Min, Max):** Mexico City: (10.8, 192.2) Santiago: (8.0, 218.6) São Paulo: (12.0, 171.3) **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** **Mexico City** >21: 0.30% (0.05, 0.56); 0; 0.39% (0.13, 0.65); 1 0.35% (-0.07, 0.76); 0-5 >65: 0.44% (0.12, 0.76); 0; 0.50% (0.17, 0.82); 1 0.31% (-0.21, 0.84); 0-5 **São Paulo** >21: 1.06% (0.73, 1.39); 0; 1.04% (0.71, 1.38); 1 1.38% (0.85, 1.91); 0-5 >65: 0.85% (0.44, 1.27); 0; 1.09% (0.68, 1.51); 1 1.17% (0.50, 1.85); 0-5 **Santiago** >21: 0.27% (0.05, 0.48); 0; 0.61% (0.40, 0.83); 1 0.86% (0.48, 1.23); 0-5 >65: 0.48% (0.23, 0.74); 0; 0.84% (0.58, 1.09); 1 1.32% (0.88, 1.75); 0-5 **Education: >21** **Mexico City** None: 0.76% (0.17, 1.360: 0; 0.62% (0.02, 1.22); 1 0.91% (-0.07, 1.89); 0-5 Primary: 0.27% (-0.19, 0.72); 0; 0.62% (0.17, 1.08); 1 0.48% (-0.27, 1.24); 0-5 Secondary: 0.19% (-0.19, 0.57); 0; 0.29% (-0.09, 0.67); 1 0.27% (-0.36, 0.90); 0-5 ≥ 12 years: 0.83% (0.03, 1.63); 0; 0.58% (-0.21, 1.38); 1 0.76% (-0.49, 2.02); 0-5 **São Paulo** None: 0.77% (-0.28, 1.82); 0; 0.70% (-0.34, 1.76); 1 0.76% (-0.91, 2.46); 0-5 Primary: 1.27% (0.78, 1.76); 0; 1.32% (-0.83, 1.82); 1 1.34% (0.55, 2.14); 0-5 Secondary: 0.93% (-0.07, 1.94); 0; 1.59% (0.58, 2.60); 1 1.91% (0.35, 3.48); 0-5 ≥ 12 years: 2.93% (2.00, 3.88); 0; 2.20% (1.27, 3.15); 1 3.59% (2.23, 4.97); 0-5 **Santiago** None: 1.44% (0.53, 2.36); 0; 2.08% (1.16, 3.01); 1 3.18% (1.60, 4.78); 0-5 Primary: 0.06% (-0.21, 0.34); 0 ;0.53% (0.25, 0.81); 1 0.58% (0.10, 1.06); 0-5 Secondary: 0.42% (0.06, 0.78); 0; 0.55% (0.19, 0.91); 1 1.10% (0.48, 1.73); 0-5 ≥ 12 years: 1.32% (0.60, 2.05); 0; 1.31% (0.59, 2.04); 1 2.00% (0.93, 3.07); 0-5 **Education: >65** **Mexico City** None: 0.41% (-0.25, 1.08); 0; 0.20% (-0.47, 0.87); 1 0.27% (-0.83, 1.38); 0-5 Primary: 0.40% (-0.15, 0.95); 0; 0.80% (0.24, 1.36); 1; 0.99% (0.07, 1.91); 0-5 Secondary: 0.50% (-0.01, 1.01); 0; 0.60% (0.09, 1.12); 1; 0.30% (-0.56, 1.16); 0-5 ≥ 12 years: 1.51% (0.39, 2.63); 0; 1.09% (-0.02, 2.22); 1; 1.83% (0.09, 3.59); 0-5 **São Paulo** None: 0.60% (-0.48, 1.70); 0; 0.62% (-0.47, 1.72); 1; 0.91% (-0.84, 2.69); 0-5 Primary: 1.59% (1.00, 2.19); 0; 1.48% (0.89, 2.07); 1; 1.73% (0.79, 2.67); 0-5 Secondary: 1.21% (-0.01, 2.44); 0; 2.31% (1.08, 3.55); 1; 3.25% (1.39, 5.16); 0-5 ≥ 12 years: 2.80% (1.67, 3.94); 0; 2.52% (1.40, 3.66); 1; 3.63% (2.01, 5.29); 0-5 **Santiago** None: 1.49% (0.54, 2.45); 0; 2.20% (1.24, 3.17); 1; 3.21% (1.54, 4.90); 0-5 Primary: 0.28% (-0.03, 0.59); 0; 0.74% (0.43, 1.05); 1; 0.92% (0.38, 1.46); 0-5 Secondary: 0.58% (0.13, 1.04); 0; 0.65% (0.20, 1.11); 1; 1.46% (0.67, 2.25); 0-5 ≥ 12 years: 2.32% (1.50, 3.15); 0; 2.20% (1.36, 3.04); 1; 4.02% (2.78, 5.27); 0-5

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Peng et al. (2005) **Period of Study:** 1987–2000 **Location:** 100 U.S. cities (NMMAPS)	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Bayesian semiparametric hierarchical models **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** 27.1 **Range (Min, Max):** (13.2, 48.7) **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Winter: -0.4% (-0.30, 0.21); 0 0.15% (-0.08, 0.39); 1 0.10% (-0.13, 0.33); 2 Spring: 0.32% (0.08, 0.56); 0 0.14% (-0.14, 0.42); 1 0.05% (-0.21, 0.32); 2 Summer: 0.13% (-0.11, 0.37); 0 0.36% (0.11, 0.61); 1 -0.03% (-0.27, 0.21); 2 Fall: 0.05% (-0.16, 0.25); 0 0.14% (-0.06, 0.34); 1 0.13% (-0.08, 0.35); 2 All Seasons: 0.09% (-0.01, 0.19); 0 0.19% (0.10, 0.28); 1 0.08% (-0.03, 0.19); 2 PM_{10} only (45 cities): Winter: 0.15% (-0.16, 0.45); 1 Spring: 0.13% (-0.21, 0.48); 1 Summer: 0.30% (-0.10, 0.69); 1 Fall: 0.07% (-0.23, 0.37); 1 PM_{10} + O_3 (45 cities): Winter: 0.18% (-0.16, 0.52); 1 Spring: 0.10% (-0.30, 0.49); 1 Summer: 0.33% (-0.14, 0.81); 1 Fall: 0.08% (-0.25, 0.41); 1 PM_{10} + O_3 (45 cities): Winter: 0.13% (-0.24, 0.49); 1 Spring: 0.1% 9(-0.18, 0.56); 1 Summer: 0.28% (-0.13, 0.70); 1 Fall: -0.01% (-0.34, 0.31); 1 PM_{10} + NO_2 (45 cities): Winter: 0.21% (-0.18, 0.60); 1 Spring: 0.19% (-0.17, 0.54); 1 Summer: 0.34% (0.01, 0.68); 1 Fall: 0.13% (-0.12, 0.39); 1
Reference: Penttinen et al. (2004) **Period of Study:** 1988–1996 **Location:** Helsinki, Finland	**Outcome:** Mortality: Total (non-accidental) (<800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** 15-64 65-74 ≥ 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** 21 **Range (Min, Max):** (0.2, 213) **Copollutant (correlation):** O_3: r = -0.09 NO_2: r = 0.50 CO: r = 0.45 SO_2: r = 0.61 TSP: r = 0.72	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Total (non-accidental) -0.23% (-1.47, 1.01); 0 0.88% (-0.32, 2.08); 1 0.11 (-0.51, 0.73); 0-3 avg Cardiovascular -1.22% (-3.00, 0.56); 0 0.63% (-1.09, 2.35); 1 0.08% (-0.96, 0.81); 0-3 avg Respiratory 3.94% (0.01, 7.87); 0 3.96% (0.11, 7.81); 1 2.13% (0.03, 4.22); 0-3 avg

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Qian et al. (2007) **Period of Study:** 2001–2004 **Location:** Wuhan, China	**Outcome:** Mortality: Total (non-accidental) (<800) Cardiovascular (390-459) Stroke (430-438) Cardiac Diseases (390-398) Respiratory (460-519) Cardiopulmonary **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, natural splines **Age Groups:** All ages <45 ≥ 45 <65 ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 141.8 [3] **Range (Min, Max):** (24.8, 477.8) **Copollutant (correlation):** NO_2 SO_2 O_3	**Increment:** 10 µg/m[3] **% Increase (Lower CI, Upper CI); lag:** **Non-accidental** 0.36% (0.19, 0.53); 0; 0.28% (0.12, 0.45); 1; 0.43% (0.24, 0.62); 0-1; 0.08% (-0.15, 0.31); 0-4 **<45** 0.28% (-0.26, 0.82); 0; 0.45% (-0.06, 0.96); 1; 0.53% (-0.08, 1.13); 0-1; 0.41% (-0.31, 1.13); 0-4 **≥ 45** 0.36% (0.19, 0.54); 0; 0.27% (0.10, 0.44); 1; 0.42% (0.22, 0.62); 0-1; 0.05% (-0.18, 0.29); 0-4 **<65** 0.20% (-0.08, 0.49); 0; 0.25% (-0.03, 0.52); 1; 0.33% (0.01, 0.66); 0-1; 0.01% (-0.38, 0.39); 0-4 **≥ 65** 0.41% (0.21, 0.61); 0; 0.30% (0.10, 0.49); 1; 0.46% (0.24, 0.69); 0-1; 0.10% (-0.16, 0.37); 0-4 **Cardiovascular** 0.51% (0.28, 0.75); 0; 0.35% (0.12, 0.58); 1; 0.58% (0.31, 0.84); 0-1; 0.35% (0.05, 0.66); 0-4 **<45** 0.59% (-0.62, 1.82); 0; 0.93% (-0.22, 2.08); 1; 1; 1.07% (-0.27, 2.42); 0-1; 1.15% (-0.40, 2.72); 0-4 **≥ 45** 0.51% (0.27, 0.75); 0; 0.33% (0.10, 0.56); 1; 0.56% (0.30, 0.83); 0-1; 0.33% (0.02, 0.63); 0-4 **<65** 0.27% (-0.23, 0.76); 0; 0.30% (-0.16, 0.77); 1; 0.42% (-0.12, 0.97); 0-1; 0.43% (-0.19, 1.06); 0-4 **≥ 65** 0.57% (0.31, 0.83); 0; 0.36% (0.11, 0.61); 1; 0.61% (0.32, 0.90); 0-1; 0.33% (0.00, 0.67); 0-4 **Stroke** 0.44% (0.16, 0.72); 0; 0.41% (0.14, 0.68); 1; 0.58% (0.27, 0.89); 0-1; 0.45% (0.09, 0.81); 0-4 **<45** 1.18% (-0.45, 2.83); 0;1.66% (0.11, 3.24); 1; 1.91% (0.10, 3.75); 0-1; 2.72% (0.58, 4.89); 0-4 **≥ 45** 0.42% (0.14, 0.70); 0; 0.37% (0.10, 0.65); 1; 0.55% (0.23, 0.86); 0-1; 0.39% (0.03, 0.76); 0-4 **<65** 0.26% (-0.35, 0.87); 0; 0.38% (-0.20, 0.96); 1; 0.48% (-0.19, 1.16); 0-1; 0.57% (-0.21, 1.35); 0-4 **≥ 65** 0.49% (0.17, 0.80); 0; 0.41% (0.11, 0.72); 1; 0.61% (0.26, 0.96); 0-1; 0.42% (0.02, 0.83); 0-4 **Cardiac** 0.49% (0.08, 0.89); 0; 0.28% (-0.11, 0.67); 1; 0.49% (0.04, 0.94); 0-1; 0.22% (-0.29, 0.74); 0-4 **<45** 0.25% (-1.64, 2.17); 0; 0.56% (-1.22, 2.38); 1; 0.61% (-1.47, 2.74); 0-1; -0.42% (-2.80, 2.02); 0-4 **≥ 45** 0.49% (0.09, 0.91); 0; 0.27% (-0.12, 0.66); 1; 0.48% (0.03, 0.94); 0-1; 0.25% (-0.27, 0.77); 0-4 **<65** 0.00% (-0.89, 0.90); 0; 0.12% (-0.73, 0.98); 1; 0.13% (-0.86, 1.13); 0-1; 0.05% (-1.08, 1.20); 0-4 **≥ 65** 0.60% (0.17, 1.03); 0; 0.32% (-0.10, 0.74); 1; 0.57% (0.09, 1.06); 0-1; 0.26% (-0.29, 0.82); 0-4 **Respiratory** 0.71% (0.20, 1.23); 0; 0.63% (0.13, 1.13); 1; 0.86% (0.28, 1.44); 0-1; 0.19% (-0.48, 0.87); 0-4 **<45** 1.74% (-1.28, 4.86); 0; 2.52% (-0.30, 5.42); 1; 2.95% (-0.41, 6.42); 0-1; 3.47% (-0.61, 7.73); 0-4 **≥ 45** 0.69% (0.18, 1.21); 0; 0.58% (0.09, 1.08); 1; 0.81% (0.23, 1.39); 0-1; 0.13% (-0.54, 0.80); 0-4 **<65** 0.06% (-1.30, 1.43); 0; -0.53% (-1.83, 0.79); 1; -0.32% (-1.84, 1.22); 0-1; -0.72% (-2.47, 1.05); 0-4 **≥ 65** 0.79% (0.27, 1.31); 0; 0.76% (0.26, 1.26); 1; 0.99% (0.41, 1.57); 0-1;

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			0.30% (-0.38, 0.98); 0-4 **Cardiopulmonary** 0.46% (0.23, 0.69); 0; 0.35% (0.13, 0.57); 1; 0.53% (0.28, 0.79); 0-1; 0.11% (-0.19, 0.42); 0-4 **<45** 0.71% (-0.48, 1.92); 0; 1.26% (0.14, 2.4); 1; 1.39% (0.06, 2.74); 0-1; 1.41% (-0.18, 3.03); 0-4 **≥ 45** 0.45% (0.23, 0.68); 0; 0.32% (0.10, 0.54); 1; 0.51% (0.25, 0.77); 0-1; 0.08% (-0.23, 0.38); 0-4 **<65** 0.14% (-0.34, 0.61); 0; 0.15% (-0.30, 0.61); 1; 0.23% (-0.30, 0.76); 0-1; 0.11% (-0.52, 0.74); 0-4 **≥ 65** 0.53% (0.28, 0.78); 0; 0.39% (0.15, 0.63); 1; 0.60% (0.32, 0.88); 0-1; 0.11% (-0.22, 0.45); 0-4 **Two-pollutant Models** **Non-accidental** PM_{10}+NO_2: 0.14% (-0.07, 0.36); 0; PM_{10}+SO_2: 0.37% (0.20, 0.55); 0; PM_{10}+O_3: 0.34% (0.17, 0.51); 0 **Cardiovascular** PM_{10}+NO_2: 0.34% (0.04, 0.63); 0; PM_{10}+SO_2: 0.53% (0.28, 0.77); 0; PM_{10}+O_3: 0.50% (0.26, 0.74); 0 **Stroke** PM_{10}+NO_2: 0.28% (-0.07, 0.63); 0; PM_{10}+SO_2: 0.49% (0.21, 0.78); 0; PM_{10}+O_3: 0.44 (0.16, 0.72); 0 **Cardiac** PM_{10}+NO_2: 0.24% (-0.27, 0.75); 0; PM_{10}+SO_2: 0.43 (0.01, 0.84); 0; PM_{10}+O_3: 0.44% (0.03, 0.85); 0 **Respiratory** PM_{10}+NO_2: 0.46% (-0.19, 1.12); 0; PM_{10}+SO_2: 0.64% (0.11, 1.18); 0; PM_{10}+O_3: 0.67% (0.15, 1.20); 0 **Cardiopulmonary** PM_{10}+NO_2: 0.26% (-0.02, 0.55); 0; PM_{10}+SO_2: 0.46% (0.23, 0.70); 0; PM_{10}+O_3: 0.44% (0.21, 0.67); 0
Reference: Qian et al. (2008) **Period of Study:** 7/2001–6/2004 **Location:** Wuhan, China	**Outcome:** Mortality: Total (non-accidental) (<800) Cardiovascular (390-459) Stroke (430-438) Cardiac diseases (390-398, 410-429) Respiratory (460-519) Cardiopulmonary (390-459, 460-519) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines and penalized splines **Age Groups:** All ages <65 ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Normal temperature: 145.7 (64.6) Low temperature: 117.3 (49.5) High temperature: 96.3 (27.9) **Range (Min, Max):** NR **Copollutant (correlation):** Normal temperature: NO_2: r = 0/72 SO_2: r = 0.59 O_3: r = 0.06 Low temperature: NO_2: r = 0.83 SO_2: r = 0.74 O_3: r = 0.19 High temperature: NO_2: r = 0.68 SO_2: r = 0.15 O_3: r = 0.65	**Increment:** 10 μg/m^3 **% Increase (Lower CI, Upper CI); lag:** **Non-accidental:** **Normal:** All ages: 0.36 (0.17, 0.56); 0-1; <65: 0.23 (-0.10, 0.56); 0-1; ≥ 65: 0.51 (0.18, 0.64); 0-1; PM_{10}+NO_2: 0.07 (-0.17, 0.30); 0-1; PM_{10}+SO_2: 0.27 (0.06, 0.47); 0-1; PM_{10}+O_3: 0.38 (0.18, 0.58); 0-1 **Low:** All ages: 0.62 (-0.09, 1.34); 0-1; <65: 1.78 (0.52, 3.05); 0-1; ≥ 65: 0.22 (-0.61, 1.05); 0-1; PM_{10}+NO_2: 0.24 (-0.49, 0.97); 0-1; PM_{10}+SO_2: 0.45 (-0.27, 1.17); 0-1; PM_{10}+O_3: 0.72 (0.00, 1.44); 0-1 **High:** All ages: 2.20 (0.74, 3.68); 0-1; <65: 2.34 (-0.09, 4.83); 0-1; ≥ 65: 2.14 (0.42, 3.89); 0-1; PM_{10}+NO_2: 1.87 (0.42, 3.35); 0-1; PM_{10}+SO_2: 2.12 (0.67, 3.60); 0-1; PM_{10}+O_3: 2.15 (0.55, 3.77); 0-1; **Cardiovascular:** **Normal:** All ages: 0.39 (0.11, 0.66); 0-1; <65: 0.17 (-0.40, 0.73); 0-1; ≥ 65: 0.44 (0.14, 0.74); 0-1; PM_{10}+NO_2: 0.11 (-0.23, 0.45); 0-1; PM_{10}+SO_2: 0.27 (-0.02, 0.55); 0-1; PM_{10}+O_3: 0.42 (0.15, 0.70) **Low:** All ages: 0.72 (-0.25, 1.70); 0-1; <65: 2.63 (0.67, 4.63); 0-1; ≥ 65: 0.24 (-0.84, 1.32); 0-1; PM_{10}+NO_2: 0.37 (-0.62, 1.38); 0-1; PM_{10}+SO_2: 0.50 (-0.47, 1.49); 0-1; PM_{10}+O_3: 0.82 (-0.16, 1.80); 0-1 **High:** All ages: 3.28 (1.24, 5.37); 0-1; <65: 4.32 (0.10, 8.71); 0-1; ≥ 65: 3.03 (0.77, 5.34); 0-1; PM_{10}+NO_2: 3.00 (0.95, 5.09); 0-1; PM_{10}+SO_2: 3.20 (1.16, 5.29); 0-1; PM_{10}+O_3: 3.71 (1.50, 5.96); 0-1 **Stroke:** **Normal:** All ages: 0.38 (0.06, 0.70); <65: 0.17 (-0.53, 0.88); 0-1; ≥ 65: 0.43 (0.07, 0.79); 0-1; PM_{10}+NO_2: 0.09 (-0.31, 0.49); 0-1; PM_{10}+SO_2: 0.31 (-0.03, 0.64); 0-1; PM_{10}+O_3: 0.38 (0.05, 0.71); 0-1 **Low:** All ages: 0.67 (-0.50, 1.85); 0-1; <65: 2.85 (0.34, 5.42); 0-1; ≥ 65: 0.11 (-1.22, 1.45); 0-1; PM_{10}+NO_2: 0.29 (-0.90, 1.51); 0-1; PM_{10}+SO_2: 0.53 (-0.65, 1.73); 0-1; PM_{10}+O_3: 0.69 (-0.48, 1.87); 0-1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			High: All ages: 2.35 (-0.03, 4.78); 0-1; <65: 4.54 (-0.79, 10.16); 0-1; ≥ 65: 1.83 (-0.83, 4.57); PM_{10}+NO_2: 2.05 (-0.34, 4.49); 0-1; PM_{10}+SO_2: 2.31 (-0.07, 4.74); 0-1; PM_{10}+O_3: 2.77 (0.25, 5.35); 0-1 **Cardiac:** **Normal:** All ages: 0.32 (-0.14, 0.79); 0-1; <65: -0.04 (-1.07, 1.01); 0-1; ≥ 65: 0.40 (-0.10, 0.91); 0-1; PM_{10}+NO_2: 0.02 (-0.57, 0.60); 0-1; PM_{10}+SO_2: 0.11 (-0.38, 0.61); 0-1; PM_{10}+O_3: 0.41 (-0.06, 0.89); 0-1 **Low:** All ages: 0.50 (-1.10, 2.13); 0-1; <65: 1.79 (-1.65, 5.35); 0-1; ≥ 65: 0.19 (-1.55, 1.95); 0-1; PM_{10}+NO_2: 0.12 (-1.53, 1.80); 0-1; PM_{10}+SO_2: 0.14 (-1.48, 1.78); 0-1; PM_{10}+O_3: 0.72 (-0.90, 2.37); 0-1 **High:** All ages: 3.31 (-0.22, 6.97); 0-1; <65: 2.71 (-4.58, 10.56); 0-1; ≥ 65: 3.45 (-0.41, 7.46); 0-1; PM_{10}+NO_2: 3.01 (-0.54, 6.69); 0-1; PM_{10}+SO_2: 3.17 (-0.37, 6.84); 0-1; PM_{10}+O_3: 4.92 (0.96, 9.03); 0-1 **Respiratory:** **Normal:** All ages: 0.80 (0.25, 1.35); 0-1; <65: -0.35 (-1.85, 1.18); 0-1; ≥ 65: 0.93 (0.38, 1.50); 0-1; PM_{10}+NO_2: 0.30 (-0.39, 0.99); 0-1; PM_{10}+SO_2: 0.64 (0.07, 1.22); 0-1; PM_{10}+O_3: 0.84 (0.28, 1.41); 0-1 **Low:** All ages: 1.07 (-0.76, 2.95); 0-1; <65: -1.13 (-6.33, 4.35); 0-1; ≥ 65: 1.30 (-0.57, 3.20); 0-1; PM_{10}+NO_2: 0.44 (-1.46, 2.36); 0-1; PM_{10}+SO_2: 0.80 (-1.05, 2.69); 0-1; PM_{10}+O_3: 1.11 (-0.73, 2.99); 0-1 **High:** All ages: 1.15 (-3.54, 6.07); 0-1; <65: -3.42 (-15.82, 10.80); 0-1; ≥ 65: 1.76 (-3.03, 6.78); 0-1; PM_{10}+NO_2: 0.63 (-4.07, 5.55); 0-1; PM_{10}+SO_2: 1.03 (-3.66, 5.94); 0-1; PM_{10}+O_3: 2.66 (-2.44, 8.02); 0-1 **Cardiopulmonary:** **Normal:** All ages: 0.45 (0.19, 0.70); 0-1; <65: 0.07 (-0.47, 0.61); 0-1; ≥ 65: 0.53 (0.25, 0.81); 0-1; PM_{10}+NO_2: 0.15 (-0.17, 0.47); 0-1; PM_{10}+SO_2: 0.34 (0.07, 0.61); 0-1; PM_{10}+O_3: 0.43 (0.17, 0.70); 0-1 **Low:** All ages: 0.69 (-0.22, 1.61); 0-1; <65: 1.95 (0.04, 3.90); 0-1; ≥ 65: 0.43 (-0.57, 1.44); 0-1; PM_{10}+NO_2: 0.33 (-0.61, 1.27); 0-1; PM_{10}+SO_2: 0.50 (-0.42, 1.43); 0-1; PM_{10}+O_3: 0.76 (-0.16, 1.68); 0-1 **High:** All ages: 3.02 (1.03, 5.04); 0-1; <65: 3.49 (-0.66, 7.81); 0-1; ≥ 65: 2.91 (0.74, 5.12); 0-1; PM_{10}+NO_2: 2.70 (0.72, 4.73); 0-1; PM_{10}+SO_2: 2.95 (0.96, 4.97); 0-1; PM_{10}+O_3: 3.32 (1.16, 5.53); 0-1
Reference: Ren et al. (2006) **Period of Study:** 1/1996–12/2001 **Location:** Brisbane, Australia	**Outcome:** Mortality: Non-accidental Cardiovascular (390-448) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, cubic spline **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 15.84 **Range (Min, Max):** (2.5, 60) **Copollutant:** O_3	The study presents quantitative results associated with an incremental increase in temperature, not PM_{10}.
Reference: Roberts (2004b) **Period of Study:** 1987–1994 **Location:** Cook County, Illinois; Allegheny County, Pennsylvania	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, smooth splines; Poisson GLM, natural cubic splines **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** Cook County Lower Temp.: 29.24 Middle Temp.: 30.03 Upper Temp.: 52.76 Allegheny County Lower Temp.: 16.50 Middle Temp.: 24.97 Upper Temp.: 55.42 Range (10th, 90th): Cook County Lower Tem.: (16.42, 46.42) Middle Temp.: (14.79,	**Increment:** 10 μg/m^3 **% Increase (SE); lag:** **GLM** **Cook** **α = 0.5** No Interaction: 0.288% (0.157); 0; Low Temp.: -0.272% (0.380); 0 Middle Temp.: 0.344% (0.165); 0 Upper Temp.: 0.281% (0.239); 0 No Interaction: 0.359% (0.149); 1 Low Temp.: -0.168% (0.372); 1 Middle Temp.: 0.361% (0.156); 1 Upper Temp.: 0.616% (0.250); 1 No Interaction: 0.465% (0.176); 0-1 ma Low Temp.: 0.043% (0.397); 0-1 ma Middle Temp.: 0.506% (0.184); 0-1 ma Upper Temp.: 0.464% (0.256); 0-1 ma No Interaction: 0.633% (0.214); 0-3 ma

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
		56.33) Upper Temp.: (30.81, 82.81) Allegheny County Lower Temp.: (5.14, 34.54) Middle Temp.: (8.91, 57.91) Upper Temp.: (30.91, 88.99)	Low Temp.: 0.365% (0.419); 0-3 ma Middle Temp.: 0.638% (0.222); 0-3 ma Upper Temp.: 0.718% (0.295); 0-3 ma **α = 1** No Interaction: 0.117% (0.157); 0 Low Temp.: -0.351% (0.406); 0 Middle Temp.: 0.161% (0.165); 0 Upper Temp.: 0.096% (0.264); 0 No Interaction: 0.141% (0.150); 1 Low Temp.: -0.366% (0.397); 1 Middle Temp.: 0.161% (0.156); 1 Upper Temp.: 0.301% (0.278); 1 No Interaction: 0.260% (0.181); 0-1 ma Low Temp.: -0.163% (0.431); 0-1 ma Middle Temp.: 0.305% (0.188); 0-1 ma Upper Temp.: 0.207% (0.291); 0-1 ma No Interaction: 0.289% (0.225); 0-3 ma Low Temp.: 0.014% (0.459); 0-3 ma Middle Temp.: 0.311% (0.231); 0-3 ma Upper Temp.: 0.301% (0.334); 0-3 ma **α = 2** No Interaction: 0.060% (0.158); 0; 0 Low Temp.: -0.464% (0.486); 0; 0 Middle Temp.: 0.115% (0.168); 0; 0 Upper Temp.: -0.022% (0.319); 0; 0 No Interaction: 0.101% (0.152); 1 Low Temp.: -0.432% (0.484); 1 Middle Temp.: 0.089% (0.160); 1 Upper Temp.: 0.455% (0.327); 1 No Interaction: 0.129% (0.184); 0-1 ma Low Temp.: -0.320% (0.546); 0-1 ma Middle Temp.: 0.157% (0.193); 0-1 ma Upper Temp.: 0.130% (0.346); 0-1 ma No Interaction: 0.090% (0.236); 0-3 ma Low Temp.: -0.319% (0.572); 0-3 ma Middle Temp.: 0.105% (0.244); 0-3 ma Upper Temp.: 0.193% (0.412); 0-3 ma **Allegheny** **α = 0.5** No Interaction: 0.078% (0.209); 0 Low Temp.: -0.759% (0.643); 0 Middle Temp.: 0.207% (0.216); 0 High Temp.: -0.367% (0.364); 0 No Interaction: 0.189% (0.206); 1 Low Temp.: -0.335% (0.691); 1 Middle Temp.: 0.293% (0.215); 1 High Temp.: -0.171% (0.349); 1 No Interaction: 0.224% (0.246); 0-1 ma Low Temp.: -0.753% (0.763); 0-1 ma Middle Temp.: 0.353% (0.253); 0-1 ma High Temp.: -0.142% (0.382); 0-1 ma No Interaction: 0.526% (0.300); 0-3 ma Low Temp.: 0.050% (0.733); 0-3 ma Middle Temp.: 0.688% (0.310); 0-3 ma High Temp.: -0.043% (0.436); 0-3 ma **α = 1** No Interaction: 0.078% (0.211); 0 Low Temp.: -0.694% (0.656); 0 Middle Temp.: 0.214% (0.219); 0 High Temp.: -0.533% (0.430); 0 No Interaction: 0.179% (0.207); 1 Low Temp.: -0.283% (0.718); 1 Middle Temp.: 0.273% (0.217); 1 High Temp.: -0.221% (0.396); 1 No Interaction: 0.221% (0.249); 0-1 ma Low Temp.: -0.731% (0.794); 0-1 ma Middle Temp.: 0.348% (0.258); 0-1 ma High Temp.: -0.253% (0.447); 0-1 ma No Interaction: 0.464% (0.309); 0-3 ma Low Temp.: 0.056% (0.780); 0-3 ma Middle Temp.: 0.626% (0.319); 0-3 ma High Temp.: -0.356% (0.516); 0-3 ma **α = 2** No Interaction: 0.034% (0.217); 0 Low Temp.: -1.059% (0.715); 0 Middle Temp.: 0.162% (0.230); 0

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			High Temp.: -0.233% (0.489); 0 No Interaction: 0.130% (0.214); 1 Low Temp.: -0.189% (0.800); 1 Middle Temp.: 0.157% (0.226); 1 High Temp.: 0.070% (0.471); 1 No Interaction: 0.183% (0.260); 0-1 ma Low Temp.: -0.918% (0.907); 0-1 ma Middle Temp.: 0.279% (0.273); 0-1 ma High Temp.: -0.001% (0.526); 0-1 ma No Interaction: 0.270% (0.331); 0-3 ma Low Temp.: -0.105% (0.898); 0-3 ma Middle Temp.: 0.394% (0.346); 0-3 ma High Temp.: -0.287% (0.615); 0-3 ma **GAM** **Cook** **α = 0.5** No Interaction: 0.438% (0.151); 0 Low Temp.: -0.178% (0.364); 0 Middle Temp.: 0.439% (0.163); 0 Upper Temp.: 0.627% (0.197); 0 No Interaction: 0.495% (0.144); 1 Low Temp.: -0.114% (0.361); 1 Middle Temp.: 0.460% (0.151); 1 Upper Temp.: 0.938% (0.208); 1 No Interaction: 0.710% (0.169); 0-1 ma Low Temp.: 0.151% (0.379); 0-1 ma Middle Temp.: 0.686% (0.180); 0-1 ma Upper Temp.: 0.952% (0.214); 0-1 ma No Interaction: 0.923% (0.203); 0-3 ma Low Temp.: 0.532% (0.402); 0-3 ma Middle Temp.: 0.855% (0.210); 0-3 ma Upper Temp.: 1.289% (0.251); 0-3 ma **α = 1** No Interaction: 0.190% (0.154); 0 Low Temp.: -0.338% (0.414); 0 Middle Temp.: 0.242% (0.162); 0 Upper Temp.: 0.161% (0.230); 0 o Interaction: 0.239% (0.146); 1 Low Temp.: -0.283% (0.406); 1 Middle Temp.: 0.248% (0.152); 1 Upper Temp.: 0.453% (0.244); 1 No Interaction: 0.353% (0.174); 0-1 ma Low Temp.: -0.074% (0.437); 0-1 ma Middle Temp.: 0.388% (0.182); 0-1 ma Upper Temp.: 0.345% (0.251); 0-1 ma No Interaction: 0.453% (0.213); 0-3 ma Low Temp.: 0.190% (0.460); 0-3 ma Middle Temp.: 0.455% (0.219); 0-3 ma Upper Temp.: 0.557% (0.294); 0-3 ma **α = 2** No Interaction: 0.071% (0.157); 0; 0 Low Temp.: -0.534% (0.478); 0; 0 Middle Temp.: 0.132% (0.165); 0; 0 Upper Temp.: 0.011% (0.264); 0; 0 No Interaction: 0.099% (0.150); 1 Low Temp.: -0.467% (0.472); 1 Middle Temp.: 0.109% (0.156); 1 Upper Temp.: 0.329% (0.278); 1 No Interaction: 0.168% (0.180); 0-1 ma Low Temp.: -0.371% (0.525); 0-1 ma Middle Temp.: 0.216% (0.188); 0-1 ma Upper Temp.: 0.116% (0.290); 0-1 ma No Interaction: 0.149% (0.227); 0-3 ma Low Temp.: -0.291% (0.557); 0-3 ma Middle Temp.: 0.174% (0.233); 0-3 ma Upper Temp.: 0.210% (0.340); 0-3 ma **Allegheny** **α = 0.5** No Interaction: 0.245% (0.203); 0 Low Temp.: -0.727% (0.648); 0 Middle Temp.: 0.314% (0.216); 0 High Temp.: 0.308% (0.287); 0 No Interaction: 0.446% (0.199); 1 Low Temp.: -0.307% (0.701); 1 Middle Temp.: 0.469% (0.211); 1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			High Temp.: 0.556% (0.285); 1 No Interaction: 0.522% (0.237); 0-1 ma Low Temp.: -0.646% (0.761); 0-1 ma Middle Temp.: 0.567% (0.251); 0-1 ma High Temp.: 0.640% (0.307); 0-1 ma No Interaction: 0.977% (0.282); 0-3 ma Low Temp.: 0.307% (0.733); 0-3 ma Middle Temp.: 1.027% (0.296); 0-3 ma High Temp.: 1.001% (0.352); 0-3 ma **α = 1** No Interaction: 0.107% (0.209); 0 Low Temp.: -0.819% (0.699); 0 Middle Temp.: 0.229% (0.219); 0 High Temp.: -0.214% (0.350); 0 No Interaction: 0.223% (0.205); 1 Low Temp.: -0.316% (0.751); 1 Middle Temp.: 0.295% (0.216); 1 High Temp.: 0.002% (0.341); 1 No Interaction: 0.267% (0.246); 0-1 ma Low Temp.: -0.797% (0.840); 0-1 ma Middle Temp.: 0.372% (0.257); 0-1 ma High Temp.: 0.035% (0.372); 0-1 ma No Interaction: 0.534% (0.302); 0-3 ma Low Temp.: 0.029% (0.810); 0-3 ma Middle Temp.: 0.660% (0.314); 0-3 ma High Temp.: 0.071% (0.431); 0-3 ma **α = 2** No Interaction: 0.061% (0.214); 0 Low Temp.: -1.048% (0.749); 0 Middle Temp.: 0.206% (0.226); 0 High Temp.: -0.332% (0.419); 0 No Interaction: 0.145% (0.211); 1 Low Temp.: -0.278% (0.816); 1 Middle Temp.: 0.210% (0.223); 1 High Temp.: -0.105% (0.394); 1 No Interaction: 0.180% (0.256); 0-1 ma Low Temp.: -1.028% (0.931); 0-1 ma Middle Temp.: 0.298% (0.269); 0-1 ma High Temp.: -0.114% (0.441); 0-1 ma No Interaction: 0.275% (0.324); 0-3 ma Low Temp.: -0.384% (0.915); 0-3 ma Middle Temp.: 0.436% (0.338); 0-3 ma High Temp.: -0.366% (0.513); 0-3 ma
Reference: Roberts (2004a) **Period of Study:** 1987–1994 **Location:** Cook County, Illinois	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Poisson GLM **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** Max = 89	The study does not present quantitative results.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Roberts (2005) **Period of Study:** \Cook County: 1987–2000. Allegheny County: 1987-1998 **Location:** Cook County, Illinois; Allegheny County, Pennsylvania	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** NR **β (SE); lag:** Standard Model Cook County 0.000127 (0.000264); 0 -0.000042 (0.000249); 1 -0.000441 (0.000246); 2 Allegheny County 0.000693 (0.000437); 0 0.000356 (0.000423); 1 0.000524 (0.000415); 2 Moving Total Model Cook County 0.000150 (0.000187); k = 2 -0.000047 (0.000153); k = 3 0.000009 (0.000133); k = 4 Allegheny County 0.000633 (0.000310); k = 2 0.000542 (0.000255); k = 3 0.000598 (0.000351); k = 4
Reference: Roberts (2006) **Period of Study:** 1987–2000 **Location:** Cook County, Illinois; Suffolk County, Massachusetts (NMMAPS)	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Poisson GLM **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Cook County: 33.7 (19.4) Suffolk County: 25.9 (11.8) Range (10th, 90th): Cook County: (13.4, 58.1) Suffolk County: (14.0, 41.7) **Copollutant (correlation):** Cook County CO: r = 0.30 NO_2: r = 0.53 SO_2: r = 0.45 O_3: r = 0.44 Suffolk County CO: r = 0.33 NO_2: r = 0.43 SO_2: r = 0.23 O_3: r = 0.36	**Increment:** Cook County: 19.4 µg/m³ Suffolk County: 14.0 µg/m³ **% Increase (SD); lag:** Cook County Standard Model: 0.49% (0.25); 0 Proposed Model: 0.29% (0.16); 0 Standard Model: 0.67% (0.25); 0-2 avg Proposed Model: 0.49% (0.25); 0-2 avg Suffolk County Standard Model: 0.88% (1.27); 0 Proposed Model: 0.85% (0.84); 0 Standard Model: 1.60% (0.71); 0-2 avg Proposed Model: 1.35% (0.73); 0-2 avg
Reference: Roberts and Martin (2006a) **Period of Study:** 1987–2000 **Location:** Cook County, Illinois (NMMAPS)	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Dose-response 1. Piecewise linear relationship (no-threshold) with change point at 25 µg/m³ and 50 µg/m³ 2. Piecewise linear relationship (threshold), exposure below 25 µg/m³ no effect, and exposures above 50 µg/m³ having a different effect then exposures between 25 µg/m³ and 50 µg/m³ **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **IQR (25th, 75th):** (23.9, 45.4) Suffolk County: (14.0, 41.7) **Copollutant (correlation):** NR	The study does not present quantitative results.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Roberts and Martin (2006b) **Period of Study:** 1987–2000 **Location:** 109 U.S. cities (NMMAPS)	**Outcome:** Mortality: Non-accidental; Cardiorespiratory **Study Design:** Time-series **Statistical Analyses:** Poisson; 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **IQR (25th, 75th):** NR **Copollutant (correlation):** NR	**Increment**: NR **β x 1000 (SE x 1000); lag:** Non-accidental Model 1 Base df: 0.079 (0.050); 0 Double df: 0.044 (0.046); 0 Half df: 0.107 (0.052); 0 Base df: 0.180 (0.044); 1 Double df: 0.149 (0.047); 1 Half df: 0.254 (0.048); 1 Base df: 0.059 (0.056); 2 Double df: 0.024 (0.056); 2 Half df: 0.143 (0.054); 2 Model 2 Base df: 0.115 (0.037); 0-2 ma Double df: 0.107 (0.034); 0-2 ma Half df: 0.145 (0.039); 0-2 ma Cardio-respiratory Model 1 Base df: 0.103 (0.068); 0 Double df: 0.056 (0.067); 0 Half df: 0.134 (0.066); 0 Base df: 0.232 (0.060); 1 Double df: 0.179 (0.067); 1 Half df: 0.309 (0.059); 1 Base df: 0.210 (0.078); 2 Double df: 0.144 (0.075); 2 Half df: 0.305 (0.079); 2 Model 2 Base df: 0.168 (0.047); 0-2 ma Double df: 0.140 (0.044); 0-2 ma Half df: 0.196 (0.051); 0-2 ma Notes: Model 1 uses current day's mortality count, while Model 2 uses a 3-day moving total mortality count.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Roberts and Martin (2007a) **Period of Study:** 1987–2000 **Location:** 8 U.S. cities and >100 U.S. cities (NMMAPS)	**Outcome:** Mortality: Total (non-accidental); Cardiorespiratory **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR	**Increment:** 10 µg/m^3 **β x 1000 (SE x 1000); lag:** 8 U.S. cities Distributed Lag Model: 0.229; 0-2 Weighted Model: 0.315; 0-2 Standard Model: 0.276; 0 -0.062; 1 0.476; 2 90 U.S. cities Total (non-accidental) Standard Model: 0.078 (0.039); 0 0.182 (0.037); 1 0.108 (0.036); 2 Moving Total Model: 0.131 (0.023); 0-2 Weighted Model: 0.274 (0.075); 0-2 Cardio-respiratory Standard Model: 0.096 (0.055); 0 0.232 (0.053); 1 0.226 (0.051); 2 Moving Total Model: 0.174 (0.032); 0-2 Weighted Model: 0.389 (0.105); 0-2 Notes: The 8 U.S. cities consist of Chicago, Cleveland, Denver, El Paso, Houston, Nashville, Pittsburgh, and Salt Lake City.
Reference: Roberts and Martin (2007b) **Period of Study:** 1987–2000 **Location:** 10 U.S. cities (NMMAPS)	**Outcome:** Mortality: Non-accidental **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Anchorage: 27.32 Chicago: 36.95 Cleveland: 39.83 Detroit: 40.78 El Paso: 40.14 Minneapolis/St. Paul: 28.01 Pittsburgh: 35.09 Salt Lake City: 37.40 Seattle: 28.72 Spokane: 34.52 **Range (Min, Max):** NR	**Increment:** NR **β Coefficient (SE); lag:** Pooled Estimates Combined Model (Unconstrained Distributed Lag Model + Piecewise Linear Dose-Response Function) Change-point: 60 µg/m^3 Slope below: 0.00130 (0.00016); 0-5 Slope above: -0.00163 (0.00026); 0-5 Change-point: 30 µg/m^3 Slope below: 0.00014 (0.00039); 0-5 Slope above: -0.00003 (0.00015); 0-5 Piecewise Linear Dose-Response Model Change-point: 60 µg/m^3 Slope below: 0.00044 (0.00011); 3-day ma Slope above: -0.00077 (0.00020); 3- day ma Change-point: 30 µg/m^3 Slope below: 0.00022 (0.00026); 3-day ma Slope above: -0.00004 (0.00011); 3-day ma Polynomial Distributed Lag Model (degree 2) 0.00046 (0.00011); 0-5

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Samoli et al. (2005) **Period of Study:** 1990–1997 **Location:** 22 European cities (APHEA-2)	**Outcome:** Mortality: All-cause (non-accidental) (<800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Time-series **Statistical Analyses:** Hierarchical modeling: 1. Poisson GAM, penalized splines; 2. Multivariate modeling **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg Median (SD) unit: Range: (Stockholm: 14 µg/m³ to Torino: 65 µg/m³) Percentile (90th): Range: (Stockholm: 27 µg/m³ to Torino: 129 µg/m³) **Copollutant (correlation):** BS	The study does not present quantitative results.
Reference: Schwartz (2004a) **Period of Study:** 1986–1993 **Location:** 14 U.S. cities	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Case-crossover; Time-series **Statistical Analyses:** Conditional logistic regression; Poisson **Age Groups:** All ages Notes: Case days matched to referent days that had the same temperature.	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m³ **% Increase (Lower CI, Upper CI); lag:** Overall: Two stage: 0.36% (0.22, 0.50); 1 Single stage: 0.33% (0.19, 0.46); 1 More winter temperature lags: Two Stage: 0.39% (0.23, 0.56); 1 One stage: 0.32% (0.19, 0.46); 1 Time stratified with temperature matching: Two Stage: 0.39% (0.19, 0.58); 1 One Stage: 0.53% (0.34, 0.72); 1 Poisson regression: 0.40% (0.18, 0.62); 1
Reference: Schwartz (2004b) **Period of Study:** 1986–1993 **Location:** 14 U.S. cities	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Case-crossover **Statistical Analyses:** Time-stratified conditional logistic regression **Age Groups:** All ages Notes: Case days matched to referent days based on concentration of gaseous air pollutants. Matched on the following conditions: 1. 24-h avg SO_2 within 1 ppb 2. Daily-maximum O_3 within 2 ppb 3. 24-h avg NO_2 within 1 ppb 4. 24-h avg CO within 0.03 ppm	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** Range: 23 to 36 µg/m³ IQR (25th, 75th): Range 25th: 17 to 24 µg/m³ Range 75th: 31 to 57 µg/m³ **Copollutant (correlation):** CO SO_2 NO_2 O_3	**Increment:** 10 µg/m³ **β x 1000 (SE x 1000); lag:** Matched on CO: 0.527 (0.251); 0-1 avg Matched on O_3: 0.451 (0.170); 0-1 avg Matched on NO_2: 0.784 (0.185); 0-1 avg Matched on SO_2: 0.811 (0.175); 0-1 avg
Reference: Sharovsky et al. (2004) **Period of Study:** 7/1996–6/1998 **Location:** São Paulo, Brazil	**Outcome:** Mortality: Myocardial infarction **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** ≥ 35	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 58.2 (25.8) **Range (Min, Max):** (23, 186) **Copollutant (correlation):** CO: r = 0.73 SO_2: r = 0.72	**Increment:** 10 µg/m³ **β (SE); lag:** PM_{10}: 0.001 (0.001) PM_{10}+CO+SO_2: 0.0004 (0.0008)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Simpson et al. (2005) **Period of Study:** 1/1996–12/1999 **Location:** 4 Australian cities	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Time-series; meta-analysis **Statistical Analyses:** Poisson GAM, natural splines; Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Brisbane: 16.60 Sydney: 16.30 Melbourne: 18.20 **Range (Min, Max):** Brisbane: (2.6, 57.6) Sydney: (3.7, 75.5) Melbourne: (3.3, 51.9) **Copollutant:** $PM_{2.5}$; CO; NO_2	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** 0.2% (-0.8, 1.2)
Reference: Slaughter et al. (2005) **Period of Study:** 1/1995–12/1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (9th, 95th):** (7.9, 41.9) µg/m^3 **Copollutant (correlation):** PM_{10} $PM_{10-2.5}$: r = 0.94 CO: r = 0.32	**Increment**:: 25 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** 1.00 (0.97, 1.03); 1 0.98 (0.95, 1.01); 2 1.00 (0.97, 1.03); 3
Reference: Staniswalis et al. (2005) **Period of Study:** 1992–1995 **Location:** El Paso, Texas	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Time-series **Statistical Analyses:** Poisson; Principal component analysis (PCA) **Age Groups:** All	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** (0.2, 133.4) **Notes:** The chemical composition and size distribution of PM was not available, therefore, the study used wind speed as a surrogate variable for the PM_{10} composition.	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Poisson regressioN: 1.7%; 3 PCA: 24-hly measurements: 2.06%; 3 Daily avg: 1.7%; 3

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Stafoggia et al. (2008) **Period of Study:** 1997–2004 **Location:** 9 Italian cities	**Outcome:** Mortality: Total (non-accidental) (<800) Cardiovascular (390-459) Respiratory (460-519) Other natural causes **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** ≥ 35	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD) unit:** Bologna: 50.4 (31.7) Florence: 37.5 (16.6) Mestre: 48.1 (26.8) Milan: 57.9 (38.0) Palermo: 36.2 (21.7) Pisa: 35.1 (14.9) Rome: 47.3 (19.9) Taranto: 59.8 (18.9) Turin: 71.5 (38.1) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** **Cardiovascular** All year: 0.63% (0.31, 1.38); 0-1 Winter: 0.15% (-0.29, 0.59); 0-1 Spring: 0.72% (-0.07, 1.52); 0-1 Summer: 2.90% (1.14, 4.69); 0-1 Fall: 1.37% (0.43, 2.32); 0-1 **Apparent Temperature** <50th Percentile: 0.31% (-0.06, 0.67); 0-1 50th-75th Percentile: 2.05% (0.47, 3.66); 0-1 >75th Percentile: 2.68% (1.20, 4.17); 0-1 **Respiratory** All year: 0.98% (0.27, 1.70); 0-1 Winter: 0.41% (-0.67, 1.51); 0-1 Spring: 2.99% (1.18, 4.83); 0-1 Summer: 3.89% (0.19, 7.73); 0-1 Fall: 0.45% (-1.11, 2.03); 0-1 **Apparent Temperature** <50th Percentile: 0.54% (-0.47, 1.57); 0-1 50th-75th Percentile: 3.15% (0.64, 5.73); 0-1 >75th Percentile: 4.12% (0.44, 7.93); 0-1 **Other natural causes** All year: 0.37% (0.09, 0.66); 0-1 Winter: 0.14% (-0.36, 0.63); 0-1 Spring: 0.29% (-0.47, 1.05); 0-1 Summer: 2.15% (0.90, 3.42); 0-1 Fall: 0.70% (-0.41, 1.83); 0-1 **Apparent Temperature** <50th Percentile: 0.07% (-0.27, 0.41); 0-1 50th-75th Percentile: 1.08% (-0.02, 2.19); 0-1 >75th Percentile: 2.30% (1.06, 3.56); 0-1 **Total (non-accidental)** All year: 0.53% (0.25, 0.80); 0-1 Winter: 0.20% (-0.08, 0.49); 0-1 Spring: 0.62% (0.14, 1.10); 0-1 Summer: 2.54% (1.31, 3.78); 0-1 Fall: 1.21% (0.37, 2.06); 0-1 **Apparent Temperature** <50th Percentile: 0.21% (-0.06, 0.47); 0-1 50th-75th Percentile: 1.60% (0.64, 2.57); 0-1 >75th Percentile: 2.55% (1.58, 3.52); 0-1 **β coefficient (SE); lag:** **Linear interaction PM_{10} and Apparent Temperature** **Cardiovascular** <50th Percentile: -0.000117 (0.000415); 0-1 50th-75th Percentile: 0.003445 (0.001407); 0-1 >75th Percentile: 0.002764 (0.001795); 0-1 **Respiratory** <50th Percentile: 0.001119 (0.000943); 0-1 50th-75th Percentile: -0.001120 (0.003480); 0-1 >75th Percentile: 0.005306 (0.004350); 0-1 **Other natural causes** <50th Percentile: 0.000411 (0.000383); 0-1 50th-75th Percentile: -0.001526 (0.001207); 0-1 >75th Percentile: 0.002564 (0.001958); 0-1 **Total (non-accidental)** <50th Percentile: 0.000246 (0.000269); 0-1 50th-75th Percentile: 0.000584 (0.000880); 0-1 >75th Percentile: 0.002396 (0.001629); 0-1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Stölzel et al. (2007) **Period of Study:** 9/1995–8/2001 **Location:** Erfurt, Germany	**Outcome:** Mortality: Total (non-accidental) (<800) Cardio-respiratory (390-459, 460-519, 785, 786) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD) unit:**: 31.9 (23.2) IQR (25th, 75th): (16.5, 39.5) **Copollutant (correlation):** $MC_{0.1\text{-}0.5}$: r = 0.85 $MC_{0.01\text{-}2.5}$: r = 0.84 NO: r = 0.54 NO_2: r = 0.62 CO: r = 0.50	**Increment:** 23 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** Total (non-accidental) 1.004 (0.980; 1.029); 0 1.004 (0.981; 1.027); 1 0.998 (0.976; 1.021); 2 0.984 (0.962; 1.006); 3 0.993 (0.972; 1.015); 4 0.990 (0.969; 1.012); 5 Cardio-respiratory 1.007 (0.981; 1.034); 0 1.006 (0.981; 1.032); 1 0.996 (0.971; 1.021); 2 0.977 (0.953; 1.002); 3 0.994 (0.970; 1.018); 4 0.993 (0.969; 1.017); 5
Reference: Sullivan et al. (2003) **Period of Study:** 1985–1994 **Location:** Western Washington	**Outcome:** Out-of-hospital cardiac arrest **Study Design:** Case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** 19-79 Study PopulatioN:Out-of-hospital cardiac arrests: 1,206	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** Lag 0: 28.05 Lag 1: 27.97 Lag 2: 28.40 **Range (Min, Max):** (7.38, 89.83) **Copollutant (correlation):** SO_2 CO **Notes:** Study used nephelometry to measure particles and equated the measurements to $PM_{2.5}$ concentrations.	**Increment::** 16.51 µg/m^3 **Odds Ratio (Lower CI, Upper CI); lag:** Overall 1.05 (0.87, 1.27); 0 0.91 (0.75, 1.11); 1 1.03 (0.82, 1.28); 2
Reference: Sunyer et al. (2002) **Period of Study:** 1985–1995 **Location:** Barcelona, Spain	**Outcome:** Mortality: Respiratory mortality **Study Design:** Case-crossover **Statistical Analyses:** Condition logistic regression **Age Groups:** >14 Study populatioN: Asthmatic individuals: 5,610	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** 61.2 **Range (Min, Max):** (17.3, 240.7) **Copollutant:** BS; NO_2; O_3; SO_2; CO	**Increment:** 32.7 µg/m^3 **Odds Ratio (Lower CI, Upper CI); lag:** Asthmatic individuals with 1 ED visit 0.884 (0.672, 1.162); 0-2 avg Asthmatic individuals with >1 ED visit 1.084 (0.661, 1.778); 0-2 avg Asthma/COPD individuals with >1 ED visit 1.011 (0.746, 1.368); 0-2 avg

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Touloumi et al. (2005) **Period of Study:** 1990–1997 **Location:** 7 European cities (London, Budapest, Stockholm, Zurich, Paris, Lyon, Madrid) (APHEA2)	**Outcome:** Mortality: Total (non-accidental) (<800) Cardiovascular (390-459) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Median (SD) unit:** London: 25.1 Budapest: 40.2 Stockholm: 13.7 Zurich: 27.5 Paris: 22.2 Lyon: 38.5 µ Madrid: 33.4 IQR (25th, 75th): London: (20.3, 33.9) Budapest: (34.3, 45.8) Stockholm: (10.3, 19.1) Zurich: (19.2, 38.5) Paris: (16.0, 33.0) Lyon: (29.7, 50.4) Madrid: (27.6, 41.0) **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **β (x 1000) (SE (x 1000)):** Total (non-accidental) No control: 0.4834 (0.1095) Reported Influenza Data Count ID: 0.4967 (0.1089) I1 ID: 0.4740 (0.1090) MI ID: 0.5019 (0.1096) RI-ID: 0.4735 (0.1091) SF ID: 0.6714 (0.1080) Estimated Influenza Data APHEA-2: 0.5550 (0.1076) I1 EID: 0.5640 (0.1073) MI EID: 0.5872 (0.1100) RI EID: 0.5872 (0.1074) SF EID: 0.6641 (0.1073) Cardiovascular No control: 0.8432 (0.1665) Reported Influenza Data Count ID: 0.8896 (0.1662) I1 ID: 0.8545 (0.1661) MI ID: 0.8693 (0.1674) RI-ID: 0.8649 (0.1665) SF ID: 1.0107 (0.1659) Estimated Influenza Data APHEA-2: 0.9389 (0.1654) I1 EID: 0.9485 (0.1648) MI EID: 1.0440 (0.1686) RI EID: 0.9718 (0.1653) SF EID: 1.0585 (0.1652) **Notes:** I1 = one indicator for all epidemics; M1 = multiple indicators, one per epidemic; R1 = indicators for intervals indicating the range of influenza counts; SF = separate smooth function during epidemic periods.
Reference: Tsai et al. (2003a) **Period of Study:** 1994–2000 **Location:** Kaohsiung, Taiwan	**Outcome:** Mortality: Total (non-accidental) (<800) Respiratory (460-519) Circulatory (390-459) **Study Design:** Bidirectional case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 81.45 **Range (Min, Max):** (20.50, 232.00) **Copollutant:** SO_2 NO_2 CO O_3	**Increment:** 67.00 µg/m^3 **Odds Ratio (Lower CI, Upper CI); lag:** Total (non-accidental) 1.000 (0.947, 1.056); 0-2 avg Respiratory 1.023 (0.829, 1.264); 0-2 avg Circulatory 0.971 (0.864, 1.092); 0-2 avg

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Vajanapoom et al. (2002) **Period of Study:** 1992–1997 **Location:** Bangkok, Thailand	**Outcome:** Mortality: Total (non-accidental) (<800) Respiratory (460-519) Cardiovascular (390-459) Other-causes **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, LOESS **Age Groups:** All ages 55-64 65-74 ≥ 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 68.0 (23.9) IQR (25th, 75th): (50.1, 80.7) **Copollutant (correlation):** NR	**Increment:** 30 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Total (non-accidental) All ages: 2.3% (1.3, 3.3); 0-4 ma 55-64: 1.5% (-0.8, 3.9); 0-4 ma 65-74: 4.2% (2.0, 6.3); 0-4 ma ≥ 75: 3.9% (2.1, 5.6); 0-4 ma Cardiovascular All ages: 0.8% (-0.9, 2.4); 0 55-64: -2.5% (-6.3, 1.3); 0 65-74: 2.9% (-0.7, 6.5); 0 ≥ 75: 1.6% (-1.8, 5.0); 0 Respiratory All ages: 5.1% (0.6, 9.6); 0-2 ma 55-64: 1.4% (-11.3, 14.2); 0-2 ma 65-74: 2.8% (-9.5, 15.2); 0-2 ma ≥ 75: 10.2% (-0.1, 20.5); 0-2 ma Other-causes All ages: 2.4% (1.3, 3.5); 0-4 ma 55-64: 1.7% (-1.1, 4.5); 0-4 ma 65-74: 5.6% (3.1, 8.1); 0-4 ma ≥ 75: 3.7% (1.8, 5.6); 0-4 ma
Reference: Vedal et al. (2003) **Period of Study:** 1/1994–12/1996 **Location:** Vancouver, British Columbia, Canada	**Outcome:** Mortality: Total (non-accidental) (<800) Respiratory (460-519) Cardiovascular (390-459) **Study Design:** Time-series **Statistical Analyses: Poisson** GAM, LOESS **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 14.4 (5.9) **Range (Min, Max):** (4.1, 37.2) **Copollutant (correlation):** O_3: r = 0.48 SO_2: r = 0.76 NO_2: r = 0.84 CO: r = 0.71	The study does not present quantitative results
Reference: Venners et al. (2003) **Period of Study:** 1/1995–12/1995 **Location:** Chongqing, China	**Outcome:** Mortality: Total (non-accidental) (<800) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, cubic spline **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 146.8 **Range (Min, Max):** (44.7, 666.2) **Copollutant:** SO_2 **Notes:** PM_{10} was measured for only 7 months of the study period.	**Increment:** 100 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** 1.00 (0.93, 1.07); 0 0.98 (0.91, 1.04); 1 1.00 (0.93, 1.07); 2 0.96 (0.90, 1.03); 3 0.97 (0.90, 1.03); 4 0.99 (0.93, 1.06); 5

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Vichit-Vadakan et al. (2008) **Period of Study:** 1/1999–12/2003 **Location:** Bangkok, Thailand	**Outcome (ICD10):** Mortality: Non-accidental (A00-R99) Cardiovascular (I00-I99) Ischemic heart diseases (I20-I25) Stroke (I60-I69) Conduction disorder (I44-I49) Respiratory (J00-J98) Lower Respiratory Infection (J10-J22) COPD (J40-J47) Asthma (J45-J46) Senility (R54) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural cubic spline **Age Groups:** All ages 0-4 5-44 18-50 45-64 ≥ 50 ≥ 65 ≥ 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 52.1 (20.1) **Range (Min, Max):** (21.3, 169.2) **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Excess Risk (Lower CI, Upper CI); lag:** **Cause-specific mortality:** Nonaccidental: 1.3% (0.8, 1.7); 0-1 Cardiovascular: 1.9% (0.8, 3.0); 0-1 Ischemic heart disease: 1.5% (–0.4, 3.5); 0-1 Stroke: 2.3% (0.6, 4.0); 0-1 Conduction disorders: –0.%3 (–5.9, 5.6); 0-1 **Cardiovascular:** ≥ 65 1.8 (0.2, 3.3); 0-1 **Respiratory** All ages: 1.0 (–0.4, 2.4); 0-1 ≤ 1: 14.6 (2.9, 27.6); 0-1 ≥ 65: 1.3 (–0.8, 3.3); 0-1 **LRI:** <5: 7.7 (–3.6, 20.3); 0-1 COPD: 1.3 (–1.8, 4.4); 0-1 Asthma: 7.4 (1.1, 14.1); 0-1 Senility: 1.8 (0.7, 2.8); 0-1 **Age-specific for non-accidental** 0–4: 0.2 (–2.0, 2.4); 0-1 5–44: 0.9 (0.2, 1.7); 0-1 18–50: 1.2 (0.5, 1.9); 0-1 45–64: 1.1 (0.4, 1.9); 0-1 ≥ 50: 1.4 (0.9, 1.9); 0-1 ≥ 65: 1.5 (0.9, 2.1); 0-1 ≥ 75: 2.2 (1.3, 3.0); 0-1 **Sex-specific for non-accidental** Male: 1.2 (0.7, 1.7); 0-1; Female: 1.3 (0.7, 1.9); 0-1 **Non-accidental** 1.2 (0.8, 1.6); 0; 0.9 (0.6, 1.3); 1; 0.9 (0.5, 1.3); 2; 0.8 (0.4, 1.2); 3; 0.3 (–0.1, 0.7); 4; 1.3 (0.8, 1.7); 0-1; 1.4 (0.9, 1.9); 0-4; **Cardiovascular** 1.5 (0.5, 2.6); 0; 1.7 (0.7, 2.7); 1; 1.6 (0.6, 2.6); 2; 0.8 (–0.1, 1.8); 3; –0.1 (–1.1, 0.9); 4; 1.9 (0.8, 3.0); 0-1; 1.9 (0.6, 3.2); 0-4 **Respiratory** 1.0 (–0.3, 2.3); 0 ; 0.8 (–0.5, 2.0); 1; 1.1 (–0.1, 2.3); 2; 1.3 (0.1, 2.6); 3; 0.7 (–0.6, 1.9); 4; 1.0 (–0.4, 2.4); 0-1; 1.9 (1.2, 2.6); 0-4 **≥ 65** 1.5 (0.9, 2.0); 0; 1.1 (0.6, 1.7); 1; 1.1 (0.6, 1.6); 2; 1.2 (0.6, 1.7); 3; 0.7 (0.2, 1.2); 4; 1.5 (0.9, 2.1); 0-1; 1.9 (1.2, 2.6); 0-4 **Sensitivity analysis:** **Nonaccidental (df):** 3: 1.3 (0.9, 1.8); 4: 1.2 (0.8, 1.7); 6: 1.3 (0.8, 1.7); 6, with SO_2: 1.2 (0.8, 1.7); 6, with NO_2: 1.0 (0.2, 1.8); 6, with O_3: 1.1 (0.6, 1.7); 9: 1.1 (0.7, 1.6); 12: 1.1 (0.6, 1.5); 15: 1.2 (0.7, 1.6) **Cardiovascular (df):** 3: 1.8 (0.8, 2.7); 4: 1.6 (0.7, 2.6); 6: 1.7 (0.7, 2.7); 6, with SO_2: 2.0 (0.9, 3.3); 6, with NO_2: 2.3 (0.2, 4.3); 6, with O_3: 1.8 (0.5, 3.2); 9: 1.7 (0.6, 2.8); 12: 1.8 (0.7 to 3.0); 15: 2.2 (0.9, 3.4)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2003) **Period of Study:** 1986–1999 **Location:** Vancouver, Canada	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular (401-440) Respiratory (460-519) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines **Age Groups:** ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Daily 14.0 Every 6th Day 19.6 **Range (Min, Max):** Daily (3.8, 52.2) Every 6th Day (3.5, 63.0) **Copollutant:** SO_2 CO NO_2 O_3 $PM_{2.5}$ $PM_{10-2.5}$	**Increment:** 15.4 μg/m^3 **% Increase (Lower CI, Upper CI); lag:** Non-accidental 3.7% (-0.5, 8.0); 0-2 avg 2.6% (-0.9, 6.1); 0 2.7% (-0.7, 6.2); 1 1.9% (-1.4, 5.3); 2 Cardiovascular 3.4% (-2.7, 9.8); 0-2 avg 5.1% (0.0, 10.4); 0 1.3% (-3.8, 6.7); 1 0.6% (-4.3, 5.7); 2 Respiratory PM_{10} 0.1% (-9.5, 10.8); 0-2 avg 1.0% (-7.5, 10.4); 0 0.4% (-7.7, 9.3); 1 -1.3% (-8.9, 7.1); 2 Cancer 1.2% (-6.9, 10.1); 0-2 avg -2.5% (-8.8, 4.3); 0 2.3% (-4.6, 9.6); 1 3.3% (-3.7, 10.8); 2
Reference: Welty et al. (2008) **Period of Study:** 1987–2000 **Location:** Chicago, Illinois	**Outcome:** Mortality: Total (non-accidental) **Study Design:** Time-series **Statistical Analyses:** Poisson–Gibbs Sampler; Bayesian Distributed Lag Model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 μg/m^3 % Excess Risk (Lower CI, Upper CI); lag: Poisson–Gibbs Sampler 0.17% (0.01, 0.34); 3 -0.24% (-0.73, 0.23); 0-14 Unconstrained: -0.19% (-0.86, 0.48); 0-14 Bayesian Distributed Lag Model -0.21% (-0.86, 0.41); 0-14

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Welty and Zeger (2005) **Period of Study:** 1987–2000 **Location:** 100 U.S. cities (NMMAPS)	**Outcome:** Mortality: Total (non-accidental) (<800) **Study Design:** Time-series **Statistical Analyses:** Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 μg/m^3 **% Increase (SE); lag:** Distributed Lag Model: Seasonally-Temporally Varying Temperature variables: 0, 1-2, 1-7, 1-14 S(t, 1 × years): 0.229 (0.053); 1 S(t, 2 × years): 0.220 (0.053); 1 S(t, 4 × years): 0.187 (0.050); 1 S(t, 8 × years): 0.178 (0.049); 1 Temperature variables: 0, 1-2, 1-7, 1-14, 0×1-2, 0×1-7, 1-2 × 1-7 S(t, 1 × years): 0.195 (0.048); 1 S(t, 2 × years): 0.200 (0.051); 1 S(t, 4 × years): 0.176 (0.050); 1 S(t, 8 × years): 0.149 (0.050); 1 Distributed Lag Model: Nonlinear Temperature variables: 0, 1-2, 1-7, 1-14 S(t, 4 × years): 0.239 (0.053); 1 Temperature variables: 0, 1-2, 1-7, 1-14, 0×1-2, 0×1-7, 1-2 × 1-7 S(t, 4 × years): 0.172 (0.045); 1 Temperature variables: S(0,2), S(1-2,2), S(1-7,2), S(1-14,2) S(t, 4 × years): 0.186 (0.046); 1 Temperature variables: S(0,2), S(1-2,2), S(1-7,2), S(1-14,2), S(0×1-2,2), S(0×1-7,2), S(1-2 × 1-7,2) S(t, 4 × years): 0.189 (0.047); 1 Temperature variables: S(0,4), S(1-2,4), S(1-7,4), S(1-14,4) S(t, 4 × years): 0.175 (0.046); 1 Temperature variables: S(0,4), S(1-2,4), S(1-7,4), S(1-14,4), S(0×1-2,4), S(0×1-7,4), S(1-2 × 1-7,4) S(t, 4 × years): 0.190 (0.048); 1 Temperature variables: 0, 1-2, 1-7 S(t, 4 × years): 0.252 (0.053); 1 Temperature variables: 0, 1-2, 1-7, 0×1-2, 0×1-7, 1-2 × 1-7 S(t, 4 × years): 0.186 (0.044); 1 Temperature variables: S(0,2), S(1-2,2), S(1-7,2) S(t, 4 × years): 0.198 (0.046); 1 Temperature variables: S(0,2), S(1-2,2), S(1-7,2), S(0×1-2,2), S(0×1-7,2), S(1-2 × 1-7,2) S(t, 4 × years): 0.201 (0.047); 1 Temperature variables: S(0,4), S(1-2,4), S(1-7,4) S(t, 4 × years): 0.189 (0.045); 1 Temperature variables: S(0,4), S(1-2,4), S(1-7,4), S(0×1-2,2), S(0×1-7,4), S(1-2 × 1-7,2) S(t, 4 × years): 0.205 (0.047); 1 Temperature variables: S(0,4), S(1-2,4) S(t, 4 × years): 0.250 (0.045); 1 Temperature variables: S(0,4), S(1-2,4), S(0×1-2,4) S(t, 4 × years): 0.253 (0.044); 1 Temperature variables: S(0,4) S(t, 4 × years): 0.220 (0.045); 1 Notes: 0 indicates current-day temperature; 1-r indicates avg of lag 1 through lag r temperature; S(, ρ) indicates a natural spline smooth with ρ degrees of freedom. S(t, α x years) indicates the natural spline smooth of time with degrees of freedom equal to α x (number of years of data).

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Wong et al. (2007) **Period of Study:** 1/1998–12/1998 **Location:** Hong Kong, China	**Outcome:** Mortality: Total (non-accidental) (<800) Cardiorespiratory (390-519) **Study Design:** Main analysis: Time-series **Sensitivity analysis:** Case-crossover, case-only **Statistical Analyses:** Main analysis: Poisson GAM **Sensitivity analysis:** Conditional logistic regression **Age Groups:** ≥ 30 ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 48.1 (24.3) **Range (Min, Max):** (15.5, 140.5) **Copollutant:** NO_2 SO_2 O_3	**Increment:** 10 µg/m^3 **% Excess Risk (Lower CI, Upper CI); lag:** Main Analysis Non-accidental Smokers: ≥ 301: .80% (0.35, 3.26); 0; 1.77% (0.46, 3.11); 2 ≥ 65: 3.20% (1.36, 5.07); 0; 2.42% (0.73, 4.13); 2 Never-smokers ≥ 30: -0.37% (-2.23, 1.52); 0; -0.03% (-1.72, 1.66); 2 ≥ 65P -0.70% (-2.81, 1.46); 0; -0.13% (-2.04, 1.80); 2 Cardiorespiratory Smokers ≥ 30: 1.43% (-0.86, 3.78); 0; 2.32% (0.24, 4.44); 2 ≥ 65: 2.98% (0.47, 5.55); 0; 2.61% (0.31, 4.95); 2 Never-smokers ≥ 30: 0.02% (-2.75, 2.87); 0; -0.79% (-3.33, 1.82); 2 ≥ 65: 0.25% (-2.62, 3.19); 0; -0.66% (-3.29, 2.04); 2 Sensitivity Analysis Poisson Regression Non-accidental ≥ 30: 1.81% (0.21, 3.44); 0; 1.93% (0.32, 3.56); 2; 1.99% (0.14, 3.87); 0-3 ≥ 65: 2.31% (0.37, 4.29); 0; 2.16% (0.20, 4.15); 2 2.57% (0.30, 4.89); 0-3 Cardiorespiratory ≥ 30: 1.04% (-1.45, 3.59); 0; 2.18% (-0.35, 4.77); 2 1.66% (-1.24, 4.64); 0-3 ≥ 65: 1.69% (-0.93, 4.37); 0; 2.44% (-0.23, 5.18); 2 2.30% (-0.80, 5.50); 0-3 Case-only: Logistic Regression Non-accidental ≥ 30: 1.79% (0.21, 3.37); 0; 1.94% (0.33, 3.56); 2 ≥ 65: 2.30% (0.42, 4.17); 0; 2.16% (0.26, 4.07); 2 Cardiorespiratory ≥ 30: 1.01% (-1.37, 3.40); 0; 2.16% (-0.28, 4.61); 2 ≥ 65: 1.65% (-0.96, 4.27); 0; 2.42% (-0.27, 5.12); 2 Case-crossover Non-accidental ≥ 30: 2.54% (0.35, 4.78); 0; 1.35% (-0.81, 3.56); 2 ≥ 65: 3.96% (1.37, 6.63); 0; 2.20% (-0.35, 4.81); 2 Cardiorespiratory ≥ 30: 0.48% (-2.74, 3.80); 0; 3.24% (-0.03, 6.61); 2 ≥ 65: 2.17% (-1.40, 5.86); 0; 3.43% (-0.13, 7.13); 2

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Wong et al. (2007) **Period of Study:** 1/1998–12/1998 **Location:** Hong Kong, China	**Outcome:** Mortality: Total (non-accidental) (<800) Cardiorespiratory (390-519) **Study Design:** Main analysis: Time-series **Sensitivity analysis:** Case-only **Statistical Analyses:** Main analysis: Poisson GAM, natural cubic spline **Sensitivity analysis:** Logistic regression **Age Groups:** ≥ 30 ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 48.1 (24.3) **Range (Min, Max):** (15.5, 140.5) **Copollutant:** NO_2 SO_2 O_3	**Increment:** 10 µg/m^3 **% Excess Risk (Lower CI, Upper CI); lag:** **Non-accidental** **Exercise** ≥ 30: 0.13% (-1.16, 1.44); 1; ≥ 65: 0.24% (-1.16, 1.67); 1 **Never-exercise** ≥ 30: 1.04% (0.07, 2.02); 1; ≥ 65: 1.26% (0.27, 2.27); 1 **Cardio-respiratory** **Exercise** ≥ 30: 0.46% (-1.43, 2.39); 1; ≥ 65: 0.30% (-1.65, 2.29); 1 **Never-exercise** ≥ 30: 0.97% (-0.36, 2.32); 1; ≥ 65: 0.98% (-0.45, 2.43); 1 **Difference in % Excess Risk (Exercise vs. Never-Exercise)** **Non-accidental** **Poisson Regression** ≥ 30: -2.86% (-4.03 to -1.67); 1; ≥ 65: -3.06% (-4.37 to -1.74); 1 **Case-only** ≥ 30: -2.91% (-4.04 to -1.77); 1; ≥ 65: -3.12% (-4.38 to -1.84); 1 **Cardiorespiratory** **Poisson regression** ≥ 30: -2.55% (-4.32 to -0.75); 1; ≥ 65: -2.64% (-4.48 to -0.76); 1 **Case-only** ≥ 30: -2.63% (-4.32 to -0.92); 1; ≥ 65: -2.73% (-4.50 to -0.92); 1 **Adjusted Case-only** **Non-accidental** **Sex** ≥ 30: -2.88% (-1.73 to -4.01); 1; ≥ 65: -3.09% (-1.82 to -4.35); 1 **Education** ≥ 30: -2.94% (-1.80 to -4.07); 1; ≥ 65: -3.18% (-1.90 to -4.44); 1 **Job** ≥ 30: -2.88% (-1.74 to -4.02); 1; ≥ 65: -3.11% (-1.83 to -4.37); 1 **Smoking** ≥ 30: -2.82% (-1.66 to -3.96); 1; ≥ 65: -2.97% (-1.68 to -4.25); 1 **Illness time** ≥ 30: -2.94% (-1.80 to -4.07); 1; ≥ 65: -3.16% (-1.88 to -4.42); 1 **Cardiorespiratory** **Sex** ≥ 30: -2.61% (-0.89 to -4.29); 1; ≥ 65: -2.71% (-0.90 to -4.48); 1 **Education** ≥ 30: -2.58% (-0.85 to -4.27); 1; ≥ 65: -2.77% (-0.95 to -4.54); 1 **Job** ≥ 30: -2.68% (-0.96 to -4.37); 1; ≥ 65: -2.68% (-0.88 to -4.46); 1 **Smoking** ≥ 30: -2.46% (-0.73 to -4.17); 1; ≥ 65: -2.50% (-0.68 to -4.29); 1 **Illness Time** ≥ 30: -2.63% (-0.91 to -4.32); 1; ≥ 65: -2.73% (-0.92 to -4.51); 1 **Case-only by Excercise Group (Never as Reference)** **Non-accidental** **≥ 30** Low: -3.34% (-5.77 to -0.85); 1; Moderate: -6.32% (-8.55 to -4.03); 1; High: -1.74% (-3.06 to -0.40); 1 **≥ 65** Low: -3.79% (-6.67 to -0.82); 1; Moderate: -7.78% (-10.39 to -5.10); 1; High: -1.77% (-3.21 to -0.31); 1 **Cardiorespiratory** **≥ 30** Low: -3.95% (-7.77, 0.04); 1; Moderate: -8.50% (-11.84 to -5.02); 1; High: -0.62% (-2.58, 1.38); 1 **≥ 65** Low: -3.97% (-8.17, 0.43); 1; Moderate: -9.42% (-13.00 to -5.69); 1; High: -0.68% (-2.71, 1.38); 1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Wong et al. (2002) **Period of Study:** 1995-1998 **Location:** Hong Kong, China	**Outcome:** Mortality: Respiratory (461-519) COPD (490-496) Pneumonia & Influenza (480-487) Cardiovascular (390-459) IHD (410-414) Cerebrovascular (430-438) **Study Design:** Time-series **Statistical Analyses:** Poisson **Age Groups:** ≥ 30 ≥ 65	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 51.53 (24.79) **Range (Min, Max):** (14.05, 163.79) **Copollutant (correlation):** NO_2: r = 0.780 SO_2: r = 0.344 O_3: r = 0.538	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** Respiratory 1.008 (1.001 to 1.014); 1 COPD 1.017 (1.002, 1.033); 0-3 Pneumonia & Influenza 1.007 (0.999, 1.015); 2 Cardiovascular 1.003 (0.998, 1.016); 2 IHD 1.013 (1.001, 1.025); 0-3 Cerebrovascular 1.007 (0.998, 1.016); 2 Respiratory PM_{10}+SO_2+O_3+NO_2: 1.005 (0.992, 1.010); 1 COPD PM_{10}+SO_2+O_3+NO_2: 0.991 (0.968, 1.015); 0-3 PM_{10}+O_3+NO_2: 0.993 (0.970, 1.016); 0-3 Pneumonia & Influenza PM_{10}+SO_2+O_3+NO_2: 1.002 (0.991, 1.013); 2 IHD 0.994 (0.978, 1.009); 0-3
Reference: Wong et al. (2008b) **Period of Study:** Bangkok: 1999–2003 Hong Kong: 1996–2002 Shanghai & Wuhan: 2001–2004 **Location:** Bangkok, Thailand; Hong Kong, Shanghai, and Wuhan, China	**Outcome (ICD10):** Mortality: Natural causes (A00-R99) Cardiovascular (I00-I99) Respiratory (J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages ≥ 65 ≥ 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Bangkok: 52.0 Hong Kong: 51.6 Shanghai: 102.0 Wuhan: 141.8 **Range (Min, Max):** Bangkok: (21.3, 169.2) Hong Kong: (13.7, 189.0) Shanghai: (14.0, 566.8) Wuhan: (24.8, 477.8) **Copollutant:** NO_2 SO_2 O_3	**Increment:** 10 µg/m^3 **% Excess Risk (Lower CI, Upper CI); lag:** Random Effects (4 cities) Natural causes: 0.55% (0.26, 0.85); 0-1 Cardiovascular: 0.58% (0.22, 0.93); 0-1 Respiratory: 0.62% (0.22, 1.02); 0-1 Random Effects (3 Chinese cities) Natural causes: 0.37% (0.21, 0.54); 0-1 Cardiovascular: 0.44% (0.19, 0.68); 0-1 Respiratory: 0.60% (0.16, 1.04); 0-1 Sensitivity Analysis Random Effects (4 cities) Omit PM_{10}>95th: 0.53% (0.27, 0.78); 0-1 Omit PM_{10}>75th: 0.53% (0.29, 0.78); 0-1 Omit PM_{10}>180 µg/m^3: 0.65% (0.24, 1.06); 0-1 Omit stations with high traffic source: 0.55% (0.26, 0.85); 0-1 Warm season-dichotomous variables: 0.86% (0.11, 1.60); 0-1 Add temperature at lag 1-2 days: 0.51% (0.23, 0.79); 0-1 Add temperature at lag 3-7 days: 0.35% (0.14, 0.57); 0-1 Daily PM_{10} defined by centering: 0.54% (0.26, 0.82); 0-1 Natural spline with (8, 4, 4)df: 0.54% (0.26, 0.81); 0-1 Penalized spline: 0.52% (0.26, 0.77); 0-1 Random Effects (3 Chinese cities) Omit PM_{10}>95th: 0.47% (0.21, 0.73); 0-1 Omit PM_{10}>75th: 0.55% (0.24, 0.85); 0-1 Omit PM_{10}>180 µg/m^3: 0.46% (0.15, 0.76); 0-1 Omit stations with high traffic source: 0.38% (0.20, 0.57); 0-1 Warm season-dichotomous variables: 0.43% (0.10, 0.76); 0-1 Add temperature at lag 1-2 days: 0.36% (0.18, 0.53); 0-1 Add temperature at lag 3-7 days: 0.25% (0.10, 0.40); 0-1 Daily PM_{10} defined by centering: 0.37% (0.21, 0.53); 0-1 Natural spline with (8, 4, 4)df: 0.36% (0.23, 0.49); 0-1 Penalized spline: 0.34% (0.23, 0.45); 0-1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Wong et al. (2008a) **Period of Study:** 1/1996–12/2002 **Location:** Hong Kong	**Outcome (ICD10):** Mortality: Non-accidental (A00-T99; Z00-Z99) Cardiovascular (I00-I99) Respiratory (J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 51.6 (25.3) **Range (Min, Max):** (13.5, 188.5) **Copollutant:** NO_2 SO_2 O_3	**Increment:** 10 µg/m^3 **% Excess Risk (Lower CI, Upper CI); lag:** **Non-accidental:** **Low SDI** 0.37 (–0.10, 0.84); 0; 0.40 (–0.04, 0.84); 1; 0.14 (–0.28, 0.57); 2; –0.12 (–0.55, 0.30); 3; –0.14 (–0.56, 0.28); 4 **Middle SDI** 0.70 (0.34, 1.07); 0; 0.48 (0.14, 0.82); 1; 0.35 (0.02, 0.68); 2; 0.18 (–0.14, 0.51); 3; 0.17 (–0.16, 0.50); 4 **High SDI** 0.22 (–0.29, 0.73); 0; 0.46 (–0.01, 0.94); 1; 0.29 (–0.17, 0.75); 2; –0.05 (–0.51, 0.40); 3; –0.06 (–0.51, 0.40); 4 **All areas** 0.45 (0.19, 0.72); 0; 0.40 (0.15, 0.64); 1; 0.22 (–0.02, 0.45); 2; 0.00 (–0.24, 0.23); 3; 0.03 (–0.20, 0.26); 4 **Cardiovascular:** **Low SDI** 0.14 (–0.77, 1.06); 0 ; 0.64 (–0.21, 1.49); 1; 0.24 (–0.58, 1.07); 2; –0.27 (–1.09, 0.55); 3; 0.01 (–0.80, 0.83); 4 **Middle SDI** 0.66 (0.00, 1.34); 0; 0.49 (–0.13, 1.12); 1; 0.80 (0.20, 1.40); 2; 0.65 (0.06, 1.25); 3; 0.52 (–0.07, 1.12); 4 **High SDI** 0.83 (–0.08, 1.75); 0; 0.89 (0.04, 1.75); 1; 0.12 (–0.70, 0.95); 2; –0.09 (–0.91, 0.73); 3; 0.04 (–0.77, 0.86); 4 **All areas** 0.52 (0.05, 1.00); 0; 0.58 (0.14, 1.03); 1; 0.43 (0.00, 0.86); 2; 0.14 (–0.28, 0.57); 3; 0.23 (–0.20, 0.65); 4 **Respiratory:** **Low SDI** 0 0.69 (–0.44, 1.82); 0; 1 0.55 (–0.50, 1.61); 1; 2 0.36 (–0.66, 1.39); 2; 3 –0.24 (–1.25, 0.78); 3; 4 –0.17 (–1.17, 0.85); 4 **Middle SDI** 0.31 (–0.50, 1.13); 0; 0.77 (0.01, 1.53); 1; 0.85 (0.12, 1.59); 2; 0.66 (–0.07, 1.39); 3; 0.69 (–0.03, 1.42); 4 **High SDI** 0.27 (–0.85, 1.40); 0 ; 0.72 (–0.32, 1.78); 1; 1.46 (0.45, 2.47); 2; 0.70 (–0.30, 1.71); 3; 0.48 (–0.52, 1.48); 4 **All areas** 0.39 (–0.20, 0.99); 0; 0.70 (0.15, 1.26); 1; 0.89 (0.36, 1.42); 2; 0.45 (–0.08, 0.98); 3; 0.43 (–0.10, 0.96); 4 **High SDI vs. Middle SDI** Non-accidental: 0.23 (-0.25, 0.72); 0-1 Cardiovascular: 0.49 (-0.40, 1.40); 0-1 Respiratory: 0.49 (-0.58, 1.58); 0-1 **High SDI vs. Low SDI** Non-accidental: 0.12 (-0.42, 0.67); 0-1 Cardiovascular: 0.82 (-0.20, 1.86); 0-1 Respiratory: -0.15 (-1.39, 1.10); 0-1 **Trend Test** Non-accidental: 0.04 (-0.15, 0.22); 0-1 Cardiovascular: 0.27 (-0.07, 0.61); 0-1 Respiratory: -0.04 (-0.46, 0.37); 0-1 SDI = Social Deprivation Index. The higher the SDI the lower the SES of the individual.
Reference: Yang et al. (2004a) **Period of Study:** 1994–1998 **Location:** Taipei, Taiwan	**Outcome:** Mortality: Non-accidental (<800) Circulatory (390-459) Respiratory (460-519) **Study Design:** Bi-directional case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** 51.99 **Range (Min, Max):** (13.71, 211.30) **Copollutant:** SO_2 NO_2 CO O_3	**Increment:** 31.43 µg/m^3 **Odds Ratio (Lower CI, Upper CI); lag:** Non-accidental 0.995 (0.971, 1.020); 0 Respiratory 0.986 (0.906, 1.074); 0 Circulatory 0.988 (0.942, 1.035)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Zanobetti et al. (2003) **Period of Study:** 1990–1997 **Location:** 10 European cities (APHEA2)	**Outcome:** Mortality: Non-accidental (<800) Circulatory (390-459) Respiratory (460-519) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** 15-64; 65-74; ≥ 75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Athens: 42.7 (12.9) Budapest: 41 (9.1) Lodz: 53.5 (15.5) London: 28.8 (13.7) Madrid: 37.8 (17.7) Paris: 22.5 (11.5) Prague: 76.2 (45.7) Rome: 58.7 (17.4) Stockholm: 15.5 (7.9) Tel Aviv: 50.3 (57.5) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 μg/m^3 **% Increase (Lower CI, Upper CI); lag:** **Cardiovascular:** 0.69% (0.31, 1.08); 0-1 avg **40-day distributed lag** 1.99% (1.44, 2.54); 4th degree; 1.97% (1.38, 2.55); Unrestricted **Respiratory:** 0.74% (-0.17, 1.66); 0-1 avg **40-day distributed lag** 4.21% (1.70, 6.79); 4th degree; 4.20% (1.08, 7.42); Unrestricted **Unrestricted distributed lags** **Cardiovascular** 1.34% (0.89, 1.79); 20; 1.72% (1.20, 2.25); 30; 1.97% (1.38, 2.55); 40 **Respiratory** 1.71% (-0.65, 4.12); 20; 2.62% (0.19, 5.11); 30; 4.20% (1.08, 7.42); 40 **40-day lags** **Non-accidental** **15-64** -0.25% (-0.87, 0.36); 4th degree; -0.01 (-0.76, 0.75); Unrestricted **65-74** 0.78% (0.23, 1.33); 4th degree; 0.74% (0.02, 1.45); Unrestricted **≥ 75** 1.84% (0.92, 2.78); 4th degree; 1.94% (1.07, 2.81); Unrestricted **Cardiovascular** **65-74** 2.06% (1.05, 3.09); 4th degree; 1.62 (0.54, 2.70); Unrestricted **≥ 75** 2.35% (1.42, 3.29); 4th degree; 2.52% (1.57, 3.48); Unrestricted **Respiratory** **≥ 75** 4.57% (1.25, 7.99); 4th degree; 4.52% (0.89, 8.28); Unrestricted

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Zeka et al. (2005) **Period of Study:** 1/1989–12/2000 **Location:** 20 U.S. cities	**Outcome (ICD10):** Mortality: All-cause (non-accidental) (V01-Y98) Heart Disease (I01-I51) IHD (I20-I25) Myocardial infarction (I21, I22) Dysrhythmias (I46-I49) Heart failure (I50) Stroke (I60-I69) Respiratory (J00-J99) Pneumonia (J12-J18) COPD (J40-J44, J47) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Birmingham: 31.9 (18.0) µg/m³ Boulder: 22.1 (11.3) Caton: 26.6 (11.5) Chicago: 33.7 (16.4) Cincinnati: 31.4 (13.9) Cleveland: 37.5 (18.7) Colorado Springs: 24.0 (13.2) Columbus: 28.5 (12.5) Denver: 28.5 (12.8) Detroit: 32.1 (17.7) Honolulu: 15.9 (6.8) Minneapolis: 24.7 (12.3) Nashville: 30.1 (12.1) New Haven: 25.4 (14.4) Pittsburgh: 30.2 (18.5) Provo: 33.7 (22.2) Seattle: 26.4 (14.7) Salt lake City: 35.0 (20.8) µ Terra Haute: 29.2 (14.6) µ Youngstown: 30.8 (13.9) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m³ **% Increase (Lower CI, Upper CI); lag:** **Single-lag model** **All-Cause (non-accidental)** 0.20% (0.08, 0.32); 0; 0.35% (0.21, 0.49); 1; 0.24% (0.14, 0.34); 2 **Respiratory** 0.34% (-0.07, 0.75); 0; 0.52% (0.15, 0.89); 1; 0.51% (0.16, 0.86); 2 **COPD** -0.06% (-0.63, 0.51); 0; 0.43% (-0.14, 1.00); 1; 0.39% (-0.16, 0.94); 2 **Pneumonia** 0.50% (0.09, 1.09); 0; 0.59% (-0.12, 1.30); 1; 0.82% (0.25, 1.39); 2 **Heart disease** 0.12% (-0.06, 0.30); 0; 0.30% (0.12, 0.48); 1; 0.37% (0.17, 0.57); 2 **IHD** 0.19% (-0.03, 0.41); 0; 0.41% (0.19, 0.63); 1; 0.43% (0.10, 0.76); 2 **Myocardial Infarction** 0.36% (-0.05, 0.77); 0; 0.17% (-0.18, 0.52); 1; 0.13% (-0.22, 0.48); 2 **Heart Failure** 0.17% (-0.63, 0.97); 0; -0.01% (-0.81, 0.79); 1; 0.78% (-0.004, 1.56); 2 **Dysrhythmias** -0.23% (-1.41, 0.95); 0; 0.37% (-0.47, 1.21); 1; 0.33% (-0.55, 1.21); 2 **Stroke** 0.09% (-0.49, 0.60); 0; 0.41% (-0.02, 0.84); 1; 0.14% (-0.27, 0.55); 2 **Unconstrained distributed lag model** **All-cause (non-accidental)** 0.45% (0.25, 0.65); 0-3 **Respiratory** 0.87% (0.38, 1.36); 0-3 **COPD** 0.43% (-0.35, 1.21); 0-3 **Pneumonia** 1.24% (0.46, 2.02); 0-3 **Heart Disease** 0.50% (0.25, 0.75); 0-3 **IHD** 0.65% (0.32, 0.98) **Myocardial Infarction** 0.36% (-0.25, 0.97); 0-3 **Heart Failure** 0.60% (-0.50, 1.70); 0-3 **Dysrhythmias** 0.20% (-1.03, 1.43); 0-3 **Stroke** 0.46% (-0.13, 1.05); 0-3
Reference: Zeka et al. (2006a) **Period of Study:** 1/1989–12/2000 **Location:** 20 U.S. cities	**Outcome (ICD10):** Mortality: All-cause (non-accidental) (V01-Y98) Heart Disease (I01-I51) Myocardial infarction (I21, I22) Stroke (I60-I69) Respiratory (J00-J99) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** All ages <65 65-75 >75	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD):** Birmingham: 31.9 (18.0) µg/m³ Boulder: 22.1 (11.3) Caton: 26.6 (11.5) Chicago: 33.7 (16.4) Cincinnati: 31.4 (13.9) Cleveland: 37.5 (18.7) Colorado Springs: 24.0 (13.2) Columbus: 28.5 (12.5) Denver: 28.5 (12.8) Detroit: 32.1 (17.7) Honolulu: 15.9 (6.8) Minneapolis: 24.7 (12.3) Nashville: 30.1 (12.1) New Haven: 25.4 (14.4) Pittsburgh: 30.2 (18.5) Provo: 33.7 (22.2) Seattle: 26.4 (14.7) Salt lake City: 35.0 (20.8)	**Increment:** 10 µg/m³ **% Increase (Lower CI, Upper CI); lag: All-cause (non-accidental)** Male: 0.46% (0.28, 0.64); 1-2 avg; Female: 0.37% (0.17, 0.57); 1-2 avg; White; 0.40% (0.22, 0.58); 1-2 avg; Black: 0.37% (-0.02, 0.76); 1-2 avg **Age:** <65: 0.25% (0.01, 0.49); 1-2 avg; 75: 0.23% (-0.06, 0.52); 1-2 avg; >75: 0.64% (0.44, 0.84); 1-2 avg **Educational Attainment:** Low (<8 yrs): 0.62% (0.29, 0.95); 1-2 avg; Medium (8–12 yrs): 0.36% (0.12, 0.60); 1-2 avg; High (>12 yrs): 0.27% (-0.004, 0.54); 1-2 avg **Location of Death:** In hospital: 0.22% (0.04, 0.40); 1-2 avg; Out of hospital: 0.71% (0.51, 0.91); 1-2 avg **Season:** Winter: 0.28% (0.04, 0.52); 1-2 avg; Summer: 0.19% (-0.22, 0.60); 1-2 avg; Transition (spring/fall): 0.49% (0.25, 0.73); 1-2 avg **Respiratory** Male: 0.71% (0.004, 1.42); 0-3; Female: 1.04% (0.33, 1.75); 0-3 White: 0.88% (0.33, 1.43); 0-3; Black: 0.71% (-0.56, 1.98); 0-3 **Age:** <65: 0.94% (-0.31, 2.19); 0-3; 65-75: 0.87% (-0.25, 1.99); 0-3 >75: 0.88% (0.17, 1.59); 0-3 **Educational Attainment:** Low (<8 yrs): 0.82% (-0.32, 1.96); 0-3; Medium (8–12 yrs): 0.88% (0.12, 1.64); 0-3; High (>12 yrs): 0.88% (-0.04, 1.80); 0-3 **Location of Death:** In hospital: 0.78% (0.17, 1.39); 0-3; Out of

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
		Terra Haute: 29.2 (14.6) Youngstown: 30.8 (13.9) **Range (Min, Max):** NR **Copollutant (correlation):** NR	hospital: 1.09% (0.25, 1.93); 0-3 **Season:** Winter: -0.007% (-0.87, 0.86); 0-3; Summer: 0.69% (-0.68, 2.06); 0-3; Transition (spring/fall): 1.57% (0.86, 2.28); 0-3 **Heart Disease** Male: 0.54% (0.23, 0.85); 2; Female: 0.46% (0.15, 0.77); 2 White; 0.50% (0.25, 0.75); 2; Black: 0.64% (0.13, 1.15); 2 **Age:** <65: 0.04% (-0.45, 0.53); 2; 65-75: 0.60% (0.13, 1.07); 2 >75: 0.65% (0.30, 1.00); 2 **Educational Attainment:** Low (<8 yrs): 0.72% (0.23, 1.21); 2; Medium (8–12 yrs): 0.38% (0.07, 0.69); 2; High (>12 yrs): 0.54% (0.13, 0.95); 2 **Location of Death:** In hospital: 0.15% (-0.14, 0.44); 2; Out of hospital: 0.93% (0.60, 1.26); 2 **Season:** Winter: 0.41% (-0.002, 0.82); 2; Summer: 0.52 (0.03, 1.01); 2; Transition (spring/fall): 0.56% (0.13, 0.99); 2 **Myocardial Infarction** Male: 0.21% (-0.40, 0.82); 0; Female: 0.59% (0.08, 1.10); 0 White; 0.24% (-0.27, 0.75); 0; Black: 0.99% (0.05, 1.93); 0 <65: 0.12% (-0.76, 1.00); 0; 65-75: 0.92% (0.21, 1.63); 0 >75: 0.16% (-0.58, 0.90); 0 **Educational Attainment:** Low (<8 yrs): 0.33% (-0.83, 1.49); 0; Medium (8–12 yrs): 0.79% (0.28, 1.30); 0; High (>12 yrs): -0.13% (-0.82, 0.56); 0 **Location of Death:** In hospital: 0.34% (-0.11, 0.79); 0; Out of hospital: 0.48% (-0.23, 1.19); 0 **Season:** Winter: 0.32% (-0.37, 1.01); 0; Summer: 0.30% (-0.82, 1.42); 0 **Transition (spring/fall):** 0.38% -0.31, 1.07); 0 **Stroke** Male: 0.11% (-0.58, 0.80); 1; Female: 0.59% (-0.04, 1.22); 1 White; 0.48% (0.01, 0.95); 1; Black: 0.13% (-0.87, 1.13); 1 **Age:** <65: 0.09% (-1.09, 1.27); 1; 65-75: -0.46% (-1.42, 0.50); 1 >75: 0.80% (0.27, 1.33); 1 **Educational Attainment:** Low (<8 yrs): 0.07% (-1.44, 1.58); 1; Medium (8–12 yrs): 0.29% (-0.32, 0.90); 1; High (>12 yrs): 0.52% (-0.28, 1.32); 1 **Location of Death:** In hospital: 0.06% (-0.49, 0.61); 1; Out of hospital: 0.87% (0.05, 1.69); 1 **Season:** Winter: -0.09% (-0.93, 0.75); 1; Summer: 0.67% (-0.31, 1.65); 1; Transition (spring/fall): 0.51% (-0.20, 1.22); 1 **Contributing causes of disease: All-cause** Secondary pneumonia present: 0.67% (0.16, 1.18); 1-2 avg Secondary pneumonia absent: 0.34% (0.16, 0.52); 1-2 avg Secondary heart failure present: 0.42% (0.01, 0.83); 1-2 avg Secondary heart failure absent: 0.37% (0.19, 0.55); 1-2 avg Secondary stroke present: 0.85% (0.30, 1.40); 1-2 avg Secondary stroke absent: 0.32% (0.14, 0.50); 1-2 avg Diabetes present: 0.57% (0.02, 1.12); 1-2 avg Diabetes absent: 0.34% (0.14, 0.54); 1-2 avg **Respiratory** Secondary pneumonia present: 1.28% (-0.33, 2.89); 0-3 Secondary pneumonia absent: 0.78% (0.15, 1.41); 0-3 Secondary heart failure present: 1.48% (0.07, 2.89); 0-3 Secondary heart failure absent: 0.79% (0.26, 1.32); 0-3 Secondary stroke present: 1.95% (-0.11, 4.01); 0-3 Secondary stroke absent: 0.80% (0.29, 1.31); 0-3 Diabetes present: 1.96% (-0.22, 4.14); 0-3 Diabetes absent: 0.82% (0.31, 1.33); 0-3 **Heart Disease** Secondary pneumonia present: 0.66% (-0.63, 1.95); 2 Secondary pneumonia absent: 0.49% (0.27, 0.71); 2 Secondary stroke present: 0.73% (-0.05, 1.51); 2 Secondary stroke absent: 0.48% (0.24, 0.72); 2 Diabetes present: 0.34% (-0.42, 1.10); 2 Diabetes absent: 0.52% (0.28, 0.76); 2 **Myocardial Infarction** Secondary pneumonia present: 1.54% (-1.05, 4.13); 0 Secondary pneumonia absent: 0.42% (0.05, 0.79); 0 Secondary stroke present: 0.50% (-1.38, 2.38); 0 Secondary stroke absent: 0.36% (-0.05, 0.77); 0 Diabetes present: 0.70% (-0.38, 1.78); 0 Diabetes absent: 0.41% (0.04, 0.78); 0 **Stroke** Secondary pneumonia present: 1.74% (0.35, 3.13); 1 Secondary pneumonia absent: 0.29% (-0.16, 0.74); 1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			Secondary heart failure present: 1.01% (-0.77, 1.79); 1 Secondary heart failure absent: 0.38% (-0.05, 0.81); 1 Diabetes present: 1.02% (-0.53, 2.57); 1 Diabetes absent: 0.37% (-0.08, 0.82); 1

Table E-18. Short-term exposure to $PM_{10-2.5}$ and mortality.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Burnett et al. (2004) **Period of Study:** 1981–1999 **Location:** 12 Canadian cities	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Time-series **Statistical Analyses:** 1. Poisson, natural splines 2. Random effects regression model **Age Groups:** All ages	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 11.4 **Range (Min, Max):** NR **Copollutant:** NO_2; O_3; SO_2; CO; PM_{10}; $PM_{2.5}$ **Note:** PM_{10} measurement calculated as the sum of $PM_{2.5}$ and $PM_{10-2.5}$ measurements.	**Increment:** 10 µg/m³ **% Increase (Lower CI, Upper CI); lag:** 1981–1999 $PM_{10-2.5}$: 0.31% (-0.66, 1.33); 1 $PM_{10-2.5}$+NO_2: 0.65% (-0.23, 1.59); 1
Reference: Kan et al. (2007a) **Period of Study:** 3/2004–12/2005 **Location:** Shanghai, China	**Outcome (ICD10):** Mortality: Total (non-accidental) (A00-R99) Cardiovascular (I00-I99) Respiratory (J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, penalized splines **Age Groups:** All ages	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 56.4 (1.34) **Range (Min, Max):** (8.3, 235.0) **Copollutant (correlation):** PM_{10}: r = 0.88 $PM_{2.5}$: r = 0.48 O_3: r = 0.07	**Increment:** 10 µg/m³ **% Increase (Lower CI, Upper CI); lag:** Total: 0.12% (-0.13, 0.36); 0-1 Cardiovascular: 0.34% (-0.05, 0.73); 0-1 Respiratory: 0.40% (-0.34, 1.13); 0-1
Reference: Kettunen et al. (2007) **Period of Study:** 1998–2004 **Location:** Helsinki, Finland	**Outcome (ICD10):** Mortality: Stroke (I60-I61, I63-I64) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, penalized thin-plate splines **Age Groups:** ≥ 65	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Median (SD) unit:** Cold Season: 6.7 Warm Season: 8.4 **Range (Min, Max):** Cold Season: (0.0, 101.4) Warm Season: (0.0, 42.0) **Copollutant:** O_3, CO, NO_2; PM_{10}; $PM_{2.5}$; UFP	**Increment:** Cold Season: 8.3 µg/m³ Warm Season: 5.7 µg/m³ **% Increase (Lower CI, Upper CI); lag:** Cold Season: -1.04% (-6.63, 4.89); 0 -2.49% (-7.57, 2.88); 1. -4.93% (-9.99, 0.41); 2 -4.33% (-9.32, 0.93); 3 Warm Season: 7.05% (-1.88, 16.80); 0 4.38% (-4.26, 13.81); 1: -1.19% (-9.45, 7.84); 2 1.42% (-6.79, 10.34); 3
Reference: Klemm et al. (2004) **Period of Study:** 8/1998–7/2000 **Location:** Fulton and DeKalb counties, Georgia (ARIES)	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular (390-459) Respiratory (460-519) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural cubic splines **Age Groups:** <65 ≥ 65	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 9.69 (3.94) **Range (Min, Max):** (1.71, 25.17) **Copollutant:** $PM_{2.5}$; O_3; NO_2; CO; SO_2; Acid; EC; OC; SO_4; Oxygenated Hydrocarbons; Nonmethane hydrocarbons; NO_3	**Increment:** NR **β (SE); lag:** Quarterly Knots: 0.00433 (0.00333); 0-1 Monthly Knots: 0.00617 (0.00360); 0-1 Biweekly Knots: 0.00516 (0.00381); 0-1
Reference: Slaughter et al. (2005) **Period of Study:** 1/1995–12/1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Non-accidental (< 800) **Study Design:** Time-series **Statistical Analyses:**Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Mean (SD) unit:**NR **Range (9th, 95th):** NR **Copollutant (correlation):** PM_1: r = 0.19 $PM_{2.5}$: r = 0.31 PM_{10}: r = 0.94 CO: r = 0.32	This study does not present quantitative results for $PM_{10-2.5}$.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Stieb et al. (2002) **Period of Study:** Publication dates of studies: 1985–12/2000 Mortality series: 1958–1999 **Location:** 40 cities (11 Canadian cities, 19 U.S. cities, Santiago, Amsterdam, Erfurt, 7 Korean cities)	**Outcome:** Mortality: All-cause (non-accidental) **Study Design:** Meta-analysis **Statistical Analyses:** Random effects model **Age Groups:** All ages	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** NR **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** Varied between studies: $PM_{2.5}$, O_3, SO_2, NO_2, CO	**Increment:** 13.0 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Single-pollutant models: 10 studies $PM_{10-2.5}$: 1.2% (0.5, 1.9) Multipollutant models: 6 studies $PM_{10-2.5}$: 0.9% (-0.3, 2.0)
Reference: Villeneuve et al. (2003) **Period of Study:** 1986–1999 **Location:** Vancouver, Canada	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular (401-440) Respiratory (460-519) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines **Age Groups:** ≥ 65	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** Daily: 6.1 Every 6th Day; 8.3 **Range (Min, Max):** Daily: (0.0, 72.0) Every 6th Day: (0.7, 35.0) **Copollutant:** $PM_{2.5}$ PM_{10} SO_2 CO NO_2 O_3	**Increment:** 11.0 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Non-accidental 1.4% (-2.5, 5.4); 0-2 avg 1.0% (-1.9, 4.0); 0 -1.1% (-4.0, 1.8); 1 2.0% (-1.0, 5.1); 2 Cardiovascular 5.9% (-0.2, 12.4); 0-2 avg 5.9% (1.1, 10.8); 0 1.4% (-3.3, 6.4); 1 2.2% (-2.0, 6.7); 2 Respiratory -1.0% (-9.8, 8.8); 0-2 avg -1.5% (-9.4, 7.1); 0 -1.5% (-8.4, 6.0); 1 0.1% (-6.4, 6.9); 2 Cancer 4.4% (-3.6, 13.1); 0-2 avg 3.1% (-2.9, 9.4); 0 -1.0% (-6.9, 5.3); 1 4.0% (-2.1, 10.4); 2
Reference: Wilson et al. (2007) **Period of Study:** 1995–1997 **Location:** Phoenix, Arizona	**Outcome:** Mortality: Cardiovascular **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, nonparametric smoothing spline **Age Groups:** >25	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Excess Risk (Lower CI, Upper CI); lag:** Central Phoenix: 2.4% (-1.2, 6.1); 0-5 ma Middle Phoenix: 3.8% (0.3, 7.5); 0-5 ma 3.4% (1.0, 5.8); 1 3.0% (0.7, 5.4); 2 Outer Phoenix: 1.6% (-1.9, 5.2); 0-5 ma

Table E-19. Short-term exposure to $PM_{2.5}$ (including PM components/sources) and mortality.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Basu et al. (2008) **Period of Study:** 5/1999–9/2003 **Location:** 9 California counties	**Outcome (ICD10):** Mortality: Non-accidental (V01-Y98) **Study Design:** (1) Main analysis: Case-crossover (2) **Sensitivity analysis:** Time-series **Statistical Analyses:** (1) Main anaylsis: conditional logistic regression (2) **Sensitivity analysis:** Poisson GAM **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SE) unit:** Contra Costa: 8.6 Fresno: 7.6 Kern: 11.3 Los Angeles: 19.8 Orange: 17.0 Riverside: 28.4 Sacramento: 8.8 San Diego: 13.4 Santa Clara: 10.8 IQR (25th, 75th): Contra Costa: (5.8, 10.1) Fresno: (3.8, 9.8) Kern: (8.0, 13.5) Los Angeles: (14.7, 23.3) Orange: (11.8, 21.0) Riverside: (17.9, 36.1) Sacramento: (5.8, 10.1) San Diego: (10.3, 15.8) Santa Clara: (7.2, 13.8) **Copollutant (correlation):** PM_{10}; r = 0.45 O_3 (1hr); r = 0.28 O_3 (8hr); r = 0.22 CO; r = 0.45 NO_2; r = 0.43	The study does not provide results quantitatively.
Reference: Dominici et al. (2007b) **Period of Study:** PM_{10}: 1987–2000. $PM_{2.5}$: 1999–2000 **Location:** 100 U.S. counties (NMMAPS)	**Outcome:** Mortality: All-cause (non-accidental) Cardiorespiratory Other-cause **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 μg/m^3 **% Increase (Lower CI, Upper CI); lag:** 1999-2000: All-cause: 0.29% (0.01, 0.57); 1 Cardiorespiratory: 0.38% (-0.07, 0.82); 1
Reference: Dominici et al. (2007a) **Period of Study:** 2000–2005 **Location:** 72 U.S. counties representing 69 communities	**Outcome:** Total mortality **Study Design:** Time-series **Statistical Analyses:** 2-stage Bayesian hierarchical model **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$, Nickel, speciated fine PM, and Vanadium **Averaging Time:** Annual avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	The study does not provide results quantitatively. **Note:** The study investigated whether county-specific short-term effects of PM_{10} on mortality are modified by long-term county-specific nickel or vanadium $PM_{2.5}$ concentrations.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Franklin et al. (2007) **Period of Study:** 1997–2002 **Location:** 27 U.S. communities	**Outcome:** Mortality: All-cause (non-accidental (<800) Cardiovascular (390-429) Respiratory (460-519) Stroke (430-438) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 15.7 µg/m^3 **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** All-cause (non-accidental): 0.67% (-0.12, 1.46); 0 1.21% (0.29, 2.14); 10.82% (0.02, 1.63); 0-1 Respiratory: 1.31% (-0.10, 2.73); 0 1.78% (0.20, 3.36); 1; 1.67% (0.19, 3.16); 0-1 Cardiovascular: 0.34% (-0.61, 1.28); 0 0.94% (-0.14, 2.02); 1. 0.54% (-0.47, 1.54); 0-1 Stroke: 0.62% (-0.69, 1.94); 0 1.03% (0.02, 2.04); 1. 0.67% (-0.23, 1.57); 0-1 Age≥ 75: All cause: 1.66% (0.62, 2.70); 1 Respiratory: 1.85% (0.27, 3.44); 1 Cardiovascular: 1.29% (0.15, 2.42); 1 Stroke: 1.52% (0.37, 2.67); 1 Age<75: All cause: 0.62% (-0.30, 1.55); 1 Respiratory: 1.53% (-0.67, 3.74); 1 Cardiovascular: 0.26% (-1.04, 1.56); 1 Stroke: -0.78% (-2.32, 0.76); 1 Male: All cause: 1.06% (0.07, 2.06); 1 Respiratory: 1.90% (0.14, 3.65); 1 Cardiovascular: 0.52% (-0.63, 1.66); 1 Stroke: 0.79% (-0.42, 2.02); 1 Female: All cause: 1.34% (0.40, 2.27); 1 Respiratory: 1.57% (-0.22, 3.35); 1 Cardiovascular: 1.30% (0.14, 2.46); 1 Stroke: 0.79% (-0.51, 2.09); 1 East: All cause: 1.95% (0.50, 3.40); 1 Respiratory: 2.66% (0.33, 5.00); 1 Cardiovascular: 1.52% (0.06, 2.98); 1 Stroke: 1.16% (-0.40, 2.73); 1 West: All cause: 0.05% (-1.80, 1.89); 1 Respiratory: 0.67% (-2.00, 3.34); 1| Cardiovascular: 0.11% (-2.03, 2.24); 1| Stroke: 0.94% (-0.38, 2.26); 1 $PM_{2.5}$>15 µg/m^3: All cause: 1.10% (-0.43, 2.64); 1 Respiratory: 1.42% (-0.84, 3.68); 1 Cardiovascular: 0.88% (-0.87, 2.62); 1 Stroke: 0.91% (-0.28, 2.10); 1 $PM_{2.5}$≤ 15 µg/m^3: All cause: 1.41% (-0.49, 3.30); 1 Respiratory: 2.46% (-0.49, 5.42); 1 Cardiovascular: 1.09% (-1.15, 3.32); 1 Stroke: 1.36% (-0.56, 3.27); 1 Effect of A/C at percentile of air conditioning prevalence: 25th percentile (45% prevalence of A/C): All cause: 1.50% (0.13, 2.88); 1 Respiratory: 2.27% (0.27, 4.27); 1 Cardiovascular: 1.04% (-0.54, 2.63); 1 Stroke: 1.04% (-0.44, 2.53); 1 75th percentile (80% prevalence of A/C): All cause: 0.85% (-0.64, 2.35); 1 Respiratory: 1.04% (-1.29, 3.37); 1 Cardiovascular: 0.81% (-0.93, 2.61); 1 Stroke: 1.03% (-0.76, 2.83); 1 Effect of A/C at percentile of air conditioning prevalence in cities with summer peaking $PM_{2.5}$ concentrations: 25th percentile (45% prevalence of A/C): All cause: 1.01% (-0.30, 2.32); 1 Respiratory: 0.76% (-1.38, 2.90); 1 Cardiovascular: 0.43% (-0.86, 1.72); 1 Stroke: -0.18% (-2.08, 1.73); 1 75th percentile (77% prevalence of A/C): All cause: -0.55% (-1.95, 0.85); 1 Respiratory: -2.08% (-4.47, 0.31); 1 Cardiovascular: -1.02% (-2.44, 0.41); 1 Stroke: 0.69% (-1.19, 2.57); 1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Franklin et al. (2008) **Period of Study:** 2000–2005 **Location:** 25 U.S. communities	**Outcome (ICD10):** Mortality: Non-accidental (V01-Y98) Respiratory (J00-J99) Cardiovascular (I01-I52) Stroke (I60-J69) **Study Design:** Time-series **Statistical Analyses:** 1st stage: Poisson, cubic spline 2nd stage: Random effects meta-analysis **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Range Mean (SD):** Winter: 9.6 to 34.4 Spring: 6.7 to 27.6 summer: 7.6 to 26.0 Fall: 9.5 to 32.1 **Range (Min, Max):** NR **Copollutant:** Al, As, Br, Cr, EC, Fe, K, Mn, Na^+, Ni, NO_3^-, NH_4, OC, Pb, Si, SO_4^{2-}, V, Zn	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Non-accidental: 0.74% (0.41, 1.07); 0-1 Cardiovascular: 0.47% (0.02, 0.92); 0-1 Respiratory: 1.01% (-0.03, 2.05); 1-2 Stroke: 0.68% (-0.21, 1.57); 0-1 Winter: 0.15% (-0.42, 0.72); 0-1 Spring: 1.88% (1.29, 2.48); 0-1 Summer: 0.99% (0.35, 1.68); 0-1 Fall: 0.19% (-0.25, 0.64); 0-1 West: 0.51% (0.10, 0.92); 0-1 East & Central: 0.92% (0.44, 1.39); 0-1 % Increase per 10 µg/m^3 increase in $PM_{2.5}$ for an IQR increase in species to $PM_{2.5}$ mass proportion Univariate analysis Al: 0.58% As: 0.55% Br: 0.38 Cr: 0.33% EC: 0.06% Fe: 0.12% K: 0.41% Mn: 0.14% Na^+: 0.20% Ni: 0.37% NO_3^-: -0.49% NH_4: 0.04% OC: -0.02% Pb: 0.17% Si: 0.41% SO_4^{2-}: 0.51% V: 0.30% Zn: 0.23% Multivariate (1) Al: 0.79% Ni: 0.34% SO_4^{2-}: 0.75% Multivariate (2) Al: 0.61% Ni: 0.35% As: 0.58%
Reference: Holloman et al. (2004) **Period of Study:** 1999–2001 **Location:** 7 North Carolina counties	Outcome (ICD10): Mortality: Cardiovascular (I00-I99) **Study Design:** Time-series **Statistical Analyses:** 3-stage Bayesian hierarchical model **Age Groups:** >16	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** 2.5% (-3.9 to 9.6); 0 4.0% (-3.3 to 12.2); 1 11.4% (2.8 to 19.8); 2 -1.1% (-7.5 to 5.2); 3

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hopke et al. (2006) **Period of Study:** Washington, DC: 8/1988–12/1997. Phoenix, Arizona: 3/1995–6/1998 **Location:** Washington, DC and surrounding counties; Phoenix, Arizona	**Outcome:** Mortality: Total (non-accidental) Cardiovascular Cardiovascular-Respiratory **Study Design:** Source-apportionment **Statistical Analyses:** Receptor modeling **Age Groups:** All ages	**Pollutant:** Source-apportioned $PM_{2.5}$: Washington, DC: Soil Traffic Secondary Sulfate Nitrate Residual Oil Wood Smoke Sea Salt Incinerator Primary Coal Phoenix, Arizona: Crustal Traffic Vegetation and Wood Burning Secondary Sulfate Metals Sea Salt Primary Coal **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	The study does not present quantitative results.
Reference: Ito et al. (2006) **Period of Study:** 8/1988–12/1997 **Location:** Washington, DC and surrounding counties	**Outcome:** Mortality: Total (non-accidental) Cardiovascular Cardiovascular-Respiratory **Study Design:** Time-series; Source-apportionment **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** Source-apportioned $PM_{2.5}$: Soil Traffic Secondary Sulfate Nitrate Residual Oil Wood Smoke Sea Salt Incinerator Primary Coal **Averaging Time:** 24-h avg **Mean (SD):** 17.8 (8.7) **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** $PM_{2.5}$ = 28.7 µg/m^3 $PM_{2.5}$ Sources 5-95th = Not reported **% Increase (Lower CI, Upper CI); lag:** Secondary sulfate (variance-weighted mean percent excess mortality) 6.7% (1.7, 11.7); 3 Primary coal-related $PM_{2.5}$ (mean percent excess mortality) 5.0% (1.0, 9.1); 3 Residual oil (mean percent excess mortality) 2.7% (-1.1, 6.5); 2 Traffic-related $PM_{2.5}$ (mean percent excess mortality) 2.6% (-1.6, 6.9); NR Soil-related $PM_{2.5}$ (mean percent excess mortality) 2.1% (-0.8, 4.9); NR **$PM_{2.5}$ Sensitivity analysis:** 2 df/year: 7.9% (3.3, 12.6); 3 4 df/year: 8.3% (3.7, 13.1); 3 8 df/year: 8.3% (3.7, 13.2); 3 16 df/year: 8.1% (3.1, 13.2); 3
Reference: Kan et al. (2007a) **Period of Study:** 3/2004–12/2005 **Location:** Shanghai, China	**Outcome (ICD10):** Mortality: Total (non-accidental) (A00-R99) Cardiovascular (I00-I99) Respiratory (J00-J98) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, penalized splines **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 52.3 (1.57) **Range (Min, Max):** (2.0, 330.3) **Copollutant (correlation):** PM_{10}: r = 0.84 $PM_{10-2.5}$: r = 0.48 O_3: r = 0.31	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Total: 0.36% (0.11, 0.61); 0-1 Cardiovascular: 0.41% (0.01, 0.82); 0-1 Respiratory: 0.95% (0.16, 1.73); 0-1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kettunen et al. (2007) **Period of Study:** 1998–2004 **Location:** Helsinki, Finland	**Outcome (ICD10):** Mortality: Stroke (I60-I61, I63-I64) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, penalized thin-plate splines **Age Groups:** ≥ 65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Median (SD) unit:** Cold Season: 8.2 Warm Season: 7.8 **Range (Min, Max):** Cold Season: (1.1, 69.5) Warm Season: (1.1, 41.5) **Copollutant:** O_3 CO NO_2 PM_{10} $PM_{10-2.5}$ UFP	**Increment:** Cold Season: 6.7 µg/m^3 Warm Season: 5.7 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Cold Season -0.19% (-3.77, 3.51); 0 -0.17% (-3.73, 3.52); 1 0.59% (-2.95, 4.26); 2 0.46% (-3.10, 4.15); 3 Warm Season 6.86% (0.37, 13.78); 0 7.40% (1.33, 13.84); 1 4.01% (-1.79, 10.14); 2 -1.72% (-7.38, 4.29); 3
Reference: Klemm et al. (2004) **Period of Study:** 8/1998–7/2000 **Location:** Fulton and DeKalb counties, Georgia (ARIES)	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular (390-459) Respiratory (460-519) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural cubic splines **Age Groups:** <65; ≥ 65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 19.62 (8.32) **Range (Min, Max):** (5.29, 48.01) **Copollutant:** $PM_{10-2.5}$; O_3; NO_2; CO; SO_2; Acid; EC; OC; SO_4; Oxygenated Hydrocarbons; Nonmethane hydrocarbons; NO_3	**Increment:** NR **β (SE); lag:** Quarterly Knots: $PM_{2.5}$: 0.00398 (0.00161); 0-1 Monthly Knots: $PM_{2.5}$: 0.00544 (0.00184); 0-1 Biweekly Knots: $PM_{2.5}$: 0.00369 (0.00201); 0-1
Reference: Lippmann et al. (2006) **Period of Study:** 2000–2003 **Location:** 60 U.S. cities (NMMAPS)	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM **Age Groups:** All ages	**Pollutant:** Speciated Fine PM: Al, Ar, Cr, Cu, EC, Fe, Mn, Ni, Nitrate, OC, Pb, Se, Si, Sulfate, V, Zn **Averaging Time:** Annual avg **Mean (SD):** R **Range (Min, Max):** NR	The study does not present quantitative results.
Reference: Mar et al. (2005b) **Period of Study:** 1995–1997 **Location:** Phoenix, Arizona	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular (390-448) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM **Age Groups:** ≥ 65	**Pollutant:** Source-apportioned $PM_{2.5}$: Soil Traffic Secondary Sulfate Nitrate Residual Oil Wood Smoke Sea Salt Incinerator Primary Coal **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR	**Increment:** $PM_{2.5}$ Sources 5-95th = NR **% Increase (median percent excess risk); lag:** Secondary sulfate: 16.0%; 0 Traffic: 13.2%; 1 Copper (Cu) smelter: 12.0%; 0 Sea salt: 10.2%; 5 Biomass/wood combustion: 8.6%; 3

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ostro et al. (2006) **Period of Study:** 1/1999–12/2002 **Location:** 9 California counties (CALFINE)	**Outcome (ICD10):** Mortality: Total mortality (respiratory, cardiovascular, ischemic heart disease, diabetes) Respiratory (J00-J98) Cardiovascular (I00-I99) Ischemic heart disease (I20-I25) Diabetes (E10-E14) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines and penalized splines **Age Groups:** All ages >65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** Contra Costa: 14 Fresno: 23 Kern: 22 Los Angeles: 21 Orange: 21 Riverside: 29 Sacramento: 14 Santa Clara: 15 San Diego: 16 **Range (Min, Max):** Contra Costa: (1, 77) Fresno: (1, 160) Kern: (1, 155) Los Angeles: (4, 85) Orange: (4, 114) Riverside: (2, 120) Sacramento: (1, 108) Santa Clara: (2, 74) San Diego: (0, 66) **Copollutant (correlation):** NO_2; r = 0.56 CO; r = 0.60 O_3 (1h); r = -0.14 O_3 (8h); r = -0.22	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Penalized splines All ages: All-cause: 0.2% (-0.2, 0.7); 2 0.6% (0.2, 1.0); 0-1 Cardiovascular: 0.3% (-0.1, 0.7); 2 0.6% (0.0, 1.1); 0-1 Respiratory: 1.3% (0.1, 2.6); 2 2.2% (0.6, 3.9); 0-1 >65: All-cause: 0.2% (-0.2, 0.7); 2 0.7% (0.2, 1.1); 0-1 Ischemic heart disease: 0.3% (-0.5, 1.0); 0-1 Males: 0.5% (-0.2, 1.2); 0-1 Females: 0.8% (0.3, 1.3); 0-1 Whites: 0.8% (0.2, 1.3); 0-1 Blacks: 0.1% (-0.9, 1.2); 0-1 Hispanics: 0.8% (-0.1, 1.6); 0-1 In hospital: 0.6% (-0.1, 1.3); 0-1 Out of hospital: 0.6% (0.1, 1.1); 0-1 High school graduates: 0.4% (0.0, 0.8); 0-1 Non-high school graduates: 0.9% (-0.1, 1.9); 0-1 Natural splines All cause 4 df: 0.5% (-0.1, 1.1); 0-1 8 df: 0.4% (-0.1, 0.9); 0-1 12 df: 0.3% (-0.1, 0.7); 0-1 Cardiovascular 4 df: 0.4% (-0.2, 0.9); 0-1 8 df: 0.1% (-0.5, 0.6); 0-1 12 df: 0.0% (-0.6, 0.6); 0-1 Respiratory 4 df: 2.1% (0.2, 4.1); 0-1 8 df: 1.6% (-0.5, 3.6); 0-1 12 df: 1.3% (-0.3, 2.9); 0-1 >65 All cause 4 df: 0.7% (0.0, 1.3); 0-1 8 df: 0.4% (-0.1, 0.9); 0-1 12 df: 0.3% (-0.1, 0.8); 0-1

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ostro et al. (2007) **Period of Study:** $PM_{2.5}$ speciation analysis: 1/2000-12/2003. $PM_{2.5}$ analysis: 1/1999-12/2003 **Location:** 6 California counties (2000–2003). 9 California counties (1999–2003) (CALFINE)	**Outcome (ICD10):** Mortality: Total (non-accidental) mortality Respiratory (J00-J98) Cardiovascular (I00-I99) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines **Age Groups:** >65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 2000–2003: 19.28 1999–2003: 18.6 **Range (Min, Max): NR** **Copollutant (correlation):** EC: r = 0.53; OC: r = 0.62; NO_3: r = 0.65; SO4: r = 0.32; Al: r = 0.02; Br: r = 0.54; Ca: r = 0.23; Cl: r = 0.15; Cu: r = 0.23; Fe: r = 0.38; K: r = 0.52; Mn: r = 0.21; Ni: r = 0.11; Pb: r = 0.27; S: r = 0.35; Si: r = 0.16; Ti: r = 0.24; V: r = 0.20; Zn: r = 0.51	**Increment:** 14.6 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Cardiovascular 1.6% (0.0, 3.1); 3 **Notes:** The study does not present all estimates quantitatively.
Reference: Ostro et al. (2008) **Period of Study:** 1/2000–12/2003 **Location:** 6 California counties	**Outcome (ICD10):** Mortality: Cardiovascular (I00-I99) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural cubic splines and natural splines **Age Groups:**	**Pollutant:** $PM_{2.5}$, EC, OC, NO_3, SO4, Ca, Cl, Cu, Fe, K, S, Si, Ti, Zn **Averaging Time:** 24-h avg **Mean (SD):** $PM_{2.5}$: 19.28 EC: 0.966 OC: 7.129 NO_3: 5.415 SO4: 1.908 Ca: 0.080 Cl: 0.094 Cu: 0.007 Fe: 0.124 K: 0.117 S: 0.648 Si: 0.168 Ti: 0.009 Zn: 0.012 **Range (95th):** $PM_{2.5}$: 46.91 EC: 2.57 OC: 15.91 NO_3: 17.46 SO4: 5.18 Ca: 0.20 Cl: 0.41 Cu: 0.02 Fe: 0.34 K: 0.26 S: 1.70 Si: 0.43 Ti: 0.02 Zn: 0.04	The study does not present quantitative results.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Rainham et al. (2005) **Period of Study:** 1981–1999 **Location:** Toronto, Canada	**Outcome:** Mortality: Total (non-accidental) (<800) Cardiorespiratory (390-459; 480-519) Other-causes **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** All years: 17.0 (8.7) Winters (Dec–Feb): 17.2 (6.8) Summers (June–Aug): 18.8 (10.2) **Range (Min, Max):** NR **Copollutant:** CO NO_2 SO_2 O_3	**Increment:** NR **% Increase (Lower CI, Upper CI); lag:** **Winter and Winter Synoptic Events** **Winter** Total: 0.998% (0.997, 1.000); 2 Cardiorespiratory: 0.998 (0.996, 1.000); 2 Other: 0.998% (0.996, 1.000); 2 **Dry Moderate** Total: 1.001% (0.996, 1.007); 1 Cardiorespiratory: 1.005 (0.998, 1.011); 1 Other: 0.997% (0.989, 1.006); 0 **Dry Polar** Total: 0.998% (0.995, 1.001); 2 Cardiorespiratory: 0.995 (0.991, 0.999); 2 Other: 1.002% (0.998, 1.005); 1 **Moist Moderate** Total: 0.998% (0.993, 1.002); 2 Cardiorespiratory: 1.003 (0.995, 1.010); 1 Other: 0.997% (0.991, 1.004); 1 **Moist Polar** Total: 1.001% (0.998, 1.005); 1 Cardiorespiratory: 1.002 (0.997, 1.007); 2 Other: 1.003% (0.999, 1.007); 0 **Moist Tropical** Total: 1.007% (0.965, 1.203); 0 Cardiorespiratory: 1.123 (1.031, 1.224); 2 Other: 1.248% (1.123, 1.387); 0 **Transition** Total: 1.003% (0.996, 1.009); 1 Cardiorespiratory: 0.996 (0.987, 1.004); 0 Other: 0.997% (0.990, 1.004); 0 **Summer and summer Synoptic Events** **Summer** Total: 1.000% (1.000, 1.001); 0 Cardiorespiratory: 1.001 (1.000, 1.002); 0 Other: 1.001% (1.000, 1.002); 0 **Dry Moderate** Total: 1.001% (0.999, 1.002); 2 Cardiorespiratory: 1.002 (0.999, 1.004); 2 Other: 0.999% (0.997, 1.002); 0 **Dry Polar** Total: 1.002% (0.999, 1.005); 2 Cardiorespiratory: 0.996 (0.991, 1.000); 0 Other: 1.003% (0.999, 1.007); 2 **Dry Tropical** Total: 1.016% (1.006, 1.027); 0 Cardiorespiratory: 1.017 (1.005, 1.030); 2 Other: 1.017% (1.003, 1.031); 0 **Moist Moderate** Total: 1.002% (1.000, 1.004); 2 Cardiorespiratory: 1.003 (0.999, 1.006); 2 Other: 1.004% (1.001, 1.006); 0 **Moist Polar** Total: 1.005% (0.998, 1.011); 1 Cardiorespiratory: 1.008 (0.997, 1.018); 0 Other: 1.003% (0.995, 1.011); 1 **Moist Tropical** Total: 0.999% (0.997, 1.001); 2 Cardiorespiratory: 0.996 (0.993, 1.000); 2 Other: 0.998% (0.995, 1.001); 1 **Transition** Total: 1.005% (0.996, 1.014); 1 Cardiorespiratory: 1.007 (0.994, 1.020); 1 Other: 1.002% (0.996, 1.008); 2

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Rosenthal et al. (2008) **Period of Study:** 7/2002–7/2006 **Location:** Indianapolis, Indiana	**Outcome:** Non-Dead on Arrival (DOA) Out-of-Hospital Cardiac Arrests (OHCA) Witnessed non-DOA OHCA **Study Design:** Case-crossover **Statistical Analyses:** Time-stratified conditional logistic regression **Age Groups:** All ages Study PopulatioN: Non-DOA OHCA: 1,374 Witnessed non-DOA OHCA: 511	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg; Hourly **Mean (SD):** NR IQR (25th, 75th): All non-DOA All heart rhythms: (9.4, 19.5) OHCA: (9.6, 19.5) Referents: (9.3, 19.5) Asystole: (9.2, 19.4) OHCA: (9.2, 19.7) Asystole: (9.2, 19.2) Witnessed non-DOA hourly All heart rhythms: (8.8, 20.7) OHCA: (8.8, 21.9) Referents: (8.8, 20.4) Asystole: (8.5, 19.8) OHCA: (9.4, 21.3) Referents: (8.3, 19.1) **Copollutant (correlation):** NR	**Increment:** 10 $\mu g/m^3$ **Hazard Ratio (Lower CI, Upper CI); lag:** Out-of-Hospital non-DOA Cardiac Arrests All 1.02 (0.94, 1.11); 0 1.00 (0.92, 1.08); 1 0.98 (0.90, 1.06); 2 1.00 (0.92, 1.08); 3 1.02 (0.92, 1.12); 0-1 avg 1.01 (0.91, 1.12); 0-2 avg 1.02 (0.91, 1.14); 0-3 avg Asystole 1.03 (0.91, 1.17); 0 1.00 (0.89, 1.13); 1 1.01 (0.90, 1.13); 2 0.98 (0.87, 1.10); 3 1.03 (0.90, 1.18); 0-1 avg 1.05 (0.90, 1.22); 0-2 avg 1.04 (0.88, 1.22); 0-3 avg Vfib 1.08 (0.92, 1.28); 0 1.02 (0.87, 1.21); 1 0.96 (0.80, 1.14); 2 1.10 (0.93, 1.31); 3 1.06 (0.88, 1.28); 0-1 avg 1.01 (0.82, 1.25); 0-2 avg 1.05 (0.83, 1.32); 0-3 avg PEA 0.92 (0.77, 1.08); 0 0.98 (0.83, 1.15); 1 0.96 (0.82, 1.14); 2 0.95 (0.82, 1.10); 3 0.96 (0.80, 1.17); 0-1 avg 0.98 (0.80, 1.21); 0-2 avg 0.98 (0.78, 1.21); 0-3 avg Witnessed Out-of-Hospital non-DOA Cardiac Arrests (lag represents h in which or h before OHCA occurred) All: 1.12 (1.01, 1.25); 0 White: 1.18 (1.03, 1.35); 0 60-75: 1.25 (1.05, 1.49); 0 Asystole: 1.22 (1.01, 1.59); 0
Reference: Schwartz et al. (2002) **Period of Study:** 1979–Late 1980's **Location:** 6 U.S. cities	**Outcome:** Mortality: Total (non-accidental) (<800) **Study Design:** Time-series **Statistical Analyses:** Hierarchical modeling: 1. Poisson GAM, LOESS; 2. Multivariate modeling **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$, $PM_{2.5}$ sources (Traffic, Coal, Residual Oil) **Averaging Time:** 24-h avg **Mean (SD):** $PM_{2.5}$ Range: (Madison: 11.3 to Steubenville: 30.5) Traffic Range: (Steubenville: 1.5 to Boston: 4.8) Coal Range: (Madison: 4.9 to Steubenville: 19.2) Residual Oil Range: (Boston: 0.5 to Steubenville: 0.9) **Range (Min, Max):** NR	The study does not present quantitative results.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Simpson et al. (2005) **Period of Study:** 1/1996–12/1999 **Location:** 4 Australian cities	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular (390-459) Respiratory (460-519) **Study Design:** Time-series; meta-analysis **Statistical Analyses:** Poisson GAM, natural splines; Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** Brisbane $PM_{2.5}$: 7.50 Sydney $PM_{2.5}$: 9.00 Melbourne $PM_{2.5}$: 9.30 Perth $PM_{2.5}$: 9.0 µg/m^3 **Range (Min, Max):** Brisbane $PM_{2.5}$: (1.9, 19.7) Sydney $PM_{2.5}$: (2.4, 35.3) Melbourne $PM_{2.5}$: (2.7, 35.1) Perth $PM_{2.5}$: (2.8, 37.3) **Copollutant:** CO, NO_2	**Increment:** 10 µg/m^3 % Increase (Lower CI, Upper CI); lag: $PM_{2.5}$ 0.9% (-0.7, 2.5)
Reference: Slaughter et al. (2005) **Period of Study:** 1/1995–12/1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (9th, 95th):** $PM_{2.5}$: (4.2, 20.2) **Copollutant (correlation):** $PM_{2.5}$: r = 0.95 PM_{10}: r = 0.62 $PM_{10-2.5}$: r = 0.31 CO: r = 0.62	**Increment:** $PM_{2.5}$: 10 µg/m^3 PM_{10}: 25 µg/m^3 Relative Risk (Lower CI, Upper CI); lag: $PM_{2.5}$ (0.97, 1.04); 1 0.99 (0.96, 1.03); 2 1.00 (0.97, 1.03); 3
Reference: Stieb et al. (2002) **Period of Study:** Publication dates of studies: 1985–12/2000 Mortality series: 1958–1999 **Location:** 40 cities (11 Canadian cities, 19 U.S. cities, Santiago, Amsterdam, Erfurt, 7 Korean cities)	**Outcome:** Mortality: All-cause (non-accidental) **Study Design:** Meta-analysis **Statistical Analyses:** Random effects model **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** Varied between studies: O_3 SO_2 NO_2 CO	**Increment:** $PM_{2.5}$: 18.3 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Single-pollutant models 18 studies $PM_{2.5}$: 2.0% (1.2, 2.7) Multipollutant models 8 studies $PM_{2.5}$: 1.3% (0.6, 1.9)
Reference: Sullivan et al. (2003) **Period of Study:** 1985–1994 **Location:** Western Washington	**Outcome:** Out-of-hospital cardiac arrest **Study Design:** Case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** 19-79 Study PopulatioN: Out-of-hospital cardiac arrests: 1,206	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg Median (SD) unit: PM_{10} Lag 0: 28.05 Lag 1: 27.97 Lag 2: 28.40 **Range (Min, Max):** PM_{10}: (7.38, 89.83) **Copollutant (correlation):** SO_2, CO Notes: Study used nephelometry to measure particles and equated the measurements to $PM_{2.5}$ concentrations.	**Increment:** PM_{10}: 16.51 µg/m^3 $PM_{2.5}$: 13.8 µg/m^3 **Odds Ratio (Lower CI, Upper CI); lag:** **Overall** **PM_{10}** 1.05 (0.87, 1.27); 0; 0.91 (0.75, 1.11); 1; 1.03 (0.82, 1.28); 2 **$PM_{2.5}$** 0.94 (0.88, 1.01); 0.94 (0.88, 1.02); 1; 1.00 (0.93, 1.08); 2 **$PM_{2.5}$: Stratified by subject characteristics** **≤ 55:** 0.95 (0.76, 1.18); 0; 0.89 (0.71, 1.12); 1; 0.95 (0.75, 1.20); 2 **>55:** 0.94 (0.88, 1.02); 0l; 0.95, (0.88, 1.03); 1; 1.01 (0.93, 1.10); 2 **Male:** 0.95 (0.87, 1.03); 0; 0.96 (0.88, 1.04); 1; 1.01 (0.93, 1.10); 2 **Female:** 0.93 (0.82, 1.06); 0; 0.92 (0.80, 1.07); 1; 0.98 (0.83, 1.15); 2

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			White: 0.93 (0.86, 1.01); 0; 0.95 (0.88, 1.03); 1; 1.03 (0.95, 1.12); 2 **Non-White:** 1.09 (0.88, 1.36); 0; 0.96 (0.75, 1.22); 1; 0.88 (0.68, 1.14); 2 **Current Smoker:** 1.05 (0.92, 1.19); 0; 0.98 (0.86, 1.12); 1; 1.06 (0.92, 1.22); 2 **Nonsmoker:** 0.93 (0.85, 1.01); 0; 0.93 (0.85, 1.02); 1; 0.97 (0.89, 1.07); 2 **Drinker:** 1.13 (0.92, 1.39); 0; 1.15 (0.94, 1.41); 1; 1.16 (0.92, 1.45); 2 **Nondrinker:** 0.94 (0.86, 1.03); 0; 0.93 (0.85, 1.02); 1; 1.00 (0.92, 1.10); 2 **Activity Level–Unrestricted:** 0.96 (0.89, 1.03); 0; 0.96 (0.89, 1.04); 1; 1.01 (0.93, 1.10); 2 **Activity Level–Limited:** 0.82 (0.56, 1.20); 0; 0.70 (0.45, 1.09); 1; 0.97 (0.65, 1.43); 2 **$PM_{2.5}$: Stratified by disease state** **Heart disease:** 0.95 (0.87, 1.04); 0; 0.97 (0.89, 1.07); 1; 1.06 (0.96, 1.16); 2 **Ischemic Heart Disease:** 0.91 (0.80, 1.04); 0; 0.97 (0.84, 1.11); 1; 1.09 (0.95, 1.26); 2 **Active Angina:** 0.98 (0.81, 1.20); 0; 1.07 (0.88, 1.31); 1; 1.08 (0.89, 1.32); 2 **Congestive Heart Failure:** 0.91 (0.80, 1.03); 0; 0.99 (0.87, 1.13); 1; 1.11 (0.97, 1.26); 2 **Supraventricular tachycardia:** 1.41 (0.97, 2.04); 0; 1.55 (1.07, 2.25); 1; 1.23 (0.84, 1.82); 2 **Bradycardia:** 0.97 (0.64, 1.46); 0; 1.29 (0.85, 1.96); 1; 1.30 (0.84, 2.01); 2 **Asthma:** (0.80, 1.27); 0; 0.92 (0.71, 1.19); 1; 0.93 (0.71, 1.22); 2 **COPD:** 1.00 (0.86, 1.17); 1.04 (0.88, 1.23); 1; 1.08 (0.92, 1.28); 2 **$PM_{2.5}$: Persons with prior recognized heart disease stratified by smoking status** **All heart disease** **Current smoke**r: 1.08 (0.92, 1.26); 0; 1.06 (0.89, 1.26); 1; 1.29 (1.06, 1.55); 2 **Nonsmoker:** 0.91 (0.82, 1.02); 0; 0.94 (0.84, 1.05); 1; 0.99 (0.88, 1.11); 2 **Ischemic Heart Disease** **Current smoker:** 1.06 (0.84, 1.34); 0; 0.99 (0.75, 1.30); 1; 1.39 (1.04, 1.86); 2 **Nonsmoker;** 0.86 (0.73, 1.02); 0; 0.93 (0.78, 1.11); 1; 0.99 (0.83, 1.18); 2 **Active Angina** **Current smoker:** 1.28 (0.88, 1.86); 0; 1.26 (0.79, 2.01); 1; 1.57 (0.99, 2.48); 2 **Nonsmoker:** 0.87 (0.68, 1.12); 0; 0.93 (0.72, 1.21); 1; 0.91 (0.70, 1.17); 2 **Congestive Heart Failure** **Current smoker:** 1.00 (0.79, 1.28); 0; 1.03 (0.78, 1.35); 1; 1.46 (1.10, 1.96); 2 **Nonsmoker:** 0.88 (0.76, 1.03); 0; 0.96 (0.82, 1.12); 1; 0.99 (0.84, 1.17); 2 **Supraventricular tachycardia** **Current smoker:** 12.80 (1.05, 156.57); 0; 2.56 (0.82, 7.99); 1; 1.15 (0.46, 2.86); 2 **Nonsmoker:** 1.19 (0.74, 1.90); 0; 1.35 (0.87, 2.10); 1; 1.15 (0.73, 1.82); 2 **Bradycardia** **Current smoker:** 0.84 (0.14, 4.95); 0; 0.42 (0.03, 5.34); 1; 0.51 (0.05, 5.79); 2 **Nonsmoker:** 0.99 (0.63, 1.55); 0; 1.42 (0.90, 2.24); 1; 1.39 (0.88, 2.20); 2

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Thurston et al. (2005) **Period of Study:** Washington, DC: 8/1988–12/1997. Phoenix, Arizona: 1995–1997 **Location:** Washington, DC and surrounding counties; Phoenix, Arizona	**Outcome:** Mortality: Total (non-accidental) (<800) Cardiovascular (390-448) **Study Design:** Time-series; Source-apportionment **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** Washington, DC: All ages Phoenix, Arizona: ≥ 65	**Pollutant:** $PM_{2.5}$, and source apportioned $PM_{2.5}$: Crustal Traffic Secondary SO4 Secondary NO_3 Wood Oil Salt Incinerator **Averaging Time:** 24-h avg **Median (SD) unit:** NR **Range (Min, Max):** NR **Copollutant:** $PM_{2.5}$ species (Na, Mg, Al, Si, P, S, Cl, K, Ca, Sc, Ti, V, Cr, Mn, Fe, Co, Ni, Cu, Zn, Ga, Ge, As, Se, Br, Rb, Sr, Y, Zr, Mo, Rh, Pd, Ag, Cd, Sn, Sb, Te, I, Cs, Ba, La, W, Au, Hg, Pb, OC, EC)	**Increment:** 10 µg/m^3 **% Increase:** Total (non-accidental): Secondary sulfate: Phoenix: 5.2% Washington, DC: 3.8% Motor vehicles: Phoenix: 0.9% Washington, DC: 4.2%

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Villeneuve et al. (2003) **Period of Study:** 1986–1999 **Location:** Vancouver, Canada	**Outcome:** Mortality: Non-accidental (<800) Cardiovascular (401-440) Respiratory (460-519) Cancer (140-239) **Study Design:** Time-series **Statistical Analyses:** Poisson, natural splines **Age Groups:** ≥ 65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** Daily $PM_{2.5}$: 7.9 Every 6th Day $PM_{2.5}$: 11.6 **Range (Min, Max):** Daily $PM_{2.5}$: (2.0, 32.0) Every 6th Day $PM_{2.5}$: (1.8, 43.0) **Copollutant:** SO_2 CO NO_2 O_3	**Increment:** $PM_{2.5}$ (Daily): 9.0 µg/m³ $PM_{2.5}$ (6th Day): 15.7 µg/m³ **% Increase (Lower CI, Upper CI); lag:** Non-accidental $PM_{2.5}$ (Daily) -0.1% (-5.1, 5.2); 0-2 avg -0.1% (-4.1, 4.1); 0 -0.3% (-4.2, 3.7); 1 0.5% (-3.3, 4.4); 2 $PM_{2.5}$ (6th Day) -2.8% (-7.5, 2.1); 0 2.0% (-2.6, 7.0); 1 4.5% (-0.3, 9.5); 2 Cardiovascular $PM_{2.5}$ (Daily) 1.5% (-6.1, 9.7); 0-2 avg 4.3% (-1.7, 10.7); 0 -1.0% (-7.0, 5.4); 1 -0.5% (-6.5, 5.9); 2 $PM_{2.5}$ (6th Day) -1.5% (-8.9, 6.5); 0 -2.0% (-9.3, 5.8); 1 3.0% (-4.2, 10.8); 2 Respiratory $PM_{2.5}$ (Daily) -0.7% (-13.1, 13.4); 0-2 avg 6.7% (-3.7, 18.3); 0 -3.0% (-12.8, 7.9); 1 -5.8% (-15.2, 4.7); 2 $PM_{2.5}$ (6th Day) 10.0% (-4.7, 26.8); 0 8.3% (-5.4, 24.0); 1 0.3% (-12.4, 14.9); 2 Cancer $PM_{2.5}$ (Daily) -0.3% (-9.4, 9.8); 0-2 avg -4.5% (-11.2, 2.8); 0 2.7% (-5.0, 11.0); 1 2.5% (-5.1, 10.7); 2 $PM_{2.5}$ (6th Day) -5.1% (-13.8, 4.5); 0 -0.3% (-9.7, 11.0); 1 0.2% (-9.1, 10.4); 2
Reference: Wilson et al. (2007) **Period of Study:** 1995–1997 **Location:** Phoenix, Arizona	**Outcome:** Mortality: Cardiovascular **Study Design:** Time-series **Statistical Analyses:** Poisson GAM, nonparametric smoothing spline **Age Groups:** >25	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m³ **% Excess Risk (Lower CI, Upper CI); lag:** $PM_{2.5}$ Central Phoenix: 11.5% (2.8, 20.9); 0-5 ma 6.6% (1.1, 12.5); 1 2.0% (-3.2, 7.5); 2 Middle Phoenix: 2.9% (-4.9, 11.4); 0-5 ma 6.4% (1.1, 11.9); 2 Outer Phoenix: 1.6% (-6.2, 10.0); 0-5 ma

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)

Table E-20. Short-term exposure to other PM size fractions and mortality.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Slaughter et al. (2005) **Period of Study:** 1/1995–12/1999 **Location:** Spokane, Washington	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Time-series **Statistical Analyses:** Poisson GLM, natural splines **Age Groups:** All ages	**Pollutant:** PM_1 **Averaging Time:** 24-h avg **Mean (SD):** NR Range (9th, 95th) PM_1: (3.3, 17.6) **Copollutant (correlation):** PM_1 $PM_{2.5}$: r = 0.95 PM_{10}: r = 0.50 $PM_{10-2.5}$: r = 0.19 CO: r = 0.63	This study does not present quantitative results for PM_1.
Reference: Stölzel et al. (2007) **Period of Study:** 9/1995–8/2001 **Location:** Erfurt, Germany	**Outcome:** Mortality: Total (non-accidental) (<800) Cardio-respiratory (390-459, 460-519, 785, 786) **Study Design:** Time-series **Statistical Analyses:** Poisson GAM **Age Groups:** All ages	**Pollutant:** $MC_{0.1-0.5}$, $MC_{0.01-2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** $MC_{0.1-0.5}$: 17.6 (14.8) $MC_{0.01-2.5}$: 22.3 (19.2) IQR (25th, 75th): $MC_{0.1-0.5}$: (8.4, 21.5) $MC_{0.01-2.5}$: (10.5, 27.3) **Copollutant (correlation):** $MC_{0.1-0.5}$ NO: r = 0.52 NO_2: r = 0.60 CO: r = 0.58 $MC_{0.01-2.5}$ NO: r = 0.51 NO_2: r = 0.58 CO: r = 0.57	**Increment:** $MC_{0.1-0.5}$: 13.1 µg/m^3 $MC_{0.01-2.5}$: 16.8 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** Total (non-accidental) $MC_{0.1-0.5}$ 1.010 (0.986; 1.034); 0 1.006 (0.983; 1.029); 1 1.007 (0.985; 1.029); 2 0.994 (0.973; 1.016); 3 1.002 (0.981; 1.023); 4 0.997 (0.976; 1.018); 5 $MC_{0.01-2.5}$ 1.007 (0.985; 1.030); 0 1.005 (0.984; 1.026); 1 1.003 (0.983; 1.023); 2 0.989 (0.970; 1.009); 3 1.002 (0.982; 1.022); 4 0.998 (0.979; 1.018); 5 Cardio-respiratory $MC_{0.1-0.5}$ 1.004 (0.977; 1.031); 0 1.004 (0.979; 1.029); 1 1.001 (0.978; 1.026); 2 0.991 (0.967; 1.014); 3 1.000 (0.977; 1.023); 4 1.000 (0.976; 1.023); 5 $MC_{0.01-2.5}$ 1.001 (0.977; 1.026); 0 0.999 (0.976; 1.022); 1 0.998 (0.976; 1.021); 2 0.985 (0.964; 1.007); 3 1.001 (0.980; 1.022); 4 1.003 (0.981; 1.024); 5

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Yamazaki et al. (2007) **Period of Study:** 1995-1998 **Location:** Hong Kong, China	**Outcome:** Mortality: Intracerebral hemorrhage (431) Ischaemic stroke (434) **Study Design:** Time-stratified case-crossover **Statistical Analyses:** Conditional logistic regression **Age Groups:** ≥ 65	**Pollutant:** PM_7 **Averaging Time:** 1-h avg **Mean (SD):** Warmer Months (April-September): 40.3 Colder Months (October-March): 39.4 **Range (Min, Max):** NR **Copollutant (correlation):** Warmer Months NO_2: r = 0.46 to 0.63 Ox: r = -0.14 to 0.20 Colder Months NO_2: 0.42 to 0.79 Ox: r = -0.36 to -0.14	**Increment:** 30 µg/m^3 Odds Ratio (Lower CI, Upper CI); lag: 24-h avg concentrations Intracerebral hemorrhage Warmer months: 1.041 (0.984, 1.102); 0 Colder months: 1.005 (0.951, 1.061); 0 Ischaemic stroke Warmer months: 1.027 (0.993, 1.062); 0 Colder months: 1.005 (0.973, 1.039); 0 Exposure measured jointly as 24-h and 1-h mean concentrations Warmer months Intracerebral hemorrhage 1-h with 200 µg/m^3 threshold: 2.397 (1.476, 3.892); 2 h 24-h: 1.019 (0.960, 1.082); 0 Ischaemic stroke 1-h with 200 µg/m^3 threshold: 1.051 (0.750, 1.472); 2 h 24-h: 1.018 (0.983, 1.055); 0 Warmer months Intracerebral hemorrhage 1-h with 200 µg/m^3 threshold: 0.970 (0.712, 1.322); 2 h 24-h: 1.015 (0.958, 1.075); 0 Ischaemic stroke 1-h with 200 µg/m^3 threshold: 1.040 (0.855, 1.265); 2 h 24-h: 1.003 (0.968, 1.039); 0

E.4. Long-Term Exposure and Cardiovascular Outcomes

Table E-21. Long-term exposure to PM_{10} and respiratory morbidity outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Baccarelli et al. (2008) **Period of Study:** 1995-2005 **Location:** Italy (Lombardy region)	**Outcome (ICD9 and ICD10):** Deep Vein Thrombosis (DVT); prothrombin time (PT); activated partial thromboplastin time (aPTT) **Age Groups:** 18-84yrs **Study Design:** Case-control (DVT outcome); Cross-sectional (PT and aPTT outcomes) **N:** 871 cases; 1210 controls (randomly selected from friends and nonblood relatives of cases; frequency matched by age to cases) **Statistical Analyses:** Unconditional logistic regression (DVT outcome); linear regression (PT and aPTT outcomes) **Covariates:** sex, area of residence, education, factor V Leiden or G20210A prothrombin mutation, current use of oral contraceptives or hormone therapy; (variables controlled using penalized regression splines with 4 df) age, BMI, day of year (for seasonality), index date, ambient temperature **Season:** covariate **Dose-response Investigated?** Yes **Statistical Package:** STATA v9.0 and R v2.2.0	**Pollutant:** PM_{10} **Averaging Time:** 1 year (immediately preceding the diagnosis date for cases or the date of examination for controls); assessed other averaging periods presented in supplements (90 days, 180 days, 270 days, 365 days, 2 yrs) **Mean (SD):** NR **Percentiles:** NR **Range (Min, Max):** Range for tertiles of exposure: 1: 12.0–44.2 2: 44.3–48.1 3: 48.2–51.5 **Monitoring Stations:** Monitors from 53 sites; exposure assigned by dividing area into 9 regions **Copollutant (correlation):** NR	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]: Estimated changes of PT associated with** PM_{10}**:** Among Controls**:** -0.12 (-0.23, 0.00) Among DVT cases: -0.06 (-0.11, 0.00) **Estimated changes of aPTT associated with** PM_{10}**:** Among Controls: -0.09 (-0.19, 0.01) Among DVT cases: 0.01 (-0.03, 0.04) **Risk of DVT associated with** PM_{10} **(avg of 1 yr preceding diagnosis/exam date) by subject characteristics:** **All subjects:** 1.70 (1.30, 2.23) **Sex:** Male: 2.07 (1.50, 2.84). Female: 1.40 (1.02, 1.92) **Age:** 18-35yrs: 1.57 (1.11, 2.24) 36-50yrs: 1.97 (1.41, 2.77). 51-84yrs: 1.54 (0.90, 2.63) **Premenopausal women with current use of oral contraceptives:** No: 1.53 (0.86, 2.72). Yes: 0.87 (0.46, 1.67) **Postmenopausal women with current use of hormone therapy:** No: 1.60 (0.72, 3.54). Yes: 0.85 (0.29, 2.45) **Current use of oral contraceptive or hormone replacement therapy:** No: 1.64 (1.05, 2.57)**.** Yes: 0.97 (0.58, 1.61) **Body Mass Index:** 13.3-22.0: 1.47 (0.97, 2.23) 22.1-24.9: 1.72 (1.17, 2.54). 25.0-53.3: 1.83 (1.03, 3.24) **Education:** Elementary/middle school: 1.93 (1.35, 2.76) High school: 1.72 (1.24, 2.39). College: 1.35 (0.74, 2.45) **Deficiencies of natural anticoagulant proteins:** None: 1.66 (1.26, 2.18). Any: 2.56 (0.91, 7.18) **Factor V Leiden or G20210A prothrombin mutation:** None: 1.69 (1.27, 2.23). Any: 1.79 (1.05, 3.05) **Hyperhomocysteinemia:** No: 1.66 (1.26, 2.19). Yes: 2.19 (1.33, 3.61) **Any cause of thrombophilia:** No: 1.59 (1.19, 2.13). Yes: 1.96 (1.34, 2.87) **Year of diagnosis:** 1995-97: 1.61 (1.06, 2.46) 1998-00: 1.34 (0.90, 1.99). 2001-05: 2.14 (1.04, 4.39) **Risk of DVT associated with** PM_{10} **over varying averaging times:** 90 days: 0.91 (0.80, 1.03). 180 days: 0.96 (0.82, 1.13). 270 days: 1.26 (1.01, 1.57). 365 days: 1.70 (1.30, 2.23). 2 years: 1.47 (1.01, 2.14) **Risk of DVT associated with** PM_{10} **(year preceding diagnosis/exam date); sensitivity analysis to evaluate the effect of different methods for adjusting for long-term trends:** **Handling of long-term time trends:** Ignored: 1.13 (0.89, 1.42) Dummy variable for each year: 1.78 (1.31, 2.44) Linear term: 1.32 (1.02, 1.69) Penalized spline, 2 df: 1.54 (1.19, 2.00) Penalized spline, 3 df: 1.64 (1.26, 2.14) Penalized spline, 4 df: 1.70 (1.30, 2.23) Penalized spline, 5 df: 1.70 (1.29, 2.22) Penalized spline, 6 df: 1.66 (1.26, 2.19) Penalized spline, 7 df: 1.60 (1.21, 2.13) Penalized spline, 8 df: 1.55 (1.15, 2.10)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Calderon-Garciduenas et al. (2007) **Period of Study:** Children recruited between Jul 2003 and Dec 2004 **Location:** Mexico (northeast or southwest Mexico city or Polotitlan)	**Outcome (ICD9 and ICD10):** Plasma Endothelin-1 (ET-1) and pulmonary arterial pressure (PAP) **Age Groups:** 6-13 years; 7.9 ± 1.3 years **Study Design:** Cross-sectional **N:** 81 children **Statistical Analyses:** Analysis of variance by parametric one-way analsis of variance and the Newman-Keuls multiple comparison post test **Covariates:** doesn't appear to have performed multivariable analyses; however, collected information on age, place and length of residency, daily outdoor time, household cooking methods, parents' occupational history, family history of atopic illnesses and respiratory disease, and personal history of otolaryngologic and respiratory symptoms **Season:** No **Dose-response Investigated?** No **Statistical Package:** STATA v8.3	**Pollutant:** PM_{10} Exposures assessed quantitatively in Mexico City only; no monitors in Polotitlan **Averaging Time:** 1, 2, and 7 days before the exam; pollutant concentrations between 0700 and 1900 h were used for the estimates **Mean (SD):** Presented only in figures **Percentiles:** NR **Range (Min, Max):** Presented only in figures **Monitoring Stations:** 4 (2 in northeast and 2 in southwest Mexico City; residence and school within 5 miles of one of these monitors) **Copollutant (correlation):** O_3	**PM Increment:** NA **Effect Estimate [Lower CI, Upper CI]:** No health effects models with measured PM concentrations were presented; used city of residence to assign exposure; no multivariable analyses presented Authors presented (statistically significantly) elevated ET-1 levels among children residing in both areas of Mexico City as compared to Polotitlan (control city): Mean ± SE (pg/mL) Control: 1.23 ± 0.06 Southwest Mexico City: 2.40 ± 0.14 Northeast Mexico City: 2.09 ± 0.10 Authors presented (statistically significantly) elevated PAP levels among children residing in both areas of Mexico City as compared to Polotitlan (control city): Mean ± SE (mmHg) Control: 14.6 ± 0.4 Southwest Mexico City: 16.7 ± 0.6 Northeast Mexico City: 18.6 ± 0.9 Among Mexico City children only, there was a positive correlation between ET-1 levels and the 7-day cumulative $PM_{2.5}$ exposure (r = 0.28, p = 0.03)
Reference: Diez Roux et al. (2008) **Period of Study:** Baseline data collected June 2000–Aug 2002; Exposure assessed retrospectively between Aug 1982 and baseline date **Location:** USA (6 field centers: Baltimore, MD; Chicago, IL; Forsyth Co, NC; Los Angeles, CA; New York, NY; St. Paul, MN	**Outcome (ICD9 and ICD10):** Three measures of subclinical atherosclerosis (common carotid intimal-medial thickness (CIMT), coronary artery calcification, and ankle-brachial index (ABI)) **Age Groups:** 44-84 yrs **Study Design:** Cross-sectional **N:** 5172 for coronary calcium analysis; 5037 for CIMT analysis; 5110 for ABI analysis **Statistical Analyses:** Generalized Additive Models (Binomial regression: presence of calcification; Linear regression: CIMT, ABI, amount of calcium) **Covariates:** age, sex, race/ethnicity, socioeconomic factors, cardiovascular risk factors (BMI, hypertension, high density lipoprotein and low density lipoprotein cholesterol, smoking, diabetes, diet, physical activity; models presented with and without adjustment for cardiovascular RFs) **Season:** NA **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 20-yr imputed mean **Mean (SD):** 34.1 (7.5) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** NR; Long-term exposure to PM estimated based on residential history reported retrospectively; all addresses geocoded; ambient AP obtained from US EPA **Copollutant (correlation):** PM_{10} 20-yr observed mean; r = 0.93 $PM_{2.5}$ 20-yr imputed mean; r = 0.73 PM_{10} 2001 mean; r = 0.75 $PM_{2.5}$ 2001 mean; r = 0.86 Due to high correlation among PM exposures, only results of mean 20-yr exposures are reported.	**PM Increment:** 21.0 µg/m^3 (approx. 10th-90th percentile) **Effect Estimate [Lower CI, Upper CI]:** **CIMT:** Relative difference (95% CI): 1.01 (1.00, 1.02) Adj. for additional CVD RFs: 1.02 (1.00, 1.03) **ABI:** Mean difference (95% CI): 0.002 (-0.005, 0.009) Adj. for additional CVD RFs: 0.001 (-0.006, 0.009) **Coronary calcium:** Relative prevalence (95% CI): 1.02 (0.96, 1.07) Adj. for additional CVD RFs: 1.02 (0.96, 1.08) **Coronary calcium (in those with calcium):** Relative difference (95% CI): 0.98 (0.84, 1.13) Adj. for additional CVD RFs: 1.01 (0.86, 1.18)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Maheswaran et al. (2005) **Period of Study:** 1994-1998 **Location:** Sheffield, United Kingdom	**Outcome (ICD9 and ICD10):** Stroke mortality (ICD9: 430-438) and Emergency hospital admissions (ICD10: I60-I69) **Age Groups:** ≥ 45 years **Study Design:** Ecological cross-sectional **N:** 1030 census enumeration districts (CEDs); 108 CEDs excluded from PM analyses due to artifacts in the emissions data **Statistical Analyses:** Poisson regression **Covariates:** age, sex, socioeconomic deprivation, and smoking prevalence; also included age-by-deprivation interaction) **Season:** NA **Dose-response Investigated?** Yes, examined quintiles of exposure **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 5-yr avg **Mean (SD):** Presented mean values and ranges for each quintile of exposure: 1: 16.0 (<16.8) 2: 17.5 (≥ 16.8, <18.2) 3: 18.8 (≥ 18.2, <19.3) 4: 19.8 (≥ 19.3, <20.6) 5: 23.3 (≥ 20.6) **Monitoring Stations:** NR **Copollutant (correlation):** CO (r = 0.82) NO_X (r = 0.87)	**PM Increment:** NA **Effect Estimate [Lower CI, Upper CI]:** **Rate Ratios (95%CI) for stroke mortality in relation to modeled outdoor air pollution quintiles** Adjusted for sex and age: 1: 1 (ref) 2: 0.95 (0.84, 1.08) 3: 1.12 (0.99, 1.27) 4: 1.16 (1.03, 1.32) 5: 1.39 (1.23, 1.58) Adjusted for sex, age, deprivation, and smoking: 1: 1 (ref) 2: 0.94 (0.83, 1.07) 3: 1.08 (0.94, 1.24) 4: 1.12 (0.97, 1.29) 5: 1.33 (1.14, 1.56) **Rate Ratios (95%CI) for emergency hospital admissions because of stroke in relation to modeled outdoor air pollution quintiles** Adjusted for sex and age: 1: 1 (ref) 2: 1.06 (0.95, 1.17) 3: 1.10 (0.99, 1.23) 4: 1.25 (1.12, 1.38) 5: 1.40 (1.26, 1.55) Adjusted for sex, age, deprivation, and smoking: 1: 1 (ref) 2: 1.01 (0.91, 1.13) 3: 0.98 (0.87, 1.10) 4: 1.08 (0.96, 1.22) 5: 1.13 (0.99, 1.29) **Rate Ratios (95%CI) for stroke mortality in relation to spatially smoothed (using a 1-km radius) modeled outdoor air pollution quintiles** Adjusted for sex, age, socioeconomic deprivation, age by deprivation interaction, and smoking prevalence: 1: 1 (ref) 2: 0.86 (0.75, 0.98) 3: 1.05 (0.92, 1.21) 4: 1.03 (0.89, 1.19) 5: 1.24 (1.05, 1.47) **Rate Ratios (95%CI) for emergency hospital admissions because of stroke in relation to spatially smoothed modeled outdoor air pollution quintiles** Adjusted for sex, age, socioeconomic deprivation, age by deprivation interaction, and smoking prevalence: 1: 1 (ref) 2: 1.05 (0.94, 1.17) 3: 1.07 (0.95, 1.20) 4: 1.06 (0.94, 1.20) 5: 1.15 (1.01, 1.31)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Maheswaran et al. (2005) **Period of Study:** 1994-1998 **Location:** Sheffield, United Kingdom	**Outcome (ICD9 and ICD10):** Coronary Heart Disease (CHD) mortality (ICD9: 410-414) and Emergency hospital admissions (ICD10: I20-I25) **Age Groups:** ≥ 45 years **Study Design:** Ecological cross-sectional **N:** 1030 census enumeration districts (CEDs); 108 CEDs excluded from PM analyses due to artifacts in the emissions data **Statistical Analyses:** Poisson regression **Covariates:** age, sex, socioeconomic deprivation, and smoking prevalence; also included age-by-deprivation interaction) **Season:** NA **Dose-response Investigated?** Yes, examined quintiles of exposure **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 5-yr avg **Mean (SD):** Presented mean values and ranges for each quintile of exposure: 1: 16.0 (<16.8) 2: 17.5 (≥ 16.8, <18.2) 3: 18.8 (≥ 18.2, <19.3) 4: 19.8 (≥ 19.3, <20.6) 5: 23.3 (≥ 20.6) **Monitoring Stations:** NR **Copollutant (correlation):** CO (r = 0.82) NO_X (r = 0.87)	**PM Increment:** NA **Effect Estimate [Lower CI, Upper CI]:** **Rate Ratios (95%CI) for CHD mortality in relation to modeled outdoor air pollution quintiles** Adjusted for sex and age: 1: 1 (ref) 2: 1.06 (0.98, 1.16) 3: 1.10 (1.01, 1.21) 4: 1.23 (1.13, 1.35) 5: 1.30 (1.19, 1.43) Adjusted for sex, age, deprivation, and smoking: 1: 1 (ref) 2: 1.03 (0.94, 1.12) 3: 1.00 (0.90, 1.11) 4: 1.08 (0.98, 1.20) 5: 1.08 (0.96, 1.20) Adjusted for sex, age, deprivation, and smoking (spatially smoothed using a 1km radius): 1: 1 (ref) 2: 0.97 (0.89, 1.07) 3: 1.00 (0.90, 1.10) 4: 1.03 (0.93, 1.15) 5: 1.07 (0.96, 1.21) **Rate Ratios (95%CI) for emergency hospital admissions from CHD in relation to modeled outdoor air pollution quintiles** Adjusted for sex and age: 1: 1 (ref) 2: 1.08 (0.98, 1.19) 3: 1.11 (1.01, 1.22) 4: 1.17 (1.07, 1.29) 5: 1.36 (1.23, 1.50) Adjusted for sex, age, deprivation, and smoking: 1: 1 (ref) 2: 1.03 (0.93, 1.13) 3: 0.96 (0.86, 1.07) 4: 0.97 (0.87, 1.08) 5: 1.01 (0.90, 1.14) Adjusted for sex, age, deprivation, and smoking (spatially smoothed using a 1km radius): 1: 1 (ref) 2: 1.01 (0.92, 1.11) 3: 1.04 (0.93, 1.15) 4: 0.97 (0.87, 1.08) 5: 1.07 (0.95, 1.20)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: O'Neill et al. (2008) **Period of Study:** 2000-2004 **Location:** USA (6 field centers: Baltimore, MD; Chicago, IL; Forsyth Co, NC; Los Angeles, CA; New York, NY; St. Paul, MN	**Outcome (ICD9 and ICD10):** Creatinine adjusted urinary albumin exretion Assessed 2 ways: continuous log urinary albumin/creatine ration (UACR) and clinically defined micro- or macro-albuminuria (UACR ≥ 25 mg/g) versus normal levels **Age Groups:** 44-84 yrs **Study Design:** Cross-sectional analyses and prospective cohort analyses **N:** 3901 participants **Statistical Analyses:** Cross-sectional: multiple linear regression (continuous outcome); binomial regression (dichotomous outcome); Cohort: repeated measures model with random subject effects (estimate 3-yr change in log UACR by levels of exposure) **Covariates:** age, gender, race, BMI, cigarette status, ETS, percent dietary protein **Season:** NA **Dose-response Investigated?** Yes, examined quartiles of exposure **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** avg of previous month, avg of previous 2 months (recent exposures); 20-yr directly monitored PM_{10} avg, 20-yr imputed PM_{10} avg (longer-term exposures) **Mean (SD):** Previous 20 years: 34.7 (7.0) Previous month: 27.5 (7.9) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** NR (used closest monitor to residence to assign exposure) **Copollutant (correlation):** $PM_{2.5}$	**PM Increment:** 10 μg/m^3 **Effect Estimate [Lower CI, Upper CI]:** **Adjusted mean differences in log UACR (mg/g) per increase in PM_{10} seen at baseline** **Previous 30 days** Full sample: -0.42 (-0.085, 0.002) Within 10 km: -0.023 (-0.079, 0.034) **Previous 60 days** Full sample: -0.056 (-0.106 to -0.005) Within 10 km: -0.040 (-0.106, 0.025) **20 yr PM_{10} (nearest monitors)** Full sample: -0.019 (-0.072, 0.033) Within 10 km: 0.009 (-0.067, 0.085) **Imputed 20 yr exposure** Full sample: -0.002 (-0.038, 0.035) Within 10 km: 0.016 (-0.033, 0.066) **Adjusted relative prevalence of microalbuminuria vs high-normal and normal levels (below 25 mg/g) per increase in PM_{10} among participants without macroalbuminuria during the baseline visit** Previous 30 days: 0.88 (0.76, 1.02) Previous 60 days: 0.83 (0.70, 0.99) 20 yr PM_{10} (nearest monitors): 0.92 (0.77, 1.08) Imputed 20 yr exposure: 0.98 (0.87, 1.10) **Adjusted mean 3-yr change (SE) in log UACR (mg/g) by quartiles of 1982-2002 exposure to PM_{10} from ambient monitors among participants seen in 2000-20004** **Full sample** Quartile: 18.5 to <29.3: 0.147 (0.024) 29.3 to <33.1: 0.159 (0.024) 33.1 to <36.3: 0.163 (0.024) 36.3 to 55.7: 0.174 (0.023) p-trend: 0.42 **Within 10 km** Quartile: 18.5 to <29.3: 0.159 (0.030) 29.3 to <33.1: 0.155 (0.031) 33.1 to <36.3: 0.167 (0.028) 36.3 to 55.7: 0.152 (0.036) p-trend: 0.99

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Rosenlund et al. (2006) **Period of Study:** 1992-1994 **Location:** Stockholm County, Sweden	**Outcome (ICD9 and ICD10):** Myocardial infarction (MI) **Age Groups:** 45-70 yrs **Study Design:** Case-control **N:** 1397 cases; 1870 controls **Statistical Analyses:** Logistic regression (main analysis); also performed multinomial logistic regression to assess cases as nonfatal, fatal in the hospital within 28 days, and out-of-hospital death within 28 days with all controls as reference **Covariates:** age, sex, and hospital catchment area (frequency mathed variables); smoking, physical inactivity, diabetes, SES; also assessed but did not include hypertension, BMI, job strain, diet, passive smoking, alcohol consumption, coffee intake, and occupational exposure to motor exhaust and other combustion products **Season:** NA **Dose-response Investigated?** No **Statistical Package:** STATA v8.2	**Pollutant:** PM_{10} (modeled traffic-related pollution; also modeled $PM_{2.5}$, but since the PM correlation was high (r = 0.998) only PM_{10} results were presented) **Averaging Time:** 30 yrs (PM only assessed during 2000, thus assumed constant levels during 1960-2000) **Median (5th–95th percentile):** **Cases:** 2.6 (0.5-6.0) **Controls:** 2.4 (0.6-5.9) **Range (Min, Max):** NR **Monitoring Stations:** NR **Copollutant (correlation):** NO_2 (r = 0.93) CO (r = 0.66) SO_2	**PM Increment:** 5 $\mu g/m^3$ (5th to 95th percentile distribution among controls) **Effect Estimate [Lower CI, Upper CI]:** Association of 30-yr avg exposure to air pollution from traffic with MI Logistic regression All cases: 1.00 (0.79, 1.27) Multinomial logistic regression Nonfatal cases: 0.92 (0.71, 1.19) Fatal cases: 1.39 (0.94, 2.07) In-hospital death: 1.21 (0.75, 1.94) Out-of-hospital death: 1.84 (1.00, 3.40)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Zanobetti & Schwartz (2007) **Period of Study:** 1985-1999 **Location:** 21 US cities (Birmingham, Alabama; Boulder, Colorado; Canton, Ohio; Chicago, Illinois; Cincinnati, Ohio; Cleveland, Ohio; Colorado Springs, Colorado; Columbus, Ohio; Denver, Colorado; Detriot, Michigan; Honolulu, Hawaii; Houston, Texas; Minneapolis-St. Paul, Minnesota; Nashville, Tennessee; New Haven, Connecticut; Pittsburgh, Pennsylvania; Provo-Orem, Utah; Salt Lake City, Utah; Seattle, Washington; Steubenville, Ohio; and Youngstown, Ohio)	**Outcome (ICD9 and ICD10):** Death, subsequent myocardial infarction (MI; ICD9 codes 410.0-410.9), and a first admission for congestive heart failure (CHF; ICD9 code 428) **Age Groups:** ≥ 65 yrs **Study Design:** Cohort **N:** 196,000 persons discharged alive following an acute MI **Statistical Analyses:** Cox's Proportional Hazards **Covariates:** age, sex, race, type of MI, number of days of coronary care and intensive care, previous diagnoses for atrial fibrillation, and secondary or previous diagnoses for COPD, diabetes, and hypertension, and for season of initial event (time period, and, sex, race, and type of MI were treated as stratification variables) **Season:** Assessed as a confounder **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Yearly averages of pollution for that year and lags up to the 3 previous years (distributed lag) **Mean (SD):** 28.8 (all cities; SD not reported) **Percentiles:** 10, 50, and 90 percentiles listed individually for each city (Table 2) **Range (Min, Max):** NR **Monitoring Stations:** NR **Copollutant (correlation):** None	**PM Increment:** 10 μg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Hazard ratio (95%CI) an increase in PM for the year of failure and for the distributed lag from the year of failure up to 3 previous years **Death** PM_{10} annual: 1.11 (1.05, 1.19) Distributed lag model Lag 0: 1.04 (0.96, 1.14) Lag 1: 1.07 (0.99, 1.14) Lag 2: 1.14 (1.10, 1.18) Lag 3: 1.06 (0.99, 1.12) Sum lags 0-3: 1.34 (1.14, 1.52) **CHF** PM_{10} annual: 1.11 (1.03, 1.21) Distributed lag model Lag 0: 1.09 (1.01, 1.18) Lag 1: 1.09 (1.01, 1.19) Lag 2: 1.13 (1.02, 1.25) Lag 3: 1.04 (0.97, 1.12) Sum lags 0-3: 1.41 (1.19, 1.66) **2nd MI** PM_{10} annual: 1.17 (1.05, 1.31) Distributed lag model Lag 0: 1.09 (0.92, 1.30) Lag 1: 1.12 (0.97, 1.30) Lag 2: 1.15 (1.08, 1.23) Lag 3: 1.01 (0.94, 1.09) Sum lags 0-3: 1.43 (1.12, 1.82) Hazard Ratio (95%CI) for an increase in PM (sum of the previous 3 yrs distributed lag) for the sensitivity analyses **Death** Subjects with follow-up starting after 2nd MI: 1.33 (1.15, 1.55) Subjects admitted between 1985-1996: 1.45 (1.26, 1.68) 2nd cohort definition (year defined at time of MI): 1.29 (1.15, 1.44) **CHF** Subjects with follow-up starting after 2nd MI: 1.42 (1.22, 1.65) Subjects admitted between 1985-1996: 1.51 (1.26, 1.81) **2nd MI** Subjects admitted between 1985-1996: 1.62 (1.23, 2.13) **Note:** Age and sex effect modification results presented in Figure 1; used meta-regression to examine predictors of heterogeneity across city and found that most predictors were not significant modifiers of PM (Table 7)

Table E-22. Long-term effects–cardiovascular– $PM_{2.5}$ (including PM components/sources)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Allen et al. (In Press) **Period of Study:** Oct 2000–Sep 2002 (exposure averaging period); outcome assessed in 2002 **Location:** 5 US communities (Chicago, Illinois; Forsyth County, North Carolina; Los Angeles, California; Northern Manhattan and the Bronx, New York; and St. Paul, Minnesota); part of MESA (Multi-ethnic Study of Atherosclerosis)	**Outcome (ICD9 and ICD10):** Abdominal aortic calcium (AAC), a marker of systemic atherosclerosis (quantitative measure of interest was the Agatston score) **Age Groups:** 46-88 years **Study Design:** Cross-sectional **N:** 1,147 participants (sensitivity analysis among 1,269 participants) **Statistical Analyses:** 2-part modeling approach: 1) modeled relative risk of having any AAC using a log link and a Gaussian error model; sensitivity analysis used modified Poisson regression with robust error variance 2) multiple linear regression of the log-transformed AAC Agatston score (among those with AAC>0); sensitivity analysis modeled all participants by adding 1 prior to log-transforming **Covariates:** age, gender, race/ethnicity, BMI, smoking status, pack-year of smoking, diabetes, education, annual income, blood lipid concentration, blood pressure, and medications; assessed impact of gender, age, diabetes, obesity, use of lipid-lowering medications, education, income, race/ethnicity, and employment status on heterogeneity of effects (or in sensitivity analyses) **Season:** NA **Dose-response Investigated?** NR **Statistical Package:** SAS v9.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2 year averaging period (Oct 2000–Sep 2002) **Mean (SD):** 15.8 (3.6) **Percentiles:** NR **Range (Min, Max):** 10.6–24.7 **Monitoring Stations:** All monitors with 1) the objective of "population exposure," "regional transport," or "general/background;) and 2) at least 50% data reporting in each of 8 3-month periods over the averaging time; used monitors located within 50 km of a study participant's residence **Copollutant (correlation):** (assessed traffic by roadway proximity)	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Results for fully adjusted models under different participant inclusion, employment status, and roadway proximity criteria. **Presense/Absence of Calcium; RR (95% CI)** Inclusion criteria: <10yrs at address: 1.04 (0.89, 1.22) ≥ 10yrs at address: 1.06 (0.96, 1.16) ≥ 10yrs at address & <10km from monitor: 1.08 (0.98, 1.18) ≥ 20yrs at address: 1.10 (0.99, 1.22) ≥ 20yrs at address & <10km from monitor: 1.11 (1.00, 1.24) <10yrs at address & employed: 1.02 (0.87, 1.20) ≥ 20yrs at address & employed: 1.07 (0.89, 1.27) <10yrs at address & not employed: 1.10 (1.00, 1.22) ≥ 20yrs at address & not employed: 1.16 (1.02, 1.31) <10yrs at address & near major road: 0.85 (0.69, 1.05) ≥ 20yrs at address & not near major road: 1.10 (0.99, 1.23) **Log-transformed Agatston Score (Agatston >0); % Change (95% CI)** Inclusion criteria: <10yrs at address: -6.6 (-64.0, 50.9) ≥ 10yrs at address: 8.0 (-29.7, 45.7) ≥ 10yrs at address & <10km from monitor: 19.7 (-19.6, 58.9) ≥ 20yrs at address: 14.4 (-32.8, 61.7) ≥ 20yrs at address & <10km from monitor: 24.6 (-24.6, 73.8) <10yrs at address & employed: 29.1 (-25.7, 83.8) ≥ 20yrs at address & employed: 43.8 (-32.4, 119.9) <10yrs at address & not employed: -15.1 (-66.3, 36.1) ≥ 20yrs at address & not employed: -14.1 (-72.6, 44.4) <10yrs at address & near major road: 34.0 (-44.2, 112.1) ≥ 20yrs at address & not near major road: 3.9 (-39.9, 47.8) **Log-transformed Agatston Score (all); % Change (95% CI)** Inclusion criteria: <10yrs at address: -8.5 (-81.3, 64.2) ≥ 10yrs at address: 40.7 (-11.5, 92.8) ≥ 10yrs at address & <10km from monitor: 60.7 (5.9, 115.4) ≥ 20yrs at address: 64.1 (-1.73, 129.9) ≥ 20yrs at address & <10km from monitor: 79.2 (10.1, 148.3) <10yrs at address & employed: 33.5 (-35.9, 102.9) ≥ 20yrs at address & employed: 55.8 (-37.2, 148.7) <10yrs at address & not employed: 54.8 (-23.8, 133.4) ≥ 20yrs at address & not employed: 89.3 (-3.7, 182.3) <10yrs at address & near major road: -30.6 (-141.3, 80.1) ≥ 20yrs at address & not near major road: 51.3 (-8.3, 110.8)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			Exploratory/sensitivity analyses (also presented in figures): Detectable AAC; RR (95%CI): Among women: 1.14 (1.00, 1.30) Among persons >65yrs: 1.10 (1.01, 1.19) Among users of lipid-lowering medications: 1.14 (1.00, 1.30) Among Hispanics: 1.22 (1.03, 1.45) Imputing missing covariates among residentially stable participants: 1.08 (0.98, 1.19) **Agatston score; % change (95%CI):** Among Hispanics: 64 (-4, 133) Among persons earning >$50,000: 72 (5, 139) **Agatston score including those with Agatston = 0; % change (95%CI):** Fully adjusted model: 41 (-12, 93) Among persons >65yrs: 75 (8, 143) Among diabetics: 149 (29, 270) Among users of lipid-lowering medications: 121 (25, 217) Among Hispanics: 141 (45, 236) Imputing missing **Covariates:** 49 (1.3, 100.1)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Auchincloss et al. (2008) **Period of Study:** Jul 2000–Aug 2002 **Location:** 6 US communities (Baltimore City and Baltimore County, Maryland; Chicago, Illinois; Forsyth County, North Carolina; Los Angeles, California; Northern Manhattan and the Bronx, New York; and St. Paul, Minnesota); part of MESA (Multi-ethnic Study of Atherosclerosis)	**Outcome (ICD9 and ICD10):** Blood pressure: systolic (SBP), diastolic (DBP), mean arterial (MAP), pulse pressure (PP); Avg of 2nd and 3rd BP measurement used for analyses **Age Groups:** 45-84 years **Study Design:** Cross-sectional (Multi-Ethnic Study of Atherosclerosis baseline examination) **N:** 5,112 persons (free of clinically apparent cardiovascular disease) **Statistical Analyses:** Linear regression; secondary analyses used log binomial models to fit a binary hypertension outcome **Covariates:** age, sex, race/ethnicity, per capita family income, education, BMI, diabetes status, cigarette smoking status, exposure to ETS, high alcohol use, physical activity, BP medication use, meteorology variables, and copollutants; examined site as a potential confounder and effect modifier; heterogeneity of effects also examined by traffic-related exposures, age, sex, type 2 diabetes, hypertensive status, cigarette use **Season:** Adjusted for temperature and barometric pressure to adjust for seasonality (because seasons vary by the study sites); Also performed sensitivity analyses adjusting for season to examine the potential for residual confounding not accounted for by weather variables **Dose-response Investigated?** Assessed nonlinear relationships–no evidence of strong threshold/nonlinear effects for $PM_{2.5}$ **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 5 exposure metrics constructed: prior day, avg of prior 2 days, prior 7 days, prior 30 days, and prior 60 days **Mean (SD):** Prior day: 17.0 (10.5) Prior 2 days: 16.8 (9.3) Prior 7 days: 17.0 (6.9) Prior 30 days: 16.8 (5.0) Prior 60 days: 16.7 (4.4) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** Used monitor nearest the participant's residence to calculate exposure metrics **Copollutant (correlation):** SO_2 NO_2 CO Traffic-related exposures (straight-line distance to a highway; total road length around a residence)	**PM Increment:** 10 µg/m3 (approx. equivalent to difference between 90th and 10th percentile for prior 30 day mean) **Effect Estimate [Lower CI, Upper CI]:** Adjusted mean difference (95% CI) in PP and SBP (mmHg) per 10 µg/m3 increase in $PM_{2.5}$ (averaged for the prior 30 days) **Pulse Pressure** Adjustment variables: Person-level **Covariates:** 1.04 (0.25, 1.84) Person-level cov., weather: 1.12 (0.28, 1.97) Person-level cov., weather, gaseous copollutants: 2.66 (1.61, 3.71) Person-level cov., study site: 0.93 (-0.04, 1.90) Person-level cov., study site, weather: 1.11 (0.01, 2.22) Person-level cov., study site, weather, gaseous copollutants: 1.34 (0.10, 2.59) **Systolic Blood Pressure** Adjustment variables: Person-level **Covariates:** 0.66 (-0.41, 1.74) Person-level cov., weather: 0.99 (-0.15, 2.13) Person-level cov., weather, gaseous copollutants: 2.8 (1.38, 4.22) Person-level cov., study site: 0.86 (-0.45, 2.17) Person-level cov., study site, weather: 1.32 (-0.18, 2.82) Person-level cov., study site, weather, gaseous copollutants: 1.52 (-0.16, 3.21) **Additional results:** Associations became stronger with longer averaging periods up to 30 days. For example: Adjusted (personal covariates and weather) mean differences in PP: Prior day: -0.38 (-0.76, 0.00) Prior 2 days: -0.22 (-0.65, 0.21) Prior 7 days: 0.52 (-0.08, 1.11) Prior 30 days: 1.12 (0.28, 1.97) Prior 60 days: 1.08 (0.11, 2.05) (Pattern held for additional adjustments and for SBP results; therefore, only results for 30-day mean differences were presented) **Additional results (not presented):** None of DBP results were statistically significant; results for MAP were similar to SBP, though weaker and generally not significant Effect modification: associations between $PM_{2.5}$ and BP were stronger for persons taking medications, with hypertension, during warmer weather, in the presence of high NO_2, residing ≤ 300m from a highway, and surrounded by a high density of roads (Fig 1); associations were not modified for age, sex, diabetes, cigarette smoking, study site, high levels of CO or SO_2, season , nor residence ≤ 400m fro a highway **Note:** supplementary material available on-line

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Calderon-Garciduenas et al. (2007) **Period of Study:** Children recruited between Jul 2003 and Dec 2004 **Location:** Mexico (northeast or southwest Mexico city or Polotitlan)	**Outcome (ICD9 and ICD10):** Plasma Endothelin-1 (ET-1) and pulmonary arterial pressure (PAP) **Age Groups:** 6-13 years; 7.9 ± 1.3 years **Study Design:** Cross-sectional **N:** 81 children **Statistical Analyses:** Analysis of variance by parametric one-way analsis of variance and the Newman-Keuls multiple comparison post test **Covariates:** doesn't appear to have performed multivariable analyses; however, collected information on age, place and length of residency, daily outdoor time, household cooking methods, parents' occupational history, family history of atopic illnesses and respiratory disease, and personal history of otolaryngologic and respiratory symptoms **Season:** No **Dose-response Investigated?** No **Statistical Package:** STATA v8.3	**Pollutant:** $PM_{2.5}$ Exposures assessed quantitatively in Mexico City only; no monitors in Polotitlan **Averaging Time:** 1, 2, and 7 days before the exam; pollutant concentrations between 0700 and 1900 h were used for the estimates **Mean (SD):** Presented only in figures **Percentiles:** NR **Range (Min, Max):** Presented only in figures **Monitoring Stations:** 4 (2 in northeast and 2 in southwest Mexico City; residence and school within 5 miles of one of these monitors) **Copollutant (correlation):** O_3	**PM Increment:** NA **Effect Estimate [Lower CI, Upper CI]:** No health effects models with measured PM concentrations were presented; used city of residence to assign exposure; no multivariable analyses presented Authors presented (statistically significantly) elevated ET-1 levels among children residing in both areas of Mexico City as compared to Polotitlan (control city): Mean ± SE (pg/mL) Control: 1.23 ± 0.06 Southwest Mexico City: 2.40 ± 0.14 Northeast Mexico City: 2.09 ± 0.10 Authors presented (statistically significantly) elevated PAP levels among children residing in both areas of Mexico City as compared to Polotitlan (control city): Mean ± SE (mmHg) Control: 14.6 ± 0.4 Southwest Mexico City: 16.7 ± 0.6 Northeast Mexico City: 18.6 ± 0.9 Among Mexico City children only, there was a positive correlation between ET-1 levels and the 7-day cumulative $PM_{2.5}$ exposure (r = 0.28, p = 0.03)
Reference: Diez Roux et al. (2008) **Period of Study:** Baseline data collected June 2000–Aug 2002; Exposure assessed retrospectively between Aug 1982 and baseline date **Location:** USA (6 field centers: Baltimore, MD; Chicago, IL; Forsyth Co, NC; Los Angeles, CA; New York, NY; St. Paul, MN	**Outcome (ICD9 and ICD10):** Three measures of subclinical atherosclerosis (common carotid intimal-medial thickness (CIMT), coronary artery calcification, and ankle-brachial index (ABI)) **Age Groups:** 44-84 yrs **Study Design:** Cross-sectional **N:** 5172 for coronary calcium analysis; 5037 for CIMT analysis; 5110 for ABI analysis **Statistical Analyses:** Generalized Additive Models (Binomial regression: presence of calcification; Linear regression: CIMT, ABI, amount of calcium) **Covariates:** age, sex, race/ethnicity, socioeconomic factors, cardiovascular risk factors (BMI, hypertension, high density lipoprotein and low density lipoprotein cholesterol, smoking, diabetes, diet, physical activity; models presented with and without adjustment for cardiovascular RFs) **Season:** NA **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 20-yr imputed mean **Mean (SD):** 21.7 (5.0) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** NR; Long-term exposure to PM estimated based on residential history reported retrospectively; all addresses geocoded; ambient AP obtained from US EPA **Copollutant (correlation):** PM_{10} 20-yr observed mean; r = 0.64 PM_{10} 20-yr imputed mean; r = 0.73 PM_{10} 2001 mean; r = 0.43 $PM_{2.5}$ 2001 mean; r = 0.64 Due to high correlation among PM exposures, only results of mean 20-yr exposures are reported.	**PM Increment:** 12.5 µg/m^3 (approx. 10th-90th percentile) **Effect Estimate [Lower CI, Upper CI]:** **CIMT:** Relative difference (95% CI): 1.01 (1.00, 1.01) Adj. for additional CVD RFs: 1.01 (1.00, 1.02) **ABI:** Mean difference (95% CI): 0.000 (-0.006, 0.006) Adj. for additional CVD RFs: -0.001 (-0.006, 0.006) **Coronary calcium:** Relative prevalence (95% CI): 1.01 (0.96, 1.05) Adj. for additional CVD RFs: 1.01 (0.96, 1.06) **Coronary calcium (in those with calcium):** Relative difference (95% CI): 0.99 (0.88, 1.12) Adj. for additional CVD RFs: 1.01 (0.89, 1.14)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hoffman et al. (2007) **Period of Study:** 2000-2003 **Location:** Ruhr area of Germany (3 large cities: Essen, Mulheim, and Bochum)	**Outcome (ICD9 and ICD10):** Coronary artery calcification (CAC) **Age Groups:** 45-74 years **Study Design:** Cross-sectional **N:** 4494 participants **Statistical Analyses:** Linear regression (outcome = natural logarithm of CAC score + 1); logistic regression (outcome = CAC score above/below the age- and gender-specific 75th percentile) **Covariates:** city and area of residence, age, sex, education, smoking, ETS, physical inactivity, waist-to-hip ratio, diabetes, blood pressure, and lipids (and household income in a subset) **Season:** NA **Dose-response Investigated?** Yes, PM was also categorized into quartiles for analyses **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** One year (2002, midpoint of the study) **Mean (SD):** Total: 22.8 (1.5) High traffic (≤ 100m): 22.9 (1.4) Low traffic (>100m): 22.8 (1.5) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** NR **Copollutant (correlation):** None (Traffic was assessed using distance to roadways)	**PM Increment:** 3.91 µg/m^3 (10th-90th percentile) **Effect Estimate [Lower CI, Upper CI]:** **Percent change (95%CI) in CAC associated with an increase in** $PM_{2.5}$ Unadjusted: 12.7 (-7.0, 36.4) Model 1 (adjusted for distance to major road): 12.3 (-7.3, 35.9) Model 2 (model 1 + city and area of residence): 29.7 (0, 68.3) Model 3 (model 2 + age, sex, education): 24.2 (0, 55.1) Model 4 (model 3 + smoking, ETS, physical inactivity, waist-to-hip ratio): 17.9 (-5.3, 46.7) Model 5 (model 4 + diabetes, blood pressure, LDL, HDL, triglycerides): 17.2 (-5.6, 45.5) **Adjusted ORs (95%CI) for the association between the top quarter of PM exposure and the low quarter of PM exposure and a CAC score abover the age- and sex-specific 75th percentiles** All: 1.22 (0.96, 1.54) No CHD: 1.22 (0.95, 1.57) Men: 1.09 (0.78, 1.53) Women: 1.37 (0.97, 1.87) Age <60 yrs: 1.18 (0.83, 1.68) Age >60 yrs: 1.27 (0.93, 1.75) Nonsmokers: 1.17 (0.89, 1.53) Current smokers: 1.30 (0.83, 2.05) Educational level Low: 1.16 (0.86, 1.57) Medium: 1.30 (0.83, 2.05) High: 1.62 (0.81, 3.25) **Additional notes:** No clear dose-response relationship demonstrated when exposure assessed in quartiles (Figure 2) Participants who had not been working full-time during the last 5 years showed stronger effects, with possible dose-response between $PM_{2.5}$ and CAC (results presented in Figure 3)
Reference: Hoffman et al. (2006) **Period of Study:** Dec 2000–Jul 2003 **Location:** Ruhr area of Germany (2 large cities: Essen, Mulheim)	**Outcome (ICD9 and ICD10):** Clinically manifest CHD (defined as self-reported history of a 'hard' coronary event, i.e. myocardial infarction or application of a coronary stent or angioplasty or bypass surgery) **Age Groups:** 45-75 years **Study Design:** Cross-sectional **N:** 3399 participants **Statistical Analyses:** Multivariable logistic regression **Covariates: Season: Dose-response Investigated?** **Statistical Package:** SAS v8.2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Yearly mean estimated with model for year 2002 (on a spatial scale of 5 km) **Mean (SD):** Total: 23.3 (1.4) High traffic: 23.4 (1.4) Low traffic: 23.3 (1.4) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** NR **Copollutant (correlation):** None (Traffic was assessed using distance to roadways)	**PM Increment:** NA **Effect Estimate [Lower CI, Upper CI]:** $PM_{2.5}$ used only as a covariate in models assessing the relationship between traffic and CHD.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kunzli et al. (2005) **Period of Study:** 1998-2003 **Location:** Los Angeles Basin	**Outcome (ICD9 and ICD10):** Carotid intima-media thickness (CIMT) **Age Groups:** Less than 40 yrs excluded; mean age = 59.2 ± 9.8 **Study Design:** Cross-sectional **N:** 798 participants **Statistical Analyses:** Linear regression **Covariates:** age, sex, education, income, smoking, ETS, blood pressure, LDL cholesterol, treatment with antihypertensives or lipid-lowering medications **Season:** NA **Dose-response Investigated?** Yes, assessed $PM_{2.5}$ in quartiles **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** GIS/geostatics model to estimate 'long-term mean ambient concentrations of $PM_{2.5}$' derived from data collected in 2000 **Mean (SD):** 20.3 ± 2.6 **Percentiles:** NR **Range (Min, Max):** 5.2, 26.9 **Monitoring Stations:** 23 monitors **Copollutant (correlation):** None	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Percent change (95%CI) in CIMT associated with an increase in $PM_{2.5}$ concentration; based on a linear model with log intima-media thickness as dependent variable **Total populatioN:** Unadjusted: 5.9 (1.0, 10.9) Adjusted for age, sex, education, income: 4.4 (0.0, 9.0) Adjusted for above + smoking, ETS, multivitamins, alcohol: 4.2 (-0.2, 8.9) **Among Females ≥ 60 years:** Unadjusted: 19.2 (8.8, 30.5) Adjusted for age, sex, education, income: 15.7 (5.7, 26.6) Adjusted for above + smoking, ETS, multivitamins, alcohol: 13.8 (4.0, 24.5) **Among those taking lipid-lowering therapy:** Unadjusted: 15.8 (2.1, 31.2) Adjusted for age, sex, education, income: 13.3 (0, 28.5) Adjusted for above + smoking, ETS, multivitamins, alcohol: 13.3 (-0.3, 28.8) Unadjusted means of CIMT across quartiles of exposure were 734, 753, 758, and 774 µm; adjusted means trend across exposure groups, p = 0.041; stratified results presented in figures
Reference: Miller et al. (2007) **Period of Study:** 1994-2003 **Location:** 36 US metropolitan areas (Women's Health Initiative)	**Outcome (ICD9 and ICD10):** First cardiovascular event (myocardial infarction, coronary revascularization, stroke, and death from either coronary heart disease [categorized as "definite" or "possible"] or cerebrovascular disease) **Age Groups:** 50-79 years **Study Design:** Cohort (median follow-up of 6 yrs) **N:** 65,893 postmenopausal women without previous cardiovascular disease **Statistical Analyses:** Cox-proportional hazards regression **Covariates:** age, race/ethnicity, smoking status, the number of cigarettes smoked per day, the number of years of smoking, systolic blood pressure, education level, household income, BMI, and presence or absence of diabetes, hypertension, or hypercholesterolemia (also evaluated ETS, occupation, physical activity, diet, alcohol consumption, waist circumference, waist-to-hip ratio, medical history, medications, and presence or absence of a family history of cardiovascular disease as possible confounders in extended models) **Season:** NA **Dose-response Investigated?** **Statistical Package:** SAS v8.0, STATA v8.0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg concentration in 2000 (used to represent long-term exposure) **Mean (SD):** Individual exposure: 13.5 (3.7) Citywide avg exposure: 1.5 (3.3) **Percentiles:** Quintile ranges: 1: 3.4, 10.9 2: 11.0, 12.4 3: 12.5, 14.2 4: 14.3, 16.4 5: 16.5, 28.3 **Range (Min, Max):** Personal exposure: 3.4, 28.3 Citywide exposure: 4.0, 19.3 **Monitoring Stations:** 573 monitors; the nearest monitor to the location of each residence was used to assign exposure (monitor within 30 mi of residence) **Copollutant (correlation):** PM_{10} SO_2 NO_2 CO O_3	**PM Increment: 10** µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Estimated Hazards Ratio (95%CI) for the time to the first cardiovascular event or death associated with an increase in $PM_{2.5}$ Any cardiovascular event (first event) Overall: 1.24 (1.09, 1.41); Between cities: 1.15 (0.99, 1.32); Within cities: 1.64 (1.24, 2.18) Coronary heart disease (first event): Overall: 1.21 (1.04, 1.42); Between cities: 1.13 (0.95, 1.35); Within cities: 1.56 (1.11, 2.19) Cerebrovascular disease (first event): Overall: 1.35 (1.08, 1.68);Between cities: 1.20 (0.94, 1.54); Within cities: 2.08 (1.28, 3.40) MI (first event): Overall: 1.06 (0.85, 1.34); Between cities: 0.97 (0.75, 1.25); Within cities: 1.52 (0.91, 2.51) Coronary revascularization (first event): Overall: 1.20 (1.00, 1.43); Between cities: 1.14 (0.93, 1.39); Within cities: 1.45 (0.98, 2.16) Stroke (first event): Overall: 1.28 (1.02, 1.61); Between cities: 1.12 (0.87, 1.45); Within cities: 2.08 (1.25, 3.48) Any death from cardiovascular cause: Overall: 1.76 (1.25, 2.47); Between cities: 1.63 (1.10, 2.40); Within cities: 2.28 (1.10, 4.75) Coronary heart disease death (definite diagnosis): Overall: 2.21 (1.17, 4.16); Between cities: 2.22 (1.06, 4.62); Within

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			cities: 2.17 (0.60, 7.89) Coronary heart disease death (possible diagnosis): Overall: 1.26 (0.62, 2.56); Between cities: 1.20 (0.54, 2.63); Within cities: 1.57 (0.29, 8.51) Cerebrovascular disease death: Overall: 1.83 (1.11, 3.00); Between cities: 1.58 (0.90, 2.78); Within cities: 2.93 (1.03, 8.38) Estimated Hazard Ratios for cardiovascular events associated with an increase in $PM_{2.5}$ according to selected characteristics (presented adjusted H and adjusted H including adjustment for city) Any cardiovascular event: H: 1.24 (1.09, 1.41); H (city): 1.69 (1.26, 2.27) Household income <$20,000: H: 1.30 (1.10, 1.53); H (city): 1.75 (1.28, 2.40) Household income $20,000-49,999: H: 1.23 (1.08, 1.41); H (city): 1.69 (1.25, 2.27) Household income ≥ $50,000: H: 1.20 (1.02, 1.40); 6 H (city): 1.66 (1.22, 2.26) Education: Not high-school graduate: H: 1.40 (1.11, 1.75); H (city): 1.88 (1.32, 2.67) Education: High school grad/trade school/GED: H: 1.33 (1.14, 1.55); H (city): 1.79 (1.32, 2.44) Education: Some college or associate degree: H: 1.26 (1.09, 1.44); H (city): 1.74 (1.29, 2.34) Education: Bachelor's degree or higher: H: 1.11 (0.94, 1.31); H (city): 1.54 (1.13, 2.10) Age <60 yr: H: 1.21 (0.84, 1.73); H (city): 1.66 (1.05, 2.61) Age 60-69 yr: H: 1.14 (0.93, 1.39); H (city): 1.53 (1.09, 2.14) Age ≥ 70 yr: H: 1.34 (1.11, 1.63); H (city): 1.85 (1.34, 2.56) Current smoker: H: 1.68 (1.06, 2.66); H (city): 2.28 (1.33, 3.92) Former smoker: H: 1.24 (1.01, 1.52); H (city): 1.71 (1.23, 2.39) Never smoked: H: 1.39 (1.07, 1.80); H (city): 1.90 (1.31, 2.78) Living with smoker currently: H: 1.28 (0.84, 1.97); H (city): 1.65 (0.99, 2.76) Living with smoker formerly: H: 1.18 (1.00, 1.38); H (city): 1.59 (1.16, 2.16) Living with smoker never: H: 1.39 (1.07, 1.80); H (city): 1.90 (1.31, 2.78) BMI <22.5: H: 0.99 (0.80, 1.21); H (city): 1.35 (0.96, 1.88) BMI 22.5-24.7: H: 1.16 (0.96, 1.40); H (city): 1.58 (1.14, 2.19) BMI 24.8-27.2: H: 1.24 (1.05, 1.45); H (city): 1.69 (1.24, 2.30) BMI 27.3-30.9: H: 1.38 (1.18, 1.61); H (city): 1.88 (1.38, 2.56) BMI >30.9: H: 1.35 (1.12, 1.64); H (city): 1.84 (1.33, 2.55) Waist-to-hip ratio <0.74: H: 1.07 (0.90, 1.29); H (city): 1.45 (1.05, 2.00) Waist-to-hip ratio 0.74-0.77: H: 1.12 (0.95, 1.31); H (city): 1.51 (1.11, 2.06) Waist-to-hip ratio 0.78-0.80: H: 1.24 (1.07, 1.44); H (city): 1.68 (1.23, 2.27) Waist-to-hip ratio 0.81-0.86: H: 1.30 (1.13, 1.50); H (city): 1.76 (1.30, 2.38) Waist-to-hip ratio >0.86: H: 1.29 (1.11, 1.50); H (city): 1.75 (1.29, 2.37) Waist circumference <73 cm: H: 1.05 (0.86, 1.27); H (city): 1.43 (1.02, 1.99) Waist circumference 73-78 cm: H: 1.20 (1.02, 1.41); H (city): 1.63 (1.19, 2.23) Waist circumference 79-85 cm: H: 1.22 (1.05, 1.41); H (city): 1.66 (1.22, 2.24) Waist circumference 86-95 cm: H: 1.33

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			(1.15, 1.53); H (city): 1.80 (1.33, 2.43) Waist circumference >95 cm: H: 1.27 (1.07, 1.51); H (city): 1.73 (1.26, 2.36) Hormone-replacement therapy-Current Use: H: 1.33 (1.09, 1.61); H (city): 1.85 (1.32, 2.58) Hormone-replacement therapy-No Current Use: H: 1.16 (0.98, 1.39); H (city): 1.57 (1.14, 2.17) Diabetes–yes: H: 0.96 (0.67, 1.37); H (city): 1.24 (0.78, 1.96) Diabetes–no: H: 1.28 (1.12, 1.47); H (city): 1.75 (1.30, 2.36) Hypertension–yes: H: 1.22 (1.02, 1.45); H (city): 1.65 (1.09, 2.27) Hypertension–no: H: 1.26 (1.05, 1.51); H (city): 1.74 (1.25, 2.40) Hypercholesterolemia–yes: H: 1.25 (0.94, 1.67); H (city): 1.71 (1.15, 2.54) Hypercholesterolemia–no: H: 1.23 (1.07, 1.42); H (city): 1.69 (1.25, 2.28) Family history of CVD– yes: H: 1.30 (1.12, 1.51); H (city): 1.80 (1.32, 2.44) Family history of CVD– no: H: 1.07 (0.83, 1.37); H (city): 1.46 (1.00, 2.12) Time lived in current state: ≥ 20 yr: H: 1.21 (1.06, 1.39); H (city): 1.66 (1.23, 2.23) Time lived in current state: 10-19 yr: H: 1.39 (1.12, 1.72)' H (city): 1.97 (1.40, 2.79) Time lived in current state: ≤ 9 yr: H: 1.54 (1.06, 2.26); H (city): 2.24 (1.39, 3.59) Health insurance coverage–yes: H: 1.22 (1.07, 1.39); H (city): 1.71 (1.27, 2.30) Health insurance coverage–no: H: 1.82 (0.81, 4.10); H (city): 2.65 (1.12, 6.28) Time spent outdoors: <30 min: H: 1.09 (0.86, 1.39); H (city): 1.56 (1.05, 2.31) Time spent outdoors: ≥ 30 min; H: 1.26 (1.05, 1.50); H (city): 1.82 (1.29, 2.57)
Reference: O'Neill et al. (2008) **Period of Study:** 2000-2004 **Location:** USA (6 field centers: Baltimore, MD; Chicago, IL; Forsyth Co, NC; Los Angeles, CA; New York, NY; St. Paul, MN	**Outcome (ICD9 and ICD10):** Creatinine adjusted urinary albumin exretion Assessed 2 ways: continuous log urinary albumin/creatine ration (UACR) and clinically defined micro- or macro-albuminuria (UACR ≥ 25 mg/g) versus normal levels **Age Groups:** 44-84 yrs **Study Design:** Cross-sectional analyses and prospective cohort analyses **N:** 3901 participants **Statistical Analyses:** Cross-sectional: multiple linear regression (continuous outcome); binomial regression (dichotomous outcome); Cohort: repeated measures model with random subject effects (estimate 3-yr change in log UACR by levels of exposure) **Covariates:** age, gender, race, BMI, cigarette status, ETS, percent dietary protein **Season:** NA **Dose-response Investigated?** Yes, examined quartiles of exposure **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** avg of previous month, avg of previous 2 months (recent exposures); 20-yr imputed $PM_{2.5}$ avg (longer-term exposures) **Mean (SD):** Previous month: 16.5 (4.8) **Percentiles:** NR **Range (Min, Max):** NR **Monitoring Stations:** NR (used closest monitor to residence to assign exposure) **Copollutant (correlation):** PM_{10}	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** **Adjusted mean differences in log UACR (mg/g) per increase in $PM_{2.5}$ seen at baseline** **Previous 30 days** Full sample: -0.017 (-0.087, 0.052) Within 10 km: 0.026 (-0.067, 0.119) **Previous 60 days** Full sample: -0.040 (-0.121, 0.042) Within 10 km: -0.013 (-0.122, 0.097) **Imputed 20 yr exposure** Full sample: 0.002 (-0.048, 0.052) Within 10 km: -0.012 (-0.076, 0.053) **Adjusted relative prevalence of microalbuminuria vs high-normal and normal levels (below 25 mg/g) per increase in $PM_{2.5}$ among participants without macroalbuminuria during the baseline visit** Previous 30 days: 0.94 (0.77, 1.16) Previous 60 days: 0.90 (0.71, 1.14) Imputed 20 yr exposure: 0.98 (0.84, 1.14)

E.5. Long-Term Exposure and Respiratory Outcomes

Table E-23. Long-term exposure to PM_{10} and respiratory morbidity outcomes

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ackermann-Liebrich et al. (1997) **Period of Study:** 1991-1993 **Location:** Switzerland (Aarau, Basel, Davos, Geneva, Lugano, Montana, Payerne, Wald)	**Outcome:** Pulmonary function **Age Groups:** 18-60 yrs **Study Design:** Cross-sectional **N:** 9651 people **Statistical Analyses:** Regression analysis **Covariates:** Age, sex, height, weight, education level, nationality, workplace exposure **Season:** NR **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Continuously measured, 12 mo. avg. used **Mean (SD):** 21.2 (7.4) **Range:** (10.1-33.4) **Copollutant (correlation):** SO_2: r = 0.93 NO_2: r = 0.91 O_3: r = -0.55 Summer Daytime O_3: r = 0.31 Excess O_3: r = 0.67 Altitude: r = -0.77	**PM Increment:** 10 µg/m³ Regression Coefficient β (Lower CI, Upper CI) for air pollutants as predictors of pulmonary function FVC: -0.0345 (-0.0407 to -0.0283); p<0.001 FEV_1: -0.0160 (-0.0225 to -0.0095); p<0.001 Percent Change (Lower CI, Upper CI) associated with increase in avg annual air pollution concentration Healthy Never-smokers FVC: -3.39; p<0.001 FEV_1: -1.59; p<0.001 All Never-smokers FVC: -3.14; p<0.001 FEV_1: -1.06; p<0.001 Former Smokers FVC: -3.03; p<0.001 FEV_1: -0.42 Current Smokers FVC: -3.21; p<0.001 FEV_1: -1.35; p<0.001 All FVC: -3.14; p<0.001 FEV_1: -1.03; p<0.001 Long-term Residents FVC: -3.16; p<0.001 FEV_1: -0.96; p<0.001
Reference: Avol et al. (2001) **Period of Study:** 1993-1998 **Location:** Southern California	**Outcome:** FVC, FEV_1, MMEF, PEFR **Age Groups:** 10 yrs **Study Design:** cohort **N:** 110 **Statistical Analyses:** Linear regression **Covariates:** Sex, race, cohort entry year, annual avg change in height, weight, BMI **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 24 h PM_{10} averaged over 1994 **Mean (SD):** 15.0-66.2	**PM Increment:** 10 µg/m³ Mean Change (Lower CI, Upper CI) FVC: -1.8 (-9.1, 5.5) FEV_1: -6.6 (-13.5, 0.3) MMEF: -16.6 (-32.1 to -1.1) PEFR: -34.9 (-59.8 to -10.0)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Bayer-Oglesby et al. (2005) **Period of Study:** 1992-2001 **Location:** Switzerland (Lugano, Zurich, Bern, Geneva, Anieres, Biel, Langnau, Payerne, & Montana)	**Outcome:** Respiratory symptoms (chronic cough, bronchitis, cold, dry cough, conjunctivitis, wheeze, sneezing, asthma, & hay fever) **Age Groups:** 6-15 yrs **Study Design:** cross-sectional **N:** 9,591 children **Statistical Analyses:** Logistic regression models **Covariates:** age, sex, nationality, parental education, number of siblings, farming status, low birth weight, breast feeding, smoking, family history of asthma, bronchitis and/or atopy, mother who smokes, indoor humidity, mode of cooking & heating, carpeting, pets, removal of carpets/pets for health reasons, completed questionnaire & month, days max temperature <0°C, mother's belief of association between environmental exposures & respiratory health **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** PM_{10} **Averaging Time:** 12 month avg **Mean (SD):** NR **Range (Min, Max):** NR **Monitoring Stations**: 9 **Copollutant (correlation):** NR	**PM Increment:** 10 µg/m^3 "Figure 2 shows that declining levels of PM_{10} were associated with declining prevalanece of chronic cough, bronchitis, common cold, nocturnal dry cough, and conjunctivitis symptoms. For wheezing, sneezing, asthma, and hay fever, no significant association could be seen with declining PM_{10} levels." "Figure 3 illustrates that, on an aggregate level, across regions the mean change in PM_{10} levels (rpearson = 0.81, p = 0.008). The strongest decline of adjusted prevalence of nocturnal dry cough was observed in Geneva, Lugano, and Anieres, where the strongest reduction of PM_{10} had also been achieved."
Reference: Burr et al. (2004a) **Period of Study:** 3 weeks in July and Jan 1997 and 2 weeks in Nov 1996 and April 1997 **Location:** North Wales, England	**Outcome:** Self-report of symptoms only for wheeze, cough, phlegm, rhinitis, and itchy eyes. **Age Groups:** all **Study Design:** Repeated measures **N:** 386 persons in congested streets and 425 in the uncongested streets in 1996/1997. Of these, 165 and 283 completed the second phase of the study.	**Pollutant:** PM_{10} **Averaging Time:** Mean hourly concentrations **Mean (SD):** SD NR Congested streets – 1996-97 35.2 1998-99 27.2 Uncongested Streets 1996-97 11.6 1998-99 8.2 **Monitoring Stations**: 1 in congested street and 1 in uncongested	Percent change PM_{10} in congested streets: 22.7 Percent change PM_{10} in uncongested streets: 28.9 Uncongested street sampling site was 20 m from the congested street sampler. The opening of the by-pass produced a reduction in pollution in the congested streets. The health effects of these changed are likely to be greater for nasal and ocular symptoms than for lower respiratory symptoms. Uncertainty about the causality arises from low reponse rates and conflicting trends in respiratory and nasal symptoms.
Reference: Calderon-Garciduenas et al. (2006) **Period of Study:** 1999, 2000 **Location:** Southwest Mexico City & Tlaxcala, Mexico	**Outcome:** Hyperinflation, interstitial markings-measured by chest radiograph, and lung function–FVC, FEV_1, PEF, FEF25-75, measured using spirometry tests **Age Groups:** 5-13 yrs **Study Design:** Cohort **N:** 249 (total), 230 (Southwest Mexico City), 19 (Tlaxcala) **Statistical Analyses:** Bayes test, Spearman rank correlation, multiple regression **Covariates:** Age, sex **Dose-response Investigated?** No **Statistical Package:** SAS 8.2	**Pollutant:** PM_{10} **Averaging Time:** 1 yr **Mean (SD):** Mexico City 1999–48 2000–45 Tlaxacala: 1994-2000: <NAAQS std **Monitoring Stations**: Southwest Mexico City–2 Tlxacala–periodic air monitoring data **Copollutant:** O_3	**PM Increment:** NR % Change: % of children with FEV_1 <80% expected value: Mexico City (n = 77): 7.8% Tlaxacala (n = 19): 0% % children with hyperinflatioN: Mexico City: 65.6% No hyperinflation: 79 Mild: 72 Moderate: 56 Severe: 23 Tlaxacala: 5.3% No hyperinflation: 18 Mild: 1 Moderate: 0 Severe: 0 % children with interstitial markings: Mexico City: 52.6% Number with: No interstitial markings: 19 Mild: 0 Moderate: 0 Severe: 0 Tlaxacala: 0% No interstitial markings: 109 Mild: 112 Moderate: 9 Severe: 0

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Calderon-Garciduenas, et al. (2003) **Period of Study:** Jan 1999-Jun 2000 **Location:** Mexico City, Tuxpam, and Tlaxcala, Mexico	**Outcome:** Respiratory system changes **Age Groups:** 5-17 yrs **Study Design:** Case-control of subjects examined for this study **N:** 174 cases, 27 controls, children **Statistical Analyses:** Chi-square test with Yates correction, Spearman's rank correlation test. **Dose-response Investigated?** No **Statistical Package:** SAS 8.2	**Pollutant:** PM_{10} **Averaging Time:** 12 h (daytime 08: 00-20: 00) and nighttime (20: 00-08: 00) **Mean (SD):** Mexico City Day/Night Jan-Jun 1999 76.0/50.0 Jul-Dec 1999 42.8/22.5 Jan-Jun 2000 75.2/47.5	Daily ambient exposure of children to a complex mixture of air pollutants produces significant chest X-ray abnormalities, a decrease in predicted values of FEF25-75, FEF75, and the FEV_1/FVC ratio in association with interstitial marking on chest X-rays, a mild restrictive pattern by spirometry, peripheral blood abnormalities, and an imbalance of serum cytokines.
Reference: Cavanagh et al. (2007) **Period of Study:** Mar-Aug 2004 **Location:** Christchurch, New Zealand	**Outcome:** A clinical study of excretion of 1-hydroypyrene (1-OHP) as a marker of PAH exposure **Age Groups:** non-smoking males aged 12-18 yr **Study Design:** Comparison of 2 high pollution events and 2 low pollution events **N:** 89 male students in a boarding school **Statistical Analyses:** Wilcoxon signed rank test for paired observations, Mann-Whitney U test **Season:** Winter **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Autumn Low Outdoor 19 Indoor NA Winter I Outdoor 43 Indoor 38 Winter II Outdoor 72 Indoor 84 Winter Low Outdoor 12 Indoor 16 **Monitoring Stations**: One inside the boarding house, and one outside	Urinary 1-OHP were raised after high-pollutions events. Peaks were slightly higher than for US non-smokers of similar ages and slightly lower than for German non-smokers of similar ages. Urinary 1-OHP was slightly higher in asthmatics compared to non-asthmatics. There were no indoor sources of PAHs (wood-burning stoves, tobacco smoke). Diet is another source of PAHs, but all students ate in the boarding house. These results suggest 1-OHP could be used as a biomarker of ambient air pollution.
Reference: Downs et al. (2007) **Period of Study:** 1991, 2002 **Location:** Switzerland	**Outcome:** FEV_1, FEV_1 as % of FVC, FEF25-75 **Age Groups:** 18-60 years **Study Design:** Prospective Cohort **N:** 4742 people **Statistical Analyses:** Linear random effects models **Covariates:** Age, sex, height, parental smoking, season, education, nationality, occupational exposure, smoking (status, pack-years), atopy, BMI **Dose-response Investigated?** Yes–linear fit best **Statistical Package:** SAS 9.1, STATA 8.2, R 2.4	**Pollutant:** PM_{10} **Averaging Time:** Annual **Mean:** Mean interval exposure: 238 µg/m³/years **Percentiles:** 25th: 197 75th: 287	**PM Increment:** 10 µg/m³ reduction in annual mean Percent / absolute reduction in annual decline in lung function over 11-year period (95% CI): Annual decline in FEV_1 reduced by 9% / 3.1 mL (0.03-6.2) Annual decline in FEF_{25-75} reduced by 16% / 11.3 mL/second (4.3-18.2) Annual decline in FEV_1 as a percentage of FVC of 0.06 (0.01-0.12) A reduction in interval exposure of 109 µg per m3 cubic meter–years (equivalent to a reduction of 10 µg/m³ in the annual avg during the mean follow-up time of 10.9 years) was associated with: A reduction of 6.9 mL (95% CI, 2.1 to 11.7) in the annual decline in FEV_1 A 22% reduction in the annual decline in $FEF_{25–75}$ (i.e., by 14.0 mL per second; 95% CI, 3.1 to 24.8)
Reference: Gauderman et al. (2000a) **Period of Study:** 1993-1997 **Location:** Southern California	**Outcome:** FVC, FEV_1, MMEF, FEF_{75} **Age Groups:** fourth, seventh, or tenth graders **Study Design:** cohort **N:** 3035 subjects **Statistical Analyses:** Linear regression **Covariates:** Height, weight, BMI, asthma, smoking, exercise, room temperature, barometric pressure **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24 h avg PM_{10} **Mean (SD):** PM_{10} 51.5 **Copollutant (correlation):** $PM_{2.5}$ r = 0.96 O_3 r = -0.32 $PM_{10-2.5}$ r = 0.92 NO_2 r = 0.65 Inorg. Acid r = 0.68	**PM_{10} Increment:** 51.5 µg/m³ % Change (Lower CI, Upper CI) PM_{10}-4th grade FVC -0.58 (-1.14 to -0.02) FEV_1 -0.85 (-1.59 to -0.10) MMEF -1.32 (-2.43 to -0.20) FEF75 -1.63 (-3.14 to -0.11) PM_{10}-7th grade FVC -0.45 (-1.03, 0.13) FEV_1 -0.44 (-1.10, 0.23) MMEF -0.48 (-2.51, 1.59) FEF_{75} -0.50 (-2.26, 1.29) PM_{10}-10th grade FVC 0.07 (-0.99, 1.13) FEV_1 -0.46 (-1.84, 0.94) MMEF -0.71 (-4.87, 3.63) FEF_{75} -1.54 (-5.61, 2.71)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Gauderman et al. (2002a) **Period of Study:** 1996–2000 **Location:** Southern California	**Outcome:** Lung function development: FEV_1, maximal midexpiratory flow (MMEF) **Age Groups:** Fourth grade children (avg age = 9.9 yrs) **Study Design:** Cohort study **N:** 1678 children, 12 communities **Statistical Analyses:** Mixed model linear regression **Covariates:** Height, BMI, doctor-diagnosed asthma and cigarette smoking in previous year, respiratory illness and exercise on day of test, interaction of each of these variables with sex, barometric pressure, temperature at test time, indicator variables for field technician and spirometer **Dose-response Investigated?** Yes **Statistical Package:** SAS (10)	**Pollutant:** PM_{10} **Averaging Time:** Annual 24 h averages **Mean (SD):** The avg levels were presented in an online data supplement (Figure E1) **Monitoring Stations**: 12 **Copollutant (correlation):** O_3 (10 AM to 6 PM) r = 0.13 O_3 r = -0.37 NO_2 r = 0.64 Acid vapor r = 0.79 $PM_{2.5}$ r = 0.95 $PM_{10-2.5}$ r = 0.95 EC r = 0.86 OC r = 0.97	**PM Increment:** 51.5 µg/m^3 Association Estimate: None of the pulmonary function tests had a statistically significant correlation with PM_{10} FEV_1 r = -0.12 p = 0.63 MMEF r = -0.22 p = 0.30
Reference: Gauderman et al. (2004) **Period of Study:** Air pollution data ascertainment: 1994-2000. Spirometry testing: spring 2001- spring 2003 **Location:** 12 Communities in Southern California	**Outcome:** Lung function FVC, FEV_1, MMEF (Maximal midexpiratory flow rate) **Age Groups:** Children, Avg age 10 years **Study Design:** Prospective Cohort Study **N:** 12 Communities 2,034 Children 24,972 child-months **Statistical Analyses:** Linear regression of changes in sex-and-community specific lung growth function and PM **Covariates:** Random effect for communities **Season:** ALL (except for $PM_{2.5}$) **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24-h measurements over each year used to create annual avg Mean: Means are presented in figures only. **Range (Min, Max):** ~15, ~65 **Monitoring Stations**: 12 **Copollutant (correlation):** O_3: r = 0.18 NO_2: r = 0.67 $PM_{2.5}$: r = 0.95 EC: r = 0.85 OC: r = 0.97	**PM Increment:** Most to least polluted community Range: PM_{10}: 51.4 µg/m^3 EC: 1.2 µg/m^3 OC: 10.5 µg/m^3 Difference in Lung Growth [Lower CI, Upper CI]; FVC -60.2 (-190.6 to 70.3) FEV_1 -82.1 (-176.9 to 12.8) MMEF -154.2 (-378.3 to 69.8) EC: FVC -77.7 (-166.7 to 11.3) FEV_1 -87.9 (-146.4 to -29.4) MMEF -165.5 (-323.4 to -7.6) OC: FVC -58.6 (-196.1 to 78.8) FEV_1 -86.2 (-185.6 to 13.3) MMEF -151.2 (-389.4 to 87.1) Correlation with % below 80% predicted Lung function (p-value) PM_{10}: 0.66 (0.02) EC: 0.74 (0.006)
Reference: Gauderman et al. (2007) **Period of Study:** 1993-2004 **Location:** 12 Southern California Communities	**Outcome:** pulmonary function tests FVC, FEV_1, $MMEF/FEF_{25-75}$ **Age Groups:** Children (mean age 10 at recruitment, followed for 8 years) **Study Design:** Cohort Study (Children's Health Study) **N:** 3677 children (1718 in cohort 1 recruited 1993 and 1959 in cohort 2 recruited 1996) 22686 pulmonary function tests. **Statistical Analyses:** Hierarchical mixed effects model with linear splines **Covariates:** Adjustments for height, height squared, BMI, BMI squared, present asthma status, exercise or respiratory illness on day of test, smoking in previous year, field technician, traffic indicator (distance from freeway, distance from major roads), random effects for participant and community. **Dose-response Investigated?** no **Statistical Package:** SAS	**Pollutant:** PM_{10} **Monitoring Stations**: 1 in each community	**PM Increment:** 51.4 µg/m^3 Pollutant effect reported as difference in 8 year lung function growth from least to most polluted community. Negative difference indicates growth deficits associated with exposure. For PM_{10} FEV growth deficit is -111

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Goss et al. (2004a) **Period of Study:** 1999-2000 **Location:** USA	**Outcome:** Cystic Fibrosis pulmonary exacerbations, FEV_1 **Age Groups: >** 6 **Study Design:** cohort **N:** 11484 patients **Statistical Analyses:** Logistic regression, t-tests, Mann-Whitney tests, Chi-squared tests, polytomous regression, multiple linear regression **Covariates:** Age, sex, lung function, weight, insurance status, pancreatic insufficiency, airway colonization, genotype, median household income by census tract, zipcode. **Dose-response Investigated?** No **Statistical Package:** STATA, SAS	**Pollutant:** PM_{10} **Averaging Time:** annual mean of 24 h averages **Mean (SD):** 24.8(7.8) mg/m^3 Percentiles: 25th: 20.3 50th(Median): 24.0 75th: 28.9 **Monitoring Stations**: 626	**PM Increment:** 10 $\mu g/m^3$ **Odds Ratio Estimate [Lower CI, Upper CI]:** Odds of having 2 or more pulmonary exacerbations as compared to 1 or less in 2000 1.08 (1.02 -1.15) Odds of having 2 or more pulmonary exacerbations as compared to noo exacerbations in 2000 1.09 (1.02 -1.17) Decrease in FEV_1 38ml(18-58)
Reference: Ho et al. (2007) **Period of Study:** Oct 1995-Mar 1996 **Location:** Taiwan, Republic of China	**Outcome:** Asthma **Age Groups:** 10-17 yrs **Study Design:** Screened junior high students for asthma, collected meteorological data to determine the relationship. **N:** 69,367 **Statistical Analyses:** Logistic regression model, the maximum likelihood estimation with Fisher's scoring algorithm, stepwise regression model, Wald statistic, Akaike criteria. General estimating equation, GENMOD **Covariates:** Wind, barometric pressure, temperature, rain, humidity **Season:** Fall-spring **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** Monthly **Monitoring Stations**: 72	**Odds Ratio from stepwise regression model:** Females (n = 32, 648) 0.993 [0.990-0.997] Males: NS Higher PM_{10} concentration resulted in less asthma prevalence. However, a higher number of rain days seemed to reduce asthma prevalence; rain days might interact with PM_{10}.
Reference: Hong et al. (2004) **Period of Study:** 2001 **Location:** Kerinci, SP7, and Pelalawan, Indonesia	**Outcome:** Respiratory symptoms **Age Groups:** <12 yrs **Study Design:** Disproportionate random sampling was used to select 100 households from each village. An interviewer interviewed all children through the caregiver/parent to obtain symptoms in the past 2 weeks (cough, cold, phlegm) and the last 12 months. **N:** 382 children **Statistical Analyses:** Chi-square test, analysis of variance, prevalence rates, adjusted odds ratios, multivariate adjusted odds ratios from multiple logistic regression models, allowing for clustering. **Covariates:** Age, gender, no. of children in household, household income, floor area of house, fuel for cooking, no. of smokers in household, personal and family medical history. **Dose-response Investigated?** No **Statistical Package:** SPSS STATA v.7	**Pollutant:** PM_{10} **Averaging Time:** 24 h measurements were taken daily from 2 weeks before the field survey to 1 month after the survey **Mean (SD):** Kerinci 102.9 (49.6) $\mu g/m^3$ SP7 73.7 (41.7) Pelalawan 26.1 (14.5) P<0.01 **Range (Min, Max):** Kerinci 25, 184 SP7 13, 138 Pelalawan 10, 66 **Monitoring Stations**: 3	**PM Increment:** Low (Pelalawan), Medium (SP7), & High (Kerinci) PM Exposure Odds Ratios (95% CI) for Symptoms by village: Cough/cold past 2 wks Pelalawan 1.00 SP7 2.03 (1.04, 3.96) Kerinci 3.17 (1.43, 7.07) Respiratory symptoms last 12 months Pelalawan 1.00 SP7 1.15 (0.58, 2.26) Kerinci 1.42 (0.62, 3.25) Ever had rhinitis w/o flu Pelalawan 1.00 SP7 2.17 (0.57, 8.29) Kerinci 0.56 (0.11, 2.83) Ever had wheezing Pelalawan 1.00 SP7 0.85 (0.35, 2.08) Kerinci 1.18 (0.46, 3.01)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Horak et al. (2002) **Period of Study:** 1994-1997 **Location:** Lower Austria	**Outcome:** Lung function growth measured by changes iN: 1. FVC (forced vital capacity) 2. FEV_1 3. MEF_{25-75} (midexpiratory flow between 25-75% of the forced vital capacity) **Age Groups:** 2-3 grade schoolchildren (mean age = 8) **Study Design:** Prospective cohort with repeated measures **N:** 975 children **Statistical Analyses:** Linear regression GEE, nonstationary M-dependent correlation structure **Covariates:** Gender, atopy, ETS exposure, baseline lung function, first height, height difference, school site **Season:** Winter, summer **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Mean (SD):** Winter: 21.0 (4.8) summer: 17.4 (2.8) **Range (Min, Max):** Winter: 9.4-30.5 summer: 11.7-28.9 **Monitoring Stations**: NR, stations were located in the immediate vicinity of each of the 8 elementary schools **Copollutant (correlation):** Winter O_3: (r = -0.581) SO_2 (r = 0.520) NO_2 (r = 0.595) summer O_3 (r = -0.429) SO_2 (r = 0.335) NO_2 (r = 0.412)	**PM Increment:** 1 µg/m^3 Mean per unit increase in PM (p-value); **Outcome:** difference per day of FVC (mL/day) Summer: 0.001 (0.938); Winter: 0.008 (0.042) Controlling for temperature: Summer: -0.007 (0.417); Winter: -0.003 (0.599) Controlling for O_3: Summer: 0.001 (0.911); Winter: 0.010 (0.019) Controlling for NO_2: Summer: -0.018 (0.056); Winter: 0.015 (0.000) Controlling for SO_2: Summer: 0.005 (0.575); Winter: 0.004 (0.492) In non-asthmatic children: Summer: -0.003 (0.710); Winter: 0.009 (0.030) In group not exposed to ETS: Summer: 0.014 (0.154); Winter: 0.012 (0.0018) In group exposed to ETS: Summer: 0.022 (0.088); Winter: 0.003 (0.656) Outcome: difference per day of FEV_1 (mL/day) Summer: -0.023 (0.003); Winter: 0.001 (0.885) Controlling for temperature: Summer: -0.034 (0.000); Winter: -0.011 (0.016) Controlling for O_3: Summer: -0.022 (0.008); Winter: 0.004 (0.338) Controlling for NO_2: Summer: -0.038 (0.000); Winter: 0.011 (0.005) Controlling for SO_2: Summer: -0.022 (0.010); Winter: -0.005 (0.358) Outcome: difference per day MEF25-75 (mL/day) Summer: -0.090 (0.000); Winter: -0.008 (0.395) Controlling for temperature: Summer: -0.112 (0.000); Winter: -0.013 (0.295) Controlling for O_3: Summer: -0.087 (0.000); Winter: -0.008 (0.434) Controlling for NO_2: Summer: -0.102 (0.000); Winter: 0.005 (0.610) Controlling for SO_2: Summer: -0.095 (0.000); Winter: -0.011 (0.474)
Reference: Hwang et al. (2006) **Period of Study:** 2001 **Location:** Taiwan	**Outcome:** Peak expiratory flow rate (PEFR), Forced Expiratory Volume in 1 second (FEV_1), Forced Vital Capacity (FVC), Self reported "frequent coughing," Self reported "shortness of breath," Self reported " irritation of respiratory tract" **Age Groups:** 24-55 years (mean = 40) **Study Design:** Cohort **N:** 120 men (60 traffic policemen and 60 controls) **Statistical Analyses:** ANOVA, odds ratios calculated from 2X2 table **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Mean (SD):** 55.58 (16.57) Percentiles: 25th: 42.96 50th(Median): 53.81 75th: 70.37 **Range (Min, Max):** 29.36, 99.58 **Monitoring Stations**: 22 **Copollutant (correlation):** NO_X (r = 0.34) SO_2 (r = 0.58) CO (r = 0.27) O_3 (r = 0.28)	**PM Increment:** 10 µg/m^3 RR Estimate [Lower CI, Upper CI]; Single pollutant model: 1.00 [0.99, 1.02] Controlling for NO_X: 0.99 [0.97, 1.00] Controlling for CO: 1.00 [0.99, 1.01] Controlling for O_3: 1.00 [0.99, 1.02]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ingle et al.(2005) **Period of Study:** May 2003-April 2004 **Location:** Jalgaon City, India	**Outcome:** Peak expiratory flow rate (PEFR), Forced Expiratory Volume in 1 second (FEV_1), Forced Vital Capacity (FVC), Self reported "frequent coughing," Self reported "shortness of breath," Self reported " irritation of respiratory tract" **Age Groups:** 24-55 years (mean = 40) **Study Design:** Cohort **N:** 120 men (60 traffic policemen and 60 controls) **Statistical Analyses:** ANOVA, odds ratios calculated from 2X2 table **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Mean (SD):** Location-specific means: Prabhat: 224 (27) Ajanta: 269 (41) Icchdevi: 229 (24) **Monitoring Stations**: 3	OR Estimate [p-value]; Self reported frequent coughing 2.96 [p<0.05] Self reported shortness of breath 1.22 [p<0.05] Self reported irritation in respiratory tract 7.5 [p<0.05] Observed/expected lung function; p-value for difference between groups: FVC (L) Traffic policemen: 0.82 Controls: 0.99 Traffic policemen: Obs = 3.03 ± 1.7 Exp = 3.70 ± 2.8 Controls: Obs = 3.18 ± 0.91 Exp = 3.19 ± 1.71 FEV_1 (L) Traffic policemen: 0.73 Controls: 1.18 Traffic policemen: Obs = 2.27 ± 1.05 Exp = 3.08 ± 2.7 Controls: Obs = 3.61 ± 0.90 Exp = 3.06 ± 0.91 PEFR (L/s) Traffic policemen: 0.66 Controls: 0.92 Traffic policemen: Obs = 6.05 ± 2.15 Exp = 9.21 ± 0.47 Controls: Obs = 5.54 ± 1.85 Exp = 6.11 ± 2.31

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kan, et al. (2007b) **Period of Study:** 1987-1992 **Location:** Four Communities in the U.S.: Forsyth County, North Carolina; Jackson, Mississippi; northwest suburbs of Minneapolis, Minnesota; and Washington County, Maryland.	**Outcome:** FEV_1 and FVC **Age Groups:** Middle-aged (mean age was 54.2 years) **Study Design:** Hierarchical regression **N:** 15,792 **Statistical Analyses:** SAS PROC MIXED **Covariates:** Distance to major roads, traffic exposure, age, ethnicity, sex, smoking, environmental tobacco smoke exposure, occupation, education, medical history, BMI. **Dose-response Investigated?** No **Statistical Package:** SPSS Version 11 for traffic density, SAS Version 9.1.2 for statistical analysis	**Pollutant:** PM_{10} **Averaging Time:** 24-h PM_{10} averaged over study period **PM Component:** Vehicle emissions **Monitoring Stations**: 0 **Copollutant:** NO_2 O_3	**RR Estimate (Lower CI, Upper CI):** (Note: for ARIC participants living <150 meters from major roads) Women FEV_1(mL) Age-adjusted model -29.5 (-52.2 to -6.9) Multivariate model -15.7 (-34.4 to -2.9) FVC (mL) Age-adjusted model -33.2 (-60.4 to -5.9) Multivariate model -24.2 (-46.2,-2.3) FEV_1/FVC (%) Age-adjusted model -0.1(-0.5,0.2) Multivariate model 0.1 (-0.3,0.4) Men FEV_1(mL) Age-adjusted model -38.4 (-76.7,0.6) Multivariate model -6.4 (-38.1,25.3) FVC (mL) Age-adjusted model -17.0(-62.0,28.0) Multivariate model 10.9(-24.7,46.5) FEV_1/FVC (%) Age-adjusted model -0.05 (-0.9,0.0) Multivariate model -0.3 (-0.7,0.2)
Reference: Kim et al. (2005b) **Period of Study:** Mar and Dec 2000 **Location:** Incheon & Ganghwa, Korea	**Outcome:** lung function (FEV_1, FVC) **Age Groups:** middle school students **Study Design:** Panel **N:** 368 children **Statistical Analyses:** Generalized liner model **Covariates:** gender, grade **Season:** Spring and fall **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** monthly **Mean (SD):** Incheon March 64 December 54 Ganghwa March 64 December 53 **Range (Min, Max):** NR	**PM Increment:** NR **OR Estimate [Lower CI, Upper CI]:** "The present study showed that the values of FEV_1 and FVC were greater in December than in March for both male and female students at all academic years…Because only the level of PM_{10} was significantly higher for March than for December in both areas, we suggest that decrements of pulmonary function in March for both areas are associated with the increased level of PM_{10}"

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kim et al. (2004) **Period of Study:** Mar-June (spring) 2001; Sep-Nov (fall) 2001 **Location:** Alameda County, CA	**Outcome:** Asthma, bronchitis **Age Groups:** Children (in grades 3-5) **Study Design:** Cross-sectional **N:** 1109 children, 871 (long term resident children), 462 (long term related females), 403 (long term related males) **Statistical Analyses:** 2-stage multiple logistic regression model **Covariates:** respiratory illness before age of 2, household mold/moisture, pests, maternal history of asthma (for asthma) Season: Spring and fall **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.2	**Pollutant:** PM_{10} **Averaging Time:** 9 weeks **Mean (SD):** Study Avg 30 **Monitoring Stations**: 10 **Copollutant (correlation):** r2 is approximately 0.9 for all copollutants–Black Carbon (BC), $PM_{2.5}$, NO_X, NO_2, NO (NO_X–NO_2)	**PM Increment:** 1.4 (IQR) **OR Estimate [Lower CI, Upper CI]:** Bronchitis All subjects: 1.03 [0.99, 1.07] LTR subjects: 1.02 [0.98, 1.07] LTR females: 1.04 [1.01, 1.09] LTR males: 1.01 [0.95, 1.06] Asthma All subjects: 1.02 [0.96, 1.09] LTR subjects: 1.04 [0.97, 1.12] LTR females: 1.09 [0.92, 1.29] LTR males: 1.02 [0.94, 1.10] Asthma excluding outlier school having a larger proportion of Hispanics All subjects: 1.06 [0.97, 1.16] LTR subjects: 1.08 [0.98, 1.19] LTR females: 1.09 [0.96, 1.24] LTR males: 1.08 [0.97, 1.19]
Reference: Kumar et al. (2004) **Period of Study:** 1999-2001 **Location:** Mandi Gobindgarh and Morinda, Punjab State, northern India	**Outcome:** Chronic respiratory sysmptoms & Spirometric ventilatory defect **Age Groups:** >15 yrs **Study Design:** Cross-sectional **N:** 3603 individuals **Statistical Analyses:** Logistic regression **Covariates:** Age, gender, migration, SES, smoking, type of cooking fuel use **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Mean (SD):** Study town 112.8 (17.9) Reference town 75.8 (2.9)	**PM_{10} Increment:** Low vs. High OR (Lower CI, Upper CI); p-value Chronic respiratory sysmptoms Low 1.00 (ref) High 1.5 (1.2, 1.8); <0.001 Spirometric ventilatory defect Low 1.00 (ref) High 2.4 (2.0-2.9); <0.001
Reference: Leonardi et al. (2000) **Period of Study:** 1996 **Location:** 17 cities of Central Europe (Bulgaria, Czech Republic, Hungary , Poland, Romania, Slovakia)	**Outcome:** Immune biomarkers **Age Groups:** 9-11 **Study Design:** Cross-sectional **N:** 366 school children **Statistical Analyses:** Linear regression **Covariates:** Age, gender, parental smoking, laboratory of analysis, recent respiratory illness **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** PM_{10} **Averaging Time:** annual PM_{10} **Mean (SD):** PM_{10}: 65 (14) **Range (Min, Max):** PM_{10}: (41, 96) 5th, median, & 95th percentile PM_{10}: 41, 63, 90	**% Change (Lower CI, Upper CI); p-value** **PM_{10}** Neutrophils -5 (-33, 36); >.20 Total lymphocytes 20 (-6, 54);.150 B lymphocytes 42 (-3, 107);.067 Total T lymphocytes 30 (-2, 73);.072 CD4+ 28 (-10, 82);.177 CD8+ 29 (-5, 75);.097 CD4/CD8 7 (-20, 43); >.20 NK 33 (-10, 97);.157 Total IgG 11 (-10, 38); >.20 Total IgM 5 (-21, 39); >.20 Total IgA11 (-16, 46); >.20 Total IgE -8 (-62, 123); >.20
Reference: Lubinski, et al. (2005) **Period of Study:** 1993-1997 **Location:** Poland	**Outcome:** Pulmonary function TLC: total lung capacity ITGV: interthoracic gas volume ITGV%TLC: ITGV percent total lung capacity Raw: airway resistance FVC: forced vital capacity FEV_1: forced expiratory volume, 1 second FEV_1%FVC: FEV_1 percent forced vital capacity PEF: peak expiratory flow FEF_{50}: forced expiratory flow **Age Groups:** 18-23 males, healthy **Study Design:** ecological cross-sectional study **N:** 1278 subjects **Statistical Analyses:** Multiple linear regression, ANOVA **Covariates:** report unclear on whether or not there was covariate control, but may include NO_2 and SO_2 **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 12 mo **Mean (SD):** A: Highest Pollution Region Katowice 67-125 Krakow 41-49 B: Moderate Pollution Region Bielsko-Biala 29-48 Opole 18-45 Lodz 23-38 Warsaw 35-45 Wroclaw 28-76 Zagan 5-35 C: Lowest Pollution Region Gizycko 5-18 Hel 12-18 Ostroda 23-33 Swinoujscie 7-16 Ustka 12-26 **Copollutant:** NO_2, SO_2	**PM Increment:** 1 µg/m³ Slope, multiple regression TLC PM_{10}: -0.05 +SO_2: 0.03 +NO_2: -0.06 ITGV PM_{10}: 0.01 +SO_2: -0.07 +NO_2: -0.07 ITGV%TLC PM_{10}: -0.06 +SO_2: 0.08 +NO_2: 0.00 Raw PM_{10}: 0.075 +SO_2: -0.08 +NO_2: 0.127 FVC PM_{10}: 0.045 +SO_2: 0.045 +NO_2: -0.14 FEV_1 PM_{10}: 0.031 +SO_2: -0.08 +NO_2: -0.12 FEV_1%FVC PM_{10}: 0.00 +SO_2: -0.14 +NO_2: -0.048 PEF PM_{10}: -0.18 +SO_2: 0.056 +NO_2: -0.09 FEF50 PM_{10}: 0.031 +SO_2: -0.11 +NO_2: -0.04

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: McConnell et al. (1999b) **Period of Study:** 1993 **Location:** Southern California	**Outcome:** Bronchitis, chronic cough, phlegm **Age Groups:** Children: 4th, 7th, & 10th graders **Study Design:** Cross-sectional **N:** 3676 people **Statistical Analyses:** Logistic regression **Covariates:** Age, sex, race, grade, health insurance **Dose-response Investigated?** Yes	**Pollutant:** PM_{10} **Averaging Time:** yearly avg 24 h PM_{10} **Mean (SD):** 34.8 **Range (Min, Max):** 13.0, 70.7 **Copollutant (correlation):** NO_2; r = 0.74 O_3; r = 0.32 Acid; r = 0.54 $PM_{2.5}$; r = 0.90 NO_2; r = 0.83 O_3; r = 0.50 Acid; r = 0.71	**PM_{10} Increment:** 19 µg/m^3 Children w/ asthma Bronchitis: 1.4 (1.1,1.8) Phlegm: 2.1 (1.4, 3.3) Cough: 1.1 (0.8, 1.7) Children w/ wheeze, no asthma Bronchitis: 0.9 (0.7, 1.3) Phlegm: 0.9 (0.6, 1.4) Cough: 1.2 (0.9, 1.8) Children w/ no wheeze, no asthma Bronchitis: 0.7 (0.4, 1.0) Phlegm: 0.8 (0.6, 1.3) Cough: 0.9 (0.7, 1.2)
Reference: McConnell et al. (2003) **Period of Study:** 1993-99 **Location:** 12 Southern CA communities	**Outcome:** bronchitis symptoms **Age Groups:** 9-19 **Study Design:** communities selected on basis of historic levels of criteria pollutants and low residential mobility. **N:** 475 children **Statistical Analyses:** 3 stage regression combined to give a logistic mixed effects model **Covariates:** sex, ethnicity, allergies history, asthma history, SES, insurance status, current wheeze, current exposure to ETS, personal smoking status, participation in team sports, in utero tobacco exposure through maternal smoking, family history of asthma, amount of time routinely spent outside by child during 2-6 pm. **Dose-response Investigated?** No **Statistical Package:** SAS Glimmix macro	**Pollutant:** PM_{10} **Averaging Time:** 4 year averages **Mean (SD):**.30.8(13.4) µg/m^3 **Range (Min, Max):** 15.7-63.5 PM Component: particulate organic carbon and elemental carbon **Copollutant (correlation):** $PM_{2.5}$: r = 0.79 $PM_{10-2.5}$: r = 0.79 Inorganic acid: r = 0.72 Organic Acid: r = 0.59 Elemental carbon: r = 0.71 Organic Carbon: r = 0.70 NO_2: r = 0.20 O_3: r = 0.64	**PM Increment:** Between community range 47.8 µg/m^3 Between community unit 1 µg/m^3 Within community 1 µg/m^3 OR Estimate [Lower CI, Upper CI] Between community per range 1.72(0.93-3.20)| Between Community per unit 1.01(1.00-1.02)| Within community per unit 1.04(0.99-1.10)
Reference: McConnell, et al. (2006) **Period of Study:** 1996-1999 **Location:** 12 Southern California communities	**Outcome:** Prevalence of bronchitic symptoms (yearly). **Age Groups:** 10-15-years-old **Study Design:** longitudinal cohort **N:** 475 asthmatic children **Statistical Analyses:** Multilevel logistic mixed effects models. **Covariates:** age, second-hand smoke; personal smoking history; sex, race. **Dose-response Investigated?** No **Statistical Package:** SAS with GLIMMIX macro	**Pollutant:** PM_{10} **Averaging Time:** 365 days **Percentiles:** Community by year (n = 48 = 12 communities · 4 years) 25th: NR 50th(Median): 3.4 75th: NR **Range (Min, Max):** Community by year (n = 48 = 12 communities · 4 years): (0.89, 8.7) **Monitoring Stations**: 12 **Copollutant:** O_3 NO_2 EC OC Acid vapor (acetic and formic acid)	**PM Increment:** 6.1 µg/m^3 OR Estimate [Lower CI, Upper CI] PM_{10} Dog (n = 292): 1.60 [1.12: 2.30] No dog (n = 183): 0.89 [0.57: 1.39] PM_{10}*Dog interaction p-value: 0.02 Cat (n = 202): 1.47 [0.96: 2.24] No Cat (n = 273): 1.20 [0.83: 1.73] PM_{10}*Cat interaction p-value: 0.41 Neither pet (n = 112): 0.91 [0.53: 1.56] Cat only (n = 71): 0.84 [0.42: 1.66] Dog only (n = 161): 1.41 [0.91: 2.19] Both pets (n = 131): 1.89 [1.15: 3.10] Results suggest that dog ownership, a source of residential exposure to endotoxin, may worsen the severity of respiratory symptoms from exposure to air pollutants in asthmatic children.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Meng et al. (2007) **Period of Study:** November 2000 and September 2001 (collection of health data) **Location:** Los Angeles and San Diego counties	**Outcome:** Poorly controlled asthma vs. controlled asthma **Age Groups:** 18-64, 65+ **Study Design:** Long-term exposure study; comparison of cases and controls **N:** 1,609 adults (represented individuals age 18+ who reported ever having been diagnosed as having asthma by a physician and had their address successfully geocoded) **Statistical Analyses:** Logistic regression to evaluate associations between TD (traffic density) and annual avg air pollution concentrations and poorly controlled asthma. Used sample weights that adjusted for unequal probabilities of selection into the CHIS sample. **Covariates:** Age, sex, race/ethnicity, family federal poverty level, county, insurance status, delay in care for asthma, taking medications, smoking behavior, self-reported health status, employment, physical activity **Dose-response Investigated?** yes	**Pollutant:** PM_{10} **Averaging Time:** 24 over 1 year **Copollutant (correlation):** O_3: r = -0.72 NO_2: r = 0.83 $PM_{2.5}$: r = 0.84 CO: r = 0.42 TD: r = 0.14	**PM Increment:** Continuous data: per 10 µg/m^3 OR Estimate [Lower CI, Upper CI]; lag: All Adults: 1.08 [0.82, 1.43] Non-Elederly Adults: 1.14 [0.84, 1.55] Elderly: 0.84 [0.41, 1.73] Women: 1.38 [0.99, 1.94]
Reference: Millstein et al. (2004) **Period of Study:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 Data were taken from the Children's Health Study **Location:** Alpine, Atascadero, Lake Arrowhead, Lake Elsinore, Lancaster, Lompoc, Long Beach, Mira Loma, Riverside, San Dimas, Santa Maria, and Upland, CA	**Outcome:** Wheezing & asthma medication use (ICD9 NR) **Age Groups:** 4th grade students, mostly 9 yrs at the time of the study **Study Design:** Cohort Study, stratified into 2 seasonal groups/ **N:** 2081 enrolled, 2034 provided parent-completed questionnaire. **Statistical Analyses:** Multilevel, mixed-effects logistic model. **Covariates:** Contagious respiratory disease, ambient airborne pollen and other allergens, temperature, sex, age race, allergies, pet cats, carpet in home, environmental tobacco smoke, heating fuel, heating system, water damage in home, education level of questionnaire signer, physician diagnosed asthma. **Season:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 **Statistical Package:** GLIMMIX SAS 8.00 macro for generalized linear mixed models. **Lags Considered:** 14	**Pollutant:** PM_{10} **Averaging Time:** Monthly means for PM_{10}. **PM Component:** Nitric acid, formic acid, acetic acid **Monitoring Stations**: 1 central location in each community **Copollutant (correlation):** O_3: r = 0.76 NO_2: r = 0.39 $PM_{2.5}$: r = 0.91	**PM Increment:** IQR 13.39 µg/m^3 Odds Ratio [lower CI, Upper CI] Annual PM_{10}: 0.93 [0.67, 1.27] March-August PM_{10}: 0.91 [0.46, 1.80] Sep-Feb PM_{10}: 0.65 [0.40, 1.06]
Reference: Oftedal et al. (2008) **Period of Study:** 2001-2002 **Location:** Oslo, Norway	**Outcome:** Lung function (PEF, $FEF_{25\%}$, $FEF_{50\%}$, FEV_1, FVC) **Age Groups:** 9-10 yrs **Study Design:** Cross-sectional **N:** 1847 children **Statistical Analyses:** Linear regression **Covariates:** Height, age, BMI, birth weight, temperature, maternal smoking, sex **Dose-response Investigated?** Yes **Statistical Package:** SPSS, STATA, S-Plus **Lags Considered:** 1-3	**Pollutant:** PM_{10} **IQR:** PM_{10} in 1st yr of life: 10.3 PM_{10} lifetime: 5.8	**PM Increment:** Per IQR β (Lower CI, Upper CI) PM_{10} in 1st yr of life PEF -72.5 (-122.3 to -22.7) $FEF_{25\%}$ -77.4 (-133.4 to -21.4) $FEF_{50\%}$ -53.9 (-102.6. to -5.2) FEV_1 -6.7 (-24.1, 10.7) FVC 0.5 (-18.5, 19.6) PM_{10} lifetime exposure PEF -66.4 (-109.5 to -23.3) $FEF_{25\%}$ -61.5 (-110.0 to -13.1) $FEF_{50\%}$ -45.6 (-87.7 to -3.5) FEV_1 -7.3 (-22.4, 7.7) FVC -2.1 (-18.6, 14.4)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Penard-Morand et al. (2005) **Period of Study:** 03/1999 -- 10/2000 Mean concentrations of NO_2, SO_2, PM_{10}, and O_3 were taken from 01/01/1998 to 12/31/2000 **Location:** 6 French cities: Bordeaux, Clermont-Ferrand, Creteil, Marseille, Strasbourg, Reims.	**Outcome:** Flexural dermatitis Asthma (493) Rhinoconjunctivitis Atopic dermatitis Wheeze Allergic rhinitis Atopy EIB (exercise-induced bronchial reactivity) **Age Groups:** 9-11 years **Study Design:** Cross-sectional **N:** 9615 Children (6672 complete examination and questionnaire info) **Statistical Analyses:** Logistic regression Marginal Model (GENMOD) **Covariates:** Age, Sex, Family history of allergy, Passive smoking Parental education **Season:** All; Excluding end of spring and during summer for clinical examinations **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 3 years **Mean (SD):** Low concentrations: 26.9 High Concentrations: 23.8 **Range (Min, Max):** Low concentrations: 10-20 High concentrations: 21.5-29.5 **Copollutant (correlation):** NO_2: r =.46 SO_2: r =.76 O_3: r = -.02 **Monitoring Stations**: 16	**PM Increment:** 10 µg/m³ (IQR) OR Estimate [Lower CI, Upper CI]: EIB (during exam): 1.43 (1.02-2.01) Flexural dermatitis (during exam): 0.79 (0.59-1.07) Wheeze (past year): 1.05 (0.72-1.54) Asthma (past year): 1.23 (0.77-1.95) Rhinoconjunctivitis (past year): 1.17 (0.86-1.59) Atopic dermatitis (past year): 1.28 (0.96-1.71) Asthma (lifetime): 1.32 (0.96-1.81) Allergic rhinitis (lifetime): 1.32 (1.04-1.68) Atopic dermatitis (lifetime): 1.09 (0.88-1.36) Atopy (lifetime): 0.98(0.80-1.22) Pollen: 1.14 (0.85-1.53) Indoor: 0.91 (0.72-1.15) Moulds: 1.00 (0.53-1.88) Highest correlated pollutant adjustments: EIB (during exam): 1.16 (0.72-1.85) Flexural dermatitis (during exam): 0.93 0(.60-1.43) Wheeze (past year): 1.31 (0.71-2.36) Asthma (past year): 1.25 (0.66-2.37) Rhinoconjunctivitis (past year): 1.22 (0.98-1.68) Atopic dermatitis (past year): 1.63 (1.07-2.49) Asthma (lifetime): 1.11 (0.70-1.74) Allergic rhinitis (lifetime): 1.19 (0.94-1.59) Atopic dermatitis (lifetime): 1.47 (1.07-2.00) Atopy (lifetime): 0.93(0.69-1.26) Pollen: 1.30 (0.98-1.57) Indoor:.83 (0.63-1.12) Moulds: 1.62 (0.64-4.09)
Reference: Peters et al., (1999) **Period of Study:** 1986-1990, 1994 **Location:** Southern California	**Outcome:** Asthma, cough, bronchitis, wheeze **Age Groups:** 4th, 7th, & 10th graders **Study Design:** cohort **N:** 3676 children **Statistical Analyses:** Stepwise logistic regression **Covariates:** Community, grade, race, sex, height, BMI, asthma in parents, hay fever, health insurance, plants in home, mildew in home, passive smoke exposure, pest infestation, carpet, vitamin supplements, active smoking, pets, gas stove, air conditioner **Dose-response Investigated?** Yes	**Pollutant:** PM_{10} **Averaging Time:** 24 h PM_{10} averaged over 1994 Mean based on data collected during 1986-1990, 1994: Alpine 37.4, 21.3 Atascadero 28.0, 20.7 Lake Elsinore 59.5, 34.7 Lake Gregory 38.3, 24.2 Lancaster 47.0, 33.6 Lompoc 30.0, 13.0 Long Beach 49.5, 38.8 Mira Loma 84.9, 70.7 Riverside 84.9, 45.2 San Dimas 67.0, 36.7 Santa Maria 28.0, 29.2 Upland 75.6, 49.0	**PM Increment:** 25 µg/m³ OR (Lower CI, Upper CI) for respiratory illness Based on 1986-1990 pollutant levels Ever asthma 0.93 (0.76, 1.13) Current asthma 1.09 (0.86, 1.37) Bronchitis 0.94 (0.74, 1.19) Cough 1.06 (0.93, 1.21) Wheeze 1.05 (0.89, 1.25) Based on 1994 pollutant levels Ever asthma 0.87 (0.67, 1.14) Current asthma 1.11 (0.81, 1.54) Bronchitis 0.90 (0.65, 1.26) Cough 1.14 (0.96, 1.35) Wheeze 1.01 (0.79, 1.29)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Pierse, et al. (2006) **Period of Study:** 2 years (once in 1998 and once in 2001—surveys) **Location:** Leicestershire, UK	**Outcome:** Cough without a cold Night time cough Current wheeze **Age Groups:** 1-5 years **Study Design:** Cross-sectional (cohorts) **N:** 4400 children **Statistical Analyses:** Binomial generalized linear models (compared with likelihood ratio tests) Spatial variograms (due to the spatial concerns) **Covariates:** Age, Gender Mother/father has asthma Coal heating the home, Smoking by household member in the home, Either parent continued education past 16 years of age, Pre-term birth, Breast feeding, Gas cooking, Presence of pets, Number of cigarettes smoked by mother, Overcrowding, Single parenthood, Diet **Dose-response Investigated?** Yes (Fig. 2 shows evidence of dose-response effect based on surveys, states in discussion). **Statistical Package:** SAS 8.2; S-Plus 6.1	**Pollutant:** PM_{10} **Averaging Time:** annual PM_{10} **Mean (SD):** 1998: 1.47 2001: 1.33 Percentiles: 25th: 1998 (.73) and 2001 (.8) 75th: 1998 (1.93) and 2001 (1.84)	**PM Increment:** 10 µg/m^3 (IQR) **Unadjusted OR estimates [Lower CI, Upper CI]:** Cough without cold (1998): 1.22 (1.10 to 1.36) Cough without cold (2001): 1.46 (1.27 to 1.68) Night-time cough (1998): 1.11 (1.01 to 1.23) Night-time cough (2001): 1.25 (1.09 to 1.43) Current wheeze (1998): 0.99 (0.89 to 1.10) Current wheeze (2001): 1.09 (0.93 to 1.30) **Adjusted OR Estimate [Lower CI, Upper CI]:** Cough without cold (1998): 1.21 (1.07 to 1.38) Cough without cold (2001): 1.56 (1.32 to 1.84) Night-time cough (1998): 1.06 (0.94 to 1.19) Night-time cough (2001): 1.25 (1.06 to 1.47) Current wheeze (1998): 0.99 (0.88 to 1.12) Current wheeze (2001): 1.28 (1.04 to 1.58) **When the child was originally asymptomatic in 1998:** Unadjusted OR estimates [Lower CI, Upper CI]: Cough without cold (2001): 1.68 (1.39 to 2.03) Night-time cough (2001): 1.21 (1.00 to 1.46) Current wheeze (2001): 1.22 (0.92 to 1.62) Adjusted OR Estimate [Lower CI, Upper CI]: Cough without cold (2001): 1.62 (1.31 to 2.00) Night-time cough (2001): 1.19 (0.96 to 1.47) Current wheeze (2001): 1.42 (1.02 to 1.97)
Reference: Qian et al. (2005) **Period of Study:** 1990-1992 **Location:** Forsythe, NC; Minneapolis, MN; Jackson, MS.	**Outcome:** FVC, FEV_1, FEV_1/FVC **Age Groups:** middle aged (avg 56.8 years) **Study Design:** cross-sectional **N:** 10,240 people **Statistical Analyses:** regression equations, multiple linear regression analyses **Covariates:** Smoking status, recent use of respiratory medication, current respiratory symptoms, chronic lung diseases, field center **Dose-response Investigated?** No **Statistical Package:** SAS software, version 9.1	**Pollutant:** PM_{10} **Averaging Time:** Annual **Mean (SD):** 27.9 (2.8) Percentiles: 25th: 25.8 50th(Median): 27.5 75th: 30.2 Range (Maximum-Minimum): 12.2 **Monitoring Stations**: 3 (Minneapolis, MN); 5 (Jackson, MS); and 9 (Forsythe, NC) **Copollutant:** O_3	**PM Increment:** 2.8 µg/m^3 (1 SD) Effect Estimate: In Never Smokers FVC ß = -0.0108, SE = 0.0026, p =.0001 FEV_1 ß = -0.0082, SE = 0.0029, p =.0047 FEV_1/FVC ß = -0.0024, SE = 0.0023, p =.2787 Smoking status Current n = 2377, FVC = -1.96, FEV_1 = -2.23, FEV_1/FVC = -0.94 Former n = 3858, FVC = -1.25, FEV_1 = -1.10, FEV_1/FVC = -0.30 Never n = 4005, FVC = -1.12, FEV_1 = -0.63, FEV_1/FVC = 0.06 Recent Use of Respiratory Medication Yes n = 424, FVC = -2.65, FEV_1 = -3.89, FEV_1/FVC = -3.00 No n = 9816, FVC = -1.41, FEV_1 = -1.20, FEV_1/FVC = -0.24 Current Respiratory Symptoms Yes n = 4340, FVC = -1.68, FEV_1 = -1.70, FEV_1/FVC = -0.63 No n = 5900, FVC = -1.05, FEV_1 = -0.63, FEV_1/FVC = 0.05 Chronic Lung Diseases Yes n = 1374, FVC = -1.95, FEV_1 = -2.31, FEV_1/FVC = -1.18 No n = 8866, FVC = -1.35, FEV_1 = -1.10, FEV_1/FVC = -0.19 Field Center Forsythe, NC n = 3504, FVC = -0.03, FEV_1 = 0.05, FEV_1/FVC = -0.33 Minneapolis, MN n = 3793, FVC = 0.50, FEV_1 = 0.54, FEV_1/FVC = -0.30 Jackson, MS n = 2943, FVC = -0.01, FEV_1 = 0.17, FEV_1/FVC = -0.32

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Rios et al., (2004) **Period of Study:** 1998-2000 **Location:** the metropolitan area of Rio de Janiero, Brazil, Duque de Caxias (DC) and Seropedica (SR)	**Outcome:** wheezing, asthma, cough at night **Age Groups:** 13-14 yrs **Study Design:** cohort **N:** 4064 students **Statistical Analyses:** chi-squared **Covariates:** sex, type of school, time of residence, domestic smoking, residents per home **Dose-response Investigated?** Yes **Statistical Package:** EpiInfo	**Pollutant:** PM_{10} **Averaging Time:** weekly measurements used to create annual PM estimate **Mean (SD):** DC 1998: 147 1999: 115 2000: 110 Total: 124 SR 1998: 37 1999: 31 2000: 37 Total: 35 **Monitoring Stations**: NR	**PM Increment:** High vs. Low Global Cut-Off Score %, p-val: DC Male: 15.0 Female: 22.3, p<.05† Private School: 16.6 Public School: 19.4, p<.05* <5yr residence: 20.9 >5yr residence: 16.8 No domestic smoking exposure: 17.6 Domestic smoking exposure: 20.4, p<.05† <5 residents per home: 18.4 5+ residents per home: 19.5 SR Male: 12.3 Female: 19.7, p<.05† Private School: 28.3, p<.05*† Public School: 14.7 <5yr residence: 10.8 >5yr residence: 16.5 No domestic smoking exposure: 14.8 Domestic smoking exposure: 18.3 <5 residents per home: 15.6 5+ residents per home: 17.4 Notes: The Global Cut-off Score encompasses replies to the asthma component of ISAAC's written questionnaire that establishes a cut-off from which is defined the presence of asthma for the Brazilian population. *comparing the cities in the same controlled variable †comparing the controlled variable in the same city

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Rojas-Martinez et al. (2007) **Period of Study:** 1996-1999 **Location:** Mexico City, Mexico	**Outcome:** Lung function: FEV_1, FVC, $FEF_{25-75\%}$ **Age Groups:** Children 8 years old at time of cohort recruitment **Study Design:** school-based "dynamic" cohort study **N:** 3170 children; 14,545 observations **Statistical Analyses:** Three-level generalized linear mixed models with unstructured variance-covariance matrix **Covariates:** age, body mass index, height, height by age, weekday spent outdoors, environmental tobacco smoke, previous-day mean air pollutant concentration, time since first test **Dose-response Investigated?** No **Statistical Package:** SA	**Pollutant:** PM_{10} **Averaging Time:** 6-mo **Mean (SD):** 6-mo averaging SD: NR Mean: 75.6 **Percentiles:** 6-mo averaging 25th: 55.8 50th(Median): 67.5 75th: 92.2 **Monitoring Stations**: 5 sites for PM_{10}, 10 for other pollutants **Copollutant:** O_3 NO_2	**PM Increment:** IQR 6-LC: 36.4 Slope [Lower CI, Upper CI] Girls One-pollutant model FVC: -39 [-47: -31] FEV: -29 [-36: -21] $FEF_{25-75\%}$: -17 [-36: 1] FEV_1/FVC: 0.12 [0.07: 0.17] Two-pollutant model: PM_{10}, 6-LC & O_3 FVC: -30 [-39: -22] FEV: -24 [-31: -16] $FEF_{25-75\%}$: -9 [-26: 9] FEV_1/FVC: 0.10 [0.06: 0.15] PM_{10}, 6-LC & NO_2 FVC: -21 [-30: -13] FEV: -17 [-25: -8] $FEF_{25-75\%}$: -23 [-43: -4] FEV_1/FVC: 0.07 [0.02: 0.13] Multipollutant model: PM_{10}, 6-LC, O_3, & NO_2 FVC: -14 [-23: -5] FEV: -11 [-20: -3] $FEF_{25-75\%}$: -7 [-27: 12] FEV_1/FVC: 0.08 [0.03: 0.13] Boys One-pollutant model FVC: -33 [-41: -25] FEV: -27 [-34: -19] $FEF_{25-75\%}$: -18 [-34: -2] FEV_1/FVC: 0.04 [-0.01: 0.09] Two-pollutant model: PM_{10}, 6-LC & O_3 FVC: -28 [-36: -19] FEV: -22 [-30: -15] $FEF_{25-75\%}$: -10 [-27: 7] FEV_1/FVC: 0.04 [-0.01: 0.09] FEV_1/FVC: 0.24 [0.13: 0.34] PM_{10}, 6-LC & NO_2 FVC: -16 [-26: -7] FEV: -19 [-27: -10] $FEF_{25-75\%}$: -26 [-44: -9] FEV_1/FVC: 0.005 [-0.06: 0.05] Multipollutant model PM_{10}, 6-LC, O_3, & NO_2 FVC: -12 [-22: -3] FEV: -15 [-23: -6] $FEF_{25-75\%}$: -12 [-30: 6] FEV_1/FVC: -0.002 [-0.06: 0.05]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Schikowski et al. (2005) **Period of Study:** 1985-1994 **Location:** Rhine-Ruhr Basin of Germany [Dortmund (1985, 1990), Duisburg (1990), Gelsenkirchen (1986, 1990), and Herne (1986)]	**Outcome:** Respiratory symptoms & pulmonary function **Age Groups:** age 54-55 **Study Design:** Cross-sectional **N:** 4757 women **Statistical Analyses:** Linear & Logistic regressions, including random effects model **Covariates:** age, smoking, SES, occupational exposure, form of heating, BMI, height **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** NR	**Pollutant:** PM_{10} **Averaging Time:** NR Min, P25, Median, Mean, P75, Max Annual Mean 35, 40, 43, 44, 47, 53 Five year Mean 39, 43, 47, 48, 53, 56 **Monitoring Stations**: 7 **Copollutant (correlation):** NR	**PM Increment:** 7 µg/m^3 OR (Lower CI, Upper CI) for asthma symptoms Annual means Chronic bronchitis 1.00 (0.85, 1.18) Chrongic cough 1.03 (0.87, 1.23) Frequent cough 1.01 (0.93, 1.10) COPD 1.37 (0.98, 1.92); p<0.1 FEV_1 0.953 (0.916, 0.989); p<0.1 FVC 0.966 (0.940, 0.992); p<0.1 FEV_1/FVC 0.989 (0.978, 1.000); p<0.1 Five year means Chronic bronchitis 1.13 (0.95, 1.34) Chrongic cough 1.11 (0.93, 1.31) Frequent cough 1.05 (0.94, 1.17) COPD 1.33 (1.03, 1.72); p<0.1 FEV_1 0.949 (0.923, 0.975); p<0.05 FVC 0.963 (0.945, 0.982); p<0.05 FEV_1/FVC 0.989 (0.980, 0.997); p<0.1
Reference: Sharma et al. (2004) **Period of Study:** 11/2002–4/2003 **Location:** 3 sections in Kanpur City, India: 1) Indian Institute of Technology Kanpur (IITK); 2) Vikas Nagar (VN); 3) Juhilal Colony (JC)	**Outcome:** Lung function **Age Groups:** 20–55 years **Study Design:** Cohort **N:** 91 people **Statistical Analyses:** Linear regression **Covariates:** NR **Season:** Fall, Winter, spring **Dose-response Investigated?** No **Statistical Package:** Microsoft Excel **Lags Considered:** 1d lag & 5d mov avg	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** IITK 184 (40) VN 295 (58) JC 293 (90) **PM Component:** Lead Nickel Cadmium Chromium Iron Zinc Benzene soluble fraction (includes polycyclic aromatic hydrocarbons [PAHs]) **Copollutant (correlation):** ΔPEF = mean daily deviations in PEF PM_{10}-ΔPEF: (-0.52) PM_{10}-$PM_{2.5}$: (0.67) PM_{10}-PM_{10} (1-day lag): (0.45) PM_{10}-$PM_{2.5}$ (1-day lag): (0.46)	**PM Increment:** 1 µg/m^3 ΔPEF (difference or change in peak expiratory flow) -0.0318 L/min
Reference: Tager et al. (2005) **Period of Study:** 4/2000-6/2000, 2/2001–6/2001, 2/2002–6/2002 **Location:** Los Angeles, California San Francisco, California	**Outcome:** Lung Function (FEV_1, FVC, PEFR, FEF75, $FEF_{25–75}$, $FEF_{25–75}$/FVC ratio) **Age Groups:** 16-21+ y/o College Freshman **Study Design:** Retrospective cohort **N:** 255 students 108 Men (M) 147 Women (W) **Statistical Analyses:** Multivariate Linear Regression **Covariates:** Sex, height, weight, area of residence, age, race, ETS exposure, respiratory disease history **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** Cumulative lifetime exposure Median: Prior to 1987: M: 73; W: 71 1987 and later: M: 36; W: 34 Lifetime: M: 48; W: 45 **Range (Min, Max):** Prior to 1987: M: 34, 117; W: 31, 124 1987 and later: M: 18, 68; W: 20, 61 Lifetime: M: 21, 80; W: 18, 71 **Monitoring Stations**: Between 1 and 3 **Copollutant (correlation):** O_3 prior to 1987: r = 0.68 O_3 1987 and later: r = 0.81 O_3–Lifetime: r = 0.57	**PM Increment:** 1 µg/m^3 Parameter Estimates (SD) (Lifetime PM_{10}, Interaction PM_{10} $FEF_{25–75}$/FVC) LnFEF75: M: -0.009 (0.0009), 0.009 (0.007) W: -0.010 (0.0007), 0.008 (0.0005)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Tamura et a. (2003) **Period of Study:** 1998-1999 **Location:** Bangkok, Thailand	**Outcome:** non-specific respiratory disease (Chronic bronchitis, acute bronchitis, bronchial asthma, dyspnea and wheezing) **Age Groups:** adults **Study Design:** Cross-sectional **N:** 1603 policemen **Statistical Analyses:** Multiple logistic regression **Covariates:** age, smoking status **Dose-response Investigated?** Yes **Statistical Package:** SPSS	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Heavily Polluted 80-190 Moderately Polluted 60-69 Control 59 **Monitoring Stations**: 13	**PM Increment:** Heavily Polluted vs. Moderately Polluted vs. Control Number and Prevalence (%) of respiratory disease among heavily polluted, moderately polluted, and control areas. **Heavily Polluted** Chronic bronchitis 16 (3.0) Acute bronchitis 19 (3.5) Bronchial asthma 5 (0.9) Dyspnea & wheezing 49 (9.2) Any 1 of above 69 (13.0) Persistent cough 11 (2.1) Persistent phlegm 27 (1.3) Cough & phlegm 6 (1.1) **Moderately Polluted** Chronic bronchitis 8 (2.4) Acute bronchitis 12 (9.0) Bronchial asthma 2 (0.6) Dyspnea & wheezing 23 (6.8) Any 1 of above 37 (10.9) Persistent cough 1 (0.3) Persistent phlegm 11 (3.3)| Cough & phlegm 1 (0.3) Control Chronic bronchitis 6 (1.9) Acute bronchitis 11 (3.3) Bronchial asthma 0 (0.0) Dyspnea & wheezing 23 (7.2) Any 1 of above 31 (9.4) Persistent cough 1 (0.3) Persistent phlegm 8 (2.4) Cough & phlegm 1 (0.3)
Reference: Wheeler and Ben-Schlomo (2005) **Period of Study:** 1995-1997 **Location:** England	**Outcome:** FEV_1 **Age Groups:** 16-79 yrs **Study Design:** Data from Health Survey for England were coupled geographically with air pollution measurements on a 1 km grid. **N:** 26,426 households with 39,251 adults **Statistical Analyses:** Logistic regression, least squares regression **Covariates:** Age, sex, height, body mass index, smoking status, household passive smoke exposure, inhaler use in the previous 24-hs, doctor diagnosis of asthma. **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** 1996 annual mean **Mean (SD):** 23.95 (3.58) **Range (Min, Max):** 17.87-43.37	β (95%CI) for Height-age standardized FEV_1 by ambient air quality index; p-value Male Good (ref) Poor -0.023 (-0.030 to -0.016); <0.001 Female Good (ref) Poor -0.019 (-0.026 to -0.013); <0.001

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Zhang et al., (2002) **Period of Study:** 1993-1996 **Location:** 4 Chinese cities (urban and suburban location in each city): Guangzhou, Wuhan, Lanzhou, Chongqing	**Outcome:** Interview-self reports of symptoms: Wheeze (ever wheezy when having a cold) Asthma (diagnosis by doctor) Bronchitis (diagnosis by doctor), Hospitalization due to respiratory disease (ever) Persistent cough (coughed for at least 1 month per year with or apart from colds) Persistent phlegm (brought up phlegm or mucus from the chest for at least 1 month per year with or apart from colds) **Age Groups:** Elementary school students; age range: 5.4–16.2 **Study Design:** Cross-sectional **N:** 7,557 returned questionnaires 7,392 included in first stage of analysis **Statistical Analyses:** 2-stage regression approach: Calculated odds ratios and 95% CIs of respiratory outcomes and covariates Second stage consisted of variance-weighted linear regressions that examined associations between district-specific adjusted prevalence rates and district-specific ambient levels of each pollutant. **Covariates:** Age, gender, breast-fed, house type, number of rooms, sleeping in own or shared room, sleeping in own or shared bed, home coal use, ventilation device used, homes smokiness during cooking, eye irritation during cooking, parental smoking, mother's education level, mother's occupation, father's occupation, questionnaire respondent, year of questionnaire administration, season of questionnaire administration, parental asthma prevalence	**Pollutant:** PM_{10} **Averaging Time:** 2 years **Mean (SD):** 151 (56) IQR: 87 **Range (Min, Max):** Gives range (max.–min.): 80 **Monitoring Stations**: 2 types: municipal monitoring stations over a period of 4 years (1993-1996); schoolyards of participating children over a period of 2 years (1995–1996)	**PM Increment:** Interquartile range corresponded to 1 unit of change. RR Estimate [Lower CI, Upper CI]; lag: Association between persistent phlegm and PM_{10}: 3.21 (1.55, 6.67); $p<0.05$ - Between and within city modeled ORs, scaled to interquartile range of concentrations for each pollutant. No associations between any type of respiratory outcome and PM_{10} When scaled to an increment of 50 $\mu g/m^3$ of PM_{10}, ORs were: Wheeze: 1.07 Asthma: 1.18 Bronchitis: 1.53 Hospitalization: 1.17 Persistent cough: 1.20 Persistent phlegm: 1.95

Table E-24. Long-term exposure to $PM_{10-2.5}$ and respiratory morbidity outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Dales et al., (2008) **Period of Study:** **Location:** Windsor, ON	**Outcome:** Pulmonary function and inflammation Age Groups: grades 4-6 **Study Design:** cross-sectional prevalence design **Statistical Analyses:** multivariate linear regression **Covariates:** Ethnic background, smokers at home, pets at home, acute respiratory illness, medication use	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** Annual Mean: 7.25 5th: 6.02 95th: 8.23 Copollutant**:** SO_2 NO_2	**Increment:** Tertiles of exposure FEV_1: <7.04: 2.18 ± 0.01 7.04-7.53: 2.19 ± 0.02 >7.53: 2.14 ± 0.01 FVC: <7.04: 2.52 ± 0.02 7.04-7.53: 2.53 ± 0.02 >7.53: 2.48 ± 0.02 eNO: <7.04: 15.48 ± 0.63 7.04-7.53: 16.73 ± 0.76 >7.53: 16.59 ± 0.79

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Gauderman et al. (2000b) **Period of Study:** 1993-1997 **Location:** Southern California	**Outcome:** FVC, FEV_1, MMEF, FEF_{75} **Age Groups:** fourth, seventh, or tenth graders **Study Design:** cohort **N:** 3035 subjects **Statistical Analyses:** Linear regression **Covariates:** Height, weight, BMI, asthma, smoking, exercise, room temperature, barometric pressure **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 24 h avg PM_{10} & annual avg of 2-week avg $PM_{2.5}$ **Mean (SD):** $PM_{10-2.5}$ 25.6 **Copollutant (correlation):** O_3; r = -0.29 NO_2 r = 0.44 Inorg. Acid r = 0.43	**Increment:** 25.6 µg/m^3 % Change (Lower CI, Upper CI) $PM_{10-2.5}$-4th grade FVC -0.57 (-1.20 to -0.06) FEV_1 -0.90 (-1.71 to -0.09) MMEF -1.37 (-2.57 to -0.15) FEF_{75} -1.62 (-3.24, 0.04) $PM_{10-2.5}$-7th grade FVC -0.35 (-1.02, 0.31) FEV_1 -0.49 (-1.21, 0.24) MMEF -0.64 (-2.83, 1.60) FEF_{75} -0.74 (-2.65, 1.20) $PM_{10-2.5}$-10th grade FVC -0.17 (-1.32, 0.99) FEV_1 -0.68 (-2.15, 0.81) MMEF -1.41 (-5.85, 3.25) FEF_{75} -2.32 (-6.60, 2.17)
Reference: Gauderman et al. (2002b) **Period of Study:** 1996–2000 **Location:** Southern California	**Outcome:** Lung function development: FEV_1, maximal mid-expiratory flow (MMEF) **Age Groups:** Fourth grade children (avg age = 9.9 yrs) **Study Design:** Cohort study **N:** 1678 children, 12 communities **Statistical Analyses:** Mixed model linear regression **Covariates:** Height, BMI, doctor-diagnosed asthma and cigarette smoking in previous year, respiratory illness and exercise on day of test, interaction of each of these variables with sex, barometric pressure, temperature at test time, indicator variables for field technician and spirometer **Dose-response Investigated?** Yes **Statistical Package:** SAS (10)	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** Annual 24 h averages **Mean (SD):** The avg levels were presented in an online data supplement (Figure E1) **Monitoring Stations**: 12 **Copollutant (correlation):** O_3 (10 AM to 6 PM) r = 0.10 O_3 r = -0.31 NO_2 r = 0.46 Acid vapor r = 0.63 PM_{10} r = 0.95 $PM_{10-2.5}$ r = 0.81 EC r = 0.71 OC r = 0.96	**PM Increment:** 29.1 µg/m^3 Association Estimate: $PM_{10-2.5}$ was not correlated with any of the pulmonary function tests that were analyzed
Reference: Leonardi et al. (2000) **Period of Study:** 1996 **Location:** 17 cities of Central Europe (Bulgaria, Czech Republic, Hungary , Poland, Romania, Slovakia)	**Outcome:** Immune biomarkers **Age Groups:** 9-11 **Study Design:** Cross-sectional **N:** 366 school children **Statistical Analyses:** Linear regression **Covariates:** Age, gender, parental smoking, laboratory of analysis, recent respiratory illness **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** subtracting $PM_{2.5}$ from PM_{10} provides avg $PM_{10-2.5}$ **Mean (SD):** $PM_{10-2.5}$: 20 (5) **Range (Min, Max):** $PM_{10-2.5}$: (12, 38) 5th, median, & 95th percentile $PM_{10-2.5}$: 12, 19, 29	**% Change (Lower CI, Upper CI); p-value** $PM_{10-2.5}$ Neutrophils 1 (-27, 38); >.20 Total lymphocytes 8 (-15, 38); >.20 B lymphocytes 22 (-16, 76); >.20 Total T lymphocytes 2 (-25, 37); >.20 CD4+ -1 (-30, 41); >.20 CD8+ 3 (-25, 41); >.20 CD4/CD8 0 (-23, 30); >.20 NK 1 (-33, 51); >.20 Total IgG -3 (-21, 18); >.20 Total IgM 19 (-9, 55); >.20 Total IgA 16 (-12, 52); >.20 Total IgE -29 (-70, 70); >.20

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: McConnell et al. (2003) **Period of Study:** 1993-99 **Location:** 12 Southern CA communities	**Outcome:** bronchitic symptoms **Age Groups:** 9-19 **Study Design:** communities selected on basis of historic levels of criteria pollutants and low residential mobility. **N:** 475 children **Statistical Analyses:** 3 stage regression combined to give a logistic mixed effects model **Covariates:** sex, ethnicity, allergies history, asthma history, SES, insurance status, current wheeze, current exposure to ETS, personal smoking status, participation in team sports, in utero tobacco exposure through maternal smoking, family history of asthma, amount of time routinely spent outside by child during 2-6 pm. **Dose-response Investigated?** No **Statistical Package:** SAS Glimmix macro	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 4 year avg **Mean (SD):** 17.0(6.4) **Range (Min, Max):** 10.2-35.0 **Copollutant (correlation):** $PM_{2.5}$: r = 0.24 PM_{10}: r = 0.79 Inorganic acid: r = 0.38 Organic Acid: r = 0.35 EC: r = 0.30 OC: r = 0.27 NO_2: r = -0.22 O_3: r = 0.29	**PM Increment:** Between community range 24.8 µg/m^3 Between community unit 1 µg/m^3 Within community 1 µg/m^3 OR Estimate [Lower CI, Upper CI] Between community per range 1.38(0.65-2.92) Between Community per unit 1.01(0.98-1.04) Within community per unit 1.02(0.95-1.10)
Reference: Millstein et al. (2004) **Period of Study:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 Data were taken from the Children's Health Study **Location:** Alpine, Atascadero, Lake Arrowhead, Lake Elsinore, Lancaster, Lompoc, Long Beach, Mira Loma, Riverside, San Dimas, Santa Maria, and Upland, CA	**Outcome:** Wheezing & asthma medication use **Age Groups:** 4th grade students, mostly 9 yrs at the time of the study **Study Design:** Cohort Study, stratified into 2 seasonal groups/ **N:** 2081 enrolled, 2034 provided parent-completed questionnaire. **Statistical Analyses:** Multilevel, mixed-effects logistic model. **Covariates:** Contagious respiratory disease, ambient airborne pollen and other allergens, temperature, sex, age race, allergies, pet cats, carpet in home, environmental tobacco smoke, heating fuel, heating system, water damage in home, education level of questionnaire signer, physician diagnosed asthma. **Season:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 **Statistical Package:** SAS 8.00 **Lags Considered:** 14	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** monthly **PM Component:** Nitric acid, formic acid, acetic acid **Monitoring Stations**: 1 central location in each community **Copollutant (correlation):** NO_2: r = 0.29 O_3: r = 0.77 $PM_{2.5}$: r = -0.08	**PM Increment:** IQR 11.44 µg/m^3 Odds Ratio [lower CI, Upper CI] Annual $PM_{10-2.5}$: 0.96 [0.74, 1.25] March-August $PM_{10-2.5}$: 0.93 [0.54, 1.59] Sep-Feb $PM_{10-2.5}$: 0.68 [0.46, 1.01]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Zhang et al. (2002) **Period of Study:** 1993-1996 **Location:** 4 Chinese cities (urban and suburban location in each city): Guangzhou, Wuhan, Lanzhou, Chongqing	**Outcome:** Interview-self reports of symptoms: Wheeze (ever wheezy when having a cold) Asthma (diagnosis by doctor) Bronchitis (diagnosis by doctor), Hospitalization due to respiratory disease (ever) Persistent cough (coughed for at least 1 month per year with or apart from colds) Persistent phlegm (brought up phlegm or mucus from the chest for at least 1 month per year with or apart from colds) **Age Groups:** Elementary school students; age range: 5.4–16.2 **Study Design:** Cross-sectional **N:** 7,557 returned questionnaires; 7,392 included in first stage of analysis **Statistical Analyses:** 2-stage regression approach: Calculated odds ratios and 95% CIs of respiratory outcomes and covariates Second stage consisted of variance-weighted linear regressions that examined associations between district-specific adjusted prevalence rates and district-specific ambient levels of each pollutant. **Covariates:** Age, gender, breast-fed, house type, number of rooms, sleeping in own or shared room, sleeping in own or shared bed, home coal use, ventilation device used, homes smokiness during cooking, eye irritation during cooking, parental smoking, mother's education level, mother's occupation, father's occupation, questionnaire respondent, year of questionnaire administration, season of questionnaire administration, parental asthma prevalence	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 2 years **Mean (SD):** 59 (28) **Percentiles:** 25th: NR 50th(Median): NR 75th: NR IQR: 42 **Range (Min, Max):** Gives range (max.–min.): 80 **Monitoring Stations**: 2 types: municipal monitoring stations over a period of 4 years (1993-1996); schoolyards of participating children over a period of 2 years (1995–1996)	**PM Increment:** Interquartile range corresponded to 1 unit of change. RR Estimate [Lower CI, Upper CI]; lag: Association between bronchitis and $PM_{10-2.5}$: 2.20 (1.14, 4.26); $p<0.05$ Association between persistent cough and $PM_{10-2.5}$: 1.46 (1.12, 1.90); $p<0.05$ Between and within city associations: Bronchitis: 3.18 (between city) Persistent phlegm (between city): 2.78 When scaled to an increment of 50 $\mu g/m^3$ of $PM_{10-2.5}$ associations (ORs) between respiratory outcome and $PM_{10-2.5}$ were: Wheeze: 1.14 Asthma: 1.34 Bronchitis: 2.56 Hospitalization: 1.58 Persistent cough: 1.57 Persistent phlegm: 3.45

Table E-25. Long-term exposure to $PM_{2.5}$ (including PM components/sources) and respiratory morbidity outcomes

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Annesi-Maesano et al. (2007) **Period of Study:** Mar 1999–Oct 2000 **Location:** France (Bordeaux, Clermont-Ferrand, Creteil, Marseille, Strasbourg,, & Reims)	**Outcome:** EIB, Fl. Atopic dermatitis, asthma, rhiniconjuctivitis, allergic rhinitis **Age Groups:** Children mean 10.4 ± 0.7 yrs **Study Design:** Semi-individual design **N:** 5338 **Statistical Analyses:** Logistic regression **Covariates:** Age, sex, family history of allergy, passive smoking **Season:** NR **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 5-day mean (Mon.-Fri.) over a 13-week to 24-week span Residential Proximity Level **Mean (SD):** Low conc: 8.7 High conc: 20.7 **Range (Min, Max):** Low conc: (1.6, 12.2) High conc: (12.5, 54.0) City Level **Mean (SD):** Low conc: 9.6 High conc: 23.0 **Range (Min, Max):** Low conc: (4.7, 12.7) High conc: (13.0, 54.5)	**PM Increment:** High vs. Low Allergic and respiratory morbidity OR Estimate (Lower CI, Upper CI) Proximity Level EIB (C) 1.35 (1.10, 1.67) Fl. Atopic dermatitis (C) 2.51 (2.06, 3.06) Asthma (P) 1.11 (0.88, 1.39) Atopic asthma (P) 1.43 (1.07, 1.91) Non-atopic asthma (P) 0.73 (0.49, 1.07) Rhiniconjunctivitis (P) 0.94 (0.77, 1.15) Atopic dermatitis (P) 1.05 (0.88, 1.27) Asthma (L) 1.00 (0.82, 1.22) Allergic Rhinitis (L) 1.09 (0.93, 1.27) Atopic dermatitis (L) 0.94 (0.82, 1.09) City Level EIB (C) 1.43 (1.15, 1.78) Fl. Atopic dermatitis (C) 2.06 (1.69, 2.51) Asthma (P) 1.31 (1.04, 1.66) Atopic asthma (P) 1.58 (1.17, 2.14) Non-atopic asthma (P) 1.00 (0.68, 1.49) Rhiniconjunctivitis (P) 0.98 (0.80, 1.20) Atopic dermatitis (P) 1.08 (0.90, 1.30) Asthma (L) 1.09 (0.89, 1.33) Allergic Rhinitis (L) 1.13 (0.97, 1.33) Atopic dermatitis (L) 0.95 (0.82, 1.09) Notes: C = Current; P = Past year; L = Lifetime Allergic sensitisation OR Estimate (Lower CI, Upper CI) Proximity Level All allergens 1.19 (1.04, 1.36) Indoor allergens 1.29 (1.11, 1.50) Outdoor allergens 1.02 (0.85, 1.23) Moulds 1.13 (0.78, 1.65) City Level All allergens 1.32 (1.15, 1.51) Indoor allergens 1.51 (1.29, 1.76) Outdoor allergens 1.06 (0.88, 1.28) Moulds 1.00 (0.69, 1.46)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Bennett et al. (2007) **Period of Study:** 1992-2005 **Location:** Melbourne, Australia	**Outcome:** Respiratory symptoms (from questionnaire) **Age Groups:** All ages, mean = 37.2 yrs **Study Design:** cohort **N:** 1446 **Statistical Analyses:** Logistic regression models **Covariates:** age, gender, use of ß2-agonists, use of inhaled corticosteroids, smoking, year of data collection, and avg daily exposure to $PM_{2.5}$ in the 12 months corresponding to the time frame of symptoms **Dose-response Investigated?** No **Statistical Package:** STATA, version 9	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 6.8 **Range (Min, Max):** (1.8-73.3) **Monitoring Stations**: up to 3	**PM Increment:** NR **Effect Estimate [Lower CI, Upper CI]:** Respiratory symptoms in last 12 months and exposure to ambient $PM_{2.5}$ over the same period Within-person (longitudinal) effects Wheeze: OR = 1.08 (0.79-1.48), p = 0.62 SOB on waking: OR = 1.34 (0.84-2.16), p = 0.22 Cough (AM): OR = 0.74 (0.47-1.15), p = 0.18 Phlegm (AM): OR = 1.55 (0.95-2.53), p = 0.08 Cough w/ phlegm (AM): OR = 1.28 (0.70-2.33), p = 0.42 Asthma attack: OR = 0.91 (0.55-1.49), p = 0.69 Between-person (cross-sectional) effects Wheeze: OR = 1.32 (0.82-2.10), p = 0.25 SOB on waking: OR = 1.29 (0.46-3.60), p = 0.63 Cough (AM): OR = 0.21 (0.07-0.62), p = 0.01 Phlegm (AM): OR = 0.49 (0.16-1.44), p = 0.19 Cough w/ phlegm (AM): OR = 0.28 (0.08-0.97), p = 0.05 Asthma attack: OR = 0.52 (0.17-1.59), p = 0.26

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Brauer, et al. (2007) **Period of Study:** 1999-2000 **Location:** The Netherlands	**Outcome:** Allergen sensitivity (any, indoor, outdoor, food, total) IgE>100 IU/mL Asthma (probable, MD-diagnosed, ever MD-diagnosed) Bronchitis (MD-diagnosed, ever MD-diagnosed) Dry cough at night Itchy rash Itchy rash/eczema Ear/Nose/Throat (ENT) infection Eczema, MD-diagnosed Eczema, ever MD-diagnosed Flu/serious cold, MD-diagnosed Wheeze (ever, early, early frequent, persistent) **Age Groups:** very young children (<4-years-old) enrolled prenatally **Study Design:** prospective birth cohort study **N:** ~4000 subjects **Statistical Analyses:** multiple logistic regression **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 12 months **Mean (SD):** SD: NR 16.9 **Percentiles:** 25th: 14.8 50th(Median): 17.3 75th: 18.1 **Range (Min, Max):** (13.5, 25.2) **Monitoring Stations**: 40 **Copollutant (correlation):** Soot: r = 0.97 NO_2: r = 0.93	**PM Increment:** IQR 3.3 μg/m^3 Notes: Traffic-related pollution ($PM_{2.5}$, soot, NO_2) was associated with respiratory infections, asthma, and allergic sensitization in children during the first four years of life. **Symptom At 4-Years-Old** Wheeze 4-years-old: 1.23 [1.00: 1.51] Early-life: 1.20 [0.99: 1.46] Asthma, MD-diagnosed 4-years-old: 1.15 [0.82: 1.62] Early-life: 1.32 [0.96: 1.83] Dry cough at night 4-years-old: 1.11 [0.94: 1.31] Early-life: 1.14 [0.98: 1.33] Bronchitis, MD-diagnosed 4-years-old: 0.88 [0.66: 1.18] Early-life: 0.86 [0.66: 1.11] ENT infection 4-years-old: 1.13 [0.98: 1.31] Early-life: 1.17 [1.02: 1.34] Flu/serious cold, MD-diagnosed 4-years-old: 1.21 [1.02: 1.42] Early-life: 1.25 [1.07: 1.46] Itchy rash 4-years-old: 0.96 [0.82: 1.11] Early-life: 0.98 [0.85: 1.14] Eczema, MD-diagnosed 4-years-old: 1.00 [0.88: 1.21] Early-life: 0.98 [0.82: 1.17] **Allergen Sensitivity At 4-Yr-Old** Allergen, any: 1.55 [1.13: 2.11] Allergen, indoor: 1.03 [0.69: 1.55] Allergen, outdoor: 0.93 [0.54: 1.58] Allergen, food: 1.75 [1.23: 2.47] Allergen, total IgE>100 IU/mL: 0.84 [0.59: 1.18] **Cumulative Allergy/Asthma Symptoms At 4-Years-Old** Wheeze, ever: 1.22 [1.06: 1.41] Asthma, ever MD-diagnosed: 1.32 [1.04: 1.69] Asthma, probable: 1.08 [0.90: 1.30] Wheeze, early: 1.16 [1.00: 1.34] Wheeze, persistent: 1.19 [0.96: 1.48] Wheeze, early frequent: 1.19 [0.96: 1.47] Bronchitis, ever MD-diagnosed: 0.96 [0.81: 1.13] Itchy rash/eczema: 0.99 [0.88: 1.13] Eczema, ever MD-diagnosed: 0.98 [0.85: 1.13]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Brauer, et al. (2007) **Period of Study:** 1999-2000 **Location:** The Netherlands	**Outcome:** Allergen sensitivity (any, indoor, outdoor, food, total) IgE>100 IU/mL Asthma (probable, MD-diagnosed, ever MD-diagnosed) Bronchitis (MD-diagnosed, ever MD-diagnosed) Dry cough at night Itchy rash Itchy rash/eczema Ear/Nose/Throat (ENT) infection Eczema, MD-diagnosed Eczema, ever MD-diagnosed Flu/serious cold, MD-diagnosed Wheeze (ever, early, early frequent, persistent) **Age Groups:** very young children (<4-years-old) enrolled prenatally **Study Design:** prospective birth cohort study **N:** ~4000 subjects **Statistical Analyses:** multiple logistic regression **Dose-response Investigated?** No	**Pollutant:** Soot (as $PM_{2.5}$ absorbance) **Averaging Time:** 12 months **Mean (SD):** 1.71 **Percentiles:** 25th: 1.33 50th(Median): 1.78 75th: 1.91 **Range (Min, Max):** (0.77, 3.68) **Unit (i.e. µg/m³)**:1E-5/m **Monitoring Stations**: 40 **Copollutant (correlation):** NO_2: r = 0.96 $PM_{2.5}$: r = 0.97	**PM Increment:** IQR 0.58 E-5/m Notes: Traffic-related pollution ($PM_{2.5}$, soot, NO_2) was associated with respiratory infections, asthma, and allergic sensitization in children during the first four years of life. **Symptom At 4-Years-Old** Wheeze 4-years-old: 1.18 [0.98: 1.41] Early-life: 1.18 [1.00: 1.40] Asthma, MD-diagnosed 4-years-old: 1.15 [0.85: 1.55] Early-life: 1.30 [0.98: 1.71] Dry cough at night 4-years-old: 1.13 [0.97: 1.30] Early-life: 1.14 [1.00: 1.31] Bronchitis, MD-diagnosed 4-years-old: 0.90 [0.69: 1.16] Early-life: 0.88 [0.69: 1.11] ENT infection 4-years-old: 1.15 [1.01: 1.31] Early-life: 1.16 [1.03: 1.31] Flu/serious cold, MD-diagnosed 4-years-old: 1.18 [1.02: 1.36] Early-life: 1.19 [1.04: 1.37] Itchy rash 4-years-old: 0.94 [0.82: 1.08] Early-life: 0.97 [0.85: 1.10] Eczema, MD-diagnosed 4-years-old: 0.99 [0.84: 1.17] Early-life: 0.97 [0.83: 1.14] **Allergen Sensitivity At 4-Yrs-Old** Allergen, any: 1.45 [1.11: 1.91] Allergen, indoor: 1.02 [0.71: 1.46] Allergen, outdoor: 0.95 [0.59: 1.52] Allergen, food: 1.64 [1.21: 2.23] Allergen, total IgE>100 IU/mL: 0.80 [0.59: 1.09] **Cumulative Allergy/Asthma Symptoms At 4-Years-Old** Wheeze, ever: 1.18 [1.04: 1.34] Asthma, ever MD-diagnosed: 1.26 [1.02: 1.56] Asthma, probable: 1.06 [0.90: 1.24] Wheeze, early: 1.11 [0.97: 1.26] Wheeze, persistent: 1.18 [0.98: 1.42] Wheeze, early frequent: 1.14 [0.95: 1.37] Bronchitis, ever MD-diagnosed: 0.95 [0.82: 1.10] Itchy rash/eczema: 0.99 [0.89: 1.11] Eczema, ever MD-diagnosed: 0.99 [0.87: 1.12]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Brauer et al. (2002) **Period of Study:** NR **Location:** The Netherlands	**Outcome:** Questionnaire derived wheezing, dry nighttime cough, ear, nose and throat infections, skin rash; Physician diagnosed asthma, bronchitis, influenza, eczema **Age Groups:** age 2 **Study Design:** Prospective cohort **N:** 4146 children **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, maternal smoking, mattress cover (allergen-free), maternal education, paternal education, gender, gas stove, gas water heater, any other siblings, ethnicity, breastfeeding, mold at home, pets, allergies in mother, allergies in father **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 4 2-week periods dispersed throughout 1 year, adjusted for temporal trend **Mean (SD):** 16.9 **Percentiles:** 10th: 14.0 25th: 15.0 50th(Median): 17.3 75th: 18.2 90th: 19.1 **Range (Min, Max):** 13.5, 25.2 **Monitoring Stations:** 40 **Copollutant (correlation):** Soot: r = 0.99 NO_2: r = 0.97	**PM Increment:** 3.2 µg/m^3 OR Estimate [Lower CI, Upper CI]; Unadjusted Wheeze 1.14 (0.99–1.30) Asthma 1.08 (0.84–1.37) Dry cough at night 1.10 (0.95–1.27) Bronchitis 1.00 (0.85–1.18) E, N, T infections 1.14 (0.99–1.33) Flu 1.15 (1.03–1.28) Itchy rash 1.07 (0.95–1.20) Eczema 1.02 (0.90–1.16) Adjusted Wheeze 1.14 (0.98–1.34) Asthma 1.12 (0.84–1.50) Dry cough at night 1.04 (0.88–1.23) Bronchitis 1.04 (0.85–1.26) E, N, T infections 1.20 (1.01–1.42) Flu 1.12 (1.00–1.27) Itchy rash 1.01 (0.88–1.16) Eczema 0.95 (0.83–1.10)
Reference: Brauer et al. (2002) **Period of Study:** NR **Location:** The Netherlands	**Outcome:** Questionnaire derived wheezing, dry nighttime cough, ear, nose and throat infections, skin rash; Physician diagnosed asthma, bronchitis, influenza, eczema **Age Groups:** age 2 **Study Design:** Prospective cohort **N:** 4146 children **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, maternal smoking, mattress cover (allergen-free), maternal education, paternal education, gender, gas stove, gas water heater, any other siblings, ethnicity, breastfeeding, mold at home, pets, allergies in mother, allergies in father **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ "soot" **Averaging Time:** 4 2-week periods dispersed throughout 1 year, adjusted for temporal trend **Mean (SD):** 16.9 10-5/m Percentiles: 10th: 1.16 25th: 1.38 50th(Median): 1.78 75th: 1.92 90th: 2.19 **Range (Min, Max):** 0.77, 3.68 **Unit (i.e. µg/m^3):** 10-5/m **Monitoring Stations**: 40 **Copollutant (correlation):** $PM_{2.5}$ (r = 0.99) NO_2 (r = 0.96)	**PM Increment:** 0.54 x 10-5/m (equivalent to 0.8 µg/m^3 elemental carbon) OR Estimate [Lower CI, Upper CI]; Unadjusted Wheeze 1.11 [0.99–1.24] Asthma 1.07 [0.87–1.31] Dry cough at night 1.08 [0.95–1.21] Bronchitis 0.98 [0.85–1.12] E, N, T infections 1.12 [0.99–1.27] Flu 1.13 [1.03–1.23] Itchy rash 1.07 [0.97–1.19] Eczema 1.01 [0.91–1.13] Adjusted Wheeze 1.11 [0.97–1.26] Asthma 1.12 [0.88–1.43] Dry cough at night 1.02 [0.88–1.17] Bronchitis 0.99 [0.84–1.17] E, N, T infections 1.15 [1.00–1.33] Flu 1.09 [0.98–1.21] Itchy rash 1.02 [0.91–1.15] Eczema 0.96 [0.85–1.08]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Brauer et al. (2006) **Period of Study:** 1997-2001 **Location:** Germany; The Netherlands	**Outcome:** Otitis Media (parental report of doctor's diagnosis prior to age 2 years) **Age Groups:** 0-2 years **Study Design:** Prospective Cohort Study **N:** 4,379 children total The Netherlands: 3,714 Germany: 665 **Statistical Analyses:** Logistic regression **Covariates:** Sex, parental atopy, maternal education, siblings, maternal smoking during pregnancy, ETS exposure at home, use of gas for cooking, indoor moulds and dampness, number of siblings, breast-feeding, and presence of pets in the home **Season:** All **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **PM Component:** Elemental Carbon (EC) **Averaging Time:** 8 weeks (4 2-week periods dispersed throughout 1 year, adjusted for temporal trends) Mean: The Netherlands: $PM_{2.5}$: 16.9 EC: 1.72 Germany: $PM_{2.5}$: 13.4 EC: 1.76 **Range (Min, Max):** The Netherlands: $PM_{2.5}$: 13.5, 25.2 EC: 0.77, 3.68 Germany: $PM_{2.5}$: 12.0, 21.9 EC: 1.40, 4.39 **Monitoring Stations**: 80 (40 for each cohort)	**PM Increment:** $PM_{2.5}$: 3 μg/m^3 (~ IQR) EC: ~0.5 μg/m^3 (~ IQR) OR Estimate [Lower CI, Upper CI] The Netherlands: $PM_{2.5}$: At age 1: 1.13 (0.98–1.32) At age 2: 1.13 (1.00–1.27) EC: At age 1: 1.11 (0.98–1.26) At age 2: 1.10 (1.00–1.22) Germany: $PM_{2.5}$: At age 1: 1.19 (0.73–1.92) At age 2: 1.24 (0.84–1.83) EC: At age 1: 1.12 (0.83–1.51) At age 2: 1.10 (0.86–1.41)
Reference: Burr et al. (2004b) **Period of Study:** 3 weeks in July and Jan 1997 and 2 weeks in Nov 1996 and April 1997 **Location:** North Wales, England	**Outcome:** Self-report of symptoms only for wheeze, cough, phlegm, rhinitis, and itchy eyes. **Age Groups:** all **Study Design:** Repeated measures **N:** 386 persons in congested streets and 425 in the uncongested streets in 1996/1997. Of these, 165 and 283 completed the second phase of the study.	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Mean hourly concentrations **Mean (SD):** Congested Streets 1996-97 21.2 1998-99 16.2 Uncongested Streets 1996-97 6.7 1998-99 4.9 **Monitoring Stations**: 1 in congested street and 1 in uncongested	% change PM_{10} in congested streets: 23.6 % change PM_{10} in uncongested streets: 26.6 Uncongested street sampling site was 20 m from the congested street sampler. The opening of the by-pass produced a reduction in pollution in the congested streets. The health effects of these changed are likely to be greater for nasal and ocular symptoms than for lower respiratory symptoms. Uncertainty about the causality arises from low reponse rates and conflicting trends in respiratory and nasal symptoms.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Calderon-Garciduenas et al. (2006) **Period of Study:** 1999, 2000 **Location:** Southwest Mexico City & Tlaxcala, Mexico	**Outcome:** Hyperinflation, interstitial markings-measured by chest radiograph, and lung function–FVC, FEV_1, PEF, FEF25-75, measured using spirometry tests **Age Groups:** 5-13 yrs **Study Design:** Cohort1999– **N:** 249 (total), 230 (Southwest Mexico City), 19 (Tlaxcala) **Statistical Analyses:** Bayes test, Spearman rank correlation, multiple regression **Covariates:** Age, sex **Dose-response Investigated?** No **Statistical Package:** SAS 8.2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 1 yr **Mean (SD):** 21 2000–19 Tlaxacala: 1994-2000: <NAAQS std Mexico City **Monitoring Stations:** Southwest Mexico City–2 Tlxacala–periodic air monitoring data Co**pollutant:** O_3	**PM Increment:** NR % Change: % of children with FEV_1 <80% expected value: Mexico City (n = 77): 7.8% Tlaxacala (n = 19): 0% % children with hyperinflatioN: Mexico City: 65.6% Number with: No hyperinflation: 79 Mild: 72 Moderate: 56 Severe: 23 Tlaxacala: 5.3% Number with: No hyperinflation: 18 Mild: 1 Moderate: 0 Severe: 0 % children with interstitial markings: Mexico City: 52.6% Number with: No interstitial markings: 19 Mild: 0 Moderate: 0 Severe: 0 Tlaxacala: 0% Number with: No interstitial markings: 109 Mild: 112 Moderate: 9 Severe: 0
Reference: Cesaroni et al. (2008) **Period of Study:** Data on PM emissions collected in 2002; cross-sectional survey carried out in 1995 **Location:** Rome, Italy	**Outcome:** Self-reported chronic bronchitis or emphysema, asthma, and rhinitis **Age Groups:** 25-59 yrs **Study Design:** Cross-sectional **N:** 9,488 subjects who had been residents in same place for at least 3 yrs and who had participated in an extension of the ISAAC initiative in Italy in 1994 & 1995 **Statistical Analyses:** GEE with a logit link **Covariates:** sex, age, smoking habits, education level, and variable to account for correlation of data for members of the same family Effect Modifiers: stratified analysis by smoking status (only presented for the traffic score variable); also stratified by education level (data not shown) Dose-response investigated: Wald test to calculate p for trend	**Pollutant:** PM emissions (estimated) Emissions estimated using a model/method based on factors such as vehicle park, driving conditions, emission factors, fuel consumption, fuel properties, road gradients, and climatic conditions **Mean:** 0.12 kg/km^2 SD: 0.081	Odds Ratios for quartiles of PM emissions: Chronic bronchitis or emphysema (n = 397): 1st: 1.00 2nd: 0.96 (0.71, 1.30) 3rd: 0.90 (0.66, 1.23) 4th: 1.05 (0.77, 1.42) p-trend = 0.871 Asthma (n = 472): 1st: 1.00 2nd: 1.10 (0.84, 1.44) 3rd: 0.94 (0.71, 1.24) 4th: 1.06 (0.80, 1.39) p-trend = 0.980 Rhinitis (n = 1227): 1st: 1.00 2nd: 1.41 (1.17, 1.69) 3rd: 1.11 (0.92, 1.34) 4th: 1.37 (1.14, 1.64) p-trend = 0.018

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Dales et al., (2008) **Period of Study:** **Location:** Windsor, ON	**Outcome:** Pulmonary function and inflammation Age Groups: grades 4-6 **Study Design:** cross-sectional prevalence design **Statistical Analyses:** multivariate linear regression **Covariates:** Ethnic background, smokers at home, pets at home, acute respiratory illness, medication use	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean:** 15.4 5th: 14.2 95th: 17.2 **Copollutant:** SO_2 NO_2	**Increment:** Tertiles of exposure FEV_1: <15.19: 2.16 ± 0.01 15.19-15.96: 2.17 ± 0.02 >15.96: 2.18 ± 0.01 FVC: <15.19: 2.51 ± 0.02 15.19-15.96: 2.50 ± 0.02 >15.96: 2.52 ± 0.02 eNO: <15.19: 16.08 ± 0.70 15.19-15.96: 15.80 ± 0.76 >15.96: 16.79 ± 0.72
Reference: Gauderman et al. (2000b) **Period of Study:** 1993-1997 **Location:** Southern California	**Outcome:** FVC, FEV_1, MMEF, FEF_{75} **Age Groups:** fourth, seventh, or tenth graders **Study Design:** cohort **N:** 3035 subjects **Statistical Analyses:** Linear regression **Covariates:** Height, weight, BMI, asthma, smoking, exercise, room temperature, barometric pressure **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** annual avg of 2-week avg $PM_{2.5}$ **Mean (SD):** $PM_{2.5}$ 25.9 **Copollutant (correlation):** 0_3: r = -0.32 $PM_{10-2.5}$: r = 0.76 NO_2: r = 0.74 Inorg. Acid: r = 0.79	**Increment:** 25.9 μg/m^3 % Change (Lower CI, Upper CI) $PM_{2.5}$-4th grade FVC -0.47 (-0.94, 0.01) FEV_1 -0.64 (-1.28, 0.01) MMEF -1.03 (-1.95 to -0.09) FEF_{75} -1.31 (-2.57 to -0.03) $PM_{2.5}$-7th grade FVC -0.42 (-0.89, 0.05) FEV_1 -0.32 (-0.88, 0.24) MMEF -0.29 (-1.99, 1.44) FEF_{75} -0.26 (-1.75, 1.25) $PM_{2.5}$-10th grade FVC 0.19 (-0.68, 1.07) FEV_1 -0.25 (-1.41, 0.93) MMEF -0.17 (-3.66, 3.46) FEF_{75} -0.79 (-4.27, 2.82)
Reference: Gauderman et al. (2002b) **Period of Study:** 1996–2000 **Location:** Southern California	**Outcome:** Lung function development: FEV_1, maximal midexpiratory flow (MMEF) **Age Groups:** Fourth grade children (avg age = 9.9 yrs) **Study Design:** Cohort study **N:** 1678 children, 12 communities **Statistical Analyses:** Mixed model linear regression **Covariates:** Height, BMI, doctor-diagnosed asthma and cigarette smoking in previous year, respiratory illness and exercise on day of test, interaction of each of these variables with sex, barometric pressure, temperature at test time, indicator variables for field technician and spirometer **Dose-response Investigated?** Yes **Statistical Package:** SAS (10)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual 24 h averages **Mean (SD):** The avg levels were presented in an online data supplement (Figure E1) **PM Component:** Elemental carbon and organic carbon. **Monitoring Stations**: 12 **Copollutant (correlation):** O_3: (10 AM to 6 PM) r = 0.14 O_3: r = -0.39 NO_2: r = 0.77 Acid vapor: r = 0.87 PM_{10}: r = 0.95 $PM_{10-2.5}$: r = 0.81 EC: r = 0.93 OC: r = 0.89	**PM Increment:** 22.2 μg/m^3 Association Estimate: Non-statistically significant negative correlation between $PM_{2.5}$ and $FEV_1$and FVC growth rates were observed. MMEF growth rates had a negative correlation with $PM_{2.5}$ (r = -0.43 p = 0.05). $PM_{2.5}$ was not signifcantly correlated to FEV_1 (r = -0.31 p = 0.25)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Gauderman et al. (2004) **Period of Study:** Air pollution data ascertainment: 1994-2000. Spirometry testing: spring 2001-spring 2003 **Location:** 12 Communities in Southern California	**Outcome:** Lung function FVC, FEV_1, MMEF (Maximal midexpiratory flow rate) **Age Groups:** Children, Avg age 10 years **Study Design:** Prospective Cohort Study **N:** 12 Communities; 2,034 children; 24,972 child-months **Statistical Analyses:** Linear regression of changes in sex-and-community specific lung growth function and PM Correlation between % with low attained FEV_1 and PM. **Covariates:** Random effect for communities **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2-week measurements used to create annual averages Mean: Means are presented in figures only. **Range (Min, Max):** ~6, ~27 **Monitoring Stations**: 12 **Copollutant (correlation):** PM_{10}: r = 0.95 O_3: r = 0.18 NO_2: r = 0.79 EC: r = 0.91 OC: r = 0.91	**PM Increment:** Most to least polluted community Range: 22.8 µg/m^3 Difference in Lung Growth [Lower CI, Upper CI]; FVC -60.1 (-166.1 to 45.9) FEV_1 -79.7 (-153.0 to ¡6.4) MMEF -168.9 (-345.5 to 7.8) Correlation with % below 80% predicted Lung function (p-value) $PM_{2.5}$: 0.79 (0.002)
Reference: Gauderman et al. (2007) **Period of Study:** 1993-2004 **Location:** 12 Southern California Communities	**Outcome:** pulmonary function tests FVC, FEV_1, $MMEF/FEF_{25-75}$ **Age Groups:** Children (mean age 10 at recruitment, followed for 8 years) **Study Design:** Cohort Study (Children's Health Study) **N:** 3677 children (1718 in cohort 1 recruited 1993 and 1959 in cohort 2 recruited 1996); 22686 pulmonary function tests. **Statistical Analyses:** Hierarchical mixed effects model with linear splines **Covariates:** Adjustments for height, height squared, BMI, BMI squared, present asthma status, exercise or respiratory illness on day of test, smoking in previous year, field technician, traffic indicator (distance from freeway, distance from major roads), random effects for participant and community. **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Monitoring Stations**: 1 in each community	**PM Increment:** 22.8 µg/m^3 Pollutant effect reported as difference in 8 year lung function growth from least to most polluted community. Negative difference indicate growth deficits associated with exposure. For $PM_{2.5}$ FEV growth deficit is -100

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Gehring et al. (2002) **Period of Study:** 1995-2002 **Location:** Munich, Germany	**Outcome:** wheezing, cough without infection, dry cough at night, obstructive, spastic or asthmoid bronchitis, respiratory infections, sneezing, runny/stuffed nose **Age Groups:** 0-2 years **Study Design:** Prospective cohort **N:** 1756 infants **Statistical Analyses:** Logistic regression **Covariates:** sex, parental atopy (yes/no), maternal education, siblings (y/n), environmental tobacco smoke at home (y/n), use of gas for cooking (y/n), home dampness (y/n), indoor moulds (y/n), keeping of dogs (y/n) and cats (y/n) study (GINI or LISA) **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Mean (SD):** $PM_{2.5}$ mass: 13.4 $PM_{2.5}$ absorb. 1.77 * 10-5/m Percentiles: $PM_{2.5}$ mass: 10th: 12.2 25th: 12.5 50th(Median): 13.1 75th: 14.0 90th: 14.9 $PM_{2.5}$ absorbance: 10th: 1.47 * 10-5 25th: 1.54 * 10-5 50th(Median): 1.70 * 10-5 75th: 1.88 * 10-5 90th: 2.13 * 10-5 **Range (Min, Max):** $PM_{2.5}$ mass: 11.9, 21.9 $PM_{2.5}$ absorbance: 1.38 to 4.39 * 10-5 $PM_{2.5}$ mass: $PM_{2.5}$ absorbance: 1/m **PM Component:** $PM_{2.5}$ mass $PM_{2.5}$ absorbance (as a marker of diesel soot) **Monitoring Stations**: 40 **Copollutant (correlation):** NO_2: r = 0.99 $PM_{2.5}$ absorbance and NO_2: r = 0.95 $PM_{2.5}$ mass and $PM_{2.5}$ absorbance: r = 0.96	**PM Increment:** $PM_{2.5}$ mass: 1.5 µg/m^3 $PM_{2.5}$ absorb. 0.4 * 10-5/m (IQR) RR Estimate [Lower CI, Upper CI]; **Wheeze ($PM_{2.5}$ mass)** Age of 1 yr: All: 0.91 (0.76–1.09) Males: 0.91 (0.72–1.16) Females: 0.94 (0.70–1.27) Age of 2 years: All: 0.96 (0.83–1.12) Males: 0.93 (0.76–1.14) Females: 1.04 (0.83–1.30) **Cough W/O Infection** ($PM_{2.5}$ mass) Age of 1 yr: All: 1.34 (1.11–1.61) Males: 1.43 (1.14–1.80); Females: 1.19 (0.84–1.70) **Dry Cough At Night** ($PM_{2.5}$ mass) Age of 1 yr: All: 1.31 (1.07–1.60) Males: 1.39 (1.08–1.78); Females: 1.17 (0.81–1.68) Age of 2 years: All: 1.20 (1.02–1.42) Males: 1.25 (1.01–1.55); Females: 1.13 (0.86–1.48) **Bronchitis** ($PM_{2.5}$ mass) Age of 1 yr: All: 0.98 (0.80–1.20) Males: 0.97 (0.76–1.25); Females: 0.98 (0.68–1.41) Age of 2 years: All: 0.92 (0.78–1.09) Males: 0.92 (0.74–1.14); Females: 0.91 (0.68–1.21) **Resp Infections** ($PM_{2.5}$ mass) Age of 1 yr: All: 1.04 (0.91–1.19) Males: 1.04 (0.87–1.25); Females: 1.06 (0.87–1.31) Age of 2 years: All: 0.98 (0.80–1.20) Males: 0.99 (0.74–1.31): Females: 0.98 (0.73–1.31) **Sneezing/Runny Nose ($PM_{2.5}$ mass)** Age of 1 yr: All: 1.01 (0.85–1.20) Males: 0.97 (0.77–1.24); Females: 1.08 (0.84–1.41) Age of 2 years: All: 0.96 (0.82–1.12) Males: 0.91 (0.73–1.12); Females: 1.04 (0.83–1.31) Wheeze ($PM_{2.5}$ absorbance) Age of 1 yr: All: 0.93 (0.78–1.12) Males: 0.91 (0.71–1.15); Females: 1.01 (0.74–1.37) Age of 2 years: All: 0.98 (0.84–1.14) Males: 0.92 (0.75–1.13); Females: 1.07 (0.85–1.36) **Cough W/O Infection** ($PM_{2.5}$ absorbance) Age of 1 yr: All: 1.32 (1.10–1.59) Males: 1.38 (1.11–1.71); Females: 1.25 (0.87–1.78) **Dry Cough At Night** ($PM_{2.5}$ absorbance) Age of 1 yr: All: 1.27 (1.04–1.55) Males: 1.31 (1.04–1.67); Females: 1.16 (0.79–1.71) Age of 2 years: All: 1.16 (0.98–1.37) Males: 1.17 (0.95–1.44); Females: 1.12 (0.84–1.48) **Bronchitis** ($PM_{2.5}$ absorbance) Age of 1 yr: All: 0.99 (0.81–1.22) Males: 1.00 (0.78–1.27); Females: 0.94 (0.63–1.39) Age of 2 years: All: 0.94 (0.79–1.12) Males: 0.91 (0.72–1.13); Females: 0.95 (0.71–1.28) **Resp Infections ($PM_{2.5}$ absorbance)** Age of 1 yr: All: 1.03 (0.90–1.18) Males: 1.03 (0.86–1.23); Females: 1.05 (0.85–1.30) Age of 2 years: All: 0.99 (0.80–1.22) Males: 0.96 (0.73–1.26); Females: 1.04 (0.75–1.43) **Sneezing/Runny Nose ($PM_{2.5}$ absorbance)** Age of 1 yr: All: 0.95 (0.79–1.14) Males: 0.90 (0.70–1.16); Females: 1.06 (0.80–1.39) Age of 2 years: All: 0.92 (0.78–1.09) Males: 0.83 (0.66–1.05); Females: 1.06 (0.83–1.34))

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Goss et al. (2004b) **Period of Study:** 1999-2000 **Location:** USA	**Outcome:** Cystic Fibrosis pulmonary exacerbations, FEV_1 **Age Groups:** Children and adults over the age of 6 **Study Design:** cohort **N:** 11484 patients **Statistical Analyses:** Logistic regression, t-tests, Mann-Whitney tests, Chi-squared tests, polytomous regression, multiple linear regression **Covariates:** Age, sex, lung function, weight, insurance status, pancreatic insufficiency, airway colonization, genotype, median household income by census tract, zipcode. **Dose-response Investigated?** No **Statistical Package:** STATA, SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** annual mean of 24 h averages **Mean (SD):** 13.7(4.2) Percentiles: 25th: 11.8 50th(Median): 13.9 75th: 15.9 **Monitoring Stations**: 713	**PM Increment:** 10 µg/m^3 Odds Ratio Estimate [Lower CI, Upper CI]: Odds of having 2 or more pulmonary exacerbations as compared to 1 or less in 2000 1.21 (1.07 -1.33) Odds of having 1 pulmonary exacerbation as compared to no exacerbations in 2000 0.70 (0.59-0.98) Decrease in FEV_1 155ml(115-194) Decrease in FEV_1 in 2000 after adjusting for FEV_1 in 1999 24ml(7-40)
Reference: Hertz-Picciotto et al. (2005) **Period of Study:** May 1994 to March 1999 **Location:** Teplice and Prachatice, Czech Republic	**Outcome:** Developmental immunotoxicity as assessed by neonatal immunophenotypes **Age Groups:** Not specified: every woman who delivered in the two aforementioned districts were asked to participate **Study Design:** Cohort study **N:** 1397 mother-infant pairs **Statistical Analyses:** Multiple linear regression with lymphocyte percentage as responding variable and pollutant exposure to 14day averaging period before the date of cord blood collection **Covariates:** Season, length of labor, parity, number of previous stillbirths, medication during delivery, working status of mother, maternal education, exposure to active and secondhand smoke, family history of allergy, self-reports of workplace exposure to dust during pregnancy, self-reported maternal chronic or severe respiratory diseases during pregnancy. Ambient temperature and season were controlled for. **Dose-response Investigated?** Yes **Statistical Package:** SUDAAN (version 8)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h 14 day averages **Mean (SD):** Overall 24 h: 24.8 14 day avg: Teplice: 30.1 Prachatice 19.8 **PM Component:** PAHs **Monitoring Stations**: 2 stations: Teplice and Prachatice	**PM Increment:** 25 µg/m^3 Adjusted for 3-day temperature and season, $PM_{2.5}$ exposure during the 14 days before birth was associated with reduced T-lymphocyte fractions CD4+, CD3+ and an increase in B-lymphocyte fraction (CD19+). The associations were not quantitatively reported anywhere else in the paper other than in Figure 2 and Table 3

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hertz-Picciotto et al. (2007) **Period of Study:** 1994-98 + follow-ups at upto 4.5 years of age for child **Location:** Czech Republic districts of Teplice and Prachatice	**Outcome:** Lower respiratory illnesses, majority being acute laryngitis, tracheitis, bronchitis. ICD10 codes J04 and J20 **Age Groups:** Birth-4.5 years of age. **Study Design:** longitudinal follow up of a stratified random sample of mother-infant pairs from previous Pregnancy Outcome Stody. Low birth weight and preterm births sampled at higher fractions. **N:** 1133 children **Statistical Analyses:** Generalized linear longitudinal models, GEE to adjust for within subject correlations, robust variance estimates were obtained. Model fit judged using Akaike Information criterion. **Covariates:** age of child, breast feeding, environmental tobacco smoke, season, day of week, year of birth, gender, birth weight, pregnancy data including age at delivery, length of gestation, maternal hypertension and diabetes, infant APGAR score, maternal work history, demographics, lifestyle, reproductive and medical histories, temperature, fuel type, other children in household **Dose-response Investigated?** No **Statistical Package:** SUDAAN version 8	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Used 3, 7, 14, 30 and 45 day averages **Mean (SD):** daily mean 22.3 (sd 16 for 3 day avg, 11 for 45 day avg)	**PM Increment:** 25 µg/m^3 **RR Estimate [Lower CI, Upper CI]; lag:** Bronchitis, birth-23 months of age Categorical model High 30 day avg $PM_{2.5}$ (greater than 50 µg/m^3) 2.26(1.81-2.82) Medium 30 day avg $PM_{2.5}$ (between 25 and 50 µg/m^3) 1.48(1.32-1.65) Continuous model 1.30(1.08-1.58) Bronchitis, 2-4.5 years of age Categorical model High 30 day avg $PM_{2.5}$ (greater than 50 µg/m^3) 3.66(2.07-6.48) Medium 30 day avg $PM_{2.5}$ (between 25 and 50 µg/m^3) 1.60(1.41-1.82) Continuous model 1.23(0.94-1.62) Notes: Results of other averaging periods shown in plots.
Reference: Hogervorst et al. (2006) **Period of Study:** NR **Location:** Maastricht, the Netherlands (six schools selected)	**Outcome:** Decreased lung function **Age Groups:** 8-13 years old **Study Design:** Multivariate linear regression (enter method) analysis **N:** 342 children **Statistical Analyses:** ANOVA, Chi square **Covariates:** Independent variables: Age, height, gender, smoking at home by parents, pets, use of ventilation hoods during cooking, presence of unvented geysers, tapestry in the home, indoor/outdoor time, education level of parents. Dependent variables: lung function indices **Dose-response Investigated?** No	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Daily **Mean (SD):** 19.0 (3.2) **Monitoring Stations**: 6 **Copollutant:** PM_{10} TSP	**PM Increment:** 10 µg/m^3 **RR Estimate [Lower CI, Upper CI]; lag:** FEV 3.62 [0.50,7.63] FVC 1.80 [-2.10, 5.80] FEF 5.93 [-2.34, 14.89]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Islam et al. (2007) **Period of Study:** 1993-2001 **Location:** 12 communities in Southern California, U.S.	**Outcome:** New onset asthma **Age Groups:** 9-10 years **Study Design:** cohort **N:** 2057 **Statistical Analyses:** Cox proportional hazard model **Covariates:** Community, sex, race/ethnicity **Season:** all **Dose-response Investigated?** No **Statistical Package:** SAS V 9.1 **Lags Considered:** 0-2 years	**Pollutant:** $PM_{2.5}$ **Range (Min, Max):** "Low" $PM_{2.5}$ Communities (5.7-8.5) "High" $PM_{2.5}$ Communities (13.7-29.5) **Monitoring Stations**: 12 **Copollutant:** NO_2, acid vapour, PM_{10} and elemental and organic carbon correlated as a "non-ozone package" of pollutants with a similar pattern relative to each other across the 12 communities.	**PM Increment:** NR **IR Estimate [Lower CI, Upper CI];** Low PM FVC ≤ 90: 19.4 (7.5, 50.5) FVC 90-110: 16.8 (7.0, 40.1) FVC >110: 7.9 (2.9, 21.9) FEV_1 ≤ 90: 23.7 (9.4, 59.4) FEV_1 90-110: 15.6 (6.5, 37.4) FEV_1 >110: 6.5 (2.3, 18.7) FEF_{25-75} ≤ 90: 21.1 (8.8, 50.5) FEF_{25-75} 90-110: 11.9 (4.7, 30.0) FEF_{25-75} >110: 6.4 (2.3, 18.2) Overall: 14.2 (7.0, 28.7) High PM FVC ≤ 90: 14.2 (5.1, 39.6) FVC 90-110: 25.6 (11.1, 59.2) FVC >110: 16.7 (6.5, 42.9) FEV_1 ≤ 90: 20.8 (8.0, 54.0) FEV_1 90-110: 23.1 (10.0, 53.7) FEV_1 >110: 18.8 (7.5, 47.3) FEF_{25-75} ≤ 90: 23.8 (10.2, 55.6) FEF_{25-75} 90-110: 23.9 (9.9, 57.7) FEF_{25-75} >110: 15.9 (6.3, 40.5) Overall: 18.4 (9.4, 35.9)
Reference: Karr et al. (2007) **Period of Study:** 1995 to 2000 **Location:** South Coast Air Basin of southern California	**Outcome:** Bronchioloitis **Study Design:** Case-control. Cases included subjects with a record of a single hospitalization with a discharge diagnosis of acute bronchiolitis.10 controls per case were matched on birth date and gestational age. **N:** 18,595 cases; 169,472 controls **Statistical Analyses:** Conditional logistic regression to estimate relative risk of hospitalization for bronchiolitis. **Covariates:** Confounders included in the model were: gender, parity, chronic lung disease, cardiac and pulmonary anomalies, SES covariates; Age, gestational age, and season of birth were controlled for by matching **Dose-response Investigated?** Yes **Statistical Package:** STATA (Version 8)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h (lifetime monthly avg from birth & 30 days preceeding cases hospitalization) **Mean (SD):** 25 **Percentiles:** 25th: 19 50th(Median): 23 75th: 29 **Range (Min, Max):** 6 to 111 **Monitoring Stations:** 17	**PM Increment:** 10 μg/m^3 **RR Estimate [Lower CI, Upper CI]** Sub-chronic and chronic exposure: OR = 1.09 (1.04-1.14) Adjusted for adjusted: Sub-chronic OR = 1.10 (1.04, 1.16) Chronic OR = 1.09 (1.03–1.15) Adjusted for CO and NO_2: Sub-chronic OR = 1.14 (1.07, 1.21) Chronic OR = 1.12 (1.06, 1.20) Adjusted for O_3, CO, and NO_2: Chronic OR = 1.15 (1.08, 1.22) Sub-chronic OR = 1.13 (1.06, 1.21)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kim et al. (2004) **Period of Study:** Mar-June (spring) 2001; Sep-Nov (fall) 2001 **Location:** Alameda County, CA	**Outcome:** Asthma, bronchitis **Age Groups:** Children (grades 3-5) **Study Design:** Cross-sectional **N:** 1109 children, 871 (long term resident children), 462 (long term related females), 403 (long term related males) **Statistical Analyses:** 2-stage multiple logistic regression model **Covariates:** respiratory illness before age of 2, household mold/moisture, pests, maternal history of asthma (for asthma) **Season:** spring and fall **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.2	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 10 weeks **Mean (SD):** Study Avg 12 **Monitoring Stations**: 10 **Copollutant (correlation):** r^2 is approximately 0.9 for all copollutants–Black Carbon (BC), PM_{10}, NO_X, NO_2, NO (NO_X–NO_2)	**PM Increment:** 0.7 (IQR) **OR Estimate [Lower CI, Upper CI]:** Bronchitis All subjects: 1.02 [1.00, 1.08] LTR subjects: 1.03 [1.01, 1.08] LTR females: 1.04 [1.02, 1.05] LTR males: 1.02 [0.99, 1.05] Asthma All subjects: 1.00 [0.96, 1.12] LTR subjects: 1.01 [0.97, 1.06] LTR females: 1.06 [0.99, 1.15] LTR males: 0.99 [0.95, 1.04] Asthma excluding outlier school having a larger proportion of Hispanics All subjects: 1.04 [0.96, 1.12] LTR subjects: 1.03 [0.94, 1.13] LTR females: 1.03 [0.91, 1.17] LTR males: 1.03 [0.94, 1.18]
Reference: Leonardi et al. (2000) **Period of Study:** 1996 **Location:** 17 cities of Central Europe (Bulgaria, Czech Republic, Hungary , Poland, Romania, Slovakia)	**Outcome:** Immune biomarkers **Age Groups:** 9-11 **Study Design:** Cross-sectional **N:** 366 school children **Statistical Analyses:** Linear regression **Covariates:** Age, gender, parental smoking, laboratory of analysis, recent respiratory illness **Dose-response Investigated?** No **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** annual $PM_{2.5}$ **Mean (SD):** $PM_{2.5}$: 46 (10) **Range (Min, Max):** $PM_{2.5}$: (29, 67) 5th, median, & 95th percentile $PM_{2.5}$: 29, 44, 67	**% Change (Lower CI, Upper CI); p-value** $PM_{2.5}$ Neutrophils -10 (-45, 46); >.20 Total lymphocytes 49 (11, 101);.008 B lymphocytes 63 (4, 155);.034 Total T lymphocytes 72 (32; 123); <.001 CD4+ 80 (34; 143); <.001 CD8+ 61 (17, 119);.003 CD4/CD8 16 (-17, 62); >.20 NK 63 (3, 158);.035 Total IgG 24 (2, 52);.034 Total IgM -9 (-32, 22); >.20 Total IgA -1 (-25, 32); >.20 Total IgE -4 (-61, 137); >.20
Reference: McConnell (1999a) **Period of Study:** 1993 **Location:** Southern California	**Outcome:** Bronchitis, chronic cough, phlegm **Age Groups:** Children: 4th, 7th, & 10th graders **Study Design:** Cross-sectional **N:** 3676 people **Statistical Analyses:** Logistic regression **Covariates:** Age, sex, race, grade, health insurance **Dose-response Investigated?** Yes	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Yearly 2 wk avg **Mean (SD):** 15.3 **Range (Min, Max):** 6.7, 31.5 **Copollutant (correlation):** NO_2; r = 0.83 O_3; r = 0.50 Acid; r = 0.71	**Chlid Respiratory symptoms OR Estimate (Lower CI, Upper CI)** **$PM_{2.5}$ Increment:** 15 µg/m^3 Children w/ asthma Bronchitis: 1.4 (0.9, 2.3) Phlegm: 2.6 (1.2, 5.4) Cough: 1.3 (0.7, 2.4) Children w/ wheeze, no asthma Bronchitis: 0.9 (0.6, 1.4) Phlegm: 1.0 (0.6, 1.8) Cough: 1.1 (0.6, 1.9) Children w/ no wheeze, no asthma Bronchitis: 0.5 (0.3, 1.0) Phlegm: 0.8 (0.4, 1.5) Cough: 0.9 (0.6, 1.3)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: McConnell et al. (2003) **Period of Study:** 1993-99 **Location:** 12 Southern CA communities	**Outcome:** bronchitic symptoms **Age Groups:** 9-19 **Study Design:** communities selected on basis of historic levels of criteria pollutants and low residential mobility. **N:** 475 children **Statistical Analyses:** 3 stage regression combined to give a logistic mixed effects model **Covariates:** sex, ethnicity, allergies history, asthma history, SES, insurance status, current wheeze, current exposure to ETS, personal smoking status, participation in team sports, in utero tobacco exposure through maternal smoking, family history of asthma, amount of time routinely spent outside by child during 2-6 pm. **Dose-response Investigated?** No **Statistical Package:** SAS Glimmix macro	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 4 year averages **Mean (SD):** 13.8(7.7) **Range (Min, Max):** 5.5-28.5 **Copollutant (correlation):** PM_{10}: r = 0.79 $PM_{10-2.5}$: r = 0.24 Inorganic acid: r = 0.76 Organic Acid: r = 0.58 EC: r = 0.83 OC: r = 0.84 NO_2: r = 0.54 O_3: r = 0.72	**PM Increment:** Between community range 23 µg/m^3 Between community unit 1 µg/m^3 Within community 1 µg/m^3 OR Estimate [Lower CI, Upper CI] Between community per range 1.81(1.14-2.88) Between Community per unit 1.03(1.01-1.05) Within community per unit 1.09(1.01-1.17)
Reference: McConnell et al. (2003) **Period of Study:** 1993-99 **Location:** 12 Southern CA communities	**Outcome:** bronchitic symptoms **Age Groups:** 9-19 **Study Design:** communities selected on basis of historic levels of criteria pollutants and low residential mobility. **N:** 475 children **Statistical Analyses:** 3 stage regression combined to give a logistic mixed effects model **Covariates:** sex, ethnicity, allergies history, asthma history, SES, insurance status, current wheeze, current exposure to ETS, personal smoking status, participation in team sports, in utero tobacco exposure through maternal smoking, family history of asthma, amount of time routinely spent outside by child during 2-6 pm. **Dose-response Investigated?** No **Statistical Package:** SAS Glimmix macro	**Pollutant:** Elemental Carbon **Averaging Time:** 4 year avg **Mean (SD):** 0.71(0.41) **Range (Min, Max):** 0.1-1.2 **Copollutant (correlation):** $PM_{2.5}$: r = 0.83 PM_{10}: r = 0.71 $PM_{10-2.5}$: r = 0.30 Inorganic acid: r = 0.82 Organic Acid: r = 0.66 Organic Carbon: r = 0.88 NO_2: r = 0.54 O_3: r = 0.68	**PM Increment:** Between community range 1.1 µg/m^3 Between community unit 1 µg/m^3 Within community 1 µg/m^3 OR Estimate [Lower CI, Upper CI] Between community per range 1.64(1.06-2.54) Between Community per unit 1.55(1.05-2.30) Within community per unit 2.63(0.83-8.33)
Reference: McConnell et al. (2003) **Period of Study:** 1993-99 **Location:** 12 Southern CA communities	**Outcome:** bronchitic symptoms **Age Groups:** 9-19 **Study Design:** communities selected on basis of historic levels of criteria pollutants and low residential mobility. **N:** 475 children **Statistical Analyses:** 3 stage regression combined to give a logistic mixed effects model **Covariates:** sex, ethnicity, allergies history, asthma history, SES, insurance status, current wheeze, current exposure to ETS, personal smoking status, participation in team sports, in utero tobacco exposure through maternal smoking, family history of asthma, amount of time routinely spent outside by child during 2-6 pm. **Dose-response Investigated?** No **Statistical Package:** SAS Glimmix macro	**Pollutant:** Organic Carbon **Averaging Time:** 4 year avg **Mean (SD):** 4.5(2.7) **Range (Min, Max):** 1.4-11.6 **Copollutant (correlation):** $PM_{2.5}$: r = 0.84 PM_{10}: r = .70 $PM_{10-2.5}$: r = 0.27 Inorganic acid: r = 0.83 Organic Acid: r = 0.69 EC: r = 0.88 NO_2: r = 0.67 O_3: r = 0.81	**PM Increment:** Between community range 10.2 µg/m^3 Between community unit 1 µg/m^3 Within community 1 µg/m^3 OR Estimate [Lower CI, Upper CI] Between community per range 1.74(0.89-3.4) Between Community per unit 1.06(0.99-1.13) Within community per unit 1.41(1.12-1.78)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: McConnell, et al. (2006) **Period of Study:** 1996-1999 **Location:** 12 Southern California communities	**Outcome:** Prevalence of bronchitic symptoms (yearly). **Age Groups:** 10-15-years-old **Study Design:** longitudinal cohort **N:** 475 asthmatic children **Statistical Analyses:** Multilevel logistic mixed effects models. **Covariates:** age, second-hand smoke; personal smoking history; sex, race. **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 365 days **Percentiles:** Community by year (n = 48 = 12 communities · 4 years) 25th: NR 50th(Median): 3.4 75th: NR **Range (Min, Max):** Community by year (n = 48 = 12 communities · 4 years): (0.89, 8.7) **Monitoring Stations**: 12 **Copollutant:** O_3 NO_2 EC OC Acid vapor (acetic and formic acid)	**PM Increment:** 3.4 µg/m^3 OR Estimate [Lower CI, Upper CI] $PM_{2.5}$ Dog (n = 292): 1.56 [1.15: 2.12] No dog (n = 183): 1.03 [0.71: 1.49] $PM_{2.5}$*Dog interaction p-value: 0.06 Cat (n = 202): 1.30 [0.90: 1.88] No Cat (n = 273): 1.36 [0.99: 1.83] $PM_{2.5}$*Cat interaction p-value: 0.87 Neither pet (n = 112): 1.11 [0.71: 1.74] Cat only (n = 71): 0.85 [0.46: 1.57] Dog only (n = 161): 1.53 [1.04: 2.25] Both pets (n = 131): 1.58 [1.02: 2.46] Results suggest that dog ownership, a source of residential exposure to endotoxin, may worsen the severity of respiratory symptoms from exposure to air pollutants in asthmatic children. Although $PM_{2.5}$ was associated at a statistically significant level with ownership of both cats and dogs, it appears that dog ownership (with or without a cat) specifically worsens the association between $PM_{2.5}$ and respiratory symptoms in asthmatic children.
Reference: Meng et al. (2007) **Period of Study:** November 2000 and September 2001 **Location:** Los Angeles and San Diego counties	**Outcome:** Poorly controlled asthma vs. controlled asthma; ICD9NR **Age Groups:** 18-64, 65+ **Study Design:** Long-term exposure study; comparison of cases and controls **N:** 1,609 adults (represented individuals age 18+ who reported ever having been diagnosed as having asthma by a physician and had their address successfully geocoded) **Statistical Analyses:** Logistic regression to evaluate associations between TD (traffic density) and annual avg air pollution concentrations and poorly controlled asthma. Used sample weights that adjusted for unequal probabilities of selection into the CHIS sample. **Covariates:** Age, sex, race/ethnicity, family federal poverty level, county, insurance status, delay in care for asthma, taking medications, smoking behavior, self-reported health status, employment, physical activity **Dose-response Investigated?** yes	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-hs **Copollutant (correlation):** O_3: r = -0.76 NO_2: r = 0.87 PM_{10}: r = 0.84 CO: r = 0.52 TD: r = 0.13	Results for $PM_{2.5}$ were nonsignificant and not reported quantitatively.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Millstein, J et al. (2004) **Period of Study:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 Data were taken from the Children's Health Study **Location:** Alpine, Atascadero, Lake Arrowhead, Lake Elsinore, Lancaster, Lompoc, Long Beach, Mira Loma, Riverside, San Dimas, Santa Maria, and Upland, CA	**Outcome:** Wheezing & asthma medication use (ICD 9 NR) **Age Groups:** 4th grade students, mostly 9 yrs at the time of the study **Study Design:** Cohort Study, stratified into 2 seasonal groups/ **N:** 2081 enrolled, 2034 provided parent-completed questionnaire. **Statistical Analyses:** Multilevel, mixed-effects logistic model. **Covariates:** Contagious respiratory disease, ambient airborne pollen and other allergens, temperature, sex, age race, allergies, pet cats, carpet in home, environmental tobacco smoke, heating fuel, heating system, water damage in home, education level of questionnaire signer, physician diagnosed asthma. **Season:** Mar-Aug, 1995, and Sep, 1995 to Feb, 1996 **Statistical Package:** GLIMMIX SAS 8.00 macro for generalized linear mixed models. **Lags Considered:** 14	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Integrated values for successive 2-wk periods **PM Component:** Nitric acid, formic acid, acetic acid **Monitoring Stations**: 1 central location in each community **Copollutant (correlation):** O_3: r = 0.09 NO_2: r = 0.28 PM_{10}: r = 0.33 $PM_{10-2.5}$: r = -0.08	**PM Increment:** IQR: 5.24 µg/m^3 **Odds Ratio [lower CI, Upper CI]** Annual $PM_{2.5}$: 1.04 [0.83, 1.29] March-August $PM_{2.5}$: 0.91 [0.64, 1.30] Sep-Feb $PM_{2.5}$: 1.18 [0.89, 1.58]
Reference: Morgenstern et al. (2007) **Period of Study:** Mar 1999-Jul 2000 **Location:** Munich, Germany	**Outcome:** Asthma, wheezing, spastic/obstructive bronchitis. Dry cough at night, respiratory infections, sneezing, runny/stuffed nose without a cold. **Age Groups:** at 1 yr & at 2 yrs **Study Design:** Cohort **N:** 3577 children for the prediction models. Respiratory data available for 3129 children at 1 yr. **Statistical Analyses:** Pearson's correlation coefficient, prediction error expressed as root mean squared error (RMSE), multiple logistic regression with confounding factors, odds ratios **Covariates:** Sex, Parental atopy (genetic predisposition to allergies), environmental tobacco smoke at home, maternal education >or <12 yrs, sibling, gas stove, home dampness, indoor mold, pets. Since it was not feasible to measure personal exposure to NO_2, $PM_{2.5}$, and $PM_{2.5}$ absorbance, exposure modeling was used. **Statistical Package:** SAS V.8.02	**Pollutant:** $PM_{2.5}$ **Averaging Time:** annual **Mean (SD):** 12.8 Percentiles: 25th: 12.5 50th(Median): 12.9 75th: 13.3 **Range (Min, Max):** 6.8, 15.3 **Monitoring Stations**: 40: traffic, n = 17 and background, n = 23. **Copollutant (correlation):** $PM_{2.5}$ absorbance r = 0.49 NO_2 r = 0.45	**PM Increment:** 1.04 µg/m^3 **Odds Ratio [Lower CI, Upper CI]** Adjusted OR for $PM_{2.5}$ and: sneezing, runny/stuffed nose during the first year of life was 1.16 [1.01, 1.34] At age 1 yr For wheezing 1.01 [0.87, 1.18] For cough without infection 1.05 [0.88, 1.25] For dry cough at night1.08 [0.86, 1.27] For asthmatic, spastic, or obstrcutive bronchitis 1.04 [0.90, 1.29] For respiratory infection1.05 [0.88, 1.22] For sneezing, runny or stuffed nose 1.16 [1.01, 1.34] At age 2 yrs For wheezing 1.10 [0.96, 1.25] For cough without infection NA, insufficient sample For dry cough at night 1.03 [0.86, 1.19] For asthmatic, spastic, or obstructive bronchitis 1.05 [0.92, 1.20] For respiratory infection 1.09 [0.94, 1.07] For sneezing, runny or stuffed nose 1.19 [1.04, 1.36]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Morgenstern et al. (2007) **Period of Study:** Ma4 1999-Jul 2000 **Location:** Munich, Germany	**Outcome:** Asthma, wheezing, spastic/obstructive bronchitis. Dry cough at night, respiratory infections, sneezing, runny/stuffed nose without a cold. **Age Groups:** at 1 yr & at 2 yrs **Study Design:** Cohort **N:** 3577 children for the prediction models. Respiratory data were available for 3129 children at 1 yr. **Statistical Analyses:** Pearson's correlation coefficient, prediction error expressed as root mean squared error (RMSE), multiple logistic regression with confounding factors, odds ratios **Covariates:** Sex, Parental atopy (genetic predisposition to allergies), environmental tobacco smoke at home, maternal education >or <12 yrs, sibling, gas stove, home dampness, indoor mold, pets. Since it was not feasible to measure personal exposure to NO_2, $PM_{2.5}$, and $PM_{2.5}$ absorbance, exposure modeling was used. **Statistical Package:** SAS V.8.02	**Pollutant:** $PM_{2.5}$ Absorbance ($PM_{2.5}$ ab) **Averaging Time:** annual **Mean (SD):** 1.7 10 –5 m -1, **Percentiles:** 25th: 1.6 10 –5 m -1 50th(Median): 1.7 10 –5 m -1 75th: 1.8 10 –5 m -1 **Range (Min, Max):** 1.3, 3.2 10 –5 m -1 **Unit (i.e. µg/m³):** 10 –5 m -1 **Monitoring Stations**: 40: traffic, n = 17 and background, n = 23.	**PM Increment:** 0.22 x 10 -5 **Odds Ratio [Lower CI, Upper CI]; no lag** At age 1 yr For wheezing 0.97 [0.77, 1.23] For cough without infection 1.16 [0.87, 1.54] For dry cough at night1.09 [0.78, 1.51] For asthmatic, spastic, or obstrcutive bronchitis 1.14 [0.88, 1.48] For respiratory infections1.03 [0.86, 1.24] For sneezing, runny or stuffed nose 1.30 [1.03, 1.65] At age 2 yrs For wheezing 1.09 [0.90, 1.33] For cough without infection NR insufficient data For dry cough at night1.18 [0.93, 1.50] For asthmatic, spastic, or obstrcutive bronchitis 0.85 [0.30, 2.34] For respiratory infections1.05 [0.79, 1.39] For sneezing, runny or stuffed nose 1.27 [1.04, 1.56]
Reference: Oftedal et al. (2008) **Period of Study:** 2001-2002 **Location:** Oslo, Norway	**Outcome:** Lung function (PEF, FEF25%, FEF50%, FEV_1, FVC) **Age Groups:** 9-10 yrs **Study Design:** Cross-sectional **N:** 1847 children **Statistical Analyses:** Linear regression **Covariates:** Height, age, BMI, birth weight, temperature, maternal smoking, se **Dose-response Investigated?** Yes **Statistical Package:** SPSS, STATA, S-Plus **Lags Considered:** 1-3	**Pollutant:** $PM_{2.5}$ IQR: $PM_{2.5}$ in 1st yr of life: 6.2 $PM_{2.5}$ lifetime: 3.6	**PM Increment:** Per IQR β (Lower CI, Upper CI) $PM_{2.5}$ in 1st yr of life PEF -76.1 (-122.2 to -30.0) FEF25% -75.6 (-127.4 to -23.8) FEF 50% -62.4 (-107.4 to -17.4) FEV_1 -12.7 (-28.8, 3.4) FVC -2.9 (-20.5, 14.7) $PM_{2.5}$ lifetime exposure PEF -57.7 (-94.4 to -21.1) FEF25% -51.8 (-93.1 to -10.6) FEF 50% -48.4 (-84.2 to -12.6) FEV_1 -10.4 (-23.2, 2.4) FVC -3.9 (-17.9, 10.1)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Sharma et al. (2004) **Period of Study:** 11/2002–4/2003 **Location:** 3 sections in Kanpur City, India 1) Indian Institute of Technology Kanpur (IITK) 2) Vikas Nagar (VN) 3) Juhilal Colony (JC)	**Outcome:** Lung function **Age Groups:** 20–55 years **Study Design:** Cohort **N:** 91 people **Statistical Analyses:** Linear regression **Covariates:** NR **Season:** Fall, Winter, spring **Dose-response Investigated?** No **Statistical Package:** Microsoft Excel **Lags Considered:** 1d lag & 5d mov avg	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** IITK 158 (22) VN 85 (30) JC 59 (9) **PM Component:** Lead Nickel Cadmium Chromium Iron Zinc Benzene soluble fraction (includes polycyclic aromatic hydrocarbons [PAHs]) **Copollutant (correlation):** ΔPEF = mean daily deviations in PEF $PM_{2.5}$-ΔPEF: -0.30 $PM_{2.5}$-PM_{10}: 0.67 $PM_{2.5}$-PM_{10} (1-day lag): 0.49 $PM_{2.5}$-$PM_{2.5}$ (1-day lag): 0.88	**PM Increment:** 1 μg/m^3 ΔPEF (difference or change in peak expiratory flow) -0.0297 L/min
Reference: Sunyer, et al (2006) **Period of Study:** initial selection: 1991-1993, follow-up June 2000-December 2001 **Location:** 21 centers in 10 European countries	**Outcome:** Chronic bronchitis **Age Groups:** Mean age (range) Males- 42.62 (38.12-45.62) Females- 42.57 (39.92-45.69) **Study Design:** Hierarchical models **N:** 6924 **Statistical Analyses:** General additive models (GAM) **Covariates:** Smoking, age at end of education, occupational group, occupational exposures, respiratory infections during childhood, rhinitis, asthma, traffic intensity at household level. **Statistical Package:** STATA-8	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 18 months **Mean (SD):** 3.7-44.9 **Copollutants:** NO_2, SO_2	**PM Increment:** NR **Odds ratio [Lower CI, Upper CI]** Chronic phlegm prevalence at follow up Males: 0.97 [0.70,1.35]
Reference: Zeka et al. (2006b) **Period of Study:** November 14, 2000 and December 31, 2004 **Location:** Boston, MA	**Outcome:** Inflammatory markers (WBC count, C-reactive protein, sediment rate, and fibrinogen) **Age Groups:** Older age groups Mean: 73.0 **Study Design:** Ecological **N:** 710 currently active subjects **Statistical Analyses:** Linear regression analyses Non-parametric regression models **Covariates:** Age, BMI, Season **Season:** spring; summer; fall; winter (reference) **Dose-response Investigated?** No **Lags Considered:** 48 h, 1 week, 4 weeks	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48 h, 1 week, and 4 weeks, using hourly measurements **Mean (SD):** 11.6 (7.95) Percentiles: 50th(Median): 9.39 75th: 14.57 90th: 21.48 **Monitoring Stations**: 2 **Copollutant (correlation):** Particle Number: r = -0.02 Black Carbon: r = 0.52 SO_4^2: r = 0.50	**PM Increment:** 1 SD increase **% Change [Lower CI, Upper CI]; lag:** Fibrinogen Lag: 48 h -0.18 (-1.93, 1.57) Lag: 1 week -1.39 (-3.46, 0.67) Lag: 4 week 1.14 (-0.60, 2.88) C-Reactive Lag: 48 h -4.88 (-13.29, 3.53) Lag: 1 week -1.37 (-10.44, 7.71) Lag: 4 weeks 4.36 (-3.25, 11.96) Sediment Rate Lag: 48 h -16.91 (-43.66, 9.84) Lag: 1 week -18.89 (-47.48, 9.70) Lag: 4 weeks 24.93 (0.68, 49.18) WBC Count Lag: 48 h -3.18 (-5.39 to -0.97) Lag: 1 week -0.51 (-3.02, 2.00) Lag: 4 weeks -0.03 (-2.17, 2.10)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Zhang et al. (2002) **Period of Study:** 1993-1996 **Location:** 4 Chinese cities (urban and suburban location in each city): Guangzhou, Wuhan, Lanzhou, Chongqing	**Outcome:** Interview-self reports of symptoms: Wheeze (ever wheezy when having a cold); asthma (diagnosis by doctor); bronchitis (diagnosis by doctor); hospitalization due to respiratory disease (ever); persistent cough (coughed for at least 1 month per year with or apart from colds); persistent phlegm (brought up phlegm or mucus from the chest for at least 1 month per year with or apart from colds). **Age Groups:** Elementary school students; age range: 5.4–16.2 **Study Design:** Cross-sectional **N:** 7,557 returned questionnaires 7,392 included in first stage of analysis **Statistical Analyses:** 2-stage regression approach: Calculated odds ratios and 95% CIs of respiratory outcomes and covariates Second stage consisted of variance-weighted linear regressions that examined associations between district-specific adjusted prevalence rates and district-specific ambient levels of each pollutant. **Covariates:** Age, gender, breastfed, house type, number of rooms, sleeping in own or shared room, sleeping in own or shared bed, home coal use, ventilation device used, homes smokiness during cooking, eye irritation during cooking, parental smoking, mother's education level, mother's occupation, father's occupation, questionnaire respondent, year of questionnaire administration, season of questionnaire administration, parental asthma prevalence.	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 2 years **Mean (SD):** 92 (31) **Percentiles:** 25th: NR 50th(Median): NR 75th: NR IQR: 39 **Range (Min, Max):** Gives range (max.–min.): $PM_{2.5}$-98 **Monitoring Stations**: 2 types: municipal monitoring stations over a period of 4 years (1993-1996); schoolyards of participating children over a period of 2 years (1995–1996)	**PM Increment:** Interquartile range corresponded to 1 unit of change. **RR Estimate [Lower CI, Upper CI]; lag:** No association between $PM_{2.5}$ and any type of respiratory morbidity. No between or within city association between $PM_{2.5}$ and any type of respiratory morbidity. When scaled to an increment of 50 μg/m^3 increase in $PM_{2.5}$, association (ORs) between respiratory outcome and $PM_{2.5}$ was: Wheeze: 1.06 Asthma: 1.29 Bronchitis: 1.68 Hospitalization: 1.08 Persistent cough: 1.24 Persistent phlegm: 3.09

Table E-26. Long-term exposure to other PM size fractions and respiratory morbidity outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kasamatsu et al. (2006) **Period of Study:** 2001-2002 **Location:** Shenyang, China	**Outcome:** FVC, FEV_1, PEF, FEF_{75} **Age Groups:** School Children aged 8-10 **Study Design:** Children in three schools in three types of areas (commercial city area, residential city area, residential suburban area) invited to participate **N:** 322 children participated, 244 have complete data. **Statistical Analyses:** Genralized estimating equations **Covariates:** age, height, **Dose-response Investigated?** no **Statistical Package:** SAS **Lags**: Considered: previous quarter.	**Pollutant:** PM_7 **Averaging Time:** avg of 4 separate 2-7 consecutive day measurements within each designated measurement month of the quarter **Mean (SD):** School A 7/2001 86.4(14.2) 10/2001 114.1(35.1) 1/2002 118.2(28.2) 4/2002 182.7(102.1) School B 7/2001 90.1(8.3) 10/2001 161.5(45.7) 1/2002 118.8(28.2) 4/2002 152.0(31.3) School C 7/2001 78.1(16.9) 10/2001 131.2(29.6) 1/2002 142.2(37.6) 4/2002 173.6(121.5) **PM Component:** mainly pollutants associated with coal heating **Monitoring Stations**: 1 at each location	**PM Increment:** 63.0 µg/m^3 **Mean change of pulmonary function value [Lower CI, Upper CI] at lag 0** Boys FVC -0.095(-0.170,-0.019) FEV_1 -0.088(-0.158,-0.019) PEF -0.170(-0.365,0.032) FEF_{75} -0.063(-0.183,0.050) Girls FVC -0.082(-0.145,-0.019) FEV_1 -0.069(-0.126,-0.006) PEF 0.095(-0.095,0.290) FEF_{75} -0.032(-0.151,0.082) Mean change of pulmonary function value [Lower CI, Upper CI] at lag 1(previous quarter) Boys FVC -0.145(-0.189,-0.095) FEV_1 -0.095(-0.139,-0.057) PEF -0.082(-0.208,0.050) FEF_{75} 0.013(-0.063,0.088) Girls FVC -0.126(-0.170,-0.088) FEV_1 -0.101(-0.139,-0.063) PEF -0.101(-0.227,0.025) FEF_{75} -0.057(-0.132,0.019)
Reference: Kasamatsu et al. (2006) **Period of Study:** 2001-2002 **Location:** Shenyang, China	**Outcome:** FVC, FEV_1, PEF, FEF_{75} **Age Groups:** School Children aged 8-10 **Study Design:** Children in three schools in three types of areas (commercial city area, residential city area, residential suburban area) invited to participate **N:** 322 children participated, 244 have complete data. **Statistical Analyses:** Genralized estimating equations **Covariates:** age, height, **Dose-response Investigated?** no **Statistical Package:** SAS **Lags:** Considered: previous quarter.	**Pollutant:** $PM_{2.1}$ **Averaging Time:** avg of 4 separate 2-7 consecutive day measurements within each designated measurement month of the quarter **Mean (SD):** School A 7/2001 47.6(6.4) 10/2001 54.2(20.5) 1/2002 68.9(15.8) 4/2002 115.8(76.7) School B 7/2001 45.6(6.5) 10/2001 74.4(27.1) 1/2002 63.3(17.9) 4/2002 96.3(27.6) School C 7/2001 42.5(9.5) 10/2001 59.7(13.1) 1/2002 76.4(22.1) 4/2002 123.0(100.9) **PM Component:** mainly pollutants associated with coal heating **Monitoring Stations**: 1 at each location	**PM Increment:** 42.1 µg/m^3 Mean change of pulmonary function value [Lower CI, Upper CI] at lag 0 Boys FVC -0.126(-0.181,-0.076) FEV_1 -0.122(-0.173,-0.076) PEF -0.164(-0.303,-0.025) FEF_{75} -0.046(-0.131,0.038) Girls FVC -0.110(-0.156,-0.067) FEV_1 -0.101(-0147,-0.059) PEF 0.008(-0.131,0.147) FEF_{75} -0.055(-0.139,0.030) Mean change of pulmonary function value [Lower CI, Upper CI] at lag 1(previous quarter) Boys FVC -0.099(-0.145,-0.053) FEV_1 -0.059(-0.106,-0.020) PEF -0.040(-0.158,0.086) FEF_{75} 0.026(-0.046,0.092) Girls FVC -0.086(-0.125,-0.046) FEV_1 -0.066(-0.106,-0.026) PEF -0.079(-0.198,0.040) FEF_{75} -0.033(-0.106,0.040)

E.6. Long-Term Exposure and Cancer

Table E-27. Long-term exposure to PM_{10} and cancer outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Abbey et al. (1999) **Period of Study:** 1977-1992 **Location:** California	**Outcome (ICD9):** Lung Cancer Mortality (162) **Age Groups:** 27-95 at baseline **Study Design:** Cohort (AHSMOG) **N:** 6,338 nonsmoking CA Seventh-Day Adventists **Statistical Analyses:** time-dependent, gender-specific, Cox proportional hazards regression models **Covariates:** age, smoking, education, occupation, BMI	**Pollutant:** PM_{10} **Averaging Time:** monthly estimates from 1966-1992 **Mean (SD):** 51.24 (16.63) **Percentiles:** IQR: 24.08 **Range (Min, Max):** 0, 83.9 **Correlations:** SO_4: r = 0.68) SO_2: r = 0.31 O_3: r = 0.77 NO_2: r = 0.56	**PM Increment:** 24.08 RR, males: 3.39 [1.57, 7.19] RR, females: 1.33 [0.60, 2.96]
Reference: Beeson et al. (1998) **Period of Study:** 1977-1992 **Location:** California	**Outcome (ICD9:** Lung Cancer Mortality (162) **Age Groups:** 27-95 at baseline **Study Design:** Cohort (AHSMOG) **N:** 6,338 nonsmoking CA Seventh-Day Adventists **Statistical Analyses:** time-dependent, gender-specific, Cox proportional hazards regression models **Covariates:** Smoking, Education, Age, Alcohol **Statistical Package:** SAS **Lags Considered:** 3 yr	**Pollutant:** PM_{10} **Averaging Time:** monthly estimates from 1966-1992 **Mean (SD):** 51 (16.52) **Percentiles:** IQR: 24 **Range (Min, Max):** 0, 84	**PM Increment:** 24 RR, males: 5.21 [1.94, 13.99] RR, females: Positive, but not statistically significant
Reference: Binkova et al. (2007) **Period of Study:** February 6-20, 2001 **Location:** Prague, Czech Republic	**Outcome:** Total DNA adducts **Age Groups:** 22-50 yrs **Study Design:** Case Control **N:** 53 exposed policemen and 52 control policemen **Statistical Analyses:** Multivariate regression **Covariates:** Smoking. Vitamin C, polymorphisms of XPD repair gene in exon 23 and 6 and GSTM 1 gene **Season:** Winter	**Pollutant:** PM_{10} **Range (Min, Max):** 32-55 **Monitoring Stations:** 2	Genetic damage was observed in city policemen working in winter outdoors in the Prague downtown area; they had slightly elevated aromatic DNA adduct levels, which was more pronounced for a distinct DNA adduct spot that could originate from ambient exposure to B[a]P
Reference: Pope et al. (2007) **Period of Study:** 1982-1998 **Location:** 50 US states, District of Columbia, and Puerto Rico	**Outcome (ICD9):** Lung cancer mortality (162) **Age Groups:** Ages >30 years **Study Design:** Longitudinal cohort **N:** 1.2 million people **Statistical Analyses:** Cox proportional hazard, generalized additive **Covariates:** Age, sex, race, education, smoking status, marital status, occupational exposure, diet, body-mass index, alcohol consumption	**Pollutant:** PM_{10} **Mean (SD):** 1982-1998: 28.8(5.9)	Effect estimates: Effect estimates were recorded in Figure 5 and not presented quantitatively anywhere else

Table E-28. Long-term exposure to $PM_{2.5}$ (including PM components/sources) and cancer outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Abbey et al. (1999) **Period of Study:** 1977-1992 **Location:** California	**Outcome:** Lung Cancer Mortality (ICD9: 162) **Age Groups:** 27-95 at baseline **Study Design: Cohort** (AHSMOG) **N:** 6,338 nonsmoking CA Seventh-Day Adventists **Statistical Analyses:** time-dependent, gender-specific, Cox proportional hazards regression models **Covariates:** age, smoking, education, occupation, BMI	**Pollutant:** SO_4 **Averaging Time:** monthly estimates from 1977-1992 **Mean (SD):** 7.24 (2.55) **Percentiles:** IQR: 2.97 **Range (Min, Max):** 0,32.11 **PM Component:** Sulfate **Correlations:** PM_{10}: r = 0.33 SO_2: r = 0.68 O_3: r = 0.53 NO_2: r = 0.76	No results presented due to inadequate lag time
Reference: Binkova et al. (2007) **Period of Study:** February 6-20, 2001 **Location:** Prague, Czech Republic	**Outcome:** Total DNA adducts **Age Groups:** 22-50 yrs **Study Design:** Case Control **N:** 53 exposed policemen and 52 control policemen **Statistical Analyses:** Multivariate regression **Covariates:** Smoking. Vitamin C, polymorphisms of XPD repair gene in exon 23 and 6 and GSTM 1 gene **Season:** Winter	**Pollutant:** $PM_{2.5}$ **Range (Min, Max):** 27-38 **c-PAHs: range** = 18-22 ng/m^3 **B[a]P: range** = 2.5-3.1 ng/m^3 **Monitoring Stations:** 2	Genetic damage was observed in city policemen working in winter outdoors in the Prague downtown area; they had slightly elevated aromatic DNA adduct levels, which was more pronounced for a distinct DNA adduct spot that could originate from ambient exposure to B[a]P
Reference: Liu et al. (2008) **Period of Study:** 1995-2005 **Location:** Taiwan	**Outcome:** Brain cancer deaths **Age Groups**: 29 yrs of age or younger **Study Design:** matched case-control **N:** 340 matched pairs **Statistical Analyses:** Conditional logistic regression **Covariates:** age, gender, urbanization level, nonpetrochemical air pollution exposure level	No direct measures of pollutants; used an index to assign petrochemical air pollution exposure (each municipality was assigned an exposure by dividing the number of workers per municipality employed in the petrochemical industry by the municipalities total population)	People who lived in the group of municipalities with the highest levels of air pollutants arising from petrochemical sources were at a statistically significant increased risk for brain cancer development compared to the group living in municipalities with the lowest petrochemical air pollution exposure index
Reference: Nafstad et al. (2004) **Period of Study:** May 1972-Dec 1973 **Location:** Oslo, Norway	**Outcome:** Lung cancer **Age Groups:** 40-49 yr old men **Study Design:** Cohort **N:** 16,209 **Statistical Analyses:** Cox regression models **Covariates:** age, smoking habits, physical activity, occupation, height, and weight **Season:** all year	PM values had small variations and were not considered in analyses. **Copollutants:** SO_2 NO_X	No effect estimates for PM

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Pope et al. (2007) **Period of Study:** 1982-1998 **Location:** 50 US states, District of Columbia, and Puerto Rico	**Outcome:** Lung cancer mortality (162) **Age Groups:** Ages >30 years **Study Design:** Longitudinal cohort **N:** 1.2 million people **Statistical Analyses:** Cox proportional hazard, generalized additive **Covariates:** Age, sex, race, education, smoking status, marital status, occupational exposure, diet, body-mass index, alcohol consumption	**Pollutant:** $PM_{2.5}$ **Mean (SD):** 1979–1983: 21.1(4.6) 1999-2000: 14.0(3.0) **Avg:** 17.7(3.7)	**PM Increment:** 10 µg/m^3 **RR Estimate [Lower CI, Upper CI]; lag:** **Lung Cancer:** 1979-1983: 1.08[1.01, 1.16] 1999-2000: 1.13[1.04, 1.22] Avg: 1.14[1.04, 1.23] RR results were also presented in Figures 2-5. Authors found that $PM_{2.5}$ had the strongest association with increased risk of all-cause, cardiopulmonary, and lung cancer mortality.
Reference: Tovalin et al. (2006) **Period of Study:** 2002 **Location:** Mexico City and Puebla	**Outcome:** DNA damage (comet tail length) **Age Groups:** 18-60 **Study Design:** Panel Study **N:** 55 male workers **Statistical Analyses:** Mann-Whitney test, Chi-square, Spearman's correlation, logistic regression **Statistical Package:** SPSS and STATA	**Pollutant:** $PM_{2.5}$ Personal monitoring values observed in this study reported in Tovalin et al. 2003	OR for being a highly damaged worker: 1.02 (1.01-1.04) Correlation between comet tail lenth and PM 2.5: 0.57
Reference: Weng et al. (2008) **Location:** Taiwan **Period of Study:** 1995-2005	**Outcome**: Childhood Leukemia deaths **Age Groups**: 19 yrs of age or younger **Study Design:** matched case-control **N:** 340 matched pairs **Statistical Analyses:** Conditional logistic regression **Covariates:** age, gender, urbanization level, nonpetrochemical air pollution exposure level	No direct measures of pollutants; used an index to assign petrochemical air pollution exposure (each municipality was assigned an exposure by dividing the number of workers per municipality employed in the petrochemical industry by the municipalities total population)	No effect estimates for PM; People who lived in the group of municipalities with the highest levels of air pollutants arising from petrochemical sources were at a statistically significant increased risk for childhood leukemia deaths compared to the group living in municipalities with the lowest petrochemical air pollution exposure index

Table E-29. Long-term exposure to other PM size fractions and cancer outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Pope et al. (2007) **Period of Study:** 1982-1998 **Location:** 50 US states, District of Columbia, and Puerto Rico	**Outcome:** Lung cancer mortality (162) **Age Groups:** Ages >30 years **Study Design:** Longitudinal cohort **N:** 1.2 million people **Statistical Analyses:** Cox proportional hazard, generalized additive **Covariates:** Age, sex, race, education, smoking status, marital status, occupational exposure, diet, body-mass index, alcohol consumption	**Pollutant:** PM_{15} **Mean (SD):** 1979-1983: 40.3(7.7)	Effect estimates were recorded in Figure 5 and not presented quantitatively anywhere else.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Pope et al. (2007) **Period of Study:** 1982-1998 **Location:** 50 US states, District of Columbia, and Puerto Rico	**Outcome:** Lung cancer mortality (162) **Age Groups:** Ages >30 years **Study Design:** Longitudinal cohort **N:** 1.2 million people **Statistical Analyses:** Cox proportional hazard, generalized additive **Covariates:** Age, sex, race, education, smoking status, marital status, occupational exposure, diet, body-mass index, alcohol consumption	**Pollutant:** $PM_{15-2.5}$ **Mean (SD):** 1979-1983: 19.2(6.1)	Effect estimates were recorded in Figure 5 and not presented quantitatively anywhere else.

E.7. Long-Term Exposure and Reproductive Effects

Table E-30. Long-term exposure to PM_{10} and reproductive outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Referentce: Bell at al. (2007) **Period of Study:** 1999-2002 **Location:** Connecticut–Fairfield, Hartford, New Haven, New London, Windham, Massachusetts–Barnstable, Berkshire, Bristol, Essex, Hampden, Middlesex, Norfolk, Plymouth, Suffolk, Worcester	**Outcome:** Low birth weight **Age Groups:** Neonates **Study Design:** Cross-sectional **N:** 358,504 deaths **Statistical Analyses:** Multiple logistic and linear regressions **Covariates:** Child's sex, mother's education, tobacco use, mother's marital status, mother's race, time prenatal care began, mother's age, birth order, gestation length **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 22.3 (5.3) **Monitoring Stations:** NR **Copollutant::** NO_2, CO, SO_2 **Gestation exposure correlation:** $PM_{2.5}$: r = 0.77 NO_2: r = 0.55	**PM Increment:** 7.4 µg/m^3 (IQR) **Difference in birth weight [Lower CI, Upper CI]; lag:** -8.2 [-11.1 to -5.3] **Difference in birth weight by race of mother [Lower CI, Upper CI]; lag:** Black: -7.9 [-16.0, 0.2] White: -9.0 [-12.2 to -5.9] **Range among trimester models for change in birth weight per IQR increase (min, max); trimester:** -6.6 to -4.7; 3rd **OR Estimate for birth weight <2500 g [Lower CI, Upper CI]; lag:** 1.027 [0.991, 1.064] **Notes**: Analyses using first births alone yielded similar results. Two pollutant models for uncorrelated pollutants were analyzed but not presented quatitatively.
Reference: Brauer et al. (2008) **Period of Study:** 1999-2002 **Location:** Vancouver, BC	**Outcome:** Fetal growth restriction, SGA, LBW **Age Groups: Study Design:** Cohort **N:** 70,249 **Statistical Analyses:** Linear regression **Covariates:** Sex, parity, month and year of birth, maternal age and smoking, neighborhood level income and education **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 12.7 **Range (Min, Max):** 5.6, 35.4 **Monitoring Stations:** 19 **Copollutant:** NO NO_2 CO SO_2 O_3	**PM Increment:** 1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** SGA: 1.02 (0.99, 1.05) LBW: 1.01 (0.95, 1.08) Preterm (<30 weeks): 1.13 (0.95, 1.35)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chen et al. (2002) **Period of Study:** 1991-1999 **Location:** Washoe County, Nevada	**Outcome:** birth weight **Age Groups:** single births with gestational age between 37-44 weeks and maternal all ages **Study Design:** retrospective cohort **N:** 39,338 single births **Statistical Analyses:** multiple linear and logistic regression **Covariates:** infant sex, maternal residential city, education, medical risk factors, active tobacco use, drug use, alcohol use, prenatal care, mother's age, race and ethnicity of mothers and weight gain of mothers **Dose-response Investigated?** No **Statistical Package:** SPSS 10.0	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 31.53 (22.32) **Percentiles: 25th:** 16.80 **50th(Median):** 26.30 **75th:** 39.35 **Range (Min, Max):** (0.97-157.32) **Monitoring Stations:** 4 **Copollutant:** CO O_3	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Using continous pollutant variables Model one–PM_{10} 1 trimester Crude model: ß = -0.186 (0.225) Adjusted model: ß = -0.082 (0.221) 2 trimester Crude model: ß = 0.045 (0.223) Adjusted model: ß = -0.020 (0.221) 3 trimester Crude model: ß = -0.509 (0.231) Adjusted model: ß = -0.395 (0.227) Whole Crude model: ß = -0.823 (0.459) Adjusted model: ß = -0.726 (0.483) Model two CO and PM_{10} 3 trimester Crude model: ß = -1.044 (0.457) Adjusted model: ß = -1.078 (0.445) O_3 and PM_{10} 3 trimester Crude model: ß = -1.035 (0.385) Adjusted model: ß = -0.966 (0.378) Model three PM_{10}, O_3, and CO 3 trimester Crude model: ß = -1.070 (0.458) Adjusted model: ß = -1.102 (0.446) Whole Crude model: ß = -1.413 (0.733) Adjusted model: ß = -1.332 (0.738) Using categorical pollutant variables–3 trimester Model 1–PM_{10} Adjusted model: ß = -10.243 (5.235) Model 2 PM_{10} and CO Adjusted model: ß = -11.883 (6.108) PM_{10} and O_3 Adjusted model: ß = 9.144 (5.860) Model 3 PM_{10}, CO, and O_3 Adjusted model: ß = -10.937 (6.222) Using logistic regression Exposure to PM_{10} at 3 trimester at >44.74 µg/m^3: OR = 1.105 (0.714-1.709) Between 19.72-44.74 µg/m^3: OR = 1.050 (0.811-1.360) **Notes:** Crude model: model with air-pollutant variables controlled with gestational age only. Adjusted model: model with air-pollutant variables controlled with confounding variables including gestational age, infant sex, maternal residential city, education, medical risk factors, active tobacco use, drug use, alcohol use, the trimester begins prenatal visits, total prenatal visits, mother's age, race and ethnicity of mother, and weight gain of mother.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Dales et al. (2004) **Period of Study:** Jan 1, 1984–Dec 31, 1999 **Location:** Canada (12 cities)	**Outcome:** SIDS (a sudden, unexplained death of a child <1 year of age for which a clinical investigation and autopsy fail to reveal a cause of death) **Age Groups:** Infants <1 yr **Study Design:** Time-series **N:** Total population of 12 cities: 10,310,309; 1556 cases of SIDS over study period **Statistical Analyses:** Random-effects regression model for count data (a linear association between air pollution and the incidence of SIDS was assumed on the logarithmic scale) **Covariates:** weather factors (daily mean temp, daily mean relative humidity, maximum change in barometric pressure, all measured on the day of death), length of time-period adjustment, seasonal indicator variables, and size-fractionated PM **Season:** Used piece-wise constant functions in time that varied by 3, 6, or 12 months **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24-hs (PM measures every 6 days; gaseous pollutants every day) **Mean (IQR):** PM_{10}: 23.43 (15.56) **Range (Min, Max):** IQR presented above **Monitoring Stations:** When data were available from more than one monitoring site, they were averaged **Copollutant:** $PM_{2.5}$ PM_{10} CO NO_2 O_3 SO_2	**Notes:** The abstract reports no association between increased daily rates of SIDS and fine particles measured every sixth day. However, no effect estimates presented for PM (only gaseous pollutants adjusted for PM).

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Dugandzic et al. (2006) **Period of Study:** 1/1/1998–12/31/2000 **Location:** Nova Scotia, Canada	**Outcome:** Low birth weight (LBW) (<2500 grams) **Age Groups:** Babies born ≥ 37 weeks (full term) **Study Design:** Retrospective cohort study **N:** 74,284 **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, parity, prior fetal death, prior neonatal death, prior low birth weight infant, smoking during pregnancy, neighborhood family income, infant gender, gestational age, weight change, year of birth **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** **Percentiles: 25th:** 14 **50th(Median):** 16 **75th:** 19 **Range (Min, Max):** Max: 53 **Monitoring Stations:** 18 **Notes:** Only three stations monitored more than one pollutant. Daily data were available for gaseous pollutants while particulate levels were measured every sixth day.	**PM Increment:** 1) IQR (5 µg/m^3) 2) Quartiles (first quartile is the reference) **Exposure period: first trimester** Unadjusted model 2nd quartile: 1.24 (0.95, 1.62) 3rd quartile: 1.25 (0.96, 1.62) 4th quartile: 1.28 (1.00, 1.65) Per IQR: 1.09 (1.00, 1.18) Adjusted model 2nd quartile: 1.24 (0.94, 1.64) 3rd quartile: 1.24 (0.95, 1.64) 4th quartile: 1.33 (1.02, 1.74) Per IQR: 1.09 (1.00, 1.18) Adjusted for Birth Year model 2nd quartile: 1.14 (0.86, 1.52) 3rd quartile: 1.08 (0.82, 1.44) 4th quartile: 1.11 (0.84, 1.48) Per IQR: 1.03 (0.94, 1.14) **Exposure period: second trimester** Unadjusted model 2nd quartile: 0.98 (0.76, 1.28) 3rd quartile: 1.09 (0.84, 1.40) 4th quartile: 1.00 (0.77, 1.28) Per IQR: 1.00 (0.91, 1.09) Adjusted model 2nd quartile: 1.02 (0.77, 1.34) 3rd quartile: 1.16 (0.89, 1.51) 4th quartile: 1.09 (0.83, 1.42) Per IQR: 1.02 (0.93, 1.12) Adjusted for Birth Year model 2nd quartile: 0.99 (0.75, 1.31) 3rd quartile: 1.10 (0.84, 1.45) 4th quartile: 1.01 (0.76, 1.34) Per IQR: 1.00 (0.90, 1.10) **Exposure period: third trimester** Unadjusted model 2nd quartile: 0.93 (0.72, 1.20) 3rd quartile: 1.07 (0.83, 1.37) 4th quartile: 0.92 (0.71, 1.18) Per IQR: 0.95 (0.87, 1.05) Adjusted model 2nd quartile: 0.96 (0.73, 1.26) 3rd quartile: 1.14 (0.88, 1.48) 4th quartile: 1.03 (0.79, 1.35) Per IQR: 0.99 (0.89, 1.09) Adjusted for Birth Year model 2nd quartile: 0.92 (0.70, 1.21) 3rd quartile: 1.04 (0.80, 1.36) 4th quartile: 0.92 (0.69, 1.22) Per IQR: 0.94 (0.85, 1.05)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Gilboa, et al. (2005) **Period of Study:** January 1, 1996-December 31, 2000 **Location:** Seven Counties in Texas, USA: (Bexar, Dallas, El Paso, Harris, Hidalgo, Tarrant, Travis	**Outcome:** Birth defects **Age Groups:** newborn babies **Study Design:** Case-control **N:** 5,338 newborn babies **Statistical Analyses:** Logistic regression **Covariates:** alcohol consumption during pregnancy, attendant of delivery (i.e.,the person who delivered the baby (physician/nursemaid-wife vs. other)), gravidity, marital status, maternal age, maternal education, maternal illness, maternal race/ethnicity, parity, place of delivery, plurality, prenatal care, season of conception, and tobacco use during pregnancy **Season:** all **Dose-response Investigated?** No **Statistical Package:** SAS v 8.2	**Pollutant:** PM_{10} **Averaging Time:** NR **Percentiles: 25th:** <19.5 **50th(Median):** 19.5-<23.8 **75th:** 23.8-<29.0 **100th:** ≥ 29.0 **Monitoring Stations:** The Environmental Protection Agency provided raw data for hourly (for gases) or daily (for PM) air pollution concentrations for the seven study counties **Copollutant:** CO NO O_3 SO	**PM Increment:** calculated as quartiles of avg concentration during weeks 3-8 of pregnancy **Isolated Cardiac Defects** **Aortic artery and valve defects:** 25th: 0.40 (0.15, 1.03); 50th: 0.45 (0.18, 1.13); 75th: 0.68 (0.28, 1.65) **Atrial septal defects:** 25th: 1.41 (0.86, 2.31); 50th: 2.13 (1.34, 3.37); 75th: 2.27 (1.43, 3.60) **Pulmonary artery and valve defects:** 25th: 1.14 (0.62, 2.10); 50th: 0.79 (0.41, 1.55); 75th: 0.68 (0.33, 1.40) **Ventricular septal defects:** 25th: 0.83 (0.61, 1.11); 50th: 1.12 (0.85, 1.48); 75th: 0.98 (0.73, 1.32) **Multiple Cardiac Defects** **Conotruncal defects:** 25th: 1.13 (0.79, 1.62); 50th: 1.20 (0.84, 1.72); 75th: 1.26 (0.86, 1.84) **Endocardial cushion and mitral valve defects:** 25th: 0.82 (0.54, 1.25); 50th: 0.66 (0.42, 1.05); 75th: 0.63 (0.38, 1.03) **Isolated Oral Clefts** **Cleft lip with or without palate:** 25th: 1.29 (0.90, 1.85); 50th: 1.45 (1.01, 2.07); 75th: 1.37 (0.94,2.00) **Cleft palate:** 25th: 0.99 (0.55, 1.78); 50th: 1.14 (0.64, 2.03); 75th: 1.11 (0.60, 2.06) **Individual Birth Defects** **Aortic valve stenosis:** 25th: 0.91 (0.53, 1.57); 50th: 0.86 (0.50, 1.50); 75th: 1.12 (0.63, 1.99) **Atrial septal defects:** 25th: 1.10 (0.89, 1.35); 50th: 1.28 (1.04, 1.57); 75th: 1.26 (1.03, 1.55) **Coarctation of the aorta:** 25th: 0.78 (0.53, 1.15); 50th: 0.68 (0.45, 1.02); 75th: 0.75 (0.48, 1.15) **Endocardial cushion defects:** 25th: 0.87 (0.49, 1.55); 50th: 1.12 (0.64, 1.96); 75th: 0.89 (0.47, 1.65) **Ostium secundum:** 25th: 1.15 (0.85, 1.55); 50th: 1.13 (0.83, 1.53); 75th: 1.06 (0.77, 1.48) **Pulmonary artery atresia without ventricular septal defects:** 25th: 1.93 (1.08, 3.45); 50th: 2.01 (1.11, 3.64); 75th: 0.86 (0.41, 1.83) **Pulmonary valve stenosis:** 25th: 1.16 (0.88, 1.55); 50th: 1.25 (0.94, 1.66); 75th: 1.27 (0.94, 1.71) **Tetralogy of Fallot:** 25th: 1.21 (0.72, 2.01); 50th: 1.40 (0.84, 2.33); 75th: 1.45 0.85, 2.48) **Ventricular septal defects:** 25th: 1.06 (0.90, 1.24); 50th: 1.10 (0.94, 1.29); 75th: 1.08 (0.92, 1.27)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Gouveia et al. (2004) **Period of Study:** 1997 **Location:** São Paulo, Brazil	**Outcome:** birth weight **Age Groups:** singleton full term live births within 1000 g to 5500 g **Study Design:** Cross sectional study **N:** 179,460 live births **Statistical Analyses:** GAM and Logistic regression models **Covariates:** maternal age, length of gestation, season, infant gender, maternal education, number of antenatal care visits, parity, and the type of delivery **Season:** All seasons **Dose-response Investigated?** Yes **Statistical Package:** S-Plus 2000	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 60.3 (25.2) **Range (Min, Max):** (25.5-153.0) **Monitoring Stations:** maximum of 12 sites **Copollutant (correlation):** CO: r = 0.9	**PM Increment:** 10 µg/m^3 **Mean [Lower CI, Upper CI]:** Changes in birth weight (in g) First trimester = -13.7 (-27.0- -0.4) Second trimester = -4.4 (-18.9- 10.1) Third trimester = 14.6 (0.0-29.2) **RR Estimate [Lower CI, Upper CI]:** (RR estimates are adjusted odds ratios for low birth weight according to quartiles of air pollution in each trimester of pregnancy.) 1st quartile First trimester = 1 (REF) Second trimester = 1 (REF) Third trimester = 1 (REF) 2nd quartile First trimester = 1.105 (0.994-1.229) Second trimester = 1.003 (0.904-1.113) Third trimester = 1.004 (0.914-1.104) 3rd quartile First trimester = 1.049 (0.903-1.219) Second trimester = 1.074 (0.920-1.254) Third trimester = 1.003 (0.861-1.169) 4th quartile First trimester = 1.144 (0.878-1.491) Second trimester = 1.252 (1.028-1.525) Third trimester = 0.970 (0.780-1.205) Multiple linear regression coefficients (SE) obtained from single, dual, and three pollutant models Single pollutant model = -1.37 (0.68) Two pollutant (PM_{10} and CO) = -0.51 (0.87) Two pollutant (PM_{10} and SO_2) = -0.94 (0.75) Three pollutant = -0.47 (0.88)
Reference: Ha et al. (2003) **Period of Study:** Jan 1995-Dec 1999 **Location:** Seoul, South Korea	**Outcome:** Post-neonate total and respiratory mortality **Age Groups:** 1 month-1 yr; 2 yr-65 yr, >65 yr **Study Design:** Cross-sectional **N:** 1045 post-neonate deaths, 67,597 2-65 yr old deaths, 100,316 >65 yr old deaths **Statistical Analyses:** Generalized additive model **Covariates:** Seasonality, temperature, relative humidity, day of the week **Dose-response Investigated?** No **Statistical Package:** S Plus **Lags Considered:** 0, 1, 2, 3, 4, 5, 6, 7, 1-5	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 69.2 (31.6) **Percentiles: 25th:** 44.8 50th(Median): 64.2 75th: 87.7 **Range (Min, Max):** 10.5 µg/m^3, 245.4 µg/m^3 **Monitoring Stations:** 27 **Copollutant (correlation):** NO_2: r = 0.73 SO_2 : r = 0.62 O_3: r = -0.02 CO: r = 0.63	**PM Increment:** 42.9 µg/m^3 **RR Estimate [Lower CI, Upper CI]; lag:** Total Mortality: 1 month-1 yr (post-neonates): 1.142 [1.096, 1.190] lag 0 2 yr-65 yr: 1.008 [1.006, 1.010] lag 0 >65 yr (elderly): 1.023 [1.023, 1.024] lag 0 Respiratory Mortality: 1 month-1 yr (post-neonates): 2.018 [1.784, 2.283] lag 0 2 yr-65 yr: 1.066 [1.044, 1.090] lag 0 >65 yr (elderly): 1.063 [1.055, 1.072] lag 0

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Hansen, et al. (2006) **Period of Study:** July 1, 2000- June 30, 2003 **Location:** Brisbane, Australia	**Outcome:** Pre-term birth (<37 weeks) **Age Groups:** newborn babies **Study Design:** Case-control **N:** 1583 live pre-terms births **Statistical Analyses:** Multiple logistic regression models **Covariates:** Neonate gender, mother's age, parity, indigenous status, number of antenatal visits, marital status, number of previous abortions/miscarriages, type of delivery, and index of SES **Season:** all **Dose-response Investigated?** No **Statistical Package:** SAS version 8.2	**Pollutant:** PM_{10} **Averaging Time:** recorded hourly, averaged daily **Mean (SD):** 19.6 (9.4) **Range (Min, Max):** 4.9, 171.7 **Monitoring Stations:** 5 **Copollutant (correlation):** Fine PM or bsp, 0.1 to <2.5 µg in diameter (0.58 to 0.76) O_3 (0.54 to 0.83) NO_2 (0.54 to 0.75) PM_{10} (0.80 to 0.93)	**PM Increment:** Trimester One 4.5 µg/m^3 Trimester Three 5.7 µg/m^3 **Odds Ratio [Lower CI, Upper CI]:** Trimester one 1.15 [1.06, 1.25] Trimester three 1.04 [0.92, 1.16]
Reference: Hansen et al. (2007) **Period of Study:** Jul 2000–Jun 2003 **Location:** Brisbane, Australia	**Outcome:** Birth weight and Small for Gestational Age (SGA; <10th percentile for age and gender); head circumference (HC) and crown-heel length (CHL) among subsample **Study Design:** Cross-sectional **N:** 26,617 births (birth weight analysis) and 21,432 (HC and CHL analyses) **Statistical Analyses:** Logistic (SGA) and linear (birth weight, HC, CHL) regressions **Covariates:** gender, gestational age (with a quadratic term), maternal age, parity, number of previous abortions/miscarriages, marital status, indigenous status, number of antenatal visits, type of delivery, an index of SES, and season of birth **Season:** assessed as a covariate **Dose-response Investigated?** Yes, assessed exposures as quartiles **Statistical Package:** SAS v8.2	**Pollutant:** PM_{10} **Averaging Time:** Trimester and monthly averages were used in analyses (calculated as the mean of daily values; hourly data was use to calculate daily means; city-wide avg used) **Mean (SD):** 19.6 (9.4) **Percentiles:** 25th: 14.6 50th: 18.1 75th: 22.7 **Range (Min, Max):** (4.9, 171.7) **Monitoring Stations:** 5 **Copollutant (correlation):** By trimesters: PM_{10} T1: PM_{10} T2: r = 0.12 PM_{10} T3: r = -0.55 O_3 T1: r = 0.77 O_3 T2: r = 0.28 O_3 T3: r = -0.61 NO_2 T1: r = 0.32 NO_2 T2: r = -0.65 NO_2 T3: r = -0.17 visibility reducing particles (bsp) T1: r = 0.82 visibility reducing particles (bsp) T2: r = 0.23 visibility reducing particles (bsp) T3: r = -0.62 PM_{10} T1: r = 0.12 PM_{10} T2: PM_{10} T3: r = 0.04 O_3 T1: r = -0.11 O_3 T2: r = 0.80 O_3 T3: r = 0.18 NO_2 T1: r = 0.77 NO_2 T2: r = 0.25 NO_2 T3: r = -0.72 visibility reducing particles (bsp) T1: r = 0.23 visibility reducing particles (bsp) T2: r = 0.80 visibility reducing particles (bsp) T3: r = -0.24 PM_{10} T1: r = -0.55 PM_{10} T2: r = 0.04	**PM Increment:** IQR (8.1 µg/m^3) **Effect Estimate [Lower CI, Upper CI]:** **Change (β) in mean birth weight (g) associated with trimester-specific exposures** **Trimester 1**: Continuous exposure: -3.2 (-11.9, 5.5) Quartiles of exposure: 1: Ref 2: -4.7 (-19.7, 10.2) 3: 4.2 (-12.9, 21.3) 4: -0.2 (-19.2, 18.8) p-trend: 0.864 **Trimester 2**: Continuous exposure: 0.4 (-9.4, 10.2) Quartiles of exposure: 1: Ref 2: 12.7 (-2.3, 27.6) 3: 7.6 (-10.6, 25.7) 4: 1.0 (-18.7, 20.7) p-trend: 0.922 **Trimester 3:** Continuous exposure: 3.6 (-6.9, 14.0) Quartiles of exposure: 1: Ref 2: 2.9 (-12.8, 18.7) 3: 18.5 (0.0, 36.9) 4: 4.3 (-15.8, 24.4) p-trend: 0.524 **ORs for SGA associated with trimester-specific exposures** **Trimester 1:** Continuous exposure: 1.04 (0.96, 1.12) Quartiles of exposure: 1: Ref 2: 1.23 (1.07, 1.42) 3: 1.12 (0.95, 1.31) 4: 1.12 (0.94, 1.34) p-trend: 0.361 **Trimester 2:** Continuous exposure: 0.95 (0.88, 1.04) Quartiles of exposure: 1: Ref 2: 0.96 (0.83, 1.11) 3: 1.06 (0.89, 1.25) 4: 0.98 (0.81, 1.18) p-trend: 0.962 **Trimester 3:** Continuous exposure: 0.93 (0.85, 1.03) Quartiles of exposure: 1: Ref 2: 0.90 (0.78, 1.04) 3: 0.81 (0.68, 0.96) 4: 0.86 (0.71, 1.04) p-trend: 0.098 **Change (β) in mean head circumference (HC; cm) associated with trimester-specific exposures**

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
		PM_{10} T3: O_3 T1: r = -0.56 O_3 T2: r = -0.18 O_3 T3: r = 0.81 NO_2 T1: r = -0.20 NO_2 T2: r = 0.75 NO_2 T3: r = 0.22 visibility reducing particles (bsp) T1: r = -0.62 visibility reducing particles (bsp) T2: r = 0.19 visibility reducing particles (bsp) T3: r = 0.79	**Trimester 1:** Continuous exposure: -0.01 (-0.04, 0.02) Quartiles of exposure: 1: Ref 2: -0.02 (-0.07, 0.04) 3: -0.02 (-0.08, 0.04) 4: -0.02 (-0.08, 0.05) p-trend: 0.605 **Trimester 2:** Continuous exposure: -0.01 (-0.04, 0.02) Quartiles of exposure: 1: Ref 2: 0.03 (-0.02, 0.08) 3: 0.00 (-0.06, 0.06) 4: -0.01 (-0.08, 0.05) p-trend: 0.538 **Trimester 3:** Continuous exposure: 0.02 (-0.02, 0.05) Quartiles of exposure: 1: Ref 2: 0.02 (-0.04, 0.07) 3: 0.07 (0.01, 0.13) 4: 0.04 (-0.03, 0.11) p-trend: 0.171 **Change (β) in mean crown-heel length (CHL; cm) associated with trimester-specific exposures** **Trimester 1:** Continuous exposure: 0.00 (-0.05, 0.05) Quartiles of exposure: 1: Ref 2: 0.02 (-0.07, 0.11) 3: 0.01 (-0.10, 0.11) 4: 0.04 (-0.07, 0.16) p-trend: 0.511 **Trimester 2:** Continuous exposure: 0.07 (0.01, 0.13) Quartiles of exposure: 1: Ref 2: 0.10 (0.01, 0.18) 3: 0.11 (0.00, 0.21) 4: 0.13 (0.01, 0.24) p-trend: 0.049 **Trimester 3:** Continuous exposure: -0.01 (-0.07, 0.05) Quartiles of exposure: 1: Ref 2: -0.02 (-0.11, 0.05) 3: 0.10 (-0.01, 0.21) 4: -0.01 (-0.13, 0.10) p-trend: 0.883

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Jalaludin et al. (2007) **Period of Study:** 1998-2000 **Location:** Sydney, Australia	**Outcome:** Gestational age (categorized: preterm birth: <37 weeks; term birth: ≥ 37 weeks but <42 weeks) **Age Groups:** infants **Study Design:** Cross-sectional **N:** 123,840 singleton births of >20 weeks gestation **Statistical Analyses:** Logistic regression **Covariates:** sex of child, maternal age, maternal smoking during pregnancy, gestational age at first antenatal visit, whether mother identifies as being Aboriginal or Torres Strait Islander, whether first pregnancy, season of conception, SES, (temperature and relative humidity were not significant in single variable models and therefore, were not included) **Season:** examined as covariate and effect modifier **Dose-response Investigated?** No **Statistical Package:** SAS v8	**Pollutant:** PM_{10} **Averaging Time:** 24 h averages used to calculate the mean concentration over the first trimester, the 3 months preceding birth, the first month after the estimated date of conception, and the month prior to delivery **Mean (SD):** (24 hr averages) All year: 16.3 (6.38) summer: 18.2 (7.20) Autumn: 17.0 (6.23) Winter: 14.5 (5.57) Spring: 15.7 (5.82) **Monitoring Stations:** 14 stations within the Sydney metropolitan area (levels averaged to provide one estimate for the entire study area) **Copollutant (correlation):** PM_{10} $PM_{2.5}$ (r = 0.83) CO (r = 0.28) NO_2 (r = 0.48) O_3 (r = 0.50) SO_2 (r = 0.42) **Notes:** Correlations between monitoring stations measuring PM_{10} ranged from 0.67 to 0.91	**PM Increment:** 1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** ORs (air pollutant concentration during the 1st trimester and preterm birth by season) Autumn: 1.462 (1.267, 1.688) Winter: 1.343 (1.190, 1.516) spring: 1.119 (0.973, 1.288) summer: 0.913 (0.889, 0.937) ORs (air pollutant concentrations during different exposure periods and preterm birth; for all of Sydney and among only those residing within 5 km of a monitoring station) 1 month preceding birth Sydney: 0.991 (0.979, 1.003) 5km: 1.008 (0.993, 1.022) 3 months preceding birth Sydney: 0.989 (0.975, 1.004) 5km: 1.012 (0.995, 1.030) 1st month of gestation Sydney: 0.983 (0.973, 0.993) 5km: 0.957 (0.914, 1.002) 1st trimester Sydney: 0.987 (0.973, 1.001) 5km: 1.009 (0.978, 1.041) **Notes:** Authors note that effect of PM_{10} on preterm birth for infants conceived during the autumn did not remain in 2 pollutant models (ORs between 0.77 and 1.04)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kaiser et al. (2004) **Period of Study:** 1995-1997 **Location:** 25 US counties (23 metropolitan areas): Jackson, AL Fresno, CA Los Angeles, CA Sacramento, CA San Diego, CA San Francisco, CA Denver, CO Hartford, CT Cook, IL Baltimore, MD Wayne, MI St. Louis, MO Bronx, NY Kings, NY New York, NY Philadelphia, PA El Paso, TX Harris, TX Dallas, TX Oklahoma, OK Tulsa, OK Providence, RI Salt Lake City, UT King, WA Milwaukee, WI	**Outcome:** Postneonatal death: All cause, SIDS (798.0) Respiratory disease (460-519) **Age Groups:** infants between 1-12 months **Study Design:** Retrospective cohort **N:** 700,000 infants (# deaths NR) **Statistical Analyses:** Risk assessment methods described iN: Kunzli et al. Public-health impact of outdoor and traffic-related air pollution: a European assessment. Lancet 2000, 356: 795-801. **Covariates:** Maternal education, maternal ethnicity, parental marital status, maternal smoking during pregnancy, infant's month and year of birth, avg temperature in the first 2 months of life **Season:** All **Dose-response Investigated?** NR **Statistical Package:** NR **Lags Considered:** Annual, county-level mean	**Pollutant:** PM_{10} **Averaging Time:** "annual mean levels" in each county **Mean (SD):** 28.4 **Range (Min, Max):** County range: 18.0, 44.8 **Monitoring Stations:** NR **Notes:** 14 out of 25 counties had PM_{10} levels >25 µg/m^3	**PM Increment:** Analysis 1: 16.4 µg/m^3 (difference between reference level of 12 µg/m^3 and observed mean level of 28.4 µg/m^3) Analysis 2: 13 µg/m^3 (difference between reference level of 12 µg/m^3 and 25 µg/m^3) **AR Estimate [Lower CI, Upper CI]:** Analysis 1: All cause 6% [3, 11] SIDS 16% [9, 23] Respiratory 24% [7, 44] Attributable # deaths per 100,000 infants: All cause 14.7 [7.3, 25.6] SIDS 11.7 [6.8, 16.6] Respiratory 2.3 [0.7, 4.1] Analysis 2: All cause 5% [2, 8] SIDS 12% [7, 18] Respiratory 19% [6, 34] Attributable # deaths per 100,000 infants: All cause 10.9 [5.5, 19.1] SIDS 9.0 [5.3, 12.8] Respiratory 1.8 [0.5, 3.2] **Notes:-**Authors did not extrapolate attributable cases below 12 µg/m^3 (i.e., reference level was set at 12 µg/m^3) -Attributable risks are based on the RRs reported by Woodruff et al, 1997 for a 10 µg/m^3 increase: All cause 1.04 [1.02-1.07] SIDS 1.12 [1.07, 1.17] Respiratory 1.20 [1.06, 1.36]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Kim et al. (2007a) **Period of Study:** May 1, 2001–May 31, 2004 **Location:** Seoul, Korea	**Outcome (ICD9 and ICD10):** LBW (low birth weight, less than 2500 g at later than gestational week 37), premature delivery (birth before the completion of the 37th week), stillbirth (intrauterine fetal death), IUGR (birth weight lower than the 10th percentile for the given gestational age), and congenital anomaly (a defect in the infant's body structure) **Age Groups:** Infants **Study Design:** Cross-sectional (women visiting the clinic for prenatal care were recruited with follow-up until discharge after delivery) **N:** 1514 observations (births) **Statistical Analyses:** multiple logistic and linear regression (in addition, for birth weight, used generalized additive model to account for long-term trends and nonlinear relationships between the response variable and the predictors, and to produce smoothed plots of the relationship between PM and birth weight) **Covariates:** Adjustment 1: infant sex, infant oder, maternal age and education, paternal education, season of birth; Adjustment 2: adjustment 1 factors plus alcohol, maternal BMI, maternal weight prior to delivery; (collected information on smoking, ETS, parity, past history of illnesses, history of illnesses during pregnancy but did not use in analyses due to small numbers or non-significance) **Season:** adjusted for season of delivery **Dose-response Investigated?** Yes **Statistical Package:** SAS 8.01, S-Plus 2000	**Pollutant:** PM_{10} **Averaging Time:** Used hourly exposure levels to calculate avg exposure levels at each trimester, each month of pregnancy, and 6 weeks before delivery from the nearest monitoring station (based on home address of mother); also created categories within each pregnancy period (<25th percentile [referent], 25th to 50th percentile, and >50th percentile) **Mean (SD):** Range of PM means across pregnancy periods: 88.7-89.7 **Monitoring Stations:** 27 stations	**LBW**: 1st Trimester Odds Ratios: Crude: 1.02 (0.93, 1.12); Adj 1: 1.03 (0.93, 1.14); Adj 2: 1.07 (0.96, 1.19) 2nd Trimester Odds Ratios: Crude: 1.03 (0.94, 1.14); Adj 1: 1.04 (0.93, 1.17); Adj 2: 1.07 (0.94, 1.22) 3rd Trimester Odds Ratios: Crude: 1.04 (0.97, 1.11); Adj 1: 1.05 (0.97, 1.14); Adj 2: 1.05 (0.96, 1.16) **IUGR**: 1st Trimester Odds Ratios: Crude: 1.07 (0.97, 1.19); Adj 1: 1.07 (0.95, 1.21); Adj 2: 1.14 (0.99, 1.31) 2nd Trimester Odds Ratios: Crude: 0.97 (0.85, 1.12); Adj 1: 0.97 (0.82, 1.13); Adj 2: 0.93 (0.77, 1.13) 3rd Trimester Odds Ratios: Crude: 0.82 (0.68, 0.99); Adj 1: 0.88 (0.72, 1.08); Adj 2: 0.85 (0.67, 1.08) **Birth defect**: 1st Trimester Odds Ratios: Crude: 1.08 (0.98, 1.20); Adj 1: 1.12 (1.00, 1.25); Adj 2: 1.08 (0.95, 1.22) 2nd Trimester Odds Ratios: Crude: 1.09 (0.99, 1.21); Adj 1: 1.11 (0.98, 1.26); Adj 2: 1.16 (1.00, 1.34) 3rd Trimester Odds Ratios: Crude: 1.00 (0.90, 1.11); Adj 1: 0.97 (0.86, 1.08); Adj 2: 0.97 (0.87, 1.10) **Stillbirth**: 1st Trimester Odds Ratios: Crude: 0.83 (0.76, 0.90); Adj 1: 0.93 (0.85, 1.02); Adj 2: 0.95 (0.85, 1.02) 2nd Trimester Odds Ratios: Crude: 0.99 (0.93, 1.05); Adj 1: 1.03 (0.95, 1.11); Adj 2: 1.07 (0.98, 1.17) 3rd Trimester Odds Ratios: Crude: 1.14 (1.10, 1.18); Adj 1: 1.09 (1.04, 1.15); Adj 2: 1.08 (1.02, 1.14) **LBW (categorical PM exposure):** 1st Trimester ORs: <25th: 1.0 25th-50th: 0.5 (0.1, 3.2) >50th: 1.0 (0.3, 3.8) 3rd Trimester ORs: <25th: 1.0 25th-50th: 1.3 (0.2, 10.4) >50th: 3.0 (0.5, 18.5) 6 wk before birth ORs: <25th: 1.0 25th-50th: 3.2 (0.3, 33.7) >50th: 5.2 (0.6, 47.6) **Changes in Birth Weight (95%CI) per 10** µg/m3 **increase in PM concentratioN:** 1st trimester: 7.8 (1.2, 14.5); 2nd trimester: -0.3 (-7.3, 6.8); 3rd trimester: -2.1 (-7.5, 3.4); 1st month: 4.4 (-1.0, 9.8); 2nd month: 6.4 (0.6, 12.2); 3rd month: 4.3 (-1.5, 10.2); 4th month: 3.0 (-3.7, 9.6); 5th month: -3.9 (-10.5, 2.7); 6th month: 0.1 (-5.7, 5.8); 7th month: 0.1 (-5.1, 5.3); 8th month: 0.0 (-4.5, 4.5); 9th month: 1.8 (-2.3, 5.9); Last 6 wk: -4.8 (-9.9, 0.4)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lee et al. (2003) **Period of Study:** Jan 1, 1996-Dec 31 1998 **Location:** Seoul, South Korea	**Outcome:** Low birth weight (LBW), <2500 g **Age Groups:** child-bearing age women and their newborn children **Study Design:** Retrospective time series **N:** 388,905 full-term single births **Statistical Analyses:** Generalized additive model, LOESS, Akaike's criterion, **Covariates:** Infant sex, birth order, maternal age, parental education level, time trend and gestational age. **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Arithmetic avg of hourly measurements at 20 stations **Mean (SD):** 71.1 (30.1) **Percentiles: 25th:** 47.4 **50th(Median):** 67.6 **75th:** 89.3 **Range (Min, Max):** 18.4, 236.9 **Monitoring Stations:** 20 **Copollutant (correlation):** 1st trimester: PM_{10}–CO: 0.47 PM_{10}–SO_2: 0.78 PM_{10}–NO_2: 0.66 2nd trimester: PM_{10}–CO: 0.68 PM_{10}–SO_2: 0.82 PM_{10}–NO_2: 0.81 3rd trimester: PM_{10}–CO: 0.69 PM_{10}–SO_2: 0.85 PM_{10}–NO_2: 0.80	**PM Increment:** IQR, 41.9 **RR Estimate [Lower CI, Upper CI]; lag:** 1st trimester: 1.03 [1.00, 1.07] 2nd trimester: 1.04 [1.00, 1.08] 3rd trimester: 1.00 [0.95, 1.04] All trimesters: 1.06 [1.01, 1.10] Low exposure in last 5 months using IQR during last 5 months: 0.94 [0.85, 1.05] Low exposure in first 5 months using IQR during first 5 months: 1.04 [1.01, 1.08] **Notes:** Birth weight was decreased by 19.6 g for an IQR increase in the 2nd trimester. The OR for LBW increased for female children, forth or higher order child, mother <20 yrs of age, and low parental education level.
Reference: Leem et al. (2006) **Period of Study:** 2001-2002 **Location:** Incheon, Korea	**Outcome (ICD9 and ICD10): Age Groups:** Pre-term delivery **Study Design:** Cross-sectional **N:** Cases: 2,082; Controls: 50,031 **Statistical Analyses:** Log-binomial regression (corrected for overdispersion; used the log link function) **Covariates:** Maternal age, parity, sex, season of birth, and education level of each parent **Season:** Controlled as a covariate **Dose-response Investigated?** Yes, assessed quartiles of exposure **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Trimesters (daily hourly data used to calculate) **Range (Min, Max):** Reported ranges within quartiles by trimester: 1st Trimester: 4: 64.57-106.39 3: 53.84-64.56 2: 45.95-53.83 1: 26.99-45.94 3rd Trimester: 4: 65.63-95.91 3: 56.07-65.62 2: 47.07-56.06 1: 33.12-47.06 **Monitoring Stations:** 27 monitoring stations; pollutant levels for each area were predicted from the levels recorded at the monitors using ordinary block kriging **Copollutant (correlation):** SO_2 (r = 0.13) NO_2 (r = 0.37) CO (r = 0.27)	**Effect Estimate [Lower CI, Upper CI]:** **Crude and Adjusted RR for preterm delivery and exposure during the 1st trimester** Crude Quartiles of exposure: 4: 1.07 (0.95, 1.21) 3: 1.02 (0.90, 1.15) 2: 1.06 (0.94, 1.20) 1: 1.00 Adjusted Quartiles of exposure: 4: 1.27 (1.04, 1.56) 3: 1.13 (0.94, 1.37) 2: 1.14 (0.97, 1.34) 1: 1.00 p-trend: 0.39 **Crude and Adjusted RR for preterm delivery and exposure during the 3rd trimester** Crude Quartiles of exposure: 4: 1.06 (0.94, 1.20) 3: 1.06 (0.94, 1.19) 2: 1.05 (0.93, 1.18) 1: 1.00 Adjusted Quartiles of exposure: 4: 1.09 (0.91, 1.30) 3: 1.04 (0.90, 1.21) 2: 1.05 (0.91, 1.20) 1: 1.00 p-trend: 0.33

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Lin et al. (2004a) **Period of Study:** 1/98-12/00 **Location:** São Paulo, Brazil	**Outcome:** Neonatal death **Age Groups:** Neonates (infants 0-28 days after birth) **Study Design:** Time series **N:** 1096 days, 6697 deaths **Statistical Analyses:** Poisson regression (GAM) **Covariates:** Non-parametric LOESS smoothers to control for: time (long term trend), temperature, humidity, and day of week Also controlled for holidays with linear term **Season:** All **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** Lag 0, "moving averages from 2 to 7 days" **Notes:** No explicit control for season apart from temperature	**Pollutant:** PM_{10} **Averaging Time:** Daily values **Mean (SD):** 48.62 (21.18) **Range (Min, Max):** 13.9, 157.3 **Monitoring Stations:** NR (indicated more than 1) **Copollutant (correlation):** CO r = 0.71 NO_2 r = 0.76 SO_2 r = 0.80 O_3 r = 0.36	**PM Increment:** 1 µg/m^3 **Log relative rate (standard error); lag** Single pollutant model 0.0017 (0.0008); lag 0 This translates to an 4.0% [95% CI: 0.3, 7.9] increase in neonatal mortality for a 23.3 µg/m^3 increase in PM_{10} Two-pollutant model 0.0000 (0.0011); lag 0 **Notes:** -In two pollutant model with PM_{10} and SO_2 (which are highly correlated), effect of PM disappeared and effect of SO_2 remained constant - results from pollutant moving averages from 2 to 7 days not reported, authors indicate effects only found for lag 0 (same day levels) - confidence intervals reported in abstract are incompatible with βs/standard errors and plotted results in text: abstract indicates a 4% increase in mortality with 95% CI: 2-6 for a 23.3 µg/m^3 increase in PM_{10}
Reference: Lin et al. (2004b) **Period of Study:** 1995-1997 **Location:** Taipei and Kaoshiung, Taiwan	**Outcome:** Low birth weight (<2500 grams) **Age Groups:** newborns **Study Design:** Retrospective cohort **N:** 92,288 infants **Statistical Analyses:** Logistic regression **Covariates:** Gender, birth order, gestational weeks, season of birth, maternal age, maternal education, copollutants **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** The 9-month pregnancy period for each infant, and each trimester	**Pollutant:** PM_{10} **Averaging Time:** NR, "daily measurements" **Mean (SD):** Reported by monitoring statioN: Taipei: 1. 48.78 2. 46.29 3. 48.79 4. 50.80 5. 52.54 Kaohsiung 1. 69.99 2. 63.39 3. 64.89 4. 75.79 5. 77.27 **Monitoring Stations:** 10 (5 in each city) **Notes:** All pregnant women/infants included in study lived within 3 km of an air quality monitoring station; Pollution assigned based on nearest air quality station to the maternal residence	**PM Increment:** Tertiles Entire pregnancy T1: <46.4 ppb T2: 46.4-63.1 ppb T3: >63.1 ppb First trimester T1: <45.8 ppb T2: 45.8-67.6 ppb T3: >67.6 ppb Second trimester T1: <44.6 ppb T2: 44.6-64.2 ppb T3: >64.2 ppb Third trimester T1: <43.7 ppb T2: 43.7-63.7 ppb T3: >63.7 ppb **RR Estimate [Lower CI, Upper CI];** Entire pregnancy T1: 1.00 T2: 0.96 [0.83, 1.11] T3: 0.87 [0.71, 1.05] First trimester T1: 1.00 T2: 0.96 [0.84, 1.09] T3: 0.97 [0.80, 1.17] Second trimester T1: 1.00 T2: 1.03 [0.90, 1.17] T3: 1.00 [0.83, 1.21] Third trimester T1: 1.00 T2: 1.02 [0.90, 1.16] T3: 0.97 [0.81, 1.17] **Notes:** RR for births in Kaoshiung vs. Taipei: 1.13 [1.03, 1.24]
Reference: Lipfert et al. (2000)	**Outcome:** Infant mortality; including respiratory mortality (traditional definition, ICD9 460-519), expanded definition (adds	**Pollutant:** PM_{10} **Averaging Time:** Yearly avg	**PM Increment:** NR (present regression coefficients) **Effect Estimate [Lower CI, Upper CI]:**

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Period of Study: 1990 **Location:** U.S.	ICD9 769 and 770) **Age Groups:** Infants **Study Design:** Cross-sectional **N:** 2,413,762 infants in 180 counties (Ns differ for various models) **Statistical Analyses:** Logistic regression **Covariates:** mother's smoking, education, marital status, and race; month of birth; and county avg heating degree days **Dose-response Investigated?** NR **Statistical Package:** NR	used **Mean (SD):** 33.1 (9.17) (based on 180 counties) **Range (Min, Max):** (16.9, 59) **Monitoring Stations:** NR **Copollutant (correlation):** PM_{10} SO_4^{2-} (r = 0.10) $NSPM_{10}$–non-sulfate portion of PM_{10} (r = 0.91) CO (r = 0.27) SO_2 (r = 0.04) **Notes:** TSP-based sulfate was adjusted for compatibility with the PM_{10}-based data	Presented regression coefficients (standard errors); (3 PM exposures regressed jointly); bold = p <0.05 Cause of death: All; Birth weight: All PM_{10}: 0.0114 (0.0015); SO_4^{2-}: -0.0002 (0.0061); $NSPM_{10}$: 0.0115 (0.0014) Cause of death: All; Birth weight: LBW PM_{10}: 0.0088 (0.0019); SO_4^{2-}: 0.0265 (0.0080); $NSPM_{10}$: 0.0086 (0.0020) Cause of death: All; Birth weight: normal PM_{10}: 0.0092 (0.0024); SO_4^{2-}: -0.0488 (0.0098); $NSPM_{10}$: 0.0096 (0.0024) Cause of death: All neonatal; Birth weight: All PM_{10}: 0.0126 (0.0018); SO_4^{2-}: 0.0267 (0.0076); $NSPM_{10}$: 0.0126 (0.0018) Cause of death: All neonatal; Birth weight: LBW PM_{10}: 0.0086 (0.0022); SO_4^{2-}: 0.0388 (0.0088); $NSPM_{10}$: 0.0093 (0.0022) Cause of death: All neonatal; Birth wt: normal PM_{10}: 0.0123 (0.0041); SO_4^{2-}: -0.0334 (0.0169); $NSPM_{10}$: 0.0125 (0.0040) Cause of death: All postneonatal; Birth wt: All PM_{10}: 0.0091 (0.0024); SO_4^{2-}: -0.0474 (0.0100); $NSPM_{10}$: 0.0096 (0.0024) Cause of death: All postneonatal; Birth wt: LBW PM_{10}: 0.0096 (0.0043); SO_4^{2-}: -0.0247 (0.0173); $NSPM_{10}$: 0.0101 (0.0042) Cause of death: All postneonatal; Birth wt: normal PM_{10}: 0.0074 (0.0030); SO_4^{2-}: -0.0569 (0.0121); $NSPM_{10}$: 0.0080 (0.0029) Cause of death: SIDS; Birth weight: All PM_{10}: 0.0138 (0.0038); SO_4^{2-}: -0.1078 (0.0151); $NSPM_{10}$: 0.0149 (0.0037) Cause of death: SIDS; Birth weight: LBW PM_{10}: 0.0115 (0.0088); SO_4^{2-}: -0.1378 (0.0337); $NSPM_{10}$: 0.0146 (0.0085) Cause of death: SIDS; Birth weight: normal PM_{10}: 0.0137 (0.0042); SO_4^{2-}: -0.0995 (0.0168); $NSPM_{10}$: 0.0147 (0.0041) Cause of death: All respiratory (ICD9: 460-519, 769, 770) Birth weight: All PM_{10}: 0.0168 (0.0034); SO_4^{2-}: 0.0706 (0.0146); $NSPM_{10}$: 0.0166 (0.0034) Cause of death: All respiratory (ICD9: 460-519, 769, 770) Birth weight: LBW PM_{10}: 0.0144 (0.0038); SO_4^{2-}: 0.0821 (0.0158); $NSPM_{10}$: 0.0139 (0.0038) Cause of death: All respiratory (ICD9: 460-519, 769, 770) Birth weight: normal PM_{10}: 0.0177 (0.0091); SO_4^{2-}: 0.0001 (0.0392); $NSPM_{10}$: 0.0118 (0.0090) Cause of death: Respiratory disease (ICD9: 460-519) Birth weight: All PM_{10}: 0.0133 (0.0089); SO_4^{2-}: 0.0093 (0.0384); $NSPM_{10}$: 0.0134 (0.0089) Cause of death: Respiratory disease (ICD9: 460-519) Birth weight: LBW PM_{10}: 0.0092 (0.0137); SO_4^{2-}: 0.0434 (0.0580); $NSPM_{10}$: 0.0089 (0.0138) Cause of death: Respiratory disease (ICD9: 460-519)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			Birth weight: normal PM_{10}: 0.0126 (0.0120); SO_4^{2-}: -0.0177 (0.0509); $NSPM_{10}$: 0.0128 (0.0119) Associations with SIDS by smoking status Smoking status: Yes; Birth weight: Normal PM_{10}: 0.0202 (0.0073); SO_4^{2-}: -0.0722 (0.0284); $NSPM_{10}$: 0.0206 (0.0071) Smoking status: No; Birth weight: Normal PM_{10}: 0.0104 (0.0051); SO_4^{2-}: -0.114 (0.021); $NSPM_{10}$: 0.0117 (0.005) Smoking status: Yes; Birth weight: LBW PM_{10}: 0.0322 (0.0130); SO_4^{2-}: -0.0958 (0.0483); $NSPM_{10}$: 0.0345 (0.0125) Smoking status: No; Birth weight: LBW PM_{10}: -0.0044 (0.012); SO_4^{2-}: -0.0172 (0.047); $NSPM_{10}$: -0.0007 (0.012) Mean risks (95%CI) between postneonatal SIDS among normal birth weight babies; pollutants regressed one at a time PM_{10}: 1.20 (1.02, 1.42); SO_4^{2-}: 0.43 (0.37, 0.51); $NSPM_{10}$: 1.33 (1.18, 1.50)
Reference: Maisonet et al. (2001) **Period of Study:** 1994-1996 **Location:** Northeastern U.S. (6 cities: Boston, Hartford, Philadelphia, Pittsburgh, Springfield, Washington DC)	**Outcome:** Low birth weight (LBW): infants with a birth weight <2,500 g and having a gestational age between 37 and 44 weeks **Age Groups:** Term live births (singleton) **Study Design:** Cross-sectional **N:** 89,557 infants **Statistical Analyses:** Logistic regression (LBW) and linear regression (for reductions in birth weight) **Covariates:** gestational age, gender, birth order, maternal age, race/ethnicity, years of education, marital status, adequacy of prenatal care, previous induced or spontaneous abortions, weight gain during pregnancy, maternal prenatal smoking, and alcohol consumption; season **Season:** Yes, as covariate **Dose-response Investigated?** Yes, categorical exposure variables assessed **Statistical Package:** STATA	**Pollutant:** PM_{10} **Averaging Time:** Trimester averages calculated using 24-h measurements taken every 6 days **Range (Min, Max):** Ranges for categories of exposure: 1st Trimester <25th: <24.821 25 to <50th: 24.821, 30.996 50 to <75th: 30.997, 36.142 75 to <95th: 36.143, 46.547 ≥ 95th: ≥ 46.548 2nd Trimester <25th: <24.702 25 to <50th: 24.702, 30.294 50 to <75th: 30.295, 35.410 75 to <95th: 35.411, 43.928 ≥ 95th: ≥ 43.929 3rd Trimester <25th: <24.702 25 to <50th: 24.702, 30.162 50 to <75th: 30.163, 35.642 75 to <95th: 35.643, 43.588 ≥ 95th: ≥ 43.589 **Monitoring Stations:** 3-4 per city **Copollutants:** CO, SO_2	**PM Increment:** 10 µg/m³ for analyses assessing exposures continuously **Effect Estimate [Lower CI, Upper CI]:** ORs for term LBW by trimester 1st Trimester Crude <25th: 1.00; 25 to <50th: 1.02 (0.90, 1.14); 50 to <75th: 0.90 (0.65, 1.24); 75 to <95th: 0.87 (0.58, 1.30); ≥ 95th: 0.89 (0.60, 1.33); Continuous: 0.93 (0.77, 1.13) 1st Trimester Adjusted <25th: 1.00; 25 to <50th: 1.02 (0.94, 1.11); 50 to <75th: 0.90 (0.78, 1.03); 75 to <95th: 0.85 (0.73, 1.00); ≥ 95th: 0.83 (0.70, 0.97); Continuous: 0.93 (0.85, 1.00) 2nd Trimester Crude <25th: 1.00; 25 to <50th: 1.01 (0.93, 1.10); 50 to <75th: 0.90 (0.66, 1.21); 75 to <95th: 0.92 (0.62, 1.34); ≥ 95th: 0.90 (0.61, 1.33); Continuous: 0.95 (0.78, 1.16) 2nd Trimester Adjusted <25th: 1.00; 25 to <50th: 1.06 (0.97, 1.15); 50 to <75th: 0.95 (0.85, 1.07); 75 to <95th: 0.91 (0.79, 1.05); ≥ 95th: 0.77 (0.63, 0.95); Continuous: 0.93 (0.85, 1.02) 3rd Trimester Crude <25th: 1.00; 25 to <50th: 0.94 (0.85, 1.05); 50 to <75th: 0.86 (0.58, 1.25); 75 to <95th: 0.86 (0.57, 1.29); ≥ 95th: 0.92 (0.61, 1.38); Continuous: 0.95 (0.75, 1.20) 3rd Trimester Adjusted <25th: 1.00; 25 to <50th: 0.98 (0.87, 1.10); 50 to <75th: 0.92 (0.76, 1.11); 75 to <95th: 0.88 (0.75, 1.04); ≥ 95th: 0.91 (0.77, 1.07); Continuous: 0.96 (0.88, 1.06) Adjusted ORs by race/ethnicity Whites: 1st Trimester <25th: 1.00; 25 to <50th: 1.13 (0.96, 1.33); 50 to <75th: 1.00 (0.92, 1.08); 75 to <95th: 1.00 (0.91, 1.09); ≥ 95th: 0.92 (0.81, 1.04); Continuous: 0.94 (0.90, 0.98) 2nd Trimester <25th: 1.00; 25 to <50th: 0.88 (0.77, 1.02); 50 to <75th: 0.95 (0.89, 1.02); 75 to <95th: 0.95 (0.84, 1.07); ≥ 95th: 0.89 (0.64, 1.26); Continuous: 0.96 (0.89, 1.04) 3rd Trimester <25th: 1.00; 25 to <50th: 0.84 (0.64, 1.11); 50 to <75th: 0.91 (0.83, 1.01); 75 to <95th: 0.80 (0.71, 0.90); ≥ 95th: 1.03 (0.86, 1.24); Continuous: 0.95 (0.90, 1.00) African Americans:

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			1st Trimester <25th: 1.00; 25 to <50th: 1.01 (0.98, 1.05); 50 to <75th: 0.88 (0.79, 0.98); 75 to <95th: 0.83 (0.70, 0.97); ≥ 95th: 0.81 (0.67, 0.99); Continuous: 0.93 (0.85, 1.01) 2nd Trimester <25th: 1.00; 25 to <50th: 1.10 (0.93, 1.30); 50 to <75th: 0.95 (0.80, 1.12); 75 to <95th: 0.88 (0.69, 1.11); ≥ 95th: 0.75 (0.54, 1.03); Continuous: 0.92 (0.80, 1.05) 3rd Trimester <25th: 1.00; 25 to <50th: 1.08 (0.92, 1.27); 50 to <75th: 0.89 (0.70, 1.12); 75 to <95th: 0.94 (0.75, 1.18); ≥ 95th: 0.83 (0.71, 0.97); Continuous: 0.99 (0.87, 1.11) Hispanics: 1st Trimester <25th: 1.00; 25 to <50th: 0.83 (0.64, 1.06); 50 to <75th: 0.86 (0.70, 1.05); 75 to <95th: 0.79 (0.68, 0.93); ≥ 95th: 1.36 (1.06, 1.75); Continuous: 0.96 (0.84, 1.09) 2nd Trimester <25th: 1.00; 25 to <50th: 1.16 (0.84, 1.61); 50 to <75th: 0.86 (0.63, 1.19); 75 to <95th: 0.98 (0.71, 1.34); ≥ 95th: 0.68 (0.38, 1.21); Continuous: 0.92 (0.81, 1.05) 3rd Trimester <25th: 1.00; 25 to <50th: 0.77 (0.55, 1.07); 50 to <75th: 1.12 (0.76, 1.66); 75 to <95th: 0.93 (0.65, 1.31); ≥ 95th: 0.90 (0.55, 1.47); Continuous: 0.96 (0.80, 1.15)
Reference: Mannes et al. (2005) **Period of Study:** January 1, 1998-December 31, 2000 **Location:** Metropolitan Sydney, Australia	**Outcome:** Risk of SGA and birth weight **Age Groups:** all singleton births >20 weeks and ≥ 400 grams birth weight and maternal all ages **Study Design:** cohort **N:** 138,056 singleton births **Statistical Analyses:** Logistic regression models **Covariates:** sex of child, maternal age, gestational age, maternal smoking, gestational age at first antenatal visit, maternal indigenous status, whether first pregnancy, season of birth, socioeconomic status **Season:** All seasons **Dose-response Investigated?** No **Statistical Package:** SAS v8.02	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 16.8 (7.1) **25th:** 12.3 **50th(Median):** 15.7 **75th:** 19.9 **Range (Min, Max):** (3.8-104.0) **Monitoring Stations:** up to 14 **Copollutants (correlations):** CO: r = 0.26 NO_2: r = 0.47 O_3: r = 0.52 $PM_{2.5}$: r = 0.81	**PM Increment:** 1 µg/m³ Risk of SGA All births One month before birth: OR = 1.01 (1.00-1.03) Third trimester: OR = 1.00 (0.99-1.013) Second trimester: OR = 1.01 (1.00-1.04) First trimester: OR = 1.00 (0.98-1.02) 5 km births One month before birth: OR = 1.00 (0.99-1.02) Third trimester: OR = 1.01 (0.99-1.02) Second trimester: OR = 1.02 (1.01-1.03) First trimester: OR = 1.01 (0.99-1.02) Change in birth weight All births One month before birth: ß = -1.21 (-2.31- -0.11) Third trimester: ß = -0.95 (-2.30–0.40) Second trimester: ß = -2.05 (-3.36- -0.74) First trimester: ß = -0.14 (-1.37- 1.09) 5 km births One month before birth: ß = -2.98 (-4.25- -1.71) Third trimester: ß = -3.84 (-5.35- -2.33) Second trimester: ß = -4.28 (-5.79- -2.77) First trimester: ß = -2.57 (-4.04- -1.10) Key second trimester findings Single pollutant model: ß = -4.28 (-5.79- -2.77) 2 pollutant (PM_{10} and CO): ß = -3.72 (-6.29- -.15) 2 pollutant (PM_{10} and NO_2): ß = -2.65 (-4.32- -0.98) 2 pollutant (PM_{10} and O_3): ß = -5.47 (-7.06- -3.88) 4 pollutant (PM_{10}, NO_2, CO and O_3): ß = -3.27 (-.05-0.51) Controlling for exposures in other pregnancy periods: ß = -3.03 (-4.85- -1.21)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Pereira et al. (1998) **Period of Study:** Jan 1991–Dec 1992 **Location:** Sao Paulo, Brazil **Notes:** Paper does not focus on PM as a pollutant of interest.	**Outcome:** Intrauterine mortality (fetuses over 28 weeks of pregnancy) **Study Design:** Time-series **N:** 730 days with PM measures **Statistical Analyses:** Poisson regression **Covariates:** Season and weather (temperature and relative humidity) **Season:** Assessed by including 23 indicator variables for month and year **Dose-response Investigated?** No **Statistical Package:** NR **Lags Considered:** Paper focuses on other pollutants (lags for PM not reported)	**Pollutant:** PM_{10} **Averaging Time:** 24 hr mean **Mean (SD):** 65.04 (27.28) **Range (Min, Max):** (14.80, 192.80) **Monitoring Stations:** 13 (averaged to provide city-wide pollutant level) **Copollutants (correlation):** NO_2 (r = 0.45) SO_2 (r = 0.74) CO (r = 0.41) O_3 (r = 0.25)	**PM Increment:** NR (reported only regression coefficients for PM) **Effect Estimate [Lower CI, Upper CI]:** Regression coefficients (standard errors) for pollutants when considered separately and simultaneously in the completed model: Separately: 0.0008 (0.0006) Simultaneously: -0.0005 (0.0010)
Reference: Ritz et al. (2000) **Period of Study:** 1989-1993 **Location:** Southern California	**Outcome:** Preterm birth (treated dichotomously as birth at <37 weeks gestation; also analyzed continuously) **Age Groups:** infants (born vaginally between 26-44 weeks of gestation) **Study Design:** Cross-sectional **N:** 97,158 births **Statistical Analyses:** Logistic and linear regression **Covariates:** maternal age, race, education, parity, interval since the previous livebirth, access to prenatal care, infant sex, previous low weight or preterm births, smoking (reported as "pregnancy complications"); to examine effect modification, authors conducted stratified analysis by region, birth and conception seasons, maternal age, race, education, and infant gender **Season:** Some models included season of birth or conception; also assessed as effect modifier in stratified analyses **Dose-response Investigated?** Examined adequacy of linear or log-linear relation using indicator terms for pollutant-avg quartiles; results presented in Fig 2 (dose-response demonstrated for last 6 weeks exposure period) **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24-h averages at 6 day intervals; averaged pollutant measures for 1, 2, 4, 6, 8, 12, and 26 weeks before birth and the whole pregnancy period **Mean (SD):** 6 weeks before birth: 47.5 (15.0) 1st month of pregnancy: 49.3 (16.9) **Range (Min, Max):** 6 weeks before birth: 12.3-152.3 1st month of pregnancy: 9.5-178.8 **Monitoring Stations:** 17 stations (PM measured at only 8 stations) **Copollutants (correlations):** 6 weeks before birth: CO (r = 0.43) NO_2 (r = 0.74) O_3 (r = 0.20) 1st month of pregnancy: CO (r = 0.37) NO_2 (r = 0.71) O_3 (r = 0.23) **Notes:** Averaged pollutant measures taken at the air monitoring station closest to the residence	**PM Increment:** 50 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** **All 8 stations** 6 weeks before birth Crude: 1.20 (1.09, 1.33) 2 exposure periods: 1.18 (1.07, 1.31) Other risk factors: 1.15 (1.04, 1.26) Other RFs plus season: 1.15 (1.03, 1.29) Multipollutant model: 1.19 (1.01, 1.40) 1st month of pregnancy Crude: 1.16 (1.06, 1.26) 2 exposure periods: 1.13 (1.04, 1.24) Other risk factors: 1.09 (1.00, 1.19) Other RFs plus season: 1.09 (0.99, 1.20) Multipollutant model: 1.12 (0.97, 1.29) **Coastal stations only** 6 weeks before birth Crude: 1.22 (1.00, 1.49) 2 exposure periods: 1.28 (1.04, 1.56) Other risk factors: 1.13 (0.93, 1.38) Other RFs plus season: 1.18 (0.92, 1.51) Multipollutant model: 1.42 (097, 2.01) 1st month of pregnancy Crude: 1.28 (1.06, 1.54) 2 exposure periods: 1.32 (1.09, 1.59) Other risk factors: 1.17 (0.97, 1.40) Other RFs plus season: 0.99 (0.79, 1.24) Multipollutant model: 1.09 (0.83, 1.41) **Inland stations only** 6 weeks before birth Crude: 1.27 (1.12, 1.44) 2 exposure periods: 1.27 (1.11, 1.44) Other risk factors: 1.19 (1.05, 1.35) Other RFs plus season: 1.27 (1.10, 1.48) Multipollutant model: 1.18 (0.97, 1.43) 1st month of pregnancy Crude: 1.16 (1.04, 1.29) 2 exposure periods: 1.16 (1.04, 1.29) Other risk factors: 1.09 (0.98, 1.21) Other RFs plus season: 1.09 (0.97, 1.24) Multipollutant model: 1.11 (0.93, 1.33) **Crude estimates for last 6 weeks exposure by season** Fall: 1.08 (0.88, 1.31) Summer: 1.06 (0.87, 1.29) Winter: 1.33 (1.07, 1.65) Spring: 1.81 (1.41, 2.31) **Reduction in mean gestation length for each increase in PM_{10} during last 6 weeks before birth (linear regression analysis)** Crude: 0.66 (± 0.24) days Adj: 0.90 (± 0.27) days **Notes:** Effect estimates remain stable when excluding SGA or LBW children or when restricting preterm births to SGA or LBW children only (results not presented)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ritz, et al. (2002) **Period of Study:** 1987-1993 **Location:** Southern California (July 1990–July 1993 for Los Angeles, 1989 for Riverside, 1988–1989 for San Bernardino, and 1987–1989 for Orange counties	**Outcome:** 1) aortic defects; 2) defects of the atrium and atrium septum; 3) endocardial and mitral valve defects; 4) pulmonary artery and valve defects; 5) conotruncal defects including tetralogy of Fallot, transposition of great vessels, truncus arteriosus communis, double outlet right ventricle, and aorticopulmonary window; and 6) ventricular septal defects not included in the conotruncal category. **Age Groups:** all live born infants and fetal deaths diagnosed between 20 weeks of gestation and 1 year after birth **Study Design:** logistic regression **N:** 10,649 infants and fetuses **Statistical Analyses:** hierarchical (two-level) regression model, polytomous logistic regression, linear model **Covariates:** gender, no prenatal care, multiple births, no siblings, maternal race, maternal age, maternal education, born before 1990, season of conception, **Season:** all **Dose-response Investigated?** Yes, for ozone and CO, study found a clear dose-response pattern for aortic septum and valve and ventricular septal defects and possibly for conotruncal and pulmonary artery and valve defects **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24 h **PM Component:** vehicle emissions **Monitoring Stations:** 30 **Copollutants (correlations):** CO: r = 0.32 NO_2	**Notes:** The authors did not observe consistently increased risks and dose-response patterns for PM_{10} after controlling for the effects of CO and ozone on these cardiac defects. (Quantitative results not shown).
Reference: Ritz et al. (2006) **Period of Study:** 1989-2000 **Location:** 389 South Coast Air Basin (SoCAB) zip codes	**Outcome:** total infant deaths during the first year of life as well as all respiratory causes of death (ICD-9 codes 460-519, 769, 770.4, 770.7, 770.8, and 770.9 and ICD-10 codes J00-J98, P22.0, P22.9, P27.1, P27.9, P28.0, P28.4, P28.5, and P28.9) and sudden infant death syndrome (SIDS) (ICD-9 code 798.0 and ICD-10 code R95). **Age Groups:** infants 0-1 yr **Study Design:** Case-control **N:** 2,975,059 births and 19,664 infant deaths **Statistical Analyses:** Conditional logistic regression analysis **Covariates:** risk factors available on birth and/or death certificates (maternal age, race/ethnicity, and education, level of prenatal care, infant gender, parity, birth country, and death season) **Season:** Death season (spring, summer, autumn, winter) **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** Two weeks before death: 46.2 One month before death: 46.3 Two months before death: 46.3 Six months before death: 46.3 **Range (Min, Max):** Two weeks before death: (21.0-83.5) One month before death: (25.0-77.2) Two months before death: (27.6-74.2) Six months before death: (31.3-69.5) **Monitoring Stations:** maximum of 31 **Copollutants (correlation):** Two weeks before death CO: r = 0.33; NO_2: r = 0.48; O_3: r = 0.12 One month before death CO: r = 0.33; NO_2: r = 0.48; O_3: r = 0.12 Two months before death CO: r = 0.32; NO_2: r = 0.48; O_3: r = 0.12 Six months before death CO: r = 0.29; NO_2: r = 0.44; O_3: r = 0.16	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** **All-cause death; 2 mo before death** Single-pollutant model: OR = 1.04 (1.01-1.06) Multiple-pollutant model: OR = 1.02 (0.99-1.05) **SIDS; 2 mo before death :** Single-pollutant model: OR = 1.03 (0.99-1.08) Multiple-pollutant model: OR = 1.01 (0.95-1.07) **Respiratory death; 2 wk before death** **Postneonatal deaths (28 d to 1 y)** Single-pollutant model: OR = 1.05 (1.01-1.10) Multiple-pollutant model: OR = 1.04 (0.98-1.09) **Postneonatal deaths (28 d to 3 mo)** Single-pollutant model: OR = 1.01 (0.95-1.08) Multiple-pollutant model: OR = 1.00 (0.92-1.09) **Post neonatal deaths (4-12 mo)** Single-pollutant model: OR = 1.12 (1.02-1.23) Multiple-pollutant model: OR = 1.07 (1.00-1.15)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Rogers et al. (2006) **Period of Study:** 1986-1988 **Location:** Georgia, USA	**Outcome**: VLBW; Term, AGA, Preterm AGA, Preterm, SGA **Age Groups:** Newborns and their mothers (<19 to ≥ 35-years-old) **Study Design:** case-control **N:** 325 infants (69 preterm SGA; 59 preterm AGA; 197 term AGA) and their mothers **Statistical Analyses:** logistic regression **Covariates:** maternal age, maternal race, maternal education, active and passive smoking, birth season, prepregnancy weight, pregnancy weight gain, maternal toxemia, anemia, asthma **Dose-response Investigated?** No **Statistical Package**: SUDAAN	**Pollutant:** PM_{10} **Averaging Time:** annual Preterm SGA: **50th(Median)**: 3.38 Preterm AGA: **50th(Median)**: 7.84 Term AGA: **50th(Median)**: 3.23 **Monitoring Stations**: NR **Percent Mothers Residing In County With Industrial Point Source** Preterm SGA: 60.9% Preterm AGA: 79.7% Term AGA: 60.4% **Percent Mothers Residing In Pm_{10} Quartile** (based on environmental transport model) Preterm SGA 1st quartile (<1.48): 31.9% 2nd quartile (1.48-3.74): 18.8% 3rd quartile (3.75-15.07): 26.1% 4th quartile (>15.07): 23.2% Preterm AGA 1st quartile (<1.48): 16.9% 2nd quartile (1.48-3.74): 22.1% 3rd quartile (3.75-15.07): 28.8% 4th quartile (>15.07): 32.2% Term AGA 1st quartile (<1.48): 24.7% 2nd quartile (1.48-3.74): 28.4% 3rd quartile (3.75-15.07): 27.9% 4th quartile (>15.07): 19.3%	**PM Increment:** Quartile **Notes:** Statistically significant increases in the odds of VLBW and preterm AGA births are associated with living in a county with a PM_{10} point source. Preterm AGA births are also associated with living in an area with very high (4th quartile) estimated PM_{10} exposure. Delivery of VLBW vs. Term AGA infant County with point source 2.54 [1.46, 4.22] PM_{10} quartile 1st quartile: reference 2nd quartile: 0.81 [0.42, 1.55] 3rd quartile: 0.85 [0.45, 1.16] 4th quartile: 1.94 [0.98, 3.83] Delivery of Preterm AGA vs. Term AGA infant County with point source 4.31 [1.88: 9.87] PM_{10} quartile 1st quartile: reference 2nd quartile: 1.56 [0.56: 4.35] 3rd quartile: 1.19 [0.44: 3.23] 4th quartile: 3.68 [1.44: 9.44] Delivery of Preterm AGA vs. Preterm SGA infant County with point source 2.07 [0.83: 5.16] PM_{10} quartile 1st quartile: reference 2nd quartile: 1.96 [0.59: 6.43] 3rd quartile: 2.10 [0.66: 6.73] 4th quartile: 2.58 [0.78: 8.51]

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Romieu et al. (2004) **Period of Study:** 1997 to 2001 **Location:** Ciudad Juarez, Mexico	**Outcome:** Respiratory-related infant mortality (460–519) **Age Groups:** <5 years **Study Design:** Case crossover **N:** 216 respiratory-related deaths; N = 412 other causes and N = 628 total deaths **Statistical Analyses:** The acute effects of air pollution was modeled on both total and respiratory-related mortality as a function of the pollution levels on the same day and preceding days and over two- and three-day averages before the date of death. Case-crossover with semi-symmetric bidirectional referent selection was the approach used. Data were stratified by day of the week and calendar month. Data were analyzed with conditional logistic regression. Second and third polynomial distributed lag models were used to study lag structure. BIC was used to determine lag length. **Covariate**: SES **Dose-response Investigated?** Yes **Statistical Package:** STATA 7.0 **Lags Considered:** 1–15 days	**Pollutant:** PM_{10} **Averaging Time:** Annual (24 h) **Mean (SD):** 1997: 33.04 (20.67) µg/m^3 1998: 35.25 (17.32) µg/m^3 1999: 45.92 (28.69) µg/m^3 2000: 43.38 (23.77) µg/m^3 2001: 39.46 (29.43) µg/m^3 **Monitoring Stations:** 5 stations in Ciudad Juarez 2 stations in El Paso (close to US-Mexico border) **Copollutant (correlation):** O_3: r = 0.01 **Notes:** Ciudad Juarez monitors measured PM_{10} every 6 days while El Paso monitors measured on a daily basis.	**PM Increment:** 20 µg/m^3 **RR Estimate [Lower CI, Upper CI]; lag:** **Total mortality:** OR = 1.02 (0.94–1.11) lag 1 OR = 1.03 (0.95-1.12) lag 2 OR = 1.03 (0.94-1.13 ac2 OR = 1.04 (0.95-1.15) ac3 **Respiratory mortality** OR = 0.95 (0.83-1.09) lag 1 OR = 1.04 (0.91-1.19) lag 2 OR = 0.98 (0.81-1.19) ac2 OR = 0.97 (0.74-1.26) ac3 **Lower SES** OR = 1.61 (0.97-2.66) lag 1 OR = 2.56 (1.06-6.17) ac2 PM_{10} was not related to infant mortality at other SES levels. **Notes:** All other odds ratios were reported in Table 4 and nowhere else in the report. ac2 and ac3 represent cumulative PM_{10} ambient levels over two or three days before death.
Reference: Sagiv et al. (2005) **Period of Study:** 1/1/1997–12/31/2001 **Location:** Allegheny county, Beaver county, Lackawanna county, Philadelphia county, Pennsylvania, U.S.A.	**Outcome:** Preterm birth (<36 weeks) **Age Groups:** Babies born between 20 and 44 weeks **Study Design:** Time series **N:** 187,997 births **Statistical Analyses:** Poisson regression; multivariable mixed-effects model with a random intercept for each county to incorporate count-level information. **Covariates:** Temperature, dew point temperature, mean 6-week level of copollutants (CO, NO_2, and SO_2), long-term preterm birth trends **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** 1, 2, 3, 4, 5, 6, 7	**Pollutant:** PM_{10} **Averaging Time:** 6-week period Daily **Mean (SD):** 6-week period 27.1 (8.3) Daily 25.3 (14.6) **Percentiles:** 6-week period **50th (Median):** 26.0 Daily **50th (Median):** 21.6 **Range (Min, Max):** 6-week period: 8.7, 68.9 Daily: 2.0, 156.3 **Monitoring Stations:** NR **Copollutant (correlation):** Daily PM_{10}–daily SO_2: r = 0.46	**PM Increment:** 1) 50 µg/m^3 2) Quartiles (first quartile is the reference) **Exposure period: 6 weeks before birth** Per 50 µg/m^3: 1.07 (0.98, 1.18) 2nd quartile: 1.00 (0.95, 1.05) 3rd quartile: 1.04 (0.99, 1.09) 4th quartile: 1.03 (0.98, 1.08) **Exposure period: 1-day acute time windows** Per 50 µg/m^3: 2-day lag: 1.10 (1.00, 1.21) 5-day lag: 1.07 (0.98, 1.18) **Notes:** Within the article, authors provide a Figure 1 displaying a graph of the relative risk (RR) and 95% confidence intervals (CI) for 1- to 7-day lags. While the authors report the 2- and 5-day lag RRs and 95% CIs in the text, the others are not specifically reported. However, the figure shows the approximate RRs per 50 µg/m^3 as indicated below: 1-day lag: 1.05 3-day lag: 1.05 4-day lag: 1.00 6-day lag: 0.97 7-day lag: 1.03

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Salam et al. (2005) **Period of Study:** 1975–1987 **Location:** Southern California	**Outcome:** Birth weight Low birth weight (LBW; <2500 g); Intrauterine growth retardation (IUGR) **Age Groups:** Children born full-term (between 37 and 44 weeks) **Study Design:** Cohort study **N:** 3901 children **Statistical Analyses:** Linear mixed-effects Logistic regression **Covariates:** Maternal age, months since last live birth, parity, maternal smoking during pregnancy, SES, marital status at childbirth, gestational diabetes, child's sex, child's race/ethnicity, child's grade in school (4th, 7th, and 10th), Julian day of birth **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** Monthly **Mean (SD):** Entire pregnancy: 45.8 (12.9) First trimester: 46.6 (15.9) Second trimester: 45.4 (14.8) Third trimester: 45.4 (15.5) **Monitoring Stations:** 1 or 3 (See notes) **Copollutant (correlation):** Entire pregnancy PM_{10}-$O_{3[10-6]}$: r = 0.54 PM_{10}-$O_{3[24\ hr]}$: r = 0.20 PM_{10}-NO_2: r = 0.55 PM_{10}-CO: r = 0.41 First trimester PM_{10}-$O_{3[10-6]}$: r = 0.54 PM_{10}-$O_{3[24\ hr]}$: r = 0.34 PM_{10}-NO_2: r = 0.48 PM_{10}-CO: r = 0.29 Second trimester PM_{10}-$O_{3[10-6]}$: r = 0.50 PM_{10}-$O_{3(24\ hr)}$: r = 0.27 PM_{10}-NO_2: r = 0.53 PM_{10}-CO: r = 0.35 Third trimester PM_{10}-$O_{3[10-6]}$: r = 0.52 PM_{10}-$O_{3[24\ hr]}$: r = 0.31 PM_{10}-NO_2: r = 0.52 PM_{10}-CO: r = 0.37 **Notes:** Exposure estimates were calculated by spatially interpolated monthly averages which were based off of three monitoring stations located within 50 km of the ZIP code region of maternal birth residences.	**PM Increment:** IQR (interquartile range) **Outcome: birth weight** Single-pollutant model Entire pregnancy 18 μg/m^3: -19.9 (-43.6, 3.8) First trimester 20 μg/m^3: -3.0 (-22.7, 16.7) Second trimester 19 μg/m^3: -15.7 (-36.1, 4.7) Third trimester 20 μg/m^3: -21.7 (-42.2 to -1.1) Multipollutant model (included O_3 (24 hr) in model; third trimester exposure) 20 μg/m^3: -10.8 (-31.8, 10.2) **Outcome: IUGR** Single-pollutant model Entire pregnancy 18 μg/m^3: 1.1 (0.9, 1.3) First trimester 20 μg/m^3: 1.0 (0.9, 1.2) Second trimester 19 μg/m^3: 1.0 (0.9, 1.2) Third trimester 20 μg/m^3: 1.1 (0.9, 1.3) **Outcome: LBW** Single-pollutant model Entire pregnancy 18 μg/m^3: 1.3 (0.8, 2.2) First trimester 20 μg/m^3: 1.0 (0.7, 1.5) Second trimester 19 μg/m^3: 1.2 (0.8, 1.7) Third trimester 20 μg/m^3: 1.3 (0.9, 1.9) **Notes:** Numbers reported for birth weight outcome are the effects on birth weight outcome (the change in birth weight in grams) across the IQR (which vary depending on air pollutant and duration of exposure measurement).
Reference: Suh et al. (2007) **Period of Study:** 2001-2004 **Location:** Seoul, Korea	**Outcome:** Birthweight **Age Groups:** prenatal followup for newborns **Study Design:** based prospective cohort study **N:** 199 pregnant mothers **Statistical Analyses:** ANCOVA, generalized linear models **Covariates:** infant's sex, maternal age, maternal and paternal education, parity, presence of illness during pregnancy, delivery month, gestational age (squared) **Dose-response Investigated?** No **Statistical Package**: SAS	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** 1st trimester: 76.41 (28.80) 2nd trimester: 77.84 (31.63) 3rd trimester: 95.61 (26.15) **Percentiles:** 1st trimester **25th:** 55.28 **50th(Median):** 71.09 **75th:** 92.38 2nd trimester **25th:** 48.65 **50th(Median):** 72.36 **75th:** 108.00 3rd trimester **25th:** 77.10 **50th(Median):** 96.35	**PM Increment:** Trimester ≥ 90th%ile compared to <90th%ile Least-square (ANCOVA) mean (SE) **All Genotypes** 1st trimester <90th%ile, N(%): 158 (90.3%): 3253 (37) ≥ 90th%ile, N(%): 17 (9.7%): 2841 (145) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.009; Adjusted, with CO: 0.041; Adjusted, with NO_2: 0.092; Adjusted, with SO_2: 0.012 2nd trimester <90th%ile, N(%): 153 (89.5%): 3253 (39) ≥ 90th%ile, N(%): 18 (10.5%): 3026 (157) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.177; Adjusted, with CO: 0.203; Adjusted, with NO_2: 0.151; Adjusted, with SO_2: 0.151

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
		75th: 116.68 **Range (Min, Max):** 1st trimester (21.00, 151.65) 2nd trimester (31.45, 139.13) 3rd trimester (23.45, 172.75) **Monitoring Stations**: 27 **Copollutant:** CO SO_2 NO_2	3rd trimester <90th%ile, N(%): 162 (90.5%): 3226 (38) ≥ 90th%ile, N(%): 17 (9.5%): 3122 (140) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.487; Adjusted, with CO: 0.748; Adjusted, with NO_2: 0.420; Adjusted, with SO_2: 0.466 **Genotype** MspI TT 1st trimester <90th%ile, N(%): 60 (34.3%): 3350 (64) ≥ 90th%ile, N(%): 5 (2.9%): 3001 (229) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.147; Adjusted, with CO: 0.186; Adjusted, with NO_2: 0.430; Adjusted, with SO_2: 0.155 2nd trimester <90th%ile, N(%): 59 (34.5%): 3335 (66) ≥ 90th%ile, N(%): 6 (3.5%): 3281 (249) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.833; Adjusted, with CO: 0.833; Adjusted, with NO_2: 0.778; Adjusted, with SO_2: 0.806 3rd trimester <90th%ile, N(%): 61 (34.1%): 3327 (65) ≥ 90th%ile, N(%): 6 (3.4%): 3227 (300) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.749; Adjusted, with CO: 0.980; Adjusted, with NO_2: 0.635; Adjusted, with SO_2: 0.687 **Genotype** MspI TC/CC 1st trimester <90th%ile, N(%): 98 (56.0%): 3193 (48) ≥ 90th%ile, N(%): 12 (6.9%): 2799 (169) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.033; Adjusted, with CO: 0.073; Adjusted, with NO_2: 0.150; Adjusted, with SO_2: 0.036 2nd trimester <90th%ile, N(%): 94 (55.0%): 3200 (52) ≥ 90th%ile, N(%): 12 (7.0%): 2933 (176) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.161; Adjusted, with CO: 0.172; Adjusted, with NO_2: 0.152; Adjusted, with SO_2: 0.158 3rd trimester <90th%ile, N(%): 101 (56.4%): 3165 (49) ≥ 90th%ile, N(%): 11 (6.2%): 3087 (147) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.626; Adjusted, with CO: 0.978; Adjusted, with NO_2: 0.551; Adjusted, with SO_2: 0.614 **Genotype** NcoI IleIle 1st trimester <90th%ile, N(%): 87 (49.7%): 3244 (52) ≥ 90th%ile, N(%): 7 (4.0%): 2983 (232) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.289; Adjusted, with CO: 0.344; Adjusted, with NO_2: 0.641; Adjusted, with SO_2: 0.293 2nd trimester <90th%ile, N(%): 82 (48.0%): 3243 (55)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			≥ 90th%ile, N(%): 11 (6.4%): 3185 (207) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.790; Adjusted, with CO: 0.783; Adjusted, with NO_2: 0.707; Adjusted, with SO_2: 0.733 3rd trimester <90th%ile, N(%): 90 (50.3%): 3239 (53) ≥ 90th%ile, N(%): 9 (5.0%): 2944 (198) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.161; Adjusted, with CO: 0.279; Adjusted, with NO_2: 0.134; Adjusted, with SO_2: 0.150 **Genotype** NcoI IleVal/ValVal 1st trimester <90th%ile, N(%): 71 (40.6%): 3262 (56) ≥ 90th%ile, N(%): 10 (5.7%): 2773 (171) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.009; Adjusted, with CO: 0.031; Adjusted, with NO_2: 0.058; Adjusted, with SO_2: 0.010 2nd trimester <90th%ile, N(%): 71 (41.5%): 3264 (61) ≥ 90th%ile, N(%): 7 (4.1%): 2862 (208) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.076; Adjusted, with CO: 0.093; Adjusted, with NO_2: 0.063; Adjusted, with SO_2: 0.061 3rd trimester <90th%ile, N(%): 72 (40.2%): 3207 (58) ≥ 90th%ile, N(%): 8 (4.5%): 3262 (180) p-Value for mean birthweight for ≥ 90th%ile PM_{10} vs. for <90th%ile PM_{10} Adjusted: 0.777; Adjusted, with CO: 0.607; Adjusted, with NO_2: 0.843; Adjusted, with SO_2: 0.791
Reference: Tsai et al. (2006) **Period of Study:** 1994-2000 **Location:** Kaohsiung, Taiwan	**Outcome:** postneonatal mortality **Age Groups:** infants more than 27 days and less than 1 year **Study Design:** Case-crossover study **N:** 1.46 million population of Taipei **Statistical Analyses:** Case-crossover technique **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package:** SAS, version 8.2	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 81.45 μg/m^3 **Percentiles: 25th:** 44.50 **50th(Median):** 79.20 **75th:** 111.50 **Range (Min, Max):** (20.50-232.00) **Monitoring Stations:** 6 **Copollutant:** SO_2 NO_2 CO O_3	**PM Increment:** 67.00 μg/m^3 **Effect Estimate [Lower CI, Upper CI]:** OR = 1.040 (0.340-3.177)
Reference: Wilhelm et al. (2005a) **Period of Study:** 1994-2000 **Location:** Los Angeles County, California, U.S.	**Outcome:** Term low birth weight (LBW) (<2500 g at ≥ 37 completed weeks gestation), Vaginal birth <37 completed weeks gestation **Age Groups:** LBW: ≥ 37 completed weeks Preterm births: <37 completed weeks **Study Design:** Cohort study **N:** For LBW: 136,134 For preterm birth: 106,483 **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, maternal race, maternal education, parity, interval since previous live birth, level of prenatal care,	**Pollutant:** PM_{10} **Averaging Time:** Entire pregnancy Trimesters of pregnancy Months of pregnancy 6 weeks before birth **Mean (SD):** First trimester: 42.2 Third trimester: 41.5 6 weeks before birth: 39.1 **Range (Min, Max):** First trimester: 26.3, 77.4 Third trimester: 25.7, 74.6	**PM Increment:** 1) 10 μg/m^3; 2) 3 levels: a) <25%ile (reference); b) 25%-75%ile; c) ≥ 75%ile **Incidence of LBW (third trimester exposure)** <17.1 μg/m^3: 2.0 (1.8, 2.2) 17.1 to <24.0 μg/m^3: 2.0 (1.9, 2.1) ≥ 24.0 μg/m^3: 2.2 (2.0, 2.4) **Incidence of preterm birth (first trimester exposure)** <32.9 μg/m^3: 8.7 (8.3, 9.2) 32.9 to <43.9 μg/m^3: 8.8 (8.5, 9.1) ≥ 43.9 μg/m^3: 8.6 (8.1, 9.0) **Incidence of preterm birth (6 weeks before birth exposure)**

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
	infant sex, previous LBW or preterm infant, birth season, other pollutants (CO, NO_2, O_3, PM_{10}) **Dose-response Investigated?** Yes **Statistical Package:** NR	6 weeks before birth: 13.0, 103.7 **Monitoring Stations:** Zip-code-level analysis: 8 Address-level analysis: 6 **Copollutant (correlation):** **First trimester:** PM_{10}-CO: r = 0.12 PM_{10}-NO_2: r = 0.29 PM_{10}-O_3: r = -0.01 PM_{10}-$PM_{2.5}$: r = 0.43 **Third trimester:** PM_{10}-CO: r = 0.32 PM_{10}-NO_2: r = 0.45 PM_{10}-O_3: r = -0.08 PM_{10}-$PM_{2.5}$: r = 0.52 **6 weeks before birth:** PM_{10}-CO: r = 0.36 PM_{10}-NO_2: r = 0.49 PM_{10}-O_3: r = -0.16 PM_{10}-$PM_{2.5}$: r = 0.60	<31.8 µg/m³: 8.8 (8.4, 9.3) 31.8 to <44.1 µg/m³: 8.6 (8.3, 8.9) ≥ 44.1 µg/m³: 8.8 (8.4, 9.2) **Outcome: LBW** **Exposure Period: Third trimester** **Address-level analysis:** Single-pollutant model: Distance ≤ 1 mile Per 10 µg/m³: 1.22 (1.05, 1.41); 33.4 to <44.7 µg/m³: 1.08 (0.76, 1.52); ≥ 44.7 µg/m³: 1.48 (1.00, 2.19) Multipollutant model: Distance ≤ 1 mile Per 10 µg/m³: 1.36 (1.12, 1.65); 33.4 to <44.7 µg/m³: 1.16 (0.77, 1.74); ≥ 44.7 µg/m³: 1.58 (0.95, 2.62) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m³: 0.98 (0.90, 1.06); 33.4 to <44.7 µg/m³: 0.95 (0.80, 1.13); ≥ 44.7 µg/m³: 0.96 (0.78, 1.18) Multipollutant model: 1 <distance ≤ 2 mile Per 10 µg/m³: 1.02 (0.92, 1.14); 33.4 to <44.7 µg/m³: 0.93 (0.77, 1.12); ≥ 44.7 µg/m³: 1.02 (0.79, 1.32) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m³: 1.03 (0.99, 1.08) 33.9 to <45.0 µg/m³: 1.04 (0.96, 1.14) ≥ 45.0 µg/m³: 1.08 (0.97, 1.20) Multipollutant model: 2 <distance ≤ 4 mile Per 10 µg/m³: 1.04 (0.98, 1.09); 33.9 to <45.0 µg/m³: 1.02 (0.92, 1.12); ≥ 45.0 µg/m³: 1.06 (0.93, 1.21) **Zip-code-level analysis** Single-pollutant model: Per 10 µg/m³: 1.03 (0.97, 1.09); 33.2 to <43.6 µg/m³: 0.98 (0.86, 1.11); ≥ 43.6 µg/m³: 1.03 (0.88, 1.21) Multipollutant model: Per 10 µg/m³: 1.07 (0.99, 1.15); 33.2 to <43.6 µg/m³: 0.97 (0.85, 1.12); ≥ 43.6 µg/m³: 1.09 (0.90, 1.31) **Outcome: LBW** **Exposure Period: Entire pregnancy period** **Address-level analysis:** Multipollutant model: Per 10 µg/m³: 1.24 (0.91, 1.70) **Outcome: Preterm Birth** **Exposure Period: First trimester of pregnancy** **Address-level analysis**: Single-pollutant model: Distance ≤ 1 mile Per 10 µg/m³: 1.00 (0.93, 1.09); 33.3 to <45.1 µg/m³: 1.07 (0.90, 1.26); ≥ 45.1 µg/m³: 1.12 (0.91, 1.38) Multipollutant model: Distance ≤ 1 mile Per 10 µg/m³: 1.00 (0.90, 1.12); 33.3 to <45.1 µg/m³: 1.12 (0.92, 1.36); ≥ 45.1 µg/m³: 1.17 (0.90, 1.50) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m³: 1.01 (0.97, 1.05); 33.7 to <45.3 µg/m³: 1.03 (0.95, 1.12); ≥ 45.3 µg/m³: 1.07 (0.97, 1.19) Multipollutant model:

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
			1 <distance ≤ 2 mile
			Per 10 μg/m^3: 1.04 (0.99, 1.10); 33.7 to <45.3 μg/m^3: 1.07 (0.98, 1.17); ≥ 45.3 μg/m^3: 1.13 (1.00, 1.27)
			Single-pollutant model:
			2 <distance ≤ 4 mile
			Per 10 μg/m^3: 1.01 (0.99, 1.03); 34.1 to <45.5 μg/m^3: 1.03 (0.99, 1.08); ≥ 45.5 μg/m^3: 1.02 (0.96, 1.07)
			Multipollutant model:
			2 <distance ≤ 4 mile
			Per 10 μg/m^3: 0.99 (0.97, 1.02); 34.1 to <45.5 μg/m^3: 0.99 (0.95, 1.04); ≥ 45.5 μg/m^3: 0.94 (0.89, 1.01)
			Zip-code-level analysis
			Single-pollutant model:
			Per 10 μg/m^3: 0.99 (0.96, 1.01); 33.3 to <44.2 μg/m^3: 1.01 (0.95, 1.08); ≥ 44.2 μg/m^3: 0.98 (0.90, 1.05)
			Multipollutant model:
			Per 10 μg/m^3: 0.99 (0.96, 1.03); 33.3 to <44.2 μg/m^3: 1.03 (0.97, 1.11); ≥ 44.2 μg/m^3: 1.01 (0.92, 1.11)
			Outcome: Preterm birth
			Exposure Period: 6 weeks before birth
			Address-level analysis:
			Single-pollutant model:
			Distance ≤ 1 mile
			Per 10 μg/m^3: 1.02 (0.95, 1.10); 32.5 to <44.8 μg/m^3: 1.09 (0.92, 1.29); ≥ 44.8 μg/m^3: 1.12 (0.92, 1.37)
			Multipollutant model:
			Distance ≤ 1 mile
			Per 10 μg/m^3: 1.06 (0.97, 1.16); 32.5 to <44.8 μg/m^3: 1.09 (0.90, 1.31); ≥ 44.8 μg/m^3: 1.17 (0.91, 1.49)
			Single-pollutant model:
			1 <distance ≤ 2 mile
			Per 10 μg/m^3: 1.00 (0.96, 1.03); 32.3 to <45.3 μg/m^3: 0.99 (0.91, 1.07); ≥ 45.3 μg/m^3: 0.99 (0.89, 1.10)
			Multipollutant model:
			1 <distance ≤ 2 mile
			Per 10 μg/m^3: 1.01 (0.97, 1.06)
			32.3 to <45.3 μg/m^3: 1.00 (0.92, 1.10)
			≥ 45.3 μg/m^3: 1.02 (0.91, 1.16)
			Single-pollutant model:
			2 <distance ≤ 4 mile
			Per 10 μg/m^3: 0.99 (0.98, 1.01); 33.1 to <45.3 μg/m^3: 1.00 (0.96, 1.05); ≥ 45.3 μg/m^3: 0.98 (0.93, 1.03)
			Multipollutant model:
			2 <distance ≤ 4 mile
			Per 10 μg/m^3: 1.00 (0.98, 1.02); 33.1 to <45.3 μg/m^3: 1.01 (0.96, 1.05); ≥ 45.3 μg/m^3: 0.98 (0.92, 1.04)
			Zip-code-level analysis
			Single-pollutant model:
			Per 10 μg/m^3: 1.02 (0.99, 1.04); 32.1 to <44.3 μg/m^3: 1.01 (0.95, 1.07); ≥ 44.3 μg/m^3: 1.04 (0.96, 1.12)
			Multipollutant model:
			Per 10 μg/m^3: 1.02 (0.99, 1.06); 32.1 to <44.3 μg/m^3: 1.02 (0.95, 1.09); ≥ 44.3 μg/m^3: 1.04 (0.95, 1.14)
			Notes: multipollutant model adds CO,NO_2, and O_3 in addition to the main pollutant of interest, PM_{10}.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Woodruff et al. (1997) **Period of Study:** 1989-1991 **Location:** 86 Metropolitan Statistical Areas in the US (counties with populations less than 100,000 were excluded)	**Outcome:** Postneonatal mortality (death of an infant between 1 month and 1 yr of age) 1) all postneonatal deaths 2) normal birth weight (NBW, ≥ 2500 g) SIDS deaths 3) NBW respiratory deaths 4) low birth weight (LBW) respiratory death Respiratory deaths: ICD9 codes 460-519; SIDS: ICD9 code 798.0 **Age Groups:** infants (1 month–1yr of age) **Study Design:** Cross-sectional **N:** 9,788,079 infants **Statistical Analyses:** Logistic regression **Covariates:** maternal education, maternal race, parental marital status, maternal smoking during pregnancy; avg temperature during the first 2 months of life; assessed race as an effect modifier (p-val for interaction terms >0.2) **Dose-response Investigated?** Yes **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Mean of 1st 2 months of life; analyzed as tertiles of exposure and as continuous exposure **Mean (SD):** 31.4 (7.8) **Range (Min, Max):** Overall: 11.9-68.8 Low category: <28.0 Medium category: 28.1-40.0 High category: >40.0 **Monitoring Stations:** NR	**PM Increment:** 10 µg/m³ (for continuous exposure analysis) **Adjusted ORs for cause-specific postneonatal mortality by pollution category (tertiles)** All causes Low: Ref Medium: 1.05 (1.01, 1.09) High: 1.10 (1.04, 1.16) SIDS, NBW: Low: Ref Medium: 1.09 (1.01, 1.17) High: 1.26 (1.14, 1.39) Respiratory death, NBW: Low: Ref Medium: 1.08 (0.87, 1.33) High: 1.40 (1.05, 1.85) Respiratory death, LBW: Low: Ref Medium: 0.93 (0.73, 1.18) High: 1.18 (0.86, 1.61) All other causes: Low: Ref Medium: 1.03 (0.97, 1.08) High: 0.97 (0.90, 1.04) **Adjusted ORs for a continuous 10 µg/m³ change in exposure** All causes: 1.04 (1.02, 1.07) SIDS, NBW: 1.12 (1.07, 1.17) Respiratory death, NBW: 1.20 (1.06, 1.36) Respiratory death, LBW: 1.05 (0.91, 1.22) All other causes: 1.00 (0.99, 1.00)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Woodruff et al (2008) **Period of Study:** 1999-2002 **Location:** US counties with >250,000 residents (96 counties)	**Outcome:** Postneonatal deaths Respiratory mortality (ICD10: J000-99, plus bronchopulmonary dysplasia [BPD] P27.1); SIDS (ICD10: R95); Ill-defined causes (R99); All other deaths evaluated as a control category **Age Groups:** Infants aged >28 days and <1 yr **Study Design:** Cross-sectional **N:** 3,583,495 births (6,639 postneonatal deaths) **Statistical Analyses:** Logistic GEE (exchangeable correlation structure) **Covariates:** maternal race/ethnicity, marital status, age, education, primiparity, county-level poverty and per capita income levels, year and month of birth dummy variables to account for time trend and seasonal effects, and region of the country; sensitivity analyses performed among only those mothers with smoking information (adjustment for smoking had no effect on the estimates) **Season:** Adjusted for year and month of birth dummy variables to account for time trend and seasonal effects **Dose-response Investigated?** Evaluated the appropriateness of a linear form from analysis based on quartiles of exposure and concluded that linear form was appropriate (data not shown) **Statistical Package:** NR	**Pollutant:** PM_{10} **Averaging Time:** Measured continuously for 24 h once every 6 days; exposure assigned by calculating avg concentration of pollutant during first 2 months of life **Median and IQR (25th-75th percentile):** Survivors: 28.9 (23.3-34.4) All causes of death: 29.1 (23.9-34.5) Respiratory: 29.8 (24.3-36.5) SIDS: 28.6 (23.5-33.8) SIDS + ill-defined: 28.8 (23.9-33.9) Other causes: 29.2 (23.9-34.5) **Percentiles:** see above **PM Component:** Not assessed, but controlled for region of the country to account for PM composition variation **Monitoring Stations:** NR **Copollutant (correlation):** PM_{10} $PM_{2.5}$ (r = 0.34) CO (r = 0.18) SO_2 (r = 0.00) O_3 (r = 0.20) **Notes:** Monthly averages calculated if there were at least 3 available measures for PM; Assigned exposures using the avg concentration of the county of residence	**PM Increment:** IQR (11 μg/m^3) **Effect Estimate [Lower CI, Upper CI]:** Adjusted ORs for single pollutant models All causes: 1.04 (0.99, 1.10) Respiratory: 1.18 (1.06, 1.31) SIDS: 1.02 (0.89, 1.16) Ill-defined + SIDS: 1.06 (0.97, 1.16) Other causes: 1.02 (0.96, 1.07) Adjusted ORs for multipollutant models (including CO, O_3, SO_2) Respiratory: 1.16 (1.04, 1.30) SIDS: 1.02 (0.90, 1.16) OR for deaths coded as BPD per increase in IQR: 1.19 (0.85, 1.65) OR for respiratory postneonatal death stratified by birth weight NBW only: 1.19 (1.05, 1.36) LBW only: 1.12 (0.95, 1.31) OR for respiratory deaths removing region of US as a confounding variable: 1.30 (1.04, 1.61) OR for respiratory deaths assessing exposure as quartiles Highest vs Lowest quartile: 1.31 (1.00, 1.71) OR for respiratory deaths among only those deaths that occurred during the first 90 days (most closely matched exposure metric of the avg over the first 2 months of life): 1.25 (1.06, 1.47)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Yang et al., (2003) **Period of Study:** January 1, 1995-December 31, 1997 **Location:** Kaohsiung, Taiwan	**Outcome:** full-term birth weight **Age Groups:** singleton live births with gestational ages between 20-50 weeks and mothers at all ages **Study Design:** retrospective cohort **N:** 13,396 **Statistical Analyses:** Multiple linear regression analysis **Covariates:** maternal age, season, marital status, maternal education, and infant gender **Season:** summer (May to October), winter (November through April) **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Percentiles: 33rd:** 1st trimester: 62.43 2nd trimester: 59.22 3rd trimester: 61.98 **67th:** 1st trimester: 100.44 2nd trimester: 98.64 3rd trimester: 100.91 **Monitoring Stations:** 6 **Copollutant (correlation):** PM_{10}, 1st-PM_{10}, 2nd: r = 0.15 PM_{10}, 1st-PM_{10}, 3rd: r = 0.73 PM_{10}, 1st-SO_2, 1st: r = 0.46 PM_{10}, 1st-SO_2, 2nd: r = 0.32 PM_{10}, 1st-SO_2, 3rd: r = 0.00 PM_{10}, 2nd –PM_{10}, 3rd: r = 0.15 SO_2, 1st-PM_{10},2nd: r = 0.11 SO_2, 1st-PM_{10},3rd: r = -0.06 SO_2, 2nd-PM_{10},2nd: r = 0.45 SO_2, 3rd-PM_{10},2rd: r = 0.31 SO_2, 3rd-PM_{10},3rd: r = 0.45 SO_2, 2nd-PM_{10},3rd: r = 0.08	**PM Increment:** 1 µg/m3 **Effect Estimate [Lower CI, Upper CI]:** **1st trimester** Low Crude RBW: comparison; Adjusted RBW: comparison Medium Crude RBW: 8.91; Adjusted RBW: 6.86 (-12.15-25.87) High Crude RBW: 26.60, p <0.05; Adjusted RBW: 25.59 (-0.50-51.68) Continuous Crude RBW: 0.41, p<0.05; Adjusted RBW: 0.52 (0.19-0.85), p<0.05 **2nd trimester** Low Crude RBW: comparison; Adjusted RBW: comparison Medium Crude RBW: 9.08; Adjusted RBW: 11.35 (-27.93-5.23) High Crude RBW: 8.27; Adjusted RBW: 11.38 (-27.69-4.93) Continuous Crude RBW: 0.11; Adjusted RBW: 0.16 (-0.36-0.04) **3rd trimester** Low Crude RBW: comparison; Adjusted RBW: comparison Medium Crude RBW: 18.71, p<0.05; Adjusted RBW: 18.97 (-38.18-0.24) High Crude RBW: 25.36, p<0.05; Adjusted RBW: 23.21 (-48.49-2.07) Continuous Crude RBW: 0.33, p<0.05; Adjusted RBW: 0.33 (-0.66-0.01)
Reference: Yang et al., 2005 **Period of Study:** 1994-2000 **Location:** Taipei, Taiwan	**Outcome:** postneonatal mortality **Age Groups:** infants more than 27 days and less than 1 year **Study Design:** Case-crossover study **N:** 2.64 million population of Taipei **Statistical Analyses:** Case-crossover technique **Covariates:** temperature, humidity **Dose-response Investigated?** No **Statistical Package:** SAS	**Pollutant:** PM_{10} **Averaging Time:** 24 h **Mean (SD):** 53.19 µg/m3 **Percentiles: 25th:** 34.70 **50th(Median):** 46.71 **75th:** 64.91 **Range (Min, Max):** (14.44-234.91) **Monitoring Stations:** 6 **Copollutant:** SO_2 NO_2 CO O_3	**PM Increment:** 30.21 µg/m3 (IQR) **Effect Estimate [Lower CI, Upper CI]:** OR = 1.031 (0.652-1.630) **Notes:** Adjusted for temperature and humidity

Table E-31. Long-term exposure to $PM_{10-2.5}$ and reproductive outcomes.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Parker and Woodruff (2008)	**Outcome:** Low birth weight **Study Design:** cohort **N:** 785,965 Singleton births delivered at 40 weeks gestation **Statistical Analyses:** GEE regression models **Covariates:** race/ethnicity, parity, maternal age **Season:** season of delivery **Dose-response Investigated?** **Statistical Package:** SUDAAN	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 9-months **Mean (SD):** 13.2 25th: 9.8 75th: 17.5 **Copollutant:** SO_2 NO_2 CO O_3	**PM Increment:** 10 µg/m^3 **Change in Birthweight:** Unadjusted: -17.5 (-22.8 to -12.3) Adjusted for maternal factors: -13.0 (-18.3 to -7.6) **Stratified by trimester:** First: -4.1 (-7.5 to -0.8) Second: -5.9 (-8.9 to -2.9) Third: -6.3 (-9.2 to -3.5) **Stratified by regioN:** Industrial Midwest: -3.5 (-14.0, 6.9) Northeast: -27.8 (-42.9 to -12.7) Northwest: -43.1 (-58.6 to -27.6) Southern CA: -13.5 (-22.4 to -4.5) Southeast: -10.2 (-19.8 to -0.5) Southwest: 4.1 (-7.8, 16.0) Upper Midwest: -24.0 (-42.2 to -5.9) **Multipollutant models:** $PM_{10-2.5}$ + $PM_{2.5}$: -13.0 (-18.3 to -7.6) $PM_{10-2.5}$+ $PM_{2.5}$ +SO_2+CO+NO_2+O_3: -14.5 (-23.4 to -5.7)

Table E-32. Long-term exposure to $PM_{2.5}$ (including PM components/sources) and reproductive outcomes.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Basu et al., (2004) **Period of Study:** 2000 **Location:** California	**Outcome:** Birth weight (continuous) **Age Groups:** pregnant women 20-30 years **Study Design:** Retrospective cohort **N:** 16693 pregnant women **Statistical Analyses:** Linear regression **Covariates:** None (the population was restricted) **Season:** All **Dose-response Investigated?** No **Statistical Package:** SAS **Lags Considered:** the 9- month period of gestation for each mother	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** Non-Hispanic whites Monitor 0-1 mile: 14.5 (5.3) Avg monitors 0-5 mile: 15.8 (4.9) County monitors: 15.6 (3.7) Hispanic Monitor 0-1 mile: 16.4 (5.4) Avg monitors 0-5 mile: 18.2 (5.0) County monitors: 16.9 (3.3) µg/m^3 **Range (Min, Max):** Non-Hispanic whites 0-1 mile: (4.4, 32.4) 0-5 mile: (4.4, 34.1) County: (4.6, 26.3) Hispanic 0-1 mile: (5.9, 33.7) 0-5 mile: (4.6, 33.9) County: (4.6, 26.3) **Monitoring Stations**: 84, all in urban areas	**PM Increment:** 1 µg/m^3 **Change in mean for unit increase in avg PM during pregnancy [Lower CI, Upper CI]:** Non-Hispanic whites Avg monitors 0-5 mile: -1.52 [-3.52, 0.48] County monitors: -4.04 [-6.71 to -1.37] Hispanic Avg monitors 0-5 mile: -2.49 [-4.53 to -0.45] County monitors: -4.35 [-7.47 to -1.23] In subset of mothers who had monitor within 1 mile of residence (n = 796 non-Hisp, 787 Hisp): Non-Hispanic whites Monitor 0-1 mile: -6.37 [-13.05, 0.31] Avg monitors 0-5 mile: -5.36 [NR] County monitors: -9.44 [-17.97 to -0.91] Hispanic: Monitor 0-1 mile: -1.37 [-7.31, 4.57] Avg monitors 0-5 mile: -0.77 [NR] County monitors: -4.06 [-12.29, 4.17] **Notes:** Sensitivity analyses testing for overly influential counties which could be driving results yielded similar estimates. Exposure assignment using monitors within 1 mile of the maternal address was limited because so few mothers lived within 1 mile of a station

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Bell at al., (2007) **Period of Study:** 1999-2002 **Location:** Connecticut–Fairfield, Hartford, New Haven, New London, Windham Massachusetts–Barnstable, Berkshire, Bristol, Essex, Hampden, Middlesex, Norfolk, Plymouth, Suffolk, Worcester	**Outcome:** Low birth weight **Age Groups:** Neonates **Study Design:** Cross-sectional **N:** 358,504 deaths **Statistical Analyses:** Multiple logistic and linear regressions **Covariates:** Child's sex, mother's education, tobacco use, mother's marital status, mother's race, time prenatal care began, mother's age, birth order, gestation length **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 11.9 (1.6) **Monitoring Stations:** NR **Copollutant:** NO_2, CO, SO_2 **Gestation exposure correlation:** PM_{10} (r = 0.77), NO_2 (r = 0.64)	**PM Increment:** 2.2 µg/m^3 (IQR) **Difference in birth weight [Lower CI, Upper CI]; lag:** -14.7 [-17.1 to -12.3] **Difference in birth weight by race of mother [Lower CI, Upper CI]; lag:** Black: -22.6 [-29.3 to -15.9] White: -14.7 [-17.3 to -12.0] **Range among trimester models for change in birth weight per IQR increase (min, max); trimester:** -7.2 to -5.4; 2nd -9.0 to -7.0; 3rd **OR Estimate for birth weight <2500 g [Lower CI, Upper CI]; lag:** 1.054 [1.022, 1.087] **Notes**: Analyses using first births alone yielded similar results. Two pollutant models for uncorrelated pollutants were analyzed, but not presented quatitatively.
Reference: Brauer et al. (2008) **Period of Study:** 1999-2002 **Location:** Vancouver, BC	**Outcome:** Fetal growth restriction, SGA, LBW **Age Groups: Study Design:** Cohort **N:** 70,249 **Statistical Analyses:** Linear regression **Covariates:** Sex, parity, month and year of birth, maternal age and smoking, neighborhood level income and education **Statistical Package:** SAS	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** 5.3 **Range (Min, Max):** 0.3, 37.0 **Monitoring Stations:** 7 **Copollutant (correlation):** NO NO_2 CO SO_2 O_3	**PM Increment:** 1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** SGA: 1.02 (0.98 1.05) LBW: 0.98 (0.92, 1.05) Preterm (<37 weeks): 1.06 (1.01, 1.11) Preterm (<35 weeks): 1.12 (1.02, 1.24) Preterm (<30 weeks): 1.13 (0.92, 1.39)
Reference: Dales et al. (2004) **Period of Study:** Jan 1, 1984–Dec 31, 1999 **Location:** Canada (12 cities)	**Outcome:** SIDS (a sudden, unexplained death of a child <1 year of age for which a clinical investigation and autopsy fail to reveal a cause of death) **Age Groups:** Infants <1 yr **Study Design:** Time-series **N:** Total population of 12 cities: 10,310,309; 1556 cases of SIDS over study period **Statistical Analyses:** Random-effects regression model for count data (a linear association between air pollution and the incidence of SIDS was assumed on the logarithmic scale) **Covariates:** weather factors (daily mean temp, daily mean relative humidity, maximum change in barometric pressure, all measured on the day of death), length of time-period adjustment, seasonal indicator variables, and size-fractionated PM **Season:** Used piece-wise constant functions in time that varied by 3, 6, or 12 months **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-hs (PM measures every 6 days; gaseous pollutants every day) **Mean (IQR):** PM_{10}: 23.43 (15.56) $PM_{2.5}$: 12.27 (8.98) $PM_{2.5-10}$: 11.28 (8.76) **Range (Min, Max):** IQR presented above **Monitoring Stations:** When data were available from more than one monitoring site, they were averaged **Copollutant:** $PM_{2.5}$ PM_{10} CO NO_2 O_3 SO_2	**Notes:** The abstract reports no association between increased daily rates of SIDS and fine particles measured every sixth day. However, no effect estimates presented for PM (only gaseous pollutants adjusted for PM).

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Huynh et al. (2006) **Period of Study:** 1999-2000 **Location:** California	**Outcome:** preterm birth **Age Groups:** infants delivered at >39 weeks gestation; maternal all ages **Study Design:** matched case-control **N:** 42,692 infants **Statistical Analyses:** Conditional logistic regression **Covariates:** maternal race/ethnicity, age, parity, marital status, and education **Dose-response Investigated?** Yes **Statistical Package:** SAS version 8.0	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** Last 2 weeks of gestation/ Preterm births: 18.6 (10.3) Last 2 weeks of gestation/ Term births: 17.9 (10.3) First month of pregnancy/ Preterm births: 18.8 (7.0) First month of pregnancy/ Term births: 18.1 (6.9) Total gestation/ Preterm births: 18.0 (5.2) Total gestation/ Term births: 17.5 (5.2) **Monitoring Stations:** NR **Copollutant (correlation):** $PM_{2.5}$ Last 2 weeks of gestation–CO Last 2 weeks of gestation: r = 0.34 $PM_{2.5}$ Last 2 weeks of gestation–CO 1st month of pregnancy: r = -0.08 $PM_{2.5}$ Last 2 weeks of gestation–CO Total gestation: r = 0.05 $PM_{2.5}$ 1st month of pregnancy–CO Last 2 weeks of gestation: r = -0.13 $PM_{2.5}$ 1st month of pregnancy–CO 1st month of pregnancy: r = 0.44 $PM_{2.5}$ 1st month of pregnancy–CO Total gestation: r = 0.18 $PM_{2.5}$ Total gestation–CO Last 2 weeks of gestation: r = 0.13 $PM_{2.5}$ Total gestation-CO 1st month of pregnancy: r = 0.08 $PM_{2.5}$ Total gestation–CO Total gestation: r = 0.32 **Notes:** Correlations also available between $PM_{2.5}$ at different stages of gestation in Table 2.	**PM Increment:** <13.4 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Total Gestation Unadjusted: OR = 1.00 (reference); Adjusted[a]: OR = 1.00 (reference); Adjusted[b]: OR = 1.00 (reference) **PM Increment:** 13.4-17.7 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Unadjusted: OR = 1.02 (0.96-1.09); Adjusted[a]: OR = 0.99 (0.92-1.06); Adjusted[b]: OR = 1.00 (0.93-1.07) **PM Increment:** 17.7-22.1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Unadjusted: OR = 1.26 (1.18-1.34); Adjusted[a]: OR = 1.13 (1.06-1.21); Adjusted[b]: OR = 1.14 (1.07-1.23) **PM Increment:** >22.1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Unadjusted: OR = 1.30 (1.22-1.39); Adjusted[a]: OR = 1.15 (1.07-1.23); Adjusted[b]: OR = 1.15 (1.07-1.24) **PM Increment:** <12.5 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** First month of gestation Unadjusted: OR = 1.00 (reference); Adjusted[a]: OR = 1.00 (reference); Adjusted[b]: OR = 1.00 (reference) **PM Increment:** 12.5-18.2 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Unadjusted: OR = 1.13 (1.06-1.21); Adjusted[a]: OR = 1.08 (1.01-1.16); Adjusted[b]: OR = 1.09 (1.01-1.17) **PM Increment:** 18.2-23.0 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Unadjusted: OR = 1.25 (1.17-1.34); Adjusted[a]: OR = 1.14 (1.06-1.22); Adjusted[b]: OR = 1.14 (1.06-1.22) **PM Increment:** >23.0 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Unadjusted: OR = 1.38 (1.29-1.49); Adjusted[a]: OR = 1.21 (1.12-1.30); Adjusted[b]: OR = 1.21 (1.12-1.30) **PM Increment:** <10.2 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Last 2 weeks of gestation Unadjusted: OR = 1.00 (reference); Adjusted[a]: OR = 1.00 (reference); Adjusted[b]: OR = 1.00 (reference) **PM Increment:** 10.2-15.6 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Unadjusted: OR = 1.16 (1.09-1.24); Adjusted[a]: OR = 1.11 (1.04-1.19); Adjusted[b]: OR = 1.11 (1.04-1.19) **PM Increment:** 15.6-23.3 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Unadjusted: OR = 1.27 (1.19-1.36); Adjusted[a]: OR = 1.18 (1.10-1.26); Adjusted[b]: OR = 1.18 (1.10-1.19) **PM Increment:** >23.3 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** Unadjusted: OR = 1.30 (1.20-1.39); Adjusted[a]: OR = 1.17 (1.08-1.26); Adjusted[b]: OR = 1.17 (1.09-1.27) Continuous measures of exposure Total gestation[c] Unadjusted: OR = 1.27 (1.26-1.27); Adjusted[a]: OR = 1.15 (1.15-1.16); Adjusted[b]: OR = 1.15 (1.15-1.16) First month of gestation[c] Unadjusted: OR = 1.22 (1.21-1.22); Adjusted[a]: OR = 1.13 (1.13-1.13); Adjusted[b]: OR = 1.13 (1.12-1.13) Last 2 weeks of gestation[c] Unadjusted: OR = 1.09 (1.09-1.10); Adjusted[a]: OR = 1.06 (1.05-1.06); Adjusted[b]: OR = 1.06 (1.05-1.06) **Notes:** Adjusted[a] for maternal age, maternal race/ethnicity, maternal education, marital status and parity. Adjusted[b] for variables in [a] and CO. [c]Per 10 µg/m^3 increase.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Jalaludin et al. (2007) **Period of Study:** 1998-2000 **Location:** Sydney, Australia	**Outcome (ICD9 and ICD10):** Gestational age (categorized: preterm birth: <37 weeks; term birth: ≥ 37 weeks but <42 weeks) **Age Groups:** infants **Study Design:** Cross-sectional **N:** 123,840 singleton births of >20 weeks gestation **Statistical Analyses:** Logistic regression **Covariates:** sex of child, maternal age, maternal smoking during pregnancy, gestational age at first antenatal visit, whether mother identifies as being Aboriginal or Torres Strait Islander, whether first pregnancy, season of conception, SES, (temperature and relative humidity were not significant in single variable models and therefore, were not included) **Season:** examined as covariate and effect modifier **Dose-response Investigated?** No **Statistical Package:** SAS v8	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h averages used to calculate the mean concentration over the first trimester, the 3 months preceding birth, the first month after the estimated date of conception, and the month prior to delivery **Mean (SD):** (24 hr averages) All year: 9.0 (3.94) summer: 8.7 (4.19) Autumn: 9.4 (3.61) Winter: 9.5 (4.22) Spring: 8.5 (3.61) **Monitoring Stations:** 14 stations within the Sydney metropolitan area (levels averaged to provide one estimate for the entire study area) **Copollutant (correlation):** PM_{10} (r = 0.83) CO (r = 0.53) NO_2 (r = 0.65) O_3 (r = 0.34) SO_2 (r = 0.43) **Notes:** Correlations between monitoring stations measuring $PM_{2.5}$ ranged from 0.66 to 0.93	**PM Increment:** 1 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** ORs (air pollutant concentration during the 1st trimester and preterm birth by season) Autumn: 1.080 (0.912, 1.281) Winter: 1.426 (1.264, 1.608) Spring: 1.156 (0.972, 1.375) summer: 0.879 (0.839, 0.922) ORs (air pollutant concentrations during different exposure periods and preterm birth; for all of Sydney and among only those residing within 5 km of a monitoring station) 1 month preceding birth Sydney: 0.984 (0.962, 1.008) 5km: 1.042 (0.997, 1.089) 3 months preceding birth Sydney: 0.981 (0.952, 1.011) 5km: 1.111 (1.037, 1.189) 1st month of gestation Sydney: 0.981 (0.962, 1.000) 5km: 1.032 (0.897, 1.188) 1st trimester Sydney: 0.978 (0.950, 1.007) 5km: 0.991 (0.929, 1.057) **Notes:** Authors note that effect of $PM_{2.5}$ on preterm birth for infants conceived during the winter did not remain in 2 pollutant models (ORs between 0.97 and 1.03)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Jedrychowski, et al., (2007) **Period of Study:** Jan 2001-Feb 2004 **Location:** Krakow, Poland	**Outcome:** Birth weight (grams), birth length (cm) **Age Groups:** pregnant women 18-35 years **Study Design:** Prospective cohort **N:** 493 women **Statistical Analyses:** Linear regression **Covariates:** Environmental tobacco smoke (# cigarettes smoked daily in presence of pregnant woman), season of birth, size of mother, parity, gestational age, gender of child, vitamin A intake **Season:** All **Dose-response Investigated?** Yes **Statistical Package:** NR **Lags Considered:** Two consecutive days in the second trimester	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48 h period **Percentiles: 50th(Median):** 35.3 **Range (Min, Max):** 10.3, 294.9 **Monitoring Stations:** No stations, personal monitoring **Notes:** PM measured during a two day period in the second trimester by Personal Environmental Monitoring Sampler (PEMS)	**PM Increment:** in 1 μg/m³ and tertiles T1: <27.0 μg/m³ T2: 27.0-46.2 μg/m³ T3: ≥ 46.2 μg/m³ **Mean [Lower CI, Upper CI]:** Birth weight (g) For ln unit PM: β = -172.39 (p = 0.02) Tertiles: T1: ref T2: β = -16.510 [-94.630, 61.610] T3: β = -109.956 [-196.649 to -23.263] In low Vitamin A group (<1,378 μg) T1: ref T2: β = -68.354 [-165.643, 28.935] T3: β = -185.070 [-293.393 to -76.747] In high Vitamin A group (>1,378 μg) T1: ref T2: β = 64.262 [-70.464, 198.988] T3: β = 38.593 [-109.853, 187.039] Birth length (cm) For ln unit PM: β = -1.39 (p = 0.00) Tertiles: T1: ref T2: β = -0.288 [-0.790, 0.214] T3: β = -0.810 [-1.367 to -0.253] In low Vitamin A group (<1,378 μg) T1: ref T2: β = -0.514 [-1.114, 0.086] T3: β = -1.100 [-1.768 to -0.432] In high Vitamin A group (>1,378 μg) T1: ref T2: β = 0.039 [-0.896, 0.974] T3: β = -0.301 [-1.326, 0.724]
Reference: Lipfert et al. (2000) **Period of Study:** 1990 **Location:** U.S.	**Outcome (ICD9 and ICD10):** Infant mortality; including respiratory mortality (traditional definition, ICD9 460-519), expanded definition (adds ICD9 769 and 770) **Age Groups:** Infants **Study Design:** Cross-sectional **N:** 2,413,762 infants in 180 counties (Ns differ for various models) **Statistical Analyses:** Logistic regression **Covariates:** mother's smoking, education, marital status, and race; month of birth; and county avg heating degree days **Dose-response Investigated?** NR **Statistical Package:** NR	**Pollutant:** SO_4^{2-}/ $NSPM_{10}$ (regressed jointly) **Averaging Time:** Yearly avg used **Mean (SD):** 33.1 (9.17) (based on 180 counties) **Range (Min, Max):** (16.9, 59) **Monitoring Stations:** NR **Copollutant:** PM_{10} $NSPM_{10}$ CO SO_2 **Notes:** TSP-based sulfate was adjusted for compatibility with the PM_{10}-based data	**PM Increment:** NR (present regression coefficients) **Effect Estimate [Lower CI, Upper CI]:** Presented regression coefficients (standard errors); (3 PM exposures regressed jointly); bold = p <0.05 Cause of death: All; Birth weight: All SO_4^{2-}: -0.0002 (0.0061); $NSPM_{10}$: 0.0115 (0.0014) Cause of death: All; Birth weight: LBW SO_4^{2-}: 0.0265 (0.0080); $NSPM_{10}$: 0.0086 (0.0020) Cause of death: All; Birth weight: normal SO_4^{2-}: -0.0488 (0.0098); $NSPM_{10}$: 0.0096 (0.0024) Cause of death: All neonatal; Birth weight: All SO_4^{2-}: 0.0267 (0.0076); $NSPM_{10}$: 0.0126 (0.0018) Cause of death: All neonatal; Birth weight: LBW SO_4^{2-}: 0.0388 (0.0088); $NSPM_{10}$: 0.0093 (0.0022) Cause of death: All neonatal; Birth wt: normal SO_4^{2-}: -0.0334 (0.0169); $NSPM_{10}$: 0.0125 (0.0040) Cause of death: All postneonatal; Birth wt: All PM_{10}: 0.0091 (0.0024); SO_4^{2-}: -0.0474 (0.0100); $NSPM_{10}$: 0.0096 (0.0024) Cause of death: All postneonatal; Birth wt: LBW SO_4^{2-}: -0.0247 (0.0173); $NSPM_{10}$: 0.0101 (0.0042) Cause of death: All postneonatal; Birth wt: normal SO_4^{2-}: -0.0569 (0.0121); $NSPM_{10}$: 0.0080 (0.0029) Cause of death: SIDS; Birth weight: All

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
			SO_4^{2-}: -0.1078 (0.0151); $NSPM_{10}$: 0.0149 (0.0037) Cause of death: SIDS; Birth weight: LBW SO_4^{2-}: -0.1378 (0.0337); $NSPM_{10}$: 0.0146 (0.0085) Cause of death: SIDS; Birth weight: normal PM_{10}: 0.0137 (0.0042); SO_4^{2-}: -0.0995 (0.0168); $NSPM_{10}$: 0.0147 (0.0041) Cause of death: All respiratory (ICD9: 460-519, 769, 770) Birth weight: All SO_4^{2-}: 0.0706 (0.0146); $NSPM_{10}$: 0.0166 (0.0034) Cause of death: All respiratory (ICD9: 460-519, 769, 770) Birth weight: LBW SO_4^{2-}: 0.0821 (0.0158); $NSPM_{10}$: 0.0139 (0.0038) Cause of death: All respiratory (ICD9: 460-519, 769, 770) Birth weight: normal PM_{10}: 0.0177 (0.0091); SO_4^{2-}: 0.0001 (0.0392); $NSPM_{10}$: 0.0118 (0.0090) Cause of death: Respiratory disease (ICD9: 460-519) Birth weight: All PM_{10}: 0.0133 (0.0089); SO_4^{2-}: 0.0093 (0.0384); $NSPM_{10}$: 0.0134 (0.0089) Cause of death: Respiratory disease (ICD9: 460-519) Birth weight: LBW PM_{10}: 0.0092 (0.0137); SO_4^{2-}: 0.0434 (0.0580); $NSPM_{10}$: 0.0089 (0.0138) Cause of death: Respiratory disease (ICD9: 460-519) Birth weight: normal SO_4^{2-}: -0.0177 (0.0509); $NSPM_{10}$: 0.0128 (0.0119) Associations with SIDS by smoking status Smoking status: Yes; Birth weight: Normal SO_4^{2-}: -0.0722 (0.0284); $NSPM_{10}$: 0.0206 (0.0071) Smoking status: No; Birth weight: Normal SO_4^{2-}: -0.114 (0.021); $NSPM_{10}$: 0.0117 (0.005) Smoking status: Yes; Birth weight: LBW SO_4^{2-}: -0.0958 (0.0483); $NSPM_{10}$: 0.0345 (0.0125) Smoking status: No; Birth weight: LBW SO_4^{2-}: -0.0172 (0.047); $NSPM_{10}$: -0.0007 (0.012) Mean risks (95%CI) between postneonatal SIDS among normal birth weight babies; pollutants regressed one at a time SO_4^{2-}: 0.43 (0.37, 0.51); $NSPM_{10}$: 1.33 (1.18, 1.50)
Reference: Liu et al., (2007) **Period of Study:** 1985-2000 **Location:** 3 Canadian cities: Calgary, Edmonton, and Montreal	**Outcome:** intrauterine growth restriction (IUGR) **Age Groups:** singleton term live births **Study Design:** retrospective cohort **N:** 386,202 singleton live births **Statistical Analyses:** Multiple logistic regression **Covariates:** maternal age, parity, infant gender, season, and city of residence **Season:** All seasons **Dose-response Investigated?** No **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 12.2 **Percentiles: 25th:** 6.3 **50th(Median):** 9.7 **75th:** 15 **PM Component:** metals and organic matter such as polycyclic aromatic hydrocarbons **Monitoring Stations:** Calgary (4), Edmonton (2), and Montreal (8) **Copollutant (correlation):** SO_2: r = 0.44, p<0.0001 NO_2: r = 0.41, p<0.0001 CO: r = 0.31, p<0.0001 O_3: r = -0.14, p<0.0001	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** 1st trimester OR = 1.07 (1.03-1.10) 2nd trimester OR = 1.06 (1.03-1.10) 3rd trimester OR = 1.06 (1.03-1.10)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Loomis et al. (1999) **Period of Study:** Jan 1, 1993–Jul 31,1995 **Location:** Mexico City (southwestern section)	**Outcome (ICD9 and ICD10):** Infant mortality (daily counts of deaths); All ICD9 codes, excluding accidents, poisoning, and violence (ICD9 ≥800) **Age Groups:** Children <1 yr of age **Study Design:** Time-series **N:** 942 deaths (days were the unit of observation) **Statistical Analyses:** Poisson regression (generalized additive model) **Covariates:** Final models controlled for mean temp of 3 days before death and nonparametrically smoothed periodic cycles **Season:** Yes (considered) **Dose-response Investigated?** Loess smoother **Statistical Package:** NR **Lags Considered:** 0-5 (also considered lags with avg exposure levels during "windows" of 2 to 4 days)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h **Mean (SD):** 27.4 (10.5) **Percentiles:** Lower quartile: 20 Median: 26 Upper quartile: 34 **Range (Min, Max):** 4, 85 **Monitoring Stations:** one **Copollutant:** O_3 NO_2 NO NO_X SO_2 **Notes:** Pearson correlation coefficients ranging from 0.52 to 0.71	**PM Increment:** 10 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** %Change in infant mortality Lags 0-5 (single day) presented in Figure 1: Lag0,1,2: No association (results not presented) Lag3: 4.8 (0.97, 8.61) Lag4: 4.2 (0.37, 7.93) %Change in mortality when avg exposure levels during "windows" of 2 to 4 days were considered Two Days: No lag: -1.36 (-5.51, 2.8) Lag1: -0.95 (-5.10, 3.20) Lag2: 2.78 (-1.33, 6.89) Lag3: 4.93 (0.86, 9.01) Three Days: No lag: -0.81 (-5.29, 3.67) Lag1: 1.99 (-2.46, 6.45) Lag2: 4.54 (0.12, 8.96) Lag3: 6.87 (2.48, 11.26) Four Days: No lag: 1.95 (-2.76, 6.66) Lag1: 3.74 (-0.95, 8.42) Lag2: 5.87 (1.21, 10.53) Multipollutant models (3-day mean w/ 3-day lag) 1 pollutant model: 6.87 (2.48, 11.26) 2 pollutant models: w/ O_3: 6.24 (1.35, 11.14) w/ NO_2: 5.91 (-0.76, 12.59) 3 pollutant model (w/ O_3 and NO_2): 6.30 (-0.54, 13.15)
Reference: Mannes et al. (2005) **Period of Study:** January 1, 1998-December 31, 2000 **Location:** metropolitan Sydney, Australia	**Outcome:** risk of small for gestational age (SGA) and birth weight **Age Groups:** all singleton births >20 weeks and ≥ 400 grams birth weight and maternal all ages **Study Design:** cohort **N:** 138,056 singleton births **Statistical Analyses:** Logistic regression models **Covariates:** sex of child, maternal age, gestational age, maternal smoking, gestational age at first antenatal visit, maternal indigenous status, whether first pregnancy, season of birth, and socioeconomic status (SES) **Season:** All seasons **Dose-response Investigated?** No **Statistical Package:** SAS System for Windows v8.02	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24 h **Mean (SD):** 9.4 (5.1) **Percentiles: 25th:** 6.5 **50th(Median):** 8.4 **75th:** 11.2 **Range (Min, Max):** (2.4- 82.1) **Monitoring Stations:** up to 14 **Copollutant (correlation):** CO: r = 0.53 NO_2: r = 0.66 O_3: r = 0.36 PM_{10}: r = 0.81	**PM Increment:** 1 µg/m^3 Risk of SGA All births One month before birth: OR = 1.01 (0.99-1.03) Third trimester: OR = 0.99 (0.97-1.02) Second trimester: OR = 1.03 (1.01-1.05) First trimester: OR = 0.99 (0.97-1.01) 5 km births One month before birth: OR = 1.01 (0.97-1.04) Third trimester: OR = 1.00 (0.95-1.05) Second trimester: OR = 1.00 (0.96-1.05) First trimester: OR = 0.99 (0.94-1.04) Change in birth weight All births One month before birth: ß = -2.48 (-4.58- -0.38) Third trimester: ß = -0.98 (-3.74–1.78) Second trimester: ß = -4.10 (-6.79- -1.41) First trimester: ß = 0.36 (-2.29- 3.01) 5 km births One month before birth: ß = -2.70 (-6.80- 1.40) Third trimester: ß = -2.83 (-9.00-3.34) Second trimester: ß = 1.54 (-4.59-7.67) First trimester: ß = 1.89 (-1.99-5.77)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Parker et al. (2005) **Period of Study:** 1999-2000 **Location:** California	**Outcome:** small for gestational age (SGA) and birth weight **Age Groups:** infants delivered at 40 weeks gestation; maternal all ages **Study Design:** cohort **N:** 18,247 singleton births **Statistical Analyses:** Linear regression models **Covariates:** maternal race, maternal Hispanic origin, marital status, parity, maternal education, and maternal age **Season:** season of delivery **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR (measurement taken every 6 days) **Mean (SD):** 15.42 (5.08) **PM Component:** metals, polycyclic aromatic hydrocarbons **Monitoring Stations:** 40 **Copollutant (correlation):** $PM_{2.5}$-CO: r = 0.60 **Notes:** Mean calculated for 9-month exposure. The following means (SDs) are calculated for trimester: First: 15.70 (6.26) Second: 15.40 (6.53) Third: 14.29 (6.35)	**PM Increment:** <11.9 µg/m3l Referent **PM Increment:** 11.9-13.9 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** First Trimester Birth weight: ß = -5.7 (-27.9-16.5) SGA: OR = 1.02 (0.84-1.23) Second Trimester Birth weight: ß = 11.3 (-12.2-34.9) SGA: OR = 0.89 (0.73-1.09) Third Trimester Birth weight: ß = 8.3 (-13.1-29.8) SGA: OR = 1.00 (0.83-1.19) **PM Increment:** 13.9-18.4 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** First Trimester Birth weight: ß = -2.5 (-24.5-19.5) SGA: OR = 1.12 (0.93-1.34) Second Trimester Birth weight: ß = -17.2 (-39.4-4.9) SGA: OR = 1.05 (0.88-1.26) Third Trimester Birth weight: ß = -8.1 (-30.2-13.9) SGA: OR = 0.98 (0.82-1.18) **PM Increment:** >18.4 µg/m^3 **Effect Estimate [Lower CI, Upper CI]:** First Trimester Birth weight: ß = -35.8 (-58.4--13.3) SGA: OR = 1.26 (1.04-1.51) Second Trimester Birth weight: ß = -46.6 (-68.6- -24.6) SGA: OR = 1.24 (1.04-1.49) Third Trimester Birth weight: ß = -31.6 (-52.0- -11.1) SGA: OR = 1.21 (1.02-1.43)
Reference: Parker and Woodruff (2008) **Period of Study:** 2001-2003 **Location:** US	**Outcome:** Low birth weight **Study Design:** cohort **N:** 785,965 Singleton births delivered at 40 weeks gestation **Statistical Analyses:** GEE regression models **Covariates:** race/ethnicity, parity, maternal age **Season:** season of delivery **Statistical Package:** SUDAAN	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 9-months **Mean (SD):** 14.5 25th: 12.1 75th: 17.6 **Copollutant (correlation):** SO_2, NO_2 CO O_3	**PM Increment:** 10 µg/m^3 **Change in Birthweight:** Unadjusted: 19.4 (9.8, 29.0) Adjusted for maternal factors: 18.4 (9.2, 27.7) **Stratified by regioN:** Industrial Midwest: -15.3 (-43.4, 12.9) Northeast: -9.8 (-11.9, 26.6) Northwest: 27.5 (5.5, 49.4) Southern CA: 5.5 (-9.6, 20.5) Southeast: 7.3 (-11.9, 26.6) Southwest: 72.3 (34.0, 110.5) Upper Midwest: -0.7 (-62.0, 60.6) **Multipollutant models:** $PM_{2.5}$ +$PM_{10-2.5}$: 14.2 (4.3, 24.1) $PM_{2.5}$ +$PM_{10-2.5}$+SO_2+CO+NO_2+O_3: 28.6 (14.2, 43.0)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ritz et al. (2007) **Period of Study:** Jan 1, 2003–Dec 31, 2003 **Location:** Los Angeles, California	**Outcome:** Preterm births (infants delivered before 37 weeks) **Age Groups:** Births **Study Design:** Case-control nested within a birth cohort (cases and controls matched on zip code and birth month) Phase 1: cross-sectional including all birth cohort Phase 2: nested case-control of survey respondents **N:** Phase 1: Birth cohort consisted of 58,316 eligible births. Phase II: 2,543 **Statistical Analyses:** Logistic regression **Covariates:** Birth certificant information: maternal age, race/ethnicity, parity, education, season of birth; survey information: maternal smoking, alcohol consumption, living with a smoker, and marital status during pregnancy; income (imputed); occupation and pregnancy weight gain considered by not included in final models **Season:** Yes **Dose-response Investigated?** Yes, examined categories of exposure **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** the entire pregnancy, the first trimester, and the last 6 weeks before delivery; only reported first trimester exposures for PM **Range (Min, Max):** NR; Ranges for 3 categories reported: Low (ref): ≤ 18.63 Mid: 18.64-21.36 High: >21.36 **Monitoring Stations:** Each zip code was linked to the nearest monitoring station (number not reported) **Copollutant (correlation):** CO NO_2 O_3 **Notes:** Daily or every 3rd day measurements used for mean calculations	**PM Increment:** Reported analyses using exposure categories **Effect Estimate [Lower CI, Upper CI]:** Birth cohort (phase I) Crude: Low: 1.0; Mid: 0.96 (0.90, 1.03) High: 1.05 (0.99, 1.12) Adj for birth cert Covariates: Low: 1.0 Mid: 1.01 (0.93, 1.09); High: 1.10 (1.01, 1.20) Survey respondents (phase II) Crude: Low: 1.0' Mid: 1.11 (0.90, 1.36) High: 1.27 (1.06, 1.53) Adj for birth cert Covariates: Low: 1.0 Mid: 1.14 (0.90, 1.46); High: 1.27 (0.99, 1.64) Adj for all Covariates: Low: 1.0 Mid: 1.15 (0.90, 1.47); High: 1.29 (1.00, 1.67) Two-phase model: * Low: 1.0 Mid: 0.98 (0.84, 1.15); High: 1.07 (0.85, 1.35) *method to reduce potential selection bias and increase statistical efficiency
Reference: Slama et al. (2007) **Period of Study:** 1/1998 -1/1999 **Location:** Munich, Germany	**Outcome:** Birth weight offspring at term **Study Design:** Cohort study **N:** 1016 births **Statistical Analyses:** Poisson model **Covariates:** Maternal passive smoking, maternal age, gestational duration, sex of child, parity, maternal education, maternal size, prepregnancy weight, other pollutants ($PM_{2.5}$, $PM_{2.5}$ absorbance, NO_2), season of conception **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Entire pregnancy period **Mean (SD):** 14.4 **Percentiles: 25th:** 13.5 **50th(Median):** 14.4 **75th:** 15.4 **Monitoring Stations:** Spatial component: 40 Temporal component: 1 **Copollutant (correlation):** p.a. = pregnancy avg trim. = trimester $PM_{2.5}$ (p.a.)–$PM_{2.5}$ (1st trim.): 0.85 $PM_{2.5}$ (p.a.)–$PM_{2.5}$ (2nd trim.): 0.77 $PM_{2.5}$ (p.a.)–$PM_{2.5}$ (3rd trim.): 0.87 $PM_{2.5}$ (p.a.)–NO_2 (p.a.): 0.45 $PM_{2.5}$ (p.a.)–NO_2 (1st trim.): 0.18 $PM_{2.5}$ (p.a.)–NO_2 (2nd trim.): 0.32 $PM_{2.5}$ (p.a.)–NO_2 (3rd trim.): 0.37 $PM_{2.5}$ (1st trim.)–$PM_{2.5}$ (2nd trim.): 0.40 $PM_{2.5}$ (1st trim.)–$PM_{2.5}$ (3rd trim.): 0.68 $PM_{2.5}$ (1st trim.)–NO_2 (p.a.): 0.48 $PM_{2.5}$ (1st trim.)–NO_2 (1st trim.): 0.15 $PM_{2.5}$ (1st trim.)–NO_2 (2nd trim.): 0.41 $PM_{2.5}$ (1st trim.)–NO_2 (3rd trim.): 0.39 $PM_{2.5}$ (2nd trim.)–$PM_{2.5}$ (3rd trim.): 0.51 $PM_{2.5}$ (2nd trim.)–NO_2 (p.a.): 0.23 $PM_{2.5}$ (2nd trim.)–NO_2 (1st trim.): -0.03 $PM_{2.5}$ (2nd trim.)–NO_2 (2nd trim.): 0.17 $PM_{2.5}$ (2nd trim.)–NO_2 (3rd trim.): 0.30 $PM_{2.5}$ (3rd trim.)–NO_2 (p.a.): 0.39 $PM_{2.5}$ (3rd trim.)–NO_2 (1st trim.): 0.33 $PM_{2.5}$ (3rd trim.)–NO_2 (2nd trim.): 0.21 $PM_{2.5}$ (3rd trim.)–NO_2 (3rd trim.): 0.23	**PM Increment:** 1) 1 μg/m^3; 2) Quartiles: a) 1st (reference) (7.2–13.5 μg/m^3); b) 2nd (13.5–14.4 μg/m^3); c) 3rd (14.4–15.4 μg/m^3); d) 4th (15.41–17.5 μg/m^3) **Prevalence ratios (PRs) of birth weight <3000 g during exposure over the whole pregnancy** **Single-pollutant models** Unadjusted models 2nd quartile: 1.07 (0.65, 1.73);3rd quartile: 1.38 (0.91, 2.09); 4th quartile: 1.45 (0.92, 2.25); Per 1 μg/m^3: 1.06 (0.95, 1.19) Adjusted models 2nd quartile: 1.08 (0.63, 1.82);3rd quartile: 1.34 (0.86, 2.13); 4th quartile: 1.73 (1.15, 2.69);Per 1 μg/m^3: 1.13 (1.00, 1.29) **Multipollutant models** Adjusted models 2nd quartile: 1.01 (0.57, 1.85); 3rd quartile: 1.12 (0.64, 1.87); 4th quartile: 1.36 (0.72, 2.45);Per 1 μg/m^3: 1.07 (0.91, 1.26) **Single-pollutant models** (restricted analysis to $PM_{2.5}$ absorbance below the median) Per 1 μg/m^3: 1.15 (0.89, 1.52) **Prevalence ratios (PRs) of birth weight <3000 g** **Multipollutant models (simultaneous adjustment of 3rd trimester** $PM_{2.5}$ **and whole pregnancy** $PM_{2.5}$**)** $PM_{2.5}$ (whole pregnancy) Per 1 μg/m^3: 0.96 (0.75, 1.19) $PM_{2.5}$ (3rd trimester) Per 1 μg/m^3: 1.17 (0.98, 1.40) **Prevalence ratios (PRs) of birth weight <3000 g during exposure over the whole pregnancy (adjustment for season of conception)** 4th quartile: 1.68 (1.05, 2.75);Per 1 μg/m^3: 1.12 (0.97, 1.28) **Prevalence ratios (PRs) of birth weight <3000 g during exposure over first trimester of pregnancy** Each trimester separately 2nd quartile: 1.14 (0.74, 1.96);3rd quartile: 1.28 (0.84, 2.10); 4th quartile: 1.65 (1.02, 2.60); Per 1 μg/m^3: 1.10 (0.99, 1.20) All trimesters adjusted simultaneously

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
			2nd quartile: 0.97 (0.60, 1.73);3rd quartile: 0.98 (0.57, 1.75); 4th quartile: 1.22 (0.71, 2.18); Per 1 μg/m3: 1.03 (0.90, 1.17) **Prevalence ratios (PRs) of birth weight <3000 g during exposure over second trimester of pregnancy** Each trimester separately 2nd quartile: 0.83 (0.52, 132);3rd quartile: 1.08 (0.71, 1.60); 4th quartile: 0.94 (0.61, 1.47); Per 1 μg/m3: 1.01 (0.92, 1.12) All trimesters adjusted simultaneously 2nd quartile: 0.75 (0.46, 1.24); 3rd quartile: 0.86 (0.56, 1.30); 4th quartile: 0.75 (0.48, 1.23); Per 1 μg/m3: 0.94 (0.84, 1.06) **Prevalence ratios (PRs) of birth weight <3000 g during exposure over third trimester of pregnancy** Each trimester separately 2nd quartile: 1.30 (0.80, 2.17); 3rd quartile: 1.44 (0.85, 2.27); 4th quartile: 1.90 (1.20, 2.82); Per 1 μg/m3: 1.14 (1.02, 1.24) All trimesters adjusted simultaneously 2nd quartile: 1.34 (0.79, 2.30); 3rd quartile: 1.48 (0.86, 2.58); 4th quartile: 1.91 (1.00, 3.20); Per 1 μg/m3: 1.14 (0.99, 1.29) **Prevalence ratios (PRs) of birth weight <3000 g during exposure over third trimester of pregnancy (adjustment for season of conception)** All trimesters adjusted simultaneously Per 1 μg/m3: 1.25 (1.04, 1.50) **Sensitivity analysis**(bootstrapped PR) 2nd quartile: 0.98 (0.63, 1.61);3rd quartile: 1.22 (0.82, 2.02); 4th quartile: 1.57 (1.02, 2.57); Per 1 μg/m3: 1.11 (0.98, 1.27) **Estimated increments in prevalence of birth weight of <3000 g during exposure 9 months after birth** Per 1 μg/m3: 7% (-7%, 22%)
Reference: Slama et al. (2007) **Period of Study:** 1/1998 -1/1999 **Location:** Munich, Germany	**Outcome:** Birth weight offspring at term **Study Design:** Cohort study **N:** 1016 births **Statistical Analyses:** Poisson model **Covariates:** Maternal passive smoking, maternal age, gestational duration, sex of child, parity, maternal education, maternal size, prepregnancy weight, other pollutants ($PM_{2.5}$, $PM_{2.5}$ absorbance, NO_2), season of conception **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ absorbance **Averaging Time:** Entire pregnancy period **Mean (SD):** 1.76 * **Percentiles: 25th:** 1.61* **50th(Median):** 1.72* **75th:** 1.89 * **Unit (i.e. μg/m3):** 10^{-5}/m **Monitoring Stations:** Spatial component: 40 Temporal component: 1 **Copollutant (correlation):** p.a. = pregnancy avg trim. = trimester abs = absorbance $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ abs (1st trim.): 0.54 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ abs (2nd trim.): 0.84 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ abs (3rd trim.): 0.55 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (p.a.): 0.69 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (1st trim.): 0.68 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (2nd trim.): 0.41 $PM_{2.5}$ abs (p.a.)–$PM_{2.5}$ (3rd trim.): 0.62 $PM_{2.5}$ abs (p.a.)–NO_2 (p.a.): 0.67 $PM_{2.5}$ abs (p.a.)–NO_2 (1st trim.): 0.34 $PM_{2.5}$ abs (p.a.)–NO_2 (2nd trim.): 0.63 $PM_{2.5}$ abs (p.a.)–NO_2 (3rd trim.): 0.36 $PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ abs (2nd trim.): 0.32	**PM Increment:** 1) 0.5 * 10^{-5}/m 2) Quartiles: a) 1st (reference) (1.29–1.61); b) 2nd (1.61–1.72); c) 3rd (1.72–1.89); d) 4th (1.89–3.10) **Prevalence ratios (PRs) of birth weight <3000 g during exposure over the whole pregnancy** **Single-pollutant models** Unadjusted models 2nd quartile: 1.19 (0.74, 1.99); 3rd quartile: 1.56 (0.98, 2.50); 4th quartile: 1.52 (0.96, 2.46); Per 0.5 * 10^{-5}/m: 1.25 (0.90, 1.70) Adjusted models 2nd quartile: 1.21 (0.73, 1.97); 3rd quartile: 1.63 (0.98, 2.57); 4th quartile: 1.78 (1.10, 2.70); Per 0.5 * 10^{-5}/m: 1.45 (1.06, 1.87) **Multipollutant models** Adjusted models 2nd quartile: 1.19 (0.70, 2.01); 3rd quartile: 1.55 (0.80, 2.80); 4th quartile: 1.40 (0.67, 2.90); Per 0.5 * 10^{-5}/m: 1.33 (0.76, 2.38) **Prevalence ratios (PRs) of birth weight <3000 g during exposure over the whole pregnancy (adjustment for season of conception)** 4th quartile: 1.72 (1.08, 2.73); Per 0.5 * 10^{-5}/m: 1.38 (0.96, 1.86) **Prevalence ratios (PRs) of birth weight <3000 g during exposure over the whole pregnancy** **Single-pollutant models** (restricted analysis to $PM_{2.5}$ below the median) Per 0.5 * 10^{-5}/m: 1.67 (0.66, 3.73)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
		$PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ abs (3rd trim.): -0.26	**Prevalence ratios (PRs) of birth weight <3000 g during exposure over first trimester of pregnancy**
		$PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (p.a.): 0.33	Each trimester separately
		$PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (1st trim.): 0.27	2nd quartile: 1.15 (0.73, 1.80); 3rd quartile: 1.01 (0.61, 1.53);
		$PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (2nd trim.): 0.08	4th quartile: 1.04 (0.70, 1.57); Per 0.5 * 10^{-5}/m: 1.03 (0.82, 1.28)
		$PM_{2.5}$ abs (1st trim.)–$PM_{2.5}$ (3rd trim.): 0.48	All trimesters adjusted simultaneously
		$PM_{2.5}$ abs (1st trim.)–NO_2 (p.a.): 0.29	2nd quartile: 0.90 (0.52, 1.58); 3rd quartile: 0.82 (0.45, 1.31);
		$PM_{2.5}$ abs (1st trim.)–NO_2 (1st trim.): 0.84	4th quartile: 0.88 (0.53, 1.42); Per 0.5 * 10^{-5}/m: 1.02 (0.77, 1.29)
		$PM_{2.5}$ abs (1st trim.)–NO_2 (2nd trim.): 0.16	**Prevalence ratios (PRs) of birth weight <3000 g during exposure over second trimester of pregnancy**
		$PM_{2.5}$ abs (1st trim.)–NO_2 (3rd trim.): -0.39	Each trimester separately
		$PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ abs (3rd trim.): 0.31	2nd quartile: 1.33 (0.85, 2.22); 3rd quartile: 1.76 (1.07, 2.91);
		$PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (p.a.): 0.48	4th quartile: 1.83 (1.11, 2.81); Per 0.5 * 10^{-5}/m: 1.27 (1.04, 1.54)
		$PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (1st trim.): 0.53	All trimesters adjusted simultaneously
		$PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (2nd trim.): 0.29	2nd quartile: 1.30 (0.77, 2.16); 3rd quartile: 1.63 (0.93, 2.73);
		$PM_{2.5}$ abs (2nd trim.)–$PM_{2.5}$ (3rd trim.): 0.36	4th quartile: 1.99 (1.12, 3.33); Per 0.5 * 10^{-5}/m: 1.21 (0.93, 1.54)
		$PM_{2.5}$ abs (2nd trim.)–NO_2 (p.a.): 0.61	**Prevalence ratios (PRs) of birth weight <3000 g during exposure over third trimester of pregnancy**
		$PM_{2.5}$ abs (2nd trim.)–NO_2 (1st trim.): 0.19	Each trimester separately
		$PM_{2.5}$ abs (2nd trim.)–NO_2 (2nd trim.): 0.85	2nd quartile: 1.30 (0.85, 2.09); 3rd quartile: 0.92 (0.55, 1.50);
		$PM_{2.5}$ abs (2nd trim.)–NO_2 (3rd trim.): 0.17	4th quartile: 1.50 (1.00, 2.27); Per 0.5 * 10^{-5}/m: 1.20 (0.98, 1.44)
		$PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (p.a.): 0.52	All trimesters adjusted simultaneously
		$PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (1st trim.): 0.51	2nd quartile: 0.99 (0.64, 1.62); 3rd quartile: 0.71 (0.40, 1.20);
		$PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (2nd trim.): 0.41	4th quartile: 1.14 (0.68, 1.91); Per 0.5 * 10^{-5}/m: 1.15 (0.92, 1.42)
		$PM_{2.5}$ abs (3rd trim.)–$PM_{2.5}$ (3rd trim.): 0.37	**Prevalence ratios (PRs) of birth weight <3000 g during exposure over first trimester of pregnancy (adjustment for season of conception)**
		$PM_{2.5}$ abs (3rd trim.)–NO_2 (p.a.): 0.40	All trimesters adjusted simultaneously
		$PM_{2.5}$ abs (3rd trim.)–NO_2 (1st trim.): -0.34	4th quartile: 0.73 (0.38, 1.38); Per 0.5 * 10^{-5}/m: 0.93 (0.41, 1.32)
		$PM_{2.5}$ abs (3rd trim.)–NO_2 (2nd trim.): 0.21	**Prevalence ratios (PRs) of birth weight <3000 g during exposure over second trimester of pregnancy (adjustment for season of conception)**
		$PM_{2.5}$ abs (3rd trim.)–NO_2 (3rd trim.): 0.88	All trimesters adjusted simultaneously
			4th quartile: 2.45 (1.22, 4.77); Per 0.5 * 10^{-5}/m: 1.14 (0.70, 1.64)
			Prevalence ratios (PRs) of birth weight <3000 g during exposure over third trimester of pregnancy (adjustment for season of conception)
			All trimesters adjusted simultaneously 4th quartile: 1.19 (0.60, 2.48); Per 0.5 * 10^{-5}/m: 1.29 (0.90, 1.75)
			Sensitivity analysis (bootstrapped PR)
			2nd quartile: 1.19 (0.76, 1.91); 3rd quartile: 1.52 (0.99, 2.34);
			4th quartile: 1.62 (1.06, 2.55); Per 0.5 * 10^{-5}/m: 1.35 (1.01, 1.83)
			Estimated increments in prevalence of birth weight <3000 g during exposure 9 months after birth Per 0.5 * 10^{-5}/m: 18% (-16%, 57%)

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Wilhelm et al. (2005a) **Period of Study:** 1994-2000 **Location:** Los Angeles County, California, U.S.	**Outcome:** Term low birth weight (LBW) (<2500 g at ≥ 37 completed weeks gestation) Vaginal birth <37 completed weeks gestation **Age Groups:** LBW: ≥ 37 completed weeks Preterm births: <37 completed weeks **Study Design:** Cohort study **N:** For LBW: 136,134 For preterm birth: 106,483 **Statistical Analyses:** Logistic regression **Covariates:** Maternal age, maternal race, maternal education, parity, interval since previous live birth, level of prenatal care, infant sex, previous LBW or preterm infant, birth season, other pollutants (not specified) **Dose-response Investigated?** Yes **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Entire pregnancy Trimesters of pregnancy Months of pregnancy 6 weeks before birth **Mean (SD):** First trimester: 21.9 Third trimester: 21.0 6 weeks before birth: 21.0 **Range (Min, Max):** First trimester: 11.8-38.9 Third trimester: 11.8-.38.9 6 weeks before birth: 9.9-48.5 **Monitoring Stations:** Zip-code-level analysis: 9 Address-level analysis: 8 **Copollutant (correlation):** First trimester $PM_{2.5}$-CO: 0.57 $PM_{2.5}$-NO_2: 0.73 $PM_{2.5}$-O_3: -0.55 $PM_{2.5}$-PM_{10}: 0.43 Third trimester: $PM_{2.5}$-CO: 0.67 $PM_{2.5}$-NO_2: 0.78 $PM_{2.5}$-O_3: -0.60 $PM_{2.5}$-PM_{10}: 0.52 6 weeks before birth: $PM_{2.5}$-CO: 0.63 $PM_{2.5}$-NO_2: 0.74 $PM_{2.5}$-O_3: -0.60 $PM_{2.5}$-PM_{10}: 0.60	**PM Increment:** 1) 10 µg/m^3; 2) 3 levels: a) <25%ile (reference); b) 25%-75%ile; c) ≥ 75%ile **Incidence of LBW (third trimester exposure)** <17.1 µg/m^3: 2.4 (2.0, 2.8); 17.1 to <24.0 µg/m^3: 2.2 (2.0, 2.5); ≥ 24.0 µg/m^3: 2.1 (1.7, 2.4) **Incidence of preterm birth (first trimester exposure)** <18.0 µg/m^3: 10.6 (9.6, 11.7); 18.0 to <25.4 µg/m^3: 8.8 (8.1, 9.5); ≥ 25.4 µg/m^3: 9.0 (8.1, 10.0) **Incidence of preterm birth (6 weeks before birth exposure)** <16.5 µg/m^3: 8.2 (7.4, 9.1); 16.5 to <24.7 µg/m^3: 8.8 (8.2, 9.4); ≥ 24.7 µg/m^3: 9.6 (8.7, 10.5) **Outcome: Preterm birth** **Exposure Period: First trimester of pregnancy** **Address-level analysis:** Single-pollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 0.85 (0.70, 1.02); 18.1 to <25.2 µg/m^3: 0.91 (0.72, 1.16); ≥ 25.2 µg/m^3: 0.83 (0.60, 1.14) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 0.85 (0.74, 0.99); 18.3 to <25.2 µg/m^3: 0.81 (0.69, 0.94); ≥ 25.2 µg/m^3: 0.79 (0.65, 0.97) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 0.83 (0.78, 0.88); 18.5 to <24.9 µg/m^3: 0.79 (0.74, 0.85); ≥ 24.9 µg/m^3: 0.76 (0.70, 0.84) **Zip-code-level analysis :** Single-pollutant model: Per 10 µg/m^3: 0.73 (0.67, 0.80); 18.0 to <25.4 µg/m^3: 0.70 (0.61, 0.80); ≥ 25.4 µg/m^3: 0.64 (0.53, 0.76) **Outcome: Preterm birth** **Exposure Period: 6 weeks before birth** **Address-level analysis:** Single-pollutant model: Distance ≤ 1 mile Per 10 µg/m^3: 1.09 (0.91, 1.30); 16.8 to <24.1 µg/m^3: 1.21 (0.97, 1.51); ≥ 24.1 µg/m^3: 1.25 (0.93, 1.68) Single-pollutant model: 1 <distance ≤ 2 mile Per 10 µg/m^3: 1.08 (0.97, 1.21); 17.2 to <24.5 µg/m^3: 0.94 (0.82, 1.08); ≥ 24.5 µg/m^3: 1.04 (0.87, 1.24) Multipollutant model1 <distance ≤ 2 mile Per 10 µg/m^3: 1.18 (0.84, 1.65) Single-pollutant model: 2 <distance ≤ 4 mile Per 10 µg/m^3: 1.05 (0.99, 1.10); 17.3 to <24.6 µg/m^3: 1.06 (1.00, 1.13); ≥ 24.6 µg/m^3: 1.08 (0.99, 1.17) **Zip-code-level analysis** Single-pollutant model: Per 10 µg/m^3: 1.10 (1.00, 1.21); 16.5 to <24.7 µg/m^3: 1.06 (0.94, 1.20); ≥ 24.7 µg/m^3: 1.19 (1.02, 1.40) **(See Notes[1])** Multipollutant model Per 10 µg/m^3: 1.12 (0.90, 1.40); ≥ 24.6 µg/m^3: 1.12 (0.82, 1.52) **Notes:** [1] In the table, the 75%ile is noted as 24.7 µg/m^3. However, the text notes the 75%ile as 24.3 µg/m^3.

Reference	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Woodruff et al. (2006) **Period of Study:** 1999-2000 **Location:** California	**Outcome (ICD10):** SIDS (R95) Respiratory mortality (J00-J99) Bronchopulmonary dysplasia (P27.1) External accidents (V01-Y98) Ill-defined and unspecified causes of mortality (R99) **Age Groups:** >28 days old **Study Design:** Matched case-control **N:** 3877 infants **Statistical Analyses:** Conditional logistic regression **Covariates:** Maternal race, education, parity, age, marital status **Dose-response Investigated?** Yes **Statistical Package:** STATA	**Pollutant:** $PM_{2.5}$ **Averaging Time:** time period between birth and postneonatal death for the infant who died and the same period for its four matched surviving infants **Percentiles:** Infants who died of all causes (cases) **25th:** 13.4 **50th(Median):** 19.2 **75th:** 23.6 Matched controls **25th:** 13.5 **50th(Median):** 18.4 **75th:** 22.7 **Monitoring Stations:** 73 (from 39 counties)	**PM Increment:** 10 µg/m^3 **RR Estimate [Lower CI, Upper CI]; lag:** All-cause mortality: Unadjusted: 1.15 (1.00, 1.32); Adjusted: 1.07 (0.93, 1.24) Cause-specific mortality: Respiratory (all): Unadjusted: 2.15 (1.15, 4.02); Adjusted: 2.13 (1.12, 4.05) Respiratory (excluding deaths due to BPD): Adjusted: 1.42 (0.66, 3.03); Respiratory (BPD alone): Unadjusted: 6.00 (1.40, 27.76) Respiratory (low birth weight infants only): Unadjusted: 3.09 (1.14, 8.40) Respiratory (normal birth weight infants only): Unadjusted: 1.66 (0.74, 3.70) Respiratory (with matched $PM_{2.5}$ averaged over all monitors in county) Adjusted: 2.28 (0.94, 5.52); Respiratory (averaging all $PM_{2.5}$ measurements in county over the 2-year study period): Adjusted: 2.26 (0.83, 6.21) SIDS: Unadjusted: 0.86 (0.61, 1.22); Adjusted: 0.82 (0.55, 1.23) SIDS (includes ICD10 code R99: ill-defined and unspecified causes of mortality): Adjusted: 1.03 (0.79, 1.35) External causes: Unadjusted: 0.91 (0.56, 1.47); Adjusted: 0.83 (0.50, 1.39) Compare against the lowest quartile, estimates for respiratory-specific mortality were provided: 2nd quartile: 1.28 (0.47, 3.51) 3rd quartile: 1.75 (0.65, 4.72) 4th quartile: 2.35 (0.85, 6.54)
Reference: Woodruff et al. (2008) **Period of Study:** 1999-2002 **Location:** US counties with >250,000 residents (96 counties)	**Outcome (ICD10):** Postneonatal deaths: Respiratory mortality (J000-99, plus bronchopulmonary dysplasia [BPD] P27.1); SIDS (R95); Ill-defined causes (R99); All other deaths evaluated as a control category **Age Groups:** Infants aged >28 days and <1 yr **Study Design:** Cross-sectional **N:** 3,583,495 births (6,639 postneonatal deaths) **Statistical Analyses:** Logistic GEE (exchangeable correlation structure) **Covariates:** maternal race/ethnicity, marital status, age, education, primiparity, county-level poverty and per capita income levels, year and month of birth dummy variables to account for time trend and seasonal effects, and region of the country; sensitivity analyses performed among only those mothers with smoking information (adjustment for smoking had no effect on the estimates) **Season:** Adjusted for year and month of birth dummy variables to account for time trend and seasonal effects **Dose-response Investigated?** Evaluated the appropriateness of a linear form from analysis based on quartiles of exposure and concluded that linear form was appropriate (data not shown) **Statistical Package:** NR	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Measured continuously for 24 h once every 6 days; exposure assigned by calculating avg concentration of pollutant during first 2 months of life **Median and IQR (25th-75th percentile):** Survivors: 14.8 (11.7-18.7) All causes of death: 14.9 (12.0-18.6) Respiratory: 14.8 (11.5-18.5) SIDS: 14.5 (12.0-17.5) SIDS + ill-defined: 14.8 (12.1-18.5) Other causes: 14.9 (12.0-18.6) **Percentiles:** See above **PM Component:** Not assessed, but controlled for region of the country to account for PM composition variation **Monitoring Stations:** NR **Copollutant (correlation):** PM_{10} (r = 0.34) $PM_{2.5}$ CO (r = 0.35) SO_2 (r = 0.21) O_3 (r = -0.10) **Notes:** Monthly averages calculated if there were at least 3 available measures for PM; Assigned exposures using the avg concentration of the county of residence	**PM Increment:** IQR (7 µg/m^3) **Effect Estimate [Lower CI, Upper CI]:** Adjusted ORs for single pollutant models All causes: 1.04 (0.98, 1.11) Respiratory: 1.11 (0.96, 1.29) SIDS: 1.01 (0.86, 1.20) Ill-defined + SIDS: 1.06 (0.97, 1.17) Other causes: 1.03 (0.96, 1.12) Adjusted ORs for multipollutant models (including CO, O_3, SO_2) Respiratory: 1.05 (0.89, 1.24) SIDS: 1.04 (0.87, 1.23) OR for respiratory deaths assessing exposure as quartiles Highest vs Lowest quartile: 1.39 (1.04, 1.85)

E.8. Long-Term Exposure and Mortality

Table E-33. Long-term exposure to PM_{10} and mortality.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chen et al. (2005) **Period of Study:** 1973-1998 **Location:** San Francisco, San Diego, Los Angeles, CA	**Outcome:** Mortality: CHD **Study Design:** Cohort **Statistical Analyses:** Cox proportion hazards model **Age Groups:** >25	**Pollutant**: PM_{10} **Averaging Time**: 25 years **Mean (SD):** 52.6 **Range (Min, Max):** NR **CopollutantS**: NO_2; O_3; SO_2	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** Males PM_{10}: 0.95 (0.81, 1.11); 0-1 PM_{10}+NO_2: 0.90 (0.76, 1.07); 0-1 PM_{10}+SO_2: 0.92 (0.78,1.09); 0-1 PM_{10}+O_3: 0.97 (0.78,1.20); 0-1 Females PM_{10}: 1.11 (0.98, 1.26); 0-1 PM_{10}+NO_2: 1.11 (0.97, 1.26); 0-1 PM_{10}+SO_2: 1.15 (1.02,1.31); 0-1 PM_{10}+O_3: 1.33 (1.12,1.59); 0-1
Reference: Chiu et al. (2006) **Period of Study:** 1994-2003 **Location:** Taiwan	**Outcome (ICD9):** Lung Cancer (Death). (162) **Age Groups:** 50-69 y/o **Study Design:** Case-control (sex, yr of birth, yr of death matched) **N:** 972 cases, 972 controls **Statistical Analyses:** Conditional logistic regression **Covariates:** Sex, year of birth, year of death, degree of urbanization **Dose-response Investigated?** Yes	**Pollutant:** PM_{10} **Averaging Time:** Annual mean of avg daily 24-h values **Mean (SD):** NR **Range (Min, Max):** NR **Air Pollution Index** (100= exact attainment of NAAQS for all five pollutants throughout the study period)	**Air Pollution Index Increment:** Categories: <0.62 (ref), 0.62-0.74, ≥ 0.75 **OR Estimate [Lower CI, Upper CI]** 0.62-0.74: 1.11 (0.88-1.40) ≥ 0.75: 1.28 (1.02-1.61) **Notes:** This association is not for PM alone, but for an exposure index of all criteria pollutants, (except lead). The exposure is an index of long-term air pollution exposure, created by dividing the annual avg of the measured values for each criteria pollutant by the NAAQS for that pollutant. The ratios for each pollutant were scaled to a 100-point scale, then averaged together to generate an index value representing the net burden of these five pollutants, with each weighted equally.
Reference: Cui et al. (2003) **Period of Study:** 6/2000-10/2002 **Location:** China–5 cities	**Outcome:** Mortality: SARS **Study Design:** Ecologic **Statistical Analyses:** SAS (Statistical Analysis Software) **Age Groups:** All ages	**Pollutant:** PM_{10} **Averaging Time:** Yearly avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutants:** O_3, SO_2, CO, NO_2	**Relative Risks (Lower CI, Upper CI); lag:** High API (Air Pollution Indexes): 1.71, (1.34, 3.33), 0-1 Moderate API: 2.26, (1.53,3.35), 0-1
Reference: Gehring et al. (2006) **Period of Study:** 1985 to 1994 **Location:** North Rhine-Westphalia Germany	**Outcome:** Mortality: Total (non-accidental) (< 800) Cardio-respiratory (390-448, 490-496, 487, 480-486, 507) Pulmonary (460-519) Cardiovascular (400-440) Lung Cancer (162) Other-causes **Study Design:** Cross-sectional **Statistical Analyses:** Cox proportional hazards model; SAS **Age Groups:** 50-59 year old women	**Pollutant:** PM_{10} **Averaging Time:** One and five year avg **Mean (SD):** 1 year avg.: 43.7 µg/m^3 5 year avg.: 48.0 µg/m^3 **Range (Min, Max):** 1 year range: (34.8, 52.5) 5 year range: (39.1, 56.1) **Copollutant (correlation):** NO_2:(1 yr avg.: 0.5, 5 yr avg.: 0.8)	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI)** All causes- Adjusted rates* Distance to road: 1.29 (0.93,1.78) 1 yr avg: 1.08 (0.94,1.25) 5 yr avg: 1.13 (0.99,1.30) Cardiopulmonary- Adjusted Rates* Distance to road: 1.70 (1.02,2.81) 1 year avg: 1.34 (1.06,1.71) 5 year avg: 1.59 (1.23,2.04) Not Cardiopulmonary- Adjusted rates* Distance to road- 1.21 (0.77,1.87) 1 year avg: 0.92 (0.76,1.10) 5 year avg: 0.91 (0.76,1.08) *Adjusted for SES and smoking
Reference: Goss et al. (2004b) **Period of Study:** 1999-2000 **Location:** United States	**Outcome:** Mortality **Study Design:** Cohort Study (Cystic Fibrosis Cohort) **Statistical Analyses:** Logistic Regression **Age Groups:** >6 yrs	**Pollutant:** PM_{10} **Averaging Time:** Annual avg **Mean (SD) unit:** PM_{10}: 24.8 (7.8) **IQR:** PM_{10}: 20.3-28.9 **Copollutant:** O_3, NO_2, SO_2, CO	**Increment:** 10 µg/m^3 PM_{10}: "no quantitative results; no clear significant association or trend"

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Maheswaran et al. (2005) **Period of Study:**1994-1998 **Location:** Sheffield, UK	**Outcome:** Mortality: Coronary Heart Disease (410-414) ER Hospital Admissions (120-125) **Study Design:** Ecological **Statistical Analyses:** Poisson Regression **Age Groups:** Men and Women: 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+	**Pollutant:** PM_{10} **Averaging Time:** 5 year avg **Mean (SD) unit:** 23.3 **Range (5th, 95th):** NR **Copollutant (correlation):** NOx: r=0.87 CO: r=0.82	**Increment:** 10 µg/m^3 **Relative Risk (CI min, CI max)** RR for mortality from coronary heart disease to modeled quintile categories PM_{10} Adjusted for sex and age (not spatially smoothed) 5: 1.30 (1.19, 1.43); 4: 1.23 (1.13, 1.35) 3: 1.10 (1.01, 1.21); 2: 1.06 (0.98, 1.16); 1: 1.00 PM_{10} Adjusted for sex, age, deprivation , and smoking (not spatially smoothed) 5: 1.08 (0.96, 1.20); 4: 1.08 (0.98, 1.20) 3: 1.00 (0.90, 1.11); 2: 1.03 (0.94, 1.12)' 1: 1.00 PM_{10} Adjusted for sex, age, deprivation, and smoking (spatially smoothed using a 1 km radius) 5: 1.07 (0.96, 1.21); 4: 1.03 (0.93, 1.15) 3: 1.00 (0.90, 1.10); 2: 0.97 (0.89, 1.07)' 1: 1.00
Reference: McDonnell et al. (2000) **Period of Study:** 1973-1977 **Location:** California	**Outcome**: Mortality **Study Design:** Cohort (AHSMOG airport cohort) **Statistical Analyses:** Cox regression models **Age Groups:** Males, 27 yrs+	**Pollutant:** PM_{10} **Averaging Time:** monthly averages **Mean (SD):** 59.2 (16.8) **IQR:** 29.5 **Copollutants (correlation):** O_3: 0.79; SO_2: 0.29; NO_2: 0.07; SO_4: 0.45	**Increment:** IQR All Cause 1.15 (0.94-1.41) Resp 1.48 (0.93-2.34) Lung Cancer 1.84 (0.59-5.67)
Reference: Naess et al. (2007b) **Period of Study:**1992-1998 **Location:** Oslo, Norway	**Outcome:** Mortality: Non-accidental (<800) Lung cancer (162) COPD (490-496) Cardiovascular (390-459) **Study Design:** Prospective Cohort **Statistical Analyses:** Cox proportional hazards regression model **Age Groups:** 51-70 71-90	**Pollutant:** PM_{10} **Averaging Time:** 4 year avg **Mean (SD) unit:** PM_{10}: 19 µg/m^3 **Range (Min, Max):** PM_{10}: (7, 30) **Copollutant (correlation):** NO_2: r=0.88	**Relative Risk (CI min, CI max)** **Increment:** 10 µg/m^3 RR for death from CVD and lung cancer Men (ages 51-70) CVDPM_{10}: 1.10 (1.06, 1.15) COPD PM_{10}: 1.33 (1.17, 1.50) Lung Cancer PM_{10}: 1.07 (0.98, 1.17) Women (ages 51-70) CVD PM_{10}: 1.14 (1.07, 1.21) COPD PM_{10}: 1.16 (1.02, 1.32) Lung Cancer PM_{10}: 1.22 (1.10, 1.37) Men (ages 71-90) CVD PM_{10}: 1.05 (1.02, 1.08) COPDPM_{10}: 1.13 (1.04, 1.24) Lung Cancer PM_{10}: 1.10 (1.00, 1.21) Women (ages 71-90) CVD PM_{10}: 1.01 (0.99, 1.04) COPD PM_{10}: 1.11 (1.01, 1.21) Lung Cancer PM_{10}: 1.18 (1.04, 1.33)
Reference: O'Neill et al. (2005b) **Period of Study:** 1996-1998 **Location:** Mexico City, Monterrey, Mexico	**Outcome:** External causes (>E800), (V01-Y89), respiratory-causes (460-520), cardiovascular causes (390-460), ICD10 cause I and J. **Age Groups:** Children (0-14 yrs) Elderly (ages > 65 yrs) **Study Design**: Time-series **N (Specify units):** Mexico city: 206,510 daily deaths Monterrey: 21.758 daily deaths **Statistical Analyses:** Robust Poisson regression **Covariates:** Day of-the-wk, public holidays, respiratory epidemics, air pollution, long-term trends **Statistical Package**: S-Plus 2000	**Pollutant:** PM_{10} **Averaging Time:** 24-h **Mean (SD):** Mexico City: 75.8 (31.4) Monterrey: 50.0 (23.5) **Range (Min, Max):** Mexico City: (18.0, 233.9) Monterrey: (6.2, 230.8) **Monitoring Stations:** 5 in Mexico City 6 in Monterrey	Authors study the effects of air pollution in general (in this case, a combination of PM_{10} and O_3). There is no specific data in regards to PM_{10} or O_3 but rather their relation together with respiratory epidemics and their associations with temperature.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Pope et al. (2002) **Period of Study**:1982–2000 **Location:** Metropolitan areas in all 50 states in the US	**Outcome (ICD 9):** Mortality: Cardiopulmonary (401-440, 460-519) Lung Cancer (162) Non-accidental (<800) **Study Design:** Prospective Cohort **Statistical Analyses:** Cox Proportional Hazards Regression **Age Groups:** >30	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD) unit:** 28.8 (5.9) $\mu g/m^3$ **Range (Min, Max):** NR **Copollutant:** SO_2, NO_2, CO, O_3	**Increment:** 10 $\mu g/m^3$ **Relative Risk (Lower CI, Upper CI)** All-Cause Mortality: 1979-1983: 1.04 (1.01, 1.08); 1999-2000: 1.06 (1.02, 1.10); Avg: 1.06 (1.02, 1.11) Cardiopulmonary Mortality: 1979-1983: 1.06 (1.02, 1.10); 1999-2000: 1.08 (1.02, 1.14); Avg: 1.09 (1.03, 1.16) Lung Cancer: 1979-1983: 1.08 (1.01, 1.16); 1999-2000: 1.13 (1.04, 1.22); Avg: 1.14 (1.04, 1.23) All Other Causes: 1979-1983: 1.01 (0.97, 1.05); 1999-2000: 1.01 (0.97, 1.06); Avg: 1.01 (0.95, 1.06)
Reference: Puett et al. (2008) **Period of Study:**1992-2002 **Location:** 13 Northeastern states (US)	**Outcome:** Mortality: All Cause and CHD **Study Design:** Nurses' Health Study–Prospective Cohort **Statistical Analyses:** General additive mixed model	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD) unit:** 21.3 (4.3) **Range (Min, Max):** NR **Copollutant:** NR	**PM Increment:** 10 $\mu g/m^3$ **All-Cause Mortality** 1 month prior to death: 1.04 (0.98, 1.11) 3 month moving avg: 1.14 (1.05, 1.23) 12 month moving avg: 1.16 (1.05, 1.28) 48 month moving avg: 1.15 (1.04, 1.28) **Fatal CHD** 1 month prior to death: 1.16 (0.98, 1.36) 3 month moving avg: 1.21 (0.98, 1.48) 12 month moving avg: 1.43 (1.10, 1.86) 48 month moving avg: 1.43 (1.09, 1.88) Stratified by BMI: BMI<30: 1.08 (0.76, 1.52); BMI≥ 30: 1.99 (1.23, 3.22)
Reference: Rosenlund et al. (2006) **Period of Study:**1992-1994 **Location:** Stockholm, Sweden	**Outcome:** Mortality: Myocardial Infarction **Study Design:** Case-control **Statistical Analyses:** Logistic Regression; STATA; GIS **Age Groups:** 45-70	**Pollutant:** PM_{10} **Averaging Time:** Annual avg **Mean (SD):** 12 **IQR (25th, 75th):** NR **Copollutant (correlation):** NO_2: r=0.93; CO: r=0.66; SO_2: r=0.49	**Relative Risk (Lower CI, Upper CI)** PM_{10} from Traffic Fatal Cases: 1.39 (0.94, 2.07) In-hospital death: 1.21 (0.75, 1.94) Out of hospital death: 1.84 (1.00, 3.40)
Reference: Samoli, et al (2005) **Period of Study:** 1990-1997. The data covered at least 3 consecutive years for each city ithin the years 1990–1997. **Location: 22** European cities: Athens, Barcelona, Basel, Bilbao, Birmingham, Budapest, Cracow, Dublin, Erfurt, Geneva, Helsinki, Ljubljana, Lodz, London, Lyon, Madrid, Marseille, Milan, Netherlands, Paris, Poznan, Prague, Rome, Stockholm, Tel Aviv, Teplice, Torino, Valencia, Wroclaw, Zurich	**Outcome:** cardiovascular mortality (ICD-9 390–459), and respiratory mortality (ICD-9 460–519) **Age Groups:** NR **Study Design:** Regression models **N (Specify units):** > 60 million people **Statistical Analyses:** Poisson regression, generalized additive models **Covariates:** Mean temperature in degrees centigrade **Dose-response Investigated?** No	**Pollutant:** PM_{10} **Averaging Time:** daily **Mean (SD):** NR **Percentiles:** **50th(Median):** 21-66 **90th:** 27-129 **Range (Min, Max):** 14, 65 **Monitoring Stations:** Daily air pollution measurements were provided by the monitoring networks established in each town participating in the APHEA-2 project.	**PM Increment:** 10 $\mu g/m^3$ Percent increase in number of deaths (as measured by exposure-response) not specified quantitatively, only represented in figures. An increase from 50 to 60 $\mu g/m^3$ is associated with an increase of about 0.4% in total deaths and with increases of about 0.5% in both cardiovascular and respiratory deaths

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Schikowski et al. (2007) **Period of Study:**1985-1994 Follow-up: 1/02-5/03 **Location:** Ruhr area-Dortmund, Duisburg, Essen, Gelsenkirchen, Herne, Borken, and Dulmen	**Outcome:** Mortality: Cardiovascular (400-440) Non-accidental (<800) **Study Design:** Cross-sectional **Statistical Analyses:** Cox's proportional hazard regression model; PHREG; SAS **Age Groups:** Women >55	**Pollutant:** PM_{10} **Averaging Time:** 24-h avg **Mean (SD) unit:** PM_{10}: 48 NO_2: 39 **Range (Min, Max):** PM_{10}: (39, 56) NO_2: (22, 55) **Copollutant:** NO_2	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** RR of cardiovascular mortality for impaired respiratory health at five and twelve years of survival time Chronic Bronchitis 5 yr: 1.53 (0.83, 2.79); 12 yr: 1.65 (0.93, 2.95) Frequent cough with phlegm production 5yr: 1.34 (0.71, 2.51); 12yr: 1.65 (0.94, 2.89) Frequent Cough 5yr: 1.17 (0.73, 1.89); 12yr: 1.21 (0.76, 1.93) FEV_1<80% of predicted value 5yr: 3.79 (1.64, 8.74); 12yr: 1.35 (0.66, 2.77) FVC<80%of predicted value 5yr: 5.03 (2.10, 12.02); 12yr: 1.89 (1.01, 3.57) PM_{10} (when exposed to a five year avg of 7 µg/m^3) Chronic bronchitis: 1.62 (1.14, 2.30) Frequent cough with phlegm: 1.62 (1.14, 2.31) Frequent cough: 1.63 (1.15, 2.32) FEV_1<80%: 1.14 (0.67, 1.95) FVC<80%: 1.13 (0.66, 1.93)
Reference: Zanobetti and Schwartz (2007) **Period of Study:** 1985-1999 **Location:** 21 US Cities	**Outcome:** Mortality: Myocardial Infarction (410) Congestive Heart Failure (428) COPD (490-496) Diabetes (250) Hypertension (401) **Study Design:** Prospective Cohort **Statistical Analyses:** Cox Proportional hazard regression **Age Groups:** >65	**Pollutant:** PM_{10} **Averaging Time:** 5 year avg **Mean (SD) unit:** PM_{10}: 28.8 **Range (Min, Max):** NR **Copollutant (correlation):** NR	**Increment:** 10 µg/m^3 **Relative Risk (Min CI, Max CI); Lag** CHF 1.09 (1.01, 1.18); 0; 1.09 (1.01, 1.19); 1 1.13 (1.02, 1.25); 2; 1.04 (0.97, 1.12); 3 Sum lags 0-3: 1.41 (1.19, 1.66) MI 1.09 (0.92, 1.30); 0; 1.12 (0.97, 1.30); 1 1.15 (1.08, 1.23); 2; 1.01 (0.94, 1.09); 3 Sum Lags 0-3: 1.43 (1.12, 1.82) Other Causes 1.04 (0.96, 1.14); 0; 1.07 (0.99, 1.14); 1 1.14 (1.10, 1.18); 2; 1.06 (0.99, 1.12); 3 Sum Lags 0-3: 1.34 (1.17, 1.52) RR for increase in PM_{10} for the sensitivity analyses CHF Subjects with subsequent MI: 1.42 (1.22, 1.65) Subjects admitted 1985-1996:1.51 (1.26, 1.81) Subsequent MI Subjects admitted 1985-1996:1.62 (1.23, 2.13) Other Causes Subjects with subsequent MI: 1.33 (1.15, 1.55) Subjects admitted 1985-1996: 1.45 (1.26, 1.68) **Estimated from Figure 1:** CHF Male: 1.08 (0.97, 1.19); Female: 1.16 (1.03, 1.28) Age 65-75: 1.04 (0.96, 1.13); Age >75: 1.12 (1.01, 1.27) MI Male: 1.13 (1.06, 1.24); Female: 1.17 (1.01, 1.34) Age 65-75: 1.09 (0.97, 1.20); Age >75: 1.22 (1.09, 1.41) Other Causes Male: 1.10 (1.02, 1.20); Female: 1.11 (1.05, 1.21) Age 65-75: 1.17 (1.02, 1.31); Age >75: 1.11 (1.07, 1.17)

Table E-34. Long-term exposure to $PM_{10-2.5}$ and mortality.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Chen et al. (2005) **Period of Study:** 1973-1998 **Location:** San Francisco, San Diego, Los Angeles, CA	**Outcome:** Mortality: CHD **Study Design:** Cohort **Statistical Analyses:** Cox proportion hazards model **Age Groups:** >25	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** 25 years **Mean (SD):** 25.4 **Range (Min, Max):** NR **Copollutant:** NO_2 O_3 SO_2	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** Males $PM_{10-2.5}$: 0.93 (0.68, 1.29); 0-1 $PM_{10-2.5}$+NO_2: 0.86 (0.62, 1.20); 0-1 $PM_{10-2.5}$+SO_2: 0.90 (0.64,1.27); 0-1 $PM_{10-2.5}$+O_3: 1.01 (0.67,1.51); 0-1 Females $PM_{10-2.5}$: 1.20 (0.95, 1.53); 0-1 $PM_{10-2.5}$+NO_2: 1.19 (0.92, 1.54); 0-1 $PM_{10-2.5}$+SO_2: 1.31 (1.03,1.68); 0-1 $PM_{10-2.5}$+O_3: 1.47 (1.10,1.96); 0-1
Reference: Goss et al. (2004b) **Period of Study:** 1999-2000 **Location:** United States	**Outcome:** Mortality **Study Design:** Cohort Study (Cystic Fibrosis Cohort) **Statistical Analyses:** Logistic Regression **Age Groups:** >6 yrs	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** Annual avg **Mean (SD) unit:** $PM_{2.5}$: 13.7 (4.2) **IQR:** $PM_{2.5}$: 11.8-15.9 **Copollutant:** O_3, NO_2, SO_2, CO	**Increment:** 10 µg/m^3 $PM_{2.5}$: 1.32 (0.91 – 1.93)
Reference: Lipfert et al (2006a) **Period of Study:** 1989-1996 **Location:** Various parts of the Untied States	**Outcome:** Mortality **Study Design:** Retrospective Cohort **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** Male US veterans between ages of 39 and 63 (Avg. age: 51)	**Pollutant:** $PM_{10-2.5}$ **Mean (SD):** 16.0 (5.1)	**Increment:** 12 1.07 (1.01, 1.13)
Reference: McDonnell et al. (2000) **Period of Study:** 1973-1977 **Location:** California	**Outcome:** Mortality **Study Design:** Cohort (AHSMOG airport cohort) **Statistical Analyses:** Cox regression models **Age Groups:** Males, 27 yrs+	**Pollutant:** $PM_{10-2.5}$ **Averaging Time:** monthly averages **Mean (SD):** $PM_{10-2.5}$: 27.3 (8.6) **IQR:** 9.7 **Copollutant:** O_3: 0.70; SO_2: 0.31; NO_2: 0.23; SO_4: 0.47	**Increment:** IQR All Cause: 1.05 (0.92-1.20) Resp: 1.19 (0.88, 1.62) Lung Cancer: 1.25 (0.63-2.49)

Table E-35. Long-term exposure to $PM_{2.5}$ (including PM components/sources) and mortality.

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Abrahamowicz et al. (2003) **Period of Study:** 1982-1989 **Location:** 151 Cities	**Outcome:** Mortality: All-causes **Study Design:** Case-cohort study **Statistical Analyses:** Cox proportion-hazards model flexible regression spline generalization **Age Groups:** >18	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean (SD):** 18.2 **Range (Min, Max):** (9.0, 33.5) **Copollutant:** Sulfates	**Relative Risk (Min CI, Max CI)** **Estimated from graph (Figure 1):** RR for a 24.5 µg/m^3 increase in $PM_{2.5}$ over time Time 0: 0.5 (-1.1, 1.6); 2: 0.6 (0.2, 0.9); 4: 0.6 (0.3, 0.8) 6: 0.8 (0.3, 1.1); 8: -1.0 (-1.5, 1.0) RR for a 19.9 µg/m^3 increase in Sulfates over time Time 0: 0.1 (-0.2, 0.7); 2: 0.1 (-0.2, 0.4); 4: 0.0 (-0.4, 0.3) 6: 0.3 (-0.1, 0.5); 8: 0.4 (-0.4, 1.6)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Ballester et al. (2003) **Period of Study:** 2001-2002 **Location:** Europe	**Outcome:** Mortality- All-causes **Study Design:** Health Impact Assessment **Statistical Analyses:** Aphesis Network **Age Groups:** >30	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean (SD):** NR **Range (Min, Max):** NR	**Potential Reduction in the total burden of mortality (min CI, max CI) for four different decreases in annual $PM_{2.5}$ using a conservative estimate** Reduction to 25 µg/m^3 - 0.4 (0.1, 0.8) Reduction to 20 µg/m^3 - 0.8 (0.2, 1.6) Reduction to 15 µg/m^3 - 1.6 (0.4, 3.1) Reduction to 10 µg/m^3 - 3.0 (0.8, 5.8)
Reference: Beelen et al. (2008) **Period of Study:** 1987-1996 **Location:** Netherlands	**Outcome:** Mortality: Total (non-accidental) (<800) Cardio-respiratory (390-448, 490-496, 487, 480-486, 507) Pulmonary (460-519) Cardiovascular (400-440) Lung Cancer (162) Other-causes **Study Design:** Case-cohort study and prospective cohort **Statistical Analyses:** Cox proportion-hazards model **Age Groups:** 55-69	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean (SD):** 28.3 (2.1) µg/m^3 **Range (Min, Max):** (23.0, 36.8) **Copollutant (correlation):** NO_2: (>0.8) BS: (>0.8) SO_2: (>0.6)	**Increment:** 11 µg/m^3 **Relative Risk (Min CI, Max CI)** **RR for the association between exposures to $PM_{2.5}$ and cause specific mortality** Natural Cause: Full cohort: 1.06 (0.97, 1.16); Case cohort: 0.86 (0.66, 1.13) Cardiovascular: Full cohort: 1.04 (0.90, 1.21); Case cohort: 0.83 (0.60, 1.15) Respiratory: Full cohort: 1.07 (0.75, 1.52); Case cohort: 1.02 (0.56, 1.88) Lung Cancer: Full cohort: 1.06 (0.82, 1.38); Case cohort: 0.87 (0.52, 1.47) Other cause: Full cohort: 1.08 (0.96, 1.23); Case cohort: 0.85 (0.65, 1.12) **RR for the association between exposures to BS and cause specific mortality** Natural Cause: Full cohort: 1.05 (1.00, 1.11); Case cohort: 0.97 (0.83, 1.13) Cardiovascular: Full cohort: 1.04 (0.95, 1.13); Case cohort: 0.98 (0.81, 1.18) Respiratory: Full cohort: 1.22 (0.99, 1.50); Case cohort: 1.29 (0.91, 1.83) Lung Cancer: Full cohort: 1.03 (0.88, 1.20); Case cohort: 1.03 (0.77, 1.38) Other cause: Full cohort: 1.04 (0.97, 1.12); Case cohort: 0.91 (0.78, 1.07)
Reference: Chen et al. (2005) **Period of Study:** 1973-1998 **Location:** San Francisco, San Diego, Los Angeles, CA	**Outcome:** Mortality: CHD **Study Design:** Cohort **Statistical Analyses:** Cox proportion hazards model **Age Groups:** >25	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 25 years **Mean (SD):** 29.0 **Range (Min, Max):** NR **Copollutant:** NO_2, O_3, SO_2	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** Males $PM_{2.5}$: 0.89 (0.69, 1.17); 0-1 $PM_{2.5}$+NO_2: 0.82 (0.61, 1.10);0-1 $PM_{2.5}$+SO_2: 0.86 (0.65,1.14); 0-1 $PM_{2.5}$+O_3: 0.92 (0.65,1.29); 0-1 Females $PM_{2.5}$: 1.19 (0.96, 1.47); 0-1 $PM_{2.5}$+NO_2: 1.18 (0.95, 1.47);0-1 $PM_{2.5}$+SO_2: 1.36 (1.05,1.74); 0-1 $PM_{2.5}$+O_3: 1.61 (1.17,2.22); 0-1
Reference: Eftim et al. (2008) **Period of Study:** 2000-2002 **Location:** USA, Same cities as six cities and ACS cohorts	**Outcome (ICD-9):** All non-accidental causes (<800) **Study Design:** Cross-sectional **Statistical Analyses:** Log-linear regression **Age Groups:** >65	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** | ACS: 13.6 (2.8) SCS: 14.1 (3.1) **Range (Min, Max):** ACS: (6.0, 25.1);SCS: (9.6, 19.1)	**Increment:** 10 µg/m^3 **% Increase in Mortality for overall exposure period and individual year (95%CI Min, 95%CI Max):** ACS (adjusted for age, sex) Overall: 10.8 (8.6, 13.0); 2000: 10.9 (8.6, 13.0) 2001: 9.1 (5.3, 12.7); 2002: 10.1 (6.0, 14.3) SCS (adjusted for age, sex) Overall: 20.8 (14.8, 27.1); 2000: 17.8 (9.8, 26.4) 2001: 16.5 (7.4, 25.0); 2002: 33.5 (19.2, 49.3)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Enstrom et al. (2005) **Period of Study:** 1973-2002 **Location:** 25 California Colonies; 11 California Colonies (EPA IPN study)	**Outcome:** Mortality: Cardiovascular-respiratory (390-448); (480-486, 487, 490-496, 507) **Study Design:** Retrospective cohort **Statistical Analyses:** Cox proportional hazards regression model, SAS PHREG **Age Groups:** 35 or older	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual **Mean (SD):** 23.4 **Range (Min, Max):** (13.1 µg/m^3, 36.1)	**Relative Risk (Lower CI, Upper CI);** **RR from causes for both sexes by county from 1973-2002** Alameda: 0.962 (0.926,0.999) Butte: 0.999 (0.910,1.096); Contra Costa: 0.999 (0.943,1.058); Fresno: 0.935 (0.872,1.002); Humboldt: 0.992 (0.900,1.092); Kern: 0.944 (0.872,1.023); Marin: 0.939 (0.867,1.016); Napa: 0.949 (0.868,1.038); Orange: 0.990 (0.948,1.034); Riverside: 0.959 (0.906,1.015); Sacramento: 0.998 (0.944,1.055); San Bernardino: 0.992 (0.938,1.049); San Diego: 0.992 (0.954, 1.033); San Francisco: 0.963 (0.914,1.014); San Joaquin: 0.925 (0.816,1.049); San Mateo: 0.949 (0.899, 1.003); Santa Barbara: 0.968 (0.878,1.068); Santa Clara: 0.955 (0.910,1.003); Santa Cruz: 0.890 (0.793,0.999); Solano: 0.901 (0.815,0.995); Sonoma: 0.968 (0.884,1.060); Stanislaus: 0.984 (0.904,1.072); Tulare: 1.047 (0.979,1.119); Ventura: 0.967 (0.872,1.072) **RR from all causes for 11 counties for both sexes (EPA IPN study)** Santa Barbara: 0.968 (0.878,1.068); Contra Costa: 0.999 (0.943,1.058); Alameda: 0.962 (0.926,0.999); Butte: 0.999 (0.910,1.096); San Francisco: 0.963 (0.914,1.014); Santa Clara: 0.955 (0.910,1.003); Fresno: 0.935 (0.872,1.002); San Diego: 0.992 (0.954,1.033); Kern: 0.944 (0.872,1.023) Riverside: 0.959 (0.906,1.015)
Reference: Filleul et al. (2005) **Period of Study:** 1974-1976 **Location:** 7 cities in France	**Outcome:** Non-accidental causes (<800), cardiopulmonary disease (401-440 and 460-519), lung cancer (162) **Age Groups:** 25–59 years **Study Design:** Cohort **N:** 14,284 people **Statistical Analyses:** Cox proportional hazard, regression **Covariates:** Sex, smoking habits, educational level, body-mass index (BMI), occupational exposure **Statistical Package:** Proc Phreg SAS	**Pollutant:** Total suspended particles (TSP) **Averaging Time:** NR **Mean (SD):** NR **Range (Min, Max):** (45, 243) **PM Component:** NR **Monitoring Stations:** 1 station **Copollutant (correlation):** BS; r = 0.87 SO_2; r = 0.17 NO; r = 0.84 NO_2; r = 0.60	**Increment:** 10 µg/m^3 Adjusted mortality rate ratios: 24 areas: All non-accidental causes: 1.00[0.99, 1.01] Lung cancer: 0.97[0.94, 1.01] Cardiopulmonary disease: 1.01[0.99, 1.03] 18 areas: All non-accidental causes: 1.05[1.02, 1.08] Lung cancer: 1.00[0.92, 1.10] Cardiopulmonary disease: 1.06[1.01, 1.12]
Reference: Fuentes et al. (2006) **Period of Study:** June 2000 **Location:** Conterminous U.S.	**Outcome:** Mortality: **Study Design:** Time-series **Statistical Analyses:** Generalized Poisson Regression **Age Groups:** 0-14, 15-64, >65 **Covariates:** temperature, pressure, dew point, wind speed, elevation, age, ethnicity	**Pollutant:** $PM_{2.5}$ **Averaging Time:** monthly **Mean (SD):** 6.60 (0.76) **Copollutant:** PM_{10}, O_3	**Increment:** 10 µg/m^3 $PM_{2.5}$: 1.066 (1.064, 1.069) PM_{10}: 1.030 (1.028, 1.032)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Janes et al. (2007) **Period of Study:** 2000 to 2002 **Location:** 113 US counties	**Outcome:** Mortality: **Study Design:** Time-series **Statistical Analyses:** Cox proportional hazards model **Age Groups:** 65-74 75-84 85+	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual Avg **Mean (SD):** NR **Range (Min, Max):** NR	**Increment:** 1 µg/m^3 **% Increase (Lower CI, Upper CI); lag:** Overall % Increase by age-sex stratum Age Category 65-74: Male: 1.48 (0.93,2.03); Female: 0.83 (0.24,1.43) 75-84: Male: 0.85 (0.34,1.35); Female: 0.77 (0.28,1.27) 85+: Male: 0.70 (0.03,1.38); Female: 0.59 (0.05,1.12) National Trend % Increase by age-sex stratum Age Category 65-74: Male: 3.55 (2.77,4.34); Female: 1.97 (1.12,2.83) 75-84: Male: 2.48 (1.83,3.14); Female: 2.29 (1.66,2.93) 85+: Male: 1.38 (0.52,2.26); Female: 1.65 (1.01,2.29) Local Trend % Increase by age-sex stratum Age Category 65-74: Male: 0.04 (-0.58,0.67); Female: -0.03 (-0.71,0.66) 75-84: Male: -0.34 (-0.87,0.19); Female: -0.31 (-0.82, 0.21) 85+: Male: <0.01 (-0.71,0.73); Female: -0.22 (-0.74,0.31) *Local trends are county specific deviations from national trends
Reference: Jerrett et al. (2003) **Period of Study:** 1982 **Location:** 151 cities from ACS	**Outcome:** Mortality **Study Design:** multilevel, individual-ecologic analysis **Statistical Analysis:** Cox proportional hazards model **Covariates:** Smoking, education, occupational exposures, BMI, marital status, alcohol consumption, gender	**Pollutant:** Sulfates **Mean (SD):** 10.6 **Range (Min, Max):** 3.6,23.5	**Increment:** 19.9 (Range) All Cause: SO_4: 1.17 (1.07, 1.27); SO_4 + CO: 1.16 (1.10, 1.23) SO_4 + NO_2: 1.16 (1.08, 1.24); SO_4 + O_3: 1.17 (1.11, 1.24) SO_4 + SO_2: 1.05 (0.98, 1.12) CPD: SO_4: 1.25 (1.16, 1.35); SO_4 + CO: 1.28 (1.18, 1.39) SO_4 + NO_2: 1.29 (1.17, 1.42); SO_4 + O_3: 1.27 (1.17, 1.38) SO_4 + SO_2: 1.13 (1.03, 1.24) Lung Cancer: SO_4: 1.31 (1.09, 1.58); SO_4 + CO: 1.26 (1.03, 1.53) SO_4 + NO_2: 1.31 (1.05, 1.65); SO_4 + O_3: 1.30 (1.07, 1.59) SO_4 + SO_2: 1.37 (1.08, 1.73)
Reference: Jerrett et al. (2005) **Period of Study:** 1982-2000 **Location:** Los Angeles, California	**Outcome:** Mortality: Non- accidental (<800) IHD (410-414) Cardiopulmonary (400-440, 460-519) Lung Cancer (162) Other Cancers (140-149,160, 161, 163-239) Other causes **Study Design:** Retrospective Cohort **Statistical Analyses:** Cox regression hazards model; kriging, radial basis function multiquadric interpolator **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** NR **Range (Min, Max):** NR **Copollutant:** O_3	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI)** All Causes - $PM_{2.5}$ Only: 1.24 (1.11,1.37) 44 Ind. Covariates together+$PM_{2.5}$: 1.17 (1.03,1.32) 44 Ind. Covariates together+ $PM_{2.5}$+O_3: 1.20 (1.07,1.34) 44 Ind. Covariates together+intersection within freeways within 500 m+ $PM_{2.5}$+O_3: 1.17 (1.05,1.31) IHD - $PM_{2.5}$ Only: 1.49 (1.20,1.85) 44 Ind. Covariates together+$PM_{2.5}$: 1.39 (1.12,1.73) 44 Ind. Covariates together+$PM_{2.5}$+O_3: 1.45 (1.15,1.82) 44 Ind. Covariates together+intersection within freeways within 500 m+ $PM_{2.5}$+O_3: 1.38 (1.11,1.72) Cardiopulmonary - $PM_{2.5}$ Only: 1.20 (1.04,1.39) 44 Ind. Covariates together+ $PM_{2.5}$+O_3: 1.19 (1.02,1.38) 44 Ind. Covariates together+intersection within freeways within 500 m+ $PM_{2.5}$+O_3: 1.13 (0.97,1.31) Lung Cancer - $PM_{2.5}$ Only: 1.60 (1.09,2.33) 44 Ind. Covariates together+$PM_{2.5}$: 1.44 (0.98,2.11) 44 Ind. Covariates together+intersection within freeways within 500 m+ $PM_{2.5}$+O_3: 1.46 (0.99,2.16) Other Cancers - $PM_{2.5}$ Only: 1.09 (0.85,1.40) 44 Ind. Covariates together+ $PM_{2.5}$+O_3: 1.08 (0.83,1.39) 44 Ind. Covariates together+intersection within freeways within 500 m+ $PM_{2.5}$+O_3: 1.08 (0.83,1.39) All Other Causes - $PM_{2.5}$ Only: 1.11 (0.74,1.67) 44 Ind. Covariates together+ $PM_{2.5}$+O_3: 0.95 (0.64,1.39) 44 Ind. Covariates together+intersection within freeways within 500 m+ $PM_{2.5}$+O_3: 1.02 (0.71,1.48)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Laden et al. (2006) **Period of Study:** 1974-1998 Period 1: 1974-1989 Period 2: 1990-1998 **Location:** Nine US Cities Watertown, MA Kingston, TN Harriman, TN St. Louis, MO Steubenville, OH Portage, WI |Wyocena, WI Pardeeville, WI Topeka, KS	**Outcome:** Total mortality Non-accidental (<800) Cardiovascular (400-440) Respiratory (485-496) Lung Cancer (162) Other **Study Design:** Prospective Cohort **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** 25-74	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** Period 1 Portage: 11.4 Topeka: 12.4 Watertown:15.4 Harriman: 20.9 St Louis: 19.2 Steubenville: 29.0 Period 2 Portage: 10.2 Topeka: 13.1 Watertown: 12.1 Harriman: 18.1 St. Louis: 13.4 Steubenville: 22.0	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI); lag:** **Period 1:** Portage: 1.00; Topeka: 1.06 (0.86, 1.31); Watertown: 1.06 (0.87, 1.28); Harriman: 1.19 (0.98, 1.44); St Louis: 1.15 (0.96, 1.38); Steubenville: 1.31 (1.10, 1.57) **Period 2:** Portage: NR; Topeka: 1.01 (0.83, 1.22); Watertown: 0.82 (0.67, 1.00); Harriman: 1.10 (0.91, 1.33); St Louis: 0.96 (0.80, 1.15); Steubenville: 1.06 (0.89, 1.27) **Complete Period:** Portage: 1.00; Topeka: 1.03 (0.89, 1.19); Watertown: 0.95 (0.83, 1.08); Harriman: 1.15 (1.01, 1.32); St. Louis: 1.05 (0.93, 1.20); Steubenville: 1.18 (1.04, 1.34) **RR for complete follow up Avg.** $PM_{2.5}$ Total Mortality: 1.16 (1.07, 1.26) Cardiovascular: 1.28 (1.13, 1.44) Respiratory: 1.08 (0.79, 1.49) Lung Cancer: 1.27 (0.96, 1.69) Other: 1.02 (0.90, 1.17) RR for period one Avg. $PM_{2.5}$ Total Mortality: 1.18 (1.09, 1.27) Cardiovascular: 1.28 (1.14, 1.43) Respiratory: 1.21 (0.89, 1.66) Lung Cancer: 1.20 (0.91, 1.58) Other: 1.05 (0.93, 1.19) **Decrease in Avg.** $PM_{2.5}$ **over the two periods** Total Mortality: 0.73 (0.57, 0.95) Cardiovascular: 0.69 (0.46, 1.01) Respiratory: 0.43 (0.16, 1.13) Lung Cancer: 1.06 (0.43, 2.62) Other: 0.85 (0.56, 1.27)
Reference: Lipfert et al. (2006a) **Period of Study:** 1989-1996 **Location:** Various parts of the Untied States	**Outcome:** Mortality **Study Design:** Retrospective Cohort **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** Male US veterans between ages of 39 and 63 (Avg. age: 51)	**Pollutant:** Sulfate **Mean (SD):** 10.7 (3.6)	**Increment:** 8 1.05 (0.94, 1.16)
Reference: Lipfert et al. (2006a) **Period of Study:** 1989-1996 **Location:** Various parts of the Untied States	**Outcome:** Mortality **Study Design:** Retrospective Cohort **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** Male US veterans between ages of 39 and 63 (Avg age 51)	**Pollutant:** $PM_{2.5}$ **Mean (SD):** 14.3 (3.2)	**Increment:** 8 1.12 (1.04, 1.20)
Reference: Lipfert et al. (2006b) **Period of Study:** 1997-2002 **Location:** Various parts of the Untied States	**Outcome:** Mortality: Non- accidental (<800) **Study Design:** Retrospective cohort **Statistical Analyses:** Cox proportional hazards regression; AIC **Age Groups:** Male US veterans between ages of 39 and 63 (Avg. age: 51)	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** 15.02 (4.80) µg/m^3 (2000-2003) **Range (Min, Max):** (3.29, 24.96) **Copollutant (correlation):** As: r = 0.443; Cr: r = 0.379; Cu: r = 0.530; Fe: r = 0.379; Pb: r = 0.489; Mn: r = 0.389; Ni: r = 0.140; Se: r = 0.312; V: r = 0.197; Zn: r = 0.420; OC: r = 0.620; EC: r = 0.544;| SO_4: r = 0.827; NO_3: r = 0.649; NO_2:r = 0.641 Peak CO: r = 0.040 Peak O_3: r = 0.222 Peak SO_2: r = 0.714	**Increment:** 10 µg/m^3 **% Increase per 10** µg/m^3 **increase in** $PM_{2.5}$ **Single-Pollutant Model** As: -5.23%; Cr: -2.11%; Cu: 2.12%; Fe: 2.81%; Pb: -2.40%; Mn: -1.20%; Ni: 3.75%; Se: -0.30%; V: 5.08%; Zn: 1.52%; OC: -0.02%; EC: 9.16%; ; SO_4: 3.04%; NO_3: 6.60%; NO_2: 6.92% Peak CO: -0.61%; Peak O_3: 4.95%; Peak SO_2: -4.20% **Multiple Pollutants model- Pollutant with traffic density** NO_3: 3.42%; SO_4: -2.73%; EC: 6.27%; Ni: 2.51%; V: 3.27% **Pollutant with NO_3** EC: 5.93%; Ni: 2.31%; V: 3.11% **Pollutant with Peak O_3** **Traffic density: 2.40%** EC: 10.79%; Fe: 5.94%; NO_3: 7.57%; $PM_{2.5}$: 8.97%; V: 4.93%; Ni: 3.65%; SO_4: 6.75%; Cu: 1.55%; OC: 0.21%

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: McDonnell et al. (2000) **Period of Study:** 1973-1977 **Location:** California	**Outcome:** Mortality **Study Design:** Cohort (AHSMOG airport cohort) **Statistical Analyses:** Cox regression models **Age Groups:** Males, 27 yrs+	**Pollutant:** $PM_{2.5}$ **Averaging Time:** monthly averages **Mean (SD):** 31.9 (10.7) **IQR:** 24.3 **Copollutants (correlation):** O_3: 0.68; SO_2: 0.18; NO_2: -0.08; SO_4: 0.33	**Increment:** IQR All Cause: 1.22 (0.95-1.58) Resp: 1.64 (0.93-2.90) Lung Cancer: 2.23 (0.56-8.94)
Reference: Miller et al. (2007) **Period of Study:** 1994-1998 **Location:** 36 US Metropolitan Areas	**Outcome:** CVD Mortality **Study Design:** Prospective Cohort (WHI) **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** postmenopausal women ages 50-79	**Pollutant:** $PM_{2.5}$ **Averaging Time:** annual avg (2000) **Mean (SD):** 13.4 **IQR:** 11.6, 18.3 **Range:** 3.4, 28.3	**Increment:** 10 µg/m^3 CVD Death: 1.76 (1.25, 2.47) CHD Death: 2.21 (1.17, 4.16) CV Death: 1.83 (1.11, 3.00)
Reference: Naess et al. (2007a) **Period of Study:** 1992-1998 **Location:** Oslo, Norway	**Outcome:** Mortality: Non-accidental (<800) Lung cancer (162) COPD (490-496) Cardiovascular (390-459) **Study Design:** Prospective Cohort **Statistical Analyses:** Cox proportional hazards regression model **Age Groups:** 51-70, 71-90	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 4 year avg **Mean (SD):** $PM_{2.5}$: 15 **Range (Min, Max):** $PM_{2.5}$: (7, 22) **Copollutant (correlation):** NO_2: r = 0.95	**Relative Risk (CI min, CI max)** RR for deaths from all causes Men (ages 51-70) $PM_{2.5}$ exposure (in µg/m^3) 6.56-11.45: 1.00 11.46-14.25: 0.96 (0.89, 1.04) 14.26-18.43: 1.12 (1.03, 1.22) 18.44-22.34: 1.48 (1.36, 1.60) Men (ages 71-90) $PM_{2.5}$ exposure (in µg/m^3) 6.56-11.45: 1.00 11.46-14.25: 0.99 (0.93, 1.06) 14.26-18.43: 1.10 (1.03, 1.17) 18.44-22.34: 1.19 (1.12, 1.27) Women (ages 51-70) $PM_{2.5}$ exposure (in µg/m^3) 6.56-11.45: 1.00 11.46-14.25: 0.96 (0.87, 1.07) 14.26-18.43: 1.08 (0.98, 1.20) 18.44-22.34: 1.44 (1.30, 1.59) Women (ages 71-90) $PM_{2.5}$ exposure (in µg/m^3) 6.56-11.45: 1.00 11.46-14.25: 1.03 (0.97, 1.09) 14.26-18.43: 1.07 (1.01, 1.12) 18.44-22.34: 1.11 (1.05, 1.16) **Increment:** 10 µg/m^3 RR for death from CVD and lung cancer Men (ages 51-70) CVD- $PM_{2.5}$: 1.11 (1.06, 1.16) COPD- $PM_{2.5}$: 1.32 (1.17, 1.49) Lung Cancer- $PM_{2.5}$: 1.07 (0.98, 1.17) Women (ages 51-70) CVD: $PM_{2.5}$: 1.16 (1.09, 1.24) COPD: $PM_{2.5}$: 1.18 (1.03, 1.34) Lung Cancer: $PM_{2.5}$: 1.23 (1.10, 1.37) Men (ages 71-90) CVD: $PM_{2.5}$: 1.06 (1.03, 1.09) COPD: PM_{10}: 1.13 (1.04, 1.24) $PM_{2.5}$: 1.14 (1.04, 1.24) Lung Cancer: $PM_{2.5}$: 1.08 (0.98, 1.19) Women (ages 71-90) CVD: $PM_{2.5}$: 1.02 (1.00, 1.05) COPD: $PM_{2.5}$: 1.09 (1.00, 1.18) Lung Cancer: $PM_{2.5}$: 1.16 (1.03, 1.31)
Reference: Naess et al. (2007b) **Period of Study:** 1992-1995 **Location:** Oslo, Norway	**Outcome:** Mortality: Lung cancer (162) COPD (490-496) Cardiovascular (390-459) Psychiatric causes (290, 292-302, 304, 306-319) Stomach cancer (151) Violence (800-999) **Study Design:** Multilevel	**Pollutant:** $PM_{2.5}$ **Averaging Time:** (Month-year) avg **Range Mean (SD):** 14.2 (3.6) **IQ Range (1st, 4th):** (6.6, 22.3) **Copollutant (correlation):** PM_{10}: r = 0.95| NO_2: r = 0.87	**Relative Risk (CI min, CI max)** **RR on All-cause mortality of $PM_{2.5}$ in Men Age 50-74** Primary Education: $PM_{2.5}$: 1.06 (1.00, 1.11); Individual: 1.34 (1.24, 1.43); Neighborhood: 1.22 (1.16, 1.28) Manual Class: $PM_{2.5}$: 1.06 (1.01, 1.12); Individual: 1.28 (1.20, 1.37); Neighborhood: 1.20 (1.14, 1.26) Income below median: $PM_{2.5}$: 1.05 (1.00, 1.12); Individual: 1.44 (1.35, 1.53); Neighborhood: 1.16 (1.11, 1.21)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
	cohort **Statistical Analyses:** WinBUGS **Age Groups:** 50-74		Not owner occupied: $PM_{2.5}$: 1.06 (1.00, 1.13); Individual: 1.24 (1.12, 1.36); Neighborhood: 1.11 (1.05, 1.17) Lives in flat dwelling: $PM_{2.5}$: 1.04 (0.98, 1.11); Individual: 1.19 (1.09, 1.31); Neighborhood: 1.10 (1.04, 1.17) More than one person per room in dwelling: $PM_{2.5}$: 1.10 (1.02, 1.18); Individual: 1.05 (0.98, 1.13); Neighborhood: 1.01 (0.96, 1.05) **RR on All-cause mortality of $PM_{2.5}$ in Women Age 50-74** Primary Education Only: $PM_{2.5}$: 1.05 (1.00, 1.11); Individual: 1.32 (1.23, 1.42); Neighborhood: 1.18 (1.12, 1.24) Manual Class: $PM_{2.5}$: 1.07 (1.01, 1.13); Individual: 1.27 (1.18, 1.36); Neighborhood: 1.18 (1.12, 1.24) Income below median: $PM_{2.5}$: 1.05 (1.01, 1.10); Individual: 1.52 (1.41, 1.63); Neighborhood: 1.13 (1.09, 1.18) Not owner occupied: $PM_{2.5}$: 1.07 (1.01, 1.14); Individual: 1.24 (1.12, 1.38); Neighborhood: 1.08 (1.02, 1.14) Lives in a flat dwelling: $PM_{2.5}$: 1.05 (0.99, 1.11); Individual: 1.21 (1.09, 1.34); Neighborhood: 1.09 (1.02, 1.15) More than one person per room in dwelling: $PM_{2.5}$: 1.11 (1.04, 1.19); Individual: 1.07 (0.99, 1.14); Neighborhood: 1.01 (0.96, 1.05) **RR for Interquartile Increase (MI) in $PM_{2.5}$ for different causes of death** CVD: Age and sex adjusted: 1.11 (1.07, 1.15) Primary education only: M1+ Individual: 1.07 (1.04, 1.11); M1+ Neighborhood: 1.03 (1.00, 1.07) Manual Class: M1+ Individual: 1.08 (1.04, 1.11); M1+ Neighborhood: 1.06 (1.02, 1.10) Income below Median: M1+ Individual: 1.07 (1.03, 1.11); M1+ Neighborhood: 1.02 (0.98, 1.05) Not owner occupied: M1+ Individual: 1.05 (1.01, 1.09); M1+ Neighborhood: 1.03 (0.99, 1.07): Living in a Flat dwelling M1+ Individual: 1.04 (1.00, 1.08); M1+ Neighborhood: 1.01 (0.97, 1.05) Crowded household: M1+ Individual: 1.10 (1.05, 1.14); M1+Neighborhood: 1.10 (1.06, 1.15) Pulmonary Cancer: Age and sex adjusted: 1.12 (1.05, 1.19) Primary education only: M1+ Individual: 1.09 (1.01, 1.17); M1+ Neighborhood: 1.05 (0.98, 1.13) Manual Class: M1+ Individual: 1.09 (1.01, 1.17); M1+ Neighborhood: 1.10 (1.06, 1.13) Income below Median: M1+ Individual: 1.09 (1.01, 1.17); M1+ Neighborhood: 1.02 (0.95, 1.10) Not owner occupied: M1+ Individual: 1.07 (1.00, 1.15); M1+ Neighborhood: 1.04 (0.97, 1.12) Living in a Flat dwelling: M1+ Individual: 1.03 (0.96, 1.11); M1+ Neighborhood: 1.00 (0.92, 1.08) Crowded household: M1+ Individual: 1.10 (1.03, 1.14); M1+Neighborhood: 1.11 (1.04, 1.20) COPD: Age and sex adjusted: 1.17 (1.09, 1.25) Primary education only: M1+ Individual: 1.13 (1.05, 1.22); M1+ Neighborhood: 1.09 (1.01, 1.19) Manual Class: M1+ Individual: 1.14 (1.05, 1.23); M1+ Neighborhood: 1.12 (1.04, 1.22) Income below Median: M1+ Individual: 1.13 (1.04, 1.22); M1+ Neighborhood: 1.06 (0.97, 1.15) Not owner occupied: M1+ Individual: 1.10 (1.02, 1.19); M1+ Neighborhood: 1.07 (0.99, 1.16) Living in a Flat dwelling: M1+ Individual: 1.08 (1.00, 1.18); M1+ Neighborhood: 1.03 (0.95, 1.13) Crowded household: M1+ Individual: 1.16 (1.07, 1.26); M1+Neighborhood: 1.16 (1.07, 1.26)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Nerriere et al. (2005) **Period of Study:** Grenoble (2001) Paris (2002) Rouen (2002-2003) Strasbourg (2003) **Location:** Four French Cities- Grenoble, Rouen, Paris, and Strasbourg	**Outcome:** Mortality: Lung Cancer (162) **Study Design:** Time-series **Statistical Analyses:** GIS **Age Groups:** 30-71 year old nonsmoking adults	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 48-h avg **Mean Range:** 17 to 49 µg/m^3	**Increment:** 10 µg/m^3 **% Increase (Lower CI, Upper CI)** % increase in lung cancer deaths attributable to $PM_{2.5}$ exposure France: 8 (1, 16) Grenoble: 10 (3, 19) Rouen: 10 (2, 19) Strasbourg: 24 (4, 40)
Reference: Ozkaynak and Thurston (1987) **Period of Study:** 1980 **Location:** U.S.	**Outcome:** Total Mortality **Study Design:** Cross-sectional **Statistical Analyses:** Multiple regression analysis	**Pollutant:** Sulfate **Averaging Time:** Annual avg **Mean Range:** Sulfate: 11.1 (3.5)	**Range of estimated total mortality effects of air pollutions:** Sulfate: 4-9% "Sulfate concentration was consistently found to be a significant predictor of mortality in the models considered. Fine particle mass coefficients were also often found to be statistically significant in the mortality regressions."
Reference: Pope et al. (2004) **Period of Study:** 1982-2000 **Location:** Metropolitan areas in all 50 states in the US	**Outcome:** Mortality: Cardiovascular Diseases (390-459) Diabetes (250) Respiratory Disease (460-519) **Study Design:** Prospective Cohort **Statistical Analyses:** Cox proportional hazards regression **Age Groups:** >30	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** 17.1 (3.7) **Range (Min, Max):** NR	**Increment:** 10 µg/m^3 **Relative Risk (Lower CI, Upper CI)** All cardiovascular disease plus diabetes: $PM_{2.5}$: 1.12 (1.08, 1.15) Former Smoker: 1.26 (1.23, 1.28); Current Smoker: 1.94 (1.90, 1.99) Ischemic Heart Disease: $PM_{2.5}$: 1.18 (1.14, 1.23) Former Smoker: 1.33 (1.29, 1.37); Current Smoker: 2.03 (1.96, 2.10) Diabetes: $PM_{2.5}$: 0.99 (0.86, 1.14) Former Smoker: 1.05 (0.94, 1.16); Current Smoker: 1.35 (1.20, 1.53) All other Cardiovascular Diseases: $PM_{2.5}$: 0.84 (0.71, 0.99) Former Smoker: 1.22 (1.09, 1.38); Current Smoker: 1.78 (1.56, 2.04) Diseases of the respiratory system: $PM_{2.5}$: 0.92 (0.86, 0.98) Former Smoker: 2.16 (2.04, 2.28); Current Smoker: 3.88 (3.66, 4.11) COPD: $PM_{2.5}$: 0.84 (0.77, 0.93) Former Smoker: 4.93 (4.48, 5.42); Current Smoker: 9.85 (8.95, 10.84) All other respiratory diseases: $PM_{2.5}$: 0.86 (0.73, 1.02) Former Smoker: 1.54 (1.36, 1.74); Current Smoker: 1.83 (1.57, 2.12)
Reference: Pope et al. (2007) **Period of Study:** 1960-1975 **Location:** New Mexico, Arizona, Utah, and Nevada	**Outcome (ICD7&8):** Mortality: Cardiovascular (ICD 7: 400-468, 331, 332 ICD 8: 390-458) Respiratory (ICD 7: 470-527 ICD 8: 460-519) Influenza/ pneumonia (ICD 7: 480-483, 490-493, ICD 8: 470-474, 480-486) **Study Design:** Retrospective Cohort **Statistical Analyses:** Poisson regression model; GAM; SAS **Age Groups:** All smelter workers >18	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** NR **Range (Min, Max):** NR	The study does not present quantitative results; results are presented in figures. The References found that the strike-related estimated percent decrease in mortality was 2.5% (1.1-4.0),

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Rainham et al. (2005) **Period of Study:** 1981-1999 **Location:** Toronto, Canada	**Outcome:** Total deaths (ICD9 <800), cardiorespiratory (390-459), non-cardiorespiratory (ICD9-NR) **Study Design:** Time-series **Statistical Analyses:** Generalized linear models were used **Season:** Winter (December–February) Summer (June–August) **Statistical Package:** S-Plus 6.1	**Pollutant:** $PM_{2.5}$ **Averaging Time:** NR **Mean (SD):** All years: 17.0 (8.7) µg/m³ Winters: 17.2 (6.8) Summers: 18.8 (10.2) Avg Winter values: Dry Moderate: 17.0 (1.0) Dry Polar: 17.5 (0.5) Dry Tropical: No Comparison Moist Moderate: 17.1 (0.8) Moist Polar: 17.5 (0.6) Moist Tropical: 16.5 (3.6) Transition: 16.7 (1.0) Avg summer values: Dry Moderate: 18.4 (0.9) Dry Polar: 19.0 (1.2) Dry Tropical: 18.5 (2.4) Moist Moderate: 19.2 (1.2) Moist Polar: 17.5 (2.0) Moist Tropical: 19.8 (1.1) Transition: 17.6 (1.5)	**Mortality risk for winter season and within winter synoptic weather categories; RR Estimate [Lower CI, Upper CI]:** Winter: Total: 0.998[0.997, 1.000] Cardioresp: 0.998[0.996, 1.000]; Other: 0.998 [0.996, 1.000] Dry Moderate: Total: 1.001[0.996, 1.007] Cardioresp: 1.005[0.998, 1.011]; Other: 1.002 [0.998, 1.005] Dry Polar: Total: 0.998[0.995, 1.001] Cardioresp: 0.995[0.991, 0.999]; Other: 1.002 [0.998, 1.005] Dry Tropical: NA Moist Moderate: Total: 0.998[0.993, 1.002] Cardioresp: 1.003[0.995, 1.010]; Other: 0.997 [0.991, 1.004] Moist Polar: Total: 1.001[0.998, 1.005] Cardioresp: 1.002[0.997, 1.007]; Other: 1.003 [0.999, 1.007] Moist Tropical: Total: 1.007[0.965, 1.203] Cardioresp: 1.123[1.031, 1.224]; Other: 1.248 [1.123, 1.387] Transition Total: 1.003[0.996, 1.009] Cardioresp: 0.996[0.987, 1.004]; Other: 0.997 [0.990, 1.004] **Mortality risk for summer season and within summer synoptic weather categories; RR Estimate [Lower CI, Upper CI]:** Summer: Total: 1.000[1.000, 1.001] Cardioresp: 1.001[1.000, 1.002]; Other: 1.001[1.000, 1.002] Dry Moderate: Total: 1.001[0.999, 1.002] Cardioresp: 1.002[0.999, 1.004]; Other: 0.999[0.997, 1.002] Dry Polar: Total: 1.002[0.999, 1.005] Cardioresp: 0.996[0.991, 1.000]; Other: 1.003[0.999, 1.007] Dry Tropical: Total: 1.016[1.006, 1.027] Cardioresp: 1.017[1.005, 1.030]; Other: 1.017 [1.003, 1.031] Moist Moderate: Total: 1.002[1.000, 1.004] Cardioresp: 1.003[0.999, 1.006]; Other: 1.004 [1.001, 1.006] Moist Polar: Total: 1.005[0.998, 1.011] Cardioresp: 1.008[0.997, 1.018]; Other: 1.003 [0.995, 1.011] Moist Tropical: Total: 0.999[0.997, 1.001] Cardioresp: 0.996[0.993, 1.000]; Other: 0.998 [0.995, 1.001] Transition: Total: 1.005[0.996, 1.014] Cardioresp: 1.007[0.994, 1.020]; Other: 1.002 [0.996, 1.008]
Reference: Roman et al. (2008) **Period of Study:** 2006 **Location:** U.S.	**Outcome:** Mortality **Study Design:** Expert Judgment Study **Statistical Analyses:** Standard best practices for expert elicitation	**Pollutant:** $PM_{2.5}$ **Averaging Time:** annual avergage **Mean (SD):** 4-30	Quantitative results are not presented in the text, but can be found graphically in Figure 3. "Most of the experts' central estimates fall at or above the 2002 ACS median (0.6% per µg/m³) and below the original Six Cities median (1.2% per µg/m³)."
Reference: Schwartz, et al (2002) **Period of Study:** 1979-1988 **Location:** Six U.S. metropolitan areas: Boston, Massachusetts; Knoxville, Tennessee; St. Louis, Missouri; Stuebenville, Ohio; Madison, Wisconsin; and Topeka, Kansas	**Outcome:** Mortality **Study Design:** Poisson regression **Statistical Analyses:** Weighted linear regression **Season:** all **Dose-response Investigated?** No **Statistical Package:** S-plus	**Pollutant:** $PM_{2.5}$ **Averaging Time:** daily **Mean (SD):** Boston-16.5 Knoxville-21.1 St. Louis-19.2 Steubenville-30.5 Madison-11.3 Topeka-12.2 SD not reported **Range (Min, Max):** (0,35) **Monitoring Stations:** 6	**PM Increment:** 10 µg/m³ The difference between mean $PM_{2.5}$ concentrations of 10 µg/m³ and 20 µg/m³ is associated with about a 1.5% increase in deaths.
Reference: Schwartz et al. (2008) **Period of Study:** 1974-1998 **Location:** Watertown, MA Kingston and Harriman, TN St Louis, MO Steubenville, OH Portage, Wyocena Pardeeville WI Topeka, KS	**Outcome:** Mortality: Non-accidental (<800) **Study Design:** Cross-sectional **Statistical Analyses:** Cox proportional hazards regression; penalized splines; Bayesian Model Averaging **Age Groups:** >18	**Pollutant:** $PM_{2.5}$ **Averaging Time:** Annual avg **Mean (SD):** 17.5 (6.8) **Range (Min, Max):** (8, 40)	**Increment:** 10 µg/m³ **Relative Risk (Lower CI, Upper CI)** **Estimated from Figure 4:** All Cause Mortality - Year before Death 0: 1.10 (1.00, 1.21); 1: 1.03 (0.98, 1.08); 2: 1.01 (1.00, 1.02) 3: 1.00 (0.99, 1.01); 4: 1.00 (0.99, 1.01); 5: 1.00 Lung Cancer Mortality - Year Before Death **Estimated from Figure 5** 0: 1.18 (1.00, 1.48); 1: 1.12 (0.98, 1.33); 2: 1.08 (0.92, 1.22) 3: 1.02 (1.01, 1.03); 4: 1.01 (1.00, 1.02); 5: 1.01 RR per 10 µg/m³ increase of $PM_{2.5}$ exposure Level Of Increase **Estimated from Figure 3** 10 µg/m³: 1.15; 20 µg/m³: 1.29; 30 µg/m³: 1.46; 40 µg/m³: 1.64

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Tainio et al. (2005) **Period of Study:** 1997-Present **Location:** Helsinki, Finland	**Outcome (ICD10):** Mortality: Cardiopulmonary (I11-I70 and J15-J47); Lung Cancer (C34); Other causes **Study Design:** Time-series simulation **Statistical Analyses:** Monte Carlo Simulation **Age Groups:** All ages	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** 10.7 **Range (Min, Max):** NR	**Estimated Deaths Per Year (Min CI, Max CI) Associated with Primary $PM_{2.5}$ Emissions from buses in Helsinki in 2020 for different bus strategies** Cardiopulmonary Mortality Current Fleet: 15.9 (0, 46.6); Modern Diesel: 7.9 (0, 23.0); Diesel with particle trap: 3.9 (0, 12); Natural gas bus: 2.3 (0, 6.8) Lung Cancer Mortality Current Fleet: 2.2 (0, 6.1); Modern Diesel: 1.1 (0, 3.0); Diesel with particle trap: 0.6 (0, 1.6); Natural gas bus: 0.3 (0, 0.9) Total Mortality Current Fleet: 18.1 (0, 55.0); Modern Diesel: 9.0 (0, 27.0); Diesel with particle trap: 4.4 (0, 14.1); Natural Gas Bus: 2.6 (0, 8.0)
Reference: Villeneuve et al. (2002) **Period of Study:** 1974-1991 **Location:** Six US Cities: Steubenville, OH, St. Louis, MO, Portage, WI, Topeka, KS, Watertown, MA, Kingston/ Harriman, TN	**Outcome (ICD10):** Mortality: Non-accidental (<800) **Study Design:** Prospective Cohort **Statistical Analyses:** Poisson, EPICURE **Age Groups:** All ages <60 ≥ 60	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 24-h avg **Mean (SD):** Portage: 10.9 (7.2) Topeka: 12.1 (7.1) Harriman: 20.7 (9.4) Watertown: 14.9 (8.4) St. Louis: 18.7 (10.6) Steubenville: 28.6 (21.0) Overall: 18.6 **Range (Min, Max):** NR	**Increment:** 18.6 µg/m^3 **Relative Risk (Min CI, Max CI)** RR of all cause mortality for exposure of $PM_{2.5}$ by age group Exposure to $PM_{2.5}$ remained fixed over entire study period <60: 1.89 (1.32, 2.69); >60: 1.21 (1.02, 1.43) Total: 1.31 (1.12, 1.52) Exposure to $PM_{2.5}$ was defined according to 13 calendar periods* (no smoothing) <60: 1.52 (1.15, 2.00); >60: 1.11 (0.95, 1.29) Total: 1.19 (1.04, 1.36) Exposure to $PM_{2.5}$ was defined according to 13 calendar periods* (smoothed) <60: 1.43 (1.10, 1.85); >60: 1.09 (0.93, 1.26) Total: 1.16 (1.02, 1.32) Time dependent estimate of $PM_{2.5}$ received during the previous two years <60: 1.42 (1.09, 1.82); >60: 1.08 (0.94, 1.25) Total: 1.16 (1.02, 1.31) Time dependent estimate of $PM_{2.5}$ received 3-5 years before current year <60: 1.35 (1.08, 1.67); >60: 1.08 (0.95, 1.22) Total: 1.14 (1.02, 1.27) Time dependent estimate of $PM_{2.5}$ received >5 years before current year <60: 1.34 (1.11, 1.59); >60: 1.09 (0.99, 1.20) Total: 1.14 (1.05, 1.23) * The calendar periods used were: 1970-1978, 1979, 1980, 1981, 1982, 1983, 1984, 1985, 1986, 1987, 1988, 1989, and 1990+. RR of all cause mortality and $PM_{2.5}$ exposure by city Portage: 1.16 (0.96, 1.39) Topeka: 1.06 (0.89, 1.27) Harriman Men: 1.04 (0.79, 1.36); Women: 0.96 (0.69, 1.31) All: 1.13 (0.95, 1.35) Watertown Men: 1.20 (0.95, 1.51); Women: 1.06 (0.78, 1.43) All: 1.32 (1.11, 1.51) St. Louis Men: 0.97 (0.76, 1.24); Women: 1.13 (0.86, 1.49) Steubenville Men: 1.39 (1.11, 1.74); Women: 1.22 (0.93, 1.61)
Reference: Willis et al. (2003) **Period of Study:** 1982-1989 **Location:** US Metropolitan areas in all 50 states	**Outcome:** Mortality: All causes Lung Cancer (162) Cardiopulmonary (401-440, 460-519) **Study Design:** Prospective Cohort **Statistical Analyses:** Cox proportional hazards model **Age Groups:** All ages	**Pollutant:** Sulfates **Averaging Time:** Annual avg **Mean (SD):** 10.6 **Range (Min, Max):** 3.6, 23.5 **Copollutant:** CO, NO_2, O_3, SO_2	All Cause, Metropolitan Scale: 1.25 (1.13, 1.37) All Cause, County Scale: 1.50 (1.30, 1.73) CPD, Metropolitan Scale: 1.29 (1.15, 1.46) CPD, County Scale: 1.75 (1.48, 2.08)

Study	Design & Methods	Concentrations	Effect Estimates (95% CI)
Reference: Zeger et al. (2007) **Period of Study:** 2000-2002 **Location:** 250 largest US counties	**Outcome:** Mortality **Study Design:** Retrospective Cohort (MCAPS) **Statistical Analyses:** log-linear regression models (GAM) **Covariates:** age, gender, race, county-level SES, education and COPD SMR **Age Groups:** 65+; 65-74, 75-84, 85+	**Pollutant:** $PM_{2.5}$ **Averaging Time:** 3 year avg	**Increment:** 10 µg/m^3 65+: 1.076 (1.044, 1.108) Eastern US: 1.125 (1.091, 1.159) Central US: 1.196 (1.115, 1.277) Western US: 1.029 (0.994, 1.064) 65-74: 1.156 (1.117, 1.196) 75-84: 1.081 (1.042, 1.121) 85+: 0.995 (0.956, 1.035)

References

Abbey DE; Nishino N; McDonnell WF; Burchette RJ; Knutsen SF; Lawrence Beeson W; Yang JX. (1999). Long-term inhalable particles and other air pollutants related to mortality in nonsmokers. American journal of respiratory and critical care medicine 159: 373-382.

Abrahamowicz M; Schopflocher T; Leffondre K; du Berger R; Krewski D. (2003). Flexible modeling of exposure-response relationship between long-term average levels of particulate air pollution and mortality in the American Cancer Society study. J Toxicol Environ Health A 66: 1625-1654.

Ackermann-Liebrich U; Leuenberger P; Schwartz J; Schindler C; Monn C; Bolognini G; Bongard JP; Brandli O; Domenighetti G; Elsasser S; Grize L; Karrer W; Keller R; Keller-Wossidlo H; Kunzli N; Martin BW; Medici TC; Perruchoud AP; Schoni MH; Tschopp JM; Villiger B; Wuthrich B; Zellweger JP; Zemp E. (1997). Lung function and long term exposure to air pollutants in Switzerland. Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team. American journal of respiratory and critical care medicine 155: 122-129.

Adamkiewicz G; Ebelt S; Syring M; Slater J; Speizer FE; Schwartz J; Suh H; Gold DR. (2004). Association between air pollution exposure and exhaled nitric oxide in an elderly population. Thorax 59: 204-209.

Adar SD; Adamkiewicz G; Gold DR; Schwartz J; Coull BA; Suh H. (2007). Ambient and microenvironmental particles and exhaled nitric oxide before and after a group bus trip. Environ Health Perspect 115: 507-512.

Aekplakorn W; Loomis D; Vichit-Vadakan N; Shy C; Plungchuchon S. (2003). Acute effects of SO2 and particles from a power plant on respiratory symptoms of children, Thailand. Southeast Asian J Trop Med Public Health 34: 906-914.

Aga E; Samoli E; Touloumi G; Anderson HR; Cadum E; Forsberg B; Goodman P; Goren A; Kotesovec F; Kriz B; Macarol-Hiti M; Medina S; Paldy A; Schindler C; Sunyer J; Tittanen P; Wojtyniak B; Zmirou D; Schwartz J; Katsouyanni K. (2003). Short-term effects of ambient particles on mortality in the elderly: results from 28 cities in the APHEA2 project. Eur Respir J Suppl 40: 28s-33s.

Allen RW; Mar T; Koenig J; Liu LJ; Gould T; Simpson C; Larson T. (2008). Changes in lung function and airway inflammation among asthmatic children residing in a woodsmoke-impacted urban area. Inhal Toxicol 20: 423-433.

Allen RW; Criqui MH; Diez Roux AV; Allison M; Shea S; Detrano R; Sheppard L; Wong N; Hinckley Stukovsky K; Kaufman JD. (In Press). Fine particulate air pollution, proximity to traffic, and aortic atherosclerosis: The Multi-Ethnic Study of Atherosclerosis. Epidemiology.

Analitis A; Katsouyanni K; Dimakopoulou K; Samoli E; Nikoloulopoulos AK; Petasakis Y; Touloumi G; Schwartz J; Anderson HR; Cambra K; Forastiere F; Zmirou D; Vonk JM; Clancy L; Kriz B; Bobvos J; Pekkanen J. (2006). Short-term effects of ambient particles on cardiovascular and respiratory mortality. Epidemiology 17: 230-233.

Andersen ZJ; Wahlin P; Raaschou-Nielsen O; Scheike T; Loft SCEH. (2007). Ambient particle source apportionment and daily hospital admissions among children and elderly in Copenhagen. J Expo Sci Environ Epidemiol 17: 625-636.

Andersen ZJ; Loft S; Ketzel M; Stage M; Scheike T; Hermansen MN; Bisgaard H. (2008a). Ambient air pollution triggers wheezing symptoms in infants. Thorax 63: 710-716.

Andersen ZJ; Wahlin P; Raaschou-Nielsen O; Ketzel M; Scheike T; Loft S. (2008b). Size distribution and total number concentration of ultrafine and accumulation mode particles and hospital admissions in children and the elderly in Copenhagen, Denmark. Occupational and environmental medicine 65: 458-466.

Anderson HR; Atkinson RW; Bremner SA; Marston L. (2003). Particulate air pollution and hospital admissions for cardiorespiratory diseases: are the elderly at greater risk? Eur Respir J Suppl 40: 39s-46s.

Annesi-Maesano I; Moreau D; Caillaud D; Lavaud F; Le Moullec Y; Taytard A; Pauli G; Charpin D. (2007). Residential proximity fine particles related to allergic sensitisation and asthma in primary school children. Respir Med 101: 1721-1729.

Arena VC; Mazumdar S; Zborowski JV; Talbott EO; He S; Chuang YH; Schwerha JJ. (2006). A retrospective investigation of PM10 in ambient air and cardiopulmonary hospital admissions in Allegheny County, Pennsylvania: 1995-2000. J Occup Environ Med 48: 38-47.

Atkinson RW. (2004). Acute effects of air pollution on admissions: reanalysis of APHEA 2. American journal of respiratory and critical care medicine 169: 1257-1258.

Auchincloss AH; Roux AV; Dvonch JT; Brown PL; Barr RG; Daviglus ML; Goff DC; Kaufman JD; O'Neill MS. (2008). Associations between Recent Exposure to Ambient Fine Particulate Matter and Blood Pressure in the Multi-Ethnic Study of Atherosclerosis (MESA). Environ Health Perspect 116: 486-491.

Avol EL; Gauderman WJ; Tan SM; London SJ; Peters JM. (2001). Respiratory effects of relocating to areas of differing air pollution levels. Am J Respir Crit Care Med 164: 2067-2072.

Babin SM; Burkom HS; Holtry RS; Tabernero NR; Stokes LD; Davies-Cole JO; DeHaan K; Lee DH. (2007). Pediatric patient asthma-related emergency department visits and admissions in Washington, DC, from 2001-2004, and associations with air quality, socio-economic status and age group. Environ Health 6: 9.

Baccarelli A; Zanobetti A; Martinelli I; Grillo P; Hou L; Lanzini G; Mannucci PM; Bartazzi PA; Schwartz J. (2007a). Air pollution, smoking, and plasma homocysteine. Environ Health Perspect 115: 176-181.

Baccarelli A; Zanobetti A; Martinelli I; Grillo P; Hou L; Giacomini S; Bonzini M; Lanzani G; Mannucci PM; Bertazzi PA; Schwartz J. (2007b). Effects of exposure to air pollution on blood coagulation. J Thromb Haemost 5: 252-260.
Baccarelli A; Martinelli I; Zanobetti A; Grillo P; Hou LF; Bertazzi PA; Mannucci PM; Schwartz J. (2008). Exposure to particulate air pollution and risk of deep vein thrombosis. Arch Intern Med 168: 920-927.
Ballester F; Saez M; Perez-Hoyos S; Iniguez C; Gandarillas A; Tobias A; Bellido J; Taracido M; Arribas F; Daponte A; Alonso E; Canada A; Guillen-Grima F; Cirera L; Perez-Boillos MJ; Saurina C; Gomez F; Tenias JM. (2002). The EMECAM project: a multicentre study on air pollution and mortality in Spain: combined results for particulates and for sulfur dioxide. Occupational and environmental medicine 59: 300-308.
Ballester F; Iniguez C; Saez M; Perez-Hoyos S; Daponte A; Ordonez JM; Barcelo MA; Taracido M; Arribas F; Bellido J; Cambra K; Canada A; Guillen JJ. (2003). Short-term relationship between air pollution and mortality in 13 Spanish cities. Med Clin (Barc) 121: 684-689.
Ballester F; Rodriguez P; Iniguez C; Saez M; Daponte A; Galan I; Taracido M; Arribas F; Bellido J; Cirarda FB; Canada A; Guillen JJ; Guillen-Grima F; Lopez E; Perez-Hoyos S; Lertxundi A; Toro S. (2006). Air pollution and cardiovascular admissions association in Spain: results within the EMECAS project. Journal of epidemiology and community health 60: 328-336.
Barnett AG; Williams GM; Schwartz J; Neller AH; Best TL; Petroeschevsky AL; Simpson RW. (2005). Air pollution and child respiratory health: a case-crossover study in Australia and New Zealand. American journal of respiratory and critical care medicine 171: 1272-1278.
Barraza-Villarreal A; Sunyer J; Hernandez-Cadena L; Escamilla-Nunez MC; Sienra-Monge JJ; Ramirez-Aguilar M; Cortez-Lugo M; Holguin F; Diaz-Sanchez D; Olin AC; Romieu I. (2008). Air pollution, airway inflammation, and lung function in a cohort study of Mexico City schoolchildren. Environ Health Perspect 116: 832-838.
Bartzokas A; Kassomenos P; Petrakis M; Celessides C. (2004). The effect of meteorological and pollution parameters on the frequency of hospital admissions for cardiovascular and respiratory problems in Athens. Indoor and Built Environment 13: 271-275.
Basu R; Woodruff TJ; Parker JD; Saulnier L; Schoendorf KC. (2004). Comparing exposure metrics in the relationship between PM2.5 and birth weight in California. Journal of exposure analysis and environmental epidemiology 14: 391-396.
Basu R; Feng WY; Ostro BD. (2008). Characterizing temperature and mortality in nine California counties. Epidemiology 19: 138-145.
Bateson TF; Schwartz J. (2004). Who is sensitive to the effects of particulate air pollution on mortality? A case-crossover analysis of effect modifiers. Epidemiology 15: 143-149.
Bayer-Oglesby L; Grize L; Gassner M; Takken-Sahli K; Sennhauser FH; Neu U; Schindler C; Braun-Fahrlander C. (2005). Decline of ambient air pollution levels and improved respiratory health in Swiss children. Environ Health Perspect 113: 1632-1637.
Bedeschi E; Campari C; Candela S; Collini G; Caranci N; Frasca G; Galassi C; Francesca G; Vigotti MA. (2007). Urban air pollution and respiratory emergency visits at pediatric unit, Reggio Emilia, Italy. J Toxicol Environ Health A 70: 261-265.
Beelen R; Hoek G; van den Brandt PA; Goldbohm RA; Fischer P; Schouten LJ; Jerrett M; Hughes E; Armstrong B; Brunekreef B. (2008). Long-term effects of traffic-related air pollution on mortality in a Dutch cohort (NLCS-AIR study). Environ Health Perspect 116: 196-202.
Beeson WL; Abbey DE; Knutsen SF. (1998). Long-term concentrations of ambient air pollutants and incident lung cancer in California adults: results from the AHSMOG study.Adventist Health Study on Smog. Environ Health Perspect 106: 813-823.
Bell ML; Ebisu K; Belanger K. (2007). Ambient air pollution and low birth weight in Connecticut and Massachusetts. Environ Health Perspect 115: 1118-1124.
Bell ML; Ebisu K; Peng RD; Walker J; Samet JM; Zeger SL; Dominic F. (2008a). Seasonal and regional short-term effects of fine particles on hospital admissions in 202 U.S. counties, 1999-2005. Am J Epidemiol In Press.
Bell ML; Levy JK; Lin Z. (2008b). The effect of sandstorms and air pollution on cause-specific hospital admissions in Taipei, Taiwan. Occupational and environmental medicine 65: 104-111.
Bennett CM; McKendry IG; Kelly S; Denike K; Koch T. (2006). Impact of the 1998 Gobi dust event on hospital admissions in the Lower Fraser Valley, British Columbia. The Science of the total environment 366: 918-925.
Bennett CM; Simpson P; Raven J; Skoric B; Powell J; Wolfe R; Walters EH; Abramson MJ. (2007). Associations between ambient PM2.5 concentrations and respiratory symptoms in Melbourne, 1998-2005. J Toxicol Environ Health A 70: 1613-1618.
Binkova B; Chvatalova I; Lnenickova Z; Milcova A; Tulupova E; Farmer PB; Sram RJ. (2007). PAH-DNA adducts in environmentally exposed population in relation to metabolic and DNA repair gene polymorphisms. Mutation Research-Fundamental and Molecular Mechanisms of Mutagenesis: Fundamental.
Boezen HM; van der Zee SC; Postma DS; Vonk JM; Gerritsen J; Hoek G; Brunekreef B; Rijcken B; Schouten JP. (1999). Effects of ambient air pollution on upper and lower respiratory symptoms and peak expiratory flow in children. Lancet 353: 874-878.
Boezen HM; Vonk JM; van der Zee SC; Gerritsen J; Hoek G; Brunekreef B; Schouten JP; Postma DS. (2005). Susceptibility to air pollution in elderly males and females. Eur Respir J 25: 1018-1024.

Bourotte C; Curl-Amarante AP; Forti MC; Pereira LAA; Braga AL; Lotufo PA. (2007). Association between ionic composition of fine and coarse aerosol soluble fraction and peak expiratory flow of asthmatic patients in Sao Paulo city (Brazil). Atmospheric Environment 41: 2036-2048.
Brauer M; Hoek G; Van Vliet P; Meliefste K; Fischer PH; Wijga A; Koopman LP; Neijens HJ; Gerritsen J; Kerkhof M. (2002). Air pollution from traffic and the develo ment of respiratory infections and asthmatic an allergic symptoms in children. American journal of respiratory and critical care medicine 166: 1092.
Brauer M; Gehring U; Brunekreef B; de Jongste J; Gerritsen J; Rovers M; Wichmann HE; Wijga A; Heinrich J. (2006). Traffic-related air pollution and otitis media. EnvironHealth Perspect 114: 1414-1418.
Brauer M; Hoek G; Smit HA; de Jongste JC; Gerritsen J; Postma DS; Kerkhof M; Brunekreef B. (2007). Air pollution and development of asthma, allergy and infections in a birth cohort. Eur Respir J 29: 879-888.
Brauer M; Lencar C; Tamburic L; Koehoorn M; Demers P; Karr C. (2008). A cohort study of traffic-related air pollution impacts on birth outcomes. Environ Health Perspect 116: 680-686.
Burnett RT; Stieb D; Brook JR; Cakmak S; Dales R; Raizenne M; Vincent R; Dann T. (2004). Associations between short-term changes in nitrogen dioxide and mortality in Canadian cities. Archives of environmental health 59: 228-236.
Burr ML; Karani G; Davies B; Holmes BA; Williams KL. (2004a). Effects on respiratory health of a reduction in air pollution from vehicle exhaust emissions. Occupational & Environmental Medicine 61: 212.
Burr ML; Karani G; Davies B; Holmes BA; Williams KL. (2004b). Effects on respiratory health of a reduction in air pollution from vehicle exhaust emissions. Occupational and environmental medicine 61: 212-218.
Cakmak S; Dales RE; Vidal CB. (2007). Air pollution and mortality in Chile: susceptibility among the elderly. Environ Health Perspect 115: 524-527.
Calderon-Garciduenas L; Maronpot RR; Torres-Jardon R; Henriquez-Roldan C; Schoonhoven R; Acuna-Ayala H; Villarreal-Calderon A; Nakamura J; Fernando R; Reed W; Azzarelli B; Swenberg JA. (2003). DNA damage in nasal and brain tissues of canines exposed to air pollutants is associated with evidence of chronic brain inflammation and neurodegeneration. Toxicologic pathology 31: 524-538.
Calderon-Garciduenas L; Vincent R; Mora-Tiscareno A; Franco-Lira M; Henriquez-Roldan C; Barragan-Mejia G; Garrido-Garcia L; Camacho-Reyes L; Valencia-Salazar G; Paredes R; Romero L; Osnaya H; Villarreal-Calderon R; Torres-Jardon R; Hazucha MJ; Reed W. (2007). Elevated plasma endothelin-1 and pulmonary arterial pressure in children exposed to air pollution. Environ Health Perspect 115: 1248-1253.
Calderón-Garcidueñas L; Mora-Tiscareño A; Fordham LA; Chung CJ; Valencia-Salazar G; Flores-Gómez S; Solt AC; Gomez-del Campo A; Jardón-Torres R; Henríquez-Roldán C. (2006). Lung Radiology and Pulmonary Function of Children Chronically Exposed to Air Pollution. Environ Health Perspect 114: 1432.
Case MW; Williams R; Yeatts K; Chen F-L; Scott J; Svendsen E; Devlin RB. (2008). Evaluation of a direct personal coarse particulate matter monitor. Atmos Environ 42: 4446-4452.
Cavanagh JA; Brown L; Trought K; Kingham S; Epton MJ. (2007). Elevated concentrations of 1-hydroxypyrene in schoolchildren during winter in Christchurch, New Zealand. Sci Total Environ 374: 51-59.
Cesaroni G; Badaloni C; Porta D; Forastiere F; Perucci CA. (2008). Comparison between several indices of exposure to traffic-related air pollution and their respiratory health impact in adults. Occupational and environmental medicine.
Chan CC; Chuang KJ; Chen WJ; Chang WT; Lee CT; Peng CM. (2008). Increasing cardiopulmonary emergency visits by long-range transported Asian dust storms in Taiwan. Environ Res 106: 393-400.
Chardon B; Lefranc A; Granados D; Gremy I. (2007). Air pollution and doctors' house calls for respiratory diseases in the Greater Paris area (2000-3). Occupational and environmental medicine 64: 320-324.
Chattopadhyay BP; Mukherjee A; Mukherjee K; Roychowdhury A. (2007). Exposure to vehicular pollution and assessment of respiratory function in urban inhabitants. Lung 185: 263-270.
Chen CH; Xirasagar S; Lin HC. (2006a). Seasonality in adult asthma admissions, air pollutant levels, and climate: a population-based study. J Asthma 43: 287-292.
Chen L; Yang W; Jennison BL; Goodrich A; Omaye ST. (2002). Air pollution and birth weight in northern Nevada, 1991-1999. Inhal Toxicol 14: 141-157.
Chen L; Verrall K; Tong S. (2006b). Air particulate pollution due to bushfires and respiratory hospital admissions in Brisbane, Australia. Int J Environ Health Res 16: 181-191.
Chen SJ; Lin WY; Huang KL; Lin CC; Hsieh LT; Kao MJ. (2004). Characteristics of particles sampled in southern Taiwan during the Asian dust storm periods in 2000 and 2001. Atmos Environ 38: 5925-5934.
Chen Y; Yang Q; Krewski D; Burnett RT; Shi Y; McGrail KM. (2005). The effect of coarse ambient particulate matter on first, second, and overall hospital admissions for respiratory disease among the elderly. Inhal Toxicol 17: 649-655.
Cheng MF; Tsai SS; Wu TN; Chen PS; Yang CY. (2007). Air pollution and hospital admissions for pneumonia in a tropical city: Kaohsiung, Taiwan. J Toxicol Environ Health A 70: 2021-2026.
Chimonas MA; Gessner BD. (2007). Airborne particulate matter from primarily geologic, non-industrial sources at levels below National Ambient Air Quality Standards is associated with outpatient visits for asthma and quick-relief medication prescriptions among children less than 20 years old enrolled in Medicaid in Anchorage, Alaska. Environ Res 103: 397-404.
Chiu HF; Cheng MH; Tsai SS; Wu TN; Kuo HW; Yang CY. (2006). Outdoor air pollution and female lung cancer in Taiwan. Inhal Toxicol 18: 1025-1031.

Choi JH; Xu QS; Park SY; Kim JH; Hwang SS; Lee KH; Lee HJ; Hong YC. (2007). Seasonal variation of effect of air pollution on blood pressure. Journal of epidemiology and community health 61: 314-318.
Chow JC. (2007). The application of thermal methods for determining chemical composition of carbonaceous aerosols: A review. Journal of Environmental Science and Health 42: 1521-1541.
Chuang KJ; Chan CC; Su TC; Lee CT; Tang CS. (2007a). The effect of urban air pollution on inflammation, oxidative stress, coagulation, and autonomic dysfunction in young adults. American journal of respiratory and critical care medicine 176: 370-376.
Chuang KJ; Chan CC; Su TC; Lin LY; Lee CT. (2007b). Associations between particulate sulfate and organic carbon exposures and heart rate variability in patients with or at risk for cardiovascular diseases. J Occup Environ Med 49: 610-617.
Crimmins BS; Baker JE. (2006). Improved GC/MS methods for measuring hourly PAH and nitro-PAH concentrations in urban particulate matter. Atmos Environ 40: 6764-6779.
Cui Y; Zhang ZF; Froines J; Zhao J; Wang H; Yu SZ; Detels R. (2003). Air pollution and case fatality of SARS in the People's Republic of China: an ecologic study. Environ Health 2: 15.
Dales R; Burnett RT; Smith-Doiron M; Adm D; Stieb DM; Brook JR. (2004). Air pollution and sudden infant death syndrome. Pediatrics 113: 628-631.
Dales R; Wheeler A; Mahmud M; Frescura AM; Smith-Doiron M; Nethery E; Liu L. (2008). The Influence of Living Near Roadways on Spirometry and Exhaled Nitric Oxide in Elementary Schoolchildren. Environmental health perspectives 116: 1423.
Dales RE; Cakmak S; Doiron MS. (2006). Gaseous air pollutants and hospitalization for respiratory disease in the neonatal period. Environ Health Perspect 114: 1751-1754.
Daniels MJ; Dominici F; Zeger SL; Samet JM. (2004). The National Morbidity, Mortality, and Air Pollution Study. Part III: PM10 concentration-response curves and thresholds for the 20 largest US cities. 1041-5505 (Print).
de Hartog JJ; Hoek G; Peters A; Timonen KL; Ibald-Mulli A; Brunekreef B; Heinrich J; Tiittanen P; van Wijnen JH; Kreyling W; Kulmala M; Pekkanen J. (2003). Effects of fine and ultrafine particles on cardiorespiratory symptoms in elderly subjects with coronary heart disease: the ULTRA study. Am J Epidemiol 157: 613-623.
De Leon SF; Thurston GD; Ito K. (2003). Contribution of respiratory disease to nonrespiratory mortality associations with air pollution. Am J Respir Crit Care Med 167: 1117-1123.
Delfino RJ; Zeiger RS; Seltzer JM; Street DH. (1998). Symptoms in pediatric asthmatics and air pollution: differences in effects by symptom severity, anti-inflammatory medication use and particulate averaging time. Environ Health Perspect 106: 751-761.
Delfino RJ; Zeiger RS; Seltzer JM; Street DH; McLaren CE. (2002). Association of asthma symptoms with peak particulate air pollution and effect modification by anti-inflammatory medication use. Environ Health Perspect 110: A607-617.
Delfino RJ; Gong H, Jr.; Linn WS; Pellizzari ED; Hu Y. (2003). Asthma symptoms in Hispanic children and daily ambient exposures to toxic and criteria air pollutants. Environ Health Perspect 111: 647-656.
Delfino RJ; Quintana PJ; Floro J; Gastanaga VM; Samimi BS; Kleinman MT; Liu LJ; Bufalino C; Wu CF; McLaren CE. (2004). Association of FEV1 in asthmatic children with personal and microenvironmental exposure to airborne particulate matter. Environ Health Perspect 112: 932-941.
Delfino RJ; Staimer N; Gillen D; Tjoa T; Sioutas C; Fung K; George SC; Kleinman MT. (2006). Personal and ambient air pollution is associated with increased exhaled nitric oxide in children with asthma. Environ Health Perspect 114: 1736-1743.
Delfino RJ; Staimer N; Tjoa T; Polidori A; Arhami M; Gillen DL; Kleinman MT; Vaziri ND; Longhurst J; Zaldivar F; Sioutas C. (2008). Circulating biomarkers of inflammation, antioxidant activity, and platelet activation are associated with primary combustion aerosols in subjects with coronary artery disease. Environ Health Perspect 116: 898-906.
DeMeo DL; Zanobetti A; Litonjua AA; Coull BA; Schwartz J; Gold DR. (2004). Ambient air pollution and oxygen saturation. American journal of respiratory and critical care medicine 170: 383-387.
Desqueyroux H; Pujet JC; Prosper M; Squinazi F; Momas I. (2002). Short-term effects of low-level air pollution on respiratory health of adults suffering from moderate to severe asthma. Environ Res 89: 29-37.
Diette GB; Hansel NN; Buckley TJ; Curtin-Brosnan J; Eggleston PA; Matsui EC; McCormack MC; Williams DL; Breysse PN. (2007). Home indoor pollutant exposures among inner-city children with and without asthma. Environ Health Perspect 115: 1665-1669.
Diez Roux AV; Auchincloss AH; Astor B; Barr RG; Cushman M; Dvonch T; Jacobs DR, Jr.; Kaufman J; Lin X; Samson P. (2006). Recent exposure to particulate matter and C-reactive protein concentration in the multi-ethnic study of atherosclerosis. Am J Epidemiol 164: 437-448.
Diez Roux AV; Auchincloss AH; Franklin TG; Raghunathan T; Barr RG; Kaufman J; Astor B; Keeler J. (2008). Long-term exposure to ambient particulate matter and prevalence of subclinical atherosclerosis in the Multi-Ethnic Study of Atherosclerosis. Am J Epidemiol 167: 667-675.
Dominici F; McDermott A; Zeger SL; Samet JM. (2003). National maps of the effects of particulate matter on mortality: exploring geographical variation. Environ Health Perspect 111: 39-44.
Dominici F; Dermott AMC; T.J. H. (2004a). Improved Semiparametric Time Series Models of Air Pollution and Mortality. Journal of the American Statistical Association 99: 938-948.
Dominici F; Zanobetti A; Zeger SL; Schwartz J; Samet JM. (2004b). Hierarchical bivariate time series models: a combined analysis of the effects of particulate matter on morbidity and mortality. Biostatistics 5: 341-360.

Dominici F; Peng RD; Bell ML; Pham L; McDermott A; Zeger SL; Samet JMCEH. (2006). Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA 295: 1127-1134.
Dominici F; Peng RD; Ebisu K; Zeger SL; Samet JM; Bell ML. (2007a). Does the effect of PM10 on mortality depend on PM nickel and vanadium content? A reanalysis of the NMMAPS data. Environ Health Perspect 115: 1701-1703.
Dominici F; Peng RD; Zeger SL; White RH; Samet JM. (2007b). Particulate air pollution and mortality in the United States: did the risks change from 1987 to 2000? Am J Epidemiol 166: 880-888.
Dong Y; Hays MD; Dean Smith N; Kinsey JS. (2004). Inverting cascade impactor data for size-resolved characterization of fine particulate source emissions. J Aerosol Sci 35: 1497-1512.
Downs SH; Schindler C; Liu LJ; Keidel D; Bayer-Oglesby L; Brutsche MH; Gerbase MW; Keller R; Kunzli N; Leuenberger P; Probst-Hensch NM; Tschopp JM; Zellweger JP; Rochat T; Schwartz J; Ackermann-Liebrich U. (2007). Reduced exposure to PM_{10} and attenuated age-related decline in lung function. N Engl J Med 357: 2338-2347.
Dubowsky SD; Suh H; Schwartz J; Coull BA; Gold DR. (2006). Diabetes, obesity, and hypertension may enhance associations between air pollution and markers of systemic inflammation. Environ Health Perspect 114: 992-998.
Dugandzic R; Dodds L; Stieb D; Smith-Doiron M. (2006). The association between low level exposures to ambient air pollution and term low birth weight: a retrospective cohort study. Environ Health 5: 3.
Ebelt ST; Wilson WE; Brauer M. (2005). Exposure to ambient and nonambient components of particulate matter: a comparison of health effects. Epidemiology 16: 396-405.
Eeva T; Lehikoinen E. (2004). Rich calcium availability diminishes heavy metal toxicity in Pied Flycatcher. Functional Ecology 18: 548-553.
Eiguren-Fernandez A; Miguel AH; Jaques PA; Sioutas C. (2003). Evaluation of a Denuder-MOUDI-PUF Sampling System to Measure the Size Distribution of Semi-Volatile Polycyclic Aromatic Hydrocarbons in the Atmosphere. Aerosol Sci Technol 37: 201-209.
El-Zein A; Nuwayhid I; El-Fadel M; Mroueh S. (2007). Did a ban on diesel-fuel reduce emergency respiratory admissions for children? The Science of the total environment 384: 134-140.
Enstrom JE. (2005). Fine particulate air pollution and total mortality among elderly Californians, 1973-2002. Inhal Toxicol 17: 803-816.
Erbas B; Kelly AM; Physick B; Code C; Edwards M. (2005). Air pollution and childhood asthma emergency hospital admissions: estimating intra-city regional variations. Int J Environ Health Res 15: 11-20.
Farhat SC; Paulo RL; Shimoda TM; Conceicao GM; Lin CA; Braga AL; Warth MP; Saldiva PH. (2005). Effect of air pollution on pediatric respiratory emergency room visits and hospital admissions. Braz J Med Biol Res 38: 227-235.
Ferdinands JM; Crawford CA; Greenwald R; Van Sickle D; Hunter E; Teague WG. (2008). Breath acidification in adolescent runners exposed to atmospheric pollution: a prospective, repeated measures observational study. Environ Health 7: 10.
Filleul L; Rondeau V; Vandentorren S; Le Moual N; Cantagrel A; Annesi-Maesano I; Charpin D; Declercq C; Neukirch F; Paris C; Vervloet D; Brochard P; Tessier JF; Kauffmann F; Baldi I. (2005). Twenty five year mortality and air pollution: results from the French PAARC survey. Occupational and environmental medicine 62: 453-460.
Fischer P; Hoek G; Brunekreef B; Verhoeff A; van Wijnen J. (2003). Air pollution and mortality in The Netherlands: are the elderly more at risk? Eur Respir J Suppl 40: 34s-38s.
Fischer PH; Brunekreef B; Lebret E. (2004). Air pollution related deaths during the 2003 heat wave in the Netherlands. Atmos Environ 38: 1083-1085.
Fischer SL; Koshland CP. (2007). Daily and peak 1 h indoor air pollution and driving factors in a rural Chinese village. EnvironSci Technol 41: 3121-3126.
Forastiere F; Stafoggia M; Picciotto S; Bellander T; D'Ippoliti D; Lanki T; von Klot S; Nyberg F; Paatero P; Peters A; Pekkanen J; Sunyer J; Perucci CA. (2005). A case-crossover analysis of out-of-hospital coronary deaths and air pollution in Rome, Italy. Am J Respir Crit Care Med 172: 1549-1555.
Forastiere F; Stafoggia M; Tasco C; Picciotto S; Agabiti N; Cesaroni G; Perucci CA. (2007). Socioeconomic status, particulate air pollution, and daily mortality: differential exposure or differential susceptibility. Am J Ind Med 50: 208-216.
Forastiere F; Faustini A. (2008). Are we understanding the respiratory effects of traffic related airborne particles? Thorax 63: 574-576.
Forsberg B; Stjernberg N; Linne R; Segerstedt B; Wall S. (1998). Daily air pollution levels and acute asthma in southern Sweden. Eur Respir J 12: 900-905.
Franklin M; Zeka A; Schwartz J. (2007). Association between PM2.5 and all-cause and specific-cause mortality in 27 US communities. Journal of Exposure Science and Environmental Epidemiology 17: 279-287.
Franklin M; Koutrakis P; Schwartz P. (2008). The role of particle composition on the association between PM2.5 and mortality. Epidemiology 19: 680-689.
Fraser MP; Yue ZW; Buzcu B. (2003). Source apportionment of fine particulate matter in Houston, TX, using organic molecular markers. Atmos Environ 37: 2117-2123.
Fuentes M; Song HR; Ghosh SK; Holland DM; Davis JM. (2006). Spatial association between speciated fine particles and mortality. Biometrics 62: 855-863.
Fung KY; Luginaah I; K.M. G; Webster G. (2005). Air Pollution and Daily Hospitalization Rates for Cardiovascular and Respiratory Diseases in London, Ontario. International Journal of Environmental Studies 62: 677-685.
Fung KY; Khan S; Krewski D; Chen Y. (2006). Association between air pollution and multiple respiratory hospitalizations among the elderly in Vancouver, Canada. Inhal Toxicol 18: 1005-1011.

Galán I; Tobias A; Banegas JR; Aranguez E. (2003). Short-term effects of air pollution on daily asthma emergency room admissions. Eur Respir J 22: 802-808.
Gauderman WJ; McConnell R; Gilliland F; London S; Thomas D; Avol E; Vora H; Berhane K; Rappaport EB; Lurmann F; Margolis HG; Peters J. (2000a). Association between air pollution and lung function growth in southern California children. Am J Respir Crit Care Med 162: 1383-1390.
Gauderman WJ; McConnell R; Gilliland F; London S; Thomas D; Avol E; Vora H; Berhane K; Rappaport EB; Lurmann F; Margolis HG; Peters J. (2000b). Association between air pollution and lung function growth in southern California children. American journal of respiratory and critical care medicine 162: 1383-1390.
Gauderman WJ; Gilliland GF; Vora H; Avol E; Stram D; McConnell R; Thomas D; Lurmann F; Margolis HG; Rappaport EB; Berhane K; Peters JM. (2002a). Association between air pollution and lung function growth in southern California children: results from a second cohort. Am J Respir Crit Care Med 166: 76-84.
Gauderman WJ; Gilliland GF; Vora H; Avol E; Stram D; McConnell R; Thomas D; Lurmann F; Margolis HG; Rappaport EB; Berhane K; Peters JM. (2002b). Association between air pollution and lung function growth in southern California children: results from a second cohort. American journal of respiratory and critical care medicine 166: 76-84.
Gauderman WJ; Avol E; Gilliland F; Vora H; Thomas D; Berhane K; McConnell R; Kuenzli N; Lurmann F; Rappaport E; Margolis H; Bates D; Peters J. (2004). The effect of air pollution on lung development from 10 to 18 years of age. The New England journal of medicine 351: 1057-1067.
Gauderman WJ; Vora H; McConnell R; Berhane K; Gilliland F; Thomas D; Lurmann F; Avol E; Kunzli N; Jerrett M; Peters J. (2007). Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. Lancet 369: 571-577.
Gehring U; Cyrys J; Sedlmeir G; Brunekreef B; Bellander T; Fischer P; Bauer CP; Reinhardt D; Wichmann HE; Heinrich J. (2002). Traffic-related air pollution and respiratory health during the first 2 yrs of life. Eur Respir J 19: 690-698.
Gent JF; Triche EW; Holford TR; Belanger K; Bracken MB; Beckett WS; Leaderer BP. (2003). Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. JAMA 290: 1859-1867.
Giglio L; van der Werf GR; Randerson JT; Collatz GJ; Kasibhatla P. (2006). Global estimation of burned area using MODIS active fire observations. Atmos Chem Phys 6: 957–974.
Gilboa SM; Mendola P; Olsham AF; Langlois PH; Savitz DA; Loomis D; Herring AH; Fixler DE. (2005). Relation between ambient air quality and selected birth defects, seven county study, Texas, 1997-2000. Am J Epidemiol 162: 238-252.
Girardot SP; Ryan PB; Smith SM; Davis WT; Hamilton CB; Obenour RA; Renfro JR; Tromatore KA; Reed GD. (2006). Ozone and PM2.5 exposure and acute pulmonary health effects: a study of hikers in the Great Smoky Mountains National Park. Environ Health Perspect 114: 1044-1052.
Goldberg MS; Burnett RT; Valois MF; Flegel K; Bailar JC, 3rd; Brook J; Vincent R; Radon K. (2003). Associations between ambient air pollution and daily mortality among persons with congestive heart failure. Environ Res 91: 8-20.
Goncalves FL; Carvalho LM; Conde FC; Latorre MR; Saldiva PH; Braga AL. (2005). The effects of air pollution and meteorological parameters on respiratory morbidity during the summer in Sao Paulo City. Environ Int 31: 343-349.
Gong H; Linn WS; Clark KW; Anderson KR; Geller MD; Sioutas C. (2005). Respiratory responses to exposures with fine particulates and nitrogen dioxide in the elderly with and without COPD. Inhalation Toxicol 17: 123-132.
Gordian ME; Choudhury AH. (2003). PM10 and asthma medication in schoolchildren. Archives of environmental health 58: 42-47.
Goss CH; Newsom SA; Schildcrout JS; Sheppard L; Kaufman JD. (2004a). Effect of ambient air pollution on pulmonary exacerbations and lung function in cystic fibrosis. Am J Respir Crit Care Med 169: 816-821.
Goss CH; Newsom SA; Schildcrout JS; Sheppard L; Kaufman JD. (2004b). Effect of ambient air pollution on pulmonary exacerbations and lung function in cystic fibrosis. American journal of respiratory and critical care medicine 169: 816-821.
Gouveia N; Bremner SA; Novaes HM. (2004). Association between ambient air pollution and birth weight in Sao Paulo, Brazil. Journal of epidemiology and community health 58: 11-17.
Grell GA; Emeis S; Stockwell WR; Schoenemeyer T; Forkel R; Michalakes J; Knoche R; Seidl W. (2000). Application of a multiscale, coupled MM5/chemistry model to the complex terrain of the VOTALP valley campaign. Atmos Environ 34: 1435-1453.
Ha EH; Lee JT; Kim H; Hong YC; Lee BE; Park HS; Christiani DC. (2003). Infant susceptibility of mortality to air pollution in Seoul, South Korea. Pediatrics 111: 284-290.
Hajat S; Anderson HR; Atkinson RW; Haines A. (2002). Effects of air pollution on general practitioner consultations for upper respiratory diseases in London. Occupational and environmental medicine 59: 294-299.
Hanigan IC; Johnston FH; Morgan GG. (2008). Vegetation fire smoke, indigenous status and cardio respiratory hospital admissions in Darwin, Australia, 1996-2005: a time-series study. Environ Health 7: 42.
Hansen C; Neller A; Williams G; Simpson R. (2006). Maternal exposure to low levels of ambient air pollution and preterm birth in Brisbane, Australia. Bjog 113: 935-941.
Hansen C; Neller A; Williams G; Simpson R. (2007). Low levels of ambient air pollution during pregnancy and fetal growth among term neonates in Brisbane, Australia. Environ Res 103: 383-389.
Hapcioglu B; Issever H; Kocyigit E; Disci R; Vatansever S; Ozdilli K. (2006). The effect of air pollution and meteorological parameters on chronic obstructive pulmonary disease at an Istanbul Hospital. Indoor and Built Environment 15: 147-153.

Harre ES; Price PD; Ayrey RB; Toop LJ; Martin IR; Town GI. (1997). Respiratory effects of air pollution in chronic obstructive pulmonary disease: a three month prospective study. Thorax 52: 1040-1044.
Hastings DL; Jardine S. (2002). The relationship between air particulate levels and upper respiratory disease in soldiers deployed to Bosnia (1997-1998). Mil Med 167: 296-303.
Hays MD; Geron CD; Linna KJ; Smith ND; Schauer JJ. (2002). Speciation of gas-phase and fine particle emissions from burning of foliar fuels. Environ Sci Technol 36: 2281-2295.
HEI. (2000). Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Health Effects Institute,
Hermann M; Wehner B; Bischof O; Han HS; Krinke T; Liu W; Zerrath A; Wiedensohler A. (2007). Particle counting efficiencies of new TSI condensation particle counters. J Aerosol Sci 38: 674-682.
Hertz-Picciotto I; Herr CEW; Yap PS; Dostál M; Shumway RH; Ashwood P; Lipsett M; Joad JP; Pinkerton KE; Šrám RJ. (2005). Air Pollution and Lymphocyte Phenotype Proportions in Cord Blood. Environ Health Perspect 113: 1391.
Hertz-Picciotto I; Baker RJ; Yap PS; Dostal M; Joad JP; Lipsett M; Greenfield T; Herr CE; Benes I; Shumway RH; Pinkerton KE; Sram R. (2007). Early childhood lower respiratory illness and air pollution. Environ Health Perspect 115: 1510-1518.
Hiltermann TJ; Stolk J; van der Zee SC; Brunekreef B; de Bruijne CR; Fischer PH; Ameling CB; Sterk PJ; Hiemstra PS; van Bree L. (1998). Asthma severity and susceptibility to air pollution. Eur Respir J 11: 686-693.
Hinwood AL; De Klerk N; Rodriguez C; Jacoby P; Runnion T; Rye P; Landau L; Murray F; Feldwick M; Spickett JCEH. (2006). The relationship between changes in daily air pollution and hospitalizations in Perth, Australia 1992-1998: a case-crossover study. Int J Environ Health Res 16: 27-46.
Ho WC; Hartley WR; Myers L; Lin MH; Lin YS; Lien CH; Lin RS. (2007). Air pollution, weather, and associated risk factors related to asthma prevalence and attack rate. Environ Res 104: 402-409.
Hoffmann B; Moebus S; Stang A; Beck EM; Dragano N; Mohlenkamp S; Schmermund A; Memmesheimer M; Mann K; Erbel R; Jockel KH. (2006). Residence close to high traffic and prevalence of coronary heart disease. European heart journal 27: 2696-2702.
Hogervorst JG; de Kok TM; Briede JJ; Wesseling G; Kleinjans JC; van Schayck CP. (2006). Relationship between radical generation by urban ambient particulate matter and pulmonary function of school children. J Toxicol Environ Health A 69: 245-262.
Holloman CH; Bortnick SM; Morara M; Strauss WJ; Calder CA. (2004). A Bayesian hierarchical approach for relating PM(2.5) exposure to cardiovascular mortality in North Carolina. Environ Health Perspect 112: 1282-1288.
Hong CY; Chia SE; Widjaja D; Saw SM; Lee J; Munoz C; Koh D. (2004). Prevalence of Respiratory Symptoms in Children and Air Quality by Village in Rural Indonesia. J Occup Environ Med 46: 1174.
Hong YC; Hwang SS; Kim JH; Lee KH; Lee HJ; Yu SD; Kim DS. (2007). Metals in particulate pollutants affect peak expiratory flow of schoolchildren. Environ Health Perspect 115: 430-434.
Hopke PK; Ito K; Mar T; Christensen WF; Eatough DJ; Henry RC; Kim E; Laden F; Lall R; Larson TV. (2006). PM source apportionment and health effects: 1. Intercomparison of source apportionment results. Journal of Exposure Science and Environmental Epidemiology 16: 275-286.
Horak F, Jr.; Studnicka M; Gartner C; Spengler JD; Tauber E; Urbanek R; Veiter A; Frischer T. (2002). Particulate matter and lung function growth in children: a 3-yr follow-up study in Austrian schoolchildren. Eur Respir J 19: 838-845.
Host S; Larrieu S; Pascal L; Blanchard M; Declercq C; Fabre P; Jusot JF; Chardon B; Le Tertre A; Wagner V; Prouvost H; Lefranc A. (2007). Short-term Associations between Fine and Coarse Particles and Cardiorespiratory Hospitalizations in Six French Cities. Occupational and environmental medicine 65: 544-551.
Host S; Larrieu S; Pascal L; Blanchard M; Declercq C; Fabre P; Jusot JF; Chardon B; Le Tertre A; Wagner V. (2008). Short-term associations between fine and coarse particles and hospital admissions for cardiorespiratory diseases in six French cities. Br Med J 65: 544.
Huynh M; Woodruff TJ; Parker JD; Schoendorf KC. (2006). Relationships between air pollution and preterm birth in California. Paediatr Perinat Epidemiol 20: 454-461.
Hwang BF; Jaakkola JJ; Lee YL; Lin YC; Guo YL. (2006). Relation between air pollution and allergic rhinitis in Taiwanese schoolchildren. Respir Res 7: 23.
Hwang JS; Chan CC. (2002). Effects of air pollution on daily clinic visits for lower respiratory tract illness. Am J Epidemiol 155: 1-10.
Ingle ST; Pachpande BG; Wagh ND; Patel VS; Attarde SB. (2005). Exposure to vehicular pollution and respiratory impairment of traffic policemen in Jalgaon City, India. IndHealth 43: 656-662.
Islam T; Gauderman WJ; Berhane K; McConnell R; Avol E; Peters JM; Gilliland FD. (2007). Relationship between air pollution, lung function and asthma in adolescents. Thorax 62: 957-963.
Issever H; Disci R; Hapcioglu B; Vatansever S; Karan M, A. ; Akkaya V; Erk O. (2005). The effect of air pollution and meteorological parameters in Istanbul on hospital admissions for acute coronary syndrome. Indoor and Built Environment 14: 157-164.
Ito K; Christensen WF; Eatough DJ; Henry RC; Kim E; Laden F; Lall R; Larson TV; Neas L; Hopke PK; Thurston GD. (2006). PM source apportionment and health effects: 2. An investigation of intermethod variability in associations between source-apportioned fine particle mass and daily mortality in Washington, DC. Journal of Exposure Science and Environmental Epidemiology 16: 300-310.

Jaffe DH; Singer ME; Rimm AA. (2003). Air pollution and emergency department visits for asthma among Ohio Medicaid recipients, 1991-1996. Environ Res 91: 21-28.
Jalaludin B; Morgan G; Lincoln D; Sheppeard V; Simpson R; Corbett S. (2006). Associations between ambient air pollution and daily emergency department attendances for cardiovascular disease in the elderly (65+ years), Sydney, Australia. Journal of Exposure Science and Environmental Epidemiology 16: 225-237.
Jalaludin B; Mannes T; Morgan G; Lincoln D; Sheppeard V; Corbett S. (2007). Impact of ambient air pollution on gestational age is modified by season in Sydney, Australia. Environ Health 6: 16.
Jalaludin BB; O'Toole BI; Leeder SR. (2004). Acute effects of urban ambient air pollution on respiratory symptoms, asthma medication use, and doctor visits for asthma in a cohort of Australian children. Environ Res 95: 32-42.
Janes H; Dominici F; Zeger SL. (2007). Trends in air pollution and mortality: an approach to the assessment of unmeasured confounding. Epidemiology 18: 416-423.
Jansen KL; Larson TV; Koenig JQ; Mar TF; Fields C; Stewart J; Lippmann M. (2005). Associations between health effects and particulate matter and black carbon in subjects with respiratory disease. Environmental Health Perspectives 113: 1741-1746.
Janssen NA; Brunekreef B; van Vliet P; Aarts F; Meliefste K; Harssema H; Fischer P. (2003). The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyperresponsiveness, and respiratory symptoms in Dutch schoolchildren. Environ Health Perspect 111: 1512-1518.
Jedrychowski W; Masters E; Choi H; Sochacka E; Flak E; Mroz E; Pac A; Jacek R; Kaim I; Skolicki Z; Spengler JD; Perera F. (2007). Pre-pregnancy dietary vitamin A intake may alleviate the adverse birth outcomes associated with prenatal pollutant exposure: epidemiologic cohort study in Poland. International journal of occupational and environmental health 13: 175-180.
Jerrett M; Burnett RT; Willis A; Krewski D; Goldberg MS; DeLuca P; Finkelstein N. (2003). Spatial analysis of the air pollution-mortality relationship in the context of ecologic confounders. J Toxicol Environ Health A 66: 1735-1777.
Jerrett M; Burnett RT; Ma R; Pope CA, 3rd; Krewski D; Newbold KB; Thurston G; Shi Y; Finkelstein N; Calle EE; Thun MJ. (2005). Spatial analysis of air pollution and mortality in Los Angeles. Epidemiology 16: 727-736.
Johnston FH; Webby RJ; Pilotto LS; Bailie RS; Parry DL; Halpin SJ. (2006). Vegetation fires, particulate air pollution and asthma: a panel study in the Australian monsoon tropics. Int J Environ Health Res 16: 391-404.
Just J; Segala C; Sahraoui F; Priol G; Grimfeld A; Neukirch F. (2002). Short-term health effects of particulate and photochemical air pollution in asthmatic children. Eur Respir J 20: 899-906.
Kabir Z; Bennett K; Clancy L. (2007). Lung cancer and urban air-pollution in Dublin: a temporal association? Ir Med J 100: 367-369.
Kaiser R; Romieu I; Medina S; Schwartz J; Krzyzanowski M; Kunzli N. (2004). Air pollution attributable postneonatal infant mortality in U.S. metropolitan areas: a risk assessment study. Environ Health 3: 4.
Kan H; Chen B. (2003). Air pollution and daily mortality in Shanghai: a time-series study. Arch Environ Health 58: 360-367.
Kan H; London SJ; Chen G; Zhang Y; Song G; Zhao N; Jiang L; Chen B. (2007a). Differentiating the effects of fine and coarse particles on daily mortality in Shanghai, China. Environ Int 33: 376-384.
Kan H; Heiss G; Rose KM; Whitsel E; Lurmann F; London SJ. (2007b). Traffic exposure and lung function in adults: the Atherosclerosis Risk in Communities study. Thorax 62: 873-879.
Kan H; London SJ; Chen G; Zhang Y; Song G; Zhao N; Jiang L; Chen B. (2008). Season, sex, age, and education as modifiers of the effects of outdoor air pollution on daily mortality in Shanghai, China: The Public Health and Air Pollution in Asia (PAPA) Study. Environ Health Perspect 116: 1183-1188.
Kan HD; Chen BH; Fu CW; Yu SZ; Mu LN. (2005). Relationship between ambient air pollution and daily mortality of SARS in Beijing. Biomed Environ Sci 18: 1-4.
Karr C; Lumley T; Schreuder A; Davis R; Larson T; Ritz B; Kaufman J. (2007). Effects of subchronic and chronic exposure to ambient air pollutants on infant bronchiolitis. Am J Epidemiol 165: 553-560.
Kasamatsu J; Shima M; Yamazaki S; Tamura K; Sun G. (2006). Effects of winter air pollution on pulmonary function of school children in Shenyang, China. Int J Hyg Environ Health 209: 435-444.
Keatinge WR; Donaldson GC. (2006). Heat acclimatization and sunshine cause false indications of mortality due to ozone. Environ Res 100: 387-393.
Kettunen J; Lanki T; Tiittanen P; Aalto PP; Koskentalo T; Kulmala M; Salomaa V; Pekkanen J. (2007). Associations of fine and ultrafine particulate air pollution with stroke mortality in an area of low air pollution levels. Stroke; a journal of cerebral circulation 38: 918-922.
Kim E; Hopke PK; Pinto JP; Wilson WE. (2005a). Spatial variability of fine particle mass, components, and source contributions during the Regional Air Pollution Study in St. Louis. Environ Sci Technol 39: 4172-4179.
Kim JH; Lim DH; Kim JK; Jeong SJ; Son BK. (2005b). Effects of particulate matter (PM10) on the pulmonary function of middle-school children. J Korean Med Sci 20: 42-45.
Kim JJ; Smorodinsky S; Lipsett M; Singer BC; Hodgson AT; Ostro B. (2004). Traffic-related air pollution near busy roads: the East Bay Children's Respiratory Health Study. American journal of respiratory and critical care medicine 170: 520-526.
Kim OJ; Ha EH; Kim BM; Park HS; Jung WJ; Lee BE; Suh YJ; Kim YJ; Lee JT; Kim H; Hong YC. (2007a). PM_{10} and pregnancy outcomes: a hospital-based cohort study of pregnant women in Seoul. J Occup Environ Med 49: 1394-1402.
Kim SY; O'Neill MS; Lee JT; Cho Y; Kim J; Kim H. (2007b). Air pollution, socioeconomic position, and emergency hospital visits for asthma in Seoul, Korea. Int Arch Occup Environ Health 80: 701-710.

Klemm RJ; Lipfert FW; Wyzga RE; Gust C. (2004). Daily mortality and air pollution in Atlanta: two years of data from ARIES. Inhal Toxicol 16 Suppl 1: 131-141.
Ko FW; Tam W; Wong TW; Lai CK; Wong GW; Leung TF; Ng SS; Hui DS. (2007a). Effects of air pollution on asthma hospitalization rates in different age groups in Hong Kong. Clin Exp Allergy 37: 1312-1319.
Ko FW; Tam W; Wong TW; Chan DP; Tung AH; Lai CK; Hui DS. (2007b). Temporal relationship between air pollutants and hospital admissions for chronic obstructive pulmonary disease in Hong Kong. Thorax 62: 780-785.
Koenig JQ; Jansen K; Mar TF; Lumley T; Kaufman J; Trenga CA; Sullivan J; Liu LJ; Shapiro GG; Larson TV. (2003). Measurement of offline exhaled nitric oxide in a study of community exposure to air pollution. Environ Health Perspect 111: 1625-1629.
Kongtip P; Thongsuk W; Yoosook W; Chantanakul S. (2006). Health effects of metropolitan traffic-related air pollutants on street vendors. Atmospheric Environment 40: 7138-7145.
Kulkarni N; Pierse N; Rushton L; Grigg J. (2006). Carbon in airway macrophages and lung function in children. The New England journal of medicine 355: 21-30.
Kumar VS; Mani U; Prasad AK; Lal K; Gowri V; Gupta A. (2004). Effect of fly ash inhalation on biochemical and histomorphological changes in rat lungs. Indian journal of experimental biology 42: 964-968.
Kunzli N; Jerrett M; Mack WJ; Beckerman B; LaBree L; Gilliland F; Thomas D; Peters J; Hodis HN. (2005). Ambient air pollution and atherosclerosis in Los Angeles. Environ Health Perspect 113: 201-206.
Kuo HW; Lai JS; Lee MC; Tai RC; Lee MC. (2002). Respiratory effects of air pollutants among asthmatics in central Taiwan. Archives of environmental health 57: 194-200.
Laden F; Schwartz J; Speizer FE; Dockery DW. (2006). Reduction in fine particulate air pollution and mortality: Extended follow-up of the Harvard Six Cities study. American journal of respiratory and critical care medicine 173: 667-672.
Lagorio S; Forastiere F; Pistelli R; Iavarone I; Michelozzi P; Fano V; Marconi A; Ziemacki G; Ostro BD. (2006). Air pollution and lung function among susceptible adult subjects: a panel study. Environ Health 5: 11.
Langley-Turnbaugh SJ; Gordon NR; Lambert T. (2005). Airborne particulates and asthma: a Maine case study. Toxicol Ind Health 21: 75-92.
Lanki T; Pekkanen J; Aalto P; Elosua R; Berglind N; D'Ippoliti D; Kulmala M; Nyberg F; Peters A; Picciotto S; Salomaa V; Sunyer J; Tiittanen P; von Klot S; Forastiere F. (2006a). Associations of traffic related air pollutants with hospitalisation for first acute myocardial infarction: the HEAPSS study. Occupational and environmental medicine 63: 844-851.
Lanki T; de Hartog JJ; Heinrich J; Hoek G; Janssen NA; Peters A; Stolzel M; Timonen KL; Vallius M; Vanninen E; Pekkanen J. (2006b). Can we identify sources of fine particles responsible for exercise-induced ischemia on days with elevated air pollution? The ULTRA study. Environ Health Perspect 114: 655-660.
Larrieu S; Jusot JF; Blanchard M; Prouvost H; Declercq C; Fabre P; Pascal L; Tertre AL; Wagner V; Riviere S; Chardon B; Borrelli D; Cassadou S; Eilstein D; Lefranc A. (2007). Short term effects of air pollution on hospitalizations for cardiovascular diseases in eight French cities: the PSAS program. The Science of the total environment 387: 105-112.
Le Tertre A; Medina S; Samoli E; Forsberg B; Michelozzi P; Boumghar A; Vonk JM; Bellini A; Atkinson R; Ayres JG; Sunyer J; Schwartz J; Katsouyanni K. (2002). Short-term effects of particulate air pollution on cardiovascular diseases in eight European cities. Journal of epidemiology and community health 56: 773-779.
Le Tertre A; Schwartz J; Touloumi G. (2005). Empirical Bayes and adjusted estimates approach to estimating the relation of mortality to exposure of PM(10). Risk Anal 25: 711-718.
Lee D; Shaddick G. (2007). Time-varying coefficient models for the analysis of air pollution and health outcome data. Biometrics 63: 1253-1261.
Lee JT; Kim H; Song H; Hong YC; Cho YS; Shin SY; Hyun YJ; Kim YS. (2002). Air pollution and asthma among children in Seoul, Korea. Epidemiology 13: 481-484.
Lee JT; Son JY; Cho YS. (2007a). A comparison of mortality related to urban air particles between periods with Asian dust days and without Asian dust days in Seoul, Korea, 2000-2004. Environ Res 105: 409-413.
Lee JT; Son JY; Cho YS. (2007b). The adverse effects of fine particle air pollution on respiratory function in the elderly. The Science of the total environment 385: 28-36.
Lee SL; Wong WH; Lau YL. (2006). Association between air pollution and asthma admission among children in Hong Kong. Clin Exp Allergy 36: 1138-1146.
Leem JH; Kaplan BM; Shim YK; Pohl HR; Gotway CA; Bullard SM; Rogers JF; Smith MM; Tylenda CA. (2006). Exposures to air pollutants during pregnancy and preterm delivery. Environ Health Perspect 114: 905-910.
Leonardi GS; Houthuijs D; Steerenberg PA; Fletcher T; Armstrong B; Antova T; Lochman I; Lochmanova A; Rudnai P; Erdei E; Musial J; Jazwiec-Kanyion B; Niciu EM; Durbaca S; Fabianova E; Koppova K; Lebret E; Brunekreef B; van Loveren H. (2000). Immune biomarkers in relation to exposure to particulate matter: a cross-sectional survey in 17 cities of Central Europe. Inhal Toxicol 12 Suppl 4: 1-14.
Letz AG; Quinn JM. (2005). Relationship of basic military trainee emergency department visits for asthma and San Antonio air quality. Allergy Asthma Proc 26: 463-467.
Lewis TC; Robins TG; Dvonch JT; Keeler GJ; Yip FY; Mentz GB; Lin X; Parker EA; Israel BA; Gonzalez L; Hill Y. (2005). Air pollution-associated changes in lung function among asthmatic children in Detroit. Environ Health Perspect 113: 1068-1075.
Liao D; Duan Y; Whitsel EA; Zheng Z-j; Heiss G; Chinchilli VM; Lin H-M. (2004). Association of Higher Levels of Ambient Criteria Pollutants with Impaired Cardiac Autonomic Control: A Population-based Study. Am J Epidemiol 159: 768-777.

Lin CA; Pereira LA; Nishioka DC; Conceicao GM; Braga AL; Saldiva PH. (2004a). Air pollution and neonatal deaths in Sao Paulo, Brazil. Braz J Med Biol Res 37: 765-770.
Lin CM; Li CY; Mao IF. (2004b). Increased risks of term low-birth-weight infants in a petrochemical industrial city with high air pollution levels. Archives of environmental health 59: 663-668.
Lin M; Chen Y; Burnett RT; Villeneuve PJ; Krewski D. (2002). The influence of ambient coarse particulate matter on asthma hospitalization in children: case-crossover and time-series analyses. Environ Health Perspect 110: 575-581.
Lin M; Stieb DM; Chen Y. (2005). Coarse particulate matter and hospitalization for respiratory infections in children younger than 15 years in Toronto: a case-crossover analysis. Pediatrics 116: e235-240.
Linares C; Diaz J; Tobias A; De Miguel JM; Otero A. (2006). Impact of urban air pollutants and noise levels over daily hospital admissions in children in Madrid: a time series analysis. Int Arch Occup Environ Health 79: 143-152.
Lipfert F; Wyzga R; Baty J; Miller J. (2006a). Traffic density as a surrogate measure of environmental exposures in studies of air pollution health effects: Long-term mortality in a cohort of US veterans. Atmospheric Environment 40: 154-169.
Lipfert FW; Zhang J; Wyzga RE. (2000). Infant mortality and air pollution: a comprehensive analysis of U.S. data 1990. J Air Waste Manag Assoc 50: 1350-1366.
Lipfert FW; Baty JD; Miller JP; Wyzga RE. (2006b). PM2.5 constituents and related air quality variables as predictors of survival in a cohort of U.S. military veterans. Inhal Toxicol 18: 645-657.
Lippmann M; Ito K; Hwang JS; Maciejczyk P; Chen LC. (2006). Cardiovascular effects of nickel in ambient air. Environ Health Perspect 114: 1662-1669.
Lipsett MJ; Tsai FC; Roger L; Woo M; Ostro BD. (2006). Coarse particles and heart rate variability among older adults with coronary artery disease in the Coachella Valley, California. Environ Health Perspect 114: 1215-1220.
Lisabeth LD; Escobar JD; Dvonch JT; Sanchez BN; Majersik JJ; Brown DL; Smith MA; Morgenstern LB. (2008). Ambient air pollution and risk for ischemic stroke and transient ischemic attack. Annals of neurology 64: 53-59.
Liu CC; Chen CC; Wu TN; Yang CY. (2008). Association of brain cancer with residential exposure to petrochemical air pollution in Taiwan. J Toxicol Environ Health A 71: 310-314.
Liu L; Ruddy TD; Dalipaj M; Szyszkowicz M; You H; Poon R; Wheeler A; Dales R. (2007). Influence of personal exposure to particulate air pollution on cardiovascular physiology and biomarkers of inflammation and oxidative stress in subjects with diabetes. J Occup Environ Med 49: 258-265.
Loomis D; Castillejos M; Gold DR; McDonnell W; Borja-Aburto VH. (1999). Air pollution and infant mortality in Mexico City. Epidemiology 10: 118-123.
Lubinski W; Toczyska I; Chcialowski A; Plusa T. (2005). Influence of air pollution on pulmonary function in healthy young men from different regions of Poland. Ann Agric Environ Med 12: 1-4.
Luttmann-Gibson H; Suh H; Coull BA; Dockery DW; Sarnat SE; Schwartz J; Stone PH; Gold DR. (2006). Short-term effects of air pollution on heart rate variability in senior adults in Steubenville, Ohio. J Occup Environ Med 48: 780-788.
Magas OK; Gunter JT; Regens JL. (2007). Ambient air pollution and daily pediatric hospitalizations for asthma. Environ Sci Pollut Res Int 14: 19-23.
Maheswaran R; Haining RP; Brindley P; Law J; Pearson T; Fryers PR; Wise S; Campbell MJ. (2005). Outdoor air pollution and stroke in Sheffield, United Kingdom: a small-area level geographical study. Stroke; a journal of cerebral circulation 36: 239-243.
Maisonet M; Bush TJ; Correa A; Jaakkola JJ. (2001). Relation between ambient air pollution and low birth weight in the Northeastern United States. Environ Health Perspect 109 Suppl 3: 351-356.
Mann JK; Tager IB; Lurmann F; Segal M; Quesenberry CP, Jr.; Lugg MM; Shan J; Van Den Eeden SK. (2002). Air pollution and hospital admissions for ischemic heart disease in persons with congestive heart failure or arrhythmia. Environ Health Perspect 110: 1247-1252.
Mannes T; Jalaludin B; Morgan G; Lincoln D; Sheppeard V; Corbett S. (2005). Impact of ambient air pollution on birth weight in Sydney, Australia. Occupational and environmental medicine 62: 524-530.
Mar TF; Larson TV; Stier RA; Claiborn C; Koenig JQ. (2004). An analysis of the association between respiratory symptoms in subjects with asthma and daily air pollution in Spokane, Washington. Inhal Toxicol 16: 809-815.
Mar TF; Jansen K; Shepherd K; Lumley T; Larson TV; Koenig JQ. (2005a). Exhaled nitric oxide in children with asthma and short-term PM2.5 exposure in Seattle. Environ Health Perspect 113: 1791-1794.
Mar TF; Koenig JQ; Jansen K; Sullivan J; Kaufman J; Trenga CA; Siahpush SH; Liu LJ; Neas L. (2005b). Fine particulate air pollution and cardiorespiratory effects in the elderly. Epidemiology 16: 681-687.
Martins LC; Latorre Mdo R; Cardoso MR; Goncalves FL; Saldiva PH; Braga AL. (2002). Air pollution and emergency room visits due to pneumonia and influenza in Sao Paulo, Brazil. Rev Saude Publica 36: 88-94.
Martins MC; Fatigati FL; Vespoli TC; Martins LC; Pereira LA; Martins MA; Saldiva PH; Braga AL. (2004). Influence of socioeconomic conditions on air pollution adverse health effects in elderly people: an analysis of six regions in Sao Paulo, Brazil. Journal of epidemiology and community health 58: 41-46.
Masjedi MR; Jamaati HR; Dokouhaki P; Ahmadzadeh Z; Taheri SA; Bigdeli M; Izadi S; Rostamian A; Aagin K; Ghavam SM. (2003). The effects of air pollution on acute respiratory conditions. Respirology 8: 213-230.
McConnell R; Berhane K; Gilliland F; London SJ; Vora H; Avol E; Gauderman WJ; Margolis HG; Lurmann F; Thomas DC. (1999a). Air pollution and bronchitic symptoms in southern California children with asthma. Environ Health Perspect 107: 757-760.

McConnell R; Berhane K; Gilliland F; London SJ; Vora H; Avol E; Gauderman WJ; Margolis HG; Lurmann F; Thomas DC; Peters JM. (1999b). Air pollution and bronchitic symptoms in Southern California children with asthma. Environ Health Perspect 107: 757-760.
McConnell R; Berhane K; Gilliland F; London SJ; Islam T; Gauderman WJ; Avol E; Margolis HG; Peters JM. (2002). Asthma in exercising children exposed to ozone: a cohort study. Lancet 359: 386-391.
McConnell R; Berhane K; Gilliland F; Molitor J; Thomas D; Lurmann F; Avol E; Gauderman WJ; Peters JM. (2003). Prospective study of air pollution and bronchitic symptoms in children with asthma. American journal of respiratory and critical care medicine 168: 790-797.
McConnell R; Berhane K; Yao L; Jerrett M; Lurmann F; Gilliland F; Kunzli N; Gauderman J; Avol E; Thomas D; Peters J. (2006). Traffic, susceptibility, and childhood asthma. Environ Health Perspect 114: 766-772.
McCreanor J; Cullinan P; Nieuwenhuijsen MJ; Stewart-Evans J; Malliarou E; Jarup L; Harrington R; Svartengren M; Han IK; Ohman-Strickland P; Chung KF; Zhang J. (2007). Respiratory effects of exposure to diesel traffic in persons with asthma. The New England journal of medicine 357: 2348-2358.
McDonnell WF; Nishino-Ishikawa N; Petersen FF; Chen LH; Abbey DE. (2000). Relationships of mortality with the fine and coarse fractions of long-term ambient PM10 concentrations in nonsmokers. Journal of exposure analysis and environmental epidemiology 10: 427-436.
McGowan JA; Hider RN; Chacko E; Town GI. (2002). Particulate air pollution and hospital admissions in Christchurch, New Zealand. Aust N Z J Public Health 26: 23-29.
Mebust MR; Eder BK; Binkowski FS; Roselle SJ. (2003). Models-3 community multiscale air quality (CMAQ) model aerosol component 2. Model evaluation. J Geophys Res 108: 4184.
Medina-Ramon M; Zanobetti A; Schwartz J. (2006). The effect of ozone and PM10 on hospital admissions for pneumonia and chronic obstructive pulmonary disease: a national multicity study. Am J Epidemiol 163: 579-588.
Meng YY; Wilhelm M; Rull RP; English P; Ritz B. (2007). Traffic and outdoor air pollution levels near residences and poorly controlled asthma in adults. Ann Allergy Asthma Immunol 98: 455-463.
Metzger KB; Tolbert PE; Klein M; Peel JL; Flanders WD; Todd K; Mulholland JA; Ryan PB; Frumkin H. (2004). Ambient air pollution and cardiovascular emergency department visits. Epidemiology 15: 46-56.
Metzger KB; Klein M; Flanders WD; Peel JL; Mulholland JA; Langberg JJ; Tolbert PE. (2007). Ambient air pollution and cardiac arrhythmias in patients with implantable defibrillators. Epidemiology 18: 585-592.
Michaud JP; Grove JS; Krupitsky DCEH. (2004). Emergency department visits and "vog"-related air quality in Hilo, Hawai'i. Environ Res 95: 11-19.
Middleton N; Yiallouros P; Kleanthous S; Kolokotroni O; Schwartz J; Dockery DW; Demokritou P; Koutrakis P. (2008). A 10-year time-series analysis of respiratory and cardiovascular morbidity in Nicosia, Cyprus: the effect of short-term changes in air pollution and dust storms. Environ Health 7: 39.
Miller KA; Siscovick DS; Sheppard L; Shepherd K; Sullivan JH; Anderson GL; Kaufman JD. (2007). Long-term exposure to air pollution and incidence of cardiovascular events in women. The New England journal of medicine 356: 447-458.
Millstein J; Gilliland F; Berhane K; Gauderman WJ; McConnell R; Avol E; Rappaport EB; Peters JM. (2004). Effects of ambient air pollutants on asthma medication use and wheezing among fourth-grade school children from 12 Southern California communities enrolled in The Children's Health Study. Archives of environmental health 59: 505-514.
Morgenstern V; Zutavern A; Cyrys J; Brockow I; Gehring U; Koletzko S; Bauer CP; Reinhardt D; Wichmann HE; Heinrich J. (2007). Respiratory health and individual estimated exposure to traffic-related air pollutants in a cohort of young children. Occup Environ Med 64: 8.
Mortimer K; Neugebauer R; Lurmann F; Alcorn S; Balmes J; Tager I. (2008). Air pollution and pulmonary function in asthmatic children: effects of prenatal and lifetime exposures. Epidemiology 19: 550-557; discussion 561-552.
Mortimer KM; Neas LM; Dockery DW; Redline S; Tager IB. (2002). The effect of air pollution on inner-city children with asthma. Eur Respir J 19: 699-705.
Moshammer H; Neuberger M. (2003). The active surface of suspended particles as a predictor of lung function and pulmonary symptoms in Austrian school children. Atmospheric Environment 37: 1737-1744.
Moshammer H; Hutter HP; Hauck H; Neuberger M. (2006). Low levels of air pollution induce changes of lung function in a panel of schoolchildren. Eur Respir J 27: 1138-1143.
Murata A; Kida K; Hasunuma H; Kanegae H; Ishimaru Y; Motegi T; Yamada K; Yoshioka H; Yamamoto K; Kudoh S. (2007). Environmental influence on the measurement of exhaled nitric oxide concentration in school children: special reference to methodology. J Nippon Med Sch 74: 30-36.
Naess O; Nafstad P; Aamodt G; Claussen B; Rosland P. (2007a). Relation between concentration of air pollution and cause-specific mortality: four-year exposures to nitrogen dioxide and particulate matter pollutants in 470 neighborhoods in Oslo, Norway. Am J Epidemiol 165: 435-443.
Naess O; Piro FN; Nafstad P; Smith GD; Leyland AH. (2007b). Air pollution, social deprivation, and mortality: a multilevel cohort study. Epidemiology 18: 686-694.
Nafstad P; Haheim LL; Wisloff T; Gram F; Oftedal B; Holme I; Hjermann I; Leren P. (2004). Urban air pollution and mortality in a cohort of Norwegian men. Environ Health Perspect 112: 610-615.
Nascimento LF; Pereira LA; Braga AL; Modolo MC; Carvalho JA, Jr. (2006). [Effects of air pollution on children's health in a city in Southeastern Brazil]. Rev Saude Publica 40: 77-82.

Nawrot TS; Torfs R; Fierens F; De Henauw S; Hoet PH; Van Kersschaever G; De Backer G; Nemery B. (2007). Stronger associations between daily mortality and fine particulate air pollution in summer than in winter: evidence from a heavily polluted region in western Europe. Journal of epidemiology and community health 61: 146-149.
Nerriere E; Zmirou-Navier D; Desqueyroux P; Leclerc N; Momas I; Czernichow P. (2005). Lung cancer risk assessment in relation with personal exposure to airborne particles in four French metropolitan areas. J Occup Environ Med 47: 1211-1217.
Neuberger M; Schimek MG; Horak F; Moshammer H; Kundi M; Frischer T; Gomiscek B; Puxbaum H; Hauck H; Auphep-Team. (2004). Acute effects of particulate matter on respiratory diseases, symptoms and functions: epidemiological results of the Austrian Project on Health Effects of Particulate Matter (AUPHEP). Atmos Environ 38: 3971-3981.
O'Connor GT; Neas L; Vaughn B; Kattan M; Mitchell H; Crain EF; Evans R, 3rd; Gruchalla R; Morgan W; Stout J; Adams GK; Lippmann M. (2008). Acute respiratory health effects of air pollution on children with asthma in US inner cities. J Allergy Clin Immunol 121: 1133-1139 e1131.
O'Neill MS; Loomis D; Borja Aburto VH; Gold D; Hertz-Picciotto I; Castillejos M. (2004). Do associations between airborne particles and daily mortality in Mexico City differ by measurement method, region, or modeling strategy? J Expo Anal Environ Epidemiol 14: 429-439.
O'Neill MS; Veves A; Zanobetti A; Sarnat JA; Gold DR; Economides PA; Horton ES; Schwartz J. (2005a). Diabetes enhances vulnerability to particulate air pollution-associated impairment in vascular reactivity and endothelial function. Circulation 111: 2913-2920.
O'Neill MS; Hajat S; Zanobetti A; Ramirez-Aguilar M; Schwartz J. (2005b). Impact of control for air pollution and respiratory epidemics on the estimated associations of temperature and daily mortality. Int J Biometeorol 50: 121-129.
O'Neill MS; Veves A; Sarnat JA; Zanobetti A; Gold DR; Economides PA; Horton ES; Schwartz J. (2007). Air pollution and inflammation in type 2 diabetes: a mechanism for susceptibility. Occupational and environmental medicine 64: 373-379.
O'Neill MS; Diez-Roux AV; Auchincloss AH; Franklin TG; Jacobs DR, Jr.; Astor BC; Dvonch JT; Kaufman J. (2008). Airborne particulate matter exposure and urinary albumin excretion: the Multi-Ethnic Study of Atherosclerosis. Occupational and environmental medicine 65: 534-540.
O'Neill MS; Diez-Roux AV; Auchincloss AH; Franklin TG; Jacobs Jnr DR; Astor BC; Dvonch JT; Kaufman J. (2007). Airborne particulate matter exposure and urinary albumin excretion: The Multi-Ethnic Study of Atherosclerosis. Occupational and environmental medicine.
Oftedal B; Nafstad P; Magnus P; Bjorkly S; Skrondal A. (2003). Traffic related air pollution and acute hospital admission for respiratory diseases in Drammen, Norway 1995-2000. European journal of epidemiology 18: 671-675.
Oftedal B; Brunekreef B; Nystad W; Madsen C; Walker SE; Nafstad P. (2008). Residential outdoor air pollution and lung function in schoolchildren. Epidemiology 19: 129-137.
Ostro B; Broadwin R; Green S; Feng WY; Lipsett M. (2006). Fine particulate air pollution and mortality in nine California counties: results from CALFINE. Environ Health Perspect 114: 29-33.
Ostro B; Feng WY; Broadwin R; Green S; Lipsett M. (2007). The effects of components of fine particulate air pollution on mortality in california: results from CALFINE. Environ Health Perspect 115: 13-19.
Ostro B; Feng WY; Broadwin R; Malig B; Green S; Lipsett M. (2008). The Impact of Components of Fine Particulate Matter on Cardiovascular Mortality in Susceptible Subpopulations. Occupational and environmental medicine.
Ozkaynak H; Thurston GD. (1987). Associations between 1980 U.S. mortality rates and alternative measures of airborne particle concentration. Risk Anal 7: 449-461.
Park K; Chow JC; Watson JG; Trimble DL; Doraiswamy P; Arnott WP; Stroud KR; Bowers K; Bode R. (2006a). Comparison of Continuous and Filter-Based Carbon Measurements at the Fresno Supersite. Journal of the Air & Waste Management Association 56: 474-491.
Park SK; O'Neill MS; Vokonas PS; Sparrow D; Schwartz J. (2005). Effects of air pollution on heart rate variability: The VA Normative Aging Study. Environ Health Perspect 113: 304-309.
Park SK; O'Neill MS; Wright RO; Hu H; Vokonas PS; Sparrow D; Suh H; Schwartz J. (2006b). HFE Genotype, Particulate Air Pollution, and Heart Rate Variability: A Gene-Environment Interaction. Circulation 114: 2798-2805.
Park SK; O'Neill MS; Vokonas PS; Sparrow D; Spiro A, 3rd; Tucker KL; Suh H; Hu H; Schwartz J. (2008). Traffic-related particles are associated with elevated homocysteine: the VA normative aging study. American journal of respiratory and critical care medicine 178: 283-289.
Parker JD; Woodruff TJ; Basu R; Schoendorf KC. (2005). Air pollution and birth weight among term infants in California. Pediatrics 115: 121-128.
Parker JD; Woodruff TJ. (2008). Influences of study design and location on the relationship between particulate matter air pollution and birthweight. Paediatr Perinat Epidemiol 22: 214-227.
Peacock JL; Symonds P; Jackson P; Bremner SA; Scarlett JF; Strachan DP; Anderson HR. (2003). Acute effects of winter air pollution on respiratory function in schoolchildren in southern England. Occupational and environmental medicine 60: 82-89.
Peel JL; Tolbert PE; Klein M; Metzger KB; Flanders WD; Todd K; Mulholland JA; Ryan PB; Frumkin H. (2005). Ambient air pollution and respiratory emergency department visits. Epidemiology 16: 164-174.
Peel JL; Metzger KB; Klein M; Flanders WD; Mulholland JA; Tolbert PE. (2007). Ambient air pollution and cardiovascular emergency department visits in potentially sensitive groups. Am J Epidemiol 165: 625-633.

Pekkanen J; Peters A; Hoek G; Tiittanen P; Brunekreef B; de Hartog J; Heinrich J; Ibald-Mulli A; Kreyling WG; Lanki T; Timonen KL; Vanninen E. (2002). Particulate air pollution and risk of ST-segment depression during repeated submaximal exercise tests among subjects with coronary heart disease: the Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air (ULTRA) study. Circulation 106: 933-938.
Peled R; Friger M; Bolotin A; Bibi H; Epstein L; Pilpel D; Scharf S. (2005). Fine particles and meteorological conditions are associated with lung function in children with asthma living near two power plants. Public Health 119: 418-425.
Penard-Morand C; Charpin D; Raherison C; Kopferschmitt C; Caillaud D; Lavaud F; Annesi-Maesano I. (2005). Long-term exposure to background air pollution related to respiratory and allergic health in schoolchildren. Clin Exp Allergy 35: 1279-1287.
Peng RD; Dominici F; Pastor-Barriuso R; Zeger SL; Samet JM. (2005). Seasonal analyses of air pollution and mortality in 100 US cities. Am J Epidemiol 161: 585-594.
Peng RD; Chang HH; Bell ML; McDermott A; Zeger SL; Samet JM; Dominici F. (2008). Coarse particulate matter air pollution and hospital admissions for cardiovascular and respiratory diseases among Medicare patients. Jama 299: 2172-2179.
Penttinen P; Tiittanen P; Pekkanen J. (2004). Mortality and air pollution in metropolitan Helsinki, 1988-1996. Scand J Work Environ Health 30 Suppl 2: 19-27.
Penttinen P; Vallius M; Tiittanen P; Ruuskanen J; Pekkanen J. (2006). Source-specific fine particles in urban air and respiratory function among adult asthmatics. Inhal Toxicol 18: 191-198.
Pereira LAA; Loomis D; Conceição GMS; Braga ALF; Arcas RM; Kishi HS; Singer JM; Böhm GM; Saldiva PHN. (1998). Association between air pollution and intrauterine mortality in São Paulo, Brazil. Environ Health Perspect 106: 325-329.
Peters A; von Klot S; Heier M; Trentinaglia I; Cyrys J; Hormann A; Hauptmann M; Wichmann HE; Lowel H. (2005). Particulate air pollution and nonfatal cardiac events. Part I. Air pollution, personal activities, and onset of myocardial infarction in a case-crossover study. Res Rep Health Eff Inst 124: 1-66; discussion 67-82, 141-148.
Peters JM; Avol E; Navidi W; London SJ; Gauderman WJ; Lurmann F; Linn WS; Margolis H; Rappaport E; Gong H; Thomas DC. (1999). A study of twelve Southern California communities with differing levels and types of air pollution. I. Prevalence of respiratory morbidity. American journal of respiratory and critical care medicine 159: 760-767.
Pierse N; Rushton L; Harris RS; Kuehni CE; Silverman M; Grigg J. (2006). Locally generated particulate pollution and respiratory symptoms in young children. Thorax 61: 216-220.
Pino P; Walter T; Oyarzun M; Villegas R; Romieu I. (2004). Fine particulate matter and wheezing illnesses in the first year of life. Epidemiology 15: 702-708.
Pirjola L; Parviainen H; Hussein T; Valli A; Hämeri K; Aaalto P; Virtanen A; Keskinen J; Pakkanen TA; Mäkelä T. (2004). "Sniffer"—a novel tool for chasing vehicles and measuring traffic pollutants. Atmos Environ 38: 3625-3635.
Pitard A; Zeghnoun A; Courseaux A; Lamberty J; Delmas V; Fossard JL; Villet H. (2004). Short-term associations between air pollution and respiratory drug sales. Environmental Research 95: 43-52.
Pope CA; Hansen ML; Long RW; Nielsen KR; Eatough NL; Wilson WE; Eatough DJ. (2004). Ambient particulate air pollution, heart rate variability, and blood markers of inflammation in a panel of elderly subjects. Environ Health Perspect 112: 339-345.
Pope CA; Dockery DW. (2006). Health effects of fine particulate air pollution: Lines that connect. J Air Waste Manag Assoc 56: 709-742.
Pope CA, 3rd; Burnett RT; Thun MJ; Calle EE; Krewski D; Ito K; Thurston GD. (2002). Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 287: 1132-1141.
Pope CA, 3rd; Muhlestein JB; May HT; Renlund DG; Anderson JL; Horne BD. (2006). Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. Circulation 114: 2443-2448.
Pope CA, 3rd; Rodermund DL; Gee MM. (2007). Mortality effects of a copper smelter strike and reduced ambient sulfate particulate matter air pollution. Environ Health Perspect 115: 679-683.
Preutthipan A; Udomsubpayakul U; Chaisupamongkollarp T; Pentamwa P. (2004). Effect of PM10 pollution in Bangkok on children with and without asthma. Pediatr Pulmonol 37: 187-192.
Puett RC; Schwartz J; Hart JE; Yanosky JD; Speizer FE; Suh H; Paciorek CJ; Neas LM; Laden F. (2008). Chronic Particulate Exposure, Mortality, and Coronary Heart Disease in the Nurses' Health Study. Am J Epidemiol.
Qian Z; Liao D; Lin HM; Whitsel EA; Rose KM; Duan Y. (2005). Lung function and long-term exposure to air pollutants in middle-aged American adults. Arch Environ Occup Health 60: 156-163.
Qian Z; He Q; Kong L; Xu F; Wei F; Chapman RS; Chen W; Edwards RD; Bascom R. (2007). Respiratory responses to diverse indoor combustion air pollution sources. IndoorAir 17: 135-142.
Qian Z; He Q; Lin HM; Kong L; Bentley CM; Liu W; Zhou D. (2008). High temperatures enhanced acute mortality effects of ambient particle pollution in the "oven" city of Wuhan, China. Environ Health Perspect 116: 1172-1178.
Rabinovitch N; Zhang L; Murphy JR; Vedal S; Dutton SJ; Gelfand EW. (2004). Effects of wintertime ambient air pollutants on asthma exacerbations in urban minority children with moderate to severe disease. J Allergy Clin Immunol 114: 1131-1137.
Rabinovitch N; Strand M; Gelfand EW. (2006). Particulate levels are associated with early asthma worsening in children with persistent disease. Am J Respir Crit Care Med 173: 1098-1105.
Rainham DG; Smoyer-Tomic KE; Sheridan SC; Burnett RT. (2005). Synoptic weather patterns and modification of the association between air pollution and human mortality. Int J Environ Health Res 15: 347-360.

Ranzi A; Gambini M; Spattini A; Galassi C; Sesti D; Bedeschi M; Messori A; Baroni A; Cavagni G; Lauriola P. (2004). Air pollution and respiratory status in asthmatic children: hints for a locally based preventive strategy. AIRE study. European journal of epidemiology 19: 567-576.
Ren C; Williams GM; Tong S. (2006). Does particulate matter modify the association between temperature and cardiorespiratory diseases? Environ Health Perspect 114: 1690-1696.
Rich DQ; Schwartz J; Mittleman MA; Link M; Luttmann-Gibson H; Catalano PJ; Speizer FE; Dockery DW. (2005). Association of short-term ambient air pollution concentrations and ventricular arrhythmias. Am J Epidemiol 161: 1123-1132.
Rich DQ; Mittleman MA; Link MS; Schwartz J; Luttmann-Gibson H; Catalano PJ; Speizer FE; Gold DR; Dockery DW. (2006a). Increased risk of paroxysmal atrial fibrillation episodes associated with acute increases in ambient air pollution. Environ Health Perspect 114: 120-123.
Rich DQ; Kim MH; Turner JR; Mittleman MA; Schwartz J; Catalano PJ; Dockery DW. (2006b). Association of ventricular arrhythmias detected by implantable cardioverter defibrillator and ambient air pollutants in the St Louis, Missouri metropolitan area. Occupational and environmental medicine 63: 591-596.
Rich KE; Petkau J; Vedal S; Brauer M. (2004). A case-crossover analysis of particulate air pollution and cardiac arrhythmia in patients with implantable cardioverter defibrillators. Inhal Toxicol 16: 363-372.
Riediker M; Devlin RB; Griggs TR; Herbst MC; Bromberg PA; Williams RW; Cascio WE. (2004). Cardiovascular effects in partrol officers are associated with fine particulate matter from brake wear and engine emissions. Particle Fibre Toxicol 1.
Riojas-Rodriguez H; Escamilla-Cejudo JA; Gonzalez-Hermosillo JA; Tellez-Rojo MM; Vallejo M; Santos-Burgoa C; Rojas-Bracho L. (2006). Personal PM2.5 and CO exposures and heart rate variability in subjects with known ischemic heart disease in Mexico City. Journal of Exposure Science and Environmental Epidemiology 16: 131-137.
Rios JL; Boechat JL; Sant'Anna CC; Franca AT. (2004). Atmospheric pollution and the prevalence of asthma: study among schoolchildren of 2 areas in Rio de Janeiro, Brazil. Ann Allergy Asthma Immunol 92: 629-634.
Ritz B; Yu F; Fruin S; Chapa G; Shaw GM; Harris JA. (2002). Ambient air pollution and risk of birth defects in Southern California. Am J Epidemiol 155: 17-25.
Ritz B; Wilhelm M; Zhao Y. (2006). Air pollution and infant death in southern California, 1989-2000. Pediatrics 118: 493-502.
Ritz B; Wilhelm M; Hoggatt KJ; Ghosh JK. (2007). Ambient air pollution and preterm birth in the environment and pregnancy outcomes study at the University of California, Los Angeles. Am J Epidemiol 166: 1045-1052.
Roberts S. (2004a). Biologically plausible particulate air pollution mortality concentration-response functions. Environ Health Perspect 112: 309-313.
Roberts S. (2004b). Interactions between particulate air pollution and temperature in air pollution mortality time series studies. Environ Res 96: 328-337.
Roberts S. (2005). Using moving total mortality counts to obtain improved estimates for the effect of air pollution on mortality. Environ Health Perspect 113: 1148-1152.
Roberts S; Martin MA. (2006a). The question of nonlinearity in the dose-response relation between particulate matter air pollution and mortality: can Akaike's Information Criterion be trusted to take the right turn? Am J Epidemiol 164: 1242-1250.
Roberts S; Martin MA. (2006b). Applying a moving total mortality count to the cities in the NMMAPS database to estimate the mortality effects of particulate matter air pollution. Occupational and environmental medicine 63: 193-197.
Roberts S. (2006). A new model for investigating the mortality effects of multiple air pollutants in air pollution mortality time-series studies. J Toxicol Environ Health A 69: 417-435.
Roberts S; Martin MA. (2007a). Methods for bias reduction in time-series studies of particulate matter air pollution and mortality. J Toxicol Environ Health A 70: 665-675.
Roberts S; Martin MA. (2007b). A distributed lag approach to fitting non-linear dose-response models in particulate matter air pollution time series investigations. Environ Res 104: 193-200.
Rodriguez C; Tonkin R; Heyworth J; Kusel M; De Klerk N; Sly PD; Franklin P; Runnion T; Blockley A; Landau L; Hinwood AL. (2007). The relationship between outdoor air quality and respiratory symptoms in young children. Int J Environ Health Res 17: 351-360.
Rogers JF; Dunlop AL. (2006). Air pollution and very low birth weight infants: a target population? Pediatrics 118: 156-164.
Rojas-Martinez R; Perez-Padilla R; Olaiz-Fernandez G; Mendoza-Alvarado L; Moreno-Macias H; Fortoul T; McDonnell W; Loomis D; Romieu I. (2007). Lung function growth in children with long-term exposure to air pollutants in Mexico City. American journal of respiratory and critical care medicine 176: 377-384.
Roman HA; Walker KD; Walsh TL; Conner L; Richmond HM; Hubbell BJ; Kinney PL. (2008). Expert judgment assessment of the mortality impact of changes in ambient fine particulate matter in the U.S. Environ Sci Technol 42: 2268-2274.
Romieu I; Ramírez-Aguilar M; Moreno-Macias H; Barraza-Villarreal A; Miller P; Hernández-Cadena L; Carbajal-Arroyo LA; Hernandez-Avila M. (2004). Infant mortality and air pollution: modifying effect by social class. J Occup Environ Med 46: 1210-1216.
Romieu I; Tellez-Rojo MM; Lazo M; Manzano-Patino A; Cortez-Lugo M; Julien P; Belanger MC; Hernandez-Avila M; Holguin F. (2005). Omega-3 Fatty Acid Prevents Heart Rate Variability Reductions Associated with Particulate Matter. Am J Respir Crit Care Med 172: 1534-1540.
Romieu I; Garcia-Esteban R; Sunyer J; Rios C; Alcaraz-Zubeldia M; Velasco SR; Holguin F. (2008). The effect of supplementation with omega-3 polyunsaturated fatty acids on markers of oxidative stress in elderly exposed to PM(2.5). Environ Health Perspect 116: 1237-1242.

Rosenlund M; Berglind N; Pershagen G; Hallqvist J; Jonson T; Bellander T. (2006). Long-term exposure to urban air pollution and myocardial infarction. Epidemiology 17: 383-390.
Rosenthal FS; Carney JP; Olinger ML. (2008). Out-of-hospital cardiac arrest and airborne fine particulate matter: a case-crossover analysis of emergency medical services data in Indianapolis, Indiana. Environ Health Perspect 116: 631-636.
Ruckerl R; Ibald-Mulli A; Koenig W; Schneider A; Woelke G; Cyrys J; Heinrich J; Marder V; Frampton M; Wichmann HE; Peters A. (2006). Air pollution and markers of inflammation and coagulation in patients with coronary heart disease. American journal of respiratory and critical care medicine 173: 432-441.
Ruckerl R; Phipps RP; Schneider A; Frampton M; Cyrys J; Oberdorster G; Wichmann HE; Peters A. (2007a). Ultrafine particles and platelet activation in patients with coronary heart disease--results from a prospective panel study. Part Fibre Toxicol 4: 1.
Ruckerl R; Greven S; Ljungman P; Aalto P; Antoniades C; Bellander T; Berglind N; Chrysohoou C; Forastiere F; Jacquemin B; von Klot S; Koenig W; Kuchenhoff H; Lanki T; Pekkanen J; Perucci CA; Schneider A; Sunyer J; Peters A. (2007b). Air pollution and inflammation (interleukin-6, C-reactive protein, fibrinogen) in myocardial infarction survivors. Environ Health Perspect 115: 1072-1080.
Sagiv SK; Mendola P; Loomis D; Herring AH; Neas LM; Savitz DA; Poole C. (2005). A time-series analysis of air pollution and preterm birth in Pennsylvania, 1997-2001. Environ Health Perspect 113: 602-606.
Sakai M; Sato Y; Sato S; Ihara S; Onizuka M; Sakakibara Y; Takahashi H. (2004). Effect of relocating to areas of reduced atmospheric particulate matter levels on the human circulating leukocyte count. J Appl Physiol 97: 1774-1780.
Salam MT; Millstein J; Li YF; Lurmann FW; Margolis HG; Gilliland FD. (2005). Birth outcomes and prenatal exposure to ozone, carbon monoxide, and particulate matter: results from the Children's Health Study. Environ Health Perspect 113: 1638-1644.
Saldiva PH; Clarke RW; Coull BA; Stearns RC; Lawrence J; Murthy GG; Diaz E; Koutrakis P; Suh H; Tsuda A; Godleski JJ. (2002). Lung inflammation induced by concentrated ambient air particles is related to particle composition. Am J Respir Crit Care Med 165: 1610-1617.
Samoli E; Analitis A; Touloumi G; Schwartz J; Anderson HR; Sunyer J; Bisanti L; Zmirou D; Vonk JM; Pekkanen J; Goodman P; Paldy A; Schindler C; Katsouyanni K. (2005). Estimating the exposure-response relationships between particulate matter and mortality within the APHEA multicity project. Environ Health Perspect 113: 88-95.
Sanchez-Carrillo CI; Ceron-Mireles P; Rojas-Martinez MR; Mendoza-Alvarado L; Olaiz-Fernandez G; Borja-Aburto VH. (2003). Surveillance of acute health effects of air pollution in Mexico City. Epidemiology 14: 536-544.
Sarnat JA; Marmur A; Klein M; Kim E; Russell AG; Sarnat SE; Mulholland JA; Hopke PK; Tolbert PE. (2008). Fine particle sources and cardiorespiratory morbidity: an application of chemical mass balance and factor analytical source-apportionment methods. Environ Health Perspect 116: 459-466.
Schikowski T; Sugiri D; Ranft U; Gehring U; Heinrich J; Wichmann HE; Kramer U. (2005). Long-term air pollution exposure and living close to busy roads are associated with COPD in women. Respir Res 6: 9921-9926.
Schikowski T; Sugiri D; Ranft U; Gehring U; Heinrich J; Wichmann HE; Kramer U. (2007). Does respiratory health contribute to the effects of long-term air pollution exposure on cardiovascular mortality? Respir Res 8: 20.
Schildcrout JS; Sheppard L; Lumley T; Slaughter JC; Koenig JQ; Shapiro GG. (2006). Ambient air pollution and asthma exacerbations in children: an eight-city analysis. Am J Epidemiol 164: 505-517.
Schwartz J; Laden F; Zanobetti A. (2002). The concentration-response relation between PM(2.5) and daily deaths. Environ Health Perspect 110: 1025-1029.
Schwartz J; Park SK; O'Neill MS; Vokonas PS; Sparrow D; Weiss S; Kelsey K. (2005a). Glutathione-S-Transferase M1, Obesity, Statins, and Autonomic Effects of Particles: Gene-by-Drug-by-Environment Interaction. Am J Respir Crit Care Med 172: 1529-1533.
Schwartz J; Litonjua A; Suh H; Verrier M; Zanobetti A; Syring M; Nearing B; Verrier R; Stone P; MacCallum G; Speizer FE; Gold DR. (2005b). Traffic related pollution and heart rate variability in a panel of elderly subjects. Thorax 60: 455-461.
Schwartz J; Coull B; Laden F; Ryan L. (2008). The effect of dose and timing of dose on the association between airborne particles and survival. Environ Health Perspect 116: 64-69.
Sharma M; Kumar VN; Katiyar SK; Sharma R; Shukla BP; Sengupta B. (2004). Effects of particulate air pollution on the respiratory health of subjects who live in three areas in Kanpur, India. Archives of environmental health 59: 348-358.
Silkoff PE; Zhang L; Dutton S; Langmack EL; Vedal S; Murphy J; Make B. (2005). Winter air pollution and disease parameters in advanced chronic obstructive pulmonary disease panels residing in Denver, Colorado. J Allergy Clin Immunol 115: 337-344.
Simpson R; Williams G; Petroeschevsky A; Best T; Morgan G; Denison L; Hinwood A; Neville GCEH. (2005). The short-term effects of air pollution on hospital admissions in four Australian cities. Aust N Z J Public Health 29: 213-221.
Slama R; Morgenstern V; Cyrys J; Zutavern A; Herbarth O; Wichmann HE; Heinrich J. (2007). Traffic-related atmospheric pollutants levels during pregnancy and offspring's term birth weight: a study relying on a land-use regression exposure model. Environ Health Perspect 115: 1283-1292.
Slaughter JC; Lumley T; Sheppard L; Koenig JQ; Shapiro GG. (2003). Effects of ambient air pollution on symptom severity and medication use in children with asthma. Ann Allergy Asthma Immunol 91: 346-353.
Slaughter JC; Kim E; Sheppard L; Sullivan JH; Larson TV; Claiborn C. (2005). Association between particulate matter and emergency room visits, hospital admissions and mortality in Spokane, Washington. Journal of exposure analysis and environmental epidemiology 15: 153-159.

Slowik JG. (2007). An Inter-Comparison of Instruments Measuring Black Carbon Content of Soot Particles. Aerosol Sci Technol 41: 295-314.
Sorensen M; Schins RP; Hertel O; Loft S. (2005). Transition metals in personal samples of PM2.5 and oxidative stress in human volunteers. Cancer Epidemiol Biomarkers Prev 14: 1340-1343.
Sørensen M; Daneshvar B; Hansen M; Dragsted LO; Hertel O; Knudsen L; Loft S. (2003). Personal PM2.5 exposure and markers of oxidative stress in blood. Environ Health Perspect 111: 161-166.
Stafoggia M; Schwartz J; Forastiere F; Perucci CA. (2008). Does temperature modify the association between air pollution and mortality? A multicity case-crossover analysis in Italy. Am J Epidemiol 167: 1476-1485.
Staniswalis JG; Parks NJ; Bader JO; Maldonado YM. (2005). Temporal analysis of airborne particulate matter reveals a dose-rate effect on mortality in El Paso: indications of differential toxicity for different particle mixtures. J Air Waste Manag Assoc 55: 893-902.
Stieb DM; Judek S; Burnett RT. (2002). Meta-analysis of time-series studies of air pollution and mortality: effects of gases and particles and the influence of cause of death, age, and season. J Air Waste Manag Assoc 52: 470-484.
Stölzel M; Breitner S; Cyrys J; Pitz M; Wolke G; Kreyling W; Heinrich J; Wichmann HE; Peters A. (2007). Daily mortality and particulate matter in different size classes in Erfurt, Germany. Journal of Exposure Science and Environmental Epidemiology 17: 458-467.
Strand M; Vedal S; Rodes C; Dutton SJ; Gelfand EW; Rabinovitch N. (2006). Estimating effects of ambient PM2.5 exposure on health using PM2.5 component measurements and regression calibration (vol 16, pg 30, 2006). Journal of Exposure Science and Environmental Epidemiology 16: 471-471.
Su TC; Chan CC; Liau CS; Lin LY; Kao HL; Chuang KJ. (2006). Urban air pollution increases plasma fibrinogen and plasminogen activator inhibitor-1 levels in susceptible patients. Eur J Cardiovasc Prev Rehabil 13: 849-852.
Suh YJ; Kim BM; Park BH; Park H; Kim YJ; Kim H; Hong YC; Ha EH. (2007). Cytochrome P450IA1 polymorphisms along with PM(10) exposure contribute to the risk of birth weight reduction. Reprod Toxicol 24: 281-288.
Sullivan J; Ishikawa N; Sheppard L; Siscovick D; Checkoway H; Kaufman J. (2003). Exposure to Ambient Fine Particulate Matter and Primary Cardiac Arrest among Persons With and Without Clinically Recognized Heart Disease. Am J Epidemiol 157: 501-509.
Sullivan J; Sheppard L; Schreuder A; Ishikawa N; Siscovick D; Kaufman J. (2005a). Relation between short-term fine-particulate matter exposure and onset of myocardial infarction. Epidemiology 16: 41-48.
Sullivan JH; Schreuder AB; Trenga CA; Liu SLJ; Larson TV; Koenig JQ; Kaufman JD. (2005b). Association between short term exposure to fine particulate matter and heart rate variability in older subjects with and without heart disease. Thorax 60: 462-466.
Sun HL; Chou MC; Lue KH. (2006). The relationship of air pollution to ED visits for asthma differ between children and adults. Am J Emerg Med 24: 709-713.
Sunyer J; Basagana X; Belmonte J; Anto JM. (2002). Effect of nitrogen dioxide and ozone on the risk of dying in patients with severe asthma. Thorax 57: 687-693.
Sunyer J; Jarvis D; Gotschi T; Garcia-Esteban R; Jacquemin B; Aguilera I; Ackerman U; de Marco R; Forsberg B; Gislason T; Heinrich J; Norback D; Villani S; Kunzli N. (2006). Chronic bronchitis and urban air pollution in an international study. Occupational and environmental medicine 63: 836-843.
Symons JM; Wang L; Guallar E; Howell E; Dominici F; Schwab M; Ange BA; Samet J; Ondov J; Harrison D; Geyh A. (2006). A Case-Crossover Study of Fine Particulate Matter Air Pollution and Onset of Congestive Heart Failure Symptom Exacerbation Leading to Hospitalization. Am J Epidemiol.
Tager IB; Balmes J; Lurmann F; Ngo L; Alcorn S; Künzli N. (2005). Chronic Exposure to Ambient Ozone and Lung Function in Young Adults. Epidemiology 16: 751.
Tainio M; Tuomisto JT; Hanninen O; Aarnio P; Koistinen KJ; Jantunen MJ; Pekkanen J. (2005). Health effects caused by primary fine particulate matter (PM2.5) emitted from buses in the Helsinki metropolitan area, Finland. Risk Anal 25: 151-160.
Tamura K; Jinsart W; Yano E; Karita K; Boudoung D. (2003). Particulate air pollution and chronic respiratory symptoms among traffic policemen in Bangkok. Archives of environmental health 58: 201-207.
Tang CS; Chang LT; Lee HC; Chan CC. (2007). Effects of personal particulate matter on peak expiratory flow rate of asthmatic children. The Science of the total environment 382: 43-51.
ten Brink H; Hoek G; Khlystov A. (2005). An approach to monitor the fraction of elemental carbon in the ultrafine aerosol. Atmos Environ 39: 6255-6259.
Thurston GD; Ito K; Mar T; Christensen WF; Eatough DJ; Henry RC; Kim E; Laden F; Lall R; Larson TV. (2005). Workgroup Report: Workshop on Source Apportionment of Particulate Matter Health Effects—Intercomparison of Results and Implications. Environmental health perspectives 113: 1768.
Timonen KL; Hoek G; Heinrich J; Bernard A; Brunekreef B; de Hartog J; Hameri K; Ibald-Mulli A; Mirme A; Peters A; Tiittanen P; Kreyling WG; Pekkanen J. (2004). Daily variation in fine and ultrafine particulate air pollution and urinary concentrations of lung Clara cell protein CC16. Occupational and environmental medicine 61: 908-914.
Timonen KL; Vanninen E; Hartog JD; Ibald-Mulli A; Brunekreef B; Gold DR; Heinrich J; Hoek G; Lanki T; Peters A; Tarkiainen T; Tiittanen P; Kreyling W; Pekkanen J. (2006). Effects of ultrafine and fine particulate and gaseous air pollution on cardiac autonomic control in subjects with coronary artery disease: The ULTRA study. JESEE 16: 332-341.

Tolbert PE; Klein M; Peel JL; Sarnat SE; Sarnat JA. (2007). Multipollutant modeling issues in a study of ambient air quality and emergency department visits in Atlanta. Journal of Exposure Science and Environmental Epidemiology 17: S29-S35.
Touloumi G; Samoli E; Quenel P; Paldy A; Anderson RH; Zmirou D; Galan I; Forsberg B; Schindler C; Schwartz J; Katsouyanni K. (2005). Short-term effects of air pollution on total and cardiovascular mortality: the confounding effect of influenza epidemics. Epidemiology 16: 49-57.
Tovalin H; Valverde M; Morandi MT; Blanco S; Whitehead L; Rojas E. (2006). DNA damage in outdoor workers occupationally exposed to environmental air pollutants. Occupational and environmental medicine 63: 230-236.
Trenga CA; Sullivan JH; Schildcrout JS; Shepherd KP; Shapiro GG; Liu LJ; Kaufman JD; Koenig JQ. (2006). Effect of particulate air pollution on lung function in adult and pediatric subjects in a Seattle panel study. Chest 129: 1614-1622.
Tsai SS; Huang CH; Goggins WB; Wu TN; Yang CY. (2003a). Relationship between air pollution and daily mortality in a tropical city: Kaohsiung, Taiwan. J Toxicol Environ Health A 66: 1341-1349.
Tsai SS; Goggins WB; Chiu HF; Yang CY. (2003b). Evidence for an association between air pollution and daily stroke admissions in Kaohsiung, Taiwan. Stroke; a journal of cerebral circulation 34: 2612-2616.
Tsai SS; Cheng MH; Chiu HF; Wu TN; Yang CY. (2006). Air pollution and hospital admissions for asthma in a tropical city: Kaohsiung, Taiwan. Inhal Toxicol 18: 549-554.
Ulirsch GV; Ball LM; Kaye W; Shy CM; Lee CV; Crawford-Brown D; Symons M; Holloway T. (2007). Effect of particulate matter air pollution on hospital admissions and medical visits for lung and heart disease in two southeast Idaho cities. Journal of Exposure Science and Environmental Epidemiology 17: 478-487.
Vajanapoom N; Shy CM; Neas LM; Loomis D. (2002). Associations of particulate matter and daily mortality in Bangkok, Thailand. Southeast Asian J Trop Med Public Health 33: 389-399.
Vallejo M; Ruiz S; Hermosillo AG; Borja-Aburto VH; Cardenas M. (2006). Ambient fine particles modify heart rate variability in young healthy adults. Journal of Exposure Science and Environmental Epidemiology 16: 125-130.
Vedal S; Brauer M; White R; Petkau J. (2003). Air pollution and daily mortality in a city with low levels of pollution. Environ Health Perspect 111: 45-52.
Vedal S; Rich K; Brauer M; White R; Petkau J. (2004). Air pollution and cardiac arrhythmias in patients with implantable cardioverter defibrillators. Inhal Toxicol 16: 353-362.
Venners SA; Wang B; Xu Z; Schlatter Y; Wang L; Xu X. (2003). Particulate matter, sulfur dioxide, and daily mortality in Chongqing, China. Environ Health Perspect 111: 562-567.
Vichit-Vadakan N; Vajanapoom N; Ostro B. (2008). The Public Health and Air Pollution in Asia (PAPA) Project: estimating the mortality effects of particulate matter in Bangkok, Thailand. Environ Health Perspect 116: 1179-1182.
Vigotti MA; Chiaverini F; Biagiola P; Rossi G. (2007). Urban air pollution and emergency visits for respiratory complaints in Pisa, Italy. J Toxicol Environ Health A 70: 266-269.
Villeneuve PJ; Goldberg MS; Krewski D; Burnett RT; Chen Y. (2002). Fine particulate air pollution and all-cause mortality within the Harvard Six-Cities Study: variations in risk by period of exposure. Ann Epidemiol 12: 568-576.
Villeneuve PJ; Burnett RT; Shi Y; Krewski D; Goldberg MS; Hertzman C; Chen Y; Brook J. (2003). A time-series study of air pollution, socioeconomic status, and mortality in Vancouver, Canada. Journal of exposure analysis and environmental epidemiology 13: 427-435.
Villeneuve PJ; Chen L; Stieb D; Rowe BH. (2006). Associations between outdoor air pollution and emergency department visits for stroke in Edmonton, Canada. European journal of epidemiology 21: 689-700.
Vinzents PS; Moller P; Sorensen M; Knudsen LE; Hertel O; Jensen FP; Schibye B; Loft S. (2005). Personal exposure to ultrafine particles and oxidative DNA damage. Environ Health Perspect 113: 1485-1490.
Violante FS; Barbieri A; Curti S; Sanguinetti G; Graziosi F; Mattioli S. (2006). Urban atmospheric pollution: Personal exposure versus fixed monitoring station measurements. Chemosphere 64: 1722-1729.
von Klot S; Wolke G; Tuch T; Heinrich J; Dockery DW; Schwartz J; Kreyling WG; Wichmann HE; Peters A. (2002). Increased asthma medication use in association with ambient fine and ultrafine particles. Eur Respir J 20: 691-702.
Ward DJ; Roberts KT; Jones N; Harrison RM; Ayres JG; Hussain S; Walters S. (2002). Effects of daily variation in outdoor particulates and ambient acid species in normal and asthmatic children. Thorax 57: 489-502.
Wellenius GA; Schwartz J; Mittleman MA. (2005a). Air pollution and hospital admissions for ischemic and hemorrhagic stroke among medicare beneficiaries. Stroke; a journal of cerebral circulation 36: 2549-2553.
Wellenius GA; Bateson TF; Mittleman MA; Schwartz J. (2005b). Particulate air pollution and the rate of hospitalization for congestive heart failure among medicare beneficiaries in Pittsburgh, Pennsylvania. Am J Epidemiol 161: 1030-1036.
Wellenius GA; Bateson TF; Mittleman MA; Schwartz J. (2005c). Particulate air pollution and the rate of hospitalization for congestive heart failure among medicare beneficiaries in Pittsburgh, Pennsylvania. Am J Epidemiol 161: 1030-1036.
Wellenius GA; Yeh GY; Coull BA; Suh HH; Phillips RS; Mittleman MA. (2007). Effects of ambient air pollution on functional status in patients with chronic congestive heart failure: a repeated-measures study. Environ Health 6: 26.
Weng HH; Tsai SS; Chiu HF; Wu TN; Yang CY. (2008). Association of childhood leukemia with residential exposure to petrochemical air pollution in taiwan. Inhal Toxicol 20: 31-36.
Westerdahl D; Fruin S; Sax T; Fine PM; Sioutas C. (2005). Mobile platform measurements of ultrafine particles and associated pollutant concentrations on freeways and residential streets in Los Angeles. Atmos Environ 39: 3597-3610.
Wheeler A; Zanobetti A; Gold DR; Schwartz J; Stone P; Suh HH. (2006). The relationship between ambient air pollution and heart rate variability differs for individuals with heart and pulmonary disease. Environ Health Perspect 114: 560-566.

Wheeler BW; Ben-Shlomo Y. (2005). Environmental equity, air quality, socioeconomic status, and respiratory health: a linkage analysis of routine data from the Health Survey for England. Journal of epidemiology and community health 59: 948-954.
Willis A; Jerrett M; Burnett RT; Krewski D. (2003). The association between sulfate air pollution and mortality at the county scale: an exploration of the impact of scale on a long-term exposure study. J Toxicol Environ Health A 66: 1605-1624.
Wilson WE; Grover BD; Long RW; Eatough NL; Eatough DJ. (2006). The measurement of fine particulate semivolatile material in urban aerosols. J Air Waste Manag Assoc 56: 384-397.
Wilson WE; Mar TF; Koenig JQ. (2007). Influence of exposure error and effect modification by socioeconomic status on the association of acute cardiovascular mortality with particulate matter in Phoenix. Journal of Exposure Science and Environmental Epidemiology 17: S11.
Wong CM; Ou CQ; Thach TQ; Chau YK; Chan KP; Ho SY; Chung RY; Lam TH; Hedley AJ. (2007). Does regular exercise protect against air pollution-associated mortality? Prev Med 44: 386-392.
Wong CM; Ou CQ; Chan KP; Chau YK; Thach TQ; Yang L; Chung RY; Thomas GN; Peiris JS; Wong TW; Hedley AJ; Lam TH. (2008a). The effects of air pollution on mortality in socially deprived urban areas in Hong Kong, China. Environ Health Perspect 116: 1189-1194.
Wong CM; Vichit-Vadakan N; Kan H; Qian Z. (2008b). Public Health and Air Pollution in Asia (PAPA): a multicity study of short-term effects of air pollution on mortality. Environ Health Perspect 116: 1195-1202.
Wong TW; Tam WS; Yu TS; Wong AH. (2002). Associations between daily mortalities from respiratory and cardiovascular diseases and air pollution in Hong Kong, China. Occupational and environmental medicine 59: 30-35.
Wong TW; Tam W; Tak Sun Yu I; Wun YT; Wong AH; Wong CM. (2006). Association between air pollution and general practitioner visits for respiratory diseases in Hong Kong. Thorax 61: 585-591.
Woodruff TJ; Grillo J; Schoendorf KC. (1997). The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. Environ Health Perspect 105.
Woodruff TJ; Parker JD; Schoendorf KC. (2006). Fine particulate matter ($PM_{2.5}$) air pollution and selected causes of postneonatal infant mortality in California. Environ Health Perspect 114: 786-790.
Woodruff TJ; Darrow LA; Parker JD. (2008). Air pollution and postneonatal infant mortality in the United States, 1999-2002. Environ Health Perspect 116: 110-115.
Xirasagar S; Lin HC; Liu TC. (2006). Seasonality in pediatric asthma admissions: the role of climate and environmental factors. Eur J Pediatr 165: 747-752.
Yang CY; Tseng YT; Chang CC. (2003). Effects of air pollution on birth weight among children born between 1995 and 1997 in Kaohsiung, Taiwan. J Toxicol Environ Health A 66: 807-816.
Yang CY; Chang CC; Chuang HY; Tsai SS; Wu TN; Ho CK. (2004a). Relationship between air pollution and daily mortality in a subtropical city: Taipei, Taiwan. Environ Int 30: 519-523.
Yang CY; Chen YS; Yang CH; Ho SC. (2004b). Relationship between ambient air pollution and hospital admissions for cardiovascular diseases in kaohsiung, taiwan. J Toxicol Environ Health A 67: 483-493.
Yang CY; Chen CC; Chen CY; Kuo HW. (2007). Air pollution and hospital admissions for asthma in a subtropical city: Taipei, Taiwan. J Toxicol Environ Health A 70: 111-117.
Yang CY; Chen CJ. (2007). Air pollution and hospital admissions for chronic obstructive pulmonary disease in a subtropical city: Taipei, Taiwan. J Toxicol Environ Health A 70: 1214-1219.
Yang CY. (2008). Air pollution and hospital admissions for congestive heart failure in a subtropical city: Taipei, Taiwan. J Toxicol Environ Health A 71: 1085-1090.
Yang Q; Chen Y; Krewski D; Shi Y; Burnett RT; McGrail KM. (2004c). Association between particulate air pollution and first hospital admission for childhood respiratory illness in Vancouver, Canada. Archives of environmental health 59: 14-21.
Yue W; Schneider A; Stolzel M; Ruckerl R; Cyrys J; Pan X; Zareba W; Koenig W; Wichmann HE; Peters A. (2007). Ambient source-specific particles are associated with prolonged repolarization and increased levels of inflammation in male coronary artery disease patients. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis 621: 50-60.
Zanobetti A; Schwartz J. (2002). Cardiovascular damage by airborne particles: are diabetics more susceptible? Epidemiology 13: 588-592.
Zanobetti A; Schwartz J; Samoli E; Gryparis A; Touloumi G; Peacock J; Anderson RH; Le Tertre A; Bobros J; Celko M; Goren A; Forsberg B; Michelozzi P; Rabczenko D; Hoyos SP; Wichmann HE; Katsouyanni K. (2003). The temporal pattern of respiratory and heart disease mortality in response to air pollution. Environ Health Perspect 111: 1188-1193.
Zanobetti A; Schwartz J. (2005). The effect of particulate air pollution on emergency admissions for myocardial infarction: a multicity case-crossover analysis. Environ Health Perspect 113: 978-982.
Zanobetti A; Schwartz J. (2006). Air pollution and emergency admissions in Boston, MA. Journal of epidemiology and community health 60: 890-895.
Zanobetti A; Schwartz J. (2007). Particulate air pollution, progression, and survival after myocardial infarction. Environ Health Perspect 115: 769-775.
Zeger S; McDermott A; Dominici F; Samet J. (2007). Mortality in the medicare population and chronic exposure to fine particulate air pollution. Johns Hopkins University, Dept of Biostatistics Working Papers 113.
Zeka A; Zanobetti A; Schwartz J. (2005). Short term effects of particulate matter on cause specific mortality: effects of lags and modification by city characteristics. Occupational and environmental medicine 62: 718-725.
Zeka A; Zanobetti A; Schwartz J. (2006a). Individual-level modifiers of the effects of particulate matter on daily mortality. Am J Epidemiol 163: 849-859.

Zeka A; Sullivan JR; Vokonas PS; Sparrow D; Schwartz J. (2006b). Inflammatory markers and particulate air pollution: characterizing the pathway to disease. Int J Epidemiol 35: 1347-1354.
Zhang JJ; Hu W; Wei F; Wu G; Korn LR; Chapman RS. (2002). Children's respiratory morbidity prevalence in relation to air pollution in four Chinese cities. EnvironHealth Perspect 110: 961-967.
Zhang KM; Wexler AS; Niemeier DA; Zhu YF; Hinds WC; Sioutas C. (2005). Evolution of particle number distribution near roadways. Part III: Traffic analysis and on-road size resolved particulate emission factors. Atmos Environ 39: 4155-4166.