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APPENDIX 8A

SHORT-TERM PM EXPOSURE-MORTALITY STUDIES: SUMMARY TABLE

TABLE 8A-1. SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States			
Samet et al. (2000a,b).* 90 largest U.S. cities. 1987-1994. PM_{10} mean ranged from 15.3 (Honolulu) to 52.0 (Riverside).	Non-accidental total deaths and cause-specific (cardiac, respiratory, and the other remaining) deaths, stratified in three age groups (<65, 65-75, 75+), were examined for their associations with PM_{10}, O_3, SO_2, NO_2, and CO (single, two, and three pollutant models) at lags 0, 1, and 2 days. In the first stage of the hierarchical model, RRs for the pollutants for each city were obtained using GAM Poisson regression models, adjusting for temperature and dewpoint (0-day and average of 1-3 days for both variables), day-of-week, seasonal cycles, intercept and seasonal cycles for three age groups. In the second stage, between-city variation in RRs were modeled within region. The third stage modeled between-region variation (7 regions). Two alternative assumptions were made regarding the prior distribution: one with possibly substantial heterogeneity and the other with less or no heterogeneity within region. The weighted second-stage regression included five types of county-specific variables: (1) mean weather and pollution variables; (2) mortality rate; (3) socio-demographic variables; (4) urbanization; (5) variables related to measurement error.	The estimated city-specific coefficients were mostly positive at lag 0, 1, and 2 days (estimated overall effect size was largest at lag 1, with the estimated percent excess death rate per 10 µg/m^3 PM_{10} being about 0.5%). The posterior probabilities that the overall effects are greater than 0 at these lags were 0.99, 1.00, and 0.98, respectively. None of the county-specific variables (effect modifiers) in the second-stage regression significantly explained the heterogeneity of PM_{10} effects across cities. In the 3-stage regression model with the index for 7 geographical regions, the effect of PM_{10} varied somewhat across the 7 regions, with the effect in the Northeast being the greatest. Adding O_3 and other gaseous pollutants did not markedly change the posterior distributions of PM_{10} effects. O_3 effects, as examined by season, were associated with mortality in summer (0.5 percent per 10 ppb increase), but not in all season data (negative in winter).	Posterior mean estimates and 95% credible intervals for total mortality excess deaths per 50 µg/m^3 increase in PM_{10} at lag 1 day: 2.3% (0.1, 4.5) for "more heterogeneity" across-city assumption; 2.2% (0.5, 4.0) for "less or no heterogeneity" across cities assumption. The largest PM_{10} effect estimated for 7 U.S. regions was for the Northeast: 4.6% (2.7, 6.5) excess deaths per 50 µg/m^3 PM_{10} increment.
Dominici et al. (2002). Re-analysis of above study.	Illustration of the issues related to GAM convergence criteria using simulation; and re-analysis of above study using stringent convergence criteria as well as comparable GLM model with natural splines.	The overall estimate was reduced but major findings of the study were not changed. Sensitivity analysis using alternative degrees of freedom for temporal trends and weather terms showed that PM_{10} risk estimates were larger when smaller number of degrees of freedom were used.	Posterior mean estimates and 95% credible intervals for total mortality excess deaths per 50 µg/m^3 increase in PM_{10} at lag 1 day: 1.4% (0.9, 1.9) using GAM with stringent convergence criteria and 1.1 (0.5, 1.7) using GLM with natural splines. Northeast still has the largest PM_{10} risk estimate.

+ = Used GAM with multiple non-parametric smooths, but have not yet re-analyzed. * = Used S-Plus Default GAM, and have re-analyzed results; GAM = Generalized Additive Model, GEE = Generalized Estimation Equations, GLM = Generalized Linear Model.

TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in μg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Dominici et al. (2000a). +20 largest U.S. cities. 1987-1994. PM_{10} mean ranged from 23.8 μg/m^3 (San Antonio) to 52.0 μg/m^3 (Riverside).	Non-accidental total deaths (stratified in three age groups: <65, 65-75, 75+) were examined for their associations with PM_{10} and O_3 (single, 2, and 3 pollutant models) at lags 0, 1, and 2 days. In the first stage of the hierarchical model, RRs for PM_{10} and O_3 for each city were obtained using GAM Poisson regression models, adjusting for temperature and dewpoint (0-day and average of 1-3 days for both variables), day-of-week, seasonal cycles, intercept and seasonal cycles for three age groups. In the second stage, between-city variation in RRs were modeled as a function of city-specific covariates including mean PM_{10} and O_3 levels, percent poverty, and percent of population with age 65 and over. The prior distribution assumed heterogeneity across cities. To approximate the posterior distribution, a Markov Chain Monte Carlo (MCMC) algorithm with a block Gibbs sampler was implemented. The second stage also considered spatial model, in which RRs in closer cities were assumed to be more correlated.	Lag 1 day PM_{10} concentration positively associated with total mortality in most locations (only 2 out of 20 coefficients negative), though estimates ranged from 2.1% to –0.4% per 10 μg/m^3 PM_{10} increase. PM_{10} mortality associations changed little with the addition of O_3 to the model, or with the addition of a third pollutant in the model. The pattern of PM_{10} effects with respiratory and cardiovascular were similar to that of total mortality. The PM_{10} effect was smaller (and weaker) with other causes of deaths. The pooled analysis of 20 cities data confirmed the overall effect on total and cardiorespiratory mortality, with lag 1 day showing largest effect estimates. The posterior distributions for PM_{10} were generally not influenced by addition of other pollutants. In the data for which the distributed lags could be examined (i.e., nearly daily data), the sum of 7-day distributed lag coefficients was greater than each of single day coefficients. City-specific covariates did not predict the heterogeneity across cities. Regional model results suggested that PM_{10} effects in West U.S. were larger than in East and South.	Total mortality excess deaths per 50 μg/m^3 increase in PM_{10}: 1.8 (-0.5, 4.1) for lag 0; 1.9 (-0.4, 4.3) for lag 1; 1.2 (-1.0, 3.4) for lag 2. Cardiovascular disease excess deaths per 50 μg/m^3 PM_{10}: 3.4 (1.0, 5.9).
Daniels et al. (2000).* The largest U.S. 20 cities, 1987-1994.	This study examined the shape of concentration-response curve. Three log-linear GAM regression models were compared: (1) using a linear PM_{10} term; (2) using a natural cubic spline of PM_{10} with knots at 30 and 60 μg/m^3 (corresponding approximately to 25 and 75 percentile of the distribution); and, (3) using a threshold model with a grid search in the range between 5 and 200 μg/m^3 with 5 μg/m^3 increment. Covariates included the smoothing function of time, temperature and dewpoint, and day-of-week indicators. These models were fit for each city separately, and for model (1) and (2) the combined estimates across cities were obtained by using inverse variance weighting if there was no heterogeneity across cities, or by using a two-level hierarchical model if there was heterogeneity.	For total and cardiorespiratory mortality, the spline curves were roughly linear, consistent with the lack of a threshold. For mortality from other causes, however, the curve did not increase until PM_{10} concentrations exceeded 50 μg/m^3. The hypothesis of linearity was examined by comparing the AIC values across models. The results suggested that the linear model was preferred over the spline and the threshold models.	
Dominici et al. (2003). Re-analysis of above study.	Re-analysis of above model using GLM/natural splines.	The shapes of concentration-response curves were similar to the original analysis.	

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in μg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Klemm et al. (2000). Replication study of the Harvard Six Cities time-series analysis by Schwartz et al. (1996).	Reconstruction and replication study of the Harvard Six Cities time-series study. The original investigators provided PM data; Klemm et al. reconstructed daily mortality and weather data from public records. Data analytical design (GAM Poisson model) was the same as that from the original study.	The combined PM effect estimates were essentially equivalent to the original results.	Total mortality percent excess risks: $PM_{10/15}$: 4.1(2.8, 5.4) per 50μg/m^3 $PM_{2.5}$: 3.3(2.3, 4.3) per 25 μg/m^3 $PM_{10-2.5}$: 1.0(-0.4, 2.4) per 25 μg/m^3
Klemm and Mason (2003). Re-analysis of the above study.	Re-analysis of the above study using GAM with stringent convergence criteria and GLM/natural splines. Sensitivity of results to alternative degrees of freedom were also examined.	When GAM with stringent convergence criteria were applied, PM effect estimates were reduced by 10 to 15%. GLM/natural splines, and increasing the degrees of freedom for temporal trends resulted in further reductions in PM coefficients.	Total mortality percent excess risks using GAM stringent convergence criteria: $PM_{10/15}$: 3.5(2.0, 5.1) per 50μg/m^3 $PM_{2.5}$: 3.0(2.1, 4.0) per 25 μg/m^3 $PM_{10-2.5}$: 0.8(-0.5, 2.0) per 25 μg/m^3 Using GLM/natural splines: $PM_{10/15}$: 2.0(0.3, 3.8) per 50μg/m^3 $PM_{2.5}$: 2.0(0.9, 3.2) per 25 μg/m^3 $PM_{10-2.5}$: 0.3(-1.2, 1.8) per 25 μg/m
Schwartz (2003a). Re-analysis of the Harvard Six Cities time-series analysis.	$PM_{2.5}$ data were re-analyzed using GAM with stringent convergence criteria, GLM/natural splines, B-splines, penalized splines, and thin-plate splines.	When GAM with stringent convergence criteria were applied, $PM_{2.5}$ effect estimates were reduced by ~5%. GLM/natural splines, B-splines, penalized splines, and thin-plate splines each resulted in further reductions in $PM_{2.5}$ excess risk estimates.	Total mortality percent excess risks using per 25 μg/m^3 $PM_{2.5}$: GAM (default):3.7(2.7, 4.7) GAM (stringent): 3.5(2.5, 4.5) Natural splines: 3.3(2.2, 4.3) B-splines: 3.0(2.0, 4.0) Penalized splines: 2.9(1.8, 4.) Thin-plate splines: 2.6(1.5, 3.8)
Zeger et al. (1999). Philadelphia, 1974-1988.	The implication of harvesting for PM regression coefficients, as observed in frequency domain, was illustrated using simulation. Three levels of harvesting, 3 days, 30 days, and 300 days were simulated. Real data from Philadelphia was then analyzed.	In the simulation results, as expected, the shorter the harvesting, the larger the PM coefficient in the higher frequency range. However, in the Philadelphia data, the regression coefficients increased toward the lower frequency range, suggesting that the extent of harvesting, if it exists, is not in the short-term range.	
Dominici et al. (2003). Re-analysis of above study.	Re-analysis of above model using GLM/natural splines.	Results were essentially unchanged.	

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in $\mu g/m^3$.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Braga et al. (2000). +Five U.S. cities: Pittsburgh, PA; Detroit, MI; Chicago, IL; Minneapolis-St. Paul, MN; Seattle, WA. 1986-1993. PM_{10} means were 35, 37, 37, 28, and 33 $\mu g/m^3$, respectively in these cities.	Potential confounding caused by respiratory epidemics on PM-total mortality associations was investigated in a subset of the 10 cities evaluated by Schwartz (2000a,b), as summarized below. GAM Poisson models were used to estimate city-specific PM_{10} effects, adjusting for temperature, dewpoint, barometric pressure, time-trend and day-of-week. A cubic polynomial was used to for each epidemic period, and a dummy variable was used to control for isolated epidemic days. Average of 0 and 1 day lags were used.	When respiratory epidemics were adjusted for, small decreases in the PM_{10} effect were observed (9% in Chicago, 11% in Detroit, 3% in Minneapolis, 5% in Pittsburgh, and 15% in Seattle).	The overall estimated percent excess deaths per 50 $\mu g/m^3$ increase in PM_{10} was 4.3% (3.0, 5.6) before controlling for epidemics and 4.0% (2.6, 5.3) after. Average of 0 and 1 day lags.
Braga et al. (2001).* Ten U.S. cities. Same as Schwartz (2000b).	The study examined the lag structure of PM_{10} effects on respiratory and cardiovascular cause-specific mortality. Using GAM Poisson model adjusting for temporal pattern and weather, three types of lag structures were examined: (1) 7-day unconstrained distributed lags; (2) 2-day average (0- and 1-day lag); and (3) 0-day lag. The results were combined across 10 cities.	The authors reported that respiratory deaths were more affected by air pollution levels on the previous days, whereas cardiovascular deaths were more affected by same-day pollution. Pneumonia, COPD, all cardiovascular disease, and myocardial infarction were all associated with PM_{10} in the three types of lags examined. The 7-day unconstrained lag model did not always give larger effect size estimates compared others.	In the 7-day unconstrained distributed lag model, the estimated percent excess deaths per 50 $\mu g/m^3$ PM_{10} were 14.2%(7.8, 21.1) , 8.8%(0.6, 17.7), 5.1%(3.0, 7.2), and 3.0%(0.0, 6.2) for pneumonia, COPD, all cardiovascular, and myocardial infarction mortality, respectively.
Schwartz (2003b). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria as well as penalized splines.	Small changes in PM risk estimates. Original findings unchanged.	Above estimates using stringent convergence criteria were: 16.5%(8.3, 25.3), 9.9%(0.6, 20.0), 5.1%(2.8, 7.5), and 3.5%(-0.7, 8.0). Corresponding numbers for penalized splines were: 11.5%(3.1, 20.6), 7.2%(-2.6, 18.0), 4.6%(2.0, 7.2), and 2.5%(-2.2, 7.5).

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United States (cont'd)			
Schwartz (2000a).* Ten U.S. cities: New Haven, CT; Pittsburgh, PA; Detroit, MI; Birmingham, AL; Canton, OH; Chicago, IL; Minneapolis-St. Paul, MN; Colorado Springs, CO; Spokane, WA; and Seattle, WA. 1986-1993. PM_{10} means were 29, 35, 36, 37, 29, 37, 28, 27, 41, and 33, respectively in these cities.	Daily total (non-accidental) deaths (20, 19, 63, 60, 10, 133, 32, 6, 9, and 29, respectively in these cities in the order shown left). Deaths stratified by location of death (in or outside hospital) were also examined. For each city, a GAM Poisson model adjusting for temperature, dewpoint, barometric pressure, day-of-week, season, and time was fitted. The data were also analyzed by season (November through April as heating season). In the second stage, the PM_{10} coefficients were modeled as a function of city-dependent covariates including copollutant to PM_{10} regression coefficient (to test confounding), unemployment rate, education, poverty level, and percent non-white. Threshold effects were also examined. The inverse variance weighted averages of the ten cities' estimates were used to combine results.	PM_{10} was significantly associated with total deaths, and the effect size estimates were the same in summer and winter. Adjusting for other pollutants did not substantially change PM_{10} effect size estimates. Also, socioeconomic variables did not modify the estimates. The effect size estimate for the deaths that occurred outside hospitals was substantially greater than that for inside hospitals. The effect size estimate was larger for subset with PM_{10} less than 50 g/m^3.	The total mortality RR estimates combined across cities per 50 µg/m^3 increase of mean of lag 0- and 1-days PM_{10}: overall 3.4 (2.7, 4.1); summer 3.4 (2.4, 4.4); winter 3.3 (2.3, 4.4); in-hospital 2.5 (1.5, 3.4); out-of-hospital 4.5 (3.4, 5.6); days < 50 µg/m^3 4.4 (3.1, 5.7); with SO_2 2.9 (1.2, 4.6); with CO 4.6 (3.2, 6.0); with O_3 3.5 (1.6, 5.3).
Schwartz (2003b). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria as well as natural splines. The case for in vs. out of hospital deaths and days PM_{10} < 50 µg/m^3 were not re-analyzed.		The total mortality RR estimates combined across cities per 50 µg/m^3 increase of mean of lag 0- and 1-days PM_{10}: overall 3.3 (2.6, 4.1); summer 3.4 (2.5, 4.4); winter 3.1 (2.0, 4.1); with SO_2 3.2 (1.7, 4.8); with CO 4.5 (2.7, 6.4); with O_3 3.5 (2.2, 4.8). Corresponding values for natural splines are: overall 2.8 (2.0, 3.6); summer 2.6 (1.6, 3.7); winter 2.9 (1.8, 4.1); with SO_2 2.8 (1.0, 4.6); with CO 3.7 (1.6, 5.8); with O_3 3.0 (1.6, 4.4).

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

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United States (cont'd)			
Schwartz (2000b).* Ten U.S. cities: New Haven, CT; Pittsburgh, PA; Birmingham, AL; Detroit, MI; Canton, OH; Chicago, IL; Minneapolis-St. Paul, MN; Colorado Springs, CO; Spokane, WA; and Seattle, WA. 1986-1993. PM_{10} means were 29, 35, 36, 37, 29, 37, 28, 27, 41, and 33, respectively in these cities.	The issue of distributed lag effects was the focus of this study. Daily total (non-accidental) deaths of persons 65 years of age and older were analyzed. For each city, a GAM Poisson model adjusting for temperature, dewpoint, barometric pressure, day-of-week, season, and time was fitted. Effects of distributed lag were examined using four models: (1) 1-day mean at lag 0 day; (2) 2-day mean at lag 0 and 1 day; (3) second-degree distributed lag model using lags 0 through 5 days; (4) unconstrained distributed lag model using lags 0 through 5 days. The inverse variance weighted averages of the ten cities' estimates were used to combine results.	The effect size estimates for the quadratic distributed model and unconstrained distributed lag model were similar. Both distributed lag models resulted in substantially larger effect size estimates than the single day lag, and moderately larger effect size estimates than the two-day average models.	Total mortality percent increase estimates combined across cities per 50 μg/m^3 increase in PM_{10}: 3.3 (2.5, 4.1) for 1-day mean at lag 0; 5.4 (4.4, 6.3) 2-day mean of lag 0 and 1; 7.3 (5.9, 8.6) for quadratic distributed lag; and 6.6 (5.3, 8.0) for unconstrained distributed lag.
Schwartz (2003b). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria as well as penalized splines. Only quadratic distributed lag and unconstrained distributed lag models were re-analyzed.	PM risk estimates were reduced but not substantially. Original findings unchanged.	Total mortality percent increase estimates combined across cities per 50 μg/m^3 increase in PM_{10}: 6.3 (4.9, 7.8) for quadratic distributed lag; and 5.8 (4.4, 7.3) for unconstrained distributed lag using stringent convergence criteria. Corresponding numbers for penalized splines were: 5.3%(4.2, 6.5) and 5.3%(3.9).
Schwartz and Zanobetti (2000). + Ten U.S. cities. Same as above.	The issue of a threshold in PM-mortality exposure-response curve was the focus of this study. First, a simulation was conducted to show that the "meta-smoothing" could produce unbiased exposure-response curves. Three hypothetical curves (linear, piecewise linear, and logarithmic curves) were used to generate mortality series in 10 cities, and GAM Poisson models were used to estimate exposure response curve. Effects of measurement errors were also simulated. In the analysis of actual 10 cities data, GAM Poisson models were fitted, adjusting for temperature, dewpoint, and barometric pressure, and day-of-week. Smooth function of PM_{10} with the same span (0.7) in each of the cities. The predicted values of the log relative risks were computed for 2 μg/m^3 increments between 5.5 μg/m^3 and 69.5 μg/m^3 of PM_{10} levels. Then, the predicted values were combined across cities using inverse-variance weighting.	The simulation results indicated that the "meta-smoothing" approach did not bias the underlying relationships for the linear and threshold models, but did result in a slight downward bias for the logarithmic model. Measurement error (additive or multiplicative) in the simulations did not cause upward bias in the relationship below threshold. The threshold detection in the simulation was not very sensitive to the choice of span in smoothing. In the analysis of real data from 10 cities, the combined curve did not show evidence of a threshold in the PM_{10}-mortality associations.	The combined exposure-response curve indicates that an increase of 50 μg/m^3 is associated with about a 4% increase in daily deaths. Avg. of 0 and 1 day lags.

+ = Used GAM with multiple non-parametric smooths, but have not yet re-analyzed. * = Used S-Plus Default GAM, and have re-analyzed results; GAM = Generalized Additive Model, GEE = Generalized Estimation Equations, GLM = Generalized Linear Model.

TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

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United States (cont'd)			
Zanobetti and Schwartz (2000).* Four U.S. cities: Chicago, IL; Detroit, MI; Minneapolis-St. Paul, MN; Pittsburgh, PA. 1986-1993. PM_{10} median = 33, 33, 25, and 31 respectively for these cities.	Separate daily counts of total non-accidental deaths, stratified by sex, race (black and white), and education (education > 12yrs or not), were examined to test hypothesis that people in each of these groups had higher risk of PM_{10}. GAM Poisson models adjusting for temperature, dewpoint, barometric pressure, day-of-week, season, and time were used. The mean of 0- and 1-day lag PM_{10} was used. The inverse variance weighted averages of the four cities' estimates were used to combine results.	The differences in the effect size estimates among the various strata were modest. The results suggest effect modification with the slope in female deaths one third larger than in male deaths. Potential interaction of these strata (e.g., black and female) were not investigated.	The total mortality RR estimates combined across cities per 50 μg/m^3 increase of mean of lag 0- and 1-days PM_{10}: white 5.0 (4.0, 6.0); black 3.9 (2.3, 5.4); male 3.8 (2.7, 4.9); female 5.5 (4.3, 6.7); education <12y 4.7 (3.3, 6.0); education > 12y 3.6 (1.0, 6.3).
Moolgavkar (2000a)* Cook County, Illinois Los Angeles County, CA Maricopa County, AZ 1987-1995 PM_{10}, CO, O_3, NO_2, SO_2 in all three locations. $PM_{2.5}$ in Los Angeles County. Cook Co: PM_{10} Median = 47 μg/m^3. Maricopa Co: PM_{10} Median = 41. Los Angeles Co: PM_{10} Median = 44; $PM_{2.5}$ Median = 22.	Associations between air pollution and time-series of daily deaths evaluated for three U.S. metropolitan areas with different pollutant mixes and climatic conditions. Daily total non-accidental deaths and deaths from cardiovascular disease (CVD), cerebrovascular (CrD), and chronic obstructive lung disease and associated conditions (COPD) were analyzed by generalized additive Poisson models in relation to 24-h readings for each of the air pollutants averaged over all monitors in each county. All models included an intercept term for day-of-week and a spline smoother for temporal trends. Effects of weather were first evaluated by regressing daily deaths (for each mortality endpoint) against temp and rel. humidity with lag times of 0 to 5 days. Then lags that minimized deviance for temp and rel. humidity were kept fixed for subsequent pollutant effect analyses. Each pollutant entered linearly into the regression and lags of between 0 to 5 days examined. Effects of two or more pollutants were then evaluated in multipollutant models. Sensitivity analyses were used to evaluate effect of degree of smoothing on results.	In general, the gases, especially CO (but not O_3) were much more strongly associated with mortality than PM. Specified pattern of results found for each county were as follows. For Cook Co., in single pollutant analyses PM_{10}, CO, and O_3 were all associated (PM_{10} most strongly on lag 0-2 days) with total mortality, as were SO_2 and NO_2 (strongest association on lag 1 day for the latter two). In joint analyses with one of gases, the coefficients for both PM_{10} and the gas were somewhat attenuated, but remained stat. sig. for some lags. With 3-pollutant models, PM_{10} coefficient became small and non-sig. (except at lag 0), whereas the gases dominated. For Los Angeles, PM_{10}, $PM_{2.5}$, CO, NO_2, and SO_2, (but not O_3), were all associated with total mortality. In joint analyses with CO or SO_2 and either PM_{10} or $PM_{2.5}$, PM metrics were markedly reduced and non-sig., whereas estimates for gases remained robust. In Maricopa Co. single-pollutant analyses, PM_{10} and each of the gases, (except O_3), were associated with total morality; in 2-pollutant models, coefficients for CO, NO_2, SO_2, were more robust than for PM_{10}. Analogous patterns of more robust gaseous pollutant effects were generally found for cause-specific (CVD, CrD, COPD) mortality analyses. Author concluded that while direct effect of individual components of air pollution cannot be ruled out, individual components best thought of as indices of overall pollutant mix.	In single pollutant models, estimated daily total mortality % excess deaths per 50 μg/m^3 PM_{10} was mainly in range of: 0.5-1.0% lags 0-2 Cook Co.; 0.25-1.0% lags 0-2 LA; 2.0% lag 2 Maricopa. Percent per 25 μg/m^3 $PM_{2.5}$ 0.5% lags 0, 1 for Los Angeles. Maximum estimated COPD % excess deaths (95% CI) per 50 μg/m^3 PM_{10}: Cook Co. 5.4 (0.3,10.7), lag 2; with O_3, 3.0 (-1.8, 8.1) lag 2; LA 5.9 (-1.6, 14.0) lag 1; Maricopa 8.2 (-4.2, 22.3) lag 1; per 25 μg/m^3 $PM_{2.5}$ in LA 2.7 (-3.4, 9.1). CVD % per 50 μg/m^3 PM_{10}: Cook 2.2 (0.4, 4.1) lag 3; with O_3, SO_2 1.99 (-0.06, 4.1) lag 3; LA 4.5 (1.7, 7.4) lag 2; with CO -0.56 (-3.8, 2.8) lag 2; Maricopa 8.9 (2.7, 15.4) lag 1; with NO_2 7.4 (-0.95, 16.3) lag 1. Percent per 25 μg/m^3 $PM_{2.5}$, LA 2.6 (0.4, 4.9) lag1; with CO 0.60 (-2.1, 3.4). CrD % per 50 μg/m^3 PM_{10}: Cook 3.3 (-0.12, 6.8) lag 2; LA 2.9 (-2.3, 8.4) lag 3; Maricopa 11.1 (0.54, 22.8) lag 5. Percent per 25 μg/m^3 $PM_{2.5}$, LA 3.6 (-0.6, 7.9) lag 3.

+ = Used GAM with multiple non-parametric smooths, but have not yet re-analyzed. * = Used S-Plus Default GAM, and have re-analyzed results; GAM = Generalized Additive Model, GEE = Generalized Estimation Equations, GLM = Generalized Linear Model.

TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Moolgavkar (2003). Re-analysis of above study, but Maricopa Co. data were not analyzed.	Re-analysis of above study using stringent convergence criteria as well as natural splines. Cerebrovascular deaths data were not analyzed. Ozone was not analyzed. In addition to the 30 degrees of freedom used for smoothing splines for temporal trends in the original analysis, results for 100 degrees of freedom were also presented. Two-pollutant model results were not reported for Cook county.	The sensitivity of results to the degrees of freedom was often greater than that to the GAM convergence criteria. The main conclusion of the original study remained the same.	Maximum estimated non-accidental deaths % excess deaths (95% CI) per 50 µg/m^3 PM_{10}: Cook Co. 2.4 (1.3,3.5), lag 0; LA 2.4 (0.5, 4.4) lag2; with CO, -1.6(-3.7, 0.6); per 25 µg/m^3 $PM_{2.5}$ in LA 1.5 (0, 3.0). Maximum estimated COPD % excess deaths (95% CI) per 50 µg/m^3 PM_{10}: Cook Co. 5.5 (0.3,11.0), lag 2; LA 4.4 (-3.1, 12.6) lag 1; per 25 µg/m^3 $PM_{2.5}$ in LA 1.9 (-10.0, 15.4). CVD % per 50 µg/m^3 PM_{10}: Cook 2.2 (0.3, 4.1) lag 3; LA 4.5 (1.6, 7.5) lag 2; Percent per 25 µg/m^3 $PM_{2.5}$, LA 2.6 (0.4, 4.9) lag1. All the estimates above are for 30 degrees of freedom cases.
Ostro et al. (1999a).+ Coachella Valley, CA. 1989-1992. PM_{10} (beta-attenuation) Mean = 56.8 µg/m^3.	Study evaluated total, respiratory, cardiovascular, non-cardiorespiratory and age >50 yr deaths (mean = 5.4, 0.6, 1.8, 3.0, and 4.8 per day, respectively). The valley is a desert area where 50-60% of PM_{10} estimated to be coarse particles. Correlation between gravimetric and beta-attenuation, separated by 25 miles, was high (r = 0.93). Beta-attenuation data were used for analysis. GAM Poisson models adjusting for temperature, humidity, day-of-week, season, and time were used. Seasonally stratified analyses were also conducted. Lags 0-3 days (separately) of PM_{10} along with moving averages of 3 and 5 days examined, as were O_3, NO_2, and CO.	Associations were found between 2- or 3-day lagged PM_{10} and all mortality categories examined, except non-cardiorespiratory series. The effect size estimates for total and cardiovascular deaths were larger for warm season (May through October) than for all year period. NO_2 and CO were significant predictor of mortality in single pollutant models, but in multi-pollutant models, none of the gaseous pollutants were significant (coefficients reduced), whereas PM_{10} coefficients remained the same and significant.	Total mortality percent excess deaths per 50 µg/m^3 PM_{10} at 2-day lag = 4.6 (0.6, 8.8). Cardiac deaths: 8.33 (2.14, 14.9) Respiratory deaths: 13.9 (3.25, 25.6)

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in μg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Ostro et al. (2000).* Coachella Valley, CA. 1989-1998. $PM_{2.5}$ = 16.8; $PM_{10-2.5}$ = 25.8 in Indio; $PM_{2.5}$ = 12.7; $PM_{10-2.5}$ = 17.9 in Palm Springs.	A follow-up study of the Coachella Valley data, with $PM_{2.5}$ and $PM_{10-2.5}$ data in the last 2.5 years. Both $PM_{2.5}$ and $PM_{10-2.5}$ were estimated for the remaining years to increase power of analyses. However, only $PM_{10-2.5}$ could be reliably estimated. Therefore, predicted $PM_{2.5}$ data were not used for mortality analysis. Thus, the incomparable sample size make it difficult to directly assess the relative importance of $PM_{2.5}$ and $PM_{10-2.5}$ in this data set.	Several pollutants were associated with all-cause mortality, including $PM_{2.5}$, CO, and NO_2. More consistent results were found for cardiovascular mortality, for which significant associations were found for $PM_{10-2.5}$ and PM_{10}, but not $PM_{2.5}$ (possibly due to low range of $PM_{2.5}$ concentrations and reduced sample size for $PM_{2.5}$ data).	Total percent excess deaths: PM_{10} (lag 0 or 2) = 2.0 (-1.0, 5.1) per 50 μg/m^3 $PM_{2.5}$ (lag 4) = 11.5 (0.2, 24.1) per 25 μg/m^3 $PM_{10-2.5}$ (lag 0 or 2) = 1.3 (-0.6, 3.5) per 25 μg/m^3 Cardio deaths: PM_{10} (lag 0) = 6.1 (2.0, 10.3) per 50 μg/m^3 $PM_{2.5}$ (lag 4) = 8.6 (-6.4, 25.8) per 25 μg/m^3 $PM_{10-2.5}$ (lag 0) = 2.6 (0.7, 4.5) per 25 μg/m^3 Respiratory deaths: PM_{10} (lag 3) = -2.0 (-11.4, 8.4) per 50 μg/m^3 $PM_{2.5}$ (lag 1) = 13.3 (-43.1, 32.1) per 25 μg/m^3 $PM_{10-2.5}$ (lag 3) = -1.3 (-6.2, 4.0) per 25 μg/m^3
Ostro et al. (2003). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria as well as natural splines. Only cardiovascular mortality data were analyzed. Additional sensitivity analyses were conducted.	The PM risk estimates were slightly reduced with stringent convergence criteria and GLM. Sensitivity analysis showed that results were not sensitive to alternative degrees of freedom for temporal trends and temperature. Multi-day averages for PM increased risk estimates.	Cardio deaths (GAM with stringent convergence criteria): PM_{10} (lag 0) = 5.5 (1.6, 9.5) per 50 μg/m^3 $PM_{2.5}$ (lag 4) = 10.2 (-5.3, 28.3) per 25 μg/m^3 $PM_{10-2.5}$ (lag 0) = 2.9 (0.7, 5.2) per 25 μg/m^3 Cardio deaths (GLM/natural splines): PM_{10} (lag 0) = 5.1 (1.2, 9.1) per 50 μg/m^3 $PM_{2.5}$ (only 0-2 day lags reported) $PM_{10-2.5}$ (lag 0) = 2.7 (0.5, 5.1) per 25 μg/m^3

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in $\mu g/m^3$.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Fairley (1999).* Santa Clara County, CA 1989-1996. $PM_{2.5}$ (13); PM_{10} (34); $PM_{10-2.5}$ (11); COH (0.5 unit); NO_3 (3.0); SO_4 (1.8)	Total, cardiovascular, and respiratory deaths were regressed on PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, COH, nitrate, sulfate, O_3, CO, NO_2, adjusting for trend, season, and min and max temperature, using Poisson GAM model. Season-specific analysis was also conducted. The same approach was also used to re-analyze 1980-1986 data (previously analyzed by Fairley, 1990).	$PM_{2.5}$ and nitrate were most significantly associated with mortality, but all the pollutants (except $PM_{10-2.5}$) were significantly associated in single poll. models. In 2 and 4 poll. models with $PM_{2.5}$ or nitrate, other pollutants were not significant. The RRs for respiratory deaths were always larger than those for total or cardiovascular deaths. The difference in risk between season was not significant for $PM_{2.5}$. The 1980-1986 results were similar, except that COH was very significantly associated with mortality.	Total mortality per 25 $\mu g/m^3$ $PM_{2.5}$ at 0 d lag: 8% in one pollutant model; 9-12% in 2 pollutant model except with NO_3(~0) . Also, 8% per 50 $\mu g/m^3$ PM_{10} in one pollutant model and 2% per 25 $\mu g/m^3$ $PM_{10-2.5}$. Cardiovascular mortality: PM_{10} = 9% per 50 $\mu g/m^3$ $PM_{2.5}$ = 13% per 25 $\mu g/m^3$ $PM_{10-2.5}$ = 3% per 25 $\mu g/m^3$ Respiratory mortality: PM_{10} = 11% per 50 $\mu g/m^3$ $PM_{2.5}$ = 7% per 25 $\mu g/m^3$ $PM_{10-2.5}$ = 16% per 25 $\mu g/m^3$
Fairley (2003). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria as well as natural splines.	PM coefficients were either unchanged, slightly decreased, or slightly increased. Original findings, including the pattern in two-pollutant models unchanged.	Percent excess mortality for GAM (stringent) and GLM/natural splines, respectively per 50 $\mu g/m^3$ for PM_{10} and 25 $\mu g/m^3$ for $PM_{2.5}$ and $PM_{10-2.5}$. Total mortality: PM_{10} =7.8(2.8,13.1); 8.3(2.9, 13.9) $PM_{2.5}$ = 8.2(1.6, 15.2); 7.1(1.4, 13.1) $PM_{10-2.5}$ = 4.5(-7.6, 18.1); 3.3(-5.3, 12.7) Cardiovascular mortality: PM_{10} = 8.5(0.6, 17.0); 8.9(1.3, 17.0) $PM_{2.5}$ = 6.4(-4.1, 18.1); 6.8(-2.5, 16.9) $PM_{10-2.5}$ = 5.1(-13.4, 27.4); (no GLM) Respiratory mortality: PM_{10} = 10.7(-3.7, 27.2); 10.8(-3.4, 27.1) $PM_{2.5}$ = 11.8(-9.9, 38.7); 13.6(-3.7, 34.1) $PM_{10-2.5}$ = 32.2(-12.1, 98.6); (no GLM)

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Schwartz et al. (1999). Spokane, WA 1989-1995 PM_{10}: "control" days: 42 µg/m^3; dust-storm days: 263	Effects of high concentration of coarse crustal particles were investigated by comparing death counts on 17 dust storm episodes to those on non-episode days on the same day of the years in other years, adjusting for temperature, dewpoint, and day-of-week, using Poisson regression.	No association was found between the mortality and dust storm days on the same day or the following day.	0% (-4.5, 4.7) for dust storm days at 0 day lag (50 µg/m^3 PM_{10}) (lagged days also reported to have no associations).
Pope et al. (1999a). + Ogden, Salt Lake City, and Provo/Orem, UT 1985-1995 PM_{10} (32 for Ogden; 41 for SLC; 38 for P/O)	Associations between PM_{10} and total, cardiovascular, and respiratory deaths studied in three urban areas in Utah's Wasatch Front, using Poisson GAM model and adjusting for seasonality, temperature, humidity, and barometric pressure. Analysis was conducted with or without dust (crustal coarse particles) storm episodes, as identified on the high "clearing index" days, an index of air stagnation.	Salt Lake City (SLC), where past studies reported little PM_{10}-mortality associations, had substantially more dust storm episodes. When the dust storm days were screened out from analysis and PM_{10} data from multiple monitors were used, comparable RRs were estimated for SLC and Provo/Orem (P/O).	Ogden PM_{10} Total (0 d) = 12.0% (4.5, 20.1) CVD (0-4 d) = 1.4% (-8.3, 12.2) Resp. (0-4 d) = 23.8 (2.8, 49.1) SLC PM_{10} Total (0 d) = 2.3% (0.47) CVD (0-4 d) = 6.5% (2.2, 11.0) Resp. (0-4 d) = 8.2 (2.4, 15.2) Provo/Ovem PM_{10} Total (0 d) = 1.9% (-2.1, 6.0) CVD (0-4 d) = 8.6% (2.4, 15.2) Resp. (0-4 d) = 2.2% (-9.8, 15.9) Note: Above % for $PM_{2.5}$ and $PM_{10-2.5}$ all per 25 µg/m^3; all PM_{10} % per 50 µg/m^3.
Schwartz and Zanobetti (2000) +Chicago 1988-1993. PM_{10}. Median = 36 µg/m^3.	Total (non-accidental), in-hospital, out-of-hospital deaths (median = 132, 79, and 53 per day, respectively), as well as heart disease, COPD, and pneumonia elderly hospital admissions (115, 7, and 25 per day, respectively) were analyzed to investigate possible "harvesting" effect of PM_{10}. GAM Poisson models adjusting for temperature, relative humidity, day-of-week, and season were applied in baseline models using the average of the same day and previous day's PM_{10}. The seasonal and trend decomposition techniques called STL was applied to the health outcome and exposure data to decompose them into different time-scales (i.e, short-term to long-term), excluding the long, seasonal cycles (120 day window). The associations were examined with smoothing windows of 15, 30, 45, and 60 days.	The effect size estimate for deaths outside of the hospital is larger than for deaths inside the hospital. All cause mortality shows an increase in effect size at longer time scales. The effect size for deaths outside of hospital increases more steeply with increasing time scale than the effect size for deaths inside of hospitals.	Mortality RR estimates per 50 µg/m^3 increase of mean of lag 0- and 1-days PM_{10}: total deaths 4.5 (3.1, 6.0); in-hospital 3.9 (2.1, 5.8); out-of-hospital 6.3 (4.1, 8.6). For total deaths, the RR approximately doubles as the time scale changes from 15 days to 60 days. For out-of-hospital deaths, it triples from15 days to 60 days time scale.

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Lippmann et al. (2000).* Detroit, MI. 1992-1994. PM_{10} = 31; $PM_{2.5}$ = 18; $PM_{10-2.5}$ = 13.	For 1992-1994 study period, total (non-accidental), cardiovascular, respiratory, and other deaths were analyzed using GAM Poisson models, adjusting for season, temperature, and relative humidity. The air pollution variables analyzed were: PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, sulfate, H^+, O_3, SO_2, NO_2, and CO.	PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ were more significantly associated with mortality outcomes than sulfate or H^+. PM coefficients were generally not sensitive to inclusion of gaseous pollutants. PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ effect size estimates were comparable per same distributional increment (5^{th} to 95^{th} percentile).	Percent excess mortality per 50 µg/m^3 for PM_{10} and 25 µg/m^3 for $PM_{2.5}$ and $PM_{10-2.5}$: Total mortality: PM_{10} (1 d) = 4.4(-1.0,10.1) $PM_{2.5}$ (3 d) = 23.1(-0.6, 7.0) $PM_{10-2.5}$ (1 d) = 4.0(-1.2, 9.4)
For 1985-1990 period TSP, PM_{10}, TSP-PM_{10}, Sulfate from TSP (TSP-$SO_4^=$)	For earlier 1985-1990 study period, total non-accidental, circulatory, respiratory, and "other" (non-circulatory or respiratory non-accidental) mortality were evaluated versus noted PM indices and gaseous pollutants.	Both PM_{10} (lag 1 and 2 day) and TSP (lag 1 day) but not TSP-PM_{10} or TSP-$SO_4^=$ significantly associated with respiratory mortality for 1985-1990 period. The simultaneous inclusions of gaseous pollutants with PM_{10} or TSP reduced PM effect size by 0 to 34%. Effect size estimates for total, circulatory, and "other" categories were smaller than for respiratory mortality.	Circulatory mortality: PM_{10} (1 d) = 6.9(-1.3, 15.7) $PM_{2.5}$ (1 d) = 3.2 (-2.3, 8.9) $PM_{10-2.5}$ (1 d) = 7.8 (0, 16.2) Respiratory mortality: PM_{10} (0 d) = 7.8(-10.2, 29.5) $PM_{2.5}$ (0 d) = 2.3 (-10.3, 16.6) $PM_{10-2.5}$ (2 d) = 7.4(-9.1, 26.9)

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Ito (2003). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria as well as natural splines. Additional sensitivity analysis examined alternative weather models and influence of the degrees of freedom in a limited data sets.	PM coefficients were often reduced (but sometimes unchanged or increased) somewhat when GAM with stringent convergence criteria or GLM/natural splines were used. The reductions in coefficients were not differential across PM components; the original conclusion regarding the relative importance of PM components remained the same.	Percent excess mortality for GAM (stringent) and GLM/natural splines, respectively per 50 µg/m^3 for PM_{10} and 25 µg/m^3 for $PM_{2.5}$ and $PM_{10-2.5}$: Total mortality: PM_{10} (1 d) = 3.3(-2.0, 8.9); 3.1(-2.2, 8.7) $PM_{2.5}$ (3 d) = 1.9 (-1.8,5.7); 2.0(-1.7, 5.8) $PM_{10-2.5}$ (1 d) = 3.2(-1.9, 8.6); 2.8(-2.2, 8.1) Circulatory mortality: PM_{10} (1 d) = 5.4(-2.6, 14.0); 4.9(-3.0, 13.5) $PM_{2.5}$ (1 d) = 2.2 (-3.2, 7.9); 2.0(-3.4, 7.7) $PM_{10-2.5}$ (1 d) = 6.7 (-1.0, 15.0); 6.0(-1.6, 14.3) Respiratory mortality: PM_{10} (0 d) = 7.5(-10.5, 29.2); 7.9(-10.2, 29.7) $PM_{2.5}$ (0 d) = 2.3 (-10.4, 16.7); 3.1(-9.7, 17.7) $PM_{10-2.5}$ (2 d) = 7.0(-9.5, 26.5); 6.4(-10.0, 25.7)

+ = Used GAM with multiple non-parametric smooths, but have not yet re-analyzed. * = Used S-Plus Default GAM, and have re-analyzed results; GAM = Generalized Additive Model, GEE = Generalized Estimation Equations, GLM = Generalized Linear Model.

TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Chock et al. (2000). 1989-1991 Pittsburgh, PA PM_{10} (daily) $PM_{2.5}$ (every 2 days)	Study evaluated associations between daily mortality and several air pollution variables (PM_{10}, $PM_{2.5}$, CO, O_3, NO_2, SO_2) in two age groups (<75 yr., 75 yr.) in Pittsburgh, PA, during 3-yr. period. Poisson GLM regression used, including filtering of data based on cubic B-spline basis functionsas adjustments for seasonal trends. Day-of-week effects, temperature was modeled as a V-shape terms. Single- and multi-pollutant models run for 0, 1, 2, and 3 day lags. $PM_{2.5}/PM_{10}$ 0.67.	Issues of seasonal dependence of correlation among pollutants, multi-collinearity among pollutants, and instability of coefficients emphasized. Single- and multi-pollutant non-seasonal models show significant positive association between PM_{10} and daily mortality, but seasonal models showed much multi-collinearity, masking association of any pollutant with mortality. Also, based on data set half the size for PM_{10}, the $PM_{2.5}$ coefficients were highly unstable and, since no consistently significant associations found in this small data set stratified by age group and season, no conclusions drawn on relative role of $PM_{2.5}$ vs. $PM_{10-2.5}$.	Total mortality percent increase per 25 µg/m^3 for aged <75 yrs: $PM_{2.5}$ = 2.6% (2.0, 7.3) $PM1_{0-2.5}$ = 0.7% (-1.7, 3.7) Total mortality percent increase per 25 µg/m^3 for aged >75 yrs: $PM_{2.5}$ = 1.5% (-3.0, 6.3) $PM1_{0-2.5}$ = 1.3% (-1.3, 3.8)
Klemm and Mason (2000). Atlanta, GA 1998-1999 $PM_{2.5}$ mean=19.9; $PM_{2.5}/PM_{10}$ =0.65. Nitrate, EC, OC, and oxygenated HC.	Reported "interim" results for 1 yr period of observations regarding total mortality in Atlanta, GA during 1998-1999. Poisson GLM model with natural splines used to assess effects of $PM_{2.5}$ vs $PM_{10-2.5}$, and for nitrate, EC, OC and oxygenated HC components.	No significant associations were found for any of the pollutants examined, possibly due to a relatively short study period (1-year). The coefficient and t-ratio were larger for $PM_{2.5}$ than for $PM_{10-2.5}$.	Total mortality percent increase per 25 µg/m^3 for: $PM_{2.5}$ = 4.8% (-3.2, 13.4) $PM_{10-2.5}$ = 1.4% (-11.3, 15.9)
Gwynn et al. (2000). +Buffalo, N.Y. 1988-1990. PM_{10} (24); COH (0.2 /1000ft); SO_4 = (62 nmoles/m3)	Total, circulatory, and respiratory mortality and unscheduled hospital admissions were analyzed for their associations with H+, SO_4=, PM_{10}, COH, O_3, CO, SO_2, and NO_2, adjusting for seasonal cycles, day-of-week, temperature, humidity, using. Poisson and negative binomial GAM models.	For total mortality, all the PM components were significantly associated, with H+ being the most significant, and COH the least significant predictors. The gaseous pollutants were mostly weakly associated with total mortality.	12% (2.6, 22.7) per 50 µg/m^3 PM_{10} at 2-day lag.
Schwartz (2000c).* Boston, MA. 1979-1986. $PM_{2.5}$ mean = 15.6.	Non-accidental total, pneumonia, COPD, and ischemic heart disease mortality were examined for possible "harvesting" effects of PM. The mortality, air pollution, and weather time-series were separated into seasonal cycles (longer than 2-month period), midscale, and short-term fluctuations using STL algorithm. Four different midscale components were used (15, 30, 45, and 60 days) to examine the extent of harvesting. GAM Poisson regression analysis was performed using deaths, pollution, and weather for each of the four midscale periods.	For COPD deaths, the results suggest that most of the mortality was displaced by only a few months. For pneumonia, ischemic heart disease, and total mortality, the effect size increased with longer time scales.	Total mortality percent increase per 25 µg/m^3 increase in $PM_{2.5}$: 5.8(4.5, 7.2) for 15-day window fluctuations; 9.6 (8.2, 11.1) for the 60 day window.

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Schwartz (2003a). Re-analysis of above study.	Reanalysis of above study using GLM/natural splines.	PM risk estimates at different time scales changed only slightly (more often increased). Increase in standard error of PM coefficients was also small (<3%). Original findings unchanged.	Total mortality percent increase per 25 µg/m^3 increase in $PM_{2.5}$: 5.8 (4.5, 7.3) for 15-day window; 9.7 (8.2, 11.2) for the 60 day window.
Lipfert et al. (2000a). Philadelphia (7 county Metropolitan area), 1992-1995. Harvard PM measurements: $PM_{2.5}$ (17.3); PM_{10} (24.1); $PM_{10-2.5}$ (6.8), sulfate (53.1 nmol/m3); H^+ (8.0 nmol/m3).	12 mortality variables, as categorized by area, age, and cause, were regressed on 29 pollution variables (PM components, O_3, SO_2, NO_2, CO, and by sub-areas), yielding 348 regression results. Both dependent and explanatory variables were pre-filtered using the19-day-weighted average filter prior to OLS regression. Covariates were selected from filtered temperature (several lagged and averaged values), indicator variables for hot and cold days and day-of-week using stepwise procedure. The average of current and previous days' pollution levels were used.	Significant associations were found for a wide variety gaseous and particulate pollutants, especially for peak O_3. No systematic differences were seen according particle size or chemistry. Mortality for one part of the metropolitan area could be associated with air quality from another, not necessarily neighboring part.	The fractional Philadelphia mortality risk attributed to the pollutant levels: "average risk" was 0.0423 for 25 µg/m^3 $PM_{2.5}$; 0.0517 for 25 µg/m^3 $PM_{10-2.5}$; 0.0609 for 50 µg/m^3 PM_{10}, using the Harvard PM indices at avg. of 0 and 1 d lags.
Laden et. al. (2000)* Six Cities (means): Watertown, MA (16.5); Kingston-Harriman, TN (21.1); St. Louis, MO (19.2); Steubenville, OH (30.5); Portage, WI (11.3); Topeka, KS (12.2). 1979-1988?. 15 trace elements in the dichot $PM_{2.5}$: Si, S, Cl, K, Ca, V, Mn, Al, Ni, Zn, Se, Br, Pb, Cu, and Fe.	Total (non-accidental), ischemic heart disease, pneumonia, and COPD (mean daily total deaths for the six cities: 59, 12, 55, 3, 11, and 3, respectively in the order shown left). A factor analysis was conducted on the 15 elements in the fine fraction of dichot samplers to obtain five common factors; factors were rotated to maximize the projection of the single "tracer" element (as in part identified from the past studies conducted on these data) for each factor; $PM_{2.5}$ was regressed on the identified factors scores so that the factor scores could be expressed in the mass scale. Using GAM Poisson models adjusting for temperature, humidity, day-of-week, season, and time, mortality was regressed on the factor scores in the mass scale. The mean of the same-day and previous day (increasing the sample size from 6,211 to 9,108 days) mass values were used. The city-specific regression coefficients were combined using inverse variance weights.	Three sources of fine particles were defined in all six cities with a representative element for each source type: Si for soil and crustal material; Pb for motor vehicle exhaust; and Se for coal combustion sources. In city-specific analysis, additional sources (V for fuel oil combustion, Cl for salt, etc.) were considered. Five source factors were considered for each city, except Topeka with the three sources. Coal and mobile sources account for the majority of fine particles in each city. In all of the metropolitan areas combined, 46% of the total fine particle mass was attributed to coal combustion and 19% to mobile sources. The strongest increase in daily mortality was associated with the mobile source factor. The coal combustion factor was positively associated with mortality in all metropolitan areas, with the exception of Topeka. The crustal factor from the fine particles was not associated with mortality.	Percent excess total mortality per 25µg/m3 increase in PM2.5 from source types: Crustal: -5.6(-13.6, 3.1) Traffic: 8.9(4.2, 13.8) Coal: 2.8(0.8, 4.8) Residual oil: 6.3(0.4, 12.5)

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
United States (cont'd)			
Schwartz (2003a). Re-analysis of above study.	Re-analysis of above study using penalized splines.	The change in risk estimates for each source-apportioned $PM_{2.5}$ in each city were either positive or negative, but the combined estimates across cities increased for traffic factor and decreased for coal factor and residual oil factor.	Percent excess total mortality per 25µg/m3 increase in PM2.5 from source types: Crustal: -5.1(-13.9, 4.6) Traffic: 9.3(4.0, 14.9) Coal: 2.0(-0.3, 4.4) Residual oil: 5.9(-0.9, 13.2)
Levy (1998). King County, WA. 1990-1994. PM_{10} Nephelometer (30); (0.59 bsp unit)	Out-of-hospital deaths (total, respiratory, COPD, ischemic heart disease, heart failure, sudden cardiac death screening codes, and stroke) were related to PM_{10}, nephelometer (0.2 - 1.0 m fine particles), SO_2, and CO, adjusting for day-of-week, month of the year, temperature and dewpoint, using Poisson GLM regression.	Nephelometer data were not associated with mortality. Cause-specific death analyses suggest PM associations with ischemic heart disease deaths. Associations of mortality with SO_2 and CO not mentioned. Mean daily death counts were small (e.g., 7.7 for total; 1.6 for ischemic heart disease). This is an apparently preliminary analysis.	Total mortality percent excess: 5.6% (-2.4, 14.3) per 50 µg/m^3 PM_{10} at avg. of 2 to 4 d lag; 7.2% (-6.3, 22.8) with SO_2 CO. 1.8% (-3.5, 7.3) per 25 µg/m^3 PM_1; -1.0 (-8.7,. 7.7) with SO_2 and CO.
Mar et al. (2000).* Phoenix, AZ. 1995-1997. PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ (TEOM), with means = 46.5, 13.0, and 33.5, respectively; and $PM_{2.5}$ (DFPSS), mean = 12.0.	Total (non-accidental) and cardiovascular deaths (mean = 8.6 and 3.9, respectively) for only those who resided in the zip codes located near the air pollution monitor were included. GAM Poisson models were used, adjusting for season, temperature, and relative humidity. Air pollution variables evaluated included: O_3, SO_2, NO_2, CO, TEOM PM_{10}, TEOM $PM_{2.5}$, TEOM $PM_{10-2.5}$, DFPSS $PM_{2.5}$, S, Zn, Pb, soil, soil-corrected K (KS), nonsoil PM, OC, EC, and TC. Lags 0 to 4 days evaluated. Factor analysis also conducted on chemical components of DFPSS $PM_{2.5}$ (Al, Si, S, Ca, Fe, Zn, Mn, Pb, Br, KS, OC, and EC); and factor scores included in mortality regression.	Total mortality was significantly associated with CO and NO_2 and weakly associated with SO_2, PM_{10}, $PM_{10-2.5}$, and EC. Cardiovascular mortality was significantly associated with CO, NO_2, SO_2, $PM_{2.5}$, PM_{10}, $PM_{10-2.5}$, OC and EC. Combustion-related factors and secondary aerosol factors were also associated with cardiovascular mortality. Soil-related factors, as well as individual variables that are associated with soil were negatively associated with total mortality.	Total mortality percent excess: 5.4 (0.1, 11.1) for PM_{10} (TEOM) 50 µg/m^3 at lag 0 d; 3.0 (-0.5, 6.6) for $PM_{10-2.5}$ (TEOM) 25 µg/m^3 at lag 0 d; 3.0 (-0.7, 6.9) for $PM_{2.5}$ (TEOM) 25 µg/m^3 at lag 0 d. Cardiovascular mortality RRs: 9.9 (1.9, 18.4) for PM_{10} (TEOM) 50 µg/m^3 at lag 0 d; 18.7 (5.7, 33.2) for $PM_{2.5}$ (TEOM) 25 µg/m^3 at lag 1 d; and 6.4 (1.4, 11.7) PM_{10} (TEOM) 25 µg/m^3 $PM_{10-2.5}$ at lag 0 d.
Mar et al. (2003). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria as well as natural splines. Only cardiovascular mortality was re-analyzed.	Reductions on PM risk estimates for PM mass concentration indices in the GAM/stringent convergence criteria or GLM/natural splines were small. The change in coefficient for source factors varied: moderate reductions for motor vehicle factor, but slight increase for regional sulfate factor. EC and OC coefficients were also slightly reduced.	Percent excess cardiovascular mortality per 50 µg/m^3 PM_{10}; 25 µg/m^3 for $PM_{2.5}$ and $PM_{10-2.5}$: GAM with stringent convergence criteria and GLM/natural splines, respectively: PM_{10} (0 d): 9.7(1.7, 18.3); 9.5(0.6, 19.3) $PM_{2.5}$ (1 d): 18.0(4.9, 32.6); 19.1(3.9, 36.4) $PM_{10-2.5}$ (0 d): 6.4(1.3, 11.7); 6.2(0.8, 12.0)

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

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United States (cont'd)			
Clyde et al. (2000). Phoenix, AZ. 1995-1998. PM_{10}, and $PM_{2.5}$, (from TEOM), with means = 45.4, and 13.8. $PM_{10-2.5}$ computed as PM_{10}-$PM_{2.5}$.	Elderly (age 65 years) non-accidental mortality for three regions of increasing size in Phoenix urban area analyzed to evaluate influence of spatial uniformity of PM_{10} and $PM_{2.5}$. All-age accidental deaths for the metropolitan area also examined as a "control". GAM Poisson models adjusting for season (smoothing splines of days), and parametric terms for temperature, specific humidity, and lags 0- to 3-d of weather variables. PM indices for lags 0-3 d considered. Bayesian Model Averaging (BMA) produces posterior mean relative risks by weighting each model (out of all possible model specifications examined) based on support received from the data.	The BMA results suggest that a weak association was found only for the mortality variable defined over the region with uniform $PM_{2.5}$, with a 0.91 probability that RR is greater than 1. The other elderly mortality variables, including the accidental deaths ("control"), had such probabilities in the range between 0.46 to 0.77. Within the results for the mortality defined over the region with uniform $PM_{2.5}$, the results suggested that effect was primarily due to coarse particles rather than fine; only the lag 1 coarse PM was consistently included in the highly ranked models.	Posterior mean RRs and 90% probability intervals per changes of 25 µg/m^3 in all lags of fine and coarse PM for elderly mortality for uniform PM_{10} region: 1.06 (1+, 1.11).
Smith et al. (2000). Phoenix, AZ. 1995-1997	Study evaluated effects of daily and 2- to 5-day average coarse ($PM_{10-2.5}$) and fine ($PM_{2.5}$) particles from an EPA-operated central monitoring site on nonaccidental mortality among elderly (65+ years), using time-series analyses for residents within city of Phoenix and, separately, for region of circa 50 mi around Phoenix. Mortality was square-root transformed.Initial model selected to represent long-term trends (using B-splines) and weather variables (e.g., ave. daily temp., max daily temp., daily mean specific humidity, etc.); then PM variables added to model one at a time to ascertain which had strongest effect. Piecewise linear analysis and spline analysis used to evaluate possible nonlinear PM-mortality relationship and to evaluate threshold possibilities. Data analyzed most likely same as Clyde's or Mar's Phoenix data.	In linear PM effect model, a statistically significant mortality association found with $PM_{10-2.5}$, but not with $PM_{2.5}$. In the model allowing for a threshold, evidence suggestive of possible threshold for $PM_{2.5}$ (in the range of 20-25 µg/m^3) found, but not for $PM_{10-2.5}$. A seasonal interaction in the $PM_{10-2.5}$ effect was also reported: the effect being highest in spring and summer when anthropogenic concentration of $PM_{10-2.5}$ is lowest.	—

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

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United States (cont'd)			
Tsai et al. (2000). Newark, Elizabeth, and Camden, NJ. 1981-1983. PM_{15}: 55.5, 47.0, 47.5; and $PM_{2.5}$: 42.1, 37.1, 39.9, for Newark, Elizabeth, and Camden, respectively.	Factor analysis-derived source type components were examined for their associations with mortality in this study. Non-accidental total deaths and cardiorespiratory deaths were examined for their associations with PM_{15}, $PM_{2.5}$ sulfate, trace metals from PM_{15}, three fractions of extractable organic matter, and CO. Data were analyzed with Poisson GEE regression models with autoregressive correlation structure, adjusting for temperature, time-of-week, and season indicator variables. Individual pollution lag days from 0 to 3, as well as the average concentrations of current and preceding 3 days were considered. Factor analysis of the trace elements, sulfate, and CO data was conducted, and mortality series were regressed on these factor scores.	Factor analysis identified several source types with tracer elements. In Newark, oil burning factor, industrial source factor, and sulfate factor were positively associated with total mortality; and sulfate was associated with cardio-respiratory mortality. In Camden, oil burning and motor vehicle factors were positively associated with total mortality; and, oil burning, motor vehicles, and sulfate were associated with cardio-respiratory mortality. In Elizabeth, resuspended dust was not associated with total mortality; and industrial source (traced by Cd) showed positive associations with cardio-respiratory mortality. On the mass basis (source-contributed mass), the RRs estimates per 10 µg/m^3 were larger for specific sources (e.g., oil burning, industry, etc.) than for total mass. The choice of lag/averaging reported to be not important.	Percent excess deaths per 50 µg/m^3 increase in current day PM_{15}: in Newark, 5.7 (4.6, 6.7) for total mortality, 7.8 (3.6, 12.1) for cardioresp. mortality; in Camden, 11.1 (0.7, 22.5) and 15.0 (4.3, 26.9); and in Elizabeth, -4.9 (-17.9, 10.9) and 3.0 (-11.0, 19.4), respectively. Percent excess deaths per 25 µg/m^3 $PM_{2.5}$; in Newark, 4.3 (2.8, 5.9) for total and 5.1 (3.1, 7.2) for cardiorespiratory mortality; in Camden, 5.7 (0.1, 11.5) and 6.2 (0.6, 12.1); in Elizabeth, 1.8 (-5.4, 9.5) and 2.3 (-5.0, 10.1), respectively.
Gamble (1998). Dallas, TX. 1990-1994. PM_{10} (25)	Relationships of total, respiratory, cardiovascular, cancer, and remaining non-accidental deaths to PM_{10}, O_3, NO_2, SO_2, and CO evaluated, adjusting for temperature, dewpoint, day-of-week, and seasonal cycles (trigonometric terms) using Poisson GLM regression.	O_3 (avg. of 1-2 day lags), NO_2 (avg.. 4 -5 day lags), and CO (avg. of lags 5- 6 days) were significantly positively associated with total mortality. PM_{10} and SO_2 were not significantly associated with any deaths.	-3.6% (-12.7, 6.6) per 50 µg/m^3 PM_{10} at 0 lag (other lags also reported to have no associations)
Ostro (1995). San Bernardino and Riverside Counties, CA, 1980-1986. $PM_{2.5}$ (estimated from visual range). Mean = 32.5.	Study evaluated total, respiratory, cardiovascular, and age > = 65 deaths (mean = 40.7, 3.8, 18.7, and 36.4 per day, respectively). $PM_{2.5}$ estimated based on airport visual range and previously published empirical formula. Autoregressive OLS (for total) and Poisson (for sub-categories) regressions used, adjusting for season (sine/cosine with cycles from 1 yr to 0.75 mo; prefiltering with 15-day moving ave.; dichotomous variables for each year and month; smooth function of day and temp.), day-of-week, temp. and dewpoint. Evaluated lags 0, 1, and 2 of estimated $PM_{2.5}$, as well as moving averages of 2, 3, and 4 days and O_3.	The results were dependent on season. No $PM_{2.5}$ – mortality association found for the full year-round period. Associations between estimated $PM_{2.5}$ (same-day) and total and respiratory deaths found during summer quarters (April - Sept.). Correlation between the estimated $PM_{2.5}$ and daily max temp. was low (r = 0.08) during the summer quarters. Ozone was also associated with mortality, but was also relatively highly correlated with temp. r = 0.73). Moving averages of $PM_{2.5}$ did not improve the associations.	Percent excess deaths per 25 µg/m3 of estimated $PM_{2.5}$, lag 0: Full year: 0.3 (-0.6, 1.2) for total; 2.1 (-0.3, 4.5) for respiratory; and 0.7 (-0.3, 1.7) for circulatory. Summer quarters: 1.6 (0.03, 3.2) for total; 5.5 (1.1, 10.0) for respiratory; and 0 (-1.0, 1.0) for circulatory.
Kelsall et al. (1997). +Philadelphia, PA 1974-1988. TSP (67)	Total, cardiovascular, respiratory, and by-age mortality regressed on TSP, SO_2, NO_2, O_3, and CO, adjusting for temporal trends and weather, using Poisson GAM model.	TSP, SO_2, O_3, and 1-day lagged CO individually showed statistically significant associations with total mortality. No NO_2 associations unless SO_2 or TSP was also considered. The effects of TSP and SO_2 were diminished when both pollutants were included.	Total mortality excess risk: 3.2% (0, 6.1) per 100 µg/m^3 TSP at 0 day lag.

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

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United States (cont'd)			
Moolgavkar and Luebeck (1996). Philadelphia, PA. 1973-1988. TSP (68)	A critical review paper, with an analysis of total daily mortality for its association with TSP, SO_2, NO_2, and O_3, adjusting for temporal trends, temperature, and also conducting analysis by season, using Poisson GAM model. (Only one non-parametric smoothing terms in GAM models)	RR results presented as figures, and seasonal difference noted. TSP, SO_2, O_3 - mortality associations varied across season. TSP associations were stronger in summer and fall. NO_2 was the most significant predictor.	Total mortality excess risk: ranged 0 (winter) to 4% (summer) per 100 µg/m^3 TSP at 1 day lag.
Murray and Nelson (2000). Philadelphia, PA, 1973-1990.	Kalman filtering used to estimate hazard function in a state space model. The model framework, which assumes harvesting effect, allows estimation of at-risk population and the effect of changes in air quality on the life expectancy of the at-risk population. The model was first verified by simulation. Combinations of TSP, linear temperature, squared temperature, and interaction of TSP and temperature were considered in six models.	Both TSP and the product of TSP and average temperature are significant, but not together. The size of at-risk population estimated was about 500 people, with its life expectancy between 11.8 to 14.3 days, suggesting that the hazard causing agent making the difference of 2.5 days in the at-risk population.	The coefficients obtained in the models cannot be directly compared to the relative risk per µg/m^3 PM obtained in other time-series models.
Smith et al. (1999). Birmingham, AL 1985-1988; Chicago (Cook Co.), IL, 1986-1990. PM_{10} median = 45 µg/m^3 for Birmingham and 37.5 µg/m^3 for Chicago.	Study evaluated associations between lagged/averaged PM_{10} and non-accidental mortality in two cities. Mortality was square root-transformed in Birmingham data, and log-transformed in Chicago data. Seasonal cycles were modeled using B-splines. Temperature was modeled using piece-wise linear terms with a change point. PM_{10} data were included in the models at lag 0 through 3 and 3-day averages at these lags. Also, to examine the possible existence of a threshold, PM_{10} was modeled using a B-spline representation, and also using parametric threshold model, with the profile log likelihood evaluated at changing threshold points. In addition, the possibility of mortality displacement was examined with a model that attempts to estimate the frail population size through Bayesian techniques using Monte Carlo sampling.	The authors reported that, while significantly positive associations were found in both cities, the results were sensitive to the choice of lags. The PM_{10}-mortality associations were more stable in Chicago (perhaps in part due to sample size). The non-linear estimates of relative risk using B-splines suggest that an increasing effect above 80µg/m^3 for Birmingham, and above 100 µg/m^3 for Chicago. The threshold model through examination of log likelihood at various possible threshold levels also suggested similar change points, but not to the extent that could achieve statistical distinctions. The mortality displacement model in Chicago data suggested that the size of the frail population was very small (mean ~765), and the mean lifetime within the frail population short (< 10 days).	Birmingham: 4.8%(t=1.98) per 50µg/m^3 change in 1 through 3 day lag average of PM_{10}. Chicago: 3.7% (t=3.17) per 50µg/m^3 change in 0 through 2 day lag average of PM_{10}.

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United States (cont'd)			
Neas et. al. (1999). Philadelphia. 1973-1980. TSP mean = 77.2.	Total, age over 65, cancer, and cardiovascular deaths analyzed for association with TSP. Conditional logistic regression analysis with case-crossover design conducted. Average values of current and previous days' TSP used. Case period is the 48-hr period ending at midnight on day of death. Control periods are 7, 14, and 21 days before and after the case period. Other covariates included temperature on the previous day, dewpoint on the same day, an indicator for hot days (> 80°F), an indicator for humid days (dewpoint > 66°F), and interaction of same-day temp. and winter season.	In each set of the six control periods, TSP was associated with total mortality. A model with four symmetric reference periods 7 and 14 days around the case period produced a similar result. A model with only two symmetric reference periods of 7 days around the case produced a larger estimate. A larger effect was seen for deaths in persons 65 years of age and for deaths due to pneumonia and to cardiovascular disease. Cancer mortality was not associated with TSP.	Odds Ratio (OR) for all cause mortality per 100 µg/m^3 increase in 48-hr mean TSP was 1.056 (1.027, 1.086). The corresponding number for those aged 65 and over was 1.074 (1.037, 1.111), and 1.063 (1.021, 1.107) for cardiovascular disease.
Schwartz (2000d). +Philadelphia. 1974-1988. TSP. Mean = 70 µg/m^3 for warm season (April through August) and 64 µg/m^3 for cold season.	Total (non-accidental) deaths analyzed. GAM Poisson models adjusting for temperature, dewpoint, day-of-week, and season applied to each of 15 warm and cold seasons. Humidity-corrected extinction coefficient, derived from airport visual range, also considered as explanatory variable. In the second stage, resulting 30 coefficients were regressed on regression coefficients of TSP on SO_2. Results of first stage analysis combined using inverse variance weighting.	When TSP controlled for, no significant association between SO_2 and daily deaths. SO_2 had no association with daily mortality when it was poorly correlated with TSP. In contrast, when SO_2 was controlled for, TSP was more strongly associated with mortality than when it was less correlated with SO_2. However, all of the association between TSP and mortality was explained by its correlation with extinction coefficient.	Total mortality excess risk estimates combined across seasons/years: 9.0 (5.7, 12.5) per 100 µg/m^3 TSP.
Levy et al. (2000). Years vary from study to study ranging between 1973 to 1994. 21 published studies included U.S., Canadian, Mexican, European, Australian, and Chilean cities. PM_{10} levels in the 19 U.S. cities (in some cases TSP were converted to PM_{10} using factor of 0.55) ranged from ~20 to ~60 uµg/m^3.	To determine whether across-study heterogeneity of PM effects could be explained by regional parameters, Levy et al. applied an empirical Bayes meta-analysis to 29 PM estimates from 21 published studies. They considered such city-specific variables as mortality rate, gaseous pollutants regression coefficients, PM_{10} levels, central air conditioning prevalence, heating and cooling degreedays. Several of the studies included were those that used GAM with multiple non-parametric smoothing terms.	Among the city-specific variables, $PM_{2.5}/PM_{10}$ ratio was a significant predictor (larger PM estimates for higher $PM_{2.5}/PM_{10}$ ratios) in the 19 U.S. cities data subsets. While the sulfate data were not available for all the 19 cities, the investigators noted that, based on their analysis of the limited data with sulfate for 10 estimates, the sulfate/PM10 ratio was highly correlated with both the mortality (r = 0.84) and with the $PM_{2.5}/PM_{10}$ ratio (r = 0.70). This indicates that the sulfate/PM_{10} ratio may be even better predictor of regional heterogeneity of PM RR estimates.	The pooled estimate from 19 U.S. cities was 0.70% (0.54, 0.84) per 10 µg/m^3 increase in PM_{10}.

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Canada			
Burnett et al. (1998a).+ 11 Canadian cities. 1980-1991. No PM index data available on consistent daily basis.	Total non-accidental deaths were linked to gaseous air pollutants (NO_2, O_3, SO_2, and CO) using GAM Poisson models adjusting for seasonal cycles, day-of-week, and weather (selected from spline-smoothed functions of temperature, dewpoint, relative humidity with 0, 1, and 2 day lags using forward stepwise procedure). Pollution variables evaluated at 0, 1, 2, and up to 3-day lag averages thereof. No PM index included in analyses because daily PM measurements not available. City-specific models containing all four gaseous pollutants examined. Overall risks computed by averaging risks across cities.	NO_2 had 4.1% increased risk per mean concentration; O_3 had 1.8%; SO_2 had 1.4%, and CO had 0.9% in multiple pollutant regression models. A 0.4% reduction in excess mortality was attributed to achieving a sulfur content of gasoline of 30 ppm in five Canadian cities. Daily PM data for fine and coarse mass and sulfates available on varying (not daily) schedules allowed ecologic comparison of gaseous pollutant risks by mean fine particle indicators mass concentrations.	Found suggestion of weak negative confounding of NO_2 and SO_2 effects with fine particles and weak positive confounding of particle effects with O_3. No quantitative RR or ER estimates reported for PM indicators.
Burnett et al. (2000).* 8 largest Canadian cities. 1986-1996. All city mean PM_{10} 25.9; $PM_{2.5}$ 13.3; $PM_{10-2.5}$ 12.6; sulfate 2.6.	Total non-accidental deaths linked to PM indices (PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, sulfate, 47 elemental component concentrations for fine and coarse fractions) and gaseous air pollutants (NO_2, O_3, SO_2, and CO). Each city's mortality, pollution, and weather variables separately filtered for seasonal trends and day-of-week patterns. The residual series from all the cities then analyzed in a GAM Poisson model. The weather model was selected from spline-smoothed functions of temperature, relative humidity, and maximum change in barometric pressure within a day, with 0 and 1 day lags using forward stepwise procedure. Pollution effects were examined at lags 0 through 5 days. To avoid unstable parameter estimates in multi-pollutant models, principal components were also used as predictors in the regression models.	O_3 was weakly correlated with other pollutants and other pollutants were "moderately" correlated with each other (the highest was r = 0.65 for NO_2 and CO). The strongest association with mortality for all pollutants considered were for 0 or 1 day lags. $PM_{2.5}$ was a stronger predictor of mortality than $PM_{10-2.5}$. The estimated gaseous pollutant effects were generally reduced by inclusion of $PM_{2.5}$ or PM_{10}, but not $PM_{10-2.5}$. Sulfate, Fe, Ni, and Zn were most strongly associated with mortality. Total effect of these four components was greater than that for $PM_{2.5}$ mass alone.	Percentage increase in daily filtered non-accidental deaths associated with increases of 50 µg/m^3 PM_{10} and 25 µg/m^3 $PM_{2.5}$ or $PM_{10-2.5}$ at lag 1 day: 3.5 (1.0, 6.0) for PM_{10}; 3.0 (1.1, 5.0) for $PM_{2.5}$; and 1.8 (-0.7, 4.4) for $PM_{10-2.5}$. In the multiple pollutant model with $PM_{2.5}$, $PM_{10-2.5}$, and the 4 gaseous pollutants, 1.9 (0.6, 3.2) for $PM_{2.5}$; and 1.2 (-1.3, 3.8) for $PM_{10-2.5}$.
Burnett and Goldberg (2003). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria as well as natural splines. In the main model of the original analysis, both dependent and independent variables were pre-filtered, but in the re-analysis, co-adjustment (i.e., more common simultaneous regression) approach was used. Additional sensitivity analysis included alternative fitting criteria and changing the extent of smoothing for temporal trends. Only PM_{10}, $PM_{2.5}$ and $PM_{10-2.5}$ were analyzed. No multiple pollutant models.	In the GAM model (stringent convergence criteria), inclusion of day-of-week variable made moderate increase in PM coefficients (up to 30%). Alternative fitting criteria and degrees of freedom for temporal trends also changed PM coefficients. Generally, larger the degrees of freedom for temporal trends, smaller the PM coefficients. $PM_{10-2.5}$ were more sensitive to alternative models than $PM_{2.5}$.	Excess total mortality in the GLM/natural splines with knot/2months, and using AIC and White-noise test fitting criteria at 1-day lag: PM_{10}: 2.7(-0.1, 5.5) per 50 µg/m^3 $PM_{2.5}$: 2.2(0.1, 4.2) per 25 µg/m^3 $PM_{10-2.5}$: 1.8(-0.6, 4.4) per 25 µg/m^3

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Canada (cont'd)			
Burnett et al. (1998b). + Toronto, 1980-1994. TSP (60); COH (0.42); SO4= (9.2 µg/m^3); PM_{10} (30, estimated); $PM_{2.5}$ (18, estimated)	Total, cardiac, and other nonaccidental deaths (and by age groups) were regressed on TSP, COH, SO_4=, CO, NO_2, SO_2, O_3, estimated PM_{10} and $PM_{2.5}$ (based on the relationship between the existing every-6th-day data and SO_4=, TSP and COH), adjusting for seasonal cycles, day-of-week, temperature, and dewpoint using Poisson GAM model.	Essentially all pollutants were significant predictors of total deaths in single pollutant models, but in two pollutant models with CO, most pollutants' estimated RRs reduced (all PM indices remained significant). Based on results from the co-pollutant models and various stepwise regressions, authors noted that effects of the complex mixture of air pollutants could be almost completely explained by the levels of CO and TSP.	Total mortality percent excess: 2.3% (0.8, 3.8) per 100 µg/m^3 TSP; 3.5% (1..8, 5.3) per 50 µg/m^3 PM_{10}; 4.8% (3.3, 6.4) per 25 µg/m^3 $PM_{2.5}$. 0 day lag for TSP and PM_{10}; Avg. of 0 and 1 day for $PM_{2.5}$.
Goldberg et al. (2000)* Montreal, Quebec 1984-95 Mean TSP = 53.1 (14.6 - 211.1) µg/m^3 PM_{10} = 32.2 (6.5 - 120.5) µg/m^3 $PM_{2.5}$ = 3.3 (0.0 - 30.0) µg/m^3	Study aimed to shed light on population subgroups that my be susceptible to PM effects. Linked data on daily deaths with other health data from the Quebec Health Insurance Plan (QHIP) (physician visits, pharmaceutical R_x, etc.) to identify individuals with presenting health conditions. PM_{10} and $PM_{2.5}$ measured by dichotomous sampler 1 in 6 days until 1992 (2 stations), then daily through 1993. PM missing days interpolated from COH, ext. coefficient, sulfates. Used quasi likelihood estimation in GAM's to assess PM associations with total and cause-specific mortality; and, also, in subgroups by age and/or preexisting health conditions. Adjusted for CO, NO_2, NO, O_3 and SO_2 in 2-pollutant and all-pollutant models.	Significant associations found for all-cause (total non-accidental) and cause-specific (cancer, CAD, respiratory disease, diabetes) with PM measures. Results reported for $PM_{2.5}$, COH and sulfates. All three PM measures associated with increases in total, resp., and "other nonaccidental", and diabetes-related mortality. No PM associations found with digestive, accidental, renal or neurologic causes of death. Also, mainly in 65+ yr group, found consistent associations with increased total mortality among persons who had cancer, acute lower resp. diseases, any cardiovascular disease, chronic CAD and congestive heart failure (CHF).	Percent excess mortality per 25 µg/m^3 estimated $PM_{2.5}$: Total deaths (3 d ave.) = 4.4% (2.5, 6.3) CV deaths (3 d ave.) = 2.6% (-0.1, 5.5) Resp deaths (3 d ave.) = 16.0% (9.7, 22.8) Coronary artery (3 d ave.) = 3.4% (-0.2, 7.1) Diabetes (3 d ave.) = 15.7% (4.8, 27.9) Lower Resp Disease (3 d ave.) = 9.7% (4.5, 15.1) Airways disease (3 d ave.) = 2.7% (-0.9, 6.4) CHF (3 d ave.) = 8.2% (3.3, 13.4)
Goldberg et al. (2001b)* Montreal, Quebec. 1984-1993. Predicted $PM_{2.5}$ mean = 17.6. CoH (1000ft) mean = 0.24, sulfate mean = 3.3.	The investigators used the universal Quebec medicare system to obtain disease conditions prior to deaths, and the roles of these respiratory and cardiovascular conditions in the PM-mortality associations were examined. GAM Poisson model adjusting for temporal pattern and weather was used.	The PM-mortality associations were found for those who had acute lower respiratory diseases, chronic coronary diseases, and congestive heart failure. They did not find PM-mortality associations for those chronic upper respiratory diseases, airways disease, cerebrovascular diseases, acute coronary artery diseases, and hypertension. Adjusting for gaseous pollutants generally attenuated PM RR estimates, but the general pattern remained. Effects were larger in summer.	The percent excess deaths estimates for non-accidental deaths per IQR (average of 0-2 day lags) for CoH, predicted $PM_{2.5}$, and sulfate were: 1.98% (1.07, 2.90), 2.17% (1.26, 3.08), and 1.29% (0.68, 1.90), respectively.
Goldberg et al. (2001). Data same as above.	Cause-specific mortality (non-accidental, neoplasm, lung cancer, cardiovascular, coronary artery disease, diabetes, renal disease, and respiratory) series were examined for their associations with O_3, using GAM Poisson model adjusting for temporal pattern and weather. Results were also reported for models with adjustments for other pollutants (SO_2, CO, NO_2, CoH, etc.).	The effect of O_3 was generally higher in the warm season and among persons aged 65 years and over. O_3 showed positive and statistically significant associations with non-accidental cause, neoplasms, cardiovascular disease, and coronary artery disease. These associations were not reduced when the model adjusted for SO_2, CO, NO_2, CoH simultaneously (or when CoH was replaced with $PM_{2.5}$ or total sulfates).	PM RRs not reported.

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Canada (cont'd)			
Goldberg and Burnett (2003). Re-analysis of above studies by Goldberg et al.	Re-analysis of above study using stringent convergence criteria as well as natural splines. Cause-specific mortality was not re-analyzed; re-analysis was focused only on the sub-groups defined using the QHIP data that showed associations with particles in the original study. Sensitivity analyses included alternative weather models and using different degrees of freedom for temporal trends.	The PM coefficients were not very sensitive to the extent of temporal smoothing but were sensitive to the functional form of weather models. Most of the originally reported associations except for congestive heart failure were highly attenuated when natural splines were used for weather model.	The percent excess deaths estimates for non-accidental deaths per IQR (average of 0-2 day lags) for CoH, predicted $PM_{2.5}$, and sulfate for GAM(stringent convergence criteria) and GLM/natural splines, respectively, were: CoH: 1.38, 0.85; Predicted $PM_{2.5}$: 1.57, 0.55; sulfate: 1.03, 0.27. Confidence bands were not given but the GAM results for predicted $PM_{2.5}$ and sulfate were indicated as significant at 0.05 level.
Özkaynak et al. (1996). Toronto, 1970-1991. TSP (80); COH (0.42 /1000ft).	Total, cardiovascular, COPD, pneumonia, respiratory, cancer, and the remaining mortality series were related to TSP, SO_2, COH, NO_2, O_3, and CO, adjusting for seasonal cycles (by high-pass filtering each series) temperature, humidity, day-of-week, using OLS regression. Factor analysis of multiple pollutants was also conducted to extract automobile related pollution, and mortality series were regressed on the resulting automobile factor scores.	TSP (0 day lag) was significantly associated with total and cardiovascular deaths. NO_2 (0-day lag) was a significant predictor for respiratory and COPD deaths. 2-day lagged O_3 was associated with total, respiratory, and pneumonia deaths. Factor analysis showed factor with high loadings for NO_2, COH, and CO (apparently representing automobile factor) as significant predictor for total, cancer, cardiovascular, respiratory, and pneumonia deaths.	Total mortality excess risk: 2.8% per 100 µg/m^3 TSP at 0 day lag.

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Europe			
Katsouyanni et al. (1997). 12 European (APHEA) cities. 1975-1992 (study years different from city to city). Median Black Smoke (BS) levels ranged from 13 in London to 73 in Athens and Kracow.	Total daily deaths regressed on BS or SO_2 using Poisson GLM models, adjusting for seasonal cycles, day-of-week, influenza epidemic, holidays, temp., humidity. Final analysis done with autoregressive Poisson models to allow for overdispersion and autocorrelation. Pollution effects examined at 0 through 3 day lags and multi-day averages thereof. When city-specific coefficients tested to be homogeneous, overall estimates obtained by computing variance-weighted means of city-specific estimates (fixed effects model). When significant heterogeneity present, source of heterogeneity sought by examining a predefined list of city-specific variables, including annual and seasonal means of pollution and weather variables, number of monitoring sites, correlation between measurements from different sites, age-standardized mortality, proportion of elderly people, smoking prevalence, and geographic difference (north-south, east-west). A random effects model was fit when heterogeneity could not be explained.	Substantial variation in pollution levels (winter mean SO_2 ranged from 30 to 330 µg/m^3), climate, and seasonal patterns were observed across cities. Significant heterogeneity was found for the effects of BS and SO_2, but only the separation between western and central eastern European cities resulted in more homogeneous subgroups. Significant heterogeneity for SO_2 remained in western cities. Cumulative effects of prolonged (two to four days) exposure to air pollutants resulted in estimates comparable with the one day effects. The effects of both SO_2 and BS were stronger during the summer and were independent.	Total mortality excess deaths per 25 µg/m^3 increase in single day BS for western European cities: 1.4 (1.0, 1.8); and 2 (1, 3) per 50 µg/m^3 PM_{10} increase. In central/eastern Europe cities, corresponding figure was 0.3 (0.05, 0.5) per 25 µg/m^3 BS.
Samoli et al. (2001). * APHEA 1 cities (see Katsouyanni (1997). At least five years between 1980-1992. The PM levels are the same as those in Katsouyanni et al. (1997).	In order to further investigate the source of the regional heterogeneity of PM effects, and to examine the sensitivity of the RRs, the APHEA data were re-analyzed by the APHEA investigators themselves (Samoli et al., 2001). Unlike previous model in which sinusoidal terms for seasonal control and polynomial terms for weather, the investigators this time used a GAM model with smoothing terms for seasonal trend and weather, which is more commonly used approach in recent years.	T he estimated relative risks for central-eastern cities were larger than those obtained from the previous model. Also, restricting the analysis to days with concentration < 150 µg/m^3 further reduced the differences between the western and central-eastern European cities. The authors concluded that part of the heterogeneity in the estimated air pollution effects between western and central eastern cities in previous publications was caused by the statistical approach and the data range.	Total mortality RRs per 50 µg/m^3 BS for all cities, western cities, and central-eastern cities using the GAM approach were: 2.5% (2.1, 2.9); 3.1% (2.3, 3.8); and, 2.3% (1.7, 2.9), respectively. In contrast, those with old method were: 1.3% (0.9, 1.7); 2.9% (2.1, 3.7); and, 0.6% (0.1, 1.1), respectively.
Samoli et al. (2003). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria as well as natural splines.	BS risk estimates using GAM were reduced by ~ 10% when stringent convergence criteria were applied. Use of GLM/natural splines resulted in further and greater reductions.	Results corresponding to above using the GAM with stringent convergence criteria were: 2.3%(1.9, 2.7); 2.7% (2.0, 3.4); and, 2.1% (1.5, 2.7), respectively. Corresponding GLM/natural splines results were: 1.2%(0.7, 1.7); 1.6%(0.8, 2.4); and, 1.0%(0.3, 1.7).

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Europe (cont'd)			
Katsouyanni et al. (2001).* 1990-1997 (variable from city to city). 29 European cities. Median PM_{10} ranged from 14 (Stockholm) to 66 (Prague). Median BS ranged from 10 (Dublin) to 64 (Athens).	The 2nd phase of APHEA (APHEA 2) put emphasis on the effect modification by city-specific factors. The first stage of city specific regressions used GAM Poisson model. The second stage regression analysis was conducted to explain any heterogeneity of air pollution effects using city-specific variables. These city-specific variables included average air pollution levels, average temperature/humidity, age-standardize mortality rate, region indicators, etc.	The authors found several effect modifiers. The cities with higher NO_2 levels showed larger PM effects. The cities with warmer climate showed larger PM effects. The cities with low standardized mortality rate showed larger PM effects.	Total mortality excess risk per 50µg/m3 increase in PM_{10}: Fixed effects model: 3.5(2.9, 4.1) Random effects model: 3.1(2.1, 4.2)
Katsouyanni et al. (2003). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria as well as natural splines and penalized splines.	The pooled estimate (random effects estimate) was reduced by 4% when stringent convergence criteria in GAM were used, by 34% when natural splines were used, and by 11% when penalized splines were used. The pattern of effect modification originally reported remained the same. The original findings were unchanged.	Total mortality excess risk per 50µg/m3 increase in PM_{10} using GAM (stringent convergence criteria): 3.3(2.7, 3.9) and 3.0(2.0, 4.1) for fixed effects and random effects models, respectively. Corresponding estimates for GLM/natural splines are: 2.1(1.5, 2.8) and 2.1(1.2, 3.0). Using penalized splines, the estimates are 2.9(2.3, 3.6) and 2.8(1.8, 3.8).
Touloumi et al. (1997). 6 European (APHEA) cities. 1977-1992 (study years different from city to city). Median Black Smoke (BS) levels ranged from 14.6 in London to 84.4 in Athens.	Results of the short-term effects of ambient NO_2 and/or O_3 on daily deaths from all causes (excluding accidents) were discussed to provide a basis for comparison with estimated SO_2 or BS effects in APHEA cities. Poisson GLM models, lag/averaging of pollution, and the computation of combined effects across the cities were done in the same way as done by Katsouyanni et al. (1997), as above.	Significant positive associations found between daily deaths and both NO_2 and O_3. Tendency for larger effects of NO_2 in cities with higher levels of BS. When BS included in the model, pooled estimate for O_3 effect only slightly reduced, but coefficient for NO_2 reduced by half. Authors speculated that short-term effects of NO_2 on mortality confounded by other vehicle-derived pollutants.	NO_2 and/or O_3 estimates only.
Zanobetti and Schwartz (2003a). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria as well as natural splines and penalized splines.	The pooled PM_{10} (average of 0 and 1 day) mortality risk estimate was reduced by 4% when stringent convergence criteria in GAM were used, by 18% when penalized splines were used. For the 4th degree polynomial distributed lag model, corresponding reductions were 10% and 26%.	Combined total mortality excess risk per 50µg/m^3 increase in the average of 0 and 1 day lag PM_{10} was 3.4(2.0, 4.8) using GAM with stringent convergence criteria. For 4th degree polynomial distributed lag model, it was 7.5(4.4, 10.7). Corresponding reductions using penalized splines were 2.9(1.4, 4.4) and 5.6(1.5, 9.8)

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Europe (cont'd)			
Zmirou et al. (1998). 10 European (APHEA) cities. 1977-1992 (study years different from city to city). Median Black Smoke (BS) levels ranged from 13 in London to 73 in Kracow.	Cardiovascular, respiratory, and digestive mortality series in10 European cities analyzed to examine cause-specificity of air pollution. The mortality series were analyzed for associations with PM (BS, except TSP in Milan and Bratislava; PM_{13} in Lyon), NO_2, O_3, and SO_2. Poisson GLM models, lag/averaging of pollution, and computation of combined effects across the cities done in the same way as by Katsouyanni et al. (1997), above.	The cardiovascular and respiratory mortality series were associated with BS and SO_2 in western European cities, but not in the five central European cities. NO_2 did not show consistent mortality associations. RRs for respiratory causes were at least equal to, or greater than those for cardiovascular causes. No pollutant exhibited any association with digestive mortality.	Pooled cardiovascular mortality percent excess deaths per 25 µg/m^3 increase in BS for western European cities: 1.0 (0.3, 1.7); for respiratory mortality, it was 2.0 (0.8, 3.2) in single lag day models (the lags apparently varied across cities).
Bremner et al. (1999). London, UK, 1992-1994. BS (13), PM_{10} (29).	Total, cardiovascular, and respiratory (by age) mortality series were regressed on PM_{10}, BS, O_3, NO_2, CO, and SO_2, adjusting for seasonal cycles, day-of-week, influenza, holidays, temperature, humidity, and autocorrelation using Poisson GLM model.	All effect size estimates (except O_3) were positive for total deaths (though not significant for single lag models). The effects of O_3 found in 1987-1992 were not replicated, except in cardiovascular deaths. Multiple day averaging (e.g., 0-1, 0-2 days) tend to give more significant effect size estimates. The effect size for PM_{10} and BS were similar for the same distributional increment.	1.9% (0.0, 3.8) per 25 µg/m^3 BS at lag 1 day; 1.3% (-1.0, 3.6) per 50 µg/m^3 PM_{10} at lag 1 d for total deaths. Resp. deaths (3 d) = 4.9% (0.5, 9.4). CVD deaths (1 d) = 3.0% (0.3, 5.7).
Prescott et al. (1998). Edinburgh, UK, 1981-1995. PM_{10} (21, by TEOM only for 1992-1995); BS (8.7).	Both mortality (total, cardiovascular, and respiratory) and emergency hospital admissions (cardiovascular and respiratory), in two age groups (<65 and >= 65), were analyzed for their associations with PM_{10}, BS, SO_2, NO_2, O_3, and CO, using Poisson GLM regression adjusting for seasonal cycles, day-of-week, temperature, and wind speed.	Among all the pollutants, BS was most significantly associated with all cause, cardiovascular, and respiratory mortality series. In the subset in which PM_{10} data were available, the RR estimates for BS and PM_{10} for all cause elderly mortality were comparable. Other pollutants' mortality associations were generally inconsistent.	3.8 (1.3, 6.4) per 25 µg/m^3 increase in BS for all cause mortality in age 65+ group, avg. of 1-3 day lags.
Rooney et al. (1998). England and Wales, and Greater London, UK PM_{10} (56, during the worst heat wave; 39, July-August mean)	Excess deaths, by age, sex , and cause, during the 1995 heat wave were estimated by taking the difference between the deaths during heat wave and the 31-day moving averages (for 1995 and 1993-94 separately). The pollution effects, additively for O_3, PM_{10}, and NO_2, were estimated based on the published season-specific coefficients from the 1987-1992 study (Anderson et al., 1996).	Air pollution levels at all the locations rose during the heat wave. 8.9% and 16.1% excess deaths were estimated for England and Wales, and Greater London, respectively. Of these excess deaths, up to 62% and 38%, respectively for these locations, may be attributable to combined pollution effects.	2.6% increase for PM_{10} in Greater London during heat wave.

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in μg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Europe (cont'd)			
Wordley et al. (1997). Birmingham, UK, 1992-1994. PM_{10} (apparently beta-attenuation, 26)	Mortality data were analyzed for COPD, pneumonia, all respiratory diseases, all circulatory diseases, and all causes. Mortality associations with PM_{10} , NO_2, SO_2, and O_3 were examined using OLS (with some health outcomes log- or square-root transformed), adjusting for day-of-week, month, linear trend, temperature and relative humidity. The study also analyzed hospital admission data.	Total, circulatory, and COPD deaths were significantly associated with 1-day lag PM_{10}. The gaseous pollutants "did not have significant associations independent from that of PM_{10}", and the results for gaseous pollutants were not presented. The impact of reducing PM_{10} to below 70 μg/m^3 was estimated to be "small" (0.2% for total deaths), but the PM_{10} level above 70 μg/m^3 occurred only once during the study period.	5.6% (0.5, 11.0) per 50 μg/m^3 PM_{10} at 1 d lag for total deaths. COPD (1 d lag) deaths = 27.6 (5.1, 54.9). Circulatory (1 d) deaths = 8.8 (1.9, 17.1)
Hoek et al. (2000). * The Netherlands, 1986-1994. PM_{10} (median 34); BS (median 10).	Total, cardiovascular, COPD, and pneumonia mortality series were regressed on PM_{10}, BS, sulfate, nitrate, O_3, SO_2, CO, adjusting for seasonal cycles, day-of-week, influenza, temperature, and humidity using Poisson GAM model. Deaths occurring inside and outside hospitals were also examined.	Particulate air pollution was not more consistently associated with mortality than were the gaseous pollutants SO_2 and NO_2. Sulfate, nitrate, and BS were more consistently associated with total mortality than was PM_{10}. The RRs for all pollutants were larger in the summer months than in the winter months.	Total mortality excess risk estimate per 50 μg/m^3 PM_{10} (average of 0-6 days): 1.2(0.2, 2.2); 0.9(-0.8, 2.7) for CVD; 5.9(0.9, 11.2) for COPD; and 10.1(3.6, 17.1) for pneumonia.
Hoek (2003). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria and natural splines.	Very little change in PM risk coefficients (often slightly increased) whether GAM with stringent convergence criteria or GLM./natural splines were used.	Total mortality excess risk estimate per 50 μg/m^3 PM_{10} (average of 0-6 days) using GAM with stringent convergence criteria: 1.4(0.3, 2.6); 0.9(-0.8, 2.7) for CVD; 6.1(1.0, 11.4) for COPD; and 10.3(3.7, 17.2) for pneumonia. Corresponding numbers using GLM/natural splines are: 1.2(-0.1, 2.5); 1.6(-0.3, 3.5); 6.0(0.4, 11.8); 10.7 (3.5, 18.3).
Hoek et al. (2001).* The Netherlands. 1986-1994. PM_{10} (median 34); BS (median 10).	This study of the whole population of the Netherlands, with its large sample size (mean daily total deaths ~ 330, allowed examination of specific cardiovascular cause of deaths. GAM Poisson regression models, adjusting for seasonal cycles, temperature, humidity, day-of-week was used.	Deaths due to heart failure, arrhythmia, cerebrovascular causes, and thrombocytic causes were more strongly (~ 2.5 to 4 times larger relative risks) associated with air pollution than the overall cardiovascular deaths (CVD) or myocardial infarction (MI) and other ischemic heart disease (IHD).	For PM_{10} (7-day mean), RRs for total CVD, MI/IHD, arrhythmia, heart failure, cerebrovascular, and thrombocytic mortality per 50 μg/m^3 increase were:0.9(-0.8, 2.7), 0.3(-2.3, 3.0), 2.5(-4.3, 9.9), 2.2(-2.5, 7.2), 1.9(-1.8, 5.8), and 0.6(-6.8, 8.7) , respectively. The RRs for BS were larger and more significant than those for PM_{10}.

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Europe (cont'd)			
Hoek (2003). Re-analysis of above study.	Re-analysis of above study using stringent convergence criteria and natural splines.	Very little change in PM risk coefficients (often slightly increased) whether GAM with stringent convergence criteria or GLM./natural splines were used.	For PM_{10} (7-day mean), RRs for total CVD, MI/IHD, arrhythmia, heart failure, cerebrovascular, and thrombocytic mortality per 50 µg/m^3 increase using GAM with stringent convergence criteria were:0.9(-0.8, 2.7), 0.4(-2.2, 3.0), 2.7(-4.2, 10.1), 2.4(-2.3, 7.4), 2.0(-1.7, 5.9), and 0.7(-6.8, 8.8) , respectively. The RRs for BS were larger and more significant than those for PM_{10}.
Pönkä et al. (1998). Helsinki, Finland, 1987-1993. TSP (median 64); PM_{10} (median 28)	Total and cardiovascular deaths, for age groups < 65 and 65 +, were related to PM_{10}, TSP, SO_2, NO_2, and O_3, using Poisson GLM model adjusting for temperature, relative humidity, day-of-week, temporal patterns, holiday and influenza epidemics.	No pollutant significantly associated with mortality from all cardiovascular or CVD causes in 65+ year age group. Only in age <65 year group, PM_{10} associated with total and CVD deaths with 4 and 5 d lags, respectively. The "significant" lags were rather "spiky". O_3 was also associated with CVD mortality <65 yr. group with inconsistent signs and late and spiky lags (neg. on d 5 and pos. on d 6).	18.8% (5.6, 33.2) per 50 µg/m^3 PM_{10} 4 day lag (other lags negative or zero).
Peters et al. (2000b). A highly polluted coal basin area in the Czech Republic and a rural area in Germany, northeast Bavaria districts. 1982-1994. TSP: mean = 121.1 and 51.6, respectively, for these two regions. PM_{10} and $PM_{2.5}$ were also measured in the coal basin during 1993-1994 (mean = 65.9 and 51.0, respectively).	Non-accidental total and cardiovascular deaths (mean = 18.2 and 12.0 per day, for the Czech and Bavaria areas, respectively). The APHEA approach (Poisson GLM model with sine/cosine, temperature as a quadratic function, relative humidity, influenza, day-of-week as covariates), as well as GLM with natural splines for temporal trends and weather terms were considered. Logarithm of TSP, SO_2, NO_2, O_3, and CO (and PM_{10} and $PM_{2.5}$ for 1993-1994) were examined at lags 0 through 3 days.	In the coal basin (i.e., the Czech Republic polluted area), on the average, 68% of the TSP was PM_{10}, and most of PM_{10} was $PM_{2.5}$ (75%). For the coal basin, associations were found between the logarithm of TSP and all-cause mortality at lag 1 or 2 days. SO_2 was also associated with all-cause mortality with slightly lower significance. PM_{10} and $PM_{2.5}$ were both associated with all-cause mortality in 1993-1994 with a lag of 1-day. NO_2, O_3 and CO were positively but more weakly associated with mortality than PM indices or SO_2. In the Bavarian region, neither TSP nor SO_2 was associated with mortality, but CO (at lag 1-day) and O_3 (at lag 0-day) were associated with all-cause mortality.	Total mortality excess deaths per 100 µg/m^3 increase in TSP for the Czech region: 3.8 (0.8, 6.9) at lag 2-day for 1982-1994 period. For period 1993-1994, 9.5 (1.2, 18.5) per 100 µg/m^3 increase in TSP at lag 1-day, and 4.8 (0.7, 9.0) per 50 µg/m^3 increase in PM_{10}; and 1.4 (-0.5, 3.4) per 25 µg/m^3 $PM_{2.5}$.

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Europe (cont'd)			
Hoek et al. (1997). +Rotterdam, the Netherlands, 1983-1991. TSP (median 42); BS (median 13).	Total mortality (also by age group) was regressed on TSP, Fe (from TSP filter), BS, O_3, SO_2, CO, adjusting for seasonal cycles, day-of-week, influenza, temperature, and humidity using Poisson GAM model.	Daily deaths were most consistently associated with TSP. TSP and O_3 effects were "independent" of SO_2 and CO. Total iron (from TSP filter) was associated "less consistently" with mortality than TSP was. The estimated RRs for PM indices were higher in warm season than in cold season.	5.5 (1.1, 9.9) per 100 µg/m^3 TSP at 1 day lag.
Kotěšovec et al. (2000). Northern Bohemia, Czech Republic, 1982-1994. TSP (121.3).	Total (excluding accidents and children younger than 1 yr), cause specific (cardiovascular and cancer), age (65 and less vs. otherwise), and gender specific mortality series were examined for their associations with TSP and SO_2 using logistic model, adjusting for seasonal cycles, influenza epidemics, linear and quadratic temperature terms. Lags 0 through 6 days, as well as a 7 day mean values were examined.	For the total mortality, TSP, but not SO_2, was associated. There were apparent differences in associations were found between men and women. For example, for age below 65 cardiovascular mortality was associated with TSP for men but not for women.	Total mortality percent excess deaths per 100 µg/m^3 increase in TSP at 2 day lag was 3.4 (0.5, 6.4).
Zanobetti et al. (2000a). Milan, Italy. 1980-1989. TSP mean = 142.	The focus of this study was to quantify mortality displacement using what they termed "GAM distributed lag models". (smoothing term was fited with Penalized Plines) Non-accidental total deaths were regressed on smooth function of TSP distributed over the same day and the previous 45 days using penalized splines for the smooth terms and seasonal cycles, temperature, humidity, day-of-week, holidays, and influenza epidemics. The mortality displacement was modeled as the initial positive increase, negative rebound (due to depletion), followed by another positive coefficients period, and the sum of the three phases were considered as the total cumulative effect.	TSP was positively associated with mortality up to 13 days, followed by nearly zero coefficients between 14 and 20 days, and then followed by smaller but positive coefficients up to the 45th day (maximum examined). The sum of these coefficients was over three times larger than that for the single-day estimate.	Total mortality percent increase estimates per IQR increase in TSP: 2.2 (1.4, 3.1) for single-day model; 6.7 (3.8, 9.6) for distributed lag model.
Anderson et al. (1996). London, UK, 1987-1992. BS (15)	Total, cardiovascular, and respiratory mortality series were regressed on BS, O_3, NO_2, and SO_2, adjusting for seasonal cycles, day-of-week, influenza, holidays, temperature, humidity, and autocorrelation using Poisson GLM model.	Both O_3 (0 day lag) and BS (1 day lag) were significant predictors of total deaths. O_3 was also positively significantly associated with respiratory and cardiovascular deaths. The effect size estimates per the same distributional increment (10% to 90%) were larger for O_3 than for BS. These effects were larger in warm season. SO_2 and NO_2 were not consistently associated with mortality.	2.8% (1.4, 4.3) per 25 µg/m^3 BS at 1-d lag for total deaths. CVD (1 d) = 1.0 (-1.1, 3.1). Resp. (1 d) = 1.1 (-2.7, 5.0).

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Europe (cont'd)			
Michelozzi et al. (1998) +Rome, Italy, 1992-1995. TSP ("PM_{13}" beta attenuation, 84).	Total mortality was related to PM_{13}, SO_2, NO_2, CO, and O_3, using Poisson GAM model , adjusting for seasonal cycles, temperature, humidity, day-of-week, and holiday. Analysis of mortality by place of residence, by season, age, place of death (in or out of hospital), and cause was also conducted.	PM_{13} and NO_2 were most consistently associated with mortality. CO and O_3 coefficients were positive, SO_2 coefficients negative. RR estimates higher in the warmer season. RRs similar for in- and out-of hospital deaths.	1.9% (0.5, 3.4) per 50 µg/m^3 PM_{13} at 0 day lag.
Garcia-Aymerich et al. (2000). Barcelona, Spain. 1985-1989. Black Smoke no data distribution was reported).	Daily total (mean = 1.8/day), respiratory, and cardiovascular mortality counts of a cohort (9,987 people) with COPD or asthma were associated with black smoke (24-hr), SO_2 (24-hr and 1-hr max), NO_2 (24-hr and 1-hr max), O_3 (1-hr max), temperature, and relative humidity. Poisson GLM regression models using APHEA protocol were used. The resulting RRs were compared with those of the general population.	Daily mortality in COPD patients was associated with all six pollution indices. This association was stronger than in the general population only for daily 1-hr max of SO_2, daily 1-hr max and daily means of NO_2. BS and daily means of SO_2 showed similar or weaker associations for COPD patients than for the general population.	Total mortality percent increase per 25 µg/m^3 increase in avg. of 0-3 day lags of BS: 2.76 (1.31, 4.23) in general population, and 1.14 (-4.4, 6.98) in the COPD cohort.
Rahlenbeck and Kahl (1996). East Berlin, 1981-1989. "SP" (beta attenuation, 97)	Total mortality (as well as deviations from long-wave cycles) was regressed (OLS) on SP and SO_2, adjusting for day-of-week, month, year, temperature, and relative humidity, using OLS, with options to log-transform pollution, and w/ and w/o days with pollution above 150 µg/m^3.	Both SP and SO_2 were significantly associated with total mortality with 2 day lag in single pollutant model. When both pollutants were included, their coefficients were reduced by 33% and 46% for SP and SO_2, respectively.	6.1% per 100 µg/m^3 "SP" at 2 day lag.
Rossi et al. (1999) + Milan, Italy, 1980-1989 TSP ("PM_{13}" beta attenuation, 142)	Specific causes of death (respiratory, respiratory infections, COPD, circulatory, cardiac, heart failure, and myocardial infarction) were related to TSP, SO_2, and NO_2, adjusting for seasonal cycles, temperature, and humidity, using Poisson GAM model.	All three pollutants were associated with all cause mortality. Cause-specific analysis was conducted for TSP only. Respiratory infection and heart failure deaths were both associated with TSP on the concurrent day, whereas the associations for myocardial infarction and COPD deaths were found for the average of 3 to 4 day prior TSP.	3.3% (2.4, 4.3) per 100 µg/m^3 TSP at 0 day lag.
Sunyer et al. (2000). Barcelona, Spain. 1990-1995. BS means: 43.9 for case period, and 43.1 for control period.	Those over age 35 who sought emergency room services for COPD exacerbation during 1985-1989 and died during 1990-1995 were included in analysis. Total, respiratory, and cardiovascular deaths were analyzed using a conditional logistic regression analysis with a case-crossover design, adjusting for temperature, relative humidity, and influenza epidemics. Bi-directional control period at 7 days was used. Average of the same and previous 2 days used for pollution exposure period. Data also stratified by potential effect modifiers (e.g., age, gender, severity and number of ER visits, etc.).	BS levels were associated with all cause deaths. The association was stronger for respiratory causes. Older women, patients admitted to intensive care units, and patients with a higher rate of ER visits were at greater risk of deaths associated with BS.	Percent increase per 25 µg/m^3 increase in 3-day average BS: 14.2 (1.6, 28.4) for all causes; 9.7 (-10.2, 34.1) for cardiovascular deaths; 23.2 (3.0, 47.4) for respiratory deaths.

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

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Europe (cont'd)			
Sunyer and Basagana (2001). Barcelona, Spain. 1990-1995. See Sunyer et al. (2000) for PM levels.	The analysis assessed any "independent" particle effects, after controlling for gaseous pollutants, on a cohort of patients with COPD (see the summary description for Sunyer et al. (2000) for analytical approach). PM_{10}, NO_2, O_3, and CO were analyzed.	PM_{10}, but not gaseous pollutants were associated with mortality for all causes. In the two-pollutant models, the PM_{10}-mortality associations were not diminished, whereas those with gaseous pollutants were.	Odds ratio for all cause mortality per IQR PM_{10} on the same-day (27 µg/m^3) was 11% (0, 24). In two pollutant models, the PM_{10} RRs were 10.5%, 12.9%, and 10.8% with NO_2, O_3, and CO, respectively.
Tobias and Campbell (1999). Barcelona, Spain. 1991-1995. Black Smoke (BS) (no data distribution was reported).	Study examined the sensitivity of estimated total mortality effects of BS to different approaches to modeling influenza epidemics: (1) with a single dummy variable; (2) with three dummy variables; (3) using daily number of cases of influenza. Poisson GLM regression used to model total daily mortality, adjusting for weather, long-term trend, and season, apparently following APHEA protocol.	Using the reported daily number of influenza cases resulted in a better fit (i.e., a lower AIC) than those using dummy variables. In the "better" model, the black smoke coefficient was about 10% smaller than those in the models with dummy influenza variables, but remained significant. Lags not reported.	Total mortality excess deaths per 25 µg/m^3 increase in BS: 1.37 (0.20, 2.56) for model using the daily case of influenza; 1.71 (0.53, 2.91) for model with three influenza dummy variables.
Alberdi Odriozola et al. (1998). Madrid, Spain, 1986-1992. "TSP" (beta attenuation, 47 for average of 2 stations)	Total, respiratory, and cardiovascular deaths were related to TSP and SO_2. Multivariate autoregressive integrated moving average models used to adjust for season, temperature, relative humidity, and influenza epidemics.	TSP (1-day lag) and SO_2 (3-day lagged) were independently associated with mortality.	4.8% (1.8, 7.7) per 100 µg/m^3 TSP at lag 1 day.
Díaz et al. (1999). Madrid, Spain. 1990-1992. TSP (no data distribution was reported).	Non-accidental, respiratory, and cardiovascular deaths (mean = 62.4, 6.3, and 23.8 per day, respectively). Auto-regressive Integrated Moving Average (ARIMA) models fit to both depend and independ. variables first to remove auto-correlation and seasonality (i.e., pre-whitening"), followed by examining cross-correlation to find optimal lags. Multivariate OLS models thus included ARIMA components, seasonal cycles (sine/cosine), V-shaped temp., and optimal lags found for pollution and weather variables. TSP, SO_2, NO_2, and O_3 examined. Season-specific analyses also conducted.	TSP was significantly associated with non-accidental mortality at lag 0 for year around and winter, but with a 1-day lag in summer. A similar pattern was seen for circulatory deaths. For respiratory mortality, a significant association with TSP was found only in summer (0-day lag). SO_2, NOx, and NO_2 showed similar associations with non-accidental deaths at lag 0 day. O_3' associations with non-accidental mortality was U-shaped, with inconsistent lags (1, 4, and 10).	For non-accidental mortality, excess deaths was 7.4% (confidence bands not reported; $p < 0.05$) per 100 µg/m^3 TSP at 0 day lag.

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

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Europe (cont'd)			
Wichmann et al., (2000) *Erfurt, Germany. 1995-1998. Number counts (NC) & mass concentrations (MC) of ultrafine particles in three size classes, 0.01 to 0.1 m, and fine particles in three size classes from 0.1 to 2.5 m diameter, using Spectrometryll Mobile Aerosol Spectrometry (MAS). MAS MC PM2.5-0.01 (mean 25.8, median 18.8, IQR 19.9). Filter measurements of PM_{10} (mean 38.2, median 31.0, IQR 27.7) and $PM_{2.5}$ (mean 26.3, median 20.2, IQR 18.5). MAS NC2.5-0.01 (mean 17,966 per cu.cm, median 14,769, IQR 13,269).	Total non-accidental, cardiovascular, and respiratory deaths (mean 4.88, 2.87, 1.08 per day, respectively) were related to particle mass concentration and number counts in each size class, and to mass concentrations of gaseous co-pollutants NO_2, CO, SO_2, using GAM regression models adjusted for temporal trends, day of week, weekly national influenza rates, temperature and relative humidity. Data analyzed by season, age group, and cause of death separately. Single-day lags and polynomial distributed lag models (PDL) used. Particle indices and pollutants fitted using linear, log-transformed, and LOESS transformations. Two-pollutant models with a particle index and a gaseous pollutant were fitted. The "best" model as used by Wichmann et al. (2000) was that having the highest t-statistic, since other criteria (e.g., log-likelihood for nested models) and AIC for non-nested models could not be applied due to different numbers of observations in each model. There should be little difference between these approaches and resulting differences in results should be small in practice. Sensitivity analyses included stratifying data by season, winter year, age, cause of death, or transformation of the pollution variable (none, logarithmic, non-parametric smooth).	Loss of stat. power by using a small city with a small number of deaths was offset by advantage of having good exposure representation from single monitoring site. Since ultrafine particles can coagulate into larger aggregates in a few hours, ultrafine particle size and numbers can increase into the fine particle category, resulting in some ambiguity. Significant associations were found between mortality and ultrafine particle number concentration (NC), ultrafine particle mass concentration (MC), fine particle mass concentration, or SO_2 concentration. The correlation between MC0.01-2.5 and NC0.01-0.1 is only moderate, suggesting it may be possible to partially separate effects of ultrafine and fine particles. The most predictive single-day effects are either immediate (lag 0 or 1) or delayed (lag 4 or 5 days), but cumulative effects characterized by PDL are larger than single-day effects. The significance of SO_2 is robust, but hard to explain as a true causal factor since its concentrations are very low. Age is an important modifying factor, with larger effects at ages < 70 than ≥ 70 years. Respiratory mortality has a higher RR than cardio-vascular mortality. A large number of models were fitted, with some significant findings of association between mortality and particle mass or number indices.	Total mortality excess deaths: Filter PM_{10} (0-4 d lag) = 6.6 (0.7, 12.8) per 50 µg/m^3. Filter $PM_{2.5}$ (0-1 d) = 3.0 (-1.7, 7.9). MC for $PM_{0.01-2.5}$ 6.2% (1.4, 11.2) for all year; by season, Winter = 9.2% (3.0, 15.7) Spring = 5.2% (-2.0, 12.8) Summer = -4.7% (-18.7, 11.7) Fall = 9.7% (1.9, 18.1) For ultrafine PM, NC 0.01-0.1 (0-4 d lag): All Year = 8.2% (0.3, 16.9) Winter = 9.7% (0.3, 19.9) Spring = 10.5% (-1.4, 23.9) Summer = -13.9% (-29.8, 5.7) Fall = 12.0% (2.1, 22.7) Best single-day lag: $PM_{0.01-0.1}$ per 25 µg/m^3: 3.6(-0.4, 7.7) $PM_{0.01-2.5}$ per 25 µg/m^3: 3.9(0.0, 8.0) $PM_{2.5}$ per 25 µg/m^3: -4.0(-7.9, 0) PM_{10} per 25 µg/m^3: 6.4(0.3, 12.9)
Stolzel et al. (2003). Re-analysis of above study.	Re-analysis of above study using GAM with stringent convergence criteria as well as GLM/natural splines. The polynomial distributed lag model was not re-analyzed.	Very little change in PM risk coefficients when GAM models with stringent convergence criteria were used. When GLM./natural splines were used, many of the coefficients for number concentrations slightly increased, but the coefficients for mass concentrations decreased slightly.	Best single-day lag using GAM (stringent): $PM_{0.01-0.1}$ per 25 µg/m^3: 3.6(-0.4, 7.7) $PM_{0.01-2.5}$ per 25 µg/m^3: 3.8(-0.1, 7.8) $PM_{2.5}$ per 25 µg/m^3: -4.0(-7.8, -0.1) PM_{10} per 25 µg/m^3: 6.2(0.1, 12.7) Best single-day lag using GLM/natural splines: $PM_{0.01-0.1}$ per 25 µg/m^3: 3.1(-1.6, 7.9) $PM_{0.01-2.5}$ per 25 µg/m^3: 3.7(-0.9, 8.4) $PM_{2.5}$ per 25 µg/m^3: -3.4(-7.9, 1.4) PM_{10} per 25 µg/m^3: 5.3(-1.8, 12.9)

+ = Used GAM with multiple non-parametric smooths, but have not yet re-analyzed. * = Used S-Plus Default GAM, and have re-analyzed results; GAM = Generalized Additive Model, GEE = Generalized Estimation Equations, GLM = Generalized Linear Model.

TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Europe (cont'd)			
Zeghnoun et al. (2001). +Rouen and Le Havre, France. 1990-1995. PM_{13} mean = 32.9 for Rouen, 36.4 for Le Havre. BS mean = 18.7 for Rouen, 16.3 for Le Havre.	Total, cardiovascular, and respiratory mortality series were regressed on BS, PM_{13}, SO_2, NO_2, and O_3 in 1- and 2-pollutant models using GAM Poisson models adjusting for seasonal trends, day-of-week, and weather.	In Rouen, O_3, SO_2, and NO_2 were each significantly associated with total, respiratory, and cardiovascular mortality, respectively. In Le Havre, SO_2 and PM_{13} were associated with cardiovascular mortality. However, the lack of statistical significance reported for most of these results may be in part due to the relatively small population size of these cities (430,000 and 260,000, respectively).	PM_{13} total mortality RRs per IQR were 0.5% (-1.1, 2.1) in Rouen (IQR=20.6, 1-day lag) and 1.9% (-0.8, 7.4) in Le Havre (IQR=23.9, 1-day lag). BS total mortality RRs per IQR were 0.5% (-1.8, 2.9) in Rouen (IQR=14.2, 1-day lag) and 0.3% (-1.6, 2.2) in Le Havre (IQR=11.5, 0-1 day lag avg.).
Roemer and Van Wijnen (2001). + Amsterdam. 1987-1998. BS and PM_{10} means in "background" = 10 and 39; BS mean in "traffic" area = 21. (No PM_{10} measurements available at traffic sites)	Daily deaths for those who lived along roads with more than 10,000 motor vehicle, as well as deaths for total population, were analyzed using data from background and traffic monitors. Poisson GAM model was used adjusting for season, day-of-week, and weather. BS, PM_{10}, SO_2, NO_2, CO, and O3 were analyzed.	Correlations between the background monitors and traffic monitors were moderate for BS (r = 0.55) but higher for NO_2 (r = 0.79) and O_3 (r = 0.80). BS and NO_2 were associated with mortality in both total and traffic population. Estimated RR for traffic population using background sites was larger than the RR for total population using background sites. The RR for total pop. using traffic sites was smaller that RRs for total population using background sites. This is not surprising since the mean BS for traffic sites were larger that for background sites.	The RRs per 100 µg/m^3 BS (at lag 1-day) were 1.383 (1.153, 1.659), 1.887 (1.207, 2.949), and 1.122 (1.023, 1.231) for total population using background sites, traffic population using background sites, and total population using traffic sites, respectively. Results for traffic pop. using traffic sites not reported)
Anderson et al. (2001). +The west Midlands conurbation, UK. 1994-1996. PM means: PM_{10} = 23, $PM_{2.5}$ = 15, $PM_{10\text{-}2.5}$ = 9, BS = 13.2, sulfate = 3.7.	Non-accidental cause, cardiovascular, and respiratory mortality (as well as hospital admissions) were analyzed for their associations with PM indices and gaseous pollutants using GAM Poisson models adjusting for seasonal cycles, day-of-week, and weather.	Daily non-accidental mortality was not associated with PM indices or gaseous pollutants in the all-year analysis. However, all the PM indices (except coarse particles) were positively and significantly associated with non-accidental mortality (age over 65) in the warm season. Of gaseous pollutants, NO_2 and O_3 were positively and significantly associated with non-accidental mortality in warm season. Two pollutant models were not considered because "so few associations were found".	Percent excess mortality for PM_{10}, $PM_{2.5}$, and $PM_{10\text{-}2.5}$ (avg. lag 0 and 1 days) were 0.2% (-1.8, 2.2) per 24.4 uµg/m^3 PM_{10}, 0.6% (-1.5, 2.7) per 17.7 uµg/m^3 $PM_{2.5}$, and –0.6% (-4.2, 2.3) per 11.3 uµg/m^3 $PM_{10\text{-}2.5}$ in all-year analysis. The results for season specific analysis were given only as figures.
Keatinge and Donaldson (2001). Greater London, England, 1976-1995. BS mean = 17.7.	The study examined potential confounding effects of atypical cold weather on air pollution/mortality relationships. First, air pollution variables (SO_2, CO and BS) were modeled as a function of lagged weather variables These variables were deseasonalized by regressing on seine and cosine variables. Mortality regression (OLS) included various lagged and averaged weather and pollution variables. Analyses were conducted in the linear range of mortality/temperature relationship (15 to 0 degrees C).	Polluted days were found to be colder and less windy and rainy than usual. In the regression of mortality on the multiple-lagged temperature, wind, rain, humidity, sumshine, SO_2, CO, and BS, cold temperature was associated with mortality increase, but not SO_2 or CO. BS suggestive evidence, though not statistically significant, of association at 0- and 1-day lag.	3% (95% CI not reported) increase in daily mortality per 17.7 µg/m^3 of BS (lag 0 and 1).

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Latin America			
Cifuentes et al. (2000).+ Santiago, Chile. 1988-1996. $PM_{2.5}$ (64.0), and $PM_{10-2.5}$ (47.3).	Non-accidental total deaths (56.6 per day) were examined for associations with $PM_{2.5}$, $PM_{10-2.5}$, O_3, CO, SO_2, and NO_2. Data analyzed using GAM Poisson regression models, adjusting for temperature, seasonal cycles. Single and two pollutant models with lag days from 0 to 5, as well as the 2- to 5-day average concentrations evaluated. They also reported results for comparable GLM model.	Both PM size fractions associated with mortality, but different effects found for warmer and colder months. $PM_{2.5}$ and $PM_{10-2.5}$ both important in whole year, winter, and summer. In summer, $PM_{10-2.5}$ had largest effect size estimate. NO_2 and CO also associated with mortality, as was O_3 in warmer months. No consistent SO_2-mortality associations.	Percent excess total deaths per 25 µg/m^3 increase in the average of previous two days for the whole year: 1.8 (1.3, 2.4) for $PM_{2.5}$ and 2.3 (1.4, 3.2) for $PM_{10-2.5}$ in single pollutant GAM models. In GLM models (whole year only), 1.4 (0.6, 2.1) for $PM_{2.5}$ and 1.6 (0.2, 3.0) for $PM_{10-2.5}$
Castillejos et al. (2000). Mexico City. 1992-1995. PM_{10} (44.6), $PM_{2.5}$ (27.4), and $PM_{10-2.5}$ (17.2).	Non-accidental total deaths, deaths for age 65 and over, and cause-specific (cardiac, respiratory, and the other remaining) deaths were examined for their associations with PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, O_3, and NO_2. Data were analyzed using GAM Poisson regression model (only one non-parametric smoothing term), adjusting for temperature (average of 1-3 day lags) and seasonal cycles. Individual pollution lag days from 0 to 5, and average concentrations of previous 5 days were considered.	All three particle size fractions were associated individually with mortality. The effect size estimate was largest for $PM_{10-2.5}$. The effect size estimate was stronger for respiratory causes than for total, cardiovascular, or other causes of death. The results were not sensitive to additions of O_3 and NO_2. In the model with simultaneous inclusion of $PM_{2.5}$ and $PM_{10-2.5}$, the effect size for $PM_{10-2.5}$ remained about the same, but the effect size for $PM_{2.5}$ became negligible.	Total mortality percent increase estimates per increase for average of previous 5 days: 9.5 (5.0, 14.2) for 50 µg/m^3 PM_{10}; 3.7 (0, 7.6) for 25 µg/m^3 $PM_{2.5}$; and 10.5 (6.4, 14.8) for 25 µg/m^3 $PM_{10-2.5}$.
Loomis et al. (1999). Mexico-City, 1993-1995. $PM_{2.5}$ (mean: 27.4 µg/m^3)	Infant mortality (avg. 3/day) related to $PM_{2.5}$, O_3, and NO_2, adjusting for temperature and smoothed time, using Poisson GAM model (same model as above, with only one non-parametric smoothing term)	Excess infant mortality associated with $PM_{2.5}$, NO_2, and O_3 in the same average/lags. NO_2 and O_3 associations less consistent in multi-pollutant models.	Infant mortality excess risk: 18.2% (6.4, 30.7) per 25 µg/m^3 $PM_{2.5}$ at avg. 3-5 lag days.
Borja-Aburto et al. (1998). Mexico-City, 1993-1995. $PM_{2.5}$ (mean: 27)	Total, respiratory, cardiovascular, other deaths, and age-specific (age >= 65) deaths were related to $PM_{2.5}$, O_3, and NO_2, adjusting for 3-day lagged temperature and smoothing splines for temporal trend, using Poisson GAM model (only one non-parametric smoothing term).	$PM_{2.5}$, O_3, and NO_2 were associated with mortality with different lag/averaging periods (1 and 4 day lags; 1-2 avg.; 1-5 avg., respectively). $PM_{2.5}$ associations were most consistently significant. SO_2 was available, but not analyzed because of its "low" levels.	For total excess deaths, 3.4% (0.4, 6.4) per 25 µg/m^3 $PM_{2.5}$ for both 0 and 4 d lags. For respiratory (4 d) = 6.4 (-2.6, 16.2); for CVD (4 d) = 5.6 (-0.1, 11.5)
Borja-Aburto et al. (1997). Mexico-City, 1990-1992. TSP (median: 204)	Total, respiratory, cardiovascular, and age-specific (age > = 65) deaths were related to O_3, TSP, and CO, adjusting for minimum temperature (temperature also fitted seasonal cycles) using Poisson GLM models. The final models were estimated using the iteratively weighted and filtered least squares method to account for overdispersion and autocorrelation.	O_3, SO_2, and TSP were all associated with total mortality in separate models, but in multiple pollutant model, only TSP remained associated with mortality. CO association weak.	Total deaths: 6% (3.3, 8.3) per 100 µg/m^3 TSP at 0 d lag. CVD deaths: 5.2% (0.9, 9.9). Resp. deaths: 9.5% (1.3, 18.4).

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Latin America (cont'd)			
Tellez-Rojo et al. (2000). Mexico City. 1994. PM10 mean = 75.1.	One year of daily total respiratory and COPD mortality series were analyzed for their associations with PM_{10} and O3 using Poisson GLM model adjusting for cold or warm months, and 1-day lagged minimum temperature. The data were stratified by the place of deaths.	The average number of daily respiratory deaths, as well as that of COPD deaths, was similar for in and out of hospital. They found that the estimated PM_{10} relative risks were consistently larger for the deaths that occurred outside medical units. The results are apparently consistent with the assumption that the extent of exposure misclassification may be smaller for those who died outside medical units.	Percent excess for total respiratory and COPD mortality were 2.9% (0.9, 4.9) and 4.1% (1.3, 6.9) per 10 uµg/m^3 increase in 3-day lag PM_{10},
Pereira et al. (1998). Sao Paulo, Brazil, 1991-1992. PM_{10} (beta-attenuation, 65)	Intrauterine mortality associations with PM_{10}, NO_2, SO_2, CO, and O_3 investigated using Poisson GLM regression adjusting for season and weather. Ambient CO association with blood carboxyhemoglobin sampled from umbilical cords of non-smoking pregnant mothers studied in separate time period.	NO_2, SO_2, and CO were all individually significant predictor of the intrauterine mortality. NO_2 was most significant in multi-pollutant model. PM_{10} and O_3 were not significantly associated with the mortality. Ambient CO levels were associated with and carboxyhemoglobin of blood sampled from the umbilical cords.	Intrauterine mortality excess risk: 4.1% (-1.8, 10.4) per 50 µg/m^3 PM_{10} at 0 day lag.
Gouveia and Fletcher (2000). Sao Paulo, Brazil. 1991-1993. PM_{10} mean = 64.3.	All non-accidental causes, cardiovascular, and respiratory mortality were analyzed for their associations with air pollution (PM_{10}, SO_2, NO_2, O_3, and CO) using Poisson GLM model adjusting for trend, seasonal cycles, and weather. Potential roles of age and socio-economic status were examined by stratifying data by these factors.	There was an apparent effect modification by age categories. Estimated PM_{10} effects were higher for deaths above age 65 (highest for the age 85+ category), and no associations were found in age group < 65 years. Respiratory excess deaths were larger than those for cardiovascular or non-accidental deaths. Other pollutants were also associated with the elderly mortality.	Percent excess for total non-accidental, cardiovascular, and respiratory mortality for those with age > 65 were 3.3% (0.6, 6.0), 3.8% (0.1, 7.6), and 6.0 (0.5, 11.8), respectively, per 64.2 uµg/m^3 increase in PM_{10} (0-, 0-, and 1-day lag, respectively).
Conceição et al. (2001) +Sao Paulo, Brazil. 1994-1997. PM_{10} mean = 66.2	Daily respiratory deaths for children under 5 years of age were analyzed for their associations with air pollution (PM_{10}, SO_2, O_3, and CO) using GAM Poisson model adjusting for seasonal cycles and weather.	Significant mortality associations were found for CO, SO_2, and PM_{10} in single pollutant models. When all the pollutants were included, PM_{10} coefficient became negative and non-significant.	Percent excess for child (age < 5) respiratory deaths: 9.7% (1.5, 18.6) per 66.2 µg/m^3 PM_{10} (2-day lag) in single pollutant model.

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in µg/m^3.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Australia			
Morgan et al. (1998). Sydney, 1989-1993. Nephelometer (0.30 bscat/104m). Site-specific conversion: $PM_{2.5}$ 9; PM_{10} 18	Total, cardiovascular, and respiratory deaths were related to PM (nephelometer), O_3, and NO_2, adjusting for seasonal cycles, day-of-week, temperature, dewpoint, holidays, and influenza, using Poisson GEE model to adjust for autocorrelation.	PM, O_3, and NO_2 all showed significant associations with total mortality in single pollutant models. In multiple pollutant models, the PM and O_3 effect estimates for total and cardiovascular deaths were marginally reduced, but the PM effect estimate for respiratory deaths was substantially reduced.	4.7% (1.6, 8.0) per 25 µg/m^3 estimated $PM_{2.5}$ or 50 µg/m^3 estimated PM_{10} at avg. of 0 and 1 day lags. (Note: converted from nephelometry data)
Simpson et al. (1997). Brisbane, 1987-1993. PM_{10} (27, not used in analysis). Nephelometer (0.26 bscat/104m, size range: 0.01-2 m).	Total, cardiovascular, and respiratory deaths (also by age group) were related to PM (nephelometer), O_3, SO_2, and NO_2, adjusting for seasonal cycles, day-of-week, temperature, dewpoint, holidays, and influenza, using Poisson GEE model to adjust for autocorrelation. Season-specific (warm and cold) analyses were also conducted.	Same-day PM and O_3 were associated most significantly with total deaths. The O_3 effect size estimates for cardiovascular and respiratory deaths were consistently positive (though not significant), and larger in summer. PM's effect size estimates were comparable for warm and cold season for cardiovascular deaths, but larger in warm season for respiratory deaths. NO_2 and SO_2 were not associated with mortality.	3.4% (0.4, 6.4) per 25 µg/m^3 1-h $PM_{2.5}$ increment at 0 d lag; and 7.8% (2.5, 13.2) per 25 µg/m^3 24-h $PM_{2.5}$ increment.
Asia			
Hong et al. (1999) +Inchon, South Korea, 1995-1996 (20 months). PM_{10} mean = 71.2.	Non-accidental total deaths, cardiovascular, and respiratory deaths were examined for their associations with PM_{10}, O_3, SO_2, CO, and NO_2. Data were analyzed using GAM Poisson regression models, adjusting for temperature, relative humidity, and seasonal cycles. Individual pollution lag days from 0 to 5, as well as the average concentrations of previous 5 days were considered.	A greater association with mortality was seen with the 5-day moving average and the previous day's exposure than other lag/averaging time. In the models that included a 5-day moving average of one or multiple pollutants, PM_{10} was a significant predictor of total mortality, but gaseous pollutants were not significant. PM_{10} was also a significant predictor of cardiovascular and respiratory mortality.	Percent excess deaths (t-ratio) per 50 µg/m^3 increase in the 5-day moving average of PM_{10}: 4.1 (0.1, 8.2) for total deaths; 5.1 (0.1, 10.4) for cardiovascular deaths; 14.4 (-3.2, 35.2) for respiratory deaths.
Lee et al. (1999). Seoul and Ulsan, Korea, 1991-1995. TSP (beta attenuation, 93 for Seoul and 72 for Ulsan)	Total mortality series was examined for its association with TSP, SO_2, and O_3, in Poisson GEE (exchangeable correlation for days in the same year), adjusting for season, temperature, and humidity.	All the pollutants were significant predictors of mortality in single pollutant models. TSP was not significant in multiple pollutant models, but SO_2 and O_3 remained significant.	5.1% (3.1, 7.2) for Seoul, and -0.1% (-3.9, 3.9) for Ulsan, per 100 µg/m^3 TSP at avg. of 0, 1, and 2 day lags.
Lee and Schwartz (1999). Seoul, Korea. 1991-1995. TSP mean = $9_{2.5}$.	Total deaths were analyzed for their association with TSP, SO_2, and O_3. A conditional logistic regression analysis with a case-crossover design was conducted. Three-day moving average values (current and two past days) of TSP and SO_2, and 1-hr max O_3 were analyzed separately. The control periods are 7 and 14 days before and/or after the case period. Both unidirectional and bi-directional controls (7 or 7 and 14 days) were examined, resulting in six sets of control selection schemes. Other covariates included temperature and relative humidity.	Among the six control periods, the two unidirectional retrospective control schemes resulted in odds ratios less than 1; the two unidirectional prospective control schemes resulted in larger odds ratios (e.g., 1.4 for 50 ppb increase in SO_2); and bi-directional control schemes resulted in odds ratios between those for uni-directional schemes. SO_2 was more significantly associated with mortality than TSP.	OR for non-accidental mortality per 100 µg/m^3 increase in 3-day average TSP was 1.010 (0.988, 1.032).

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TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

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Asia (cont'd)			
Xu et al. (2000). Shenyang, China, 1992. TSP (430).	Total (non-accidental), CVD, COPD, cancer and other deaths examined for their associations with TSP and SO_2,using Poisson (GAM, and Markov approach to adjust for mortality serial dependence) models, adjusting for seasonal cycles, Sunday indicator, quintiles of temp. and humidity. Ave. pollution values of concurrent and 3 preceding days used. While GAM models were used in the process, the risk estimates presented were for a fully parametric model (i.e., GLM).	Total deaths were associated with TSP and SO_2 in both single and two pollutant models. TSP was significantly associated with CVD deaths, but not with COPD. SO_2 significantly associated with COPD, but not with CVD deaths. Cancer deaths not associated with TSP or SO_2.	Percent total excess deaths per 100 µg/m^3 increase in 0-3 day ave. of TSP = 1.75 (0.65, 2.85); with SO_2 = 1.31 (0.14, 2.49) COPD TSP = 2.6 (-0.58, 5.89); with SO_2 = 0.76 (-2.46, 4.10). CVD TSP = 2.15 (0.56, 3.71); with SO_2 = 1.95 (1.19, 3.74). Cancer TSP = 0.87 (-1.14, 2.53); with SO_2 = 1.07 (-1.05, 3.23). Other deaths TSP = 3.52 (0.82, 6.30); with SO_2 = 2.40 (-0.51, 5.89).
Ostro et al. (1998). Bangkok, Thailand, 1992-1995 PM_{10} (beta attenuation, 65)	Total (non-accidental), cardiovascular, respiratory deaths examined for associations with PM_{10} (separate measurements showed 50% of PM_{10} was $PM_{2.5}$),using Poisson GAM model (only one non-parametric smoothing term in the model) adjusting for seasonal cycles, day-of-week, temp., humidity.	All the mortality series were associated with PM_{10} at various lags. The effects appear across all age groups. No other pollutants were examined.	Total mortality excess risk: 5.1% (2.1, 8.3) per 50 µg/m^3 PM_{10} at 3 d lag (0 and 2 d lags also significant). CVD (3 d ave.) = 8.3 (3.1, 13.8) Resp. (3 d ave.) = 3.0 (-8.4, 15.9)
Cropper et al. (1997). Delhi, India, 1991-1994 TSP (375)	Total (by age group), respiratory and CVD deaths related to TSP, SO_2, and NOx, using GEE Poisson model (to control for autocorrelation), adjusting for seasonal cycles (trigonometric terms), temperature, and humidity. 70% deaths occur before age 65 (in U.S., 70% occur after age 65).	TSP was significantly associated with all mortality series except with the very young (age 0-4) and the "very old" (age >= 65). The results were reported to be unaffected by addition of SO_2 to the model. The authors note that, because those who are affected are younger (than Western cities), more life-years are likely to be lost per person from air pollution impacts.	2.3% (significant at 0.05, but SE of estimate not reported) per 100 µg/m^3 TSP at 2 day lag.
Kwon et al. (2001) +Seoul, South Korea, 1994-1998. PM10 mean = 68.7.	The study was planned to test the hypothesis that patients with congestive heart failure are more susceptible to the harmful effects of ambient air pollution than the general population. GAM Poisson regression models, adjusting for seasonal cycles, temperature, humidity, day-of-week, as well as the case-crossover design, with 7 and 14 days before and after the case period, were applied	The estimated effects were larger among the congestive heart failure patients than among the general population (2.5 ~ 4.1 times larger depending on the pollutants). The case-crossover analysis showed similar results. In two pollutant models, the PM_{10} effects were much lower when CO, NO_2, or SO_2 were included. O_3 had little impact on the effects of the other pollutants.	The RRs for PM_{10} (same-day) using the GAM approach for the general population and for the cohort with congestive heart failure were 1.4% (0.6, 2.2) and 5.8 (-1.1, 13.1), respectively, per 42.1 µg/m^3. Corresponding ORs using the case-crossover approach were 0.1% (-0.9, 1.2) and 7.4% (−2.2, 17.9), respectively.

+ = Used GAM with multiple non-parametric smooths, but have not yet re-analyzed. * = Used S-Plus Default GAM, and have re-analyzed results; GAM = Generalized Additive Model, GEE = Generalized Estimation Equations, GLM = Generalized Linear Model.

TABLE 8A-1 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE MORTALITY EFFECTS STUDIES

Reference, Location, Years, PM Index, Mean or Median, IQR in $\mu g/m^3$.	Study Description: Outcomes, Mean outcome rate, and ages. Concentration measures or estimates. Modeling methods: lags, smoothing, and covariates.	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes.	PM Index, lag, Excess Risk% (95% LCL, UCL), Co-pollutants.
Asia (cont'd)			
Lee et al. (2000) +Seven major cities, Korea. 1991-1997. TSP mean = 77.9.	All non-accidental deaths were analyzed for their associations with TSP, SO_2, NO_2, O_3, and CO using GAM Poisson model adjusting for trend, seasonal cycles, and weather. Pollution relative risk estimates were obtained for each city, and then pooled.	In the results of pooled estimates for multiple pollutant models, the SO_2 relative risks were not affected by addition of other pollutants, whereas the relative risks for other pollutants, including TSP, were. The SO_2 levels in these Korean cities were much higher than the levels observed in the current U.S. For example, the 24-hr mean SO_2 levels in the Korean cities ranged from 12.1 to 31.4 ppb, whereas, in Samet et al.'s 20 largest U.S. cities, the range of 24-hr mean SO_2 levels were 0.7 to 12.8 ppb.	Percent excess deaths for all non-accidental deaths was 1.7% (0.8, 2.6) per 100 $\mu g/m^3$ 2-day moving average TSP.

+ = Used GAM with multiple non-parametric smooths, but have not yet re-analyzed. * = Used S-Plus Default GAM, and have re-analyzed results; GAM = Generalized Additive Model, GEE = Generalized Estimation Equations, GLM = Generalized Linear Model.

APPENDIX 8B

PARTICULATE MATTER-MORBIDITY STUDIES: SUMMARY TABLES

Appendix 8B.1: PM-Cardiovascular Admissions Studies

TABLE 8B-1. ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes. Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
United States			
Samet et al. (2000a,b) 14 US cities 1985-1994, but range of years varied by city PM_{10} ($\mu g/m^3$) mean, median, IQR: Birmingham, AL: 34.8, 30.6, 26.3 Boulder, CO: 24.4, 22.0, 14.0 Canton, OH: 28.4, 25.6, 15.3 Chicago, Il: 36.4, 32.6, 22.4 Colorado Springs, CO: 26.9, 22.9, 11.9 Detroit, MI: 36.8, 32.0, 28.2 Minneapolis/St. Paul, MN: 27.4, 24.1, 17.9 Nashville, TN: 31.6, 29.2, 17.9 New Haven, CT: 29.3h, 26.0, 20.2 Pittsburgh, PA: 36.0, 30.5, 27.4 Provo/Orem, UT: 38.9, 30.3, 22.8 Seattle, WA: 31.0, 26.7, 20.0 Spokane, WA: 45.3, 36.2, 33.5 Youngstown, OH: 33.1, 29.4, 18.6	Daily medicare hospital admissions for total cardiovascular disease, CVD (ICD9 codes 390-429), in persons 65 or greater. Mean CVD counts ranged from 3 to 102/day in the 14 cities. Covariates: SO_2, NO_2, O_3, CO, temperature, relative humidity, barometric pressure. Stats: In first stage, performed city-specific, PM10-ONLY, generalized additive robust Poisson regression with seasonal, weather, and day of week controls. Repeated analysis for days with PM_{10} less than 50 $\mu g/m^3$ to test for threshold. Lags of 0-5 considered, as well as the quadratic function of lags 0-5. Individual cities analyzed first. The 14 risk estimates were then analyzed in several second stage analyses: combining risks across cities using inverse variance weights, and regressing risk estimates on potential effect-modifiers and slopes of PM_{10} on co-pollutants.	City-specific risk estimates for a 10 $\mu g/m^3$ increase in PM_{10} ranged from -1.2% in Canton to 2.2% in Colorado Springs. Across-city weighted mean risk estimate was largest at lag 0, diminishing rapidly at other lags. Only the mean of lags 0 and 1 was significantly associated with CVD. There was no evidence of statistical heterogeniety in risk estimates across cities for CVD. City-specific risk estimates were not associated with the percent of the population that was non-white, living in poverty, college educated, nor unemployed. No evidence was observed that PM_{10} effects were modified by weather. No association was observed between the city-specific PM_{10} risk estimates and the city-specific correlation between PM_{10} and co-pollutants. However, due to the absence of multi-pollutant regression results, it is not clear whether this study demonstrates an independent effect of PM_{10}.	Percent Excess CVD Risk (95% CI), combined over cities per 50 $\mu g/m^3$ change in PM_{10}. PM_{10}: 0 d lag. 5.5% (4.7, 6.2) PM_{10}: 0–1d lag. 6.0% (5.1, 6.8) $PM_{10} < 50$ $\mu g/m^3$: 0–1 d lag. 7.6% (6.0, 9.1)
Zanobetti and Schwartz (2003b)	Statistical reanalysis using GAM with improved convergence criterion (New GAM), GLM with natural splines (GLM NS), and GLM with penalized splines (GLM PS). Lag structure: average of lags 0 and 1.		Default GAM: 5.9% (5.1-6.7) New GAM: 4.95% (3.95-5.95) GLM NS: 4.8% (3.55-6.0) GLM PS: 5.0% (4.0-5.95)

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
United States (cont'd)			
Janssen et al. (2002) 14 U.S. cities studied in Samet et al. (2000a,b) above PM_{10} ($\mu g/m^3$) — Mean Summer/Winter — Ratio Birmingham 40.0/27.4 0.69 Boulder* 26.8/36.3 1.35 Canton 36.6/25.8 0.70 Chicago 42.5/30.4 0.71 Colorado Springs* 21.3/37.3 1.75 Detroit 42.8/32.8 0.77 Minneapolis 30.5/23.0 0.75 Nashville 40.1/31.9 0.80 New Haven 30.3/31.6 1.04 Pittsburgh 46.6/29.4 0.63 Seattle* 23.8/43.3 1.82 Spokane* 32.7/42.2 1.29 Provo-Urem* 31.4/66.3 2.11 Youngstown 40.7/30.1 0.74	Examined same database as Samet et al. (2000a,b) to evaluate whether differences in prevalence in air conditioning (AC) and/or the contribution of different sources to total PM_{10} emissions could partially explain the observed variability in exposure effect relations. Variables included 24-hr means of temperature. Cities were characterized and analyzed as either winter or nonwinter peaking. Rations between mean concentrations during summer (June, July August) and winter (January, February, March) were calculated. (*Winter peaking PM_{10} concentration.)	Analysis of city groups of winter peaking, PM_{10} and nonwinter peaking PM_{10} yielded coefficients for CVD-related hospitalization admissions that decreased significantly with increasing percentage of central AC for both city groups. Four source related variables coefficients for hospital admissions for CVD increased significantly with increasing percentage of PM_{10} from highway vehicles, highway diesels, oil combustion, metal processing, increasing population, and vehicle miles traveled (VMT) per sq mile and with decreasing percentage of PM_{10} from fugitive dust. For COPD and pneumonia association were less significant but the pattern of association were similar to that for CVD.	**Homes with AC** β CVD % change (SE) All cities −15.2 (14.8) Nonwinter peak cities −50.3** (17.4) Winter peak cities −51.7** (13.8) **Source PM_{10} from highway vehicles** % change (SE) β CVD 58.0* (9.9) [**p <0.05]
Zanobetti and Schwartz (2003b)	Statistical reanalysis of Janssen et al., 2002 findings using GLM with natural splines (GLM NS), and GLM with penalized splines (GLM PS). Lag structure: average of lags 0 and 1.	Zanobetti and Schwartz (2003b) reanalyzed the main findings from this study using alternative methods for controlling time and weather covariates. While the main conclusions of the study were not significantly altered, some changes in results are worth noting. The effect of air conditioning use on PM10 effect estimates was less pronounced and no longer statistically significant for the winter PM10-peaking cities using natural splines or penalized splines in comparison to the original Janssen et al. GAM analysis. The effect of air conditioning remained significant for the non-winter PM10-peaking cities. The significance of highway vehicles and diesels on PM10 effect sizes remained significant, as did oil combustion.	**Homes with AC** β CVD % change (SE) All cities GLM NS: −13.55 (14.9) GLM PS: -12.0 (14.1) Nonwinter peaking cities GLM NS: −44.1** (20.15) GLM PS: -38.4** (17.8) Winter peaking cities GLM NS: −6.1 (40.3) GLM PS: -41.5 (39.6) **Source PM_{10} from highway vehicles** % change (SE) β CVD GLM NS: 51.1** (14.7) GLM PS: 35.1** (14.3) [**p <0.05]

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
United States (cont'd)			
Zanobetti et al. (2000b) 10 US cities 1986-1994 PM_{10} ($\mu g/m^3$) median, IQR: Canton, OH: 26, 15 Birmingham, AL: 31, 26 Chicago, Il: 33, 23 Colorado Springs, CO: 23, 13 Detroit, MI: 32, 28 Minneapolis/St. Paul, MN: 24, 18 New Haven, CT: 26, 21 Pittsburgh, PA: 30, 28 Seattle, WA: 27, 21 Spokane, WA: 36, 34	Derived from the Samet et al. (2000a,b) study, but for a subset of 10 cities. Daily hospital admissions for total cardiovascular disease, CVD (ICD9 codes 390-429), in persons 65 or greater. Median CVD counts ranged from 3 to 103/day in the 10 cities. Covariates: SO_2, O_3, CO, temperature, relative humidity, barommetric pressure. Stats: In first stage, performed single-pollutant generalized additive robust Poisson regression with seasonal, weather, and day of week controls. Repeated analysis for days with PM_{10} less than 50 $\mu g/m^3$ to test for threshold. Lags of 0-5 considered, as well as the quadratic function of lags 0-5. Individual cities analyzed first. The 10 risk estimates were then analyzed in several second stage analyses: combining risks across cities using inverse variance weights, and regressing risk estimates on potential effect-modifiers and pollutant confounders.	Same basic pattern of results as in Samet et al. (2000a,b). For distributed lag analysis, lag 0 had largest effect, lags 1 and 2 smaller effects, and none at larger lags. City-specific slopes were independent of percent poverty and percent non-white. Effect size increase when data were restricted to days with PM_{10} less than 50 $\mu g/m^3$. No multi-pollutant models reported; however, no evidence of effect modification by co-pollutants in second stage analysis. As with Samet et al. 2000., it is not clear whether this study demonstrates an independent effect of PM_{10}. This study used the old GAM model. Results have not been explicitly reanalyzed, but note that the 14 cities noted above in Zanobetti and Schwartz (2003b) include these 10 cities.	Percent Excess Risk (SE) combined over cities: Effects computed for 50 $\mu g/m^3$ change in PM_{10}. PM_{10}: 0 d. 5.6 (4.7, 6.4) PM_{10}: 0–1 d. 6.2 (5.4, 7.0) $PM_{10} < 50$ $\mu g/m^3$: 0–1 d. 7.8 (6.2, 9.4)
Schwartz (1999) 8 US metropolitan counties 1988-1990 median, IQR for PM_{10} ($\mu g/m^3$): Chicago, IL: 35, 23 Colorado Springs, CO: 23, 14 Minneapolis, MN: 28, 15 New Haven, CT: 37, 25 St. Paul, MN: 34, 23 Seattle, WA: 29, 20 Spokane, WA: 37, 33 Tacoma, WA: 37, 27	Daily hospital admissions for total cardiovascular diseases (ICD9 codes 390-429) among persons over 65 years. Median daily hospitalizations: 110, 3, 14, 18, 9, 22, 6, 7, alphabetically by city. Covariates: CO, temperature, dewpoint temp. Stats: robust Poisson regression after removing admission outliers; generalized additive models with LOESS smooths for control of trends, seasons, and weather. Day of week dummy variables. Lag 0 used for all covariates.	In single-pollutant models, similar PM_{10} effect sizes obtained for each county. Five of eight county-specific effects were statistically significant, as was the PM_{10} effect pooled across locations. CO effects significant in six of eight counties. The PM_{10} and CO effects were both significant in a two pollutant model that was run for five counties where the PM_{10}/CO correlation was less than 0.5. Results reinforce those of Schwartz, 1997. This study used the old GAM model. No reanalysis has been reported.	Percent Excess Risk (95% CI): Effects computed for 50 $\mu g/m^3$ change in PM_{10}. PM_{10}: 0d. Individual counties: Chicago: 4.7 (2.6, 6.8) CO Spng: 5.6 (−6.8, 19.0) Minneap: 4.1 (−3.6, 12.5) New Hav: 5.8 (2.1, 9.7) St. Paul: 8.6 (2.9, 14.5) Seattle: 3.6 (−0.1, 7.4) Spokane: 6.7 (0.9, 12.8) Tacoma: 5.3 (3.1, 7.6) Pooled: 5.0 (3.7, 6.4) 3.8 (2.0, 5.5) w. CO

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
United States (cont'd)			
Linn et al. (2000) Los Angeles 1992-1995 mean, SD: $PM_{10\ est}$ (µg/m^3): 45, 18	Hospital admissions for total cardiovascular diseases (CVD), congestive heart failure (CHF), myocardial infarction (MI), cardiac arrhythmia (CA) among all persons 30 years and older, and by sex, age, race, and season. Mean hospital admissions for CVD: 428. Covariates: CO, NO_2, O_3, temperature, rainfall. Daily gravimetric PM_{10} estimated by regression of every sixth day PM_{10} on daily real-time PM_{10} data collected by TEOM. Poisson regression with controls for seasons and day of week. Reported results for lag 0 only. Results reported as Poisson regression coefficients and their standard errors. The number of daily CVD admissions associated with the mean PM_{10} concentration can be computed by multiplying the PM_{10} coefficient by the PM_{10} mean and then exponentiating. Percent effects are calculated by dividing this result by the mean daily admission count for CVD.	In year-round, single-pollutant models, significant effects of CO, NO_2, and PM_{10} on CVD were reported. PM_{10} effects appeared larger in winter and fall than in spring and summer. No consistent differences in PM_{10} effects across sex, age, and race. CO risk was robust to including PM_{10} in the model; no results presented on PM_{10} robustness to co-pollutants. This study did not use the GAM model in developing its main findings.	% increase with PM_{10} change of 50 µg/m^3: $PM_{10\ est}$: 0 d. CVD ages 30+ 3.25% (2.04, 4.47) MI ages 30+ 3.04% (0.06, 6.12) CHF ages 30+ 2.02% (−0.94, 5.06) CA ages 30+ 1.01% (−1.93, 4.02)
Morris and Naumova (1998) Chicago, IL 1986-1989 mean, median, IQR, 75th percentile: PM_{10} (µg/m^3): 41, 38, 23, 51	Daily hospital admissions for congestive heart failure, CHF (ICD9 428), among persons over 65 years. Mean hospitalizations: 34/day. Covariates: O_3, NO_2, SO_2, CO, temperature, relative humidity. Gases measured at up to eight sites; daily PM_{10} measured at one site. Stats: GLM for time series data. Controlled for trends and cycles using dummy variables for day of week, month, and year. Residuals were modeled as negative binomial distribution. Lags of 0-3 days examined.	CO was only pollutant statistically significant in both single- and multi-pollutant models. Exposure misclassification may have been larger for PM_{10} due to single site. Results suggest effects of both CO and PM_{10} on congestive heart failure hospitalizations among elderly, but CO effects appear more robust. This study did not use the GAM model.	Percent Excess Risk (95% CI) per 50 µg/m^3 change in PM_{10}. PM_{10}: 0 d. 3.92% (1.02, 6.90) 1.96% (−1.4, 5.4) with 4 gaseous pollutants

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR µg/m^3	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
United States (cont'd)			
Schwartz (1997) Tucson, AZ 1988-1990 mean, median, IQR: PM_{10} (µg/m^3): 42, 39, 23	Daily hospital admissions for total cardiovascular diseases (ICD9 codes 390-429) among persons over 65 years. Mean hospitalizations: 13.4/day. Covariates: O_3, NO_2, CO, SO_2, temperature, dewpoint temperature. Gases measured at multiple sites; daily PM_{10} at one site. Stats: robust Poisson regression; generalized additive model with LOESS smooth for controlling trends and seasons, and regression splines to control weather. Lags of 0-2 days examined.	Both PM_{10} (lag 0) and CO significantly and independently associated with admissions, whereas other gases were not. Sensitivity analyses reinforced these basic results. Results suggest independent effects of both PM_{10} and CO for total cardiovascular hospitalizations among the elderly. This study used the old GAM model. No reanalysis has been reported.	Percent Excess Risk (95% CI) per 50 µg/m^3 change in PM_{10}. PM_{10}: 0 d. 6.07% (1.12, 1.27) 5.22% (0.17, 10.54) w. CO
Gwynn et al (2000) Buffalo, NY mn/max PM_{10} = 24.1/90.8 µg/m^3 SO_4^- = 2.4/3.9 H^+ = 36.4/38.2 nmol/m^3 CoH = 0.2/0.9 10^{-3} ft	Air pollution health effects associations with total, respiratory, and CVD hospital admissions (HA's) examined using Poisson model controlling for weather, seasonality, long-wave effects, day of week, holidays.	Positive, but non-significant assoc. found between all PM indices and circulatory hospital admissions. Addition of gaseous pollutants to the model had minimal effects on the PM RR estimates. This study used the old GAM model. No reanalysis has been reported.	Percent excess CVD HA risks (95% CI) per PM_{10} = 50 µg/m^3; SO_4 = 15 µg/m^3; H^+ = 75 nmoles/m^3; COH = 0.5 units/1,000 ft: PM_{10} (lag 3) = 5.7% (−3.3, 15.5) SO_4 (lag 1) = 0.1% (−0.1, 0.4) H^+ (lag 0) = 1.9% (−0.3, 4.2) COH (lag 1) = 2.2% (−1.9, 6.3)
Lippmann et al. (2000) Detroit (Wayne County), MI 1992-1994 mean, median, IQR: $PM_{2.5}$ (µg/m^3): 18, 15, 11 PM_{10} (µg/m^3): 31, 28, 19 $PM_{10-2.5}$ (µg/m^3): 13, 12, 9	Various cardiovascular (CVD)-related hospital admissions (HA's) for persons 65+ yr. analyzed, using GAM Poisson models, adjusting for season, day of week, temperature, and relative humidity. The air pollution variables analyzed were: PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, sulfate, H^+, O_3, SO_2, NO_2, and CO. However, this study site/period had very low acidic aerosol levels. As noted by the authors 85% of H^+ data was below detection limit (8 nmol/m^3).	For heart failure, all PM metrics yielded significant associations. Associations for IHD, dysrhythmia, and stroke were positive but generally non-sig. with all PM indices. Adding gaseous pollutants had negligible effects on various PM metric RR estimates. The general similarity of the $PM_{2.5}$ and $PM_{10-2.5}$ effects per µg/m^3 in this study suggest similarity in human toxicity of these two inhalable mass components in study locales/periods where $PM_{2.5}$ acidity not usually present. However, small sample size limits power to distinguish between pollutant-specific effects.	Percent excess CVD HA risks (95% CI) per 50 µg/m^3 PM_{10}, 25 µg/m^3 $PM_{2.5}$ and $PM_{10-2.5}$: IHD: $PM_{2.5}$ (lag 2) 4.3 (−1.4, 10.4) PM_{10} (lag 2) 8.9 (0.5, 18.0) $PM_{10-2.5}$ (lag 2) 10.5 (2.7, 18.9) Dysrhythmia: $PM_{2.5}$ (lag 1) 3.2 (−6.5, 14.0) PM_{10} (lag 1) 2.9 (−6.8, 13.7) $PM_{10-2.5}$ (lag 0) 0.2 (−12.2, 14.4) Heart Failure: $PM_{2.5}$ (lag 1) 9.1 (2.4, 16.2) PM_{10} (lag 0) 9.7 (0.2, 20.1) $PM_{10-2.5}$ (lag 0) 5.2 (−3.3, 14.5) Stroke: $PM_{2.5}$ (lag 0) 1.8 (−5.3, 9.4) PM_{10} (lag 1) 4.8 (−5.5, 16.2) $PM_{10-2.5}$ (lag 1) 4.9 (−4.7, 15.5)

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
United States (cont'd)			
Ito 2003 Detroit (Wayne County), MI	Statistical reanalysis using GAM with improved convergence criterion (New GAM), and GLM with natural splines (GLM NS). Same model structure as before.		IHD: New GAM: 8.0% (-0.3-17.1) GLM NS: 6.2% (-2.0-15.0) New GAM: 3.65% (-2.05-9.7)* GLM NS: 3.0% (-2.7-9.0)* New GAM: 10.2% (2.4-18.6)** GLM NS: 8.1% (0.4-16.4)** Dysrhythmias: New GAM: 2.8% (-10.9-18.7) GLM NS: 2.0% (-11.7-17.7) New GAM: 3.2% (-6.6-14.0)* GLM NS: 2.6% (-7.1-13.3)* New GAM: 0.1% (-12.4-14.4)** GLM NS: 0.0% (-12.5-14.3)** Heart Failure: New GAM: 9.2% (-0.3-19.6) GLM NS: 8.4% (-1.0-18.7) New GAM: 8.0% (1.4-15.0)* GLM NS: 6.8% (0.3-13.8)* New GAM: 4.4% (-4.0-13.5)** GLM NS: 4.9% (-3.55-14.1)**

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
Moolgavkar (2000b) Three urban counties: Cook, IL; Los Angeles, CA; Maricopa, AZ. 1987-1995 Pollutant median, IQR: Cook: PM_{10}: 35, 22 LA: PM_{10}: 44, 26 $PM_{2.5}$: 22, 16 Maricopa: PM_{10}: 41, 19	Analysis of daily hospital admissions for total cardiovascular diseases, CVD, (ICD9 codes 390-429) and cerebrovascular diseases, CRD, (ICD9 430-448) among persons aged 65 and over. For Los Angeles, a second age group, 20-64, was also analyzed. Median daily CVD admissions were 110, 172, and 33 in Cook, LA, and Maricopa counties, respectively. PM_{10} available only every sixth day in LA and Maricopa counties. In LA, every-sixth-day $PM_{2.5}$ also was available. Covariates: CO, NO_2, O_3, SO_2, temperature, relative humidity. Stats: generalized additive Poisson regression, with controls for day of week and smooth temporal variability. Single-pollutant models estimated for individual lags from 0 to 5. Two-pollutant models also estimated, with both pollutants at same lag.	In single-pollutant models in Cook and LA counties, PM was significantly associated with CVD admissions at lags 0, 1, and 2, with diminishing effects over lags. $PM_{2.5}$ also was significant in LA for lags 0 and 1. For the 20-64 year old age group in LA, risk estimates were similar to those for 65+. In Maricopa county, no positive PM_{10} associations were observed at any lag. In two-pollutant models in Cook and LA counties, the $PM_{10}/PM_{2.5}$ risk estimates diminished and/or were rendered non-significant. Little evidence observed for associations between CRD admissions and PM. These results suggest that PM is not independently associated with CVD or CRD hospital admissions.	Percent Excess CVD Risk (95% CI) Effects computed for 50 μg/m^3 change in PM_{10} and 25 μg/m^3 change in $PM_{2.5}$. Cook 65+: PM_{10}, 0 d. 4.2 (3.0, 5.5) PM_{10}, 0 d. w/NO_2. 1.8 (0.4, 3.2) LA 65+: PM_{10}, 0 d. 3.2 (1.2, 5.3) PM_{10}, 0 d. w/CO −1.8 (−4.4, 0.9) $PM_{2.5}$, 0 d. 4.3 (2.5, 6.1) $PM_{2.5}$, 0 d. w/CO 0.8 (−1.3, 2.9) LA 20−64 years old: PM_{10}, 0 d. 4.4 (2.2, 6.7) PM_{10}, 0 d. w/CO 1.4 (−1.3, 4.2) $PM_{2.5}$, 0 d. 3.5 (1.8, 5.3) $PM_{2.5}$, 0 d., w/CO) 2.3 (−0.2, 4.8) Maricopa: PM_{10}, 0 d. −2.4 (−6.9, 2.3)

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
United States (cont'd)			
Moolgavkar (2003)	Statistical reanalysis using GAM with improved convergence criterion (New GAM), and GLM with natural splines (GLM NS). New analyses were run with variable and in some cases more extensive control of time than in original analysis.		Cook County, IL: New GAM100df: 4.05% (2.9-5.2) GLM NS100df: 4.25% (3.0-5.5) Los Angeles County, CA: New GAM30df: 3.35% (1.2-5.5) New GAM100df: 2.7% (0.6-4.8) GLM NS100df: 2.75% (0.1-5.4) New GAM30df: 3.95% (2.2-5.7)* New GAM100df: 2.9% (1.2-4.6)* GLM nspline100df: 3.15% (1.1-5.2)*
Zanobetti et al. (2000a) Cook County, IL 1985-1994 Median, IQR: PM_{10} ($\mu g/m^3$): 33, 23	Total cardivascular hospital admissions in persons 65 and older (ICD 9 codes390-429) in relation to PM_{10}. Data were analyzed to examine effect modification by concurrent or preexisting cardiac and/or respiratory conditions, age, race, and sex. No co-pollutants included.	Evidence seen for increased CVD effects among persons with concurrent respiratory infections or with previous admissions for conduction disorders.	Percent Excess CVD Risk (95% CI) Effects computed for 50 $\mu g/m^3$ PM_{10}, 0-1 D. AVG. CVD: 6.6 (4.9-8.3)

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
Tolbert et al. (2000a) Atlanta Period 1: 1/1/93-7/31/98 Mean, median, SD: PM_{10} (µg/m^3): 30.1, 28.0, 12.4 Period 2: 8/1/98-8/31/99 Mean, median, SD: PM_{10} (µg/m^3): 29.1, 27.6, 12.0 $PM_{2.5}$ (µg/m^3): 19.4, 17.5, 9.35 CP (µg/m^3): 9.39, 8.95, 4.52 10-100 nm PM counts (count/cm^3): 15,200, 10,900, 26,600 10-100 nm PM surface area (um^2/cm^3): 62.5, 43.4, 116 $PM_{2.5}$ soluble metals (µg/m^3): 0.0327, 0.0226, 0.0306 $PM_{2.5}$ Sulfates (µg/m^3): 5.59, 4.67, 3.6 $PM_{2.5}$ Acidity (µg/m^3): 0.0181, 0.0112, 0.0219 $PM_{2.5}$ organic PM (µg/m^3): 6.30, 5.90, 3.16 $PM_{2.5}$ elemental carbon (µg/m^3): 2.25, 1.88, 1.74	Preliminary analysis of daily emergency department (ED) visits for dysrhythmias, DYS, (ICD 9 code 427) and all cardiovascular diseases, CVD, (codes 402, 410-414, 427, 428, 433-437, 440, 444, 451-453) for persons aged 16 and older in the period before (Period 1) and during (Period 2) the Atlanta superstation study. ED data analyzed here from just 18 of 33 participating hospitals; numbers of participating hospitals increased during period 1. Mean daily ED visits for dysrhythmias and all CVD in period 1 were 6.5 and 28.4, respectively. Mean daily ED visits for dysrhythmias and all CVD in period 2 were 11.2 and 45.1, respectively. Covariates: NO_2, O_3, SO_2, CO temperature, dewpoint, and, in period 2 only, VOCs. PM measured by both TEOM and Federal Reference Method; unclear which used in analyses. For epidemiologic analyses, the two time periods were analyzed separately. Poisson regression analyses were conducted with cubic splines for time, temperature and dewpoint. Day of week and hospital entry/exit indicators also included. Pollutants were treated a-priori as three-day moving averages of lags 0, 1, and 2. Only single-pollutant results reported.	In period 1, significant negative association (p=0.02) observed between CVD and 3-day average PM_{10}. There was ca. 2% drop in CVD per 10 µg/m^3 increase in PM_{10}. CVD was positively associated with NO_2 (p=0.11) and negatively associated with SO_2 (p=0.10). No association observed between dysrhythmias and PM_{10} in period 1. However, dysrhythmias were positively associated with NO_2 (p=0.06). In period 2, i.e., the first year of operation of the superstation, no associations seen with PM_{10} or $PM_{2.5}$. However, significant positive associations observed between CVD and elemental carbon (p=0.005) and organic matter (p=0.02), as well as with CO (p=0.001). For dysrhythmias, significant positive associations observed with elemental carbon (p=0.004), CP (p=0.04), and CO (p=0.005). These preliminary results should be interpreted with caution given the incomplete and variable nature of the databases analyzed.	Percent Excess Risk (p-value): Effects computed for 50 µg/m^3 change in PM_{10}; 25 µg/m^3 for CP and $PM_{2.5}$; 25,000 counts/cm^3 for 10-100 nm counts. Period 1: PM_{10}, 0-2 d. avg. CVD: −8.2 (0.02) DYS: 4.6 (0.58) Period 2: 0-2 d. avg. in all cases CVD % effect; DYS % effect: PM_{10}: 5.1 (−7.9, 19.9); 13.1 (−14.1, 50.0) $PM_{2.5}$: 6.1 (−3.1, 16.2); 6.1 (−12.6, 28.9) CP: 17.6 (−4.6, 45.0); 53.2 (2.1, 129.6) 10-100 nm counts: −11.0 (0.17); 3.0 (0.87)

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
Canada			
Burnett et al. (1995) Ontario, Canada 1983-1988 Sulfate Mean: 4.37 µg/m^3 Median: 3.07 µg/m^3 95th percentile: 13 µg/m^3	168 Ontario hospitals. Hospitalizations for coronary artery disease, CAD (ICD9 codes 410,413), cardiac dysrhythmias, DYS (code 427), heart failure, HF (code 428), and all three categories combined (total CVD). Mean total CVD rate: 14.4/day. 1986 population of study area: 8.7 million. All ages, <65, >=65. Both sexes, males, females. Daily sulfates from nine monitoring stations. Ozone from 22 stations. Log hospitalizations filtered with 19-day moving average prior to GEE analysis. Day of week effects removed. 0-3 day lags examined. Covariates: ozone, ozone^2, temperature, temperature^2. Linear and quadratic sulfate terms included in model.	Sulfate lagged one day significantly assoc. with total CVD admissions with and without ozone in the model. Larger associations observed for coronary artery disease and heart failure than for cardiac dysrhythmias. Suggestion of larger associations for males and the sub-population 65 years old and greater. Little evidence for seasonal differences in sulfate effects after controlling for covariates.	Effects computed for 95th percentile change in SO_4 SO_4, 1d, no covariates: Total CVD: 2.8 (1.8, 3.8) CAD: 2.3 (0.7, 3.8) DYS: 1.3 (−2.0, 4.6) HF: 3.0 (0.6, 5.3) Males: 3.4 (1.8, 5.0) Females: 2.0 (0.2, 3.7) <65: 2.5 (0.5, 4.5) >=65: 3.5 (1.9, 5.0) SO_4, 1d, w. temp and O_3: Total CVD: 3.3 (1.7,4.8)
Burnett et al. (1997a) Canada's 10 largest cities 1981-1994 COH daily maximum Mean: 0.7 10^3 ln feet Median: 0.6 10^3 ln feet 95th percentile: 1.5 10^3 ln feet	Daily hospitalizations for congestive heart failure (ICD9 code 427) for patients over 65 years at 134 hospitals. Average hospitalizations: 39/day. 1986 population of study area: 12.6 million. Regressions on air quality using generalized estimating equations, controlling for long-term trends, seasonality, day of week, and inter-hospital differences. Models fit monthly and pooled over months. Log hospitalizations filtered with 19-day moving average prior to GEE analysis. 0-3 day lags examined. Covariates: CO, SO_2, NO_2, O_3, temperature, dewpoint temperature.	COH significant in single-pollutant models with and without weather covariates. Only *ln*CO and *ln* NO_2 significant in multi-pollutant models. COH highly colinear with CO and NO_2. Suggests no particle effect independent of gases. However, no gravimetric PM data were included.	Effects computed for 95% change in COH: 0 d lag: 5.5% (2.5, 8.6) 0 d lag w/weather: 4.7% (1.3, 8.2) 0 d lag w/CO, NO_2, SO_2, O_3: −2.26 (−6.5, 2.2)

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
Canada (cont'd)			
Burnett et al. (1997b) Metro-Toronto, Canada 1992-1994 Pollutant: mean, median, IQR: COH (10^3 ln ft): 0.8, 0.8, 0.6 H+ (nmol/m^3): 5, 1, 6 SO_4 (nmol/m^3): 57, 33, 57 PM_{10} (µg/m^3): 28, 25, 22 $PM_{2.5}$ (µg/m^3): 17, 14, 15 $PM_{10\text{-}2.5}$ (µg/m^3): 12, 10, 7	Daily unscheduled cardiovascular hospitalizations (ICD9 codes 410-414,427, 428) for all ages. Average hospital admissions: 42.6/day. Six cities of metro-Toronto included Toronto, North York, East York, Etobicoke, Scarborough, and York, with combined 1991 population of 2.36 million. Used same stat model as in Burnett et al., 1997c. 0- 4 day lags examined, as well as multi-day averages. Covariates: O_3, NO_2, SO_2, CO, temperature, dewpoint temperature.	Relative risks > 1 for all pollutants in univariate regressions including weather variables; all but H+ and FP statistically significant. In multivariate models, the gaseous pollutant effects were generally more robust than were particulate effects. However, in contrast to Burnett et al. (1997A), COH remained significant in multivariate models. Of the remaining particle metrics, CP was the most robust to the inclusion of gaseous covariates. Results do not support independent effects of FP, SO_4, or H+ when gases are controlled.	Percent excess risk (95% CI) per 50 µg/m^3 PM_{10}, 25 µg/m^3 $PM_{2.5}$ and $PM_{10\text{-}2.5}$, and IQR for other indicators. COH: 0–4 d. 6.2 (4.0, 8.4) 5.9 (2.8, 9.1) w. gases H+: 2–4 d. 2.4 (0.4, 4.5) 0.5 (–1.6, 2.7) w. gases SO_4: 2–4 d. 1.7 (–0.4, 3.9) –1.6 (–4.4, 1.3) w. gases PM_{10} : 1–4 d. 7.7 (0.9, 14.8) –0.9 (–8.3, 7.1) w. gases $PM_{2.5}$: 2–4 d. 5.9 (1.8, 10.2) –1.1 (–7.8, 6.0) w. gases $PM_{10\text{-}2.5}$: 0–4 d. 13.5 (5.5, 22.0) 8.1 (–1.3, 18.3) w. gases

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
Canada (cont'd)			
Burnett et al. (1999) Metro-Toronto, Canada 1980-1994 Pollutant: mean, median, IQR: FP_{est} (µg/m^3): 18, 16, 10 CP_{est} (µg/m^3): 12, 10, 8 $PM_{10\ est}$ (µg/m^3): 30, 27, 15	Daily hospitalizations for dysrhythmias, DYS (ICD9 code 427; mean 5/day); heart failure, HF (428; 9/d); ischemic heart disease, IHD (410-414; 24/d); cerebral vascular disease, CVD (430-438; 10/d); and diseases of the peripheral circulation, DPC (440-459; 5/d) analyzed separately in relation to environmental covariates. Same geographic area as in Burnett et al., 1997b. Three size-classified PM metrics were estimated, not measured, based on a regression on TSP, SO_4, and COH in a subset of every 6th-day data. Generalized additive models used and non-parametric LOESS prefilter applied to both pollution and hospitalization data. Day of week controls. Tested 1-3 day averages of air pollution ending on lags 0-2. Covariates: O_3, NO_2, SO_2, CO, temperature, dewpoint temperature, relative humidity.	In univariate regressions, all three PM metrics were associated with increases in cardiac outcome (DVS, HF, IHD). No associations with vascular outcomes, except for CPest with DPC. In multi-pollutant models, PM effects estimates reduced by variable amounts (often >50%) for specific endpoints and no statistically significant (at $p<0.05$) PM associations seen with any cardiac or circulatory outcome (results not shown). Use of estimated PM metrics limits interpretation of pollutant-specific results. However, results suggest that linear combination of TSP, SO_4, and COH does not have a strong independent association with cardiovascular admissions when full range of gaseous pollutants also modeled.	Single pollutant models: Percent excess risk (95% CI) per 50 µg/m^3 PM_{10}; 25 µg/m^3 $PM_{2.5}$; and 25 µg/m^3 $PM_{10-2.5}$. All cardiac HA (lags 2-5 d): $PM_{2.5}$ 1-poll = 8.1 (2.45, 14.1) $PM_{2.5}$ w/4 gases = −1.6 (−10.4, 8.2); w/CO = 4.60 (−3.39, 13.26) PM_{10} 1-poll = 12.07 (1.43, 23.81) w/4 gases = −1.40 (−12.53, 11.16) w/CO = 10.93 (0.11, 22.92) $PM_{10-2.5}$ 1-poll = 20.46 (8.24, 34.06) w/4 gases = 12.14 (−1.89, 28.2); w/CO = 19.85 (7.19, 34.0) DYS: FP_{est} (0 d): 6.1 (1.9, 10.4) CP_{est} (0 d): 5.2 (−0.21, 1.08) $PM_{10\ est}$: (0 d): 8.41 (2.89, 14.2) HF: FP_{est} (0−2 d): 6.59 (2.50, 10.8) CP_{est} (0−2 d): 7.9 (2.28, 13) $PM_{10\ est}$ (0−2 d): 9.7 (4.2, 15.5) IHD: FP_{est} (0−2 d): 8.1 (5.4, 10.8) CP_{est} (0 d): 3.7 (1.3, 6.3) $PM_{10\ est}$ (0−1 d): 8.4 (5.3, 11.5)

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
Canada (cont'd)			
Stieb et al. (2000) Saint John, Canada 7/1/92-3/31/96 mean and S.D.: PM_{10} (µg/m^3): 14.0, 9.0 $PM_{2.5}$ (µg/m^3): 8.5, 5.9 **HOSPITAL ADMISSIONS** H+ (nmol/m^3): 25.7, 36.8 Sulfate (nmol/m^3): 31.1, 29.7 COH mean (10^3 ln ft): 0.2, 0.2 COH max (10^3 ln ft): 0.6, 0.5	Study of daily emergency department (ED) visits for angina/myocardial infarction (mean 1.8/day), congestive heart failure (1.0/day), dysrhythmia/conduction disturbance (0.8/day), and all cardiac conditions (3.5/day) for persons of all ages. Covariates included CO, H_2S, NO_2, O_3, SO_2, total reduced sulfur (TRS), a large number of weather variables, and 12 molds and pollens. Stats: generalized additive models with LOESS prefiltering of both ED and pollutant variables, with variable window lengths. Also controlled for day of week and LOESS-smoothed functions of weather. Single-day, and five day average, pollution lags tested out to lag 10. The strongest lag, either positive or negative, was chosen for final models. Both single and multi-pollutant models reported. Full-year and May-Sep models reported.	In single-pollutant models, significant positive associations observed between all cardiac ED visits and PM_{10}, $PM_{2.5}$, H_2S, O_3, and SO_2. Significant negative associations observed with H+, sulfate, and COH max. PM results were similar when data were restricted to May-Sep. In multi-pollutant models, no PM metrics were significantly associated with all cardiac ED visits in full year analyses, whereas both O_3 and SO_2 were. In the May-Sep subset, significant negative association found for sulfate. No quantitative results presented for non-significant variables in these multi-pollutant regressions. In cause-specific, single-pollutant models, PM tended to be positively associated with dysrhythmia/conductive disturbances but negatively associated with congestive heart failure (no quantitative results presented). The objective decision rule used for selecting lags reduced the risk of data mining; however, the biological plausibility of lag effects beyond 3-5 days is open to question. Rich co-pollutant data base. Results imply no effects of PM independent of co-pollutants.	Percent Excess Risk (p-value) computed for 50 µg/m^3 PM_{10}, 25 µg/m^3 $PM_{2.5}$ and mean levels of sulfate and COH. Full year results for all cardiac conditions, single pollutant models: PM_{10}: 3d. 29.3 (P=0.003) $PM_{2.5}$: 3d. 14.4 (P=0.055) H+: 4–9 d. avg. –1.8 (0.010) Sulfate: 4d. –6.0 (0.001) COH max: 7d. –5.4 (0.027) Full year results for all cardiac conditions, multi-pollutant models: No significant PM associations.

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
Europe			
Le Tertre et al. (2002) Eight-City - APHEA 2 Study mean (SD) PM_{10} µg/m^3 Barcelona - 1/94-12/96 55.7 (18.4) Birmingham - 3/92-12/94 24.8 (13.1) London - 1/92-12/94 28.4 (12.3) Milan - No PM_{10} Netherlands - 1/92-9/95 39.5 (19.9) Paris - 1/92-9/96 PM_{13} - 22.7 (10.8) Rome - No PM_{10} Stockholm - 3/94-12/96 15.5 (7.2)	Examined the association between measures of PM to include PM_{10} and hospital admissions for cardiac causes in eight European cities with a combined population of 38 million. Examined age factors and ischemic heart disease and studies also stratified by age using autoregressive Poisson models controlled for long-term trends, season, influenza, epidemics, and meteorology, as well as confounding by other pollutants. In a second regression examined, pooled city-specific results for sources of heterogeneity.	Pooled results were reported for the cardiac admissions results in table format. City-specific and pooled results were depicted in figures only. Found a significant effect of PM_{10} and black smoke on admissions for cardiac causes (all ages) and cardiac causes and ischemic heart disease for people over 65 years with the impact of PM_{10} per unit of pollution being half that found in the United States. PM_{10} did not seem to be confounded by O_3 or SO_2. The effect was reduced when CO was incorporated in the regression model and eliminated when controlling for NO_2. There was little evidence of an impact of particles on hospital admissions for ischemic heart disease for people below 65 years or stroke for people over 65 years. The authors state results were consistent with a role for traffic exhaust/diesel in Europe.	For a 10 µg/m^3 increase in PM_{10} Cardiac admissions/all ages 0.5% (0.2, 0.8) Cardiac admissions/over 65 years 0.7% (0.4, 1.0) Ischemic heart disease/over 65 years 0.8% (0.3, 1.2) For cardiac admissions for people over 65 years: All the city-specific estimates were positive with London, Milan, and Stockholm significant at the 5% level.
Atkinson et al. (1999b) Greater London, England 1992-1994 Pollutant: mean, median, 90-10 percentile range: PM_{10} (µg/m^3): 28.5, 24.8, 30.7 Black Smoke (µg/m^3): 12.7, 10.8, 16.1	Daily emergency hospital admissions for total cardiovascular diseases, CVD (ICD9 codes 390-459), and ischemic heart disease, IHD (ICD9 410-414), for all ages, for persons less than 65, and for persons 65 and older. Mean daily admissions for CVD: 172.5 all ages, 54.5 <65, 117.8 ≥65; for IHD: 24.5 <65, 37.6 ≥65. Covariates: NO_2, O_3, SO_2, CO, temperature, relative humidity. Poisson regression using APHEA methodology; sine and cosine functions for seasonal control; day of week dummy variables. Lags of 0-3, as well as corresponding multi-day averages ending on lag 0, were considered.	In single-pollutant models, both PM metrics showed positive associations with both CVD and IHD admissions across age groups. In Two-pollutant models, the BS effect, but not the PM_{10} effect, was robust. No quantitative results provided for two-pollutant models. Study does not support a PM_{10} effect independent of co-pollutants.	Effects computed for 50 µg/m^3 PM_{10} and 25 µg/m^3 BS PM_{10} 0 d. All ages: CVD: 3.2 (0.9, 5.5) 0–64 yr: CVD: 5.6 (2.0, 9.4) IHD: 6.8 (1.3, 12.7) 65+ yr: CVD: 2.5 (–0.2, 5.3) IHD: 5.0 (0.8, 9.3) Black Smoke 0 d. All ages: CVD: 2.95 (1.00, 4.94) 0–64 yr: CVD: 3.12 (0.05, 6.29) IHD: 2.78 (–1.88, 7.63) 65+ yr: CVD: 4.24 (1.89, 6.64) IHD (lag 3): 4.57 (0.86, 8.42)

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
Europe (cont'd)			
Prescott et al. (1998) Edinburgh, Scotland 1981-1995 (BS and SO_2) 1992-1995 (PM_{10}, NO_2, O_3, CO) Means for long and short series: BS: 12.3, 8.7 PM_{10}: NA, 20.7	Daily emergency hospital admissions for cardiovascular disease (ICD9 codes 410-414, 426-429, 434-440) for persons less than 65 years and for persons 65 or older. Separate analyses presented for long (1981-1995) and short (1992-1995) series. Mean hospital admissions for long and short series: <65, 3.5, 3.4; 65+, 8.0, 8.7. Covariates: SO_2, NO_2, O_3, CO, wind speed, temperature, rainfall. PM_{10} measured by TEOM. Stats: Poisson log-linear regression; trend and seasons controlled by monthly dummy variables over entire series; day of week dummy variables; min daily temperature modeled using octile dummies. Pollutants expressed as cumulative lag 1-3 day moving avg.	In long series, neither BS nor NO_2 were associated with CVD admissions in either age group. In the short series, only 3-day moving average PM_{10} was positively and significantly associated with CVD admissions in single-pollutant models, and only for persons 65 or older. BS, SO_2, and CO also showed positive associations in this subset, but were not significant at the 0.05 level. The PM_{10} effect remained largely unchanged when all other pollutants were added to the model, however quantitative results were not given. Results appear to show an effect of PM_{10} independent of co-pollutants.	Percent Excess Risk (95% CI): Effects computed for 50 μg/m^3 change in PM_{10} and 25 μg/m^3 change in BS. Long series: BS, 1–3 d. avg. <65: −0.5 (−5.4, 4.6) 65+: −0.5 (−3.8, 2.9) Short series: BS, 1–3 d. avg. <65: −9.5 (−24.6, 8.0) 65+: 5.8 (−4.9, 17.8) PM_{10}, 1–3 d. avg. <65: 2.0 (−12.5, 19.0) 65+: 12.4 (4.6, 20.9)
Wordley et al. (1997) Birmingham, UK 4/1/92-3/31/94 mean, min, max: PM_{10} (μg/m^3): 26, 3, 131	Daily hospital admissions for acute ischemic heart disease (ICD9 codes 410-429) for all ages. Mean hospitalizations: 25.6/day. Covariates: temperature and relative humidity. Stats: Linear regression with day of week and monthly dummy variables, linear trend term. Lags of 0-3 considered, as well as the mean of lags 0-2.	No statistically significant effects observed for PM_{10} on ischemic heart disease admissions for any lag. Note that PM_{10} was associated with respiratory admissions and with cardiovascular mortality in the same study (results not shown here).	% change (95% CI) per 50 μg/m^3 change PM_{10} IHD admissions: PM_{10} 0-d lag: 1.4% (−4.4, 7.2) PM_{10} 1-d lag: −1.3% (−7.1, 4.4)
Díaz et al. (1999) Madrid, Spain 1994-1996 TSP by beta attenuation Summary statistics not given.	Daily emergency hospital admissions for all cardiovascular causes (ICD9 codes 390-459) for the Gregorio Maranon University Teaching Hospital. Mean admissions: 9.8/day. Covariates: SO_2, NO_2, O_3, temperature, pressure, relative humidity, excess sunlight. Stats: Box-Jenkins time-series methods used to remove autocorrelations, followed by cross-correlation analysis; sine and cosine terms for seasonality; details unclear.	No significant effects of TSP on CVD reported.	No quantitative results presented for PM.

TABLE 8B-1 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND CARDIOVASCULAR HOSPITAL ADMISSIONS

Reference citation. Location, Duration PM Index, Mean or Median, IQR	Study Description: Health outcomes or codes, Mean outcome rate, sample or population size, ages. Concentration measures or estimates. Modeling methods: lags, smoothing, co-pollutants, covariates, concentration-response	Results and Comments. Design Issues, Uncertainties, Quantitative Outcomes	PM Index, Lag, Excess Risk % (95% LCL, UCL), Co-Pollutants
Australia			
Morgan et al. (1998) Sydney, Australia 1990-1994 mean, median, IQR, 90-10 percentile range: Daily avg. bscat/10^4m: 0.32, 0.26, 0.23, 0.48 Daily max 1-hr bscat/10^4m: 0.76, 0.57, 60, 1.23	Daily hospital admissions for heart disease (ICD9 codes 410, 413, 427, 428) for all ages, and separately for persons less than 65 and persons 65 or greater. Mean daily admissions: all ages, 47.2; <65, 15.4; 65+, 31.8. PM measured by nephelometry (i.e., light scattering), which is closely associated with $PM_{2.5}$. Authors give conversion for Sydney as $PM_{2.5}$ =30 × bscat. Covariates: O_3, NO_2, temperature, dewpoint temperature. Stats: Poisson regression; trend and seasons controlled with linear time trend and monthly dummies; temperature and dewpoint controlled with dummies for eight levels of each variable; day of week and holiday dummies. Single and cumulative lags from 0-2 considered. Both single and multi-pollutant models were examined.	In single-pollutant models, NO_2 was strongly associated with heart disease admissions in all age groups. PM was more weakly, but still significantly associated with admissions for all ages and for persons 65+. The NO_2 association in the 65+ age group was unchanged in the multi-pollutant model, whereas the PM effect disappeared when NO_2 and O_3 were added to the model.. These results suggest that PM is not robustly associated with heart disease admissions when NO_2 is included, similar to the sensitivity of PM to CO in other studies.	Percent Excess Risk (95% CI): Effects computed for 25 μg/m^3 $PM_{2.5}$ (converted from bscat). 24-hr avg. $PM_{2.5}$ 0 d. <65: 1.8 (−2.9, 6.7) 65+: 4.9 (1.6, 8.4) All: 3.9 (1.1, 6.8) 24-hr $PM_{2.5}$, 0 d w. NO_2 and O_3. 65+: 0.12 (−1.3, 1.6) 1-hr $PM_{2.5}$, 0 d. <65: 0.19 (−1.6, 2.0) 65+: 1.8 (0.5, 3.2) All: 1.3 (0.3, 2.3)
Asia			
Wong et al. (1999a) Hong Kong 1994-1995 median, IQR for PM_{10} (μg/m^3): 45.0, 34.8	Daily emergency hospital admissions for cardiovascular diseases, CVD (ICD9 codes 410-417, 420-438, 440-444), heart failure, HF (ICD9 428), and ischemic heart disease, IHD (ICD9 410-414) among all ages and in the age categories 5-64, and 65+. Median daily CVD admissions for all ages: 101. Covariates: NO_2, O_3, SO_2, temperature, relative humidity. PM_{10} measured by TEOM. Stats: Poisson regression using the APHEA protocol; linear and quadratic control of trends; sine and cosine control for seasonality; holiday and day of week dummies; autoregressive terms. Single and cumulative lags from 0-5 days considered.	In single-pollutant models, PM_{10}, NO_2, SO_2, and O_3 all significantly associated with CVD admissions for all ages and for those 65+. No multi-pollutant risk coefficients were presented; however, the PM_{10} effect was larger when O_3 was elevated (i.e., above median). A much larger PM_{10} effect was observed for HF than for CVD or IHD. These results confirm the presence of PM_{10} associations with cardiovascular admissions in single-pollutant models, but do not address the independent role of PM_{10}.	Percent Excess Risk (95% CI): Effects computed for 50 μg/m^3 change in PM_{10}. PM_{10}, 0−2 d. avg. CVD: 5−64: 2.5 (−1.5, 6.7) 65+: 4.1 (1.3, 6.9) All: 3.0 (0.8, 5.4) HF (PM_{10}, 0-3 d ave.): All: 26.4 (17.1, 36.4) IHD (PM_{10}, 0-3 d ave.): All: 3.5 (−0.5, 7.7)

Appendix 8B.2. PM-Respiratory Hospitalization Studies

TABLE 8B-2. ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States			
Samet et al, (2000a,b)* Study Period: 84- 95 14 U.S. Cities: Birmingham, Boulder, Canton, Chicago, Col. Springs, Detroit, Minn./St. Paul, Nashville, New Haven, Pittsburgh, Provo/Orem, Seattle, Spokane, Youngstown. Mean pop. aged 65+ yr per city =143,000 PM_{10} mean = 32.9 µg/m^3 PM_{10} IQR = NR	Hospital admissions for adults 65+ yrs. for CVD (mean=22.1/day/city), COPD (mean=2.0/day/city), and Pneumonia (mean=5.6/day/city) related to PM_{10}, SO_2, O_3, NO_2, and CO. City-specific Poisson models used with adjustment for season, mean temperature (T) and relative humidity (RH) (but not their interaction), as well as barometric pressure (BP) using LOESS smoothers (span usually 0.5). Indicators for day-of-week and autoregressive terms also included.	PM_{10} positively associated with all three hospital admission categories, but city specific results ranged widely, with less variation for outcomes with higher daily counts. PM_{10} effect estimates not found to vary with co-pollutant correlation, indicating that results appear quite stable when controlling for confounding by gaseous pollutants. Analyses found little evidence that key socioeconomic factors such as poverty or race are modifiers, but it is noted that baseline risks may differ, yielding differing impacts for a given RR.	PM_{10} = 50 µg/m^3 <u>COPD HA's for Adults 65+ yrs.</u> Lag 0 ER = 7.4% (CI: 5.1, 9.8) Lag 1 ER = 7.5% (CI: 5.3, 9.8) 2 day mean (lag0,lag1) ER = 10.3% (CI: 7.7, 13) <u>Pneumonia HA's for Adults 65+ yrs.</u> Lag 0 ER =8.1% (CI: 6.5, 9.7) Lag 1 ER = 6.7% (CI: 5.3, 8.2) 2 day mean (lag0, lag1) = 10.3% (CI: 8.5, 12.1)
Reanalysis of Samet et al (2000) by Zanobetti and Schwartz (2003b)	Re-analyses of Samet et al. (2000) with more stringent GAM convergence criteria and alternative models.	Results differ somewhat from original analyses, especially for pneumonia. Results indicate that the stricter convergence criteria results in about a 14% lower GAM effect than in the originally published analyses method. Authors recommend the penalized spline model results.	COPD 2 day mean (lag 0, lag1): Default GAM ER=9.4 (5.9, 12.9) Strict GAM ER = 8.8 (4.8, 13.0) NS GLM ER=6.8 (2.8, 10.8) PS GLM ER = 8.0 (4.3, 11.9) Pneumonia 2 day mean (lag 0, lag1): Default GAM ER=9.9 (7.4, 12.4) Strict GAM ER =8.8(5.9, 11.8) NS GLM ER=2.9 (0.2,5.6) PS GLM ER = 6.3 (2.5,10.3)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States (cont'd)			
Zanobetti et al. (2000b)+ 10 U.S. Cities	Derived from the Samet et al. (2000a,b) study, but for a subset of 10 cities. Daily hospital admissions for total cardiovascular and respiratory disease in persons aged 65 yr. Covariates: SO_2, O_3, CO, temperature, relative humidity, barometric pressure. In first stage, performed single-pollutant generalized additive robust Poisson regression with seasonal, weather, and day of week controls. Repeated analysis for days with PM_{10} less than 50 µg/m^3 to test for threshold. Lags of 0-5 d considered, as well as the quadratic function of lags 0-5. Individual cities analyzed first. The 10 risk estimates were then analyzed in several second stage analyses: combining risks across cities using inverse variance weights, and regressing risk estimates on potential effect-modifiers and pollutant confounders.	Same basic pattern of results as in Samet et al. (2000a,b). For distributed lag analysis, lag 0 had largest effect, lags 1 and 2 smaller effects, and none at larger lags. City-specific slopes were independent of percent poverty and percent non-white. Effect size increase when data were restricted to days with PM_{10} less than 50 µg/m^3. No multi-pollutant models reported; however, no evidence of effect modification by co-pollutants in second stage analysis. Suggests association between PM_{10} and total respiratory hospital admissions among the elderly.	Percent excess respiratory risk (95% CI) per 50 µg/m^3 PM_{10} increase: COPD (0-1 d lag) = 10.6 (7.9, 13.4) COPD (unconstrained dist. lag) = 13.4 (9.4, 17.4) Pneumonia (0-1 d lag) = 8.1 (6.5, 9.7) Pneumonia (unconstrained dist. lag) = 10.1 (7.7, 12.6)
Jamason et al. (1997) New York City, NY (82 - 92) Population = NR PM_{10} mean = 38.6 µg/m^3	Weather/asthma relationships examined using a synoptic climatological multivariate methodology. Procedure relates homogenous air masses to daily counts of overnight asthma hospital admission.	Air pollution reported to have little role in asthma variations during fall and winter. During spring and summer, however, the high risk categories are associated with high concentration of various pollutants (i.e., PM_{10}, SO_2,NO_2, O_3).	NR
Chen et al. (2000)+ Reno-Sparks, NV (90 - 94) Population = 307,000 B-Gauge PM_{10} mean=36.5 µg/m^3 PM_{10} IQR = 18.3-44.9 µg/m^3 PM_{10} maximum = 201.3 µg/m^3	Log of COPD (mean=1.72/day) and gastroenteritis (control) admissions from 3 hospitals analyzed using GAM regression, adjusting for effects of day-of-week, seasons, weather effects (T, WS), and long-wave effects. Only one LOESS used with GAM, so the default convergence criteria may be satisfactory in this case. No co-pollutants considered.	PM_{10} positively associated with COPD admissions, but no association with gastroenteritis (GE) diseases, indicating biologically plausible specificity of the PM_{10}-health effects association. Association remained even after excluding days with PM_{10} above 150 µg/m^3.	<u>COPD All age Admissions</u> 50 µg/m^3 IQR PM_{10} (single pollutant): ER = 9.4% (CI: 2.2, 17.1)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States (cont'd)			
Gwynn et al. (2000)+ Buffalo, NY (5/88-10/90) PM_{10} mn./max. = 24.1/90.8 µg/m^3 PM_{10} IQR = 14.8-29.2 µg/m^3 $SO_4^=$ mn./max. = 2.4/3.9 µg/m^3 $SO_4^=$ IQR = 23.5 – 7.5 µg/m^3 H^+ mn/max = 36.4/382 nmol/m^3 H^+ IQR = 15.7-42.2 nmol/m^3 CoH mn/max = 0.2/0.9 10 3 ft. CoH IQR = 0.1-0.3	Air pollutant-health effect associations with total, respiratory, and circulatory hospital admissions and mortality examined using Poisson methods controlling for weather, seasonality, long-wave effects, day of week, and holidays using GAM with LOESS terms.	Strongest associations found between $SO_4^=$ and respiratory hospital admissions, while secondary aerosol H^+ and $SO_4^=$ demonstrated the most coherent associations across both respiratory hospital admissions and mortality. Addition of gaseous pollutants to the model had minimal effects on the PM RR estimates. CoH weakness in associations may reflect higher toxicity by acidic sulfur containing secondary particles versus carbonaceous primary particles.	<u>Respiratory Hospital Admissions(all ages) PM Index (using standardized conc. increment)</u> -Single Pollutant Models For PM_{10} = 50 µg/m^3; SO_4 = 15 µg/m^3; H^+ = 75nmoles/m^3;COH = 0.5 units/1000ft PM_{10}(lag 0) ER = 11% (CI: 4.0, 18) $SO_4^=$(lag 0) ER = 8.2% (CI: 4.1, 12.4) H^+(lag 0) ER = 6% (CI: 2.8, 9.3) CoH(lag0) ER = 3% (CI: -1.2, 7.4)
Gwynn and Thurston (2001)+ New York City, NY 1988, 89, 90 PM_{10} 37.4 µg/m^3 mean	Respiratory hospital admissions, race specific for PM_{10}, H^+, O_3, $SO_4^=$. LOESS GAM regression model used to model daily variation in respiratory hospital admissions, day-week, seasonal, and weather aspects addressed in modeling.	Greatest difference between the white and non-white subgroups was observed for O_3. However, within race analyses by insurable coverage suggested that most of the higher effects of air pollution found for minorities were related to socio-economic studies.	PM_{10} (max-min) increment 1 day lag white 1.027 (0.971-1.074) non-white (1.027 (0.988-1.069)
Jacobs et al. (1997) Butte County, CA (83 - 92) Population = 182,000 PM_{10} mean = 34.3 µg/m^3 PM_{10} min/max = 6.6 / 636 µg/m^3 CoH mean = 2.36 per 1000 lin. ft. CoH min/max = 0 / 16.5	Association between daily asthma HA's (mean = 0.65/day) and rice burning using Poisson GLM with a linear term for temperature, and indicator variables for season and yearly population. Co-pollutants were O_3 and CO. PM_{10} estimated for 5 of every 6 days from CoH.	Increases in rice straw burn acreage found to correlate with asthma HA's over time. All air quality parameters gave small positive elevations in RR. PM_{10} showed the largest increase in admission risk.	Asthma HA's (all ages) For an increase of 50 µg/m^3 PM_{10}: ER = 6.11% (not statistically significant)
Linn et al. (2000) Los Angeles, CA (92 - 95) Population = NR PM_{10} mean = 45.5 µg/m^3 PM_{10} Min/Max = 5/132 µg/m^3	Pulmonary hospital admissions (HA's) (mean=74/day) related to CO, NO_2, PM_{10}, and O_3 in Los Angeles using GLM Poisson model with long-wave spline, day of week, holidays, and weather controls.	PM_{10} positively associated with pulmonary admissions year-round, especially in winter. No association with cerebro-vascular or abdominal control diseases. However, use of linear temperature, and with no RH interaction, may have biased effect estimates downwards for pollutants here most linearly related to temperature (i.e., O_3 and PM_{10}).	<u>Pulmonary HA's (>29 yrs.)</u> PM_{10} = 50 µg/m^3 (Lag 0)ER = 3.3% (CI: 1.7, 5)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States (cont'd)			
Moolgavkar et al. (1997)+ Minneapolis-St. Paul 86 - 91 Population.= NR Birmingham, AL '86-'91 Population. = NR PM_{10} mean = 34 µg/m^3 (M-SP) PM_{10} IQR =22-41 µg/m^3 (M-SP) PM_{10} mean =43.4 µg/m^3(Birm) PM_{10} IQR =26-56 µg/m^3(Birm)	Investigated associations between air pollution (PM_{10}, SO_2, NO_2 O_3, and CO) and hospital admissions for COPD (mean/day=2.9 in M-SP; 2.3 in Birm) and pneumonia (mean=7.6 in M-SP; 6.0 in Birm) among older adults (>64 yrs.). Poisson GAM's used, controlling for day-of-week, season, LOESS of temperature (but neither RH effects nor T-RH interaction considered).	In the M-SP area, PM_{10} significantly and positively associated with total daily COPD and pneumonia admissions among elderly, even after simultaneous inclusion of O_3. When four pollutants included in the model (PM_{10}, SO_2, O_3, NO_2), all pollutants remained positively associated. In Birm., neither PM_{10} nor O_3 showed consistent associations across lags. The lower power (fewer counts) and lack of T-RH interaction weather modeling in this Southern city vs. M-SP may have contributed to the differences seen between cities.	<u>COPD + Pneumonia Admissions (>64yrs.)</u> In M-SP, For PM_{10} = 50 µg/m^3 (max lg) ER(lg 1) = 8.7% (CI: 4.6, 13) With O_3 included simultaneously: ER(lg1)= 6.9% (95 CI: 2.7, 11.3) In Birm, For PM_{10}=50 µg/m^3 (max lg.) ER(lg 0) = 1.5% (CI: -1.5, 4.6) With O_3 included simultaneously: ER(lg0) = 3.2% (CI: -0.7, 7.2)
Nauenberg and Basu (1999) Los Angeles (91 - 94) Wet Season = 11/1-3/1 Dry Season = 5/1-8/15 Population .= 2.36 Million PM_{10} Mean = 44.81 µg/m^3 PM_{10} SE = 17.23 µg/m^3	The effect of insurance status on the association between asthma-related hospital admissions and exposure to PM_{10} and O_3 analyzed, using GLM Poisson regression techniques with same day and 8-day weighted moving average levels, after removing trends using Fourier series. Compared results during wet season for all asthma HA's (mean = 8.7/d), for the uninsured (mean=0.77/d), for MediCal (poor) patients (mean = 4.36/d), and for those with other private health or government insurance (mean = 3.62/d).	No associations found between asthma admissions and O_3. No O_3 or PM_{10} associations found in dry season. PM_{10} averaged over eight days associated with increase in asthma admissions, with even stronger increase among MediCal asthma admissions in wet season. The authors conclude that low income is useful predictor of increased asthma exacerbations associated with air pollution. Non-respiratory HA's showed no such association with PM_{10}.	<u>All Age Asthma HA's</u> PM_{10} = 50 µg/m^3, no co-pollutant, during wet season (Jan. 1 - Mar. 1): <u>All Asthma Hospital Admissions</u> 0-d lag PM_{10} ER = 16.2 (CI: 2.0, 30) 8-d avg. PM_{10} ER = 20.0 (CI: 5.3, 35) <u>MediCal Asthma Hospital Admissions</u> 8-d avg. PM_{10} ER = 13.7 (3.9, 23.4) <u>Other Insurance Asthma HA's</u> 8-d avg. PM_{10} ER = 6.2 (-3.6, 16.1)
Schwartz et al. (1996b) Cleveland (Cayahoga County), Ohio (88 - 90) PM_{10} mean = 43 µg/m^3 PM_{10} IQR = 26 – 56 µg/m^3	Review paper including an example drawn from respiratory hospital admissions of adults aged 65 yr and older (mean = 22/day) in Cleveland, OH. Categorical variables for weather and sinusoidal terms for filtering season employed.	Hospital admissions for respiratory illness of persons aged 65 yr and over in Cleveland strongly associated with PM_{10} and O_3, and marginally associated with SO_2 after control for season, weather, and day of the week effects.	<u>Respiratory HA's for persons 65+ years</u> 50 µg/m^3 PM_{10} ER = 5.8% (CI: 0.5, 11.4)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States (cont'd)			
Zanobetti, et al. (2000a)+ Study Period: 86 - 94 Chicago (Cook Count), IL Population = 633,000 aged 65+ PM_{10} mean = 33.6 µg/m^3 PM_{10} range = 2.2, 157.3 µg/m^3	Analyzed HA's for older adults (65 + yr) for COPD (mean = 7.8/d), pneumonia (mean = 25.5/d), and CVD, using GLM Poisson regression controlling for temperature, dew point, barometric pressure, day of week, long wave cycles and autocorrelation, to evaluate whether previous admission or secondary diagnosis for associated conditions increased risk from air pollution. Effect modification by race, age, and sex also evaluated.	Air pollution- associated CVD HA's were nearly doubled for those with concurrent respiratory infections (RI) vs. those without concurrent RI. For COPD and pneumonia admissions, diagnosis of conduction disorders or dysrhythmias (Dyshr.) increased PM_{10} RR estimate. The PM_{10} RR effect size did not vary significantly by sex, age, or race, but baseline risks across these groups differ markedly, making such sub-population RR inter-comparisons difficult to interpret.	PM_{10} = 50 µg/m^3(average of lags 0,1) <u>COPD (adults 65+ yrs.)</u> W/o prior RI. ER = 8.8% (CI: 3.3, 14.6) With prior RI ER = 17.1% (CI: -6.7, 46.9) <u>COPD (adults 65+ yrs.)</u> W/o concurrent Dys. ER = 7.2% (CI: 1.3, 13.5) With concurrent Dys. ER = 16.5%(CI: 3.2, 31.5) <u>Pneumonia (adults 65+ yrs.)</u> W/o pr. Asthma ER = 11% (CI: 7.7, 14.3) With pr. Asthma ER = 22.8% (CI: 5.1, 43.6) <u>Pneumonia (adults 65+ yrs.)</u> W/o pr. Dyshr. ER = 10.4% (CI: 6.9, 14) With pr. Dyshr. ER = 18.8% (CI: 6.3, 32.7)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States (cont'd)			
Lippmann et al. (2000)* Detroit, MI ('92-'94) Population = 2.1 million PM_{10} Mean = 31 µg/m^3 (IQR= 19, 38 µg/m^3; max=105 µg/m^3) $PM_{2.5}$ Mean = 18 µg/m^3 (IQR= 10, 21 µg/m^3; max=86 µg/m^3) $PM_{10-2.5}$ Mean = 12 µg/m^3 (IQR= 8, 17 µg/m^3; max=50 µg/m^3) $SO_4^=$Mean = 5 µg/m^3 (IQR=1.8, 6.3 µg/m^3; max=34.5 µg/m^3) H^+ Mean = 8.8 nmol/m^3 = 0.4 µg/m^3 (IQR=0, 7nmol/m^3;max=279)	Respiratory (COPD and Pneumonia) HA's for persons 65 + yr. analyzed, using GAM Poisson models, adjusting for season, day of week, temperature, and relative humidity using LOESS smooths. The air pollution variables analyzed were: PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, sulfate, H^+, O_3, SO_2, NO_2, and CO. However, this study site/period had very low acidic aerosol levels. As noted by the authors 85% of H^+ data was below detection limit (8 nmol/m^3).	For respiratory HA's, all PM metrics yielded RR's estimates >1, and all were significantly associated in single pollutant models for pneumonia. For COPD, all PM metrics gave RR's >1, with H^+ being associated most significantly, even after the addition of O_3 to the regression. Adding gaseous pollutants had negligible effects on the various PM metric RR estimates. The most consistent effect of adding co-pollutants was to widen the confidence bands on the PM metric RR estimates: a common statistical artifact of correlated predictors. Despite usually non-detectable levels, H^+ had strong association with respiratory admissions on the few days it was present. The general similarity of the $PM_{2.5}$ and $PM_{10-2.5}$ effects per µg/m^3 in this study suggest similarity in human toxicity of these two inhalable mass components in study locales/periods where $PM_{2.5}$ acidity is usually not present.	<u>Pneumonia HA's for 65+ yrs.</u> <u>No co-pollutant:</u> PM_{10} (50 µg/m^3) 1d lag ER = 22% (CI: 8.3, 36) $PM_{2.5}$ (25 µg/m^3) 1d lag: ER = 13% (CI: 3.7, 22) $PM_{2.5-10}$ (25 µg/m^3) 1d lag: ER = 12% (CI: 0.8, 24) H^+ (75 nmol/m^3) 3d lag: ER = 12% (CI: 0.8, 23) <u>O_3 co-pollutant (lag 3) also in model:</u> PM_{10} (50 µg/m^3) 1d lag, ER = 24% (CI: 8.2, 43) $PM_{2.5}$ (25 µg/m^3) 1d lag: ER = 12% (CI: 1.7, 23) $PM_{2.5-10}$ (25 µg/m^3) 1d lag: ER = 14% (CI: 0.0, 29) H^+ (75 nmol/m^3) 3d lag: ER = 11% (CI: -0.9, 24) <u>COPD Hospital Admissions for 65+ yrs.</u> <u>No co-pollutant:</u> PM_{10} (50 µg/m^3) 3d lag ER = 9.6% (CI: -5.1, 27) $PM_{2.5}$ (25 µg/m^3) 3d lag: ER = 5.5% (CI: -4.7, 17) $PM_{2.5-10}$ (25 µg/m^3) 3d lag: ER = 9.3% (CI: -4.4, 25) H^+ (75 nmol/m^3) 3d lag: ER = 13% (CI: 0.0, 28) <u>O_3 co-pollutant (lag 3) also in model:</u> PM_{10} (50 µg/m^3) 3d lag, ER = 1.0% (-15, 20) $PM_{2.5}$ (25 µg/m^3) 3d lag: ER = 2.8% (CI: -9.2, 16) $PM_{2.5-10}$ (25 µg/m^3) 3d lag: ER = 0.3% (CI: -14, 18) H^+ (75 nmol/m^3) 3d lag: ER = 13% (CI: -0.6, 28)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States (cont'd)			
Reanalysis by Ito (2003)	Re-analyses of Lippmann et al. (2000) with more stringent GAM convergence criteria and alternative models.	More stringent GAM generally, but not always, resuled in reduced RR estimates, but effect sizes not significantly different from originals. Extent fo reuction independent of risk estimate size. The reductions were not differential across PM components, so study conclusions unchanged.	**Pneumonia** (PM_{10}= 50 ug/m^3, LAG= 1D, No Co Poll): Default GAM: ER= 21.5 (8.3, 36) Strict GAM: ER=18.1 (5.3, 32.5) NS GLM: ER=18.6 (5.6, 33.1) **COPD** (PM_{10}= 50 ug/m^3, LAG= 3D, No Co Poll): Default GAM: ER= 9.6 (-5.3, 26.8) Strict GAM: ER=6.5 (-7.8, 23.0) NS GLM: ER=4.6 (-9.4, 20.8) **COPD** ($PM_{2.5}$,=25 ug/m^3, Lag=1D, No Co Poll): Default GAM: ER =5.5 (-4.7, 16.8) Strict GAM: ER=3.0(-6.9, 13.9) NS GLM: ER=0.3(-9.3, 10.9) **Pneumonia** ($PM_{2.5}$,=25 ug/m^3, LAG= 1D,No Co Poll): Default GAM: ER = 12.5 (3.7, 22.1) Strict GAM:ER = 10.5 (1.8, 19.8) NS GLM: 10.1 (1.5, 19.5)
Lumley and Heagerty (1999) Seattle (King Cty.), WA (87-94) Population = NR PM_1 daily mean = NR PM_{1-10} daily mean = NR From Sheppard et al, 1999: PM_{10} mean = 31.5 µg/m^3 PM_{10} IQR = 19-39 µg/m^3 $PM_{2.5}$ mean = 16.7 µg/m^3 $PM_{2.5}$ IQR = 8-21 µg/m^3	Estimating equations based on marginal generalized linear models (GLM) applied to respiratory HA's for persons <65 yrs. of age (mean ˜ 8/day) using class of variance estimators based upon weighted empirical variance of the estimating functions. Poisson regression used to fit a marginal model for the log of admissions with linear temperature, day of week, time trend, and dummy season variables. No co-pollutants considered.	PM_1 at lag 1 day associated with respiratory HA's in children and younger adults (<65), but not PM_{10-1}, suggesting a dominant role by the submicron particles in $PM_{2.5}$–asthma HA associations reported by Sheppard et al. (1999). 0-day lag PM_1 and 0 and 1 day lag PM_{1-10} had RR near 1 and clearly non-significant. Authors note that model residuals correlated at r=0.2, suggesting the need for further long-wave controls in the model (e.g., inclusion of the LOESS of HA's).	<u>Respiratory HA's for persons <65 yrs. old</u> PM_1 = 25 µg/m^3, no co-pollutant: 1-d lag ER = 5.9 (1.1, 11.0)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States (cont'd)			
Moolgavkar et al. (2000)+ King County, WA (87 - 95) Population = NR PM_{10} mean = 30.0 µg/m^3 PM_{10} IQR =18.9-37.3 µg/m^3 $PM_{2.5}$ mean =18.1 µg/m^3 $PM_{2.5}$ IQR =10-23 µg/m^3	Association between air pollution and hospital admissions (HA's) for COPD (all age mean=7.75/day; 0-19 yrs. mean=2.33/day) investigated using Poisson GAM's controlling for day-of-week, season, and LOESS of temperature. Co-pollutants addressed: O_3, SO_2, CO, and pollens. $PM_{2.5}$ only had one monitoring site versus multiple sites averaged for other pollutants.	Of the PM metrics, PM_{10} showed the most consistent associations across lags (0-4 d). $PM_{2.5}$ yielded the strongest positive PM metric association at lag3 days, but gave a negative association at lag4 days. That $PM_{2.5}$ only had one monitoring site may have contributed to its effect estimate variability. Residual autocorrelations (not reported) may also be a factor. Adding gaseous co-pollutants or pollens decreased the $PM_{2.5}$ effect estimate less than PM_{10}. Analyses indicated that asthma HA's among the young were driving the overall COPD-air pollution associations.	<u>COPD HA's all ages</u> (no co-pollutant) PM_{10} (50 µg/m^3, lag 2) ER = 5.1% (CI: 0, 10.4) $PM_{2.5}$ (25 µg/m^3, lag 3) ER = 6.4% (CI: 0.9, 12.1) COPD HA's all ages (CO as co-pollutant) PM_{10} (50 µg/m^3, lag 2) ER = 2.5% (CI: -2.5, 7.8) $PM_{2.5}$ (25 µg/m^3, lag 3) ER = 5.6% (CI: 0.2, 11.3)
Moolgavkar (2000a)* Study Period: 1987-1995 <u>Chicago (Cook County), IL</u> Population = NR PM_{10} median = 35 µg/m^3 PM_{10} IQR = 25-47 µg/m^3 <u>Los Angeles (LA County), CA</u> Population = NR PM_{10} median = 44 µg/m^3 PM_{10} IQR = 33-59 µg/m^3 $PM_{2.5}$ median = 22 µg/m^3 $PM_{2.5}$ IQR = 15-31 µg/m^3 <u>Phoenix (Maricopa County), AZ</u> Population = NR PM_{10} median = 41 µg/m^3 PM_{10} IQR = 32-51 µg/m^3	Investigated associations between air pollution (PM_{10}, O_3, SO_2, NO_2, and CO) and COPD Hospital Admissions (HA's). $PM_{2.5}$ also analyzed in Los Angeles. HA's for adults >65 yr.: median=12/day in Chicago, =4/d in Phoenix; =20/d in LA. Analyses employed 30df to fit long wave. In LA, analyses also conducted for children 0-19 yr. (med.=17/d) and adults 20-64 (med.=24/d). Poisson GAM's used controlling for day-of-week, season, and splines of temperature and RH (but not their interaction) adjusted for overdispersion. PM data available only every 6th day (except for daily PM_{10} in Chicago), vs. every day for gases. Power likely differs across pollutants, but number of sites and monitoring days not presented. Two pollutant models forced to have same lag for both pollutants. Autocorrelations or intercorrelations of pollutant coefficients not presented or discussed.	For >64 adults, CO, NO_2 and O_3 (in summer) most consistently associated with the HA's. PM effects more variable, especially in Phoenix. Both positive and negative significant associations for PM and other pollutants at different lags suggest possible unaddressed negative autocorrelation. In LA, PM associated with admissions in single pollutant models, but not in two pollutant models. The forcing of simultaneous pollutants to have the same lag (rather than maximum lag), which likely maximizes intercorrelations between pollutant coefficients, may have biased the two pollutant coefficients, but information not presented. Analysis in 3 age groups in LA yielded similar results. Author concluded that "the gases, other than ozone, were more strongly associated with COPD admissions than PM, and that there was considerable heterogeneity in the effects of individual pollutants in different geographic areas".	Most Significant Positive ER Single Pollutant Models: <u>COPD HA's (>64 yrs.)</u> (50 µg/m^3 PM_{10}): Chicago: Lag 0 ER =2.4% (CI: -0.2, 4.3) LA: Lag 2 ER = 6.1% (CI: 1.1, 11.3) Phoenix: Lag 0 ER = 6.9% (CI: -4.1, 19.3) <u>LA COPD HA's</u> (50 µg/m^3 PM_{10}, 25 µg/m^3 $PM_{2.5}$ or $PM_{2.5-10}$) (0-19 yrs.): PM_{10} lg2=10.7%(CI: 4.4, 17.3) (0-19 yrs.): $PM_{2.5}$ lg0=4.3%(CI: -0.1, 8.9) (0-19 yrs.): $PM_{(2.5-10)}$ lg2=17.1%(CI: 8.9, 25.8) (20-64 yrs.): PM_{10} lg2=6.5%(CI: 1.7, 11.5) (20-64 yrs.): $PM_{2.5}$ lg2=5.6%(CI: 1.9, 9.4) (20-64 yrs.): $PM_{2.5-10}$ lg2=9%(CI: 3, 15.3) (> 64 yrs): PM_{10} lg2 = 6.1% (1.1, 11.3) (> 64 yrs): $PM_{2.5}$ lg2 = 5.1% (0.9, 9.4) (>64 yrs.): $PM_{2.5-10}$ lg3=5.1% (CI: -0.4, 10.9) (>64 yr) 2 Poll. Models (CO = co-poll.) PM_{10}: Lag 2 ER = 0.6% (CI: -5.1, 6.7) $PM_{2.5}$: Lag 2 ER = 2.0% (-2.9, 7.1)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States (cont'd)			
Reanalysis by Moolgavkar (2003)	Re-analyses of Moolgavkar (2000a) with more stringent GAM convergence criteria and alternative models.	GAM effect estimates virtually unchanged from originals using when GAM stringent criteria applied in LA (direct comparisons not possible in Chicago). In LA, changes in spline degrees of freedom had much more influence on effect size than the change in convergence criteria, especially for PM_{10}. In Chicago, small insignificant association of PM_{10} in the original work actually increased and became significant with the 100df model. Authors conclude the "basic qualitative conclusions unchanged".	LA COPD (all ages), LAG= 2D, PM_{10} =50ug/m3 Default GAM:30df** ER= 7.36% (CI:4.32-11.39) Strict GAM:30df ER= 7.78% (CI:4.32-10.51) Strict GAM: 100df ER = 7.78% (CI:4.32-10.51) NS GLM: 100df ER=5.00% (CI:1.22, 8.91) LA COPD (all ages), LAG=2D, $PM_{2.5}$ =25 ug/m3 Default GAM:30df** ER=4.82% (CI:2.44, 7.25) Strict GAM:30df ER=4.69% (CI:2.06, 7.38) Strict GAM: 100df ER=2.87% (CI:0.53, 5.27) NS GLM: 100df ER=2.59% (CI:-0.29, 5.56) Chicago COPD (>64yrs) LAG= 0D, PM_{10}=50ug/m^3 Default GAM (30df) ER =2.4% (CI: --0.2, 4.3) Default GAM (100df) not provided for comparison Strict GAM (100df) ER=3.24% (CI:0.031-6.24)
Sheppard et al. (1999)* Seattle, WA, Pop. = NR 1987-1994 PM_{10} mean = 31.5 µg/m^3 PM_{10} IQR = 19-39 µg/m^3 $PM_{2.5}$ mean = 16.7 µg/m^3 $PM_{2.5}$ IQR = 8-21 µg/m^3 $PM_{2.5-10}$ mean = 16.2 µg/m^3 $PM_{2.5-10}$ IQR = 9-21 µg/m^3	Daily asthma hospital admissions (HA's) for residents aged <65 (mean=2.7/day) regressed on PM_{10}, $PM_{2.5}$, $PM_{2.5-10}$, SO_2, O_3, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects. Appendicitis HA's analyzed as a control. Except O_3 in winter, missing pollutant measures estimated in a multiple imputation model. Pollutants varied in number of sites available for analysis, CO the most (4) vs. 2 for PM.	Asthma HA's significantly associated with PM_{10}, $PM_{2.5}$, and $PM_{10-2.5}$ mass lagged 1 day, as well as CO. Authors found PM and CO to be jointly associated with asthma admissions. Highest increase in risk in spring and fall. Results conflict with hypothesis that wood smoke (highest in early study years and winter) would be most toxic. Associations of CO with respiratory HA's taken by authors to be an index of incomplete combustion, rather than direct CO biological effect.	<u>Asthma Admissions (ages 0-64)</u> PM_{10} (lag=1day); 50 µg/m^3 ER = 13.7% (CI: 5.5%, 22.6) $PM_{2.5}$ (lag=1day); 25 µg/m^3 ER = 8.7% (CI: 3.3%, 14.3) $PM_{2.5-10}$ (lag=1day); 25 µg/m^3 ER = 11.1% (CI: 2.8%, 20.1)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States (cont'd)			
Reanalysis by Sheppard (2003)	Re-analyses of Sheppard et al. (1999) with more stringent GAM convergence criteria and alternative models.	The author notes that "While the biases from computational details of the fitting were small, they are not completely trivial given the small effects of interest." She concludes that: "Overall the results did not change meaningfully".	Asthma (ages 0-64) LAG=1day, PM_{10}=50 ug/m^3 No Co-Poll: Default GAM: ER = 13.7% (CI: 5.5%, 22.6) Strict GAM: ER= 8.1 (0.1, 16.7) NS GLM : ER=10.9 (2.8, 19.6) Asthma (all ages) LAG=1day, $PM_{2.5}$=25 ug/m^3 No Co-Poll: Default GAM : ER= 8.7% (3.3, 14.3) Strict GAM: ER=6.5% (1.1,12.0) NS GLM: ER= 8.7% (3.3,14.4) With Co-poll: Strict GAM: ER=6.5 (2.1, 10.9) NS GLM: ER=6.5 (2.1, 10.9)
Freidman et al. (2001) Atlanta, GA Summer 1996/control vs. Olympics PM_{10} decrease for 36.7 µg/m^3 to 30.8 µg/m^3	Asthma events in children aged 1 to 16 years were related to pollutant levels contrasting those during the Summer Olympics games during a 17 day period to control periods before and after the Olympics. GEE Poisson regression with autoregressive terms employed.	Asthma events were reduced during the Olympic period. A significant reduction in asthma events was associated with ozone concentration. The high correlation between ozone and PM limit the ability to determine which pollutants may have accounted for the reduction in asthma events.	3 day cumulative exposure PM_{10} per 10 µg/m^3 1.0 (0.80-2.48)
Zanobetti and Schwartz (2001)+ Cook County, Illinois 1988-1994 PM_{10}: 33 µg/m^3 median	Respiratory admissions for lung disease in persons with or without diabetes as a co-morbidity related to PM_{10} measures. The generalized additive model used nonparametric LOESS functions to estimate the relation between the outcome and each predictor. The covariates examined were temperature, prior day's temperature, relative humidity, barometric pressure, and day of week.	Weak evidence that diabetes modified the risks of PM_{10} induced respiratory hospital admissions while diabetes modified the risk of PM_{10} induced COPD admissions in older people. Found a significant interaction with hospital admissions for heart disease and PM with more than twice the risk in diabetics as in persons without diabetes.	<u>COPD</u> PM_{10} 10 µg/m^3 with diabetes 2.29 (-0.76-5.44) without diabetes 1.50 (0.42-2.60)
Janssen et al. (2002)+ 14 U.S. cities 1985-1994 see Samet et al. (2000a,b)	Regression coefficients of the relation between PM_{10} and hospital admissions for respiratory disease from Samet et al. (2000a,b) and prevalence of air conditioning (AC).	Regression coefficients of the relation between ambient PM_{10} and hospital admissions for COPD decreased with increasing percentage of homes with central AC.	—

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Canada (cont'd)			
Burnett et al. (1997b) Toronto, Canada (1992-1994), Pop. = 4 mill. $PM_{2.5}$ mean = 16.8 µg/m^3 $PM_{2.5}$ IQR = 8-23 µg/m^3 $PM_{2.5-10}$ mean = 11.6 µg/m^3 $PM_{2.5-10}$ IQR = 7-14 µg/m^3 PM_{10} mean = 28.4 µg/m^3 PM_{10} IQR = 16-38 µg/m^3 CoH mean = 0.8 (per 10^3 lin. ft.) CoH IQR = 0.5-1.1(per 10^3 lin ft) SO_4 mean = 57.1 nmole/m^3 SO_4 IQR = 14-71 nmole/m^3 H^+ mean = 5 nmole/m^3 H^+ IQR = 0-6 nmole/m^3	Hospital admissions (HA's) for respiratory diseases (tracheobronchitis, chronic obstructive long disease, asthma, pneumonia) analyzed using Poisson regression (adjusting for long-term temporal trends, seasonal variations, effects of short-term epidemics, day-of-week, ambient temperature and dew point). Both linear prefiltering Poisson regression and LOESS GAM models applied. Daily particle measures: $PM_{2.5}$, coarse particulate mass($PM_{10-2.5}$), PM_{10}, SO_4, H^+, and gaseous pollutants (O_3, NO_2, SO_2, and CO) evaluated.	Positive air pollution-HA associations found, with ozone being pollutant least sensitive to adjustment for co-pollutants. However, even after the simultaneous inclusion of O_3 in the model, the association with the respiratory hospital admissions were still significant for PM_{10}, $PM_{2.5}$, $PM_{2.5-10}$, $CoH_,$, SO_4, and H^+.	<u>Respiratory HA's all ages</u>(no co-pollutant) PM_{10} (50 µg/m^3, 4d avg. lag 0) ER = 10.6% (CI: 4.5 - 17.1) $PM_{2.5}$ (25 µg/m^3, 4d avg. lag 1) ER = 8.5% (CI: 3.4, 13.8) $PM_{2.5-10}$ (25 µg/m^3, 5d avg. lag 0) ER = 12.5% (CI: 5.2, 20.0) <u>Respiratory HA's all ages</u>(O_3 co-pollutant) PM_{10} (50 µg/m^3, 4d avg. lag 0) ER = 9.6% (CI: 3.5, 15.9) $PM_{2.5}$ (25 µg/m^3, 4d avg., lag 1) ER = 6.2% (1.0, 11.8) $PM_{2.5-10}$ (25 µg/m^3, 5d avg. lag 0) ER = 10.8% (CI: 3.7, 18.1)
Burnett et al. (1999)+ Metro-Toronto, Canada 1980-1994 Pollutant: mean, median, IQR: FP_{est} (µg/m^3): 18, 16, 10 CP_{est} (µg/m^3): 12, 10, 8 $PM_{10\ est}$ (µg/m^3): 30, 27, 15	Daily hospitalizations for asthma (493, mean 11/day), obstructive lung disease (490-492, 496, mean 5/day), respiratory infection (464, 466, 480-487, 494, mean 13/day) analyzed separately in relation to environmental covariates. Same geographic area as in Burnett et al., 1997b. Three size-classified PM metrics were <u>estimated</u>, not measured, based on a regression on TSP, SO_4, and COH in a subset of every 6th-day data. Generalized additive models. Applied with non-parametric LOESS prefilter applied to both pollution and hospitalization data. Day of week controls. Tested 1-3 day averages of air pollution ending on lags 0-2. Covariates: O_3, NO_2, SO_2, CO, temperature, dewpoint temperature, relative humidity.	In univariate regressions, all three PM metrics were associated with increases in respiratory outcome. In multi-pollutant models, there were no significant PM associations with any respiratory outcome (results not shown). Use of estimated PM metrics limits the interpretation of pollutant-specific results reported. However, results suggest that a linear combination of TSP, SO_4, and COH does not have a strong independent association with cardiovascular admissions when a full range of gaseous pollutants are also modeled.	Percent excess risk (95% CI) per 50 µg/m^3 PM_{10}; 25 µg/m^3 $PM_{2.5}$ and $PM_{(10-2.5)}$: <u>Asthma</u> $PM_{2.5}$ (0-1-2 d): 6.4 (2.5, 10.6) PM_{10} (0-1 d): 8.9 (3.7, 14.4) $PM_{10-2.5}$ (2-3-4 d): 11.1 (5.8, 16.6) <u>COPD</u> $PM_{2.5}$: 4.8 (-0.2, 10.0) PM_{10}: 6.9 (1.3, 12.8) $PM_{10-2.5}$ (2-3-4 d): 12.8 (4.9, 21.3) <u>Resp. Infection:</u> $PM_{2.5}$: 10.8 (7.2, 14.5) PM_{10}: 14.2 (9.3, 19.3) $PM_{10-2.5}$ (0-1-2 d): 9.3 (4.6, 14.2)
Burnett et al. (1997c) 16 Canadian Cities('81-91) Population=12.6 MM CoH mean=0.64(per 10^3 lin. ft) CoH IQR=0.3-0.8(per 10^3 lin ft)	Air pollution data were compared to respiratory hospital admissions (mean=1.46/million people/day) for 16 cities across Canada. Used a random effects regression model, controlling for long-wave trends, day of week, weather, and city-specific effects using a linear prefiltered random effects relative risk regression model.	The 1 day lag of 0_3 was positively associated with respiratory admissions in the April to December period, but not in the winter months. Daily maximum 1-hr. CoH from 11 cities and CO also positively associated with HA's, even after controlling for O_3.	<u>Respiratory HA's all ages (with O_3,CO)</u> CoH IQR = 0.5, lag 0: CoH ER = 3.1% (CI: 1.0-4.6%)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Canada (cont'd)			
Burnett et al. (2001b)+ Toronto, Canada 1980-1994 $PM_{2.5}$: 18 µg/m^3 $PM_{10\text{-}2.5}$: 16.2 µg/m^3 (both estimated values)	Respiratory admissions in children aged <2 years relates to mean pollution levels. O_3, NO_2, SO_2, and CO (ICD-9: 493 asthma; 466 acute bronchitis; 464.4 croup or pneumonia, 480-486). Time-series analysis adjusted with LOESS.	Summertime urban air pollution, especially ozone, increases the risk that children less than 2 years of age will be hospitalized for respiratory disease.	$PM_{2.5}$ lag 0 15.8% (t=3.29) $PM_{2.5}$ lag 0 with O_3 1.4% (0.24) $PM_{10\text{-}2.5}$ lag 1 18.3% (t=3.29) with O_3 4.5% (0.72)
Europe			
Atkinson et al. (1999a) London (92 - 94) Population = 7.2 MM PM_{10} Mean = 28.5 10^{th}-90^{th} IQR = 15.8-46.5 µg/m^3 BS mean = 12.7 µg/m^3 10^{th}-90^{th} IQR = 5.5-21.6 µg/m^3	All-age respiratory (mean=150.6/day), all-age asthma (38.7/day), COPD plus asthma in adults >64 yr. (22.9/day), and lower respiratory (64.1/day) in adults >64 yr (16.7/day) hospital admissions in London hospitals considered. Counts for ages 0-14, 15-64, and >64 yr also examined. Poisson GLM regression used, controlling for season, day-of-week, meteorology, autocorrelation, overdispersion, and influenza epidemics.	Positive associations found between respiratory-related emergency hospital admissions and PM_{10} and SO_2, but not for O_3 or BS. When SO_2 and PM_{10} included simultaneously, size and significance of each was reduced. Authors concluded that SO_2 and PM_{10} are both indicators of the same pollutant mix in this city. SO_2 and PM_{10} analyses by temperature tertile suggest that warm season effects dominate. Overall, results consistent with earlier analyses for London, and comparable with those for North America and Europe.	PM_{10} (50 µg/m^3), no co-pollutant. All Respiratory Admissions: All age (lag 1d) ER = 4.9% (CI: 1.8, 8.1) 0-14 y (lag 1d) ER = 8.1% (CI: 3.5, 12.9) 15-64y (lag 2d) ER = 6.9% (CI: 2.1, 12.9) 65+ y (lag 3d) ER = 4.9% (CI: 0.8, 9.3) Asthma Admissions: All age (lag 3d) ER = 3.4% (CI: -1.8, 8.9) 0-14 y (lag 3d) ER = 5.4% (CI: -1.2, 12.5) 15-64 y(lag 3d) ER = 9.4% (CI: 1.1, 18.5) 65+ y.(lag 0d) ER = 12% (CI: -1.8, 27.7) COPD & Asthma Admissions (65+yrs.) (lag 3d) ER = 8.6% (CI: 2.6, 15) Lower Respiratory Admissions (65+ yrs.) (lag 3d) ER = 7.6% (CI: 0.9, 14.8)
Wordley et al. (1997) Study Period: 4/92 –3/94 Birmingham, UK Population = NR PM_{10} daily values: Mean = 25.6 µg/m^3 range = 2.8, 130.9 µg/m^3 PM_{10} 3 day running. mean: Mean = 25.5 µg/m^3 range = 7.3, 104.7 µg/m^3	Relation between PM_{10} and total HA's for respiratory (mean = 21.8/d), asthma (mn.=6.2/d), bronchitis (mn.=2.4/d), pneumonia (mn.=3.4/d), and COPD (mn.=3.2/d) analyzed, using log-linear regression after adjusting for day of week, month, linear trend, RH, and T (but not T-RH interaction). RR's compared for various thresholds vs. mean risk of HA.	PM_{10} positively associated with all HA's for respiratory, asthma, bronchitis, pneumonia, and COPD. Pneumonia, all respiratory, and asthma HA's also significantly positively associated with the mean of PM_{10} over the past three days, which gave 10 to 20% greater RR's per 10 µg/m^3, as expected given smaller day to day deviations. Other air pollutants examined but not presented, as "these did not have a significant association with health outcomes independent from that of PM_{10}".	50 µg/m^3 in PM_{10} All Respiratory HA's (all ages) (lag0d) ER = 12.6% (CI: 5.7, 20) Asthma HA's (all ages) (lag2d) ER = 17.6% (CI: 3, 34.4) Bronchitis HA's (all ages) (lag0d) ER= 32.6% (CI: 4.4, 68.3) Pneumonia HA's (all ages) (lag3d) ER = 31.9% (CI: 15, 51.4) COPD HA's (all ages) (lag1d) ER = 11.5% (CI: -3, 28.2)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Europe (cont'd)			
Prescott et al. (1998) Edinburgh (10/92-6/95) Population = 0.45 MM PM_{10} mean. =20.7 µg/m^3 PM_{10} min/max=5/72 µg/m^3 PM_{10} 90th% - 10th% = 20 µg/m^3	Poisson log-linear regression models used to investigate relation of daily HA's with NO_2, O_3, CO, and PM_{10}. Adjustments made for seasonal and weekday variation, daily T (using 8 dummy variables), and wind speed. Separate analyses for age<65 yr. (mean resp HA = 3.4/day) and age >64 yr. (mean resp HA = 8.7/day), and for subjects with multiple HA's.	The two strongest findings were for cardiovascular HA's of people aged >64, which showed a positive association with PM_{10} as a mean of the 3 previous days. PM_{10} was consistently positively associated with Respiratory HA's in both age groups, with the greatest effect size in those >64, especially among those with >4 HA's during '81-'95. Weak significances likely contributed to by low population size.	Single Pollutant Models PM_{10} = 50 µg/m^3, mean of lags 1-3 Respiratory HA's (age<65) ER = 1.25 (-12.8, 17.5) Respiratory HA's (age>64) ER = 5.33 (-9.3, 22.3) Respiratory HA's (age>64, >4 HA's) ER = 7.93 (-19.0, 43.7)
McGregor et al. (1999) Birmingham, UK. Population = NR Mean PM_{10} = 30.0 µg/m^3	A synoptic climatological approach used to investigate linkages between air mass types (weather situations), PM_{10}, and all respiratory hospital admissions (mean= 19.2/day) for the Birmingham area.	Study results show distinct differential responses of respiratory admission rates to the six winter air mass types. Two of three types of air masses associated with above- average admission rates also favor high PM_{10} levels. This is suggestive of possible linkage between weather, air quality, and health.	NR
Hagen et al. (2000)+ Drammen, Sweden(11/94-12/97) Population = 110,000 PM_{10} mean = 16.8 µg/m^3 PM_{10} IQR = 9.8-20.9 µg/m^3	Examined PM_{10}, SO_2, NO_2, VOC's, and O_3 associations with respiratory hospital admissions from one hospital (mean = 2.2/day). Used Poisson GAM controlling for temperature and RH (but not their interaction), long-wave and seasonality, day-of-week, holidays, and influenza epidemics.	As a single pollutant, the PM_{10} effect was of same order of magnitude as reported in other studies. The PM_{10} association decreased when other pollutants were added to the model. However, the VOC's showed the strongest associations.	Respiratory Hospital Admissions(all ages) For IQR=50 µg/m^3 -Single Pollutant Model: PM_{10} (lag 0) ER = 18.3% (CI: -4.2, 46) -Two Pollutant Model (with O_3): PM_{10} (lag 0) ER = 18.3% (CI: -4.2, 45.4) -Two Pollutant Model (with Benzene): PM_{10} (lag 0) ER = 6.5% (CI:-14 , 31.8)
Dab et al. (1996) Paris, France (87 - 92) Population = 6.1 MM PM_{13} mean = 50.8 µg/m^3 PM_{13} 5th-95th range = 19.0-137.3 BS mean = 31.9 µg/m^3 BS 5th-95th Range =11.0-123.3	Daily mortality and general admissions to Paris public hospitals for respiratory causes were considered (means/day: all resp.=79/d, asthma=14/d, COPD=12/d). Time series analysis used linear regression model followed by a Poisson regression. Epidemics of influenza A and B, temperature, RH, holidays, day of week, trend, long-wave variability, and nurses' strike variables included. No two pollutant models considered.	For the all respiratory causes category, the authors found "the strongest association was observed with PM_{13}" for both hospital admissions and mortality, indicating a coherence of association across outcomes. Asthma was significantly correlated with NO_2 levels, but not PM_{13}.	For PM_{13} = 50 µg/m^3; BS = 25 µg/m^3; Respiratory HA's (all ages): PM_{13} Lag 0 ER = 2.2% (CI: 0.2, 4.3) BS Lag 0 ER = 1.0% (0.2, 1.8) COPD HA's (all ages): PM_{13} Lag 2 ER = 2.3% (CI: -6.7, 2.2) BS Lag 2 ER = 1.1% (-2.9, 0.6) Asthma HA's (all ages): PM_{13} Lg 2 ER = 1.3% (CI: -4.6, 2.2) BS Lg 0 ER = 1.2% (-0.5, 2.9)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Europe (cont'd)			
Anderson et al. (1997) Amsterdam(77 - 89) Barcelona (86- 92) London (87 - 91) Milan (80- 89) Paris (87 - 92) Rotterdam (77 - 89) Populations .= 0.7(A), 1.7(B), 7.2(L),1.5(M),6.5(P),0.6(R)MM BS Means = 6, 41, 13, -, 26, 22 TSP Means = 41,155, -, 105, -,41	All-age daily hospital admissions (HA's) for COPD considered in 6 APHEA cities; Mean/day = 1.1(A), 11(B), 20(L), 5(M), 11(P), 1.1(R). Poisson GLM regression controlling for day of week, holidays, seasonal and other cycles, influenza epidemics, temperature, RH, and autocorrelation. Overall multi-city estimates made using inverse variance wts., allowing for inter-city variance.	Ozone gave the most consistent associations across models. Multi-city meta-estimates also indicated associations for BS and TSP. The warm/cold season RR differences were important only for ozone, having a much stronger effect in the warm season. COPD effect sizes found were much smaller than in U.S. studies, possibly due to inclusion of non-emergency admissions or use of less health-relevant PM indices.	BS (25 $\mu g/m^3$) 1d lag, no co-pollutant: All Age COPD Hospital Admissions ER = 1.7% (0.5, 2.97) TSP (100 $\mu g/m^3$) 1d lag, no co-pollutant: All Age COPD Hospital Admissions ER = 4.45% (CI: -0.53, 9.67)
Díaz et al. (1999) Madrid (94 - 96) Population = NR TSP mean 40 $\mu g/m^3$	ARIMA modeling used to analyze emergency respiratory and circulatory admissions (means/day=7.8,7.6) from one teaching hospital. Annual, weekly, and 3 day periodicities controlled, but no time trend included, and temperature crudely fit with v-shaped linear relationship.	Although TSP correlated at zero lag with admissions in winter and year-round, TSP was never significant in ARIMA models; so effect estimates not reported for TSP. Also, found biologically implausible u-shaped relationship for O_3, possibly indicating unaddressed temperature effects.	N/A
Spix et al. (1998) London (L) (87 - 91) Pop. =7.2 Million (MM) BS Mean = 13 $\mu g/m^3$ Amsterdam (A) (77 - 89) Pop. =0.7 MM BS Mean = 6 $\mu g/m^3$ TSP mean = 41 $\mu g/m^3$ Rotterdam (R) (77 - 89) Pop. =0.6MM BS Mean = 22 $\mu g/m^3$ TSP mean = 41 $\mu g/m^3$ Paris (P) (87 - 92), Pop.= 6.14 MM BS Mean = 26 $\mu g/m^3$ Milano (M) (80 - 89) Pop. = 1.5 MM TSP Mean =120 ($\mu g/m^3$)	Respiratory (ICD9 460-519) HA's in age groups 15-64 yr and 65 + yrs. related to SO_2, PM (BS or TSP), O_3, and NO_2 in the APHEA study cities using standardized Poisson GLM models with confounder controls for day of week, holidays, seasonal and other cycles, temperature, RH, and autocorrelation. PM lag considered ranged from 0-3 day, but varied from city to city. Quantitative pooling conducted by calculating the weighted means of local regression coefficients using a fixed-effects model when no heterogeneity could be detected; otherwise, a random-effects model employed.	Pollutant associations noted to be stronger in areas where more than one monitoring station was used for assessment of daily exposure. The most consistent finding was an increase of daily HA's for respiratory diseases (adults and elderly) with O_3. The SO_2 daily mean was available in all cities, but SO_2 was not associated consistently with adverse effects. Some significant PM associations were seen, although no conclusion related to an overall particle effect could be drawn. The effect of BS was significantly stronger with high NO_2 levels on the same day, but NO_2 itself was not associated with HA's. Authors concluded that "there was a tendency toward an association of respiratory admissions with BS, but the very limited number of cities prevented final conclusions."	Respiratory Admissions (BS = 25 $\mu g/m^3$) BS (L, A, R, P) 15-64 yrs: 1.4% (0.3, 2.5) 65+ yrs: 1.0% (-0.2, 2.2) TSP (A, R, M) (100 $\mu g/m^3$) 15-64 yrs: 2.0 (-2.1, 6.3) 65+ yrs: 3.2 (-1.2, 7.9) Respiratory HA's BS (L, A, R, P): Warm (25 $\mu g/m^3$) 15-64 yrs: -0.5% (-5.2, 4.4) 65+ yrs: 3.4% (-0.1, 7.1) BS (L, A, R, P): Cold (25 $\mu g/m^3$) 15-64 yrs: 2,0% (0.8, 3.2) 65+ yrs: 0% (-2.2, 2.3) TSP (A, R, M): Warm (100 $\mu g/m^3$) 15-64 yrs: 6.1% (0.1, 12.5) 65+ yrs: 2.0% (-3.9, 8.3) TSP (A, R, M): Cold (100 $\mu g/m^3$) 15-64 yrs: -5.9% (-14.2, 3.2) 65+ yrs: 4.0% (-0.9, 9.2)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Europe (cont'd)			
Vigotti et al. (1996) Study Period.: 80 - 89 Milan, IT Population = 1.5 MM TSP mean = 139.0 µg/m^3 TSP IQR = 82.0, 175.7 µg/m^3	Association between adult respiratory HA's (15-64 yr mean =11.3/day, and 65 + yr mean =8.8/day) and air pollution evaluated, using the APHEA protocol. Poisson regression used with control for weather and long term trend, year, influenza epidemics, and season	Increased risk of respiratory HA was associated with both SO_2 and TSP. The relative risks were similar for both pollutants. There was no modification of the TSP effect by SO_2 level. There was a suggestion of a higher TSP effect on hospital admissions in the cool months.	Young Adult (15-64 yrs.) Resp. HA's 100 µg/m^3 increase in TSP Lag 2 ER = 5% (CI: 0, 10) Older Adult (65+ yrs.) Resp. HA's 100 µg/m^3 increase in TSP Lag 1 ER = 5% (CI: -1, 10)
Anderson et al. (1998) London (87 - 92) Population = 7.2 MM BS daily mean = 14.6 µg/m^3 BS 25-75th IQR = 24-38	Poisson GLM log-linear regression used to estimate the RR of London daily asthma hospital admissions associated with changes in O_3, SO_2, NO_2 and particles (BS) for all ages and for 0-14 yr. (mean=19.5/d), 15-64 yr. (mean=13.1/d) and 65 + yr. (mean =2.6/d). Analysis controlled for time trends, seasonal factors, calendar effects, influenza epidemics, RH, temperature, and auto-correlation. Interactions with co-pollutants and aeroallergens tested via 2 pollutant models and models with pollen counts (grass, oak and birch).	Daily hospital admissions for asthma found to have associations with O_3, SO_2, NO_2, and particles (BS), but there was lack of consistency across the age groups in the specific pollutant. BS association was strongest in the 65 + group, especially in winter. Pollens not consistently associated with asthma HA's, sometimes being positive, sometimes negative. Air pollution associations with HA's not explained by airborne pollens in simultaneous regressions, and there was no consistent pollen-pollutant interaction.	Asthma Admissions. BS=25 µg/m^3 BS Lag = 0-3 day average concentration All age ER = 5.98% (0.4, 11.9) <15yr. ER = 2.2% (-4.6, 9.5) 15-64yr ER = 1.2% (-5.3, 8.1) 65+ yr. ER = 22.8% (6.1, 42.5) BS=50 µg/m^3, 2d lag & co-pollutant: Older Adult (>64 yrs.) Asthma Visits: BS alone: ER = 14.6% (2.7, 27.8) &O_3: ER = 20.0% (3.0, 39.8) & NO_2: ER = 7.4% (-8.7, 26.5) SO_2: ER = 11.8% (-2.2, 27.8)
Kontos et al. (1999) Piraeus, Athens GR (87 - 92) Population = NR BS mean =46.5 µg/m^3 BS max =200 µg/m^3	Relation of respiratory HA's for children (0-14 yrs.) (mean = 4.3/day) to BS, SO_2, NO_2, and O_3 evaluated, using a nonparametric stochastic dynamical system approach and frequency domain analyses. Long wave and effects of weather considered, but non-linearity and interactions of T and RH relation with HA's not addressed.	Pollution found to explain significant portion of the HA variance. Of pollutants considered, BS was consistently among most strongly explanatory pollutants across various reported analyses.	NR

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Europe (cont'd)			
Ponce de Leon et al. (1996) London (4/87-2/92) Population = 7.3 million BS mean. =14.6 µg/m^3 BS 5th-95th %=6 - 27 µg/m^3	Poisson GLM log-linear regression analysis of daily counts of HA's (means/day: all ages=125.7; Ages 0-14=45.4; Ages 15-64=33.6; Ages 65+=46.7). Effects of trend, season and other cyclical factors, day of the week, holidays, influenza epidemic, temperature, humidity, and autocorrelation addressed. However, temperature modeled as linear, with no RH interaction. Pollution variables were BS, SO_2, O_3, and NO_2, lagged 0-3 days.	O_3 associated with increase in daily HA's, especially in the "warm" season. However, u-shape of the O_3 dose-response suggests that linear temperature control was not adequate. Few significant associations with other pollutants, but these tended to be positive (especially in cold season, Oct-March, and for older individuals for BS).	<u>Respiratory HA's (all ages)</u> Single Pollutant Models For Oct-Mar. BS = 25 µg/m^3 Lag 1 ER = 0.2% (–1.9, 2.3) For Apr-Sep. BS = 25 µg/m^3 Lag 1 ER = -2.7% (-6.0, 0.8) <u>Respiratory HA's (>65)</u> Single Pollutant Models For Oct-Mar. BS = 25 µg/m^3 Lag 2 ER = 1.2% (-2.1, 4.5) For Apr-Sep. BS = 25 µg/m^3 Lag 2 ER = 4.5% (-1.0, 10.4)
Schouten et al. (1996) Amsterdam/Rotterdam (77 - 89) Amsterdam Pop. = 0.69 Million Rotterdam Pop. = 0.58 Million Amsterdam, NE BS mean. =11 µg/m^3 BS 5th-95th% = 1 - 37 µg/m^3 Rotterdam, NE BS mean. =26 µg/m^3 BS 5th-95th %= 6 -61 µg/m^3	Daily emergency HA's for respiratory diseases (ICD 460-519), COPD (490-492, 494, 496), and asthma (493). The mean HA/d (range) for these were: 6.70 (0-23), 1.74 (0-9) and 1.13 (0-7) respectively in Amsterdam and 4.79 (0-19), 1.57 (0-9), and 0.53 (0-5) in Rotterdam. HA associations with BS, O_3, NO_2, and SO_2 analyzed, using autoregressive Poisson GLM regression allowing for overdispersion and controlling for season, day of week, meteorological factors, and influenza epidemics.	BS did not show any consistent effects in Amsterdam; but in Rotterdam BS was positively related to HA's. Most consistent BS associations in adults >64 yrs. in winter. Positive O_3 association in summer in people aged >64 in Amsterdam and Rotterdam. SO_2 and NO_2 did not show any clear effects. Results not changed in pollutant interaction analyses. The authors concluded short-term air pollution-emergency HA's association is not always consistent at these individual cities' relatively low counts of daily HA's and low levels of air pollution. Analyses for all ages of all the Netherlands gave a strong BS-HA association in winter.	Single Pollutant Models For BS=25 µg/m^3, 2 day lag For all of the Netherlands: <u>Respiratory HA's (all ages)</u> Winter: ER = 2.0% (-1.5, 5.7) Summer: ER = 2.4% (0.6, 4.3)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Europe (cont'd)			
Sunyer et al. (1997) Barcelona (86 - 92) Population = NR BS Median: 40 µg/m^3 BS Range: 11-258 (B Helsinki (86 - 92) Population = NR BS Median: - BS Range: - Paris (86 - 92) Population = NR BS Median: 28 µg/m^3 BS Range: 4-186 µg/m^3 London (86 - 92) Population = NR BS Median: 13 µg/m^3 BS Range: 3-95 µg/m^3	Evaluated relations of BS, SO_2, NO_2, and O_3 to daily counts of asthma HA's and ED visits in adults [ages 15-64 years: mean/day = 3.9 (B); 0.7 (H); 13.1 (H); 7.3 (P)] and children [ages < 15 years: mean/day = 0.9 (H); 19.8 (L); 4.6 (P)]. Asthma (ICD9=493) studied in each city, but the outcome examined differed across cities: ED visits in Barcelona; emergency hospital asthma admissions in London and Helsinki, and total asthma admissions in Paris. Estimates from all cities obtained for entire period and also by warm or cold seasons, using Time-series GLM regression, controlling for temperature and RH, viral epidemics, day of week effects, and seasonal and secular trends applied using the APHEA study approach. Combined associations were estimated using meta-analysis.	Daily admissions for asthma in adults increased significantly with increasing ambient levels of NO_2, and positively (but non-significantly) with BS. The association between asthma admissions and pollution varied across cities, likely due to differing asthma outcomes considered. In children, daily admissions increased significantly with SO_2 and positively (but non-significantly) with BS and NO_2, though the latter only in cold seasons. No association observed in children for O_3. Authors concluded that "In addition to particles, NO_2 and SO_2 (by themselves or as a constituent of a pollution mixture) may be important in asthma exacerbations".	ER per 25 µg/m^3 BS (24 h Average) <u>Asthma Admissions/Visits:</u> <15 yrs.: London ER = 1.5% (lg 0d) Paris ER = 1.5% (lg 2d) Total ER = 1.5% (-1.1, 4.1) 15-64 yrs: Barcelona ER = 1.8% (lg 3d) London ER = 1.7% (lg 0d) Paris ER = 0.6% (lg 0d) Total ER = 1.0% (-0.8, 2.9) <u>Two Pollutant (per 25 µg/m^3 BS)</u> <u>Asthma Admissions (24 h Avg)</u> <15 yrs, (BS & NO_2): London ER = 0.6% (lg 0d) Paris ER = 2.9% (lg 2d) Total ER = 1.8% (-0.6, 4.3) <15 yrs, (BS & SO_2): London ER = -1.1% (lg 0d) Paris ER = -1.4% (lg 2d) Total ER = -1.3 (-5.0, 2.5) 15-64 yrs, (BS & NO_2): Barcelona ER = 1.5% (lg 0d) London ER = -4.7% (lg 0d) Paris ER = -0.7% (lg 1d) Total ER = -0.5% (-5.1, 4.4)
Tenías et al (1998) Study Period.: 94 - 95 Valencia, Spain Hosp. Cachment Pop. =200,000 BS mean = 57.7 µg/m^3 BS IQR = 25.6-47.7 µg/m^3	Associations between adult (14+ yrs.) emergency asthma ED visits to one city hospital (mean =1.0/day) and BS, NO_2, O_3, SO_2 analyzed, using GLM Poisson auto-regressive modeling, controlling for potential confounding weather and time (e.g., seasonal) and trends using the APHEA protocol.	Association with asthma was positive and more consistent for NO_2 and O_3 than for BS or SO_2. Suggests that secondary oxidative-environment pollutants may be more asthma relevant than primary reduction-environment pollutants (e.g., carbonaceous particles). NO_2 had greatest effect on BS in co-pollutant models, but BS became significant once 1993 was added, showing power to be a limitation of this study.	<u>Adult Asthma HA's</u>, BS = 25 µg/m^3 For 1993-1995: Lag 0 ER = 10.6% (0.9, 21.1) For 1994-1995: Lag 0 ER = 6.4% (-4.8, 18.8)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Europe (cont'd)			
Anderson et al. (2001) West Midland, England (October 1994-December 1996) Population = 2.3 million PM_{10} mean = 23.3 µg/m^3 $PM_{2.5}$ mean = 14.5 µg/m^3 $PM_{10-2.5}$ = 9.0 µg/m^3 (by subtraction)	Respiratory hospital admissions (mean = 66/day) related to PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, BS, SO_4^-, NO_2, O_3, SO_2, CO. GLM regression with quasi-likelihood approach, controlling for seasonal patterns, temp, humidity, influenza episodes, day week. Adjusted for residual serial correlation and over-dispersion.	Respiratory admissions (all ages) not associated with any pollutant. Analyses by age revealed some associations to PM_{10} and $PM_{2.5}$ and respiratory admissions in the 0-14 age group. There was a striking seasonal interaction in the cool season versus the warm season. $PM_{10-2.5}$ effects cannot be excluded. Two pollutant models examined particulate measures. $PM_{2.5}$ effects reduced by inclusion of black smoke.	Respiratory HA - lag 0+1 days PM_{10} Increment10-90% (11.4-38.3 µg/m^3) All ages: 1.5 (-0.7 to 3.6) Ages 0-14: 3.9 (0.6 to 7.4) Ages 15-64: 0.1 (-4.0 to 4.4) Ages 65: -1.1 (-4.3 to 2.1) $PM_{2.5}$ (6.0-25.8) All ages: 1.2 (-0.9 to 3.4) Ages 0-14: 3.4 (-0.1 to 7.0) Ages 15-64: -2.1 (-6.4 to 2.4) Ages 65: -1.3 (-4.7 to 2.2) $PM_{10-2.5}$ (4.1 to 15.2) All ages: 0.2 (-2.5 to 3.0) Ages 0-14: 4.4 (-0.3 to 9.4) Ages 15-64: -4.9 (-9.9 to 0.4) Ages 65: -1.9 (-6.0 to 2.5) COPD (ICD-9 490-492, 494-496) PM_{10} Age 65: -1.8 (-6.9 to 3.5) $PM_{2.5}$ Age 65: -3.9 (-9.0 to 1.6) $PM_{10-2.5}$ Age 65: -1.7 (-8.9 to 5.3) Asthma (ICD- 9-493) (mean lag 0+1) PM_{10} Ages 0-14: 8.3 (1.7 to 15.3) Ages 15-64: -2.3 (-10.0 to 6.1) $PM_{2.5}$ Ages 0-14: 6.0 (-0.9 to 13.4) Ages 15-64: -8.4 (-16.4 to 0.3) $PM_{10-2.5}$ Ages 0-14: 7.1 (-2.1 to 17.2) Ages 15-64: -10.7 (-19.9 to -0.5)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Europe (cont'd)			
Atkinson et al. (2001)+ Eight city study: Median/range Barcelona 1/94 - 12/96 PM_{10} 53.3 µg/m^3 (17.1, 131.7) Birmingham 3/92 -12/94 PM_{10} 21.5 µg/m^3 (6.5, 115) London 1/92 - 12/94 PM_{10} 24.9 µg/m^3 (7.2, 80.4) Milan -No PM_{10} Netherlands 1/92 - 9/95 PM_{10} 33.4 µg/m^3 (11.3, 130.8) Paris 1/92 - 9/96 PM_{10} 20.1 µg/m^3 (5.8, 80.9) Rome - No PM_{10} Stockholm 3/94 - 12/96 PM_{10} 13.6 µg/m^3 (4.3, 43.3)	As part of the APHEA 2 project, association between PM_{10} and daily counts of emergency hospital admissions for Asthma (0-14 and 15-64 yrs), COPD and all-respiratory disease (65+ yrs) regressed using GAM, controlling for environmental factors and temporal patterns.	This study reports that PM was associated with daily admissions for respiratory disease in a selection of European cities. Average daily ozone levels explained a large proportion of the between-city variability in the size of the particle effect estimates in the over 65 yr age group. In children, the particle effects were confounded with NO_2 on a day-to-day basis.	For 10 µg/m^3 increase **Asthma Admission Age 0-14 yrs**: PM_{10} for cities ranged from -0.9% (-2.1, 0.4) to 2.8% (0.8, 4.8) with an overall effect estimate of 1.2% (0.2, 2.3) **Asthma Admission Age 15-64 yrs:** Overall PM 1.1% (0.3, 1.8) **Admission of COPD and Asthma Age 65+ years:** Overall PM 1.0% (0.4, 1.5) **Admission All Respiratory Disease Age 65+ years:** Overall PM 0.9% (0.6, 1.3)
Thompson et al. (2001) Belfast, Northern Ireland 1/1/93 – 12/31/95. PM_{10} µg/m^3 mean (SD) May – October 24.9 (13.7) November – April 31.9 (24.3)	The rates of acute asthma admission to children's emergency was studied in relation to day-to-day fluctuation of PM_{10} and other pollutants using GLM Poisson regression.	A weak, but significant association between PM10 concentration and asthma emergency-department admissions was seen. After adjusting for multiple pollutants only the benzene level was independently associated with asthma emergency department admission. Benzene was highly correlated to PM_{10}, SO_2 and NO_2 levels.	—
Fusco et al. (2001)+ Rome, Italy 1995-1997 PM – suspended particles measured	Daily counts of hospital admissions for total respiratory conditions, acute respiratory infection including pneumonia, COPD, and asthma was analyzed in relation to PM measures and gaseous pollutants using generalized additive GAM models controlling for mean temperature, influenza, epidermics, and other factors using spline smooths.	No effect was found for PM. Total respiratory admission were significantly associated with same-day level of NO_2 and CO. There was no indication that the effects of air pollution were present at lags >2 days. Among children, total respiratory and asthma admissions were strongly associated with NO_2 and CO. Multipollutant model analysis yielded weaker and more unstable results.	—

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Latin America			
Braga et al. (1999) São Paulo, Brazil (92 - 93) Population = NR PM_{10} mean = 66.3 µg/m^3 PM_{10} Std. Deviation = 26.1 PM_{10} Min./Max. = 26.7/165.4	Pediatric (<13 yrs.) hospital admissions (mean=67.6/day) to public hospitals serving 40% of the population were regressed (using both GLM and GAM) on air pollutants, controlling for month of the year, day-of-week, weather, and the daily number of non-respiratory admissions (mean=120.7/day). Air pollutants considered included PM_{10}, O_3, SO_2, CO, and NO_2.	PM_{10} and O_3 were the two pollutants found to exhibit the most robust associations with respiratory HA's. SO_2 showed no correlation at any lag. Simultaneous regression of respiratory HA's on PM_{10}, O_3, and CO decreased effect estimates and their significance, suggesting that "there may not be a predominance of any one pollutant over the others". Associations ascribed primarily to auto emissions by the authors.	PM_{10} (50 µg/m^3), no-co-pollutant Respiratory Hospital Admissions (<13 yr.) GLM Model: (0-5day lg avg.) ER = 8.9% (CI: 4.6, 13.4) GAM Model (0-5day lg avg.) ER = 8.3% (CI: 4.1, 12.7)
Gouveia and Fletcher (2000) Study Period. 92-94 Sao Paulo, Brazil Population = 9.5 MM x 66% PM_{10} mean = 64.9 µg/m^3 PM_{10} IQR = 42.9-75.5 µg/m^3 $PM_{10}10/90^{th}$% =98.1 µg/m^3 PM_{10} 95^{th}% = 131.6 µg/m^3	Daily public hospital respiratory disease admissions for children (mean resp. < 5y = 56.1/d; mean pneumonia <5y =40.8/d; mean asthma <5 y = 8.5/d; mean pneum.<1y=24.0) and daily levels air pollutants (PM_{10}, SO_2, NO_2, O_3, and CO) and were analyzed with Poisson regression. GLM Models adjusted for time trends, seasonal patterns, weekdays, holidays, weather, and serial correlation. PM_{10} measured by Beta-gauge. Private hospitals serving wealthier citizens not in database.	Children's HA's for total respiratory and pneumonia positively associated with O_3, NO_2, and PM_{10}. Effects for pneumonia greater than for all respiratory diseases. Effects on infants (<1 yr. old) gave higher estimates. Similar results for asthma, but estimates higher than for other causes. Results noted to agree with other reports, but smaller RR's. This may be due to higher baseline admission rates in this poor sub-population vs. other studies, but this was not intercompared by the authors.	PM_{10} = 50 µg/m^3: All Respiratory HA's for children < 5yrs. ER = 2.0% (-0.8, 4.9) Pneumonia HA's for children <5 yrs. ER = 2.5% (-0.8, 6.0) Asthma HA's for children <5 yrs. ER = 2.6% (-4.0, 9.7) Pneumonia HA's for children <1 yrs. ER = 4.7% (0.7, 8.8)
Rosas et al. (1998) SW Mexico City (1991) Population = NR PM_{10} mean. =77 µg/m^3 PM_{10} min/max= 25/183 µg/m^3	Log-regression GLM analysis of relations between emergency hospital admissions for asthma for children <15 yrs (mean=2.5/day), adults (mean=3.0/day), and adults >59 yrs (mean=0.65/day) and lag 0-2 d pollen, fungal spores, air pollutants (O_3, NO_2, SO_2, and PM_{10}) and weather factors. Long wave controlled only by separating the year into two seasons: "dry" and "wet". Day-of-week not included in models.	Few statistical associations were found between asthma admissions and air pollutants. Grass pollen was associated with child and adult admissions, and fungal spores with child admissions. Authors conclude that aeroallergens may be more strongly associated with asthma than air pollutants, and may act as confounding factors in epidemiologic studies. Results are limited by low power and the lack of long-wave auto-correlation controls in the models.	NR

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Australia			
Morgan et al. (1998) Sydney, AU (90 - 94) Population = NR $PM_{2.5}$ 24 h mean = 9.6 µg/m^3 $PM_{2.5}$ 10th-90th% = 3.6-18 µg/m^3 $PM_{2.5}$ max-1 h mean = 22.8 µg/m^3 $PM_{2.5}$ 10th-90th% = 7.5-44.4 µg/m^3	A Poisson analysis, controlled for overdispersion and autocorrelation via generalized estimating equations (GEE), of asthma (means: 0-14 yrs.=15.5/day; 15-64=9/day), COPD (mean 65+yrs =9.7/day), and heart disease HA's. $PM_{2.5}$ estimated from nephelometry. Season and weather controlled using dummy variables.	Childhood asthma was primarily associated with NO_2, while COPD was associated with both NO_2 and PM. 1-hr. max $PM_{2.5}$ more consistently positively related to respiratory HA's than 24-h avg $PM_{2.5}$. Adding all other pollutants lowered PM effect sizes, although pollutant inter-correlations makes many pollutant model interpretations difficult. No association found between asthma and O_3 or PM. The authors cited the error introduced by estimating $PM_{2.5}$ and the low PM levels as possible reasons for the weak PM-respiratory HA associations.	<u>Asthma HA's</u> <u>Single Pollutant Model:</u> For 24 hr $PM_{2.5}$ = 25 µg/m^3 1-14 yrs.(lag1) ER = -1.5% (CI: -7.8, 5.3) 15-64 yrs.(lag0) ER = 2.3% (CI: -4, 9) For 1h $PM_{2.5}$ =25 µg/m^3 1-14 yrs.(lag1) ER = + 0.5% (CI: -1.9, 3.0) 15-64 yrs.(lag0) ER = 1.5% (CI: -0.9, 4) <u>Multiple Pollutant Model:</u> For 24h $PM_{2.5}$ = 25 µg/m^3 1-14 yrs.(lag1) ER = -0.6% (CI: -7.4, 6.7) <u>COPD (65+yrs.)</u> Single Pollutant Model: For 24h $PM_{2.5}$ = 25 µg/m^3 (lag 0) ER =4.2% (CI: -1.5, 10.3) For 1h $PM_{2.5}$ = 25 µg/m^3 (lag 0) ER = 2% (CI: -0.3, 4.4) <u>Multiple Pollutant Model:</u> For 1h $PM_{2.5}$ = 25 µg/m^3 (lag 0) ER = 1.5% (CI: -0.9, 4)
Asia			
Tanaka et al. (1998) Stdy Pd.:1/92-12/93 Kushiro, Japan Pop. = 102 adult asthmatics PM_{10} mean = 24.0 µg/m^3 PM_{10} IQR = NR	Associations of HA's for asthma (in 44 non-atopic and 58 atopic patients) with weather or air pollutants (NO, NO_2, SO_2,PM_{10}, O_3, and acid fog) evaluated. Odds ratios (OR) and 95% CI's calculated between high and low days for each environmental variable. Poisson GLM regression was performed for the same dichototomized variables.	Only the presence of acid fog had a significant OR >1.0 for both atopics and non-atopics. PM_{10} associated with a reduction in risk (OR<1.0) for both atopics and non-atopics. Poisson regression gave a non-significant effect by PM_{10} on asthma HA's. However, no long-wave or serial auto-correlation controls applied, so the opposing seasonalities of PM vs. HA's indicated in time series data plots are likely confounding these results.	For same-day (lag=0) PM_{10} Adult Asthma HA's OR for <30 vs. >30 µg/m^3 PM_{10}: Non-atopic OR = 0.77 (CI: 0.61, 0.98) Atopic OR = 0.87 (CI: 0.75, 1.02) Poisson Coefficient for PM_{10} > 30 µg/m^3 Non-atopic = -0.01 (SE = 0.15) Atopic = -0.002 (SE = 0.09)

TABLE 8B-2 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY HOSPITAL ADMISSIONS STUDIES

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Asia (cont'd)			
Wong et al. (1999a) Study Period.: 94 - 95 Hong Kong Population = NR PM_{10} mean = 50.1 µg/m^3 PM_{10} median = 45.0 µg/m^3 PM_{10} IQR = 30.7, 65.5 µg/m^3	Poisson GLM regression analyses were applied to assess association of daily NO_2, SO_2, O_3, and PM_{10} with emergency HA's for all respiratory (median = 131/day) and COPD (median = 101/day) causes. Effects by age groups (0-4, 5-64, and 65+ yrs.) also evaluated. Using the APHEA protocol, models accounted for time trend, season and other cyclical factors, T, RH, autocorrelation and overdispersion. PM_{10} measured by TEOM, which likely underestimates mass.	Positive associations were found for HA's for all respiratory diseases and COPD with all four pollutants. PM_{10} results for lags 0-3 cumulative. Admissions for asthma, pneumonia, and influenza were associated with NO_2, O_3, and PM_{10}. Those aged > or = 65 years were at higher risk, except for PM_{10}. No significant respiratory HA interactions with PM_{10} effect were found for high NO_2, high O_3, or cold season.	PM_{10} = 50 µg/m^3 (Lags = 0-3 days) Respiratory HA's All age: ER = 8.3% (CI: 5.1, 11.5) 0-4yrs.: ER = 9.9% (CI: 5.4, 14.5) 5-64yrs.: ER = 8.8% (CI: 4.3, 13.4) 65+ yrs.: ER = 9.3% (CI: 5b.1, 13.7) Asthma HA's (all ages) ER = 7.7% (1.0, 14.9) COPD HA's (all ages) ER = 10.0% (5.6, 14.3) Pneumonia and Influenza HA's (all ages) ER = 13.1% (7.2, 19.4)

+ = Used GAM with multiple smooths, but have not yet reanalyzed. * = Used S-Plus Default GAM, and have reanalyzed results.
GAM=Generalized Additive Model, GLM=Generalized Linear Model; NS= Natural Spline, PS=Penalized Spline.

Appendix 8B.3: PM-Respiratory Visits Studies

TABLE 8B-3. ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY MEDICAL VISITS

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States			
Choudhury et al. (1997) Anchorage, Alaska (90 - 92) Population = 240,000 PM_{10} mean = 41.5 µg/m^3 PM_{10} (SD) = 40.87 PM_{10} maximum=565 µg/m^3	Using insurance claims data for state employees and dependents living in Anchorage, Alaska, number of daily medical visits determined for asthma (mean = 2.42/day), bronchitis, and upper respiratory infections. Used GLM regression, including a time-trend variable, crude season indicator variables (i.e., spring, summer, fall, winter), and a variable for the month following a volcanic eruption in 1992.	Positive association observed between asthma visits and PM_{10}. Strongest association with concurrent-day PM_{10} levels. No co-pollutants considered. Temperature and RH did not predict visits, but did interact with the PM_{10} association. Morbidity relative risk higher with respect to PM_{10} pollution during warmer days.	Asthma Medical Visits (all ages): For mean = 50 µg/m^3 PM_{10} (single poll.) Lag = 0 days ER = 20.9% (CI: 11.8, 30.8)
Lipsett et al. (1997) Santa Clara County, CA Population = NR (Winters 88 - 92) PM_{10} mean = 61.2 µg/m^3 PM_{10} Min/Max = 9/165 µg/m^3	Asthma emergency department (ER) visits from 3 acute care hospitals (mean=7.6/day) related to CoH, NO_2, PM_{10}, and O_3 using Poisson GLM model with long-wave, day of week, holiday, and weather controls (analysis stratified by minimum T). Analyses using GAM also run for comparison. Every other day PM_{10} estimated from CoH. Residential wood combustion (RWC) reportedly a major source of winter PM. Gastro-enteritis (G-E) ER admissions also analyzed as a control disease.	Consistent relationships found between asthma ER visits and PM_{10}, with greatest effect at lower temperatures. Sensitivity analyses supported these findings. For example, .GAM model gave simi.lar, though sometimes less significant, results. NO_2 also associated, but in simultaneous regressions only PM_{10} stayed associated. ER visits for gastroenteritis not significantly associated with air pollution. Results demonstrate an association between wintertime ambient PM_{10} and asthma exacerbations in an area where RWC is a principal PM source.	Asthma ED Visits (all ages) PM_{10} = 50 µg/m^3 (2 day lag): GLM Results: At 20 F, ER = 34.7% (CI: 16, 56.5) At 30 F, ER = 22% (CI: 11, 34.2) At 41 F, ER = 9.1% (CI: 2.7, 15.9)
Norris et al. (1999)+ Seattle, WA (9/95-12/96) Pop. Of Children <18= 107,816 PM_{10} mean. =21.7 µg/m^3 PM_{10} IQR = 11.6 µg/m^3 $_{sp}$ mean = 0.4 m 1/10 4 (12.0 µg/m^3 $PM_{2.5}$) $_{sp}$ IQR = 0.3 m 1/10 4 (= 9.5 µg/m^3 $PM_{2.5}$)	The association between air pollution and childhood (<18 yrs.) ED visits for asthma from the inner city area with high asthma hospitalization rates (0.8/day, 23/day/10K persons) were compared with those from lower hospital utilization areas(1.1/day, 8/day/10K persons). Daily ED counts were regressed against PM_{10}, light scattering ($_{sp}$), CO, SO_2, and NO_2 using a semiparametric S-Plus Poisson regression model with spline smooths for season and weather variables, evaluated for over-dispersion and auto-correlation.	Associations found between ED visits for asthma in children and fine PM and CO. CO and PM_{10} highly correlated with each other (r=.74) and K, an indicator of woodsmoke pollution. There was no stronger association between ED visits for asthma and air pollution in the higher hospital utilization area than in the lower utilization area in terms of RR's. However, considering baseline risks/10K population indicates a higher PM attributable risk (AR) in the inner city.	Children's (<18 yrs.) Asthma ED Visits Single Pollutant Models: 24h PM_{10} =50 µg/m^3 Lag1 ER = 75.9% (25.1, 147.4) For 25 µg/m^3 $PM_{2.5}$ Lag1 ER = 44.5% (CI: 21.7, 71.4) Multiple Pollutant Models: 24h PM_{10} =50 µg/m^3 Lag1 ER = 75.9% (CI: 16.3, 166) For 25µg/m^3 $PM_{2.5}$ Lag1 ER = 51.2% (CI: 23.4, 85.2)

TABLE 8B-3 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY MEDICAL VISITS

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States (cont'd)			
Norris et al. (2000)+ Spokane, WA (1/95 - 3/97) Population = 300,000 PM_{10} mean. = 27.9 µg/m^3 PM_{10} Min/Max =4.7/186.4 µg/m^3 PM_{10} IQR = 21.4 µg/m^3 Seattle, WA (9/95 - 12/96) Pop. Of Children <18 = 107,816 PM_{10} mean. = 21.5 µg/m^3 PM_{10} Min/Max = 8/69.3 µg/m^3 PM_{10} IQR = 11.7 µg/m^3	Associations investigated between an atmospheric stagnation index (# of hours below median wind speed), a "surrogate index of pollution", and asthma ED visits for persons <65 yr. (mean=3.2/d) in Spokane and for children <18 yr. (mean=1.8/d) in Seattle. Poisson GAM model applied, controlling for day of week, long-wave effects, and temperature and dew point (as non-linear smooths). Factor Analysis (FA) applied to identify PM components associated with asthma HA's.	Stagnation persistence index was strongly associated with ED visits for asthma in both cities. Factor analysis indicated that products of incomplete combustion (especially wood-smoke related K, OC, EC, and CO) are the air pollutants driving this association. Multi-pollutant models run with "stagnation" as the "co-pollutant" indicated importance of general air pollution over any single air pollutant index, but not of the importance of various pollutants relative to each other.	<u>Asthma ED Visits</u> Single Pollutant Models Persons<65 years (Spokane) For PM_{10} IQR = 50 µg/m^3 Lag 3 ER = 2.4% (CI: -10.9, 17.6) Persons<18 years (Seattle) For PM_{10} IQR = 50 µg/m^3 Lag 3 ER = 56.2% (95 CI: 10.4 , 121.1)
Tolbert et al. (2000b) Atlanta, GA (92 - 94 Summers) Population = 80% of children in total population of 3 million PM_{10} mn. (SE) = 38.9 (15.5) µg/m^3 PM_{10} Range = 9, 105 µg/m^3	Pediatric (<17 yrs. of age) ED visits (mean = 467/day) related to air pollution (PM_{10}, O_3, NO_x, pollen and mold) using GEE and logistic regression and Bayesian models. Autocorrelation, day of week, long-term trend terms, and linear temperature controls included.	Both PM_{10} and O_3 positively associated with asthma ED visits using all three modeling approaches. In models with both O_3 and PM_{10}, both pollutants become non-significant because of high collinearity of the variables (r=0.75).	<u>Pediatric (<17 yrs. of age) ED Visits</u> PM_{10} = 50 µg/m^3 Lag 1 day ER = 13.2% (CI: 1.2, 26.7) With O_3 8.2 (-7.1, 26.1)

TABLE 8B-3 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY MEDICAL VISITS

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
United States (cont'd)			
Tolbert et al. (2000a) Atlanta Period 1: 1/1/93-7/31/98 Mean, median, SD: PM_{10} (µg/m^3): 30.1, 28.0, 12.4 Period 2: 8/1/98-8/31/99 Mean, median, SD: PM_{10} (µg/m^3): 29.1, 27.6, 12.0 $PM_{2.5}$ (µg/m^3): 19.4, 17.5, 9.35 CP (µg/m^3): 9.39, 8.95, 4.52 10-100 nm PM counts (count/cm^3): 15,200, 10,900, 26,600 10-100 nm PM surface area (um^2/cm^3): 62.5, 43.4, 116 $PM_{2.5}$ soluble metals (µg/m^3): 0.0327, 0.0226, 0.0306 $PM_{2.5}$ Sulfates (µg/m^3): 5.59, 4.67, 3.6 $PM_{2.5}$ Acidity (µg/m^3): 0.0181, 0.0112, 0.0219 $PM_{2.5}$ organic PM (µg/m^3): 6.30, 5.90, 3.16 $PM_{2.5}$ elemental carbon (µg/m^3): 2.25, 1.88, 1.74	Preliminary analysis of daily emergency department (ED) visits for asthma (493), wheezing (786.09) COPD (491, 492, 4966) LRI 466.1, 480, 481, 482, 483, 484, 485, 486), all resp disease (460-466, 477, 480-486, 491, 492, 493, 496, 786.09) for persons 16 yr in the period before (Period 1) and during (Period 2) the Atlanta superstation study. ED data analyzed here from just 18 of 33 participating hospitals; numbers of participating hospitals increased during period 1. Mean daily ED visits for dysrhythmias and all DVD in period 1 were 6.5 and 28.4, respectively. Covariates: NO_2, O_3, SO_2, CO temperature, dewpoint, and, in period 2 only, VOCs. PM measured by both TEOM and Federal Reference Method; unclear which used in analyses. For epidemiologic analyses, the two time periods were analyzed separately. Poisson GLM regression analyses were conducted with cubic splines for time, temperature and dewpoint. Day-of-week and hospital entry/exit indicators also included. Pollutants	In period 1, observed significant COPD association with 3-day average PM_{10}. COPD was also positively associated with NO_2, O_3, CO and SO_2. No statistically significant association observed between asthma and PM_{10} in period 1. However, asthma positively associated with ozone (p=0.03). In period 2, i.e., the first year of operation of the superstation, no statistically significant associations observed with PM_{10} or $PM_{2.5}$. These preliminary results should be interpreted with caution given the incomplete and variable nature of the databases analyzed.	Period 1: PM_{10} (0-2 d): asthma: 5.6% (-8.6, 22.1) COPD: 19.9% (0.1, 43.7) Period 2: (all 0-2 day lag) PM_{10}: asthma 18.8% (-8.7, 54.4) COPD -3.5% (-29.9, 33.0) $PM_{2.5}$: asthma 2.3% (-14.8, 22.7) COPD 12.4% (-7.9, 37.2) $PM_{10-2.5}$: asthma 21.1% (-18.2, 79.3) COPD -23.0% (-50.7, 20.1)
Yang et al (1997) Study Period: 92 - 94 Reno-Sparks, Nevada Population = 298,000 PM_{10} mean = 33.6 µg/m^3 PM_{10} range = 2.2, 157.3 µg/m^3	Association between asthma ER visits (mean = 1.75/d, SD=1.53/d) and PM_{10}, CO and O_3 assessed using linear WLS and ARIMA GLM regression, including adjustments for day-of-week, season, and temperature (but not RH or T-RH interaction). Season adjusted only crudely, using month dummy variable.	Only O_3 showed significant associations with asthma ER visits. However, the crude season adjustment and linear model (rather than Poisson) may have adversely affected results. Also, Beta-gauge PM_{10} mass index used, rather than direct gravimetric mass measurements.	NR

TABLE 8B-3 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY MEDICAL VISITS

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Canada			
Delfino et al. (1997) Montreal, Canada Population= 3 million 6-9/92, 6-9/93 1993 Means (SD): PM_{10}= 21.7 µg/m^3 (10.2) $PM_{2.5}$= 12.2 µg/m^3 (7.1) $SO_4^=$= 34.8 nmol/m^3 (33.1) H^+= 4 nmol/m^3 (5.2)	Association of daily respiratory emergency department (ED) visits (mean = 98/day from 25 of 31 acute care hospitals) with O_3, PM_{10}, $PM_{2.5}$, $SO_4^=$, and H^+ assessed using GLM regression with controls for temporal trends, auto-correlation, and weather. Five age sub-groups considered.	No associations with ED visits in '92, but 33% of the PM data missing then. In '93, only H^+ associated for children <2, despite very low H^+ levels. H^+ effect stable in multiple pollutant models and after excluding highest values. No associations for ED visits in persons aged 2-64 yrs. For patients >64 yr, O_3, PM_{10}, $PM_{2.5}$, and $SO_4^=$ positively associated with visits ($p < 0.02$), but PM effects smaller than for O_3.	<u>Respiratory ED Visits</u> Adults >64: (pollutant lags = 1 day) 50 µg/m^3 PM_{10} ER = 36.6% (10.0, 63.2) 25 µg/m^3 $PM_{2.5}$ ER = 23.9% (4.9, 42.8)
Delfino et al. (1998) Montreal, Canada 6-8/89,6-8/90 Mean PM_{10}= 18.6 µg/m^3 (SD=9.3, 90th% = 30.0 µg/m^3)	Examined the relationship of daily ED visits for respiratory illnesses by age (mean/day: <2yr.=8.9; 2-34yr.=20.1; 35-64yr.=22.6; >64yr.=20.3) with O_3 and estimated $PM_{2.5}$. Seasonal and day-of-week trends, auto-correlation, relative humidity and temperature were addressed in linear time series GLM regressions.	There was an association between $PM_{2.5}$ and respiratory ED visits for older adults (>64), but this was confounded by both temperature and O_3. The fact that $PM_{2.5}$ was estimated, rather than measured, may have weakened its relationship with ED visits, relative to O_3.	<u>Older Adults(>64 yr) Respiratory ED Visits</u> Estimated $PM_{2.5}$ = 25 µg/m^3 Single Pollutant: (lag 1 $PM_{2.5}$) ER = 13.2 (-0.2, 26.6) With Ozone (lag 1 $PM_{2.5}$): Est. $PM_{2.5}$ (lag1) ER = 0.8% (CI: -14.4, 15.8)
Stieb et al. (1996) St. John, New Brunswick, Canada Population = 75,000 May-Sept. 84 - 92 SO_4^{2-} Mean = 5.5 µg/m^3 Range: 1-23, 95th% =14 µg/m^3 TSP Mean = 36.7 µg/m^3 Range:5-108, 95th% =70 µg/m^3	Asthma ED visits (mean=1.6/day) related to daily O_3 and other air pollutants (SO_2, NO_2, SO_4^{2-}, and TSP). PM measured only every 6th day. Weather variables included temperature, humidex, dewpoint, and RH. ED visit frequencies were filtered to remove day of week and long wave trends. Filtered values were GLM regressed on pollution and weather variables for the same day and the 3 previous days.	Positive, statistically significant ($p < 0.05$) association observed between O_3 and asthma ED visits 2 days later; strength of the association greater in nonlinear models. Ozone effect not significantly influenced by addition of other pollutants. However, given limited number of sampling days for sulfate and TSP, it was concluded that "a particulate effect could not be ruled out".	<u>Emergency Department Visits (all ages)</u> Single Pollutant Model 100 µg/m^3 TSP = 10.7% (-66.4, 87.8)

TABLE 8B-3 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY MEDICAL VISITS

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Canada (cont'd)			
Stieb et al. (2000)+ Saint John, New Brunswick, Canada 7/1/92-3/31/96 mean and S.D.: PM_{10} (µg/m^3): 14.0, 9.0 $PM_{2.5}$ (µg/m^3): 8.5, 5.9 H+ (nmol/m^3): 25.7, 36.8 Sulfate (nmol/m^3): 31.1, 29.7 COH mean (10^3 ln ft): 0.2, 0.2 COH max (10^3 ln ft): 0.6, 0.5	Study of daily emergency department (ED) visits for asthma (mean 3.5/day), COPD (mean 1.3/day), resp infections (mean 6.2/day), and all respiratory conditions (mean 10.9/day) for persons of all ages. Covariates included CO, H_2S, NO_2, O_3, SO_2, total reduced sulfur (TRS), a large number of weather variables, and 12 molds and pollens. Stats: generalized additive models with LOESS prefiltering of both ED and pollutant variables, with variable window lengths. Also controlled for day of week and LOESS-smoothed functions of weather. Single-day, and five day average, pollution lags tested out to lag 10. The strongest lag, either positive or negative, was chosen for final models. Both single and multi-pollutant models reported. Full-year and May-Sep models reported.	In single-pollutant models, significant positive associations were observed between all respiratory ED visits and PM_{10}, $PM_{2.5}$, H_2S, O_3, and SO_2. Significant negative associations were observed with H+, and COH max. PM results were similar when data were restricted to May-Sep. In multi-pollutant models, no PM metrics significantly associated with all cardiac ED visits in full year analyses, whereas both O_3 and SO_2 were. In the May-Sep subset, significant negative association found for sulfate. No quantitative results presented for non-significant variables in these multi-pollutant regressions.	$PM_{2.5}$, (lag 3) 15.1 (-0.2, 32.8) PM_{10}, (lag 3) 32.5 (10.2, 59.3)
Europe			
Atkinson et al. (1999b) London (92 - 94) Population = NR PM10 Mean = 28.5 µg/m^3 10^{th}-90^{th} IQR = 15.8-46.5 µg/m^3 BS mean =12.7 µg/m^3 10^{th}-90^{th} IQR = 5.5-21.6 µg/m^3	All-age Respiratory (mean=90/day), Asthma (25.9/day), and Other Respiratory (64.1/day) ED visits from 12 London hospitals considered, but associated population size not reported. Counts for ages 0-14, 15-64, and >64 also examined. Poisson GLM regression used, controlling for season, day of week, meteorology, autocorrelation, overdispersion, and influenza epidemics.	PM_{10} positively associated, but not BS, for all-age/all-respiratory category. PM_{10} results driven by significant children and young adult associations, while older adult visits had negative (but non-significant) PM_{10}-ED visit relationship. PM_{10} positively associated for all ages, children, and young adults for asthma ED visits. However, PM_{10}-asthma relationship couldn't be separated from SO_2 in multi-pollutant regressions. Older adult ED visits most strongly associated with CO. No O_3-ED visits relationships found (but no warm season analyses attempted).	PM_{10} (50 µg/m^3) No co-pollutant: <u>All Respiratory ED visits</u> All age(lag 1d)ER = 4.9% (CI: 1.3, 8.6) <15yrs(lag 2d)ER = 6.4% (CI: 1, 12.2) 15-64yr(lag1d)ER = 8.6% (CI: 3.4, 14) <u>Asthma ED visits</u> All age (lag 1d) ER = 8.9% (CI: 3, 15.2) <15yrs (lag 2d) ER = 12.3% (CI: 3.4, 22) 15-64yr (lg 1d) ER = 13% (CI: 4.6, 22.1) PM_{10} (50 µg/m^3) 2d lag & co-pollutant: Children's (<15 yrs.) Asthma ED Visits: PM alone: ER = 12.3% (CI: 3.4, 22) &NO_2: ER = 7.8% (CI: -1.2, 17.6) & O_3: ER = 10.5% (CI: 1.6, 20.1) & SO_2: ER = 8.1% (CI: -1.1, 18.2) & CO: ER = 12.1% (CI: 3.2, 21.7)

TABLE 8B-3 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY MEDICAL VISITS

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Europe (cont'd)			
Hajat et al. (1999) London, England (92 - 94) Population = 282,000 PM_{10} mean = 28.2 µg/m^3 PM_{10} 10^{th}-90^{th}%=16.3-46.4 µg/m^3 BS mean = 10.1 µg/m^3 BS 10^{th}-90^{th}%=4.5-15.9 µg/m^3	Examined associations of PM_{10}, BS, NO_2, O_3, SO_2, and CO, with primary care general practitioner asthma and "other LRD" consultations. Asthma consultation means per day = 35.3 (all ages); 14.(0-14 yrs,); 17.7 (15-64 yrs.); 3.6 (>64 yrs.). LRD means = 155 (all ages); 39.7(0-14 yrs,); 73.8 (15-64 yrs.); 41.1 (>64 yrs.). Time-series analyses of daily numbers of consultations performed, controlling for time trends, season factors, day of week, influenza, weather, pollen levels, and serial correlation.	Positive associations, weakly significant and consistent across lags, observed between asthma consultations and NO_2 and CO in children, and with PM_{10} in adults, and between other LRD consultations and SO_2 in children. Authors concluded that there are associations between air pollution and daily concentrations for asthma and other lower respiratory disease in London. In adults, the authors concluded that the only consistent association was with PM_{10}. Across all of the various age, cause, and season categories considered, PM_{10} was the pollutant most coherent in giving positive pollutant RR estimates for both asthma and other LRD (11 of 12 categories positive) in single pollutant models considered.	<u>Asthma Doctor's Visits:</u> 50 µg/m^3 PM_{10} -Year-round, Single Pollutant: All ages (lg 2): ER = 5.4% (CI: -0.6, 11.7) 0-14 yrs.(lg 1): ER = 6.4% (-1.5, 14.6) 15-64 yrs.(lg 0): ER = 9.2% (CI: 2.8, 15.9) >64yrs.(lg 2): ER = 11.7% (-1.8, 26.9) -Year-round, 2 Pollutant, Children (0, 14): (PM_{10} lag = 1 day) PM_{10} ER's: W/NO_2: ER = 0.8% (CI: -8.7, 11.4) W/O_3: ER = 5.5% (-2.1, 13.8) W/SO_2: ER = 3.2% (CI: -6.4, 13.7) <u>Other Lower Resp. Dis. Doctor's Visits:</u> 50 µg/m^3 PM_{10} -Year-round, Single Pollutant: All ages (lg 2): ER = 3.5% (CI: 0, 7.1) 0-14 yrs.(lg 1): ER = 4.2% (CI: -1.2, 9.9) 15-64 yrs.(lg 2): ER= 3.7% (CI: 0.0, 7.6) >64yrs.(lg 2): ER = 6.2% (CI: 0.5, 12.9)
Hajat et al. (2001)+ London (1992-1994) 44,406-49,596 registered patients <1 to 14 years PM_{10} mean 28.5 (13.9)	Daily physician consultations (mean daily 4.8 for children; 15.3 for adults) for allergic rhinitis (ICD-9, 477), SO_2, O_3, NO_2, CO, PM_{10}, and pollen using generalized additive models with nonparametric smoother.	SO_2 and O_3 show strong associations with the number of consultations for allergic rhinitis. Estimates largest for a lag of 3 or 4 days prior to consultations, with cumulative measures stronger than single day lags. Stronger effects were found for children than adults. The two-pollutant analysis of the children's model showed that PM_{10} and NO_2 associations disappeared once either SO_2 or O_3 was incorporated into the model.	PM_{10} - Increment (10-90%) (15.8-46.5) Age <1-14 years lag 3: 10.4 (2.0 to 19.4) Cum 0-3: 17.4 (6.8 to 29.0) Ages 15-64 years lag 2: 7.1 (2.6 to 11.7) Cum 0-6: 20.2 (14.1 to 26.6)

TABLE 8B-3 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY MEDICAL VISITS

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Europe (cont'd)			
Medina et al. (1997)+ Greater Paris 91 - 95 Population.= 6.5 MM Mean PM_{13} = 25 µg/m^3 PM_{13} min/max = 6/95 µg/m^3 Mean BS = 21 µg/m^3 BS min/max = 3/130 µg/m^3	Evaluated short-term relationships between PM_{13} and BS concentrations and doctors' house calls (mean=8/day; 20% of city total) in Greater Paris. Poisson regression used, with non-parametric smoothing functions controlling for time trend, seasonal patterns, pollen counts, influenza epidemics, day-of-week, holidays, and weather.	A relationship between all age (0-64 yrs.) asthma house calls and PM_{13}, BS, SO_2, NO_2, and O_3 air pollution, especially for children aged 0-14 (mean = 2/day). In two-pollutant models including BS with, successively, SO_2, NO_2, and O_3, only BS and O_3 effects remained stable. These results also indicate that air pollutant associations noted for hospital ED visits are also applicable to a wider population that visits their doctor.	Doctor's Asthma House Visits: 50 µg/m^3 PM_{13} Year-round, Single Pollutant: All ages (lg 2): ER = 12.7% (CI: 4.1, 21.9) 0-14 yrs.(lg 0-3): ER = 41.5% (CI: 20, 66.8) 15-64 yrs.(lg 2): ER = 6.3% (CI: -4.6, 18.5)
Damiá et al. (1999) Valencia, Spain (3/94-3/95) Population = NR BS mean = 101 µg/m^3 BS range = 34-213 µg/m^3	Associations of BS and SO_2 with weekly total ED admissions for asthma patients aged > 12 yrs (mean = 10/week) at one hospital over one year assessed, using linear stepwise GLM regression. Season-specific analyses done for each of 4 seasons, but no other long-wave controls. Linear T, RH, BP, rain, and wind speed included as crude weather controls in ANOVA models.	Both BS and SO_2 correlated with ED admissions for asthma (SO_2: r=0.32; BS: r=0.35), but only BS significant in stepwise multiple regression. No linear relationship found with weather variables. Stratified ANOVA found strongest BS-ED association in the autumn and during above average temperatures. Uncontrolled autocorrelation (e.g., within-season) and weather effects likely remain in models.	Asthma ED Visits (all ages): BS = 40 µg/m^3 (single pollutant) BS as a lag 0 weekly average: ER = 41.5% (CI = 39.1, 43.9)
Pantazopoulou et al. (1995) Athens, GR (1988) Population = NR Winter (1/88-3/88,9/88-12/88) BS mean. =75 µg/m^3 BS 5^{th}-95^{th} %=26 - 161 µg/m^3 Summer (3/22/88-3/88,9/21/88) BS mean. =55 µg/m^3 BS 5^{th}-95^{th} %=19 - 90 µg/m^3	Examined effects of air pollution on daily emergency outpatient visits and admissions for cardiac and respiratory causes. Air pollutants included: BS, CO, and NO_2. Multiple linear GLM regression models used, controlling for linear effects of temperature and RH, day of week, holidays, and dummy variables for month to crudely control for season, separately for winter and summer.	Daily number of emergency visits related positively with each air pollutant, but only reached nominal level of statistical significance for NO_2 in winter. However, the very limited time for each within-season analysis (6 mo.) undoubtably limited the power of this analysis to detect significant effects. Also, possible lagged pollution effects were apparently not investigated, which may have reduced effect estimates.	Single Pollutant Models For Winter (BS = 25 µg/m^3) Outpatient Hospital Visits ER = 1.1% (-0.7, 2.3) Respiratory HA's ER = 4.3% (0.2, 8.3) For Summer, BS = 25 µg/m^3) Outpatient Hospital Visits ER = 0.6% (-4.7, 6.0) Respiratory HA's ER = 5.5% (-3.6, 14.7)

TABLE 8B-3 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY MEDICAL VISITS

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Europe (cont'd)			
Garty et al. (1998) PM_{10} mean 45 µg/m^3 Tel Aviv, Israel (1993)	Seven day running mean of asthma ED visits by children (1-18 yrs.) to a pediatric hospital modeled in relation to PM_{10} in Tel Aviv, Israel.	No PM_{10} associations found with ED visits. The ER visits–pollutant correlation increased significantly when the September peak was excluded. Use of a week-long average and associated uncontrolled long-wave fluctuations (with resultant autocorrelation) likely prevented meaningful analyses of short –term PM associations with ED visits.	N/A
Latin America			
IIabaca et al. (1999) Santiago, Chile February 1995-August 1996 PM_{10}: warm: 80.3 µg/m^3 cold: 123.9 µg/m^3 $PM_{2.5}$: warm: 34.3 µg/m^3 cold: 71.3 µg/m^3	Number of daily respiratory emergency visits (REVs) related to PM by Poisson GLM model with longer- and short-term trend terms. SO_2, NO_2, O_3.	Stronger coefficients for models including $PM_{2.5}$ than for models including PM_{10} or $PM_{10\text{-}2.5}$. Copollutant effects were significantly associated with REVs. For respiratory patients, the median number of days between the onset of the first symptoms and REV was two to three days. For the majority of patients (70%) this corresponded to the lag observed in this study indicating that the timing of the pollutant effect is consistent with the temporal pattern of REV in this population.	REV, lag 2 Cold $PM_{2.5}$, lag 2 OR: 1.027 (1.01 to 1.04) for a 45 µg/m^3 increment PM_{10}, lag 2 OR: 1.02 (1.01 to 1.04) for a 76 µg/m^3 increment $PM_{2.5}$, lag 2 OR: 1.01 (1.00* to 1.03) for a 32 µg/m^3 increment Pneumonia, lag 2 PM_{10}: 1.05 (1.00* to 1.10) 64 µg/m^3 increment $PM_{2.5}$: 1.04 (1.00* to 1.09) 45 µg/m^3 increment $PM_{10\text{-}2.5}$: 10.5 (1.00* to 1.10) 32 µg/m^3 increment *decimals <1.00

TABLE 8B-3 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY MEDICAL VISITS

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Latin America (cont'd)			
Lin et al. (1999) Sao Paulo, BR (91-93) Population=NR PM_{10} mean =65 µg/m^3 PM_{10} SD=27 µg/m^3 PM_{10} range=15-193 µg/m^3	Respiratory ED visits by children (0-12 yrs.) To a major pediatric hospital (mean=56/day) related to PM_{10}, SO_2, NO_2, CO, and O_3 using various GLM models: Gaussian linear regression modeling, Poisson modeling, and a polynomial distributed lag model. Lower respiratory (mean = 8/day) and upper respiratory (mean = 9/day) all evaluated. Analyses considered effects of season, day of week, and extreme weather (using T, RH dummy variables).	PM_{10} was found to be "the pollutant that exhibited the most robust and stable association with all categories of respiratory disease". O_3 was the only other pollutant that remained associated when other pollutants all simultaneously added to the model. However, some pollutant coefficients went negative in multiple pollutant regressions, suggesting coefficient intercorrelations in the multiple pollutant models. More than 20% increase in ED visits found on the most polluted days, "indicating that air pollution is a substantial pediatric health concern".	50 µg/m^3 PM_{10} (0-5-day lag mean) <u>Respiratory ED Visits (<13 yrs.)</u> Single pollutant model: PM_{10} ER=21.7% (CI: 18.2, 25.2) All pollutant models: PM_{10} ER=28.8% (CI: 21.4, 36.7) <u>Lower Respiratory ED Visits (<13 yrs.)</u> Single pollutant model: PM_{10} ER=22.8% (CI: 12.7, 33.9) All pollutant models: PM_{10} ER=46.9% (CI: 27.9, 68.8)
Ostro et al. (1999b)+ Santiago, CI (7/92—12/93) <2 yrs. Population 20,800 3-14 yrs. Population 128,000 PM_{10} mean. =108.6 µg/m^3 PM_{10} Min/Max=18.5/380 µg/m^3 PM_{10} IQR = 70.3 – 135.5 µg/m^3	Analysis of daily visits to primary health care clinics for upper (URS) or lower respiratory symptoms (LRS) for children 2-14 yr (mean LRS=111.1/day) and < age 2 (mean LRS=104.3/day). Daily PM_{10} and O_3 and meteorological variables considered. The multiple regression GAM included controls for seasonality (LOESS smooth), temperature, day of week, and month.	Analyses indicated an association between PM_{10} and medical visits for LRS in children ages 2-14 and in children under age 2 yr. PM_{10} was not related to non-respiratory visits (mean =208/day). Results unchanged by eliminating high PM_{10} (>235 µg/m^3) or coldest days (<8°C). Adding O_3 to the model had little effect on PM_{10}-LRS associations.	<u>Lower Resp. Symptoms Clinic Visits</u> PM_{10} = 50 µg/m^3 Single Pollutant Models: -Children<2 years Lag 3 ER = 2.5% (CI: 0.2, 4.8) -Children 2-14 years Lag 3 ER = 3.7% (CI: 0.8, 6.7%) Two Pollutant Models (with O_3): -Children<2 years Lag 3 ER = 2.2% (CI: 0, 4.4) -Children 2-14 years Lag 3 ER = 3.7% (CI: 0.9, 6.5)

TABLE 8B-3 (cont'd). ACUTE PARTICULATE MATTER EXPOSURE AND RESPIRATORY MEDICAL VISITS

Reference/Citation, Location, Duration, PM Index/Concentrations	Study Description:	Results and Comments	PM Index, Lag, Excess Risk %, (95% CI = LCI, UCL) Co-Pollutants
Australia			
Smith et al. (1996) Stdy Pd.: 12/92-1/93,12/93-1/94 West Sydney, AU Population = 907,000 -Period 1 (12/92-1/93) B_{scatt} median = 0.25 10 4/m B_{scatt} IQR = 0.18-0.39 10 4/m B_{scatt} 95th% = 0.86 10 4/m -Period 2 (12/93-1/94) B_{scatt} median = 0.19 10 4/m B_{scatt} IQR = 0.1-0.38 10 4/m B_{scatt} 95th5% = 3.26 10 4/m PM_{10} median = 18 µg/m^3 PM_{10} IQR = 11.5-28.8 µg/m^3 PM_{10} 95th% = 92.5 µg/m^3	Study evaluated whether asthma visits to emergency departments (ED) in western Sydney (mean10/day) increased as result of bushfire-generated PM (B_{scatt} from nephelometry) in Jan., 1994 (period 2). Air pollution data included nephelometry (B_{scatt}), PM_{10}, SO_2, and NO_2. Data analyzed using two methods: (1) calculation of the difference in proportion of all asthma ED visits between the time periods, and; (2) Poisson GLM regression analyses. Control variables included T, RH, BP, WS, and rainfall.	No difference found in the proportion of all asthma ED visits during a week of bushfire-generated air pollution, compared with the same week 12 months before, after adjusting for baseline changes over the 12-month period. The max. B_{scatt} reading was not a significant predictor of the daily asthma ED visits in Poisson regressions. However, no long-wave controls applied, other than indep. vars., and the power to detect differences was weak (90% for a 50% difference). Thus, the lack of a difference may be due to low statistical strength or to lower toxicity of particles from burning vegetation at ambient conditions vs. fossil fuel combustion.	<u>ED Asthma Visits (all ages)</u> Percent change between bushfire and non bushfire weeks: PM_{10} = 50 µg/m^3 ER = 2.1% (CI: -0.2, 4.5)
Asia			
Ye et al. (2001) Tokyo, Japan Summer months July-August, 1980-1995 PM_{10} 46.0 mean	Hospital emergency transports for respiratory disease for >65 years of age were related to pollutant levels NO_2, O_3, PM_{10}, SO_2, and CO.	For chronic bronchitis PM_{10} with a lag time of 2 days was the most statistically significant model covariate.	Asthma (ICD-9-493) Coefficienct estimate (SE) 0.003 (0.001)
Chew et al. (1999) Singapore (90 - 94) Population = NR TSP mean = 51.2 µg/m^3 TSP SD = 20.3 µg/m^3 TSP range = 13-184 µg/m^3	Child (3-13 yrs.) ED visits (mean = 12.8/day) and HA's (mean = 12.2/day) for asthma related to levels of SO_2, NO_2, TSP, and O_3 using GLM linear regression with weather, day-of-week controls. Auto-correlation effects controlled by including prior day response variable as a regression variable. Separate analyses done for adolescents (13-21 yrs.) (mean ED=12.2, mean HA=3.0/day).	Positive associations found between TSP, SO_2, and NO_2, and daily HA and ED visits for asthma in children, but only with ED visits among adolescents. Lack of power (low counts) for adolescents' HA's appears to have been a factor in the lack of associations. When ED visits stratified by year, SO_2 and TSP remained associated in every year, but not NO_2. Analyses for control diseases (appendicitis and urinary tract infections) found no associations.	TSP(100 µg/m^3) No co-pollutant: <u>Child (3-13 yrs.)Asthma ED visits</u> Lag 1d ER = 541% (CI: 198.4, 1276.8)

+ = Used GAM with multiple smooths, but have not yet reanalyzed. * = Used S-Plus Default GAM, and have reanalyzed results

Appendix 8B.4: Pulmonary Function Studies

TABLE 8B-4. SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF ASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
United States			
Thurston et al. (1997) Summers 1991-1993. O_3, H+, sulfate	Three 5-day summer camps conducted in 1991, 1992, 1993. Study measured symptoms and change in lung function (morning to evening). Poisson regression for symptoms.	The O_3-ΔPEFR relationship was seen as the strongest.	—
Canada			
Vedal et al. (1998) Port Alberni, BC PM_{10} via a Sierra-Anderson dichotomous sampler. PM_{10} ranged from 1 to 159 µg/m^3.	Study of 206 children aged 6 to 13 years living in Port Alberni, British Columbia. 75 children had physician-diagnosed asthma, 57 had an exercised induced fall in FEV1, 18 children with airway obstruction, and 56 children without any symptoms. Respiratory symptom data obtained from diaries. An autoregressive model was fitted to the data, using GEE methods. Covariates included temp., humidity, and precipitation.	Ozone, SO_2 and sulfate levels low due to low vehicle emissions. PM_{10} associated with change in peak flow.	Lag 0, PM10 average PEF = 0.27 (−0.54, −0.01) per 10 µg/m^3 increment
Europe			
Gielen et al. (1997) Amsterdam, NL Mean PM_{10} level: 30.5 µg/m^3 (16, 60.3). Mean maximum 8 hr O_3: 67 µg/m^3.	Study evaluated 61 children aged 7 to 13 years living in Amsterdam, The Netherlands. 77 percent of the children were taking asthma medication and the others were being hospitalized for respiratory problems. Peak flow measurements were taken twice daily. Associations of air pollution were evaluated using time series analyses. The analyses adjusted for pollen counts, time trend, and day of week.	The strongest relationships were found with ozone, although some significant relationships found with PM_{10}.	Lag 0, PM_{10}: Evening PEF = −0.08 (−2.49, 2.42) Lag 1, PM_{10}: Morning PEF = 1.38 (−0.58, 3.35) Lag 2, PM_{10}: Morning PEF = 0.34 (−1.78, 2.46) Evening PEF = −1.46 (−3.23, 0.32)
Hiltermann et al. (1998) Leiden, NL July-Oct, 1995 O_3, NO_2, SO_2, BS, and PM_{10} ranged from 16.4 to 97.9 µg/m^3)	270 adult asthmatic patients from an out-patient clinic in Leiden, The Netherlands were studied from July 3 to October 6, 1995. Peak flow measured twice daily. An autoregressive model was fitted to the data. Covariates included temp. and day of week. Individual responses not modeled.	No relationship between ozone or PM_{10} and PFT was found	Lag 0, PM_{10}: Average PEF = −0.80 (−3.84, 2.04) 7 day ave., PM_{10}: Average PEF = −1.10 (−5.22, 3.02)

TABLE 8B-4 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF ASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 $PM_{2.5}$). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Europe (cont'd)			
Peters et al. (1996) Erfurt and Weimar, Germany SO_2, TSP, PM_{10}, sulfate fraction, and PSA. Mean PM_{10} level was 112 µg/m^3. PM was measured by a Marple-Harvard impactor.	Panel of 155 asthmatic children in the cities of Erfurt and Weimar, E. Germany studied. Each panelist's mean PEF over the entire period subtracted from the PEF value to obtain a deviation. Mean deviation for all panelists on given day was analyzed using an autoregressive moving average. Regression analyses done separately for adults and children in each city and winter; then combined results calculated.	Five day average SO_2 was associated with decreased PEF. Changes in PEF were not associated with PM levels.	—
Peters et al. (1997b) Erfurt, Germany PM fractions measured over range of sizes from ultrafine to fine, including PM_{10}. Particles measured using size cuts of 0.01 to 0.1, 0.1 to 0.5, and 0.5 to 2.5 µm. Mean PM_{10} level: 55 µg/m^3 (max 71). Mean SO_2: 100 µg/m^3 (max 383). PM was measured using a Harvard impactor. Particle size distributions were estimated using a conduction particle counter.	Study of 27 non-smoking adult asthmatics living in Erfurt, Germany during winter season of 1991-1992. Morning and evening peak flow readings recorded. An auto-regressive model was used to analyze deviations in individual peak flow values, including terms for time trend, temp., humidity, and wind speed and direction.	Strongest effects on peak flow found with ultrafine particles. The two smallest fractions, 0.01 to 0.1 and 0.1 to 0.5 were associated with a decrease of PEF.	Lag 0, PM_{10}: Evening PEF = −0.38 (−1.83, 1.08) Lag 1, PM_{10}: Morning PEF = −1.30 (−2.36, 0.24) 5 Day Mean, PM_{10}: Morning PEF = −1.51 (−3.20, 0.19) Evening PEF = −2.31 (−4.54, −0.08) Lag 0, $PM_{2.5}$: Evening PEF = −0.75 (−1.66, 0.17) Lag 1, $PM_{2.5}$: Morning PEF = −0.71 (−1.30, 0.12) 5 Day Mean, $PM_{2.5}$: Morning PEF = −1.19 (−1.81, 0.57) Evening PEF = −1.79 (−2.64, −0.95)
Peters et al. (1997c) Sckolov, Czech Republic Winter 1991-1992 PM_{10}, SO_2, TSP, sulfate, and particle strong acid. Median PM_{10} level: 47 µg/m^3 (29, 73). Median SO_2: 46 µg/m^3 (22, 88). PM was measured using a Harvard impactor. Particle size distributions were estimated using a conduction particle counter.	89 children with asthma in Sokolov, Czech Republic studied. Subjects kept diaries and measured peak flow for seven months during winter of 1991-2. The analysis used linear regression for PFT. First order autocorrelations were observed and corrected for using polynomial distributed lag (PDL) structures.	Five day mean SO_2, sulfates, and particle strong acidity were also associated with decreases in PM PFT as well as PM_{10}.	Lag 0, PM_{10}: Morning PEF = −0.71 (−2.14, 0.70) Evening PEF = −0.92 (−1.96, 0.12) 5 Day mean PM_{10}: Evening PEF = −1.72 (−3.64, 0.19) Morning PEF = −0.94 (−2.76, 0.91

TABLE 8B-4 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF ASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 $PM_{2.5}$). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Europe (cont'd)			
Timonen and Pekkanen (1997) Kupio, Finland PM_{10}, BS, NO_2, and SO_2. The intequartile range on PM_{10} was 8 to 23.	Studied 74 asthmatic children (7 to 12 yr) in Kuoio, Finland. Daily mean PEF deviation calculated for each child. Values were analyzed, then using linear first-order autoregressive model. PM was measured using single stage Harvard Impactors.	Lagged concentrations of NO_2 related to declines in morning PEF as well as PM_{10} and BS.	
Penttinen et al. (2001) studied adult asthmatics for 6 months in Helsinki, Findland. PM was measured using a single-stage Harvard impactor. Particle number concentrations were measured using an Electric Aerosol Spectrometer. NO_2PM_{10} ranged from 3.8 to 73.7 µg/m^3. $PM_{2.5}$ ranged from 2.4 to 38.3 µg/m^3.	57 asthmatics were followed with daily PEF measurements and symptom and medications diaries from November 1996 to April 1997. PEF deviations from averages were used as dependent variables. Independent variables included PM_1, $PM_{2.5}$, PM_{10}, particle counts, CO, NO, and	The strongest relationships were found between PEF deviations and PM particles below 0.1 µm. No associations were found between particulate pollution and respiratory symptoms.	AM PEF = -.115 (-.448, .218) $PM_{2.5}$ lag one day AM PEF = -.001 (-.334, .332) $PM_{2.5}$ lag two days
Pekkanen et al. (1997) Kuopio, Finland PM fractions measured over range of sizes from ultrafine to fine, including PM_{10}. Mean PM_{10} level: 18 µg/m^3 (10, 23). Mean NO_2 level: 28 µg/m^3.	Studied 39 asthmatic children aged 7-12 years living in Kuopio, Finland. Changes in peak flow measurements were analyzed using a linear first-order autoregressive model. PM was measured using single stage Harvard impactors.	Changes in peak flow found to be related to all measures of PM, after adjusting for minimum temperature. PN0.032−0.10 (1/cm^3) and PN1.0−3.2 (1/cm^3) were most strongly associated with morning PEF deviations.	Lag 0, PM_{10}: Evening PEF = −0.35 (−1.14, 0.96) Lag 1, PM_{10}: Morning PEF = −2.70 (−6.65, 1.23) Lag 2, PM_{10}: Morning PEF = −4.35 (−8.02, −0.67) Evening PEF = −1.10 (−4.70, 2.50) Small sized particles had relationships similar to those of PM_{10} for morning and evening PEF.
Segala et al. (1998) Paris, France Nov. 1992 - May 1993. BS, SO_2, NO_2, PM_{13} (instead of PM_{10}), measured. Mean PM_{13} level: 34.2 µg/m^3 (range 8.8, 95). Mean SO_2 level: 21.7 µg/m^3 (range 4.4, 83.8). Mean NO_2 level: 56.9 µg/m^3 (range 23.8, 121.9). PM was measured by β-radiometry.	Study of 43 mildly asthmatic children aged 7-15 years living in Paris, France from Nov. 15, 1992 to May 9, 1993. Peak flow measured three times a day. Covariates in the model included temperature and humidity. An autoregressive model was fitted to the data using GEE methods.	Effects found related to PM_{10} were less than those found related to the other pollutants. The strongest effects were found with SO_2.	Lag 4, PM_{13}: Morning PEF = −0.62 (−1.52, 0.28)

TABLE 8B-4 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF ASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Europe (cont'd)			
Gauvin et al. (1999) Grenoble, France Summer 1996, Winter 1997 Mean (SD) µg/m^3 PM_{10} Summer 23 (6.7) PM_{10} Winter 38 (17.3) Sunday 15.55 (5.12) Weekday 24.03 (7.2)	Two panels: mild adult asthmatics, ages 20-60 years, (summer-18 asthmatics, 20 control subjects; winter-19 asthmatics, 21 control subjects) were examined daily for FEV_1 and PEF. Bronchial reactivity was compared Sunday vs. weekday. Temperature and RH controlled.	Respiratory function decreased among asthmatic subjects a few days (lag 2/4 days) after daily PM_{10} levels had increased. Bronchial reactivity was not significantly different between the weekdays and weekends. No copollutant analysis conducted.	For a 10 µg/m^3 increase in PM_{10} Summer FEV_1 −1.25% (−0.58 to −1.92) PEF −0.87% (−0.1 to −1.63)
Agócs et al. (1997) Budapest, Hungary SO_2 and TSP were measured. TSP was measured by beta reactive absorption methods.	Panel of 60 asthmatic children studied for two months in Budapest, Hungary. Mixed model used relating TSP to morning and evening PEFR measurements, adjusting for SO_2, time trend, day of week, temp., humidity		No significant TSP-PEFR relationships found.
Australia			
Jaulaludin et al. (2000) Sydney, Austrlia 1 February 1994 to 31 December 1994 Six PM_{10} TEOM monitors PM_{10} Mean - 22.8 ±13.9 µg/m^3 (max 122.8 µg/m^3)	Population regression and GEE models used a cohort of 125 children (mean age of 9.6 years) in three groups; two with doctor's diagnoses of asthma. This study was designed to examine effects of ambient O_3 and peak flow while controlling for PM_{10}.	In Syndey, O_3 and PM_{10} poorly correlated (0.13). For PM_{10} with O_3, 0.0051 (0.0124) p-0.68 peak flow	PM_{10} only B(SE) = 0.0045 (0.0125) p-0.72 peak flow
Rutherford et al. (1999) Brisbane, Australia PM_{10}, TSP, and particle diameter. PM_{10} ranged form 11.4 to 158.6 µg/m^3. Particle sizing was done by a Coulter Multisizer.	Study examined effects of 11 dust events on peak flow and symptoms of people with asthma in Brisbane, Australia. PEF data for each individual averaged for a period of 7 days prior to the identified event. This mean was compared to the average for several days of PEF after the event, and the difference was tested using a paired t-test.	The paired t-tests were stat. significant for some days, but not others. No general conclusions could be drawn.	—

TABLE 8B-4 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF ASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 $PM_{2.5}$). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Latin America			
Romieu et al. (1996) Mexico City, Mexico During study period, maximum daily 1-h O_3 ranged from 40 to 370 ppb (mean 190 ppb, SD = 80 ppb). 24 h ave, PM_{10} levels ranged from 29 to 363 µg/m^3 (mean 166.8 µg/m^3, SD 72.8 µg/m^3). For 53 percent of study days, PM_{10} levels exceeded 150 µg/m^3. PM_{10} was measured by a Harvard impactor.	Study of 71 children with mild asthma aged 5-7 years living in the northern area of Mexico City. Morning and evening peak flow measurements recorded by parents. Peak flow measurements were standardized for each person and a model was fitted using GEE methods. Model included terms for minimum temperature.	Ozone strongly related to changes in morning PEF as well as PM_{10}.	Lag 0, PM_{10}: Evening PEF = −4.80 (−8.00, −1.70) Lag 2, PM_{10}: Evening PEF = −3.65 (−7.20, 0.03) Lag 0, $PM_{2.5}$: Evening PEF = −4.27 (−7.12, −0.85) Lag 2, $PM_{2.5}$: Evening PEF = −2.55 (−7.84, 2.74) Lag 1, PM_{10} Morning PEF = −4.70 (−7.65, −1.7) Lag 2, PM_{10} Morning PEF = −4.90 (−8.4, −1.5)
Romieu et al. (1997) Mexico City, Mexico During study period, maximum daily 1-h ozone ranged from 40 to 390 ppb (mean 196 ppb SD = 78 ppb) PM_{10} daily average ranged from 12 to 126 µg/m^3. PM_{10} was measured by a Harvard impactor.	Study of 65 children with mild asthma aged 5-13 yr in southwest Mexico City. Morning and evening peak flow measurements made by parents. Peak flow measurements standardized for each person and model was fitted using GEE methods. Model included terms for minimum temperature.	Strongest relationships were found between ozone (lag 0 or 1) and both morning and evening PFT.	Lag 0, PM_{10}: Evening PEF = −1.32 (−6.82, 4.17) Lag 2, PM_{10}: Evening PEF = −0.04 (−4.29, 4.21) Morning PEF = 2.47 (−1.75, 6.75) Lag 0, PM_{10}: Morning PEF = 0.65 (−3.97, 5.32)

Appendix 8B.5: Short-Term PM Exposure Effects On Symptoms in Asthmatic Individuals

TABLE 8B-5. SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON SYMPTOMS IN STUDIES OF ASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 $PM_{2.5}$). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
United States			
Delfino et al. (1996) San Diego, CA Sept-Oct 1993 Ozone and $PM_{2.5}$ measured. PM was measured by a Harvard impactor. $PM_{2.5}$ ranged from 6 to 66 µg/m^3 with a mean of 25.	Study of 12 asthmatic children with history of bronchodilator use. A random effects model was fitted for ordinal symptoms scores and bronchodilator use in relation to 24-hr $PM_{2.5}$.	Pollen not associated with asthma symptom scores. 12-hr personal O_3 but not ambient O_3 related to symptoms.	No significant relationships with PM_{10}.
Delfino et al. (1997) San Diego County, CA PM_{10} and ozone PM was measured using a tapered-element oscillating microbalance. PM_{10} ranged from 6 to 51 µg/m^3 with a mean of 26.	A panel of 9 adults and 13 children were followed during late spring 1994 in semi-rural area of San Diego County at the inversion zone elevation of around 1,200 feet. A random effects model was fitted to ordinal symptom scores, bronchodilator use, and PEF in relation to 24-hour PM_{10}. Temp., relative humidity, fugal spores, day of week and O_3 evaluated	Although PM_{10} never exceeded 51 µg/m^3, bronchodilator use was significantly associated with PM_{10}(0.76 [0.027, 0.27]) puffs per 50 µg/m^3. Fungal spores were associated with all respiratory outcomes.	—
Delfino et al. (1998) So. California community Aug. - Oct. 1995 Highest 24-hour PM_{10} mean: 54 µg/m^3. PM_{10} and ozone PM was measured using a tapered-element oscillating microbalance. PM_{10} ranged from 6 to 51 µg/m^3 with a mean of 26.	Relationship of asthma symptoms to O_3 and PM_{10} examined in a So. California community with high O_3 and low PM. Panel of 25 asthmatics ages 9 - 17 followed daily, Aug. - Oct., 1995. Longitudinal regression analyses utilized GEE model controlling for autocorrelation, day of week, outdoor fungi and weather.	Asthma symptoms scores significantly associated with both outdoor O_3 and PM_{10} in single pollutant and co-regressions. 1-hr and 8-hr maxi PM_{10} had larger effects than 24-hr mean.	24-h - 1.47 (0.90-2.39) 8-h - 2.17 (1.33-3.58) 1-h - 1.78 (1.25-2.53)
Yu et al. (2000) study of a panel of 133 children aged 5-12 years in Seattle, WA. PM was measured by gravimetric and nephelometry methods. $PM_{1.0}$ ranged from 2 to 62 µg/m^3 with a mean of 10.4. PM_{10} 9 to 86 µg/m^3 mean 24.7.	Daily diary records were collected from November 1993 through August 1995 during screening for the CAMP study. A repeated measures logistic regression analysis was used applied using GEE methods	One day lag CO and PM_{10} levels and the same day PM_{10} and SO_2 levels had the strongest effects on asthma symptoms after controlling for subject specific variables and time-dependent confounders.	OR symptom = 1.18 (1.05, 1.33) (PM_{10} same day) OR symptom = 1.17 (1.04, 1.33) (PM_{10} one day lag)
Ostro et al. (2001) studied exacerbation of asthma in African-American children in Los Angeles. PM was measured by a beta-attenuated Andersen monitor. PM_{10} ranged from 21 to 119 µg/m^3 with a mean of 51.8.	138 children aged 8 to 13 years who had physician diagnosed asthma were included. A daily diary was used to record symptoms and medication use. GEE methods were used to estimate the effects of air pollution on symptoms controlling for meteorological and temporal variables.	Symptoms were generally related to PM_{10} and NO_2, but not to ozone. Reported associations were for pollutant variables lagged 3 days. Results for other lag times were not reported.	24-h OR wheeze = 1.02 (0.99, 106))PM_{10} lag 3 days) OR cough = 1.06 (1.02, 1.09) (PM_{10} lag 3 days) OR shortness of breath = 1.08 (1.02, 1.13) (PM_{10} lag 3 days) 1-h OR cough = 1.05 (1.02, 1.18) lag 3 days

TABLE 8B-5 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON SYMPTOMS IN STUDIES OFASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 $\mu g/m^3$ PM_{10} (25 $\mu g/m^3$ $PM_{2.5}$). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
United States (cont'd)			
Delfino et al. (2002) PM_{10}, ozone, NO_2, fungi, pollen, temperature, relative humidity	22 asthmatic children aged 9-19 were followed March through April of 1996. Study used an asthma symptom score.	No relationship between PM_{10} and symptom score was found	Lag 0 Score OR = 1.17 (0.53, 2.59) 3 Day moving average Score OR = 1.49 (0.71, 2.59) all for 50 $\mu g/m^3$ increase in PM_{10}
Mortimer et al. (2002) Eight U.S. urban areas Daily PM10 were collected in Chicago, Cleveland, and Detroit with an average intra-diary range of 53 $\mu g/m^3$ from the Aerometric Information Retrieval System of EPA.	Study of 846 asthmatic children in the eight urban area National Cooperative Inner City Asthma study. Peak flow and diary symptom data are the outcome measures. Morning symptoms consist of cough, chest tightness, and wheeze. Mixed linear and GEE models were used.	In the three cities with PM_{10} data, a stronger association was seen for PM_{10} than ozone for respiratory symptoms.	Morning symptoms PM_{10} - 2day ave. OR = 1.26 (1.0-1.59)
Thurston et al. (1997) Summers 1991-1993. O_3, H+, sulfate, pollen, daily max temp. measured.	Three 5-day summer camps conducted in 1991, 1992, 1993. Study measured symptoms and change in lung function (morning to evening). Poisson regression for symptoms.	Ozone related to respiratory symptoms No relationship between symptoms and other pollutants.	—
Canada			
Vedal et al. (1998) PM_{10} measured by Sierra-Anderson dichotomous sampler PM_{10} range: −1 to 159 $\mu g/m^3$ Port Alberrni British, Columbia	206 children aged 6 to 13 years, 75 with physician's diagnosis of asthma. Respiratory symptom data from diaries, GEE model. Temp., humidity.	PM_{10} associated with respiratory symptoms.	Lag 0 Cough OR = 1.08 (1.00, 1.16) per 10 $\mu g/m^3$ PM_{10} increments
Europe			
Gielen et al. (1997) Amsterdam, NL PM_{10} and ozone. PM_{10} was measured using a Sierra-Anderson dichotomous sampler. PM_{10} ranged from 15 to 60 $\mu g/m^3$.	Study of 61 children aged 7 to 13 years living in Amsterdam, NL. 77 percent were taking asthma medication and the others were being hospitalized for respiratory problems. Respiratory symptoms recorded by parents in diary. Associations of air pollution evaluated using time series analyses, adjusted for pollen counts, time trend, and day of week.	Strongest relationships found with O_3, although some significant relationships found with PM_{10}.	Lag 0, Symptoms: Cough OR = 2.19 (0.77, 6.20) Bronch. Dial. OR = 0.94 (0.59, 1.50) Lag 2, Symptoms: Cough OR = 2.19 (0.47, 10.24) Bronch. Dial. OR = 2.90 (1.80, 4.66)

TABLE 8B-5 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON SYMPTOMS IN STUDIES OF ASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 $PM_{2.5}$). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Europe (cont'd)			
Hiltermann et al. (1998) Leiden, NL July-Oct 1995. Ozone, PM_{10}, NO_2, SO_2, BS PM_{10} ranged from 16 to 98 µg/m^3 with a mean of 40.	Study of 270 adult asthmatic patients from an out-patient clinic in Leiden, NL from July 3, to October 6, 1995. Respiratory symptom data obtained from diaries. An autoregressive model was fitted to the data. Covariates included temperature and day of week.	PM_{10}, O_3, and NO_2 were associated with changes in respiratory symptoms.	Lag 0, Symptoms: Cough OR = 0.93 (0.83, 1.04) Short. breath OR = 1.17 (1.03, 1.34) 7 day average, Symptoms: Cough OR = 0.94 (0.82, 1.08) Short. breath OR = 1.01 (0.86, 1.20)
Hiltermann et al. (1997) The Netherlands Ozone and PM_{10} PM_{10} averaged 40 µg/m^3,	Sixty outpatient asthmatics examined for nasal inflammatory parameters in The Netherlands from July 3 to October 6, 1995. Associations of log transformed inflammatory parameters to 24-h PM_{10} analyzed, using a linear regression model. Mugwort-pollen and O_3 were evaluated.	Inflammatory parameters in nasal lavage of patients with intermittent to severe persistent asthma were associated with ambient O_3 and allergen exposure, but not with PM_{10} exposure.	—
Peters et al. (1997a) Erfurt, Germany PM fractions measured over range of sizes from ultrafine to fine, including PM_{10}. Mean PM_{10} level: 55 µg/m^3 (max 71). Mean SO_2: 100 µg/m^3 (max 383). PM was measured using a Harvard impactor.	Study of 27 non-smoking adult asthmatics living in Erfurt, Germany during winter season 1991-1992. Diary used to record presence of cough. Symptom information analyzed using multiple logistic regression analysis.	Weak associations found with 5 day mean sulfates and respiratory symptoms.	Lag 0, PM_{10}: Cough OR = 1.32 (1.16, 1.50) Feeling ill OR = 1.20 (1.01, 1.44) 5 Day Mean, PM_{10}: Cough OR = 1.30 (1.09, 1.55) Feeling ill OR = 1.47 (1.16, 1.86) Lag 0, $PM_{2.5}$: Cough OR = 1.19 (1.07, 1.33) Feeling ill OR = 1.24 (1.09, 1.41) 5 Day Mean, $PM_{2.5}$: Cough OR = 1.02 (0.91, 1.15) Feeling ill OR = 1.21 (1.06, 1.38)
Peters et al. (1997b) Sokolov, Czech Republic Winter 1991-1992 PM_{10}, SO_2, TSP, sulfate, and particle strong acid. Median PM_{10}: 47 µg/m^3 (29, 73). Median SO_2: 46 µg/m^3 (22, 88). PM was measured using a Harvard impactor. Particle size distributions were estimated using a conduction particle counter.	Study of 89 children with asthma in Sokolov, Czech Republic. Subjects kept diaries and measured peak flow for seven months during winter of 1991-2. Logistic regression for binary outcomes used. First order autocorrelations were observed and corrected for using polynomial distributed lag structures.	Significant relationships found between TSP and sulfate with both phlegm and runny nose.	Lag 0, Symptoms: Cough OR = 1.01 (0.97, 1.07) Phlegm OR = 1.13 (1.04, 1.23) 5 Day Mean, Symptoms: Cough OR = 1.10 (1.04, 1.17) Phlegm OR = 1.17 (1.09, 1.27)

TABLE 8B-5 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON SYMPTOMS IN STUDIES OF ASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 μg/m^3 PM_{10} (25 μg/m^3 $PM_{2.5}$). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Europe (cont'd)			
Peters et al. (1997c) Sokolov, Czech Republic PM_{10} one central site. SO_4 reported. Mean PM_{10}: 55 μg/m^3, max 177 μg/m^3. SO_4 - fine: mean 8.8 μg/m^3, max 23.8 μg/m^3. PM was measured using a Harvard impactor. Particle size distributions were estimated using a conduction particle counter.	Role of medication use evaluated in panel study of 82 children, mean ages 9.8 yr., with mild asthma in Sokolov, Czech Republic Nov. 1991 - Feb 1992. Linear and logistic regression evaluated PM_{10}, SO_2, temp, RH relationships to respiratory symptoms.	Medicated children, as opposed to those not using asthma medication, increased their beta-agonist use in direct association with increases in 5-day mean of SO_4 particles <2.5 μm, but medication did not prevent decrease in PEF and increase in prevalence of cough attributable to PM air pollution.	Cough 1.16 (1.00, 1.34) 6.5 μg/m^3 increase 5-day mean SO_4 5-d Mean SO_4/increase of 6.5 μg/m^3 Beta-Agonist Use 1.46 (1.08, 1.98) Theophylline Use 0.99 (0.77, 1.26) No PM_{10} analysis
Neukirch et al. (1998) Paris, France SO_2, NO_2, PM_{13} and BS. PM was measured by radiometry. PM_{13} ranged from 9 to 95 μg/m^3 with a mean of 34.	Panel of 40 nonsmoking adult asthmatics in Paris studied. GEE models used to associate health outcomes with air pollutants. Models allowed for time-dependent covariates, adjusting for time trends, day of week, temp. and humidity.	Significant relationships found for incidence of respiratory symptoms and three or more day lags of SO_2, and NO_2. Only selected results were given.	Significant relationships found between incidence of respiratory symptoms and three or more day lags of PM_{13}.
Segala et al. (1998) Paris, France SO_2, NO_2, PM_{13} (instead of PM_{10}), and BS. PM was measured by β-radiometry.	Study of 43 mildly asthmatic children aged 7-15 yr in Paris. Patients followed Nov. 15, 1992 to May 9, 1993. Respiratory symptoms recorded daily in diary. An autoregressive model fitted to data using GEE methods. Covariates included temp. and humidity.	Effects found related to PM_{13} were less than those found related to the other pollutants.	Lag 2, Symptoms: Short. Breath OR = 1.22 (0.83, 1.81) Resp. Infect. OR = 1.66 (0.84, 3.30)
Güntzel et al. (1996) Switzerland SO_2, NO_2, TSP	An asthma reporting system was used in connection with pollutant monitoring in Switzerland from fall of 1988 to fall 1990. A Box-Jenkins ARIMA time series model was used to relate asthma to TSP, O_3, SO_2, and NO_2 after adjusting for temperature.	No significant relationships found.	—
Taggart et al. (1996) Northern England SO_2, NO_2 and BS.	Panel of 38 adult asthmatics studied July 17 to Sept. 22, 1993 in northern England. Used generalized linear model to relate pollutants to bronchial hyper-responsiveness, adjusting for temperature.	Small effects seen in relation to NO_2 and BS.	—
Just et al. (2002) PM_{13}, SO_2, NO_2, O_3	82 medically diagnosed asthmatic children living in Paris, followed for 3 months. Study measured asthma attacks and nocturnal cough, symptoms, and PEF	PM_{13} was only associated with eye irritation.	Lag 0 Asthma episodes OR = 1.34 (0.08, 20.52) for 50 μg/m^3 PM_{13}.

TABLE 8B-5 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON SYMPTOMS IN STUDIES OF ASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 $PM_{2.5}$). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Von Klot et al. (2002) $PM_{2.5-10}$, PM_{10}, NO_2, SO_2, CO, temperature	53 adult asthmatics in Erfurt, Germany in the winter 1996/1997. Study measured inhaled medication use, wheezing, shortness of breath, phlegm and cough	Medication use and wheezing were associated with $PM_{2.5-10}$	5 Day mean Corticosteroid use OR = 1.12 (1.04-1.20) for 12 µg/m^3 $PM_{2.5-10}$. Wheezing OR = 1.06 (0.98, 1.15) for 12 µg/m^3 $PM_{2.5-10}$.
Desqueyroux et al. (2002) PM_{10}, O_3, SO_2, and NO_2	60 severe asthmatic adults in Paris were followed for 13 months. Study measured incident asthma attacks	Attacks were associated with PM_{10} for lags 4 and 5 but not for lags 1, 2, and 3	Lag 1 Attack OR = 0.50 (0.18, 1.34) Lag 2 Attack OR = 0.67 (0.33, 1.47) Lag 3 Attack OR = 1.69 (0.90, 3.18) Lag 4 Attack OR = 2.19 (1.16, 4.16) Lag 5 Attack OR = 2.10 (1.05, 4.32) all for 50 µg/m^3 increase in PM_{10}
Latin America			
Romieu et al. (1997) Mexico City, Mexico During study period, max daily 1-h O_3 range: 40 to 390 ppb (mean 196 ppb SD = 78 ppb) PM_{10} daily average range: 12 to 126 µg/m^3. PM was measured by a Harvard impactor.	Study of 65 children with mild asthma aged 5-13 yr living in southwest Mexico City. Respiratory symptoms recorded by the parents in daily diary. An autoregressive logistic regression model used to analyze presence of respiratory symptoms.	Strongest relationships found between O_3 and respiratory symptoms.	Lag 0, Symptoms: Cough OR = 1.05 (0.92, 1.18) Phlegm OR = 1.05 (0.83, 1.36) Diff. Breath OR = 1.13 (0.95, 1.33) Lag 2, Symptoms: Cough OR = 1.00 (0.92, 1.10) Phlegm OR = 1.00 (0.86, 1.16) Diff. Breath OR = 1.2 (1.1, 1.36)
Romieu et al. (1996) During study period, max daily range: 40 to 370 ppb (mean 190 ppb, SD = 80 ppb). 24 h ave. PM_{10} levels range: 29 to 363 µg/m^3 (mean 166.8 µg/m^3, SD 72.8 µg/m^3). PM_{10} levels exceeded 150 µg/m^3 for 53% of study days. 24-h ave. $PM_{2.5}$ levels range 23-177 µg/m^3 (mean 85.7 µg/m^3) PM was measured by a Harvard impactor.	Study of 71 children with mild asthma aged 5-7 yr living in northern Mexico City. Respiratory symptoms recorded by parents in daily diary. An autoregressive logistic regression model was used to analyze the presence of respiratory symptoms.	Cough and LRI were associated with increased O_3 and PM_{10} levels.	PM_{10} (lag 0) increase of 50 µg/m^3 related to: LRI = 1.21 (1.10, 1.42) Cough = 1.27 (1.16, 1.42) Phlegm = 1.21 (1.00, 1.48) $PM_{2.5}$ (lag 0) increase of 25 µg/m^3 related to: LRI = 1.18 (1.05, 1.36) Cough = 1.21 (1.05, 1.39) Phlegm = 1.21 (1.03, 1.42)

Appendix 8B.6: Short-Term PM Exposure Effects On Pulmonary Function in Nonasthmatics

TABLE 8B-6. SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF NONASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
United States			
Hoek et al. (1998) (summary paper)	Results summarized from several other studies reported in the literature. These included: asymptomatic children in the Utah Valley (Pope et al., 1991), children in Bennekom, NL (Roemer et al., 1993), children in Uniontown, PA (Neas et al., 1995), and children in State College, PA (Neas et al., 1996). Analyses done using a first-order autoregressive model with adjustments for time trend and ambient temp.	Other pollutants not considered.	Significant decreases in peak flow found to be related to PM_{10} increases.
Lee and Shy (1999) North Carolina Mean 24 h PM_{10} conc. over two years: 25.1 µg/m^3.	Study of the respiratory health status of residents whose households lived in six communities near an incinerator in southwestern North Carolina. Daily PEFR measured in the afternoon was regressed against 24 hour PM_{10} level lagged by one day. Results were adjusted for gender, age, height, and hypersensitivity.	PM_{10} was not related to variations in respiratory health as measured by PEFR.	—
Korrick et al. (1998) Mt. Washington, NH O_3 levels measured at 2 sites near top of the mountain. $PM_{2.5}$ measured near base of the mountain. PM was measured by a Harvard impactor.	Study of the effects of air pollution on adult hikers on Mt. Washington, NH. Linear and non-linear regressions used to evaluate effects of pollution on lung function.	$PM_{2.5}$ had no effect on the O_3 regression coefficient.	—
Naeher et al. (1999) Virginia PM_{10}, $PM_{2.5}$, sulfate fraction, H+, and ozone	Daily change in PEF studied in 473 non-smoking women in Virginia during summers 1995-1996. Separate regression models run, using normalized morning and evening PEF for each individual.	Ozone was only pollutant related to evening PEF.	Morning PEF decrements were associated with PM_{10}, $PM_{2.5}$, and H+. Estimated effect from $PM_{2.5}$ and PM_{10} was similar. No PM effects found for evening PEF.
Neas et al. (1996) State College, PA $PM_{2.1}$: mean 23.5; max 85.8 µg/m^3.	Study of 108 children in State College, PA, during summer of 1991 for daily variations in symptoms and PEFRs in relation to $PM_{2.1}$ An autoregressive linear regression model was used. The regression was weighted by reciprocal number of children of each reporting period. Fungus spore conc., temp., O_3 and SO_2 were examined.	Spore concentration associated with deficient in morning PERF.	$PM_{2.1}$ (25 µg/m^3) related to RR of: PM PFER (lag 0) = −0.05 (−1.73, 0.63) PM PEFR (lag 1) = −0.64 (−1.73, 0.44)

TABLE 8B-6 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF NONASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 μg/m^3 PM_{10} (25 μg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
United States (cont'd)			
Neas et al. (1999) Philadelphia, PA Median PM_{10} level: 31.6 in SW camps, 27.8 in NE camps (IQR ranges of about 18). Median $PM_{2.5}$ level: 22.2 in the SW camps, 20.7 in NE camps (IQR ranges about 16.2 and 12.9, respectively). Particle-strong acidity, fine sulfate particle, and O_3 also measured.	Panel study of 156 normal children attending YMCA and YWCA summer camps in greater Philadelphia area in 1993. Children followed for at most 54 days. Morning and evening deviations of each child's PEF were analyzed using a mixed-effects model adjusting for autocorrelation. Covariates included time trend and temp. Lags not used in the analysis.	Analyses that included sulfate fraction and O_3 separately also found relationship to decreased flow. No analyses reported for multiple pollutant models.	Lag 0, PM_{10}: Morning PEF = -8.16 (−14.81, −1.55) Evening PEF = −1.44 (−7.33, 4.44) 5 day ave, PM_{10} Morning PEF = 2.64 (−6.56, 11.83) Evening PEF = 1.47 (−7.31, 10.22) Lag 0, $PM_{2.5}$ Morning PEF = −3.28 (−6.64, 0.07) Evening PEF = −0.91 (−4.04, 2.21) 5 day ave., $PM_{2.5}$ Morning PEF = 3.18 (−2.64, 9.02) Evening PEF = 0.95 (−4.69, 6.57)
Schwartz and Neas (2000) Eastern U.S. $PM_{2.5}$ and CM ($PM_{10-2.5}$) measured. Summary levels not given.	Analyses for 1844 school children in grades 2-5 from six urban areas in eastern U.S. and from separate studies from Uniontown and State College, PA. Lower resp. symptoms, cough and PEF used as endpoints. The authors replicated models used in the original analyses. CM and were used individually and jointly in the analyses. Sulfate fractions also used in the analyses. Details of models not given.	Sulfate fraction was highly correlated with $PM_{2.5}$ (0.94), and, not surprisingly, gave similar answers.	Uniontown Lag 0,$PM_{2.5}$: Evening PEF = −1.52 (−2.80, −0.24) State College Lag 0, $PM_{2.5}$: Evening PEF = −0.93 (−1.88, 0.01) Results presented for CM showed no effect. Results for PM_{10} were not given.
Linn et al. (1996) So. California NO_2 ozone, and PM_5 measured. PM_5 was measured using a Marple low volume sampler PM_5 ranged from 1-145 μg/m^3 with a mean of 24.	Study of 269 school children in Southern California twice daily for one week in fall, winter and spring for two years. A repeated measures analysis of covariance was used to fit an autoregressive model, adjusting for year, season, day of week, and temperature.	Morning FVC was significantly decreased as a function of PM_5 and NO_2	—

TABLE 8B-6 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF NONASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 μg/m^3 PM_{10} (25 μg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Canada			
Vedal et al. (1998) Port Alberni, BC PM_{10} via a Sierra-Anderson dichotomous sampler. PM_{10} ranged from 1 to 159 μg/m^3.	Study of 206 children aged 6 to 13 years living in Port Alberni, British Columbia. 75 children had physician-diagnosed asthma, 57 had an exercised induced fall in FEV1, 18 children with airway obstruction, and 56 children without any symptoms. Respiratory symptom data obtained from diaries. An autoregressive model was fitted to the data, using GEE methods. Covariates included temp., humidity, and precipitation.	No consistent evidence for adverse health effects was seen in the nonasthmatic control group.	—
Europe			
Boezen et al. (1999) Netherlands PM_{10}, BS, SO_2, and NO_2 measured, but methods were not given. PM_{10} ranged from 4.8 to 145 μg/m^3 with site means ranging from 26 to 54 μg/m^3.	Data collected from children during three winters (1992-1995) in rural and urban areas of The Netherlands. Study attempted to investigate whether children with bronchial hyperresponsiveness and high serum Ige levels were more susceptible to air pollution. Prevalence of a 10 percent PEF decrease was related to pollutants for children with bronchial hyperresponsiveness and high serum Ige levels.	No consistent pattern of effects observed with any of the pollutants for 0, 1, and 2 day lags.	—
Frischer et al. (1999) Austria PM_{10} measured gravimettrically for 14-d periods. Annual mean PM_{10} levels range: 13.6 - 22.9 μg/m^3. O_3 range: 39.1 ppb - 18.5 pbs between sites.	At nine sites in Austria during 1994, 1995, and 1996, a longitudinal study designed to evaluate O_3 was conducted. During 1994 - 1996, children were measured for FVC, FEV_1 and MEF_{50} six times, twice a year in spring and fall. 1060 children provided valid function tests. Mean age 7.8 ± 0.7 yr. GEE models used. PM_{10}, SO_2, NO_2, and temp. evaluated.	Small but consistent lung function decrements in cohort of school children associated with ambient O_3 exposure.	PM_{10} showed little variation in exposure between study site. For PM_{10}, positive effect seen for winter exposure but was completely confounded by temperature. PM_{10} Summertime β = 0.003 SE 0.012 p=0.77
Grievink et al. (1999) Netherlands PM_{10} and BS. PM_{10} ranged from 12 to 123 μg/m^3 with a mean of 44.	A panel of adults with chronic respiratory symptoms studied over two winters in The Netherlands starting in 1993/1994. Logistic regression analysis was used to model the prevalence of large PEF decrements. Individual linear regression analysis of PEF on PM was calculated and adjusted for time trends, influenza incidence, and meteorological variables.	Subjects with low levels of serum β-carotene more often had large PEF decrements when PM_{10} levels were higher, compared with subjects with high serum β-carotene. Results suggested serum β-carotene may attenuate the PM effects on decreased PEF.	—

TABLE 8B-6 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF NONASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 μg/m^3 PM_{10} (25 μg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Europe (cont'd)			
Künzli et al. (2000)	Ackermann-Liebrich et al. (1997) data reanalyzed. Authors showed that a small change in FVC (-3.14 percent) can result in a 60% increase in number of subjects with FVC less than 80 percent of predicted.	The results were for two hypothetical communities, A and B.	—
Roemer et al. (2000) PM_{10} means for 17 panels ranged 11.2 to 98.8 μg/m^3. SO_2, NO_2, and elemental content of PM also measured. Measurement methods were not described.	Combined results from 1208 children divided among 17 panels studied. Separate results reported by endpoints included symptoms as reported in a dairy and PEF. Individual panels were analyzed using multiple linear regression analysis on deviations from mean PEF adjusting for auto-correlation. Parameter estimates were combined using a fixed-effects model where heterogeneity was not present and a random-effects model where it was present.	Daily concentrations of most elements were not associated with the health effects.	PM_{10} analyses not focus of this paper.
Scarlett et al. (1996) PM_{10}, O_3, and NO_2 measured.	In study of 154 school children, pulmonary function was measured daily for 31 days. Separate autoregressive models for each child were pooled, adjusting for pollen, machine, operator, time of day, and time trend.	PM_{10} was related to changes in FEV and FVC	—
van der Zee et al. (1999) Netherlands PM_{10} averages ranged 20 to 48 μg/m^3. BS, sulfate fraction, SO_2, and NO_2 also measured.	Panel study of 795 children aged 7 to 11 years, with and without chronic respiratory symptoms living in urban and nonurban areas in the Netherlands. Peak flow measured for three winters starting in 1992/1993. Peak flow dichotomized at 10 and 20% decrements below the individual median. Number of subjects was used as a weight. Minimum temperature day of week, and time trend variables were used as covariates. Lags of 0, 1 and 2 days were used, as well as 5 day moving average.	In children with symptoms, significant associations found between PM_{10}, BS and sulfate fraction and the health endpoints. No multiple pollutant models analyses reported.	Lag 0, PM_{10}, Urban areas Evening PEF OR = 1.15 (1.02, 1.29) Lag 2, PM_{10}, Urban areas Evening PEF OR = 1.07 (0.96, 1.19) 5 day ave, PM_{10}, Urban areas Evening PEF = 1.13 (0.96, 1.32)

TABLE 8B-6 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF NONASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 μg/m^3 PM_{10} (25 μg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Europe (cont'd)			
van der Zee et al. (2000) Netherlands PM_{10} averages ranged 24 to 53 μg/m^3. BS, sulfate fraction, SO_2, and NO_2 also measured. PM_{10} was measured using a Sierra Anderson 241 dichotomous sampler.	Panel study of 489 adults aged 50-70 yr, with and without chronic respiratory symptoms, living in urban and nonurban areas in the Netherlands. Resp. symptoms and peak flow measured for three winters starting in 1992/1993. Symptom variables analyzed as a panel instead of using individual responses. The analysis was treated as a time series, adjusting for first order autocorrelation. Peak flow dichotomized at 10 and 20% decrements below the individual median. The number of subjects used as a weight. Minimum temp., day of week, and time trend variables used as covariates. Lags of 0, 1 and 2 days used, as well as 5 day moving average.	BS tended to have the most consistent relationship across endpoints. Sulfate fraction also related to increased respiratory effects. No analyses reported for multiple pollutant models. Relationship found between PM_{10} and the presence of 20% decrements in symptomatic subjects from urban areas.	Lag 0, PM_{10}, Urban areas Morning large decrements OR = 1.44 (1.02, 2.03) Lag 2, PM_{10}, Urban areas Morning large decrements OR = 1.14 (0.83, 1.58) 5 day ave, PM_{10}, Urban areas Morning large decrements OR = 1.16 (0.64, 2.10) Results should be viewed with caution because of problems in analysis.
Tiittanen et al. (1999) Kupio, Finland Median PM_{10} level: 28 (25^{th}, 75^{th} percentiles = 12, 43). Median $PM_{2.5}$ level: 15 (25^{th}, 75^{th} percentiles = 9, 23). Black carbon, CO, SO_2, NO_2, and O_3 also measured. PM was measured using single stage Harvard samplers.	Six-week panel study of 49 children with chronic respiratory disease followed in the spring of 1995 in Kuopio, Finland. Morning and evening deviations of each child's PEF analyzed, using a general linear model estimated by PROC MIXED. Covariates included a time trend, day of week, temp., and humidity. Lags of 0 through 3 days were used, as well as a 4-day moving average. Various fine particles were examined.	Ozone strengthened the observed associations. Introducing either NO_2 or SO_2 in the model did not change the results markedly. Effects varied by lag. Separating effects by size was difficult.	Lag 0, PM_{10}: Morning PEF = 1.21 (−0.43, 2.85) Evening PEF = 0.72 (−0.63, 1.26) 4 day ave, PM_{10} Morning PEF = −1.26 (−5.86, 3.33) Evening PEF = 2.33 (−2.62, 7.28) Lag 0, $PM_{2.5}$ Morning PEF = 1.11 (−0.64, 2.86) Evening PEF = 0.70 (−0.81, 2.20) 4 day ave., $PM_{2.5}$ Morning PEF = −1.93 (−7.00, 3.15) Evening PEF = 1.52 (−3.91, 6.94)

TABLE 8B-6 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF NONASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 μg/m^3 PM_{10} (25 μg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Europe (cont'd)			
Ward et al. (2000) West Midlands, UK Daily measurements of PM_{10}, $PM_{2.5}$, SO_2, CO, O_3, and oxides of nitrogen. Details on PM monitoring were inomplete.	Panel study of 9 yr old children in West Midlands, UK for two 8-week periods representing winter and summer conditions. Individual PEF values converted to z-values. Mean of the z-values analyzed in a linear regression model, including terms for time trend, day of week, meteorological variables, and pollen count. Lags up to four days also used.	Results on effects of pollution on lung function to be published elsewhere.	—
Osunsanya et al. (2001) studied 44 patients aged > 50 with COPD in Aberdeen, UK. PM was measured using tapered element oscillating microbalance. Particle sizes were measured a TSI model 3934 scanning particle sizer. PM_{10} ranged from 6 to 34 μg/m^3 with a median of 13.	Symptom scores, bronchodilator use, and PEF were recorded daily for three months. GEE methods were used to analyze the dichotomous outcome measures. PEF was converted to a dichotomous measure by defining a 10 percent decrement as the outcome of interest.	No associations were found between actual PEF and $_{PM10}$ or ultrafine particles. A change of $_{PM10}$ from 10 to 20 μg/m^3 was associated with a 14 percent decrease in the rate of high scores of shortness of breath. A similar change in PM_{10} was associated with a rate of high scores of cough.	The endpoint was measured in terms of scores rather than L/min.
Cuijpers et al. (1994) Maastricht, NL SO_2, NO_2, BS, ozone, and H+ measured. PM measurements were made with a modified Sierra Anderson sampler. PM_{10} ranged from 23 to 54 μg/m^3.	Summer episodes in Maastricht, The Netherlands studied. Paired t tests used for pulmonary function tests.	Small decreases in lung function found related to pollutants.	Quantitative results not given.
Latin America			
Gold et al. (1999) Mexico City, Mexico Mean 24 h O_3 levels: 52 ppb. Mean $PM_{2.5}$: 30 μg/m^3. Mean PM_{10}: 49 μg/m^3.	Peak flow studied in a panel of 40 school-aged children living in southwest Mexico City. Daily deviations from morning and afternoon PEFs calculated for each subject. Changes in PEF regressed on individual pollutants allowing for autocorrelation and including terms for daily temp., season, and time trend.	O_3 significantly contributed to observed decreases in lung function, but there was an independent PM effect.	Both $PM_{2.5}$ and PM_{10} significantly related to decreases in morning and afternoon peak flow. Effects of the two pollutants similar in magnitude when compared on percent change basis.

TABLE 8B-6 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON PULMONARY FUNCTION TESTS IN STUDIES OF NONASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
New Zealand			
Harré et al. (1997) Christchurch, NZ SO_2, NO_2, PM_{10}, and CO measured. Details on monitoring methods and pollutant ranges were not given.	Study of 40 subjects aged over 55 years with COPD living in Christchurch, New Zealand conducted during winter of 1994. Subjects recorded their peak flow measurements. A log-linear regression model with adjustment for first order auto-correlation was used to analyze peak flow data and a Poisson regression model was used to analyze symptom data.	Few significant associations found between the health endpoints and the pollutants.	Lag 0, PM_{10}: PEF = −0.86 (−2.33, 0.61)
Jalaludin et al. (2000) studied PEF in 148 children 6 primary schools in Sydney, Australia. PM was measured by tapered element oscillating microbalance. Mean PM_{10} was 22.8 +- 13.9 µg/m^3.	148 children in grades 3-5 were followed for 11 months, recording PEF twice daily. The normalized change in PEF was analyzed using GEE methods. PEF was related to SO_3, PM_{10}, NO_2, as well as meteorological variables.	Daily mean deviations in PEF were related to ozone, but no relationships were found with PM_{10} or NO_2. Multiple pollutant models gave similar results to those given by the single pollutant models.	Change from AM to PM PEF = 0.045 (−.205, 2.95) lag one day
Asia			
Chen et al. (1999) Taiwan Beta-gauge PM_{10} ranged 44.5 to 189.0 µg/m^3 for peak concentrations.	In 3 Taiwan communities in 1995, PM_{10} by B-gauge measured at selected primary schools in each community. Spirometry tests (FVC, $FEV_{1.0}$, $FEF_{25-75\%}$, PEF) obtained in period May 1995 to Jan. 1996 using ATS protocol in study pop. aged 8 to 13 yr. 895 children were analyzed. Study was designed to investigate short-term effect of ambient air pollution in cross-sectional survey. Multivariate linear model analysis used in both one pollutant and multipollutant models, with 1-, 2-, and 7-day lags. SO_2, CO, O_3, NO_2 and PM_{10} examined, as were meteorol. variables.	In the one-pollutant model, daytime peak O_3 conc. with a 1-day lag significantly affected both FVC and FEV_1. NO_2, SO_2, CO affected FVC. PM_{10} showed nonsignificant decrement. No significant result demonstrated in the model for the exposure with 7 days lag. In the multi-pollutant model, only peak O_3 conc. with 1-day lag showed sig. effect on FVC and $FEV_{1.0}$.	One pollutant model daytime average PM_{10} – 2 day lag FVC −0.37 se 0.39
Tan et al. (2000) Southeast Asian smoke-haze event 9/29 – 10/27 1997 PM_{10} mean daily was 125.4 ± 44.9 µg/m^3 ultra range of 47 to 216 µg/m^3 in Singapore	Examined the association between acute air pollution caused by biomass burning and peripheral UBC counts in human serial measurement made during the event were compared with a period after the haze cleared (Nov. 21 – Dec. 5, 1997)	Indices of atmospheric pollution were significantly associated in the elevated band neutrophil counts expressed as a percentage of total polymonphonuclear leukocytes (PMN). No statistically significant difference in FEU_1 and FUC were observed during and after haze exposure.	

Appendix 8B.7: Short-Term PM Exposure Effects On Symptoms in Nonasthmatics

TABLE 8B-7. SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON SYMPTOMS IN STUDIES OF NONASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
United States			
Schwartz and Neas (2000) Eastern U.S. $PM_{2.5}$ and CM ($PM_{10-2.5}$ by substation).. Summary levels not given	Reported on analysis of 1844 school children in grades 2–5 from six urban areas in the eastern U.S., and from separate studies from Uniontown and State College, PA. Lower respiratory symptoms, and cough used as endpoints. The authors replicated the models used in the original analyses. CM and $PM_{2.5}$ were used individually and jointly in the analyses. Sulfates fractions were also used in the analyses. Details of the models were not given.	Sulfate fraction was highly correlated with $PM_{2.5}$ (0.94), and not surprisingly gave similar answers.	$PM_{2.5}$ was found to be significantly related to lower respiratory symptoms even after adjusting for CM, whereas the reverse was not true. However, for cough, CM was found to be significantly related to lower respiratory symptoms even after adjusting for $PM_{2.5}$, whereas the reverse was not true.
Zhang et al. (2000) Vinton, Virginia 24- h PM_{10}, $PM_{2.5}$, sulfate and strong acid measured in 1995.	In southwestern Virginia, 673 mothers were followed June 10 to Aug. 31, 1995 for the daily reports of present or absence of runny or stuffy nose. PM indicator, O_3, NO_2 temp., and random sociodemographic characteristics considered.	Of all pollutants considered, only the level of coarse particles as calculated (PM_{10} - $PM_{2.5}$) independently related to incidence of new episode of runny noses.	—
Canada			
Vedal et al. (1998) Port Alberni, BC PM_{10} via a Sierra-Anderson dichotomous sampler. PM_{10} ranged from 1 to 159 µg/m^3.	Study of 206 children aged 6 to 13 years living in Port Alberni, British Columbia. 75 children had physician-diagnosed asthma, 57 had an exercised induced fall in FEV1, 18 children with airway obstruction, and 56 children without any symptoms. Respiratory symptom data obtained from diaries. An autoregressive model was fitted to the data, using GEE methods. Covariates included temp., humidity, and precipitation.	No consistent evidence for adverse health effects was seen in the nonasthmatic control group.	—
Long et al. (1998) Winnepeg, CN PM_{10}, TSP, and VOC measured. Methods for PM monitoring not given. Ranges of values also not given.	Study of 428 participants with mild airway obstruction conducted during a Winnepeg pollution episode. Gender specific odds ratios of symptoms were calculated for differing PM_{10} levels using the Breslow-Day test.	Cough, wheezing, chest tightness, and shortness of breath were all increased during the episode	—
Europe			
Boezen et al. (1998) Amsterdam, NL PM_{10}, SO_2, and NO_2 measured. PM_{10} ranged from 7.9 to 242.2 µg/m^3 with a median of 43.	Study of 75 symptomatic and asymp. adults near Amsterdam for three months during winter 1993-1994. An autoregressive logistic model was used to relate PM_{10} to respiratory symptoms, cough, and phlegm, adjusting for daily min. temp., time trend, day of week.	No relationship found with pulmonary function. Some significant relationships with respiratory disease found in subpopulations	—

TABLE 8B-7 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON SYMPTOMS IN STUDIES OF NONASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 µg/m^3 PM_{10} (25 µg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Europe (cont'd)			
Howel et al. (2001) study of children's respiratory health in 10 non-urban communities of northern England. PM levels were measured using a single continuous real-time monitor. PM_{10} levels ranged from 5 to 54 µg/m^3.	The study included 5 pairs of non-urban communities near and not so near 5 coal mining sites. 1405 children aged 1-11 years were included. 275 of the children reported having asthma. Diaries of respiratory symptoms were collected over a 6 week period. PM_{10}, measured by a single continuous real-time monitor, ranged from 5 to 54 µg/m^3.	The associations found between daily PM_{10} levels and respiratory symptoms were frequently small and positive and sometimes varied by community.	OR wheeze = 1.16 (1.05, 1.28 (PM_{10}) OR cough = 1.09 (1.02, 1.16) (PM_{10}) OR reliever use = 1.00 (0.94, 1.06) (PM_{10})
Roemer et al. (1998) Mean PM_{10} levels measured at local sites ranged 11.2 to 98.8 µg/m^3 over the 28 sites.	Pollution Effects on Asthmatic Children in Europe (PEACE) study was a multi-center study of PM_{10}, BS, SO_2, and NO_2 on respiratory health of children with chronic respiratory symptoms. Results from individual centers were reported by Kotesovec et al. (1998), Kalandidi et al. (1998), Haluszka et al. (1998), Forsberg et al. (1998), Clench-Aas et al. (1998), and Beyer et al. (1998). Children with chronic respiratory symptoms were selected into the panels. The symptom with one of the larger selection percentages was dry cough (range over sample of study communities 29 to 92% [22/75; 84/91] with most values over 50%). The group as a whole characterized as those with chronic respiratory disease, especially cough.	These studies modeled group rates and are an example of the panel data problem.	—
Roemer et al. (2000) PM_{10} means for the 17 panels ranged 11.2 to 98.8 µg/m^3. SO_2, NO_2, and PM elemental content also measured. Measurement methods were not described.	Combined results from 1208 children divided among 17 panels studied. Endpoints included symptoms as reported in a dairy and PEF. Symptom variables analyzed as a panel instead of using individual responses. The analysis was treated as a time series, adjusting for first order autocorrelation. Parameter estimates were combined using a fixed-effects model where heterogeneity was not present and a random-effects model where it was present.	Daily concentrations of most elements were not associated with the health effects.	The analysis of PM_{10} was not a focus of this paper.

TABLE 8B-7 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON SYMPTOMS IN STUDIES OF NONASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 μg/m^3 PM_{10} (25 μg/m^3 $PM_{2.5}$). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
Europe (cont'd)			
van der Zee et al. (1999) Netherlands PM_{10} averages ranged 20 to 48 μg/m^3. BS, sulfate fraction, SO_2, and NO_2 also measured.	A panel study of 795 children aged 7 to 11 yr, with and without chronic respiratory symptoms, living in urban and nonurban areas in the Netherlands. Respiratory symptoms measured for 3 winters starting 1992/1993. Symptom variables analyzed as a panel instead of using individual responses. The analysis was treated as a time series, adjusting for first order autocorrelation. The number of subjects was used as a weight. Minimum temp., day of week, and time trend variables used as covariates. Lags of 0, 1 and 2 days used, as well as 5 day moving average.	In children with symptoms, significant associations found between PM_{10}, BS and sulfate fraction and the health endpoints. No analyses reported with multiple pollutant models.	Lag 0, PM_{10}, Urban areas Cough OR = 1.04 (0.95, 1.14) Lag 2, PM_{10}, Urban areas Cough OR = 0.94 (0.89, 1.06) 5 day ave, PM_{10}, Urban areas Cough OR = 0.95 (0.80, 1.13)
van der Zee et al. (2000) Netherlands Daily measurements of PM_{10}, BS, fine sulfate, nitrate, ammonium and strong acidity. PM_{10} was measured using a Sierra Anderson 241 dichotomous sampler.	Panel study of adults aged 50 to 70 yr during 3 consecutive winters starting in 1992/1993. Symptom variables analyzed as a panel instead of using individual responses. Analysis treated as a time series, adjusting for first order autocorrelation. Number of subjects used as a weight. Min. temp., day of week, time trend variables used as covariates. Lags 0, 1 and 2 days used, as well as 5 day moving average.	BS was associated with upper respiratory symptoms.	Lag 0, Symptoms, Urban areas LRS OR = 0.98 (0.89, 1.08) URS OR = 1.04 (0.96, 1.14) Lag 2, Symptoms, Urban areas LRS OR = 1.01 (0.93, 1.10) URS OR = 1.04 (0.96, 1.13) 5 day ave, Symptoms, Urban areas LRS OR = 0.95 (0.82, 1.11) URS OR = 1.17 (1.00, 1.37)
Tiittanen et al. (1999) Kupio, Finland Median PM_{10} level: 28 (25^{th}, 75^{th} percentiles = 12, 43). Median $PM_{2.5}$: 15 (25^{th} and 75^{th} percentiles of 9 and 23). Black carbon, CO, SO_2, NO_2, and O_3 also measured. PM was measured using single stage Harvard samplers.	Six-week panel study of 49 children with chronic respiratory disease followed in spring 1995 in Kuopio, Finland. Cough, phlegm, URS, LRS and medication use analyzed, using a random effects logistic regression model (SAS macro GLIMMIX). Covariates included a time trend, day of week, temp., and humidity. Lags of 0 to 3 days used, as well as 4-day moving average.	Ozone strengthened the observed associations. Introducing either NO_2 or SO_2 in the model did not change the results markedly.	Lag 0, PM_{10}: Cough OR = 1.00 (0.87, 1.16) 4 day ave, PM_{10} Cough OR = 1.58 (0.87, 2.83) Lag 0, $PM_{2.5}$ Cough OR = 1.04 (0.88, 1.23) 4 day ave., $PM_{2.5}$ Cough OR = 2.01 (1.04, 3.89)
Keles et al. (1999) Istanbul, Turkey Nov. 1996 to Jan. 1997. TSP levels ranged from annual mean of 22 μg/m^3 in unpolluted area to 148.8 μg/m^3 in polluted area.	Symptoms of rhinitis and atopic status were evaluated in 386 students grades 9 and 10 using statistical package for the social sciences, Fisher tests, and multiple regression model as Spearman's coefficient of correlation.	No difference found for atopic status in children living in area with different air pollution levels.	—

TABLE 8B-7 (cont'd). SHORT-TERM PARTICULATE MATTER EXPOSURE EFFECTS ON SYMPTOMS IN STUDIES OF NONASTHMATICS

Reference citation, location, duration, pollutants measured, summary of values	Type of study, sample size, health outcomes measured, analysis design, covariates included, analysis problems, etc.	Results and Comments Effects of co-pollutants	Effect measures standardized to 50 μg/m^3 PM_{10} (25 μg/m^3 PM_{2-5}). Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest PM effects
New Zealand			
Harré et al. (1997) Christchurch, NZ SO_2, NO_2, PM_{10}, and CO measured. Details on monitoring methods and pollutant ranges were not given.	Study of 40 subjects aged 55 years with COPD living in Christchurch, New Zealand during winter 1994. Subjects recorded completed diaries twice daily. Poisson regression model used to analyze symptom data.	NO_2 was associated with increased bronchodilator use.	PM_{10} was associated with increased nighttime chest symptoms.
Asia			
Awasthi et al. (1996) India Suspended particulate matter, SO_2, nitrates, coal, wood, PM and kerosene measured. SPM was measured using a high-volume sampler.	A cohort of 664 preschool children studied for two weeks each in northern India. Ordinary least squares was used to relate a respiratory symptom complex pollutants.	A significant regression coefficient between PM and symptoms was found	—

Appendix 8B.8: Long-Term PM Exposure Effects On Respiratory Health Indicators, Symptoms, and Lung Function

TABLE 8B-8. LONG-TERM PARTICULATE MATTER EXPOSURE RESPIRATORY HEALTH INDICATORS: RESPIRATORY SYMPTOM, LUNG FUNCTION

Reference citation, location, duration, type of study, sample size, pollutants measured, summary of values	Health outcomes measured, analysis design, covariates included, analysis problems	Results and Comments Effects of co-pollutants	Effect estimates as reported by study authors. Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest effects of PM
United States			
Abbey et al. (1998) California Communities 20 year exposure to respirable particulates, suspended sulfates, ozone, and PM_{10}. PM_{10} ranged from 1 to 145 µg/m^3 with a mean value of 32.8.	Sex specific multiple linear regressions were used to relate lung function measures to various pollutants in long-running cohort study of Seven Day Adventists (ASHMOG Study).	Sulfates were associated with decreases in FEV.	Frequency of days where PM_{10} > 100 µg/m^3 associated with FEV decrement in males whose parents had asthma, bronchitis, emphysema, or hay fever. No effects seen in other subgroups.
Berglund et al.. (1999) California communities	Cohort study of Seventh Day Adventists. Multivariate logistic regression analysis of risk factors (e.g., PM) for chronic airway disease in elderly non-smokers, using pulmonary function test and respiratory symptom data.	Significant risk factors identified: childhood respiratory illness, reported ETS exposure, age, sex and parental history.	For PM_{10} > 100µg/m^3, 42 d/yr: RR = −1.09 CT (0.92, 1.30) for obstructive disease determined by pulmonary function tests.
Peters et al. (1999a,b) 12 southern California communities 5 year exposure to PM_{10}, ozone, NO_2, acid levels. PM_{10} annual averages ranged from 13 to 70 µg/m^3.	Asthma, bronchitis, cough and wheeze rates were adjusted for individual covariates. Community rates were then regressed on pollutant averages for 1986-1990.	Wheeze was associated with NO_2 and acid levels. No symptoms were associated with PM_{10} levels.	OR for PM_{10} (per 25 µg/m^3): 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)
Avol et al. (2001) Subjects living in Southern California in 1993 that moved to other western locations in 1998. Pollutants O_3, NO_2, PM_{10} differences 15 to 66 µg/m^3.	Studied 110 children who were 10 yrs of age at enrollment and 15 at follow-up who had moved from communities filled out health questions and underwent spirometry. Linear regression used to determine whether annual average change in lung function correlated with average changes in PM.	As a group, subjects who moved to areas of lower PM_{10} showed increased growth in lung function and subjects who moved to communities with a higher PM_{10} showed decreased growth in lung function.	PM_{10} 24 hr average PERF ml/s per 10 µg/m^3 mean = −34.9 95% CI −59.8, −10.1
Gauderman et al. (2000) 12 So. California communities 1993 to 1997 Pollutants: O_3, NO_2, PM_{10}, and $PM_{2.5}$. PM_{10} levels ranged from 16.1 to 67.6 µg/m^3 across the communities.	Studies of lung function growth of 3035 children in 12 communities within 200-mile radius of Los Angeles during 1993 to 1997. Cohorts of fourth, seventh, and tenth-graders studied. By grade cohort, a sequence of linear regression models were used to determine over the 4yr of follow-up, if average lung function growth rate of children was associated with average pollutant levels. Adjustment were made for height, weight, body mass index, height by age interaction, report of asthma activity or smoking. Two-pollutant models also used.	Lung growth rate for children in most polluted community, as compared to least polluted, was estimated to result in cumulative reduction of 3.4% in FEV_1 and 5.0% in MMEF over 4-yr study period. Estimated deficits mostly larger for children spending more time outdoors. Due to the high correlation in concentrations across communities, not able to separate effects of each pollutant. No sig. associations seen with O_3.	From the lowest to highest observed concentration of each pollutant, the predicted differences in annual growth rates were: −0.85% for PM_{10} (p = 0.026); −0.64% for $PM_{2.5}$ (p = 0.052); −0.90% for $PM_{10-2.5}$ (p = 0.030); −0.77% for NO_2 (p = 0.019); and −0.73% for inorganic acid vapor (p = 0.042).

TABLE 8B-8 (cont'd). LONG-TERM PARTICULATE MATTER EXPOSURE RESPIRATORY HEALTH INDICATORS: RESPIRATORY SYMPTOM, LUNG FUNCTION

Reference citation, location, duration, type of study, sample size, pollutants measured, summary of values	Health outcomes measured, analysis design, covariates included, analysis problems	Results and Comments Effects of co-pollutants	Effect estimates as reported by study authors. Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest effects of PM
United States (cont'd)			
Gauderman et al. (2002) Follow-up on 12 southern California communities 5 year exposure to PM_{10}, ozone, NO_2, acid levels. PM_{10} annual averages ranged from 5 to 27 µg/m^3.	Linear regression analysis was used to estimate the individual lung function growth adjusted for height, weight, body mass index, and smoking. Growth rates were then adjusted for individual covariates to obtain community adjusted growth rates. These rates were then related to pollutant averages for 1996-1999.	Lung function growth was related to total acid.	From the lowest to highest observed concentration of each pollutant, the predicted differences in annual growth rates of FEV1 were: PM_{10} −0.21 (−1.04, 0.64), ozone −0.55 (−1.27, 0.16), NO_2 −0.48 (−1.12, 0.17), $PM_{2.5}$ −0.39 (−1.06, 0.28), total acid −0.63 (−1.21, 0.17)
McConnell et al. (1999) 12 Southern California communities 1994 air monitoring data. PM_{10} (mean 34.8; range 13.0 - 70.7 µg/m^3). $PM_{2.5}$ (yearly mean 2 week averaged mean 15.3 µg/m^3; range 6.7 - 31.5 µg/m^3).	Cross-sectional study of 3,676 school children whose parents completed questionnaires in 1993 that characterized the children's history of respiratory illness. Three groups examined: (1) history of asthma; (2) wheezing but no asthma; and (3) no history of asthma or wheezing. Logistic regression model used to analyze PM, O_3, NO_2, acid vapor effects. This study also described in Peters et al. (1999b,c).	Positive association between air pollution and bronchitis and phlegm observed only among children with asthma. As PM_{10} increased across communities, a corresponding increase in risk of bronchitis per interquartile range occurred. Strongest association with phlegm was for NO_2. Because of high correlation of PM air pollution, NO_2, and acid, not possible to distinguish clearly which most likely responsible for effects.	PM_{10} Asthma Bronchitis 1.4 CI (1.1 – 1.8) Phlegm 2.1 (1.4 – 3.3) Cough 1.1 (0.8 – 1.7) No Asthma/No Wheeze Bronchitis 0.7 (0.4 – 1.0) Phlegm 0.8 (0.6 – 1.3) Cough 0.9 (0.7 – 1.2)
McConnell et al. (2002) 12 Southern California communities 1994-1997 4-year mean conc. PM_{10} µg/m^3 High community: 43.3 (12.0) Low community: 21.6 (3.8)	In 3,535 children assessed, the association of playing team sports with subsequent development of asthma during 4 yrs of follow-up. Comparing high pollutant communities to low pollutant communities. Relative risks of asthma adjusted for ethnic origin were evaluated for every pollutant with a multivariate proportional hazards model. See also Peters et al. (1999b,c).	Across all communities there was a 1.8-fold increased risk (95% CI 1.2-2.8) for asthma in children who had played three or more team sports in the previous year. In high ozone (10:00 h to 18:00 h mean concentration) communities, there was a 3.3-fold increase risk of asthma in children playing three or more sports, an increase not seen in low ozone communities.	The effect of team sports was similar in communities with high and low PM with a small increase in asthma among children playing team sports.
Dockery et al. (1996) 24 communities in the U. S. and Canada. PM_{10}, $PM_{2.5}$, sulfate fraction, H^+, ozone, SO_2, and other measures of acid were monitored. PM was measured using a Harvard impactor. PM_{10} ranged form 15.4 to 32.7 with a mean of 23.8. $PM_{2.5}$ ranged form 5.8 to 20.7 µg/m^3 with a mean of 14.5.	Respiratory health effects among 13,369 white children aged 8 to 12 yrs analyzed in relation to PM indices. Two-stage logistic regression model used to adjust for gender, history of allergies, parental asthma, parental education, smoking in home.	Although bronchitis endpoint was significantly related to fine PM sulfates, no endpoints were related to PM_{10} levels.	—

TABLE 8B-8 (cont'd). LONG-TERM PARTICULATE MATTER EXPOSURE RESPIRATORY HEALTH INDICATORS: RESPIRATORY SYMPTOM, LUNG FUNCTION

Reference citation, location, duration, type of study, sample size, pollutants measured, summary of values	Health outcomes measured, analysis design, covariates included, analysis problems	Results and Comments Effects of co-pollutants	Effect estimates as reported by study authors. Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest effects of PM
United States (cont'd)			
Raizenne et al. (1996) 24 communities in the U.S. and Canada Pollutants measured for at least one year prior to lung function tests: PM_{10}, $PM_{2.1}$, particle strong acidity, O_3, NO_2, and SO_2. PM was measured with a Harvard impactor. For pollutant ranges, see Dockery et al. (1996).	Cross-sectional study of lung function. City specific adjusted means for FEV and FVC calculated by regressing the natural logarithm of the measure on sex, ln height, and ln age. These adjusted means were then regressed on the annual pollutant means for each city.	PM measures (e.g., particle strong acidity) associated with FEV and FVC decrement.	—
Europe			
Ackermann-Liebrich et al. (1997) Eight Swiss regions Pollutants: SO_2, NO_2, TSP, O_3, and PM_{10}. PM was measured with a Harvard impactor. PM_{10} ranged from 10 to 53 µg/m^3 with a mean of 37.	Long-term effects of air pollution studied in cross-sectional population-based sample of adults aged 18 to 60 yrs. Random sample of 2,500 adults in each region drawn from registries of local inhabitants. Natural logarithms of FVC and FEV_1 regressed against natural logarithms of height, weight, age, gender, atopic status, and pollutant variables.	Significant and consistent effects on FVC and FEV were found for PM_{10}, NO_2 and SO_2.	Estimated regression coefficient for PM_{10} versus FVC = −0.035 (95% CI −0.041, −0.028). Corresponding value for FEV_1 −0.016 (95% CI −0.023 to −0.01). Thus, 10 µg/m^3 PM_{10} increase estimated to lead to estimated 3.4 percent decrease in FVC and 1.6 percent decrease in FEV_1.
Braun-Fahrländer et al. (1997) 10 Swiss communities Pollutants: PM_{10}, NO_2, SO_2, and O_3. PM was measured with a Harvard impactor. PM_{10} ranged from 10 to 33 µg/m^3.	Impacts of long-term air pollution exposure on respiratory symptoms and illnesses were evaluated in cross-sectional study of Swiss school children, (aged 6 to 15 years). Symptoms analyzed using a logistic regression model including covariates of family history of respiratory and allergic diseases, number of siblings, parental education, indoor fuels, passive smoking, and others.	Respiratory endpoints of chronic cough, bronchitis, wheeze and conjunctivitis symptoms were all related to the various pollutants. The colinearity of the pollutants including NO_2, SO_2, and O_3, prevented any causal separation.	PM_{10} Chronic cough OR 11.4 (2.8, 45.5) Bronchitis OR 23.2 (2.8, 45.5) Wheeze OR 1.41 (0.55, 3.58)
Zemp et al. (1999) 8 study sites in Switzerland. Pollutants: TSP, PM_{10}, SO_2, NO_2, and O_3. PM was measured with a Harvard impactor. PM_{10} ranged from 10 to 33 µg/m^3 with a mean of 21.	Logistic regression analysis of associations between prevalences of respiratory symptoms in random sample of adults and air pollution. Regressions adjusted for age, BMI, gender, parental asthma, education, and foreign citizenship.	Chronic cough and chronic phlegm and breathlessness were related to TSP, PM_{10} and NO_2.	Chronic cough, chronic phlegm and breathlessness were related to PM_{10}, and TSP.

TABLE 8B-8 (cont'd). LONG-TERM PARTICULATE MATTER EXPOSURE RESPIRATORY HEALTH INDICATORS: RESPIRATORY SYMPTOM, LUNG FUNCTION

Reference citation, location, duration, type of study, sample size, pollutants measured, summary of values	Health outcomes measured, analysis design, covariates included, analysis problems	Results and Comments Effects of co-pollutants	Effect estimates as reported by study authors. Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest effects of PM
Europe (cont'd)			
Heinrich et al. (1999) Bitterfeld, Zerbstand Hettstedt areas of former East Germany, During Sept. 1992 to July 1993 TSP ranged from 44 to 65 µg/m^3; PM_{10} measured October 1993 - March 1994 ranged from 33 to 40; and BS ranged from 26 to 42 µg/m^3. PM was measured with a Harvard impactor.	Parents of 2470 school children (5-14 yr) completed respiratory health questionnaire. Children excluded from analysis if had lived < 2 years in their current home, yielding an analysis group of 2,335 children. Outcomes studied: physician diagnosis for asthma, bronchitis, symptom, bronchial reactivity, skin prick test, specific IgE. Multiple logistic regression analyses examined regional effects.	Controlling for medical, socio-demographic, and indoor factors, children in more polluted area had circa 50% increase for bronchitic symptoms and physician-diagnosed allergies compared to control area and circa twice the respiratory symptoms (wheeze, shortness of breath and cough). Pulmonary function tests suggested slightly increased airway reactivity to cold for children in polluted area.	No single pollutant could be separated out as being responsible for poor respiratory health.
Heinrich et al. (2000) Three areas of former E. Germany Pollution measures: SO_2, TSP, and some limited PM_{10} data. TSP decreased from 65, 48, and 44 µg/m^3 to 43, 39, and 36 µg/m^3 in the three areas. PM was measured with a Harvard impactor.	Cross-sectional study of children (5-14 yr). Survey conducted twice, in 1992-1993 and 1995-1996; 2,335 children surveyed in first round, and 2,536 in second round. Only 971 children appeared in both surveys. The frequency of bronchitis, otitus media, frequent colds, febrile infections studied. Because changes measured over time in same areas, covariate adjustments not necessary.	PM and SO_2 levels both decreased in the same areas; so results are confounded.	The prevalence of all respiratory symptoms decreased significantly in all three areas over time.
Heinrich et al. (2002) Surveyed children aged 5-14 in 1992-3, 1995-6, 1998-9. Annual TSP levels ranged from 25-79 µg/m^3. Smallparticles ($NC_{0.01\text{-}2.5}$ per $10^3 cm^{-3}$) remained relatively constant.	A two-stage logistic regression model was used to analyze the data which adjusted for age, gender, educational level of parents, and indoor factors. The model included fixed area effects, random deviations, and errors from the adjustments. Parameters were estimated using GEE methods.	The study found bronchitis and frequency of colds were significantly related to TSP.	An increment of 50 µg/m^3 TSP was associated with an odds ratio for bronchitis of 3.02 (1.72-5.29) and an odds ratio of 1.90 (1.17-3.09) for frequency of colds.
Krämer et al. (1999) Six East and West Germany communities (Leipzig, Halle, Maddeburg, Altmark, Duisburg, Borken) Between 1991 and 1995 TSP levels in six communities ranged from 46 to 102 µg/m^3. Each East Germany community had decrease in TSP between 1991 and 1995. TSP was measured using a low volume sampler.	The study assessed relationship between TSP and airway disease and allergies by parental questionnaires in yearly surveys of children (5-8 yr) between February and May. The questions included pneumonia, bronchitis ever diagnosed by physician, number of colds, frequent cough, allergic symptoms. In all, 19,090 children participated. Average response was 87%. Analyses were conducted on 14,144 children for whom information on all covariates were available. Variables included gender; parent education, heating fuel, ETS. Logistic regression used to allow for time trends and SO_2 and TSP effects. Regression coefficients were converted to odds ratios.	TSP and SO_2 simultaneously included in the model. Bronchitis ever diagnosed showed a significant association. A decrease in raw percentage was seen between the start of the study and the end for bronchitis. Bronchitis seemed to be associated only with TSP in spite of huge differences in mean SO_2 levels.	Bronchitis ever diagnosed TSP per 50 µg/m^3 OR 1.63 CI (1.37 – 1.93) Halle (East) TSP µg/m^3 / % Bronchitis 1991 102 60.5 1992 73 54.7 1993 62 49.6 1994 52 50.4 1995 46 51.9

TABLE 8B-8 (cont'd). LONG-TERM PARTICULATE MATTER EXPOSURE RESPIRATORY HEALTH INDICATORS: RESPIRATORY SYMPTOM, LUNG FUNCTION

Reference citation, location, duration, type of study, sample size, pollutants measured, summary of values	Health outcomes measured, analysis design, covariates included, analysis problems	Results and Comments Effects of co-pollutants	Effect estimates as reported by study authors. Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest effects of PM
Europe (cont'd)			
Baldi et al. (1999) 24 areas of seven French towns 1974-1976 Pollutants: TSP, BS, and SO_2, NO_4 3-year average TSP-mean annual values ranging 45-243 µg/m^3. TSP was measured by the gravimetric method.	Reanalysis of Pollution Atmospheric of Affection Respiratory Chroniques (PAARC) survey data to search for relationships between mean annual air pollutant levels and prevalence of asthma in 1291 adult (25-59 yrs) and 195 children (5-9 yrs) asthmatics. Random effects logistic regression model used and included age, smoking, and education level in the final model.	Only an association between SO_2 and asthma in adults observed. No other pollutant was associated. Nor was relationship with children seen. Meteorological variables and O_3 not evaluated.	For a 50 µg/m^3 increase in TSP Adult asthma prevalence OR 1.01 CI 0.92–1.11 SO_2 Adult asthma prevalence OR 1.26 CI 1.04–1.53
Zeghnoun et al. (1999) La Havre, France during 1993 and 1996. Daily mean BS levels measured in three stations ranged 12 - 14 µg/m^3.	Respiratory drug sales for mucolytic and anticough medications (most prescribed by a physician) were evaluated versus BS, SO_2, and NO_2 levels. An autoregressive Poisson regression model permitting overdispersion control was used in the analysis.	Respiratory drug sales associated with BS, NO_2, and SO_2 levels. Both an early response (0 to 3 day lag) and a longer one (lags of 6 and 9 days) were associated.	—
Leonardi et al. (2000) 17 cities of Central Europe Yearly average concentration (Nov. 1995 - Oct. 1996) across the 17 study areas varied from 41 to 96 µg/m^3 for PM_{10}, from 29 to 67 µg/m^3 for $PM_{2.5}$, and from 12 to 38 µg/m^3 for $PM_{10\text{-}2.5}$.	Cross-sectional study collected blood and serum samples from 10-61 school children aged 9 to 11 in each community 11 April to 10 May 1996. Blood and serum samples examined for parameters in relation to PM. Final analysis group of 366 examined for peripheral lymphocyte type and total immunoglobulin classes. Association between PM and each log transformed biomarker studied by linear regression in two-stage model with adjustment for confounding factors (age, gender, number of smokers in house, laboratory, and recent respiratory illness). This survey was conducted within the frame work of the Central European study of Air Quality and Respiratory Health (CEASAR) study.	Number of lymphocytes (B, $CD4^+$, $CD8^d$, and NK) increased with increasing concentration of PM adjusted for confounders. The adjusted regression slopes are largest and statistically significant for $PM_{2.5}$ as compared to PM_{10}, but small and non statistically signif. for $PM_{10\text{-}2.5}$. Positive relationship found between concentration of IgG in serum and $PM_{2.5}$ but not for PM_{10} or $PM_{10\text{-}2.5}$. Two other models produced similar outcomes: a multi-level linear regression model and an ordinal logistic regression model.	Adjusted Regression slope $PM_{2.5}$ $CD4^+$ 80% 95% CI (34; 143) $p < 0.001$ Total IgG 24% 95% CI (2; 52) p 0.034

TABLE 8B-8 (cont'd). LONG-TERM PARTICULATE MATTER EXPOSURE RESPIRATORY HEALTH INDICATORS: RESPIRATORY SYMPTOM, LUNG FUNCTION

Reference citation, location, duration, type of study, sample size, pollutants measured, summary of values	Health outcomes measured, analysis design, covariates included, analysis problems	Results and Comments Effects of co-pollutants	Effect estimates as reported by study authors. Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest effects of PM
Europe (cont'd)			
Turnovska and Kostiranev (1999) Dimitrovgrad, Bulgaria, May 1996 Total suspended particulate matter (TSPM) mean levels were 520 ± I 61 µg/m^3 in 1986 and 187 ± 9 µg/m^3 in 1996. SO_2, H_2S, and NO_2 also measured.	Respiratory function of 97 schoolchildren (mean age 10.4 ± 0.6 yr) measured in May 1996 as a sample of 12% of all four-graders in Dimitrovgrad. The obtained results were compared with reference values for Bulgarian children aged 7 to 14 yr, calculated in the same laboratory in 1986 and published (Gherghinova et al., 1989; Kostianev et al., 1994). Variation analysis technique were used to treat the data.	Vital capacity and FEV_1 were significantly lower (mean value. = 88.54% and 82.5% respectfully) comparing values between 1986 and 1996. TSPM pollution had decreased by 2.74 times to levels still higher than Bulgarian and WHO standards.	—
Jedrychowski et al. (1999) In Krakow, Poland in 1995 and 1997 Spacial distributions for BS and SO_2 derived from network of 17 air monitoring stations. BS 52.6 µg/m ± 53.98 in high area and 33.23 ± 35.99 in low area.	Effects on lung function growth studied in preadolescent children. Lung function growth rate measured by gain in FVC and FEV_1 and occurrence of slow lung function growth (SLFG) over the 2 yr period defined as lowest quintile of the distribution of a given test in gender group. 1129 children age 9 participated in first year and 1001 in follow-up 2 years later. ATS standard questionnaire and PFT methods used. Initially univariate descriptive statistics of pulmonary function indices and SLFG were established, followed by multivariate linear regression analyses including gender, ETS, parental education, home heating system and mold. SO_2 also analyzed.	Statistically significant negative association between air pollution level and lung function growth (FVC and FEV_1) over the follow up in both gender groups. SLFG was significantly higher in the more polluted areas only among boys. In girls there was consistency in the direction of the effect, but not stat. significant. Could not separate BS and SO_2 effects on lung function growth. Excluding asthma subjects subsample (size 917) provided similar results.	<u>Boys</u> SLFG (FVC) OR = 2.15 (CI 1.25 – 3.69) SLFG (FEV_1) OR = 1.90 (CI 1.12 – 3.25) <u>Girls</u> FVC OR = 1.50 (CI 0.84 – 2.68) FEV1 OR = 1.39 (CI 0.78 – 2.44)
Jedrychowski and Flak (1998) In Kracow Poland, in 1991-1995 Daily 24 h concentration of SPM (black smoke) measured at 17 air monitoring stations. High areas had 52.6 µg/m^3 mean compared to low areas at 33.2 µg/m^3.	Respiratory health survey of 1,129 school children (aged 9 yr). Respiratory outcomes included chronic cough, chronic phlegm, wheezing, difficulty breathing and asthma. Multi-variable logistic regression used to calculate prevalence OR for symptoms adjusted for potential confounding.	The comparison of adjusted effect estimates revealed chronic phlegm as unique symptom related neither to allergy nor to indoor variable but was associated significantly with outdoor air pollution category (APL). No potential confounding variable had major effect.	It was not possible to assess separately the contribution of the different sources of air pollutants to the occurrence of respiratory symptoms. ETS and household heating (coal vs. gas vs. central heating) appeared to be of minimal importance.
Horak et al. (2002) Frischer et al. (1999) Eight communities in lower Austria between 1994-1997. PM_{10} mean summer value of 17.36 µg/m^3 and winter value of 21.03 µg/m^3.	Lung function assessed in 975 school children in grade 2-3. A several step analysis included GEE and sensitivity analyses.	Concluded that long term exposure to PM_{10} had a significant negative effect on lung function with additional evidence for a further effect for O_3 and NO_2.	After adjusting for confounders an increase in PM_{10} by 10 µg/m^3 was associated with a decrease in FEV_1 growth at 84 mL/yr and 329 mL/5 yr for MEF_{25-75}.

TABLE 8B-8 (cont'd). LONG-TERM PARTICULATE MATTER EXPOSURE RESPIRATORY HEALTH INDICATORS: RESPIRATORY SYMPTOM, LUNG FUNCTION

Reference citation, location, duration, type of study, sample size, pollutants measured, summary of values	Health outcomes measured, analysis design, covariates included, analysis problems	Results and Comments Effects of co-pollutants	Effect estimates as reported by study authors. Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest effects of PM
Europe (cont'd)			
Gehring et al. (2002) In Munich, Germany December 1997 - January 1999 Annual $PM_{2.5}$ levels determined by 40 sites and a GIS predictor for model. Mean $PM2^{-5}$ annual average of 13.4 µg/m^3 with range of 11.90 to 21.90 µg/m^3	Effect of traffic-related air pollutants. $PM_{2.5}$ and NO_2 on respiratory health outcomes wheeze, cough, bronchitis, respiratory infections, and runny nose were evaluated using multiple logistic regression analyses of l, 756 children during the first and second year of life adjusting for potential confounding factors.	There was some indication of an association between $PM_{2.5}$ and symptoms of cough but not other outcomes. In the second year of life most effects were attenuated.	—
Latin America			
Calderón-Garcidueñas et al. (2000) Southwest Metropolitan Mexico City (SWMMC) winter of 1997 and summer of 1998.	Study of 59 SWMMC children to evaluate relationship between exposure to ambient pollutants (O_3 and PM_{10}) and chest x-ray abnormalities. Fishers exact test used to determine significance in a 2x2 task between hyperinflation and exposure to SWMMC pollutant atmosphere and to control, low-pollutant city atmosphere.	Bilateral symmetric mild lung hyperinflation was significantly associated with exposure to the SWMMC air pollution mixture ($p>0.0004$). This raises concern for development of chronic disease outcome in developing lungs.	—
Australia			
Lewis et al. (1998) Summary measures of PM_{10} and SO_2 estimated for each of 10 areas in steel cities of New South Wales. PM_{10} was measured using a high volume sampler with size-selective inlets.	Cross-sectional survey of children's health and home environment between Oct 1993 and Dec 1993 evaluated frequency of respiratory symptoms (night cough, chest colds, wheeze, and diagnosed asthma). Covariates included parental education and smoking, unflued gas heating, indoor cats, age, sex, and maternal allergy. Logistic regression analysis used allowing for clustering by GEE methods.	SO_2 was not related to differences in symptom rates, but adult indoor smoking was.	Night cough OR 1.34 (1.18, 1.53) Chest colds OR 1.43 (1.12, 1.82) Wheeze OR 1.13 (0.93, 1.38)
Asia			
Wong et al. (1999b) Hong Kong, 1989 to 1991 Sulfate concentrations in respirable particles fell by 38% after implementing legislation reducing fuel sulfur levels.	3405 nonsmoking, women (mean age 36.5 yr; SD ± 3.0) in a polluted district and a less polluted district were studied for six respiratory symptoms via self-completed questionnaires. Binary latent variable modeling used.	Comparison was by district; no PM measurements reported. Results suggest control regulation may have had some (but not statistically significant) impact.	—

TABLE 8B-8 (cont'd). LONG-TERM PARTICULATE MATTER EXPOSURE RESPIRATORY HEALTH INDICATORS: RESPIRATORY SYMPTOM, LUNG FUNCTION

Reference citation, location, duration, type of study, sample size, pollutants measured, summary of values	Health outcomes measured, analysis design, covariates included, analysis problems	Results and Comments Effects of co-pollutants	Effect estimates as reported by study authors. Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest effects of PM
Asia (cont'd)			
Wang et al. (1999) Kaohsiung and Panting, Taiwan October 1995 to June 1996 TSP measured at 11 stations, PM_{10} at 16 stations. PM_{10} annual mean ranged from 19.4 to 112.81 $\mu g/m^3$ (median = 91.00 $\mu g/m^3$) TSP ranged from 112.81 to 237.82 $\mu g/m^3$ (median = 181.00). CO, NO_2, SO_2, hydrocarbons and O_3 also measured.	Relationship between asthma and air pollution examined in cross-sectional study among 165,173 high school students (11- 16 yr). Evaluated wheeze, cough and asthma diagnosed by doctor. Video determined if student displayed signs of asthma. Only 155,283 students met all requirements for study analyses and, of these, 117,080 were covered by air monitoring stations. Multiple logistic regression analysis used to determine independent effects of risk factors for asthma after adjusting for age, gender, ETS, parents education, area resident, and home incense use.	Asthma significantly related to high levels of TSP, NO_2, CO, O_3 and airborne dust. However PM_{10} and SO_2 not associated with asthma. The lifetime prevalence of asthma was 18.5% and the 1-year prevalence was 12.5%.	Adjusted OR PM_{10} 1.00 (0.96–1.05) TSP 1.29 (1.24–1.34)
Guo et al. (1999) Taiwan, October 1955 and May 1996 PM_{10} measured by beta-gauge. Also monitoring for SO_2, NO_2, O_3, CO. PM_{10} ranged from 40 to 110 $\mu g/m^3$ with a mean of 69.	Study of asthma prevalence and air pollutants. Survey for respiratory disease and symptoms in middle-school students age < 13 to ≥ 15 yr. Total of 1,018,031 (89.3%) students and their parents responded satisfactorily to the questionnaire. Schools located with 2 km of 55 monitoring sites. Logistic regression analysis conducted, controlling for age, hx eczema, parents education.	Because of close correlation among air pollutants, not possible to separate effects of individual ones. Factor analysis used to group into two classes (traffic-related and stationary fossil fuel-related). No association found between lifetime asthma prevalence and nontraffic related air pollutants (SO_2, PM_{10}).	—
Wang et al. (1999) Chongquing, China April to July 1995 Dichot samplers used to measure $PM_{2.5}$. Mean $PM_{2.5}$ level high in both urban (143 $\mu g/m^3$) and suburban (139 $\mu g/m^3$) area. SO_2 also measured	Study examined relationship between PFT and air pollution. Pulmonary function testing performed on 1,075 adults (35 - 60 yr) who had never smoked and did not use coal stoves for cooking. Generalized additive model used to estimate difference, between two areas for FEV_1, FVC, and FEV_1/FVC% with adjustment for confounding factors (gender; age, height, education, passive smoking, and occupational exposures).	Mean SO_2 concentration in the urban and suburban area highly statistically significant different (213 and 103 $\mu g/m^3$ respectfully). $PM_{2.5}$ difference was small, while levels high in both areas. Estimated effects on FEV1 statistically different between the two areas.	Difference between urban and suburban area excluding occupational exposures: $\underline{FEV_1}$ B – 119.79 SE 28.17 t – 4.25 $p < 0.01$ $\underline{FVC}$ B – 57.89 SE 30.80 t – 1.88 $p < 0.05$

TABLE 8B-8 (cont'd). LONG-TERM PARTICULATE MATTER EXPOSURE RESPIRATORY HEALTH INDICATORS: RESPIRATORY SYMPTOM, LUNG FUNCTION

Reference citation, location, duration, type of study, sample size, pollutants measured, summary of values	Health outcomes measured, analysis design, covariates included, analysis problems	Results and Comments Effects of co-pollutants	Effect estimates as reported by study authors. Negative coefficients for lung function and ORs greater than 1 for other endpoints suggest effects of PM
Asia (cont'd)			
Zhang et al. (1999) 4 areas of 3 Chinese Cities (1985 - 1988) TSP levels ranged from an annual arithmetic mean 137 µg/m^3 to 1250 µg/m^3 using gravimetric methods.	A pilot study of 4 districts of 3 Chinese cities in for the years 1985-1988, TSP levels and respiratory health outcomes studied. 4,108 adults (< 49 yrs) examined by questionnaires for couth, phlegm, wheeze, asthma, and bronchitis. Categorical logistic—regression model used to calculate odds ratio. SO_2 and NO_2 were also examined. Other potential confounding factors (age, education level, indoor ventilation, and occupation) examined in the multiple logistic regression model.	Results suggested that the OR's for cough, phlegm, persistent cough and phlegm and wheeze increased as outdoor TSP concentrations did. .	Wheeze produced largest OR for both mothers and fathers in all locations.
Qian et al. (2000) 4 China cities The 4 year average TSP means were 191, 296, 406, and 1067 µg/m^3. SO_2 and NO_2 measurements were also available. TSP was measured gavimetrically.	Pilot cross-sectional survey of 2789 elementary school children in four Chinese communities chosen for their PM gradient. Frequency of respiratory symptoms (cough, phlegm, wheeze, and diagnosed asthma, bronchitis, or pneumonia) assessed by questionnaire. Covariates included parental occupation, education and smoking. The analysis used logistic regression, controlling for age, sex, parental smoking, use of coal in home, and home ventilation.	Results not directly related to pollution levels, but symptom rates were highest in highest pollution area for cough, phlegm, hospitalization for respiratory disease, bronchitis, and pneumonia. No gradient correlating with pollution levels found for the three lower exposure communities.	—