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Air Quality Criteria for Lead

Volume II of II

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Air Quality Criteria for Lead

Volume II

National Center for Environmental Assessment-RTP Division
Office of Research and Development
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Research Triangle Park, NC

PREFACE

National Ambient Air Quality Standards (NAAQS) are promulgated by the United States Environmental Protection Agency (EPA) to meet requirements set forth in Sections 108 and 109 of the U.S. Clean Air Act. Those two Clean Air Act sections require the EPA Administrator (1) to list widespread air pollutants that reasonably may be expected to endanger public health or welfare; (2) to issue air quality criteria for them that assess the latest available scientific information on nature and effects of ambient exposure to them; (3) to set "primary" NAAQS to protect human health with adequate margin of safety and to set "secondary" NAAQS to protect against welfare effects (e.g., effects on vegetation, ecosystems, visibility, climate, manmade materials, etc); and (5) to periodically review and revise, as appropriate, the criteria and NAAQS for a given listed pollutant or class of pollutants.

Lead was first listed in the mid-1970's as a "criteria air pollutant" requiring NAAQS regulation. The scientific information pertinent to Pb NAAQS development available at the time was assessed in the EPA document *Air Quality Criteria for Lead*; published in 1977. Based on the scientific assessments contained in that 1977 lead air quality criteria document (1977 Lead AQCD), EPA established a 1.5 $\mu g/m^3$ (maximum quarterly calendar average) Pb NAAQS in 1978.

To meet Clean Air Act requirements noted above for periodic review of criteria and NAAQS, new scientific information published since the 1977 Lead AQCD was later assessed in a revised Lead AQCD and Addendum published in 1986 and in a Supplement to the 1986 AQCD/Addendum published by EPA in 1990. A 1990 Lead Staff Paper, prepared by EPA's Office of Air Quality Planning and Standards (OPQPS), drew upon key findings and conclusions from the 1986 Lead AQCD/Addendum and 1990 Supplement (as well as other OAQPS-sponsored lead exposure/risk analyses) in posing options for the EPA Administrator to consider with regard to possible revision of the Pb NAAQS. However, EPA chose not to revise the Pb NAAQS at that time. Rather, as part of implementing a broad 1991 U.S. EPA Strategy for Reducing Lead Exposure, the Agency focused primarily on regulatory and remedial clean-up efforts to reduce Pb exposure from a variety of non-air sources that posed more extensive public health risks, as well as other actions to reduce air emissions.

The purpose of this revised Lead AQCD is to critically assess the latest scientific information that has become available since the literature assessed in the 1986 Lead

AQCD/Addendum and 1990 Supplement, with the main focus being on pertinent new information useful in evaluating health and environmental effects of ambient air lead exposures. This includes discussion in this document of information regarding: the nature, sources, distribution, measurement, and concentrations of lead in the environment; multimedia lead exposure (via air, food, water, etc.) and biokinetic modeling of contributions of such exposures to concentrations of lead in brain, kidney, and other tissues (e.g., blood and bone concentrations, as key indices of lead exposure).; characterization of lead health effects and associated exposure-response relationships; and delineation of environmental (ecological) effects of lead. This final version of the revised Lead AQCD mainly assesses pertinent literature published or accepted for publication through December 2005.

The First External Review Draft (dated December 2005) of the revised Lead AQCD underwent public comment and was reviewed by the Clean Air Scientific Advisory Committee (CASAC) at a public meeting held in Durham, NC on February 28-March 1, 2006. The public comments and CASAC recommendations received were taken into account in making appropriate revisions and incorporating them into a Second External Review Draft (dated May, 2006) which was released for further public comment and CASAC review at a public meeting held June 28-29, 2006. In addition, still further revised drafts of the Integrative Synthesis chapter and the Executive Summary were then issued and discussed during an August 15, 2006 CASAC teleconference call. Public comments and CASAC advice received on these latter materials, as well as Second External Review Draft materials, were taken into account in making and incorporating further revisions into this final version of this Lead AQCD, which is being issued to meet an October 1, 2006 court-ordered deadline. Evaluations contained in the present document provide inputs to an associated Lead Staff Paper prepared by EPA's Office of Air Quality Planning and Standards (OAQPS), which poses options for consideration by the EPA Administrator with regard to proposal and, ultimately, promulgation of decisions on potential retention or revision, as appropriate, of the current Pb NAAQS.

Preparation of this document has been coordinated by staff of EPA's National Center for Environmental Assessment in Research Triangle Park (NCEA-RTP). NCEA-RTP scientific staff, together with experts from academia, contributed to writing of document chapters. Earlier drafts of document materials were reviewed by scientists from other EPA units and by non-EPA experts in several public peer consultation workshops held by EPA in July/August 2005.

NCEA acknowledges the valuable contributions provided by authors, contributors, and reviewers and the diligence of its staff and contractors in the preparation of this document. The constructive comments provided by public commenters and CASAC that served as valuable inputs contributing to improved scientific and editorial quality of the document are also acknowledged by NCEA.

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Abbreviations and Acronyms

αFGF	α-fibroblast growth factor
AA	arachidonic acid
AAL	active avoidance learning
AAS	atomic absorption spectroscopy
ABA	β-aminoisobutyric acid
ACBP	Achenbach Child Behavior Profile
ACE	angiotensin converting enzyme
ACh	acetylcholine
AChE	acetylcholinesterase
ACR	acute-chronic ratio
AD	adult
ADC	analog digital converter
ADP	adenosine diphosphate
AE	anion exchange
AEA	*N*-arachidonylethanolamine
AFC	antibody forming cells
2-AG	2-arachidonylglycerol
A horizon	uppermost layer of soil (litter and humus)
AHR	aryl hydrocarbon receptor
AI	angiotensin I
ALA	δ-aminolevulinic acid
ALAD	δ-aminolevulinic acid dehydratase
ALAS	aminolevulinic acid synthetase
ALAU	urinary δ-aminolevulinic acid
ALD	aldosterone
ALS	amyotrophic lateral sclerosis
ALT	alanine aminotransferase
ALWT	albumin weight
AMEM	Alpha Minimal Essential Medium
AMP	adenosine monophosphate
ANCOVA	analysis of covariance
ANF	atrial natriuretic factor
Ang II	angiotensin II
ANOVA	analysis of variance

ANP	atrial natriuretic peptide
AP	alkaline phosphatase
AP-1	activated protein-1
ApoE	apolipoprotein E
AQCD	Air Quality Criteria Document
Arg	arginine
AS52	cells derived from the CHO cell line
ASGP-R	aceyl glycoprotein receptor
AST	aspartate aminotransferase
ASV	anode stripping voltammetry
3-AT	3-aminotriazole; 3-amino triazide
ATP	adenosine triphosphate
ATP1A2	sodium-potassium adenosine triphosphase α2
ATPase	adenosine triphosphatase
ATSDR	Agency for Toxic Substances and Disease Research
AVCD	atrioventricular conduction deficit
AVS	acid volatile sulfide
AWQC	ambient water quality criteria
β	beta-coefficient; slope of an equation
βFGF	β-fibroblast growth factor
17β–HS	17β-hydroxysteriod
3β-HSD	3β-hydroxysteriod dehydrogenase
17β-HSDH	17β-hydroxysteriod dehydrogenase
6β-OH-cortisol	6-β-hydroxycortisol
B	both
BAEP	brainstem auditory-evoked potentials
BAER	brainstem auditory-evoked responses
BAF	bioaccumulation factor
B cell	B lymphocyte
BCFs	bioconcentration factors
BCS	bovine calf serum
BDNF	brain derived neurotrophic factor
BDWT	body weight changes
BEI	biological exposure index
BFU-E	blood erythroid progenitor

BLL	blood lead level
BLM	biotic ligand model
BM	basement membrane
BMI	body mass index
BDNF	brain-derived neurotrophic factor
BOTMP	Bruinicks-Oseretsky Test of Motor Proficiency
BP	blood pressure
BPb	blood lead concentration
BSA	bovine serum albumin
BSI	Brief Symptom Inventory
BTQ	Boston Teacher Questionnaire
BUN	blood urea nitrogen
bw, b. wt., BW	body weight
C3H10T/12	mouse embryo cell line
C3, C4	complement proteins
CA	chromosome aberration
CA3	cornu ammonis 3 region of hippocampus
^{45}Ca	calcium-45 radionuclide
Ca-ATP	calcium-dependent adenosine triphosphate
Ca-ATPase	calcium-dependent adenosine triphosphatase
$CaCO_3$	calcium carbonate
CaEDTA	calcium disodium ethylenediaminetetraacetic acid
CAL	calcitonin
CaM	calmodulin
Ca-Mg-ATPase	calcium-magnesium-dependent adenosine triphosphatase
cAMP	cyclic adenosinemonophosphate
$CaNa_2$ EDTA	calcium disodium ethylenediaminetetraacetic acid
CANTAB	Cambridge Neuropsychological Testing Automated Battery
CAT	catalase; Cognitive Abilities Test
CBCL	Achenbach Child Behavior Checklist
CBCL-T	Total Behavior Problem Score
CBL	cumulative blood lead
CBLI	cumulative blood lead index
CCB	cytochalasin B
CCD	charge-coupled device

CCE	Coordination Center for Effects
CCL	carbon tetrachloride
CCS	cosmic calf serum
C-CV_{RSA}	coefficient of component variance of respiratory sinus arrhythmia
Cd	cadmium
^{109}Cd	cadmium-109 radionuclide
CdU	urinary cadmium
CEC	cation exchange capacity
CESD, CES-D	Center for Epidemiologic Studies Depression (scale)
GFAP	glial fibrillary acidic protein
CFU-E	colony forming unit blood-erythroid progenitor (cell count)
CFU-GEMM	colony forming unit blood-pluripotent progenitor (cell count)
CFU-GM	blood granulocyte/macrophage progenitor (cell count)
cGMP	cyclic guanosine-3',5'-monophosphate
ChAT	choline acetyltransferase
CHD	coronary heart disease
CHO	Chinese hamster ovary cell line
CI	confidence interval
CLE-SV	competitive ligand-exchange/stripping voltammetry
CLRTAP	Convention on Long-Range Transboundary of Air Pollution
CLS	Cincinnati Lead Study
CMC	criterion maximum concentration
CMI	cell-mediated immunity
CNS	central nervous system
COH	cation-osmotic hemolysis
ConA	concanavalin A
COR	cortisol
CoTx	cotreatment
COX-2	cyclooxygenase-2
CP	coproporphryn
CPT	current perception threshold
cr	creatinine
CRAC	calcium release activated calcium reflux
CREB	cyclic AMP-response element binding protein
CRF	chronic renal failure

CRI	chronic renal insufficiency
CSF	cerebrospinal fluid
CuZn-SOD	copper and zinc-dependent superoxide dismutase
CV	conduction velocity
CVLT	California Verbal Learning Test
CV_{R-R}	coefficient of variation of the R-R interval
CYP	cytochrome (e.g., CYP1A, CYP-2A6, CYP3A4, CYP450)
CYP3a11	cytochrome P450 3a11
D	D-statistic
DA	dopamine; dopaminergic
dbcAMP	dibutyryl cyclic adenosine-3',5'-monophosphate
DCV	distribution of conduction velocities
DEAE	diethylaminoethyl (chromatography)
DET	diffusive equilibrium thin films
DEYO	death of young
DFS	decayed or filled surfaces, permanent teeth
dfs	covariate-adjusted number of caries
DG	dentate gyrus
DGT	diffusive gradient thin films
DL	DL-statistic
DMEM	Dulbecco's Minimal Essential Medium
DMEM/F12	Dulbecco's Minimal Essential Medium/Ham's F12
DMFS	decayed, missing, or filled surfaces, permanent teeth
DMPS	2,3-dimercaptopropane 1-sulfonate
DMSA	2,3-dimercaptosuccinic acid
DMT	Donnan membrane technique
DMTU	dimethylthiourea
DNA	deoxyribonucleic acid
DO	distraction osteogenesis
DOC	dissolved organic carbon
DOM	dissolved organic carbon
DOPAc	3,4-dihydroxyphenylacetic acid
DPASV	differential pulse anodic stripping voltammetry
dp/dt	rate of left ventricular isovolumetric pressure
DPPD	*N*-*N*-diphenyl-*p*-phynylene-diamine

DR	drinking water
DSA	delayed spatial alternation
DTC	diethyl dithiocarbomate complex
DTH	delayed type hypersensitivity
DTPA	diethylenetriaminepentaacetic acid
DTT	dithiothreitol
dw	dry weight
E	embryonic day
E_2	estradiol
EBE	early biological effect
EBV	Epstein-Barr virus
EC	European Community
EC_{50}	effect concentration for 50% of test population
eCB	endocannabinoid
ECG	electrocardiogram
Eco-SSL	ecological soil screening level
EDS	energy dispersive spectrometers
EDTA	ethylenediaminetetraacetic acid
EEDQ	*N*-ethoxycarbonyl-2-ethoxy-1,2-dihydroquinone
EEG	electroencephalogram
EG	egg
EGF	epidermal growth factor
EGG	effects on eggs
EGPN	egg production
EKG	electrocardiogram
electro	electrophysiological stimulation
EM/CM	experimental medium-to-control medium (ratio)
EMEM	Eagle's Minimal Essential Medium
eNOS	endothelial nitric oxide synthase
EP	erythrocyte protoporphyrin
EPA	U.S. Environmental Protection Agency
Epi	epinephrine
EPMA	electron probe microanalysis
EPO	erythropoietin
EPSC	excitatory postsynaptic currents

EPT	macroinvertebrates from the Ephemeroptera (mayflies), Plecoptera (stoneflies), and Trichoptera (caddisflies) group
ERG	electroretinogram; electroretinographic
ERL	effects range – low
ERM	effects range – median
EROD	ethoxyresorufin-*O*-deethylase
ESCA	electron spectroscopy for chemical analysis
ESRD	end-stage renal disease
EST	estradiol
ESTH	eggshell thinning
ET	endothelein; essential tremor
ETOH	ethyl alcohol
EXAFS	extended X-ray absorption fine structure
EXANES	extended X-ray absorption near edge spectroscopy
F	F-statistic
F344	Fischer 344 (rat)
FAV	final acute value
FBS	fetal bovine serum
FCS	fetal calf serum
FCV	final chronic value
FD	food
FEF	forced expiratory flow
FEP	free erythrocyte protoporphyrin
FERT	fertility
FEV_1	forced expiratory volume in one second
FGF	fibroblast growth factor (e.g., βFGF, αFGF)
FI	fixed interval (operant conditioning)
FIAM	free ion activity model
FMLP	*N*-formyl-L-methionyl-L-leucyl-L-phenylalanine
fMRI	functional magnetic resonance imaging
FR	fixed-ratio operant conditioning
FSH	follicle stimulating hormone
FT3	free triiodothyronine
FT4	free thyroxine
FTES	free testosterone

FTII	Fagan Test of Infant Intelligence
FTPLM	flow-through permeation liquid membranes
FURA-2	1-[6-amino-2-(5-carboxy-2-oxazolyl)-5-benzofuranyloxy]-2-(2-amino-5-methylphenoxy) ethane-*N*,*N*,*N*′,*N*′-tetraacetic acid
FVC	forced vital capacity
γ-GT	γ-glutamyl transferase
G	gestational day
GABA	gamma aminobutyric acid
GAG	glycosaminoglycan
G12 CHV79	cells derived from the V79 cell line
GCI	General Cognitive Index
GD	gestational day
GDP	guanosine diphosphate
GEE	generalized estimating equations
GFAAS	graphite furnace atomic absorption spectroscopy
GFR	glomerular filtration rate
GGT	γ-glutamyl transferase
GH	growth hormone
GI	gastrointestinal
GIME-VIP	gel integrated microelectrodes combined with voltammetric in situ profiling
GIS	geographic information system
GLU	glutamate
GMAV	genus mean acute value
GMCV	genus mean chronic value
GMP	guanosine monophosphate
GMPH	general morphology
GnRH	gonadotropin releasing hormone
GOT	aspartate aminotransferase
GP	gross productivity
G6PD, G6PDH	glucose-6-phosphate dehydrogenase
GPEI	glutathione *S*-transferase P enhancer element
$gp91^{phox}$	NAD(P)H oxidase
GPT	glutamic-pyruvic transaminase
GPx	glutathione peroxidase
GRO	growth

GRP78	glucose-regulated protein 78
GSD	geometric standard deviation
GSH	reduced glutathione
GSIM	gill surface interaction model
GSSG	glutathione disulfide
GST	glutathione-*S*-transferase
GSTP	placental glutathione transferase
GTP	guanosine triphosphate
GV	gavage
H^+	acidity
^3H	hydrogen-3 radionuclide (tritium)
HA	humic acid; hydroxyapatite
Hb	hemoglobin
HBEF	Hubbard Brook Experimenatl Forest
HBSS	Hank's Balanced Salt Solution
HCG; hCG	human chorionic gonadotropin
Hct	hematocrit
HDL	high-density lipoprotein (cholesterol)
HEP	habitat evaluation procedure
HET	Binghamton heterogeneous stock
HFPLM	hollow fiber permeation liquid membranes
Hgb	hemoglobin
HGF	hepatocyte growth factor
HH	hydroxylamine hydrochloride
H-H	high-high
HHANES	Hispanic Health and Nutrition Examination Survey
H-L	high-low
HLA	human leukocyte antigen
H-MEM	minimum essential medium/nutrient mixture–F12-Ham
HMP	hexose monophosphate shunt pathway
HNO_3	nitric acid
H_2O_2	hydrogen peroxide
HOME	Home Observation for Measurement of Environment
HOS TE	human osteosarcoma cells
HPLC	high-pressure liquid chromatography

H_3PO_4	phosphoric acid
HPRT	hypoxanthine phosphoribosyltransferase (gene)
HR	heart rate
HSI	habitat suitability indices
H_2SO_4	sulfuric acid
HSPG	heparan sulfate proteoglycan
Ht	hematocrit
HTC	hepatoma cells
hTERT	catalytic subunit of human telomerase
HTN	hypertension
IBL	integrated blood lead index
IBL × WRAT-R	integrated blood lead index × Wide Range Achievement Test-Revised (interaction)
ICD	International Classification of Diseases
ICP	inductively coupled plasma
ICP-AES	inductively coupled plasma atomic emission spectroscopy
ICP-MS, ICPMS	inductively coupled plasma mass spectrometry
ID-MS	isotope dilution mass spectrometry
IFN	interferon (e.g., IFN-γ)
Ig	immunoglobulin (e.g., IgA, IgE, IgG, IgM)
IGF-1	insulin-like growth factor 1
IL	interleukin (e.g., IL-1, IL-1β, IL-4, IL-6, IL-12)
ILL	incipient lethal level
immuno	immunohistochemical staining
IMP	inosine monophosphate
iNOS	inducible nitric oxide synthase
i.p., IP	intraperitoneal
IPSC	inhibitory postsynaptic currents
IQ	intelligence quotient
IRT	interresponse time
ISEL	in situ end labeling
ISI	interstimulus interval
i.v., IV	intravenous
IVCD	intraventricular conduction deficit
JV	juvenile

KABC	Kaufman Assessment Battery for Children
KTEA	Kaufman Test of Educational Achievement
KXRF, K-XRF	K-shell X-ray fluorescence
LA	lipoic acid
LB	laying bird
LC	lactation
LC_{50}	lethal concentration at which 50% of exposed animals die
LC_{74}	lethal concentration at which 74% of exposed animals die
LD_{50}	lethal dose at which 50% of exposed animals die
LDH	lactate dehydrogenase
LDL	low-density lipoprotein (cholesterol)
L-dopa	3,4-dihydroxyphenylalanine (precursor of dopamine)
LE	Long Evans (rat)
LET	linear energy transfer (radiation)
LH	luteinizing hormone
LHRH	luteinizing hormone releasing hormone
LN	lead nitrate
L-NAME	L-N^G-nitroarginine methyl ester
LOAEL	lowest-observed adverse effect level
LOEC	lowest-observed-effect concentration
LOWESS	locally weighted scatter plot smoother
LPO	lipoperoxide
LPP	lipid peroxidation potential
LPS	lipopolysaccharide
LT	leukotriene
LT_{50}	time to kill 50%
LTER	Long-Term Ecological Research (sites)
LTP	long term potentiation
LVH	left ventricular hypertrophy
µPIXE	microfocused particle induced X-ray emission
µSXRF	microfocused synchrotron-based X-ray fluorescence
MA	mature
MA-10	mouse Leydig tumor cell line
MANCOVA	multivariate analysis of covariance
MAO	monoamine oxidase

MATC	maximum acceptable threshold concentration
MDA	malondialdehyde
MDA-TBA	malondialdehyde-thiobarbituric acid
MDCK	kidney epithelial cell line
MDI	Mental Development Index (score)
MDRD	Modification of Diet in Renal Disease (study)
MEM	Minimal Essential Medium
MG	microglobulin
Mg-ATPase	magnesium-dependent adenosine triphosphatase
MiADMSA	monoisamyl dimercaptosuccinic acid
Mi-DMSA	mi monoisoamyl dimercaptosuccinic acid
MK-801	NMDA receptor antagonist
MLR	mixed lymphocyte response
MMSE	Mini-Mental State Examination
MMTV	murine mammary tumor virus
MN	micronuclei formation
MND	motor neuron disease
MNNG	*N*-methyl-*N*′-nitro-*N*-nitrosoguanidine
MPH	morphology
MRI	magnetic resonance imaging
mRNA	messenger ribonucleic acid
MROD	methoxyresorufin-*O*-demethylase
MRS	magnetic resonance spectroscopy
MS	mass spectrometry
MSCA	McCarthy Scales of Children's Abiltities
mSQGQs	mean sediment quality guideline quotients
MT	metallothionein
MVV	maximum voluntary ventilation
MW	molecular weight (e.g., high-MW, low-MW)
N, n	number of observations
N/A	not available
NAAQS	National Ambient Air Quality Standards
NAC	*N*-acetyl cysteine
NAD	nicotinamide adenine dinucleotide
NADH	reduced nicotinamide adenine dinucleotide

NADP	nicotinamide adenine dinucleotide phosphate
NAD(P)H, NADPH	reduced nicotinamide adenine dinucleotide phosphate
NADS	nicotinamide adenine dinucleotide synthase
NAF	nafenopin
NAG	*N*-acetyl-β-D-glucosaminidase
Na-K-ATPase	sodium-potassium-dependent adenosine triphosphatase
NAWQA	National Water-Quality Assessment
NBT	nitro blue tetrazolium
NCBP	National Contaminant Biomonitoring Program
NCD	nuclear chromatin decondensation (rate)
NCS	newborn calf serum
NCTB	Neurobehavioral Core Test Battery
NCV	nerve conduction velocity
ND	non-detectable; not detected
NDI	nuclear divison index
NE	norepinephrine
NES	Neurobehavioral Evaluation System
NF-κB	nuclear transcription factor-κB
NGF	nerve growth factor
NHANES	National Health and Nutrition Examination Survey
NIOSH	National Institute for Occupational Safety and Health
NIST	National Institute for Standards and Technology
NK	natural killer
NMDA	*N*-methyl-D-aspartate
NMDAR	*N*-methyl-D-aspartate receptor
NMR	nuclear magnetic resonance
NO	nitric oxide
NO_2	nitrogen dioxide
NO_3	nitrate
NOAEC	no-observed-adverse-effect concentration
NOAEL	no-observed-adverse-effect level
NOEC	no-observed-effect concentration
NOEL	no-observed-effect level
NOM	natural organic matter
NORs	nucleolar organizing regions

NOS	nitric oxide synthase; not otherwise specified
NO_x	nitrogen oxides
NP	net productivity
NPSH	nonprotein sulfhydryl
NR	not reported
NRC	National Research Council
NRK	normal rat kidney
NS	nonsignificant
NSAID	non-steroidal anti-inflammatory agent
NT	neurotrophin
NTA	nitrilotriacetic acid
O_2	oxygen
ODVP	offspring development
OH	hydroxyl
7-OH-coumarin	7-hydroxy-coumarin
1,25-$(OH)_2$-D, 1,25-$(OH)_2$ D_3	1,25-dihydroxyvitamin D
24,25-$(OH)_2$-D_3	24,25-dihydroxyvitamin D
25-OH-D_3	25-hydroxyvitamin D
8-OHdG	8-hydroxy-2'-deoxyguanosine
O horizon	forest floor
OR	odds ratio; other oral
OSWER	Office of Solid Waste and Emergency Response
P, p	probability value
P300	event-related potential
P450 1A1	cytochrome P450 1A1
P450 1A2	cytochrome P450 1A2
P450 CYP3a11	cytochrome P450 3a11
PAD	peripheral arterial disease
PAH	polycyclic aromatic hydrocarbon
PAI-1	plasminogen activator inhibitor-1
PAR	population attributable risk
Pb	lead
^{203}Pb	lead-203 radionuclide
^{204}Pb, ^{206}Pb, ^{207}Pb, ^{208}Pb	stable isotopes of lead-204, -206, -207, -208, respectively
^{210}Pb	lead-210 radionuclide

$Pb(Ac)_2$	lead acetate
PbB	blood lead concentration
$PbCl_2$	lead chloride
$Pb(ClO_4)_2$	lead chlorate
PBG-S	porphobilinogen synthase
PBMC	peripheral blood mononuclear cells
$Pb(NO_3)_2$	lead nitrate
PbO	lead oxides (or litharge)
PBP	progressive bulbar paresis
PbS	galena
PbU	urinary lead
PC12	pheochromocytoma cell
PCR	polymerase chain reaction
PCV	packed cell volume
PDE	phosphodiesterase
PDGF	platelet-derived growth factor
PDI	Psychomotor Development Index
PEC	probable effect concentration
PEF	expiratory peak flow
PG	prostaglandin (e.g., PGE_2, PGF_2); prostate gland
PHA	phytohemagglutinin A
Pi	inorganic phosphate
PIXE	particle induced X-ray emission
PKC	protein kinase C
pl NEpi	plasma norepinephrine
PMA	progressive muscular atrophy
PMN	polymorphonuclear leucocyte
PMR	proportionate mortality ratio
PN	postnatal (day)
P5N	pyrimidine 5'-nucleotidase
PND	postnatal day
p.o., PO	per os (oral administration)
POMS	Profile of Mood States
ppb	parts per billion
ppm	parts per million

PPVT-R	Peabody Picture Vocabulary Test-Revised
PRA	plasma renin activity
PRL	prolactin
PROG	progeny counts or numbers
PRR	prevalence rate ratio
PRWT	progeny weight
PST	percent transferrin saturation
PTH	parathyroid hormone
PTHrP	parathyroid hormone-related protein
PVC	polyvinyl chloride
PWM	pokeweed mitogen
PRWT	progeny weight
QA/QC	quality assurance/quality control
Q/V	flux of air (Q) divided by volume of culture (V)
r	Pearson correlation coefficient
R^2	multiple correlation coefficient
r^2	correlation coefficient
^{226}Ra	most stable isotope of radium
R/ALAD	ratio of ALAD activity before and after reactivation
RAVLT	Rey Auditory Verbal Learning Test
^{86}Rb	rubidium-86 radionuclide
RBA	relative bioavailablity
RBC	red blood cell; erythrocyte
RBF	renal blood flow
RBP	retinol binding protein
RBPH	reproductive behavior
RCPM	Ravens Colored Progressive Matrices
REL	rat epithelial (cells)
REP	reproduction
RHIS	reproductive organ histology
^{222}Rn	most stable isotope of radon
RNA	ribonucleic acid
ROS	reactive oxygen species
ROS 17.2.8	rat osteosarcoma cell line
RPMI 1640	Roswell Park Memorial Institute basic cell culture medium

RR	relative risk; rate ratio
RT	reaction time
RSEM	resorbed embryos
RSUC	reproductive success (general)
RT	reproductive tissue
∑SEM	sum of the molar concentrations of simultaneously extracted metal
SA7	simian adenovirus
SAB	Science Advisory Board
SAM	*S*-adenosyl-L-methionine
SBIS-4	Stanford-Binet Intelligence Scale-4th edition
s.c., SC	subcutaneous
SCAN	Test for Auditory Processing Disorders
SCE	selective chemical extraction; sister chromatid exchange
SCP	stripping chronopotentiometry
SD	Spraque-Dawley (rat); standard deviation
SDH	succinic acid dehydrogenase
SDS	sodium dodecyl sulfate; Symbol Digit Substitution
SE	standard error; standard estimation
SEM	standard error of the mean
SES	socioeconomic status
sGC	soluble guanylate cyclase
SH	sulfhydryl
SHBG	sex hormone binding globulin
SHE	Syrian hamster embryo cell line
SIMS	secondary ion mass spectrometry
SIR	standardized incidence ratio
SLP	synthetic leaching procedure
SM	sexually mature
SMAV	species mean acute value
SMR	standardized mortality ratio
SNAP	Schneider Neonatal Assessment for Primates
SNP	sodium nitroprusside
SO_2	sulfur dioxide
SOD	superoxide dismutase

SOPR	sperm-oocyte penetration rate
SPCL	sperm cell counts
SPCV	sperm cell viability
SQGs	sediment quality guidelines
SRA	Self Reported Antisocial Behavior scale
SRD	Self Report of Delinquent Behavior
SRIF	somatostatin
SRM	Standard Reference Material
SRT	simple reaction time
SSADMF	Social Security Administration Death Master File
SSB	single-strand breaks
SSEP	somatosensory-evoked potential
StAR	steroidogenic acute regulatory protein
STORET	STOrage and RETrieval
SVC	sensory conduction velocity
SVRT	simple visual reaction time
T	testosterone
TA	tail
TABL	time-averaged blood lead
T&E	threatened and endangered (species)
TAT	tyrosine aminotransferase
TB	tibia
TBARS	thiobarbituric acid-reactive species
TBPS	Total Behavior Problem Score
TCDD	methionine-choline-deficient diet
T cell	T lymphocyte
TCLP	toxic characteristic leaching procedure
TE	testes
TEC	threshold effect concentration
TEDG	testes degeneration
TEL	tetraethyl lead
TES	testosterone
TEWT	testes weight
TF	transferrin, translocation factor
TG	6-thioguanine

TGF	transforming growth factor
TH	tyrosine hydroxylase
^{232}Th	stable isotope of thorium-232
TLC	Treatment of Lead-exposed Children (study)
TNF	tumor necrosis factor (e.g., TNF-α)
TOF	time-of-flight
tPA	plasminogen activator
TPRD	total production
TRH	thyroid releasing hormone
TRV	toxicity reference value
TSH	thyroid stimulating hormone
TSP	triple-super phosphate
TT3	total triiodothyronine
TT4	serum total thyroxine
TTES	total testosterone
TTR	transthyretin
TU	toxic unit
TWA	time-weighted average
TX	tromboxane (e.g., TXB_2)
U	uriniary
^{235}U, ^{238}U	uranium-234 and -238 radionuclides
UCP	urinary coproporphyrin
UDP	uridine diphosphate
UNECE	United Nations Economic Commission for Europe
Ur	urinary
USFWS	U.S. Fish and Wildlife Service
USGS	United States Geological Survey
UV	ultraviolet
V79	Chinese hamster lung cell line
VA	Veterans Administration
VC	vital capacity; vitamin C
VDR	vitamin D receptor
VE	vitamin E
VEP	visual-evoked potential
VI	variable-interval

vit C	vitamin C
vit E	vitamin E
VMA	vanilmandelic acid
VMI	Visual-Motor Integration
VSM	vascular smooth muscle (cells)
VSMC	vascular smooth muscle cells
WAIS	Wechsler Adult Intelligence Scale
WDS	wavelength dispersive spectrometers
WHO	World Health Organization
WISC	Wechsler Intelligence Scale for Children
WISC-R	Wechsler Intelligence Scale for Children-Revised
WO	whole organism
WRAT-R	Wide Range Achievement Test-Revised
WT	wild type
WTHBF-6	human liver cell line
ww	wet weight
XAFS	X-ray absorption fine structure
XANES	X-ray absorption near edge spectroscopy
XAS	X-ray absorption spectroscopy
XPS	X-ray photoelectron spectroscopy
X-rays	synchrotron radiation
XRD	X-ray diffraction
XRF	X-ray fluorescence
ZAF	correction in reference to three components of matrix effects: atomic number (Z), absorption (A), and fluorescence (F)
$ZnNa_2$ DTPA	zinc disodium diethylenetriaminepentaacetic acid
$ZnNa_2$ EDTA	zinc disodium ethylenediaminetetraacetic acid
ZPP	zinc protoporphyrin

AX4. CHAPTER 4 ANNEX

ANNEX TABLES AX4

Table AX4-1. Analytical Methods for Determining Lead in Blood, Urine, and Hair

Sample Matrix	Preparation Method	Analytical Method	Sample Detection Limit	Accuracy (percent recovery)	Reference
Blood	Wet ashing with acid mixtures; residue dissolution in dilute $HClO_4$	ASV with mercury-graphite electrode (NIOSH Method 195)	40 µg/L	95–105	NIOSH (1977b)
Blood	Wet ashing with HNO_3; residue dissolution in dilute HNO_3	GFAAS (NIOSH Method 214)	100 µg/L	No data	NIOSH (1977e)
Blood	Dilution with Triton X-100®; addition of nitric acid and diammonium phosphate	GFAAS	2.4 µg/L	93–105	Aguilera de Benzo et al. (1989)
Blood	Dilution of sample with ammonium solution containing Triton X-100	ICP/MS	15 µg/L	96–111	Delves and Campbell (1988)
Blood	Dilution of sample in 0.2% Triton X-100 and water	GFAAS	≈15 µg/L	97–150	Que Hee et al. (1985)
Blood	Wet ashing, dilution	ICP-MS	0.1 ppb	94–100	Zhang et al. (1997)
		GFAAS	4 ppb	90–108	
Blood and urine	Mixing of urine sample with HNO_3; filtration, chelation of lead in whole blood or filtered urine with APDC, extraction with MIBK	AAS (NIOSH Method 8003)	0.05 µg/g (blood); 50 µg/L (urine)	99 (±10.8%)	NIOSH (1994)
Blood and urine	Wet ashing of sample with HNO_3, complexation with diphenylthio-carbazone, and extraction with chloroform	Spectrophotometry (NIOSH Method 102)	30 µg/L (blood); 12 µg/L (urine)	97 97	NIOSH (1977a)
Blood and urine	^{206}Pb addition and sample acid digestion; lead coprecipitation by addition of $Ba(NO_3)_2$, followed by electrodeposition on platinum wire	IDMS	No data	98–99	Manton and Cook (1984)
Blood and tissue	Digestion of sample with $HNO_3/HClO_4/H_2SO_4$; heat	ICP-AES (Method 8005)	0.01 µg/g (blood); 0.2 µg/g (tissue)	113	NIOSH (1984)

Table AX4-1 (cont'd). Analytical Methods for Determining Lead in Blood, Urine, and Hair

Sample Matrix	Preparation Method	Analytical Method	Sample Detection Limit	Accuracy (percent recovery)	Reference
Blood	Addition of 50 µL of blood into reagent, mixing, and transferring to sensor strip (commercial test kit)	Gold electrode sensor	1.4 µg/dL	No data	ESA (1998)
Urine	Collect 50 mL urine sample and add 5 mL concentrated HNO_3 as preservative; filter samples through cellulose membrane, adjust pH to 8, ash filters and resins in low temperature oxygen plasma for 6 hours	ICP-AES (Method 8310)	5 µg/L	100	NIOSH (1994)
Serum, blood, and urine	Filtration of sample if needed; blood requires digestion in a Parr bomb; dilution of serum or urine with acid or water	ICP-AES	10–50 µg/L	85 (serum) >80 (urine, blood)	Que Hee and Boyle (1988)
Urine	Wet ashing of sample with acid mixture and dissolution in dilute $HClO_4$	ASV with mercury-graphite electrode (Method 200)	4 µg/L	90–110	NIOSH (1977c)
Hair	Cleaning of sample with acetone/ methanol; digestion with acid mixture and heat; diammonium phosphate addition as matrix modifier	GFAAS	0.16 µg/g	99	Wilhelm et al. (1989)
Hair	Cleaning with lauryl sulfate and water; digestion with heated nitric acid	ICP-AES	1 µg/g	No data	DiPietro et al. (1989)
Hair	Cleaning with water; digestion with heated nitric acid and H_2O_2	ET-AAS	<0.026 µg/g	>90	Drash et al. (1997)
Hair	Cleaning with acetone/water	XRF	0.5 µg/g	No data	Gerhardsson et al. (1995a)

AAS, atomic absorption spectroscopy; APDC, ammonium pyrrolidine dithiocarbamate; ASV, anode stripping voltammetry; $Ba(NO_3)_2$, barium nitrate; ET-AAS, electro-thermal atomic absorption spectrometry; GFAAS, graphite furnace atomic absorption spectroscopy; H_2O_2, hydrogen peroxide; H_2SO_4, sulfuric acid; $HClO_4$, perchloric acid; HNO_3, nitric acid; ICP-AES, inductively coupled plasma/atomic emission spectroscopy; ICP-MS, inductively coupled plasma-mass spectrometry; IDMS, isotope dilution mass spectrometry; MIBK, methyl isobutyl ketone; NIOSH, National Institute for Occupational Safety and Health; ^{206}Pb, lead 206; XRF, X-ray fluorescence.

Table AX4-2. Summary of Selected Measurements of PbB Levels in Humans

Reference, Study Location, and Period	Study Description	PbB Measurement			Comment
United States					
CDC (2005) U.S. 1999-2002	Design: National survey (NHANES IV) stratified, multistage probability cluster design Subjects: Children and adults (≥1 yrs, n = 16, 915) in general population Biomarker measured: PbB Analysis: ICP-MS	Units: µg/dL Geometric mean (95% CI)			Data from NHANES IV Phase 1 (1999-2000) and 2 (2001-2002).
		Age (yr)	**1999-2000**	**2001-2002**	
		1-5:	1.66 (1.60, 1.72)	1.45 (1.39, 1.40)	
		n:	7, 970	8, 945	
		6-11:	1.51 (1.36, 1.66)	1.25 (1.14, 1.36)	
		n:	905	1,044	
		12-19:	1.10 (1.04, 1.17)	0.94 (0.90, 0.99)	
		n:	2, 135	2, 231	
		≥20:	1.75 (1.68, 1.81)	1.56 (1.49, 1.62)	
		n:	4, 207	4, 772	
		Males:	2.01 (1.93, 2.09)	1.78 (1.71, 1.86)	
		n:	3, 913	4, 339	
		Females:	1.37 (1.32, 1.43)	1.19 (1.14, 1.25)	
		n:	4,057	4, 606	
Brody et al. (1994) Pirckle et al. (1998) U.S. 1988-1994	Design: National survey (NHANES III) stratified multistage probability cluster design. Subjects: Children and adults (≥1 yrs, n = 29, 843) in general population Biomarker measured: PbB Analysis: GFAAS	Units: µg/dL Geometric mean (95% CI)			Comparison of data from NHANES III Phase 1 (1988-1991) and Phase 2 (1991-1994) indicated declining PbB concentrations in children.
		Age (yr)	**1988-1991**	**1991-1994**	
		1-5:	3.6 (3.3, 4.0)	2.7 (2.5, 3.0)	
		n:	2, 234	2, 392	
		6-11:	2.5 (2.2, 2.7)	1.9 (1.8, 2.1)	
		n:	1, 587	1, 345	
		12-19:	1.6 (1.4, 1.9)	1.5 (1.4, 1.7)	
		n:	1, 376	1, 615	
		20-49:	2.6 (2.5, 2.8)	2.1 (2.0, 2.2)	
		n:	4, 320	4, 716	
		50-69	4.0 (3.8, 4.2)	3.1 (2.9, 3.2)	
		n:	2,071	2,026	
		≥70	4.0 (3.7, 4.3)	3.4 (3.3, 3.6)	
		n:	1, 613	1, 548	
		Males:	3.7 (3.5, 3.9)	2.8 (2.6, 2.9)	
		n:	6,051	6, 258	
		Females:	2.1 (2.0, 2.2)	1.9 (1.8, 2.)	
		n:	6,068	7, 384	

Table AX4-2 (cont'd). Summary of Selected Measurements of PbB Levels in Humans

Reference, Study Location, and Period	Study Description	PbB Measurement	Comment
United States (cont'd)			
Nash et al. (2003) U.S. 1988-1994	Design: National survey (NHANES III) stratified, multistage probability cluster design Subjects: Women(n = 2, 575), age range: 40-59 yrs, in general population Biomarker measured: PbB Analysis: GFAAS	Units: µg/dL Geometric mean (95% CI, n) Premenopausal: 1.9 (1.7, 2.0, 1, 222) Surgically menopausal: 2.7 (2.4, 3.2, 139) Naturally menopausal: 2.9 (2.5, 3.2, 653)	Geometric mean PbB concentrations were significantly lower in premenopausal women. Increasing PbB concentrations were significantly associated with decreased bone mineral density.
Pirkle et al. (1994) U.S. 1976-1980	Design: National survey (NHANES II, III) stratified, multistage probability cluster design Subjects: Children and adults (≥1 yrs, n = 29, 843) in general population Biomarker measured: PbB Analysis: GFAAS	Units: µg/dL Geometric mean (95% CI) **Age (yr) / 1976-1980 / 1988-1991** 1-5: 15.0 (14.2, 15.8) / 3.6 (3.3, 4.0) n: 2, 271 / 2, 234 6-19: 11.7 (11.2, 12.4) / 1.9 (1.7, 2.2) n: 2,024 / 2, 963 20-74: 13.1 (12.7, 13.7) / 3.0 (2.8, 3.2) n: 5, 537 / 6, 922 *Males:* 15.0 (14.5, 15.5) / 3.7 (3.5, 3.9) n: 4, 895 / 6,051 *Females:* 11.1 (10.6, 11.5) / 2.1 (2.0, 2.2) n: 4, 937 / 6,068	Comparison of data from NHANES II (1976-1980) and Phase 1 of NHANES III (1988-1991) indicated declining PbB concentrations in U.S. population.
Symanski and Hertz-Picciotto (1995) U.S. 1982-1984	Design: National survey (HHANES) multistage-area probability sample Subjects: Adults, females (n = 3, 137), age range 20-60 yrs, in general Hispanic population Biomarker measured: PbB Analysis: GFAAS	Units: µg/dL Arithmetic mean (SE, n) All Premenopausal: 7.5 (0.07, 1, 984) Menopausal: 8.9 (0.11, 1, 152) Mexican-American: Premenopausal: 7.2 (0.13, 1, 219) Menopausal: 8.4 (0.20, 624)	Mean difference between premenopausal and postmenopausal (≤4 yrs) was 1.4 µg/dL (95% CI: 0.20, 2.7).

Table AX4-2 (cont'd). Summary of Selected Measurements of PbB Levels in Humans

Reference, Study Location, and Period	Study Description	PbB Measurement				Comments
United States (cont'd)						
Yassin et al. (2004) U.S. 1988-1994	Design: National survey (NHANES III) stratified, multistage probability cluster design Subjects: Adults (n = 11, 126) in general population, age range: 18-64 yr), stratified by occupational category Biomarker measured: PbB Analysis: GFAAS	Units: µg/dL				
		Occupation	GM	GSD	Maximum	n
		Vehicle mechanics	4.80	3.88	28.1	169
		Food service workers	2.00	2.69	27.0	700
		Management, professional technical, and sales workers	2.13	4.05	39.4	4, 768
		Personal service workers	2.48	4.52	25.9	1, 130
		Agricultural workers	2.76	4.02	23.4	498
		Production workers: machine operators, material movers, etc.	2.88	4.24	52.9	1, 876
		Laborers other than in construction	3.47	3.36	21.8	137
		Transportation workers	3.49	5.19	22.3	530
		Mechanics other than vehicle mechanics	3.50	4.91	16.6	227
		Construction trades people	3.66	4.64	16.9	470
		Construction laborers	4.44	7.84	36.0	122
		Health service workers	1.76	2.24	22.4	499
		All	2.42	6.93	52.9	11, 126
Mexico						
Hernandez-Avila et al. (2002) Mexico 1993-1995	Design: Cross-sectional Subjects: Adults females (n = 903) in general population, age range: 36-70 yr Biomarker measured: PbB Analysis: GFAAS	Units: µg/dL Arithmetic mean (SD, n) Premenopausal: 10.63 (5.46, 463) Menopausal: 11.39 (2.65, 437) Surgically menopausal: 10.23 (4.92, 115) Naturally menopausal: 11.30 (5.88, 322)				Mean difference between premenopausal and menopausal; was 0.76 µg/dL (95% CI: 0.224, 1.48).

PbB, blood lead; GFAAS, graphite furnace atomic absorption spectroscopy; ICP-MS, inductively coupled plasma-mass spectrometry; NR, not reported.

Table AX4-3. Bone Lead Measurements in Cadavers

Reference, Study Location, and Period	Study Description	Lead Measurement	Findings, Interpretation
United States			
Wittmers et al. (1988) Minnesota 1976-82	Lead in tibia, skull, iliac crest, rib, and vertebrae. 81 Caucasian males and 53 male cadavers ranging in age from 0 to 98 yr. Ashing, nitric acid, AAS.	Mean and SEM (μg/g bone ash) >75 yr: Tibia 29.0 ±3.4 (n = 28), ilium 17.0 ± 2.6 (n = 29), rib 20.5 ± 2.4 (n = 31), vertebra 18.8 ± 2.6 (n = 30), skull 26.1 ± 3.2 (n = 28) 51-75 yr: Tibia 24.2 ± 2.3 (n = 38), ilium 19.2 ± 2.4 (n = 15), rib 22.3 ± 2.6 (n = 40), vertebra 22.4 ± 2.6 (n = 41), skull 22.8 ± 2.9 (n = 29) 36-50 yr: Tibia 16.6 ±4.1 (n = 14), ilium 9.9 ± 1.6 (n = 15), rib 9.7 ± 1.7 (n = 15), vertebra 11.9 ± 2.1 (n = 15), skull 15.2 ± 3.3 (n = 15) 21-35 yr: Tibia 5.9 ±1.2 (n = 18), ilium 5.3 ± 1.2 (n = 16), rib 5.0 ± 1.2 (n = 18), vertebra 6.3 ± 1.3 (n = 17), skull 4.9 ± 1.1 (n = 17) 14-20 yr: Tibia 2.3 ±1.0 (n = 13), ilium 2.3 ± 0.9 (n = 13), rib 2.9 ± 1.4 (n = 12), vertebra 3.8 ± 1.4 (n = 12), skull 3.2 ± 1.7 (n = 10) 0-2 yr: Tibia 0.3 ±0.2 (n = 11), ilium 0.0 ± 0.0 (n = 11), rib 0.7 ± 0.4 (n = 12), vertebra 0.6 ± 0.6 (n = 12), skull 0.6 ± 0.4 (n = 12)	Ratio of lead in tibia and skull/iliac/rib/vertebrae <1 from age 0 to 35 yrs then >1 from 36 to 75 yrs and greater than 75 yrs. Evidence of differential distribution amongst bones with age; the earliest difference is apparent during adolescence when trabecular bone of the vertebral body accumulates significantly more lead than that of the other 4 sites.
Saltzman et al. (1990) Cincinnati, OH 1970-71	29 tissues from 55 cadavers, mean age 50 yrs. Muffle furnace ashing. Pb concentrations by dithazone method.	Higher concentrations of Pb in tibia compared with rib and vertebrae and higher values for males compared with females. Males (n = 46): Ribs 6.70 ± 3.96 (μg/g, wet weight), tibia 12.55 ± 10.65, vertebrae 4.12 ± 2.49. Females (n = 8): Ribs 3.17 ± 0.91 (μg/g, wet weight), tibia 4.54 ± 2.04, vertebrae 2.01 ± 0.72.	Bone Pb increased with age. Results were similar to those of Barry (1978) and Wittmers et al. (1988).
Canada			
Samuels et al. (1989) Canada 1965-69	Ashed vertebral bones from male and female cadavers from three Canadian cities. AAS method.	Changes for different age ranges in Pb concentration for the period 1965-1969: 0-11 months: 3.98 μg/g (n = 28) 1-4 yrs: 10.02 μg/g (n = 32) 5-11 yrs: 12.91 μg/g (n = 26) 12-19 yrs: 7.11 μg/g (n = 26) ≥20 yrs: 14.77 μg/g (n = 25)	For period 1965 to 1969 levels vary over age groups (p = 0.0001) but there was little gender difference. For the period 1980 to 1998 for Winnipeg, values were approximately half to one third those prevailing earlier.

Table AX4-3 (cont'd). Bone Lead Measurements in Cadavers

Reference, Study Location, and Period	Study Description	Lead Measurement	Findings, Interpretation
Europe			
Drasch et al. (1987) Germany 1983-85	Bone Pb in temporal bone, cortical part of the mid-femur, and pelvic bone from 120 female and 120 male adult cadavers. AAS.	Geometric means: Males: Pelvic 1.95 ± 1.00 (µg/g, wet weight), mid-femur 4.75 ± 2.53, temporal 6.24 ± 3.17. Females: Pelvic 1.41 ± 0.74 (µg/g), mid-femur 3.14 ± 1.89, temporal 5.00 ± 2.66.	Found cortical lead > trabecular lead. Limited difference in Pb for younger males and females; much higher Pb in bones of men >50 yr old compared with women
Drasch and Ott (1988) Germany 1984	Bone Pb in temporal bone, cortical part of the mid femur, and pelvic bone from 82 child cadavers. Nitric acid digestion, AAS.	(values in µg/g wet weight)	Negligible difference for 0 to 1 yr olds, for pre-school children (1-6 yrs) and for 10 to 20 yr olds; mean values for cortical bones showed higher Pb concentrations than trabecular bone; mean Pb in the mid femur and temporal bone was not statistically different for each of three age groups.
Hac et al. (1997) Poland	Pb in rib bone and hair from 59 cadavers, aged 1-87 yrs. Perchloric acid digestion, AAS.	Bone Pb 3.0 (±1.5) µg/g (n = 54).	Small increases to age 50 yrs in rib bone. Number of samples for each age group not stated.
Asia			
Noda et al. (1993) Japan 1976, 1981, and 1986	76 cadavers, age range 0 to 83 yrs.	Age 0 yrs (1.25 µg/g wet weight) to 59 yrs (4.5 µg/g) after which there was a decrease (~2.5 µg/g). For the age range 10-49 yrs, there was no significant difference in mean values of 2.8 to 3.1 µg/g.	Found no significant gender difference but levels in 1986 were significantly lower than in 1976.

Age	**0-1 yrs**		**1-6 yrs**		**10-20 yrs**		**0-20 yrs**	
Sex	Male	Female	Male	Female	Male	Female	Male	Female
n	9	16	9	9	18	16	39	42
Temporal	0.331	0.334	0.530	0.732	1.770	1.740	0.858	0.749
Pelvic bone	0.230	0.278	0.461	0.522	0.748	0.511	0.455	0.404
Mid-femur	0.333	0.327	0.642	0.858	1.342	1.010	0.768	0.632

Table AX4-4. Bone Lead Measurements in Environmentally-Exposed Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
United States			
Kim et al. (1996) Boston, MA 1989-90	Examination of the relationship between tooth Pb in children and bone Pb levels in young adults. Members of a cohort of young adults (n = 63, ~20 yr of age) were reassessed 13 yr after initial examination. Dentine Pb by anodic stripping voltammetry. Bone K-shell XRF. LOWESS smoothing, multiple linear regression.	No PbB. Tibia Pb 1.3 (± 4.4), patella Pb 5.4 (±8.4). Dentine Pb 13.4 (±10.7). Approximately one-third of tibia and one-fourth of patella estimates were negative values.	A 10 µg/g increase in dentine Pb levels in childhood was predictive of a 1 µg/g increase in tibia Pb levels and a 5 µg/g increase in patella PbB levels, and a 3 µg/g increase in mean bone Pb levels among the young adults. They concluded that Pb exposure in early life may be used to predict elevated body burden up to 13 yr later.
Hu et al. (1990) Boston, MA Unknown	To evaluate if K-shell XRF can be used to assess low-level Pb burdens in 34 employees (26 males, 8 females) ranging in age from 21 to 58 yr of a biomedical company with no known history of excessive Pb exposure. Medical environmental history questionnaire. Multiple linear regression.	18 (53%) of subjects had bone Pb levels included 0 or less within the estimate of uncertainty. Highest bone Pb 21 ± 4 µg/g bone mineral. For 16 young adults, age and year of home construction had a positive but statistically insignificant effect ($p > 0.05$) on bone Pb.	K-shell XRF may be useful for assessing low-level Pb burdens in epidemiological studies.
Hu et al. (1996) Boston, MA 1991+	Normative Aging Study. Subjects were middle-aged and elderly men who had community (nonoccupational) exposures to lead. Cross-sectional. Backwards elimination multivariate regression models that considered age, race, education, retirement status, measures of both current and cumulative smoking, and alcohol consumption.	47-59 yrs (n = 116): PbB 5.8 (±3.7), tibia 14.6 (±8.3), patella 23.6 (±12.4) 60-69 yrs (n = 360): PbB 6.3 (±4.2), tibia 21.1 (±11.4), patella 30.5 (±16.9) >70 yrs (n = 243): PbB 6.5 (±4.5), tibia 27 (±15.6), patella 38.8 (±23.5)	Factors that remained significantly related to higher levels of both tibia and patella Pb were higher age and measures of cumulative smoking, and lower levels of education. An increase in patella Pb from the median of the lowest to the median of the highest quintiles (13-56 µg/g) corresponded to a rise in PbB of 4.3 µg/dL. Bone Pb levels comprised the major source of circulating lead in these men.
Campbell et al. (2004) New York Unknown	Investigated the relationship between bone mineral density and environmental Pb exposure in 35 African American children.	High Pb exposure: PbB levels (mean 23.6 µg/dL; n = 19); low Pb exposure (mean 6.5 µg/dL; n = 16).	Unexpectedly, they found that children with high Pb exposure had a significantly higher bone mineral density than children with low Pb exposure. They hypothesized that this arises from the effect of Pb on accelerating bone maturation by inhibition of parathyroid hormone-related peptide.

Table AX4-4 (cont'd). Bone Lead Measurements in Environmentally-Exposed Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
United States (cont'd)			
Rosen et al. (1989) Bronx, NY Unknown	Comparison of L-shell XRF measures and EDTA provocation test in lead-toxic children 1-6 yr old. Eligible if PbB 25-55 µg/dL and erythrocyte protoporphyrin >35 µg/dL.	Negative EDTA test results (n = 30): PbB 30 ± 5 µg/dL, tibia Pb 12 ± 2 µg/g (range 7-52). Positive EDTA test results (n = 29): PbB 39 ± 8 µg/dL, tibia Pb 37 ± 3 µg/g (range 7-200).	From PbB and LXRF alone, 90% of Pb-toxic children were correctly classified as being EDTA-positive or -negative. LXRF may be capable of replacing EDTA testing.
Kosnett et al. (1994) Dickson City, PA 1991	Aim to determine the influence of demographic, exposure and medical factors on the bone Pb concentration of subjects with environmental Pb exposure. 101 subjects (49 males, 52 females; aged 11 to 78 yrs) recruited from 49 of 123 households geographically located in a suburban residential neighborhood. Tibia. Multiple regression.	Mean (SD) bone Pb12.7 (14.6). Log-transformed bone Pb highly correlated with age ($r = 0.71$; $p \leq 0.0001$). Gender differences in log-transformed bone Pb values were insignificant up until the 6th decade.	Bone Pb showed no significant change up to age 20 yr, increased with the same slope in men and women between ages 20 and 55 yrs, and then increased at a faster rate in men older than 55 yrs.
Rosen et al. (1993) Moosic and Throop, PA 1989-91	Suburban population (Throop, n = 269) exposed to unusually high emissions during 1963-81 from nearby battery recycling/secondary smelter. Moosic served as control community. Approximately 9% children aged 5-12 yr, 15% 13-17 yr, 40% ≥ 18 yr. Soil and PbB, L-shell XRF.	No significant differences in tibia Pb found among three age groups in Moosic or Throop. Mooaic: means 5-12 yr, 6 µg/g; 13-17 yr, 8 µg/g; ≥18 yr, 7 µg/g Throop: means 5-12 yr, 12 ± 1 µg/g; 13-17 yr, 15 ± 2 µg/g; ≥18 yr, 12 ± 1 µg/g.	No change in bone Pb with age. Baseline values for bone Pb in the environmentally exposed population of Moosic can serve as a reference baseline for contemporary bone Pb levels in similar communities in the USA.
Stokes et al. (1998) Bunker Hill, ID; Spokane, WA 1994	Examined whether environmental exposure to Pb during childhood was associated with current adverse neurobehavioral effects. K-shell XRF. Formerly exposed as children 19-30 yr (n = 238, age 19-30 yr). Referents (n = 258)	Exposed group: PbB 2.9 µg/dL; tibia Pb 4.6 µg/g. Referent group: PbB 1.6 µg/dL; tibia Pb 0.6 µg/g.	

Table AX4-4 (cont'd). Bone Lead Measurements in Environmentally-Exposed Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
United States (cont'd)			
McNeill et al. (2000) Idaho and Washington 1994	To determine if high Pb exposure in childhood persisted until adulthood. 262 exposed subjects and 268 age and sex matched controls aged 19 – 29 yr. Tibia bone Pb, cumulative PbB index. Inverse weighted group mean data, linear regressions.	Group inverse weighted mean (SEM). Males: Exposed 4.54 (0.31); controls 0.03 (0.31) µg Pb/g bone mineral. Females: Exposed 5.61 (0.43); controls 1.67 (0.43) µg Pb/g bone mineral.	Lead from exposure in early childhood had persisted in the bone matrix until adulthood. Bone Pb significantly correlated with age for exposed groups. No significant correlation in regressions for control groups with age. Exposed subjects had group bone Pb levels significantly higher ($p < 0.005$) than control subjects in 7 of 11 age groups. Exposed subjects had increased current PbB concentrations that correlated significantly with bone Pb values. Incorporation rate of Pb into bone 0.039 (0.003) (μg Pb/g bone mineral)/ µg/dL yr).
Mexico			
Farias et al. (1998) Mexico City and suburbs 1995-96	Examined the relation of blood and tibia bone Pb levels to Pb determinants in 100 adolescents aged 11 to 21 yr. LOWESS smoothing, multivariate regressions.	Females (n = 62): PbB 6.4 (±3.2), tibia 5.5(±8.6). Males (n = 36): PbB 9.1 (±5.5), tibia 3.8 (±5.5). 25 subjects had bone Pb < 0. Bone Pb accounted for 4.1% of variation in PbB. Increase in bone Pb of 21.6 µg/g was associated with an increase in PbB of 1.2 µg/dL.	Predictors of bone Pb included higher traffic density near the home, mother's smoking history, and time spent outdoors. Predictors of log-transformed PbB included bone Pb levels, male sex, use of Pb-glazed ceramics, and living in Mexico City. Bone Pb accumulated over time constitutes a moderate source of circulating Pb during adolescence

PbB = blood lead.

Table AX4-5. Bone Lead Measurements in Occupationally-Exposed Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
United States			
Hu et al. (1994) U.S. 1991	Construction workers aged 23 to 67 yr (n = 19). Examination of Bone Pb and PbB as predictors of blood pressure in construction workers. Multivariate linear regression, LOWESS smoothing.	PbB 8.3 (±4.0), tibia Pb 9.8 (±9.5), patella Pb 13.9 (±13.6).	
Schwartz et al. (2000b) U.S. 1995	Retired organolead employees (n = 543). Aim to determine influence of PbB, chelatable Pb, and tibial Pb on systolic and diastolic blood pressure.	PbB 4.6 (±2.6), tibia Pb14.4 (±9.3).	Tibia Pb was not associated with any blood pressure measures.
Popovich et al. (2005) Idaho	108 former female smelter employees and 99 referents to assess the PbB versus bone Pb relationship.	Exposed: PbB 2.73 (±2.39), tibia 14.4 (±0.5) Referents: PbB 1.25 (±2.10), tibia 3.22 (±0.50) Pb concentrations in tibia and blood significantly higher in the exposed group. Endogenous release rate (µg Pb per dL blood/µg Pb/g bone) in postmenopausal women was double the rate found in premenopausal women (0.132 ± 0.019 vs. 0.067 ± 0.014).	Higher tibia bone Pb (and PbB) was associated with use of estrogen (present or former) in both the whole referent group and postmenopausal women in the referent group.
Canada			
Fleming et al. (1997) Canada 1994	Primary smelter workers, 367 active and 14 retired. PbB in 204 workers returning after a 10-mo strike ended in 1991. Cumulative PbB index, K-shell measures with ^{109}Cd source.	Active (1975-81) median PbB 16.0, (1987-92) median PbB 8.0, tibia range 0-150, calcaneus 0-250. Retired tibia range 20-120, calcaneus 40-220. Bone Pb-cumulative PbB index slopes larger for retired compared with active workers, but not significant.	Nonlinearities in cumulative PbB index and tibia and calcaneus Pb suggest differences in Pb transfer from whole blood to bone among smelter employees. Contribution to PbB from bone stores at any instant in time is similar for all occupationally exposed populations, active or retired. Age-related variations in bone turnover are not a dominant factor in endogenous exposure of male lead workers. More rapid absorption of Pb in calcaneus than tibia.

Table AX4-5 (cont'd). Bone Lead Measurements in Occupationally-Exposed Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Canada (cont'd)			
Fleming et al. (1998) Canada 1994	Primary smelter. ALAD 1-1 (n = 303) and ALAD1-2, 2-2 (n = 65). PbB, serum Pb, cumulative PbB index, ALAD genotype, K-shell measures with ^{109}Cd source.	1-1: PbB 22.9, tibia 41.2, calcaneus 71.6 1-2, 2-2: PbB 25.2, tibia 42.7, calcaneus 72.3. Slopes of linear relations of bone Pb to cumulative PbB index were greater for workers homoallelic for ALAD 1, indicating more efficient uptake of lead from blood into bone; effect most significant in calcaneus bone and for workers hired since improved safety measures enacted in 1977 [ALAD1-1: 0.0528 ± 0.0028 and ALAD1-2 or 2-2: 0.0355 ± 0.0031 ($p < 0.001$)].	Decreased transfer of PbB into bone in individuals expressing the ALAD2 allele contrasted with increased PbB. ALAD genotype affected lead metabolism and potentially modified lead delivery to target organs including the brain but ALAD genotype did not significantly affect the net accumulation of lead in bone.
Brito et al. (2000) Canada 1993-98	Aims were to: (i) investigate the long-term human Pb metabolism by measuring the change of Pb concentration in the tibia and calcaneus between 1993 and 1998; and (ii) assess whether improved industrial hygiene was resulting in a slow accumulation of Pb in an exposed workforce. 101 workers in a secondary lead smelter, 51 subjects had similar bone Pb measurements in 1993. Most other subjects had been hired since 1993. Cumulative PbB index. Linear regressions.	Repeats (n = 51) 1993: Tibia 39 (±19), calcaneus 64 (±36). 1998: Tibia 33 (±18), calcaneus 65 (±38). Non-repeats (n = 50) 1998: Tibia 15 (±16), calcaneus 13 (±18). Tibia Pb decreased significantly ($p < 0.001$) in the 51 subjects with repeated bone Pb measurements. Tibia Pb in 1993 and changes in cumulative PbB index were significant predictors of changes in tibia Pb. An overall half-life of 15 yr (95% CI: 9, 55 yr) was estimated. Adding continuing lead exposure and recirculation of bone lead stores to the regression models produced half-life estimates of 12 and 9 yr, respectively, for release of lead from the tibia. Repeat subjects showed no net change in calcaneus Pb after 5 yr.	The decrease in new exposure coupled to release of previously stored bone Pb resulted in a significant decrease in tibia Pb in the repeat subjects. The rate of clearance of Pb from the tibia of 9 to 15 yr is towards the more rapid end of previous estimates. The lack of a significant change in the calcaneus Pb was surprising and if confirmed would have implications for models of Pb metabolism.

Table AX4-5 (cont'd). Bone Lead Measurements in Occupationally-Exposed Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Canada (cont'd)			
Brito et al. (2002) Canada 1994, 1999	Evaluated endogenous release of Pb from bone to blood in 204 exposed subjects resuming their duties after a 10-mo strike in a primary lead smelter in 1991. Bone Pb (^{109}Cd source) measured in the tibia and calcaneus in 1994 (Fleming et al., 1997) and 1999. A linear model used to predict the current PbB upon the level of lead in bone. 327 subjects available on both occasions. Group H higher PbB and Group L lower PbB.	Group H: PbB 22.0, tibia 19.2 (n = 120) Group L: PbB 20.6, tibia 82.8 (n = 45) Group H: PbB 24.2, calcaneus 41.4 (n = 90) Group L: PbB 20.2,calcaneus 138.2 (n = 45) Structural analysis of data gave slopes for tibia (2.0, 95% CI: 1.66-2.54) and calcaneus (0.19, 95% CI: 0.16-0.23) that were significantly higher than those predicted by the commonly used simple linear regression method, for tibia (0.73, 95%, CI: 0.58-0.88) and calcaneus (0.08, 95% CI: 0.06-0.09).	Suggested that more Pb than previously predicted by regression analysis is released from bone to blood.
Europe			
Somervaille et al. (1988) England	K-shell measures with ^{109}Cd source on diverse Pb workers and controls Crystal glass (n = 87); Battery plant (n = 88); Precious metals (n = 15); Laboratory (n = 20). Cumulative PbB index.	Crystal glass: PbB 48.1, tibia 31.0 Battery plant: PbB 32.3, tibia 32.3 Precious metals: PbB 51.4, tibia 54.8 Laboratory: PbB 13.1, tibia 16.7	Correlation coefficients between tibia lead and duration of employment were consistently higher at all three factories respectively ($r = 0.86$, $p < 0.0001$; $r = 0.61$, $p < 0.0001$; $r = 0.80$, $p < 0.0001$). Strong relation between tibia Pb and cumulative PbB index among workers in factories from which PbB histories were available.
Christoffersson et al. (1984) Sweden Unknown	Lead smelter employees Active (n = 75); Former plant (n = 32) Finger bone measurement with ^{57}Co source.	Active: median PbB 53.8 (15.5), mean tibia 43 (<20, 122) Former: median PbB 24.9 (7.0), mean tibia 59.0 (<20, 135)	Increase of bone Pb with time of employment, no association between bone Pb and current PbB in active workers, in retired workers PbB rose with increasing bone Pb.
Christoffersson et al. (1986) Sweden 1978-84	Retired lead workers. Group 1: 7 smelter, 1 storage battery monitored for 2-5 yr directly after end of exposure. Group 2: 6 battery, bone Pb measured 7-13 yr after end of exposure. Finger bone measurement with ^{57}Co source from 4 to 9 times.	Group 1: mean initial bone Pb 97 (61, 131), decreasing bone Pb with time half-life 6.7 yr (3.4, 15) Group 2: mean initial bone Pb 72 (37, 96), mean half-life 8.2 yr (2.4,∞)	Decrease of lead in bone after the end of exposure considerably faster than estimated earlier from various data on lead metabolism.

Table AX4-5 (cont'd). Bone Lead Measurements in Occupationally-Exposed Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Europe (cont'd)			
Hanninen et al. (1998) Finland Unknown	Storage battery workers Grouped into those whose PbB exceeded 50 µg/dL [High PbB (n = 28; 21 males)], and never [Low PbB (n = 26; 22 males)]. Evaluation of neuropsychological dysfunction.	High PbB: average PbB 39.3 (±8.3), tibia 35.3 (±16.6), calcaneus 100.4 (±43.1) Low PbB: average PbB 29.0 (±6.2), tibia 19.8 (±13.7), calcaneus 78.6(±62.4)	No relation was found between the neuropsychological test battery and tibial Pb.
Erkkilä et al. (1992) Finland Unknown	K-shell measures with ^{109}Cd source on acid battery employees and controls Active (n = 91); Former plant (n = 16); Office (n = 38); Laboratory (n = 26). K-shell XRF.	Active: PbB 30.0 (9.5), tibia 21.1 (17), calcaneus 76.6 (55.3) Former plant: PbB 12.2 (6.2), tibia 32.4 (34.9), calcaneus 73.5 (57.7) Office: PbB 6.4 (3.3), tibia 7.7 (11.3), calcaneus 14.2 (15.6) Laboratory: PbB 3.7 (1.7), tibia 3.5 (10.8), calcaneus 1.2 (10.6)	Tibia Pb concentration increased consistently both as a function of intensity of exposure and duration of exposure. Calcaneal Pb concentration strongly dependent on the intensity rather than duration of exposure. Biological half-life of Pb in calcaneus <7-8 yr periods into which the duration of exposure was split. Retired workers: endogenous exposure to Pb arising from skeletal burdens accumulated over a working lifetime can easily produce the dominant contribution to systemic Pb concentrations once occupational exposure has ceased.
Nilsson et al. (1991) Sweden 1980s	Group A: 7 retired smelter workers and 1 battery worker monitored for ~10 yr with 11-17 finger bone measurements with ^{57}Co. Group B: 6 retired battery workers monitored for up to 18.5 yr with 7-13 finger bone measurements.	Bone Pb values decreased over time. A mono-exponential retention model was used. Group A: estimated half-life for bone Pb was 6.2-27 yr. Group B: half-life was 11-470 yr.	The "shared" half-life for bone Pb was 16 (CI: 12, 23) yr. These values are longer than ones of Christoffersson et al. (1986) for the same two groups; no "background" values were subtracted in the latter case.

Table AX4-5 (cont'd). Bone Lead Measurements in Occupationally-Exposed Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Europe (cont'd)			
Gerhardsson et al. (1993) Sweden Unknown	Pb smelter and truck assembly (referent) workers; Active smelter (n = 70); Retired smelter (n = 30); Truck assembly (n = 31); Retired truck assembly (n = 10). K-shell measures with ^{109}Cd source.	Median values presented. Active smelter: PbB 31.9 (5.0, 47.4), tibia 13.0 (−4.1, 72.8), calcaneus 48.6 (0.4, 217.8) Retired smelter: PbB 9.9 (3.3, 21), tibia 39.3 (2.9, 73.4), calcaneus 100.2 (34.8, 188.9) Truck: PbB 4.1 (1.7, 12.4), tibia 3.4 (−9.4, 13.3), calcaneus 12.2 (−12.7, 43.0) Retired truck: PbB 3.5 (2.2, 12.2), tibia 12.0 (−6.7, 23.7), calcaneus 30.2 (−7.1, 56.7)	Higher calcaneus Pb than tibia Pb in active lead workers suggested more rapid absorption over time in this mainly trabecular bone. Estimated biological half times were 16 yr in calcaneus (95% CI: 11, 29 yr) and 27 yr in tibia (95% CI: 16, 98 yr). Strong positive correlation between bone Pb and cumulative PbB index.
Börjesson et al. (1997) Sweden 1992	Pb smelter and referent male metal workers Active smelter (n = 71); Retired smelter (n = 18); Referent active (n = 27); Referent retired (n = 8). Similar cohort to Gerhardsson et al. (1993). Finger bone measurement with ^{57}Co source. Cumulative PbB index.	Median values presented. Active smelter: PbB 33.1 (8.3, 93), bone Pb 23.0 (−13, 99) Retired smelter: PbB 17.2 (8.9, 33.1), bone Pb 55 (3, 88) Active referent: PbB 3.7 (0.8, 7.0), bone Pb 3 (−21, 16) Retired referent: PbB 3.9 (3.1, 6.2), bone Pb 1.5 (−3, 12)	Multiple regression analyses showed bone Pb was best described by the cumulative PbB index, which explained 29% of the observed variance (multiple r^2) in bone Pb in active workers and about 39% in retired workers. Estimated biological half-life of bone Pb among active lead workers was 5.2 yr (95% CI: 3.3-13.0 yr).
Bergdahl et al. (1998) Sweden 1986	Secondary Pb smelter Exposed (n = 77); Referents (n = 24). K-shell measures with ^{109}Cd source. Cumulative PbB index and (calculated) plasma Pb.	Exposed: PbB 35.0 (14, 57), tibia 25 (5, 193), calcaneus 52 (−20, 458) Referents: PbB 5.0 (2.9, 16). tibia 10 (−6, 36), calcaneus 11(−12, 61)	Strong relationships between the tibia Pb (r^2 = 0.78) and calcaneus (r^2 = 0.80) and cumulative PbB index. Half-lives of Pb in tibia 13-24 yr and calcaneus 12-19.
Erfurth et al. (2001) Sweden	Secondary smelter Active (n = 62); Retired (n = 15); Referents (n = 26). Evaluation of effects of Pb on the endocrine system. Finger bone measures with ^{57}Co source.	Median values presented. Active: PbB 33.2 (8.3, 93.2), tibia 21 (−13, 99) Retired: PbB 18.6 (10.4, 49.7), tibia 55 (3, 88) Referents: PbB 4.1 (0.8, 6.2), tibia 2 (−21, 14)	No significant associations between bone Pb and pituitary and thyroid hormones, serum testosterone, gonadotropin-releasing hormone and thyroid releasing hormone.

Table AX4-5 (cont'd). Bone Lead Measurements in Occupationally-Exposed Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Europe (cont'd)			
Roels et al. (1995) Belgium	Pb smelter and others. Active production (n = 73); Other departments (n = 50). K-shell measures with ^{109}Cd source. Cumulative PbB index.	Active: PbB 42.0, tibia 66.5 Others: PbB 14.5, tibia 31.4	Strong relationship between bone Pb and cumulative PbB index in smelter populations ($r = 0.80$, $p < 0.0001$; age explained ≤9.5% of variance). Slope of regression equation of log bone Pb versus log cumulative PbB index showed that doubling of cumulative PbB index corresponds to doubling of bone Pb.
Mexico			
Juarez-Perez et al. (2004) Mexico City 1996-7	Lithographic print shop workers; Males, n = 59, 10 females; mean age 47 yrs Plasma Pb by ultraclean ICP-MS methods. K-shell measures with ^{109}Cd source.	PbB 11.9 (±5.8), tibia 27.6 (±18.1; ND-73.8), patella 46.8 (±29.3; ND-139)	Statistically significant associations between: plasma Pb and PbB, patella Pb, tibia Pb, age, education, use of Pb-glazed ceramics but not air Pb, hand Pb or hygiene index at work. Multiple linear regression models with patella and tibia Pb as main predictors and adjusting for PbB and hygiene index explained 57% of variability in plasma Pb. Negative association between plasma Pb and hygiene index suggest oral exposure and gastrointestinal uptake of Pb predominant source of Pb exposure in these subjects.
Asia			
Schwartz et al. (2001) Korea 1997-99	Korean Pb workers (798, 639 male, 164 female) and controls (135, 124 male, 1 female). Evaluation of associations between PbB, tibia Pb, chelatable Pb, and neurobehavioral functions. K-shell measures with ^{109}Cd source.	Active: PbB 32 (±15), tibia 37.2 (±40.4) Controls: PbB 5.3 (±1.8), tibia 5.8 (±7.0).	After adjustment for covariates, tibia Pb was not associated with neurobehavioral test scores.

Table AX4-5 (cont'd). Bone Lead Measurements in Occupationally-Exposed Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Asia (cont'd)			
Todd et al. (2001) Korea	Korean Pb workers active (n = 723), retired (n = 79), controls (n = 135). Evaluation of associations between PbB, tibia Pb, chelatable Pb. K-shell measures with ^{109}Cd source.	Active: median PbB 31.7, tibia 24.4 (−7.4, 337.6) Retired: median PbB 13.5, tibia 26.4 (−6.7, 196.7) Controls: median PbB 5.1, tibia 5.0 (−10.9, 26.6)	Control women higher bone Pb than men. Job duration, body mass index, and age were positive predictors of tibial Pb. Rate of increase in tibia Pb with age itself increased with increasing age. Tibial Pb stores in older subjects are less bioavailable and may contribute less to PbB than tibial stores in younger subjects.

PbB = blood lead.

Table AX4-6. Bone Lead Contribution to PbB

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
United States			
Korrick et al. (2002) Boston, MA 1990-95	Nurses' Health Study. Cross-sectional study of 264 elderly women; 46-54 yr (n = 80) 55-64 yr (n = 102), 65-74 yr (n = 82). Tibia and patella Pb. Multivariate linear regression models.	46-54 yr: PbB 2.7 (SE ±0.3), tibia 10.5 (±1.0), patella 14.9 (±1.2) 55-64 yr: PbB 3.4 (±0.2), tibia 12.7 (±0.9), patella 17.0 (±1.1) 65-74 yr: PbB 3.3 (±0.3), tibia 16.4 (±0.9), patella 19.8 (±1.2). An increase from the first to the fifth quintile of tibia Pb level (19 µg/g) was associated with a 1.7 µg/dL increase in PbB (p 0.0001).	Tibia and patella Pb values were significantly and positively associated with PbB but only among postmenopausal women who were not using estrogens. Older age and lower parity were associated with higher tibia Pb; only age was associated with patella Pb. They suggested the observed interaction of bone Pb with estrogen status in determining PbB supports the hypothesis that increased bone resorption, as occurs postmenopausally because of decreased estrogen production, results in heightened release of bone Pb stores into blood.
Popovic et al. (2005) Bunker Hill, ID 1994	108 former female smelter employees and 99 referents to assess the PbB versus bone Pb relationship	Exposed: PbB 2.73 (±2.39), tibia 14.4 (±0.5) Referents: PbB 1.25 (±2.10), tibia 3.22 (±0.50) Pb concentrations in tibia and blood significantly higher in the exposed group. Endogenous release rate (µg Pb per dL blood/µ Pb/g bone) in postmenopausal women was double the rate found in premenopausal women (0.132 ± 0.019 vs. 0.067 ± 0.014).	Higher tibia bone Pb (and PbB) was associated with use of estrogen (present or former) in both the whole referent group and postmenopausal women in the referent group.

Table AX4-6 (cont'd). Bone Lead Contribution to PbB

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Canada			
Brito et al. (2000) Canada 1993-98	Aims were to: (i) investigate the long term human Pb metabolism by measuring the change of Pb concentration in the tibia and calcaneus between 1993 and 1998; and (ii) assess whether improved industrial hygiene was resulting in a slow accumulation of Pb in an exposed workforce. 101 workers in a secondary lead smelter, 51 subjects had similar bone Pb measurements in 1993. Most other subjects had been hired since 1993. Cumulative PbB index. Linear regressions.	Repeats (n = 51) 1993: Tibia 39 (±19), calcaneus 64 (±36). 1998: Tibia 33 (±18), calcaneus 65 (±38). Non-repeats (n = 50) 1998: Tibia 15 (±16), calcaneus 13 (±18). Tibia Pb decreased significantly ($p < 0.001$) in the 51 subjects with repeated bone Pb measurements. Tibia Pb in 1993 and changes in cumulative PbB index were significant predictors of changes in tibia Pb. An overall half-life of 15 yr (95% CI: 9, 55 yr) was estimated. Adding continuing lead exposure and recirculation of bone lead stores to the regression models produced half-life estimates of 12 and 9 yr, respectively, for release of lead from the tibia. Repeat subjects showed no net change in calcaneus Pb after 5 yr.	The decrease in new exposure coupled to release of previously stored bone Pb resulted in a significant decrease in tibia Pb in the repeat subjects. The rate of clearance of Pb from the tibia of 9 to 15 yr is towards the more rapid end of previous estimates. The lack of a significant change in the calcaneus Pb was surprising and if confirmed would have implications for models of Pb metabolism.
Brito et al. (2002) Canada 1994, 1999	Evaluated endogenous release of Pb from bone to blood in 204 exposed subjects resuming their duties after a 10-mo strike in a primary lead smelter in 1991. Bone Pb (^{109}Cd source) measured in the tibia and calcaneus in 1994 (Fleming et al., 1997) and 1999. A linear model used to predict the current PbB upon the level of lead in bone. 327 subjects available on both occasions. Group H higher PbB and Group L lower PbB.	Group H: PbB 22.0, tibia 19.2 (n = 120) Group L: PbB 20.6, tibia 82.8 (n = 45) Group H: PbB 24.2, calcaneus 41.4 (n = 90) Group L: PbB 20.2,calcaneus 138.2 (n = 45) Structural analysis of data gave slopes for tibia (2.0, 95% CI: 1.66, 2.54) and calcaneus (0.19, 95% CI: 0.16, 0.23) that were significantly higher than those predicted by the commonly used simple linear regression method, for tibia (0.73, 95% CI: 0.58, 0.88) and calcaneus (0.08, 95% CI: 0.06, 0.09).	Suggested that more Pb than previously predicted by regression analysis is released from bone to blood.

Table AX4-6 (cont'd). Bone Lead Contribution to PbB

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Mexico			
Brown et al. (2000) Mexico City 1994-5	Investigated determinants of bone Pb and PbB of 430 lactating Mexican women during the early postpartum period and contribution of bone Pb to PbB. Linear regression analyses.	PbB 9.5 (±4.5), tibia 10.2 (±10.1), patella 15.2 (±15.1).	Older age, use of Pb glazed pottery, and higher proportion of life spent in Mexico City were main predictors of higher tibia and patella Pb. Women in the 90th percentile for patella Pb had an untransformed predicted mean PbB 3.6 µg/dL higher than those in the 10th percentile.
Téllez-Rojo et al. (2002) Mexico City 1994-95	Evaluated the hypothesis that lactation stimulates Pb release from bone to blood. Cross–sectional examination of breastfeeding patterns and bone Pb as determinants of PbB among 425 lactating women (mean age 24.8 ± 5.3 yr) for 7 mo after delivery. Bone Pb at 1 mo postpartum. Maternal blood samples and questionnaire information collected at delivery and at 1, 4, and 7 mo postpartum. Generalized estimating equations.	Mean PbB decreased with time postpartum: 1 mo 9.4 (±4.4), 4 mo 8.9 (±4.0), 7 mo 7.9 (±3.3). Tibia 10.6 (11.6 after correction for negative values), patella 15.3 (16.9 after correction). After adjustment for bone Pb and environmental exposure, women who exclusively breastfed their infants had PbB levels that were increased by 1.4 µg/dL and women who practiced mixed feeding had levels increased by 1.0 µg/dL, in relation to those who had stopped lactation. A 10 µg Pb/g increment in patella and tibia bone Pb increased PbB by 6.1% (95% CI: 4.2, 8.1) and 8.1% (95% CI: 5.2, 11.1), respectively.	They concluded that their results support the hypothesis that lactation is directly related to the amount of Pb released from bone.
Garrido-Latorre et al. (2003) Mexico City 1995	Aim was to examine the relationship of PbB levels to menopause and bone lead levels in 232 perimenopausal and postmenopausal women from Mexico City. Measured bone mineral density in addition to bone Pb. Information regarding reproductive characteristics and known risk factors for PbB was obtained using a standard questionnaire by direct interview. Mean age of the population was 54.7 yrs (±9.8). Linear regression analyses.	PbB 9.2 (±4.7), tibia 14.85 (±10.1), patella 22.73 (±14.9). A change of 10 µg Pb/g bone mineral in postmenopausal subjects was associated with an increase in PbB of 1.4 µg/dL, whereas a similar change in bone lead among premenopausal women was associated with an increase in PbB of 0.8 µg/dL.	Found that postmenopausal women using hormone replacement therapy had lower PbB levels and higher tibia and patella bone Pb levels than non-users; patella Pb explained the greatest part of variations in PbB. Found no association with PbB levels and did not describe any relationships between bone lead and bone density.

PbB = blood lead.

Table AX4-7. Bone Lead Studies in Pregnant and Lactating Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
United States			
Hu et al. (1996) Boston, MA 1990	Cord PbB measured in 223 women, 41 bone Pb measured at 1-4 postpartum. ANOVA.	Values omitted if measurement uncertainty was >10 µg/g for tibia and 15 µg/g for patella. Cord PbB 1.19 (±1.32), maternal PbB 2.9 (±2.6), tibia 4.5 (±4.0) patella 5.8 (±4.5). Maternal age was the only factor marginally associated with combined bone Pb ($p = 0.08$) but not individually with tibia or patella Pb.	Umbilical cord PbB among women served by this Boston hospital declined dramatically from 1980 to 1990.
Rothenberg et al (2000) Los Angeles, CA 1995-98	Examined bone Pb contribution to PbB in a group of 311 immigrant women (mean age 27.8 ±7.5 yr), 99% from Latin America, during the 3rd trimester of pregnancy, and 1 to 2 mo after delivery. Multiple regression, variance-weighted least squares regression, structural equation modeling.	Prenatal PbB 2.2 (+4.8/−1.0, geometric mean), postnatal PbB 2.8 (+4.9/−1.2) ($p < 0.0001$), tibia 6.7(±12.5), calcaneus 8.4 (±13.2). Variance-weighted multiple regression and structural equation models showed that both calcaneus and tibia Pb were directly associated with prenatal PbB but only calcaneus Pb was associated with postnatal PbB. Increasing natural log yrs in the United States independently predicted decreasing calcaneus and 3rd trimester PbB.	Suggest that while some exogenous Pb sources and modulators of PbB, such as use of Pb-glazed pottery and calcium in the diet, control Pb exposure during and after pregnancy, endogenous Pb sources from past exposure before immigration continue to influence PbB levels in this cohort.
Rothenberg et al. (2002) Los Angeles, CA 1995-2001	Examined the effects of blood and bone PbB on hypertension and elevated blood pressure in the 3rd trimester and postpartum among 1,006 mostly Latina and Afro-American women. Multiple and logistic regression.	Returned and eligible: 3rd trimester PbB (n = 720) 1.9 (+3.6/−1.0), postpartum PbB (n = 704) 2.3 (+4.3/−1.2), tibia (n = 700) 8.0 (±11.4), calcaneus (n = 700) 10.7 (±11.9). Returned but ineligible: 3rd trimester PbB (n = 279) 1.9 (+4.2/−0.8), postpartum PbB (n = 274) 2.3 (+4.7/−1.1), tibia (n = 263) 8.7 (±13.9), calcaneus (n = 262) 11.2 (±15.1). For each 10 µg/g increase in calcaneus Pb level, the odds ratio for 3rd trimester hypertension (systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg) was 1.86 (95% CI: 1.04, 3.32). In normotensive subjects, each 10 µg/g increase in calcaneus Pb level was associated with a 0.70 mmHg (95% CI: 0.04, 1.36) increase in 3rd trimester systolic blood pressure and a 0.54 mmHg (95% CI: 0.01, 1.08) increase in diastolic blood pressure after adjusting for postpartum hypertension, education, immigration status, current smoking, current alcohol use, parity, age, and body mass index. Tibia bone Pb was not related to hypertension or elevated blood pressure either in the 3rd trimester or postpartum, nor was calcaneus Pb related to postpartum hypertension or elevated blood pressure.	The authors concluded that past Pb exposure influences hypertension and elevated blood pressure during pregnancy and controlling blood pressure may require reduction of Pb exposure long before pregnancy.

Table AX4-7 (cont'd). Bone Lead Studies in Pregnant and Lactating Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Mexico			
Hernandez-Avila et al. (1996) Mexico City	Cross-sectional investigation of the interrelationships between environmental, dietary, and lifestyle histories, blood and bone Pb levels, among 98 recently postpartum women. Multivariate linear regression. Age 25.6 (±6.8) yr.	14-20 yr (n = 24): PbB 10.4 (±4.1), tibia 11.8 (±14.9), patella 14.1 (±13.3). 21-29 yr (n = 44): PbB 10.3 (±4.8), tibia 10.7 (± 10.9), patella 17.1 (±13.4) 30-43 yr (n = 27): PbB 7.8 (± 3.7), tibia 16.3 (±8.4), patella 18.1 (±12.7). A 34 µg/g increase in patella Pb (from the medians of the lowest to the highest quartiles) was associated with an increase in PbB of 2.4 µg/dL. Significant predictors of bone Pb included years living in Mexico City, lower consumption of high calcium content foods, and nonuse of calcium supplements for the patella and years living in Mexico City, older age, and lower calcium intake for tibia bone. Low consumption of milk and cheese, as compared to the highest consumption category (every day), was associated with an increase in tibia Pb of 9.7 µg/g.	Suggest that patella bone is a significant contributor to PbB during lactation and that consumption of high calcium content foods may protect against the accumulation of Pb in one.
González-Cossío et al. (1997) Mexico City Unknown	Examined relationship of Pb levels in cord blood and maternal bone to birth weight. Umbilical cord and maternal venous blood samples and anthropometric and sociodemographic data were obtained at delivery and 1 mo postpartum. Bone Pb at 1 mo postpartum. Multiple regression, LOWESS. Background information for calcium supplementation study Hernandez-Avila et al. (2003). Mother-infant pairs (n = 272).	Maternal PbB 8.9 (±4.1), cord PbB 7.1 (±3.5), tibia 9.8 (±8.9), patella 14.2 (±13.2). After adjustment for other determinants of birth weight, tibia Pb was the only Pb biomarker clearly related to birth weight. The decline in birth weight associated with increments in tibia Pb was nonlinear and accelerated at the highest tibia Pb quartile. In the upper quartile, neonates were on average, 156 g lighter than those in the lowest quartile.	Bone-lead burden is inversely related to birth weight.

Table AX4-7 (cont'd). Bone Lead Studies in Pregnant and Lactating Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Mexico (cont'd)			
Brown et al. (2000) Mexico City 1994-5	Investigated determinants of bone Pb and PbB of 430 lactating Mexican women during the early postpartum period and contribution of bone Pb to PbB. Linear regression analyses.	PbB 9.5 (±4.5), tibia 10.2 (±10.1), patella 15.2 (±15.1).	Older age, use of Pb glazed pottery, and higher proportion of life spent in Mexico City were main predictors of higher tibia and patella Pb. Women in the 90th percentile for patella Pb had an untransformed predicted mean PbB 3.6 µg/dL higher than those in the 10th percentile.
Chuang et al. (2001) Mexico City 1994-95	Aim to estimate the contribution of maternal whole PbB and bone Pb, and environmental Pb to umbilical cord PbB (as a measure of fetal Pb exposure). Maternal and umbilical cord blood samples within 12 hr of each infant's delivery. Structural equation modeling.	Bone Pb measured within 1 mo after delivery. PbB 8.45 (±3.94, n = 608), tibia 9.67 (±9.21, n = 603), patella 14.24 (±14.19, n = 575). Tibia and patella Pb, use of Pb glazed ceramics, and mean air Pb level contributed significantly to plasma Pb. An increase in patella Pb and tibia Pb was associated with increases in cord PbB of 0.65 and 0.25 µg/dL, respectively.	Suggested that maternal plasma Pb varies independently from maternal whole PbB. Contributions from endogenous (bone) and exogenous (environmental) sources were approximately the same. (Plasma Pb not measured).
Ettinger et al. (2004) Mexico City 1994-95	Aim to quantify the relation between maternal blood and bone Pb and breast-feeding status among 310 lactating women in Mexico City, Mexico, at 1 mo postpartum. Breast milk measured. Multiple linear regression, LOWESS smoothing.	Breastfeeding: PbB 9.3 (±4.4, n = 310), tibia 9.6 (±10.1, n = 303), patella 14.5 (±14.9, n = 294). Non breastfeeding: PbB 9.3 (±4.9, n = 319), tibia 10.5 (±10.2, n = 306), patella 15.2 (±16.1, n = 289). Breast milk geometric mean 1.1 (range 0.21-8.02) µg/L. Breast milk Pb significantly correlated with umbilical cord Pb and maternal PbB at delivery and with maternal PbB and patella Pb at 1 mo postpartum. An interquartile range increase in patella Pb (20 µg/g) was associated with a 14% increase in breast milk lead (95% CI: 5, 25%). An IQR increase in tibia Pb (12.0 µg/g) was associated with a 5% increase in breast milk lead (95% CI: −3, 14).	Suggest that even among a population of women with relatively high lifetime Pb exposure, breast milk Pb levels are low, influenced both by current Pb exposure and by redistribution of bone Pb accumulated from past environmental exposures.

Table AX4-7 (cont'd). Bone Lead Studies in Pregnant and Lactating Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Mexico (cont'd)			
Sanín et al. (2001) Mexico City 1994-95	Examined early postnatal growth in a cohort of healthy breastfed newborns in relation to maternal bone Pb burden. 329 mother-infant pairs sampled for umbilical cord blood at birth and maternal and infant venous blood at 1 mo postpartum. Maternal evaluations at 1 mo postpartum included Pb measures in blood and bone. Primary endpoints were attained weight 1 mo of age, and weight gain from birth to 1 mo of age. Linear regression.	Included in analyses (n = 329): Infant: cord PbB 6.8 (±3.9), PbB 1 mo 5.7 (±3.0) Maternal: PbB 9.7 (±5.2), tibia Pb 10.1 (±10.3), patella Pb 15.2 (±15.2) Excluded from analyses (n = 276): Infant: cord PbB 6.3 (±3.0), PbB 1 mo 5.5 (±3.3) Maternal: PbB 8.8 (±3.9), tibia Pb 9.75 (±10.3), patella Pb 14.2 (±17.3). Infant PbB were inversely associated with weight gain, with an estimated decline of 15.1 g/µg/dL of PbB. Children who were exclusively breastfed had significantly higher weight gains; however, this gain decreased significantly with increasing levels of patella Pb. Multivariate regression analysis predicted a 3.6 g decrease in weight at 1 mo of age/µg Pb/g bone mineral increase in maternal patella Pb levels.	The authors concluded that maternal Pb burden is negatively associated with infant attained weight at 1 mo of age and to postnatal weight gain from birth to 1 mo of age.
Gomaa et al. (2002) Mexico City Unknown	Aim to compare umbilical cord PbB and maternal bone Pb as independent predictors of infant mental development (n = 197). Prospective design. At 24 mo of age, each infant was assessed using the Bayley Scales of Infant Development-II (Spanish Version). Multiple linear regression.	Cord PbB 6.7 (±3.4), tibia 11.5 (±11.0), patella 17.9 (±15.2). After adjustment for confounders, Pb levels in umbilical cord blood and patella bone were significantly, independently, and inversely associated with the Mental Development Index (MDI) scores of the Bailey Scale. In relation to the lowest quartile of patella Pb, the 2nd, 3rd, and 4th quartiles were associated with 5.4-, 7.2-, and 6.5-point decrements in adjusted MDI scores. A 2-fold increase in cord PbB (e.g., from 5-10 µg/dL) was associated with a 3.1-point decrement in MDI score.	Suggest that higher maternal patella bone Pb levels constitute an independent risk factor for impaired mental development in infants at 24 mo of age. This effect is probably attributable to mobilization of maternal bone Pb stores.

Table AX4-7 (cont'd). Bone Lead Studies in Pregnant and Lactating Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Mexico (cont'd)			
Hernandez-Avila et al. (2002) Mexico City 1994	Aim to evaluate the effects of maternal bone Pb stores on anthropometry at birth in 223 mother-infant pairs. Anthropometric data were collected within the first 12 hr following delivery. Maternal information was obtained 1 mo after delivery (mean age 24.4 ± 5.4 yr). Transformed anthropometric measurements to an ordinal 5-category scale, and association of measurements with other factors evaluated with ordinal logistic-regression models. Cumulative Odds Model.	Cord blood 7.01 (±3.5), maternal PbB 8.82 (± 4.0), tibia 10.70 (±7.58, adjusted for negative values), patella 15.39 (±11.18, adjusted for negative values). Maternal PbB increased linearly by 0.096/µg of tibia Pb and 0.078/µg patella Pb. Umbilical cord PbB increased by 0.111/µg tibia Pb and 0.061/µg patella Pb. Birth length of newborns decreased as tibia Pb levels increased (odds ratio of 1.03/µg/g bone mineral [95% CI: 1.01, 1.06]).	Compared with women in the lower quintiles of the distribution of tibia Pb, those in the upper quintile had a 79% increase in risk of having a lower birth length newborn (OR ratio 1.79; 95% CI: 1.10, 3.22). Patella Pb was positively related to the risk of a low head circumference score; this score remained unaffected by inclusion of birth weight. The increased risk was 1.02/ μg Pb/g bone mineral (95% CI: 1.01, 1.04). Odds ratios did not vary substantially after the authors adjusted for birth weight and other important determinants of head circumference.
Téllez-Rojo et al. (2002) Mexico City 1994-95	Evaluated the hypothesis that lactation stimulates Pb release from bone to blood. Cross-sectional examination of breastfeeding patterns and bone Pb as determinants of PbB among 425 lactating women (mean age 24.8 ±5.3 yr) for 7 mo after delivery. Bone Pb at 1 mo postpartum. Maternal blood samples and questionnaire information collected at delivery and at 1, 4, and 7 mo postpartum. Generalized estimating equations.	Mean PbB decreased with time postpartum: 1 mo 9.4 (±4.4), 4 mo 8.9 (±4.0), 7 mo 7.9 (±3.3). Tibia 10.6 (11.6 after correction for negative values), patella 15.3 (16.9 after correction). After adjustment for bone Pb and environmental exposure, women who exclusively breastfed their infants had PbB levels that were increased by 1.4 µg/dL and women who practiced mixed feeding had levels increased by 1.0 µg/dL, in relation to those who had stopped lactation. A 10 µg Pb/g increment in patella and tibia bone Pb increased PbB by 6.1% (95% CI: 4.2, 8.1) and 8.1% (95% CI: 5.2, 11.1), respectively.	They concluded that their results support the hypothesis that lactation is directly related to the amount of Pb released from bone.

Table AX4-7 (cont'd). Bone Lead Studies in Pregnant and Lactating Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Mexico (cont'd)			
Hernandez-Avila et al. (2002) Mexico City 1994	Evaluated the effects that maternal bone Pb has on anthropometry at birth in 223 mother-infant pairs. Anthropometric data (birth length, head circumference) collected within the first 12 hr following delivery. Maternal information was obtained 1 mo postpartum. Transformed anthropometric measurements to an ordinal 5-category scale, ordinal logistic-regression models.	Participants (n = 223) Cord blood 7.01 (±3.5), maternal PbB 8.82 (±4.0), tibia 9.83 (±8.9), patella 14.14 (±13.0). Nonparticipants (n = 494): Cord blood 6.75 (±3.50), PbB 8.47 (±4.19). Birth length of newborns decreased as tibia Pb levels increased. Compared with women in the lower quintiles of the distribution of tibia Pb, those in the upper quintile had a 79% increase in risk of having a lower birth length newborn (odds ratio 1.79; 95% CI: 1.10, 3.22). The effect was attenuated– but nonetheless significant- even after adjustment for birth weight. Patella Pb was positively and significantly related to the risk of a low head circumference score; this score remained unaffected by inclusion of birth weight.	The authors estimated the increased risk of having a low head-circumference score to be 1.02/ µg Pb/g bone mineral (95% CI: 1.01, 1.04). Odds ratios did not vary substantially after the authors adjusted for birth weight and other important determinants of head circumference.
Hernandez-Avila et al. (2003) Mexico City 1994-95	Tested the hypothesis that in a randomized trial of lactating women a dietary calcium supplement will lower PbB levels. Lactating women (mean age 24 yr) were randomly assigned to receive either calcium carbonate (1200 mg of elemental calcium daily) or placebo in a double-blind trial. Blood samples were obtained at baseline, and 3 and 6 mo after the trial began. Primary endpoint was change in maternal PbB in relation to supplement use and other covariates with multivariate generalized linear models for longitudinal observations.	Lactating calcium group (n = 296): PbB 9.2 (±4.2), tibia 10.7 (±9.8), patella 16.2 (±15.7) Lactating placebo (n = 321): PbB 9.4 (± 5.0), tibia 9.6 (±10.3), patella 13.5 (± 15.1) Women randomized to the calcium supplements experienced a small decline in PbB of 0.29 µg/dL (95% CI: −0.85, −0.26). The effect was more apparent among women who were compliant with supplement use and had high patella Pb of ≥5 µg/g. Among this subgroup, supplement use was associated with an estimated reduction in mean PbB of 1.16 µg/dL (95% CI: −2.08, −0.23), an overall reduction of 16.4%.	Among lactating women with relatively high Pb burden, calcium supplementation was associated with a modest reduction in PbB levels.

Table AX4-7 (cont'd). Bone Lead Studies in Pregnant and Lactating Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Mexico (cont'd)			
Téllez-Rojo et al. (2004) Mexico City 1997-99	Tested the hypotheses that maternal bone Pb burden is associated with increasing maternal whole PbB and plasma Pb over the 3 trimesters of pregnancy and that this association is modified by rates of maternal bone resorption. Urine was analyzed for cross-linked N-telopeptides (NTx) of type I collagen, a biomarker of bone resorption. Patella and tibia Pb at 1 mo postpartum. Mixed models.	Participants (n = 193): PbB (µg/dL): initial 7.10 (±1.72), 1st trimester 6.47 (± 0.17), 2nd trimester5.80 (± 0.17), 3rd trimester 6.05 (± 0.17). Plasma (µg/L): 1st trimester 0.13 (±1.88), 2nd trimester 0.12 (± 1.95), 3rd trimester 0.12 (± 1.88) (geometric means and SD) Bone Pb during pregnancy: Tibia 11.35 (±8.82, adjusted for negative values), patella 13.82 (±10.97, adjusted for negative values). Nonparticipants (n = 134): PbB 6.82 (±1.75), tibia 13.71 (±9.17, adjusted for negative values), patella 11.79 (±9.75, adjusted for negative values). Found an increasing trend for plasma Pb among women with the highest bone Pb (≥median level of 12.1 µg/g) but a decreasing trend among less-exposed women(below the median level). The observed increase reached its maximum among women with both the highest bone Pb and the highest bone resorption. In comparison with women with a low bone Pb and a high NTx level, those with a high bone Pb and a high NTx level had, on average, an 80% higher mean plasma Pb. In the cross-sectional analyses for each trimester of pregnancy, there was an increasingly stronger association between bone Pb and plasma Pb (log-transformed) as pregnancy progressed. An increase in patella lead of 10 µg/g would be associated with 9% ($p = 0.07$), 24% ($p < 0.01$), and 25% ($p < 0.01$) increases in plasma Pb in the 1st, 2nd, and 3rd trimesters of pregnancy, respectively. The corresponding values for tibia lead were 8% ($p = 0.16$), 19% ($p < 0.01$), and 13% ($p = 0.01$), respectively. Dietary calcium intake was inversely associated with plasma lead.	They concluded that the results support the hypothesis of a biologic interaction between bone Pb burden and bone resorption. They also suggest that as pregnancy progresses, bone Pb may be mobilized increasingly into plasma.

Table AX4-7 (cont'd). Bone Lead Studies in Pregnant and Lactating Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Mexico (cont'd)			
Moline et al. (2000) Morelos, Mexico 1999	Pilot study to assess the body burden of lead in 24 Mexican women (age 21-34 yr) who were lactating. Demographic and reproductive characteristics of women and potential sources of lead exposure were gathered by a direct interview. Multiple regression. Average time of lactation 22 (±17) months.	PbB 4.6 (± 2.0, geometric mean), tibia 9.2 (±4.2), patella 14.8 (±8.0), calcaneus 11.7 (±11.2). An inverse relationship was noted between months of lactation and age-adjusted calcaneus lead level ($p = 0.001$). No association was observed between age-adjusted patella or tibia lead level and months of lactation ($p = 0.15$).	This pilot study provides further limited evidence for the hypothesis that Pb mobilization occurs during lactation.

PbB = blood lead.

Table AX4-8. Bone Lead Studies of Menopausal and Middle-aged to Elderly Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
United States			
Hu et al. (1996) Boston, MA 1991+	Normative Aging Study. Subjects were middle-aged and elderly men who had community (nonoccupational) exposures to lead. Cross-sectional. Backwards elimination multivariate regression models that considered age, race, education, retirement status, measures of both current and cumulative smoking, and alcohol consumption.	47-59 yr: (n = 116): PbB 5.8 (±3.7), tibia 14.6 (±8.3), patella 23.6 (±12.4) 60-69 yr: (n = 360): PbB 6.3 (±4.2), tibia 21.1 (±11.4), patella 30.5 (±16.9) >70 yr: (n = 243): PbB 6.5 (±4.5), tibia 27 (±15.6), patella 38.8 (±23.5)	Factors that remained significantly related to higher levels of both tibia and patella Pb were higher age and measures of cumulative smoking, and lower levels of education. An increase in patella Pb from the median of the lowest to the median of the highest quintiles (13-56 µg/g) corresponded to a rise in PbB of 4.3 µg/dL. Bone Pb levels comprised the major source of circulating lead in these men.
Kim et al. (1997) Boston, MA 1991-95	Normative Aging Study (n = 70). Aim to examine age and secular trends in bone and PbB levels of community-exposed men aged 52-83 yr. Bone and PbB levels measured twice, with a 3-yr interval.	PbB 6.7 (±1.8), tibia 17.5 (±2.0), patella 29.1 (±1.8) 3 yr later: PbB 5.1 (±1.4), tibia 17.9 (±1.7), patella 22.2 (±1.8)	Cross-sectional analysis of each set of measurements indicated that, on average, a 1-year-older individual would have 2.7% and 2.4-3.2% higher levels of Pb in patella and tibia, respectively. Secular trend over time was decreasing for patella Pb levels and stable for tibia Pb levels.
Cheng et al. (1998) Boston, MA 1991-95	Normative Aging Study (n = 747). Aim to examine relationships of nutritional factors to body Pb burden. Cross-sectional. Multiple regression models adjusting for age, education level, smoking, and alcohol consumption.	PbB 6.2 (± 4.1), tibia 21.9 (± 13.3), patella 32.0 (±19.5). Multiple regression models men in the lowest quintile of total dietary intake levels of vitamin D (including vitamin supplements) (<179 i.u./day) had mean tibia and patella Pb levels 5.6 µg/g and 6.0 µg/g/ higher than men with intake in the highest quintile (≥589 i.u./day). Higher calcium intake was associated with lower bone Pb levels, but this relation became insignificant when adjustment was made for vitamin D. Subjects in the lowest vitamin C intake quintile (<109 mg/day) had a mean PbB level 1.7 µg/dL higher than men in the highest quintile (≥339 mg/day), while men in the lowest iron intake quintile (<10.9 mg/day) had a mean PbB level 1.1 µg/dL higher than men in the highest quintile (≥23.5 mg/day).	Also observed inverse associations of PbB levels with total dietary intake of vitamin C and iron. Suggested that low dietary intake of vitamin D may increase Pb accumulation in bones, while lower dietary intake of vitamin C and iron may increase PbB.

Table AX4-8 (cont'd). Bone Lead Studies of Menopausal and Middle-aged to Elderly Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
United States (cont'd)			
Hu et al. (2001) Boston, MA 1991+	Normative Aging Study. Aim to determine if ALAD polymorphism is associated with altered levels of lead in bone and blood. Multivariate linear regression models controlling for age, education, smoking, alcohol ingestion, and vitamin D intake.	ALAD 1-1 (n = 608): PbB 6.3(±4.1), tibia 22.2 (±13.9), patella 32.2 (±19.9) ALAD 1-2/2-2 (n = 118): PbB 5.7 (±4.2), tibia 21.2 (±10.9), patella 30.4 (±17.2) ALAD 1- 1 genotype was associated with cortical bone lead levels that were 2.55 µg/g (95% CI: 0.05, 5.05) higher than those of the variant allele carriers.	No significant differences by genotype with respect to Pb levels in trabecular bone or blood. In stratified analyses and a multivariate regression model that tested for interaction, the relationship of trabecular bone Pb to PbB appeared to be significantly modified by ALAD genotype, with variant allele carriers having higher PbB levels, but only when trabecular bone Pb levels >60 µg/g. The authors suggest that the variant ALAD-2 allele modifies lead kinetics possibly by decreasing lead uptake into cortical bone and increasing the mobilization of lead from trabecular bone.
Oliveira et al. (2002) Boston, MA 1991-98	Normative Aging Study. To determine if seasonal fluctuations in PbB levels are related to increased mobilization of bone Pb stores during the winter months. Measurements of blood and bone Pb during the high sun exposure months of May-August (summer; n = 290); the intermediate sun exposure months of March, April, September, and October (spring/fall; n = 283); and the low sun exposure months of November-February (winter; n = 191).	Mean PbB levels were slightly lower in summer (5.8 ± 3.4 µg/dL) compared with winter (6.6 ± 4.7 µg/dL). Mean bone Pb levels were higher during the summer than the winter months: 23.9 (±15.2) and 20.3 (±11.3) µg/g respectively for the tibia and 34.3 (±22.8) and 29.0 (±16.2) µg/g respectively for patella.	Found a significant interaction between season and bone Pb with bone Pb during the winter months exerting an almost 2-fold greater influence on PbB levels than during the summer months. The authors attributed this to increased mobilization of endogenous bone Pb stores arising potentially from decreased exposure to sunlight, lower levels of activated vitamin D and enhanced bone resorption.

Table AX4-8 (cont'd). Bone Lead Studies of Menopausal and Middle-aged to Elderly Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
United States (cont'd)			
Korrick et al. (2002) Boston, MA 1990-95	Nurses' Health Study. Cross-sectional study of 264 elderly women; 46-54 yr (80) 55-64 yr (102), 65-74 yr (82). Tibia and patella Pb. Multivariate linear regression models.	46-54 yr: PbB 2.7 (SE ±0.3), tibia 10.5 (±1.0), patella 14.9 (±1.2) 55-64 yr: PbB 3.4 (±0.2), tibia 12.7 (±0.9), patella 17.0 (±1.1) 65-74 yr: PbB 3.3 (±0.3), tibia 16.4 (±0.9), patella 19.8 (±1.2). An increase from the first to the fifth quintile of tibia Pb level (19 µg/g) was associated with a 1.7 µg/dL increase in PbB ($p = 0.0001$).	Tibia and patella Pb values were significantly and positively associated with PbB but only among postmenopausal women who were not using estrogens. Older age and lower parity were associated with higher tibia Pb; only age was associated with patella Pb. They suggested the observed interaction of bone Pb with estrogen status in determining PbB supports the hypothesis that increased bone resorption, as occurs postmenopausally because of decreased estrogen production, results in heightened release of bone Pb stores into blood.
Tsaih et al. (1999) Boston, MA 1991-97	Normative Aging Study. Aim to evaluate hypothesis that bone and erythrocyte Pb make independent contributions to urine Pb excreted over 24 hour. Urine used as a proxy for plasma Pb. Age range 53-82 yr (n = 71). Generalized additive model.	PbB: 5.94 (±3.0), tibia 21.7 (±10.9), patella 31.1 (±15.1), urinary Pb 5.69 (±1.9) μg/day. Both erythrocyte Pb and bone Pb variables remained independently and significantly associated with urinary Pb.	Finding suggests that bone influences plasma Pb in a manner that is independent of the influence of erythrocytic lead on plasma Pb. Reinforces superiority of bone Pb over PbB in predicting some chronic forms of toxicity may be mediated through bone's influence on plasma Pb. Urinary lead might be useful as a proxy for plasma Pb.
Wright et al. (2004) Boston, MA 1991-97	Normative Aging Study. Aim to evaluate if hemochromatosis gene (HFE) was associated with body lead burden. Tibia and patella bone Pb. DNA samples genotyped. Multivariate linear regression analyses.	Of 730 subjects, 94 (13%) carried the C282Y variant and 183 (25%) carried the H63D variant. In multivariate analyses that adjusted for age, smoking, and education, having an HFE variant allele was an independent predictor of significantly lower patella Pb levels ($p < 0.05$).	Suggested that HFE variants have altered kinetics of Pb accumulation after exposure and these effects may be mediated by alterations in Pb toxicokinetics via iron metabolic pathways regulated by the HFE gene product and body iron stores.

Table AX4-8 (cont'd). Bone Lead Studies of Menopausal and Middle-aged to Elderly Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
United States (cont'd)			
Lin et al. (2004) Boston, MA 1999-2000	Community Lead Study. Measured PbB and bone Pb levels among minority individuals from Boston. Compared with earlier studies of predominantly white subjects, the 84 volunteers in this study (33:67 male to female ratio; 31-72 yrs of age) had similar educational, occupational, and smoking profiles and mean blood, tibia, and patella Pb levels. LOWESS smoothing curves. Multiple linear regression analyses to predict blood, tibia and patella Pb.	<45 yr (n = 28): PbB 2.0 (±1.2), tibia 8.3 (±8.4), patella 8.9 (±14.3) 46-60 yr (n = 41): PbB 2.8 (±1.7), tibia 10.8 (±11.5), patella 11.8 (±11.4) 61-75 yr (n = 15): PbB 5.3 (±3.2),tibia 21.7 (±8.6), patella 30.9 (±15.7) Slopes of the univariate regressions of blood, tibia, and patella lead versus age were 0.10 µg/dL/yr ($p < 0.001$), 0.45 µg/g/yr ($p < 0.001$), and 0.73 µg/g/yr ($p < 0.001$), respectively.	Analyses of smoothing curves and regression lines for tibia and patella Pb suggested an inflection point at 55 yr of age, with slopes for subjects ≥55 yr of age that were not only steeper than those of younger subjects but also substantially steeper than those observed for individuals >55 yr of age in studies of predominantly white participants.
Berkowitz et al. (2004) New York 1994-99	Longitudinal study of 91 premenopausal and perimenopausal women aged ≥ 30 yrs of age from New York who were undergoing surgical menopause (baseline; n 84) to determine if bone Pb values decrease and PbB values increase during menopause. Tibia Pb concentrations measured at baseline, 6 mo (70) and 18 mo (62) post surgery.	Baseline: Median PbB 2.5 (0.3, 11.7), tibia 6.1 (−22.2, 36.4) 6 mo: PbB 3.2 (0.4, 12.0), tibia 6.8 (−14.2, 29.0) 18 mo: PbB 3.1 (0.5, 9.1), tibia 5.8 (−15.4, 24.2)	Marginal decline in tibia Pb values between 6 and 18 mo post surgery for women who took estrogen replacement therapy (ERT) but not for those who did not take ERT. They concluded that there was no substantial mobilization of Pb (from the tibia) during menopause but common ERT use may have masked this effect, the amounts of Pb released were too low to detect in blood, or the numbers of subjects was too small to detect an effect.
Schafer et al. (2005) Baltimore, MD	Evaluated the relations among PbB, tibia Pb, and homocysteine levels by cross-sectional analysis among subjects in the Baltimore Memory Study, a longitudinal study of 1, 140 randomly selected residents in Baltimore, MD, aged 50-70 yr and 66.0% female, 53.9% white, and 41.4% black or African American. Multiple linear regression analyses.	PbB 3.5 (±2.4) µg/dL, tibia 18.9 (±12.5) µg/g, homocysteine 10.0 (±4.1) μmol/L. Tibia lead was modestly correlated with PbB (Pearson $r = 0.12$, $p < 0.01$) but was not associated with homocysteine levels.	Suggested that homocysteine could be a mechanism that underlies the effects of lead on the cardiovascular and central nervous systems, possibly offering new targets for intervention to prevent the long-term consequences of lead exposure.

Table AX4-8 (cont'd). Bone Lead Studies of Menopausal and Middle-aged to Elderly Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
United States (cont'd)			
Kosnett et al. (1994) Dickson City, PA	Aim to determine the influence of demographic, exposure and medical factors on the bone Pb concentration of subjects with environmental Pb exposure. 101 subjects (49 males, 52 females; aged 11 to 78 yrs) recruited from 49 of 123 households geographically located in a suburban residential neighborhood.	Log-transformed bone Pb highly correlated with age ($r = 0.71$; $p \leq 0.0001$).	Bone Pb showed no significant change up to age 20 yr, increased with the same slope in men and women between ages 20 and 55 yr, and then increased at a faster rate in men older than 55 yr.
Popovic et al. (2005) Bunker Hill, ID 1994	108 former female smelter employees and 99 referents to assess the PbB versus bone Pb relationship.	Exposed: PbB 2.73 (±2.39), tibia 14.4 (±0.5) Referents: PbB 1.25 (±2.10), tibia 3.22 (±0.50) Pb concentrations in tibia and blood significantly higher in the exposed group. Endogenous release rate (µg Pb per dL blood/ µg Pb/g bone) in postmenopausal women was double the rate found in premenopausal women (0.132 ± 0.019 versus 0.067 ± 0.014).	Higher tibia bone Pb (and PbB) was associated with use of estrogen (present or former) in both the whole referent group and postmenopausal women in the referent group.
Canada			
Webber et al. (1995) Canada Unknown	Tested hypothesis that women on hormone replacement therapy should have higher bone Pb content and lower plasma Pb as hormone replacement therapy would suppress the transfer of endogenous Pb to the circulation. 56 women, some using hormone replacement therapy over ~4 yrs.	Low dose hormone replacement therapy (n = 15): PbB 4.08 (±1.60), tibia 19.37 (±8.62), calcaneus 24.02 (±10.88) Moderate dose hormone replacement therapy (n = 11): PbB 5.22 (±3.36), tibia 16.80 (±11.68), calcaneus 23.83 (±14.18) Calcium only (n = 22): PbB 4.6 (±1.59), tibia 11.13 (±6.22), calcaneus 21.12 (±13.55)	Women not taking hormones had significantly lower Pb values in cortical bone compared to all women on hormone replacement therapy ($p = 0.007$). Showed higher tibia Pb levels but no increase in calcaneus Pb level or decrease in PbB.

Table AX4-8 (cont'd). Bone Lead Studies of Menopausal and Middle-aged to Elderly Subjects

Reference, Study Location, and Period	Study Description	Lead Measurement (SD or range) PbB in µg/dL, Bone Pb in µg/g Bone Mineral	Findings, Interpretation
Mexico			
Garrido-Latorre et al. (2003) Mexico City 1995	Aim was to examine the relationship of PbB levels to menopause and bone lead levels in 232 perimenopausal and postmenopausal women from Mexico City. Measured bone mineral density in addition to bone Pb. Information regarding reproductive characteristics and known risk factors for PbB was obtained using a standard questionnaire by direct interview. Mean age of the population was 54.7 yrs (±9.8). Linear regression analyses.	PbB 9.2 (±4.7), tibia 14.85 (±10.1), patella 22.73 (±14.9). A change of 10 µg Pb/g bone mineral in postmenopausal subjects was associated with an increase in PbB of 1.4 µg/dL, whereas a similar change in bone lead among premenopausal women was associated with an increase in PbB of 0.8 µg/dL.	Found that postmenopausal women using hormone replacement therapy had lower PbB levels and higher tibia and patella bone Pb levels than non-users; patella Pb explained the greatest part of variations in PbB. Found no association with PbB levels and did not describe any relationships between bone lead and bone density.
Australia			
Gulson et al. (2002) Sydney, Australia 2000	Environmentally exposed females (n = 7) and males (n = 3) aged 44-70 yr. Treated for 6 mo with the bisphosphonate alendronate. PbB and isotopic ratios measured by TIMS for 6 mo prior to treatment and 12 mo post-treatment. Bone mineral density and bone markers including NT_x measured.	Found a decrease in PbB concentrations and changing PbB isotopic composition in the direction predicted during treatment. Upon cessation of treatment, PbB increased and the isotopic compositions changed.	Results consistent with changes in bone remodeling associated with bisphosphonate use.

PbB = blood lead.

Table AX4-9. Lead in Deciduous Teeth from Urban and Remote Environments

Reference, Study Location, and Period	Study Description	Lead Measurement	Findings, Interpretation
Canada			
Tsuji et al. (2001) Ontario, Canada	Dentine chips from schoolchildren living in a remote area.	Mean value of 9.2 µg/g dry weight (n = 61)	Attributed the high values to consumption of lead contaminated game meat.
Europe			
Tvinnereim et al. (1997) Norway 1990-94	2,746 deciduous whole teeth.	Mean 1.27 ± 1.87 µg/g of dry tooth substance	Observed an ~50% reduction in lead concentrations since the 1970s.
Lyngbye et al. (1991) Denmark	In 2,033 teeth from 1, 848 children.	Geometric mean for the largest group from Arhus to be 8.4 µg/g (wet weight) with similar values from Copenhagen suburbs with a secondary lead smelter (9.6 µg/g) and a lead battery factory (9.9 µg/g).	Concluded that automobile exhausts and indirect occupational exposure were important sources for the lead in dentine.
Gil et al. (1996) Coruna, Spain	220 whole deciduous and permanent teeth (one per subject).	Permanent teeth showed higher mean values (13.1 ±1.1 µg/g) than deciduous teeth (4.0 ± 1.1 µg/g)	Found no gender differences.
Nowak and Chmielnicka (2000) Poland	Compared permanent teeth from two cohorts, one from the highly polluted Katowice district and a control town of Beskid.	In the control teeth, they observed decreases in lead for incisors (41.8 µg/g) to canines (37.5 µg/g) to molars (35.3 µg/g) to premolars (32.0 µg/g). However, there was no difference in the mean values for the two centers: Katowice 36.5 ± 16.3 µg/g and Beskid 36.3 ± 11.5 µg/g.	These values are very high compared with most other studies.

Table AX4-9 (cont'd). Lead in Deciduous Teeth from Urban and Remote Environments

Reference, Study Location, and Period	Study Description	Lead Measurement	Findings, Interpretation
Mexico			
Hernandez-Guerrero et al. (2004) Mexico City	100 healthy deciduous teeth collected from 2 to 13 yr old children.	Higher mean concentrations of lead in the 10-13 yr old group (7.7 µg/g) than in other age groups and the mean concentrations were higher in girls (7.3 µg/g) than boys (6.3 µg/g).	No association between pollution intensity and tooth lead.
Frank et al. (1990) Alsace, Mexico	Circular biopsies 500 μm in diameter punched in the vertical sections of the crown and cervical third of each root. The age of the European subjects ranged from 10 to 80 yrs in Europe and 12 to 29 yrs in Mexico City. Energy-dispersive X-ray fluorescence method to compare lead in enamel and dentine of premolars and permanent molars.	Compared with the European values, there were ~6 times higher inner coronal dentine and 7 to 9 times higher pulpal root dentine concentrations for samples from Mexico City.	The authors found no significant difference in the relationship between traffic and mean lead values for enamel and dentine in the European communities but a significantly higher lead concentration in relation to age. The differences were attributed to traffic exposure.
Asia			
Karakaya et al. (1996) Ankara, Turkey	103 whole deciduous teeth from primary school aged children aged 7 to 12 yrs.	Significant differences in lead for urban (4.99 ± 0.46 µg/g dry weight) compared with suburban children (1.69 ± 0.25 µg/g).	

Table AX4-10. Lead in Deciduous Teeth from Polluted Environments

Reference, Study Location, and Period	Study Description	Lead Measurement	Findings, Interpretation
Europe			
Begerow et al. (1994) Germany 1991	790 children aged 6 yrs old living in urban and rural areas in western and eastern Germany. Incisors sampled.	Lead levels of 1.50 to 1.74 µg/g from the western sector and from 1.51 to 2.72 µg/g in the eastern sector.	Major decrease (40-50%) since 1976.
Cikrt et al. (1997) Czech Republic	Compared tooth (n = 162) and PbB levels in children living at various distances from a lead smelter.	Significant difference in the mean tooth lead for children from the most contaminated zone less than 0.5 km from the smelter (6.44 µg/g; n = 13) and those >5 km from the smelter (1.45 µg/g; n = 36). PbB levels varied from 15.42 µg Pb/100 ml (n = 6; 95% CI: 7.17, 33.17) close to the smelter to 4.66 µg/100 ml (n = 165, 95% CI: 4.30, 5.04) at larger distances.	No descriptions of the teeth type were available.
Australia			
Gulson (1996) Broken Hill, Australia	36 exposed and nonexposed children from Broken Hill lead-zinc mining community. Sectioned teeth into mainly enamel (incisal section) and mainly dentine (cervical section). Lead isotope ratios and lead concentrations by TIMS with isotope dilution.	For subjects with low exposure (n = 13), lead concentrations in the incisal section ranged from 0.4 to 3.5 µg/g with a mean and standard deviation of 1.2 ± 0.8 µg/g (n = 13). For the cervical sections in low exposure children, the values ranged from 0.8 to 8.3 and mean 3.7 ± 2.4 µg/g. For subjects with high exposure (n = 23), lead concentrations in the incisal section ranged from 1.0 to 8.9 µg/g with a mean and standard deviation of 2.6 ± 1.8 µg/g. For the cervical sections in high exposure children the values ranged from 1.5 to 31.5 µg/g and mean 13.7 ± 8.0 µg/g.	The isotopic results in dentine were interpreted to reflect an increased lead exposure from the lead-zinc-silver orebody during early childhood, probably associated with hand-to-mouth activity.
Gulson et al. (2004) Lake Macquarie, Australia	10 children from six houses in a primary zinc-lead smelter community at North Lake Macquarie, New South Wales, Australia. Sectioned deciduous teeth compared with environmental samples. Lead isotope ratios and lead concentrations by TIMS with isotope dilution.	PbB levels in the children ranged from 10 to 42 µg/dL and remained elevated for a number of years. Median lead level in the enamel section of the teeth was 2.3 µg/g with a range from 0.6 to 7.4 µg/g; in dentine the median value was 5.3 µg/g with a range from 1.4 to 19.9 µg/g.	Approximately 55 to 100% of lead could be derived from the smelter.

PbB = blood lead.

Table AX4-11. Summary of Selected Measurements of Urine Lead Levels in Humans

Reference, Study Location, and Period	Study Description	Urine Lead Measurement			Comment
United States					
CDC (2005) U.S. 1999-2002	Design: national survey (NHANES IV) stratified, multistage probability cluster design Subjects: children and adults (≥6 yrs, n = 5140) in general population Biomarker measured: urine lead Analysis: ICP-MS	Units: µg/g creatinine Geometric mean (95% CI)			Geometric mean PbB concentrations in age strata ranged from 0.94 to 1.51 µg/dL.
		Age (yr)	**1999-2000**	**2001-2002**	
		≥6:	0.72 (0.70, 0.74)	0.64 (0.60, 0.68)	
		n:	2465	2689	
		6-11:	1.17 (0.98, 1.41)	0.92 (0.84, 1.00)	
		n:	340	368	
		12-19:	0.50 (0.46, 0.54)	0.40 (0.38, 0.43)	
		n:	719	762	
		≥20:	0.72 (0.68, 0.76)	0.66 (0.621, 0.70)	
		n:	1406	1559	
		Males:	0.72 (0.68, 0.76)	0.64 (0.61, 0.67)	
		n:	1227	1334	
		Females:	0.72 (0.68, 0.76)	0.64 (0.59, 0.69)	
		n:	1238	1355	
Schwartz et al. (1999, 2000b) U.S. 1993-1997	Design: prospective Subjects: adult male (n = 543) former TEL manufacture workers (age range: 42-74 yrs) Biomarker measured: DMSA (10 mg/kg)-provoked urine lead Analysis: GFAAS	Units: µg/4 hr Arithmetic mean (SD): ≥2 yr exposure: 17.1 (15.7) <2 yr exposure: 20.4 (17.9)			Arithmetic mean (SD) PbB (µg/dL) was 5.0 (2.8) for workers exposed ≥2 yr and 2.8 (1.9) for workers exposed <2yr. PbB was strongest predictor or DMSA-provoked urine lead. Arithmetic mean (SD) tibia lead (µg/g, XRF) was 15.6 (9.8) for workers exposed ≥2 yr and 12.1 (7.7) for workers exposed <2yr.
Rabinowitz et al. (1976) New York NR	Design: experimental study Subjects: adult (n:5) males, age range 25-53 yrs, ingested 300 µg Pb/day (~50% as ^{204}Pb) for 10-210 days Biomarker measured: urine lead Analysis: MS	Units: µg/day Arithmetic mean (range): 36 (36-41)			Arithmetic mean (range) PbB (µg/dL) was 19.4 (16.7-25.1). Blood-to-urine clearance estimate was 0.19 (range 0.15-0.23) L/day (from Diamond, 1992).

Table AX4-11 (cont'd). Summary of Selected Measurements of Urine Lead Levels in Humans

Reference, Study Location, and Period	Study Description	Urine Lead Measurement	Comment
United States (cont'd)			
Berger et al. (1990) Ohio 1983-1986	Design: cross-sectional, convenience sample Subjects: children (n = 39), age range not reported. Biomarker measured: timed urine lead Analysis: AAS	Units: µg/day range: 5-70	PbB range was 22-55 µg/dL. Blood-to urine clearance estimate was 0.07 L/day (from Diamond, 1992).
Europe			
Chamberlain et al. (1978) United Kingdom 1975-1976	Design: experimental Subjects: adult males (n = 6), intravenous injection of ^{203}Pb tracer Biomarker: urinary lead clearance Analysis: gamma spectrometer (^{203}Pb)	Units: L/day Arithmetic mean (range) Blood-to-urine: 0.09 (0.08-0.10) Plasma-to-urine: 20	
Brockhaus et al. (1988) Germany 1982-1986	Design: cross-sectional Subjects: children (n = 184), age range 4-11 yrs residing in 2 areas impacted by smelting operations Biomarker measured: urine lead Analysis: GFAAS	Units: µg/g creatinine Geometric mean (GSD, range) Stolberg (n = 106): 9.6 (2.3, 0.2-43.0) Dortmund (n = 78): 6.7 (2.0, 1.6-41.0)	Geometric mean PbB levels were ~7 µg/dL.
Koster et al. (1989) Germany NR	Design: cross-sectional Subjects: adult (n = 46, 40 males) hospital workers, age range 20-78 yr. Biomarker measured: urine lead Analysis: GFAAS	Units: µg/24 hr-1.73 m^2 (adult body surface area) Arithmetic mean (range): 6.8 (2.3-18.9)	Arithmetic mean (range) PbB (µg/dL) was 7.6 (2.6-18.7). Blood-to-urine clearance estimate was 0.15 L/day (from Diamond, 1992).
Australia			
Gulson et al. (2000) Australia	Design: longitudinal Subjects: women (n = 58) during pregnancy, age range 18-35 yrs Biomarker measured: blood-to-urine clearance Analysis: TIMS	Units: µg/h Arithmetic mean (SD, range): 3.2 (0.8-10.2) Geometric mean: 2.7	Reported blood-to-urine clearance corresponds to ~0.08 L/day.

Table AX4-11 (cont'd). Summary of Selected Measurements of Urine Lead Levels in Humans

Reference, Study Location, and Period	Study Description	Urine Lead Measurement	Comment
Asia			
Araki et al. (1986, 1990) Japan NR	Design: cross-sectional Subjects: adult (n = 19) male, gun metal foundry workers, age range 34-59 yr. Biomarker measured: urine lead Analysis: AAS	Units: µg/24 hr Arithmetic mean (range): 94 (37-171)	Arithmetic mean plasma concentration was 0.67 µg/dL (range 0.37-0.92). Plasma-to urine clearance estimate was 22 L/day. Blood-to-urine clearance estimate was 0.33 L/day (from Diamond, 1992).
Lee et al. (1990) Korea NR	Design: cross-sectional Subjects: adults (n = 95) male workers in lead smelting, battery manufacture, PVC-stabilizer manufacture facilities, age range: 19-64 yrs; reference subjects (n = 13), age range 22-54 yr. Biomarker measured: DMSA (10 mg/kg)-provoked urine lead Analysis: GFAAS	Units: µg/4 hr Arithmetic mean (SD, range) Lead workers: 288.7 (167.7, 32.4-789) Reference: 23.7 (11.5, 10.5-43.5)	Arithmetic mean (SD, range) PbB concentration (µg/dL) was 44.6 (12.6, 21.4-78.4) in lead workers and 5.9 (1.2, 4.0-7.2) in reference subjects. PbB was strongest predictor or DMSA-provoked urine lead.
Schwartz et al. (2000a), Lee et al. (2001) Korea 1997-1999	Design: Cross-sectional Subjects: Adult lead (inorganic) workers (n = 798, 634 males), age range 18-65 yrs. Biomarker measured: MSA (10 mg/kg)-provoked urine lead Analysis: GFAAS	Units: µg/4 hr Arithmetic mean (SD, range) 186 (208, 4.8-2100)	Arithmetic mean (SD, range) PbB (µg/dL) was 32.0 (15, 4-86). PbB was strongest predictor or DMSA-provoked urine lead. Arithmetic mean (SD, range) tibia lead (µg/g, XRF) was 37.1 (40.4, -7 to 338).

AAS - atomic absorption spectroscopy; PbB = blood lead; ET-AAS - electro-thermal atomic absorption spectrometry; GFAAS - graphite furnace atomic absorption spectroscopy; ICP-AES - inductively coupled plasma/atomic emission spectroscopy; ICP-MS - inductively coupled plasma-mass spectrometry; MS - mass spectrometry; NR - not reported; Pct - percentile; TEL -tetraethyl lead; TIMS - thermal ionization mass spectrometry.

Table AX4-12. Summary of Selected Measurements of Hair Lead Levels in Humans

Reference, Study Location, and Period	Study Description	Hair Lead Measurement	Comment
United States			
DiPietro et al. (1989) GA, SC, TX, VA 1976-1980	Design: Cross-sectional (random sample from NHANES II, HHANES Stands) Subjects: adults (n = 270, 200 males; age range: 20-73 yrs) from general population Biomarker measured: Hair lead Analysis: ICP-AES	Units: µg/g Geometric mean (10-90th Pct range) 2.42 (<1.0-10.8)	Hair lead level varied with hair treatment (e.g., shampoo, coloring).
Tuthill (1996) MA NR	Design: Cross-sectional Subjects: children (n = 277, 141 males, age range 6.5-7.5 yrs) Biomarker measured: Hair lead Analysis: ICP-AES	Units: µg/g <0.1-0.9 : 13.5% 1-1.9: 40.8% 2-2.9: 25.6% 3-3.9: 9.0% ≥4: 11.1%	Study examined associations between hair lead levels and attention-deficit behaviors.
Europe			
Annesi-Maesano et al. (2003) France 1985, 1991-1992	Design: cross-sectional Subjects: mother (mean age 29 yr)-infant pairs (n:374) Biomarker measured: hair lead Analysis: ICP-AES	Units: µg/g Arithmetic mean (SD): Infant: 1.38 (1.26) Mother: 5.16 (6.08)	Mean PbB concentrations were 96 µg/dL (SD 58) in mothers and 67 (SD 48) in infant cord blood. Infant hair-cord PbB correlation (Spearman, r) was 0.21 ($p < 0.01$).
Drasch et al. (1997) Germany 1993-1994	Design: cross-sectional Subjects: adults (n = 150, 75 males; age range: 16-93 yrs) from general population with no known occupational exposure Biomarker measured: hair lead (post-mortem) Analysis: ET-AAS	Units: µg/g Median (range): 0.76 (0.026-20.6) 25-75th Pct range: 0.45-1.48	Median PbB (µg/dL) was 2.8 (range <0.9-16.1). Median temporal bone lead was 2.84 µg/g (range 0.25-22.3), Hair lead correlation (Spearman r) was 0.35 ($p < 0.001$) for blood, 0.10 ($p > 0.05$) for temporal bone, and 0.16 ($p > 0.05$) for body burden.0.512 for liver ($p = 0.003$) and 0.57 ($p = 0.001$) for kidney.
Gerhardsson et al. (1995b) Sweden NR	Design: cross-sectional Subjects: adult male smelter workers (n = 32) and referents (n = 10) Biomarker measured: hair lead (post-mortem) Analysis: XRF	Units: µg/g Median (range): Active workers: 8.0 (1.5-29,000) Retired workers: 2.6 (0.6-9.3) Reference: 2.05 (0.3-96)	Based on reported a cumulative annual PbB index of 1,374 µg/dL and average duration of employment of 31.4 yrs, average PbB may have been ~44 µg/dL in workers. Hair lead correlation (Spearman, r).

Table AX4-12 (cont'd). Summary of Selected Measurements of Hair Lead Levels in Humans

Reference, Study Location, and Period	Study Description	Hair Lead Measurement	Comment
Europe (cont'd)			
Esteban et al. (1999) Russia 1996	Design: cross-sectional Subjects: children (n = 189, 110 females; age rage 1.9-10.6 yr) living in the vicinity of lead battery and leaded glass manufacture facilities. Biomarker measured: hair lead Analysis: ICP-AES	Units: ng/g Geometric mean (range): 5.4 (1-39.2) 90^{th} Pct: ~15	Geometric mean PbB was 8.5 µg/dL (range 3.1-35.7); log PbB = 1.44 + 0.35 (log hair) + 0.24 (gender), $r^2 = 0.20$.

AAS - atomic absorption spectroscopy; PbB = blood lead; ET-AAS - electro-thermal atomic absorption spectrometry; GFAAS - graphite furnace atomic absorption spectroscopy; HHANES - Hispanic Health and Nutrition Examination Survey; ICP-AES - inductively coupled plasma/atomic emission spectroscopy; ICP-MS - inductively coupled plasma-mass spectrometry; NR - not reported; Pct - percentile.

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