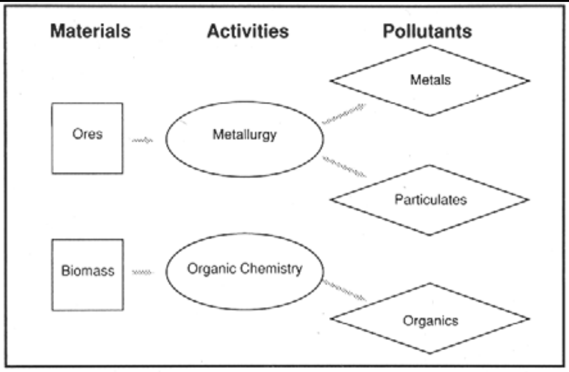


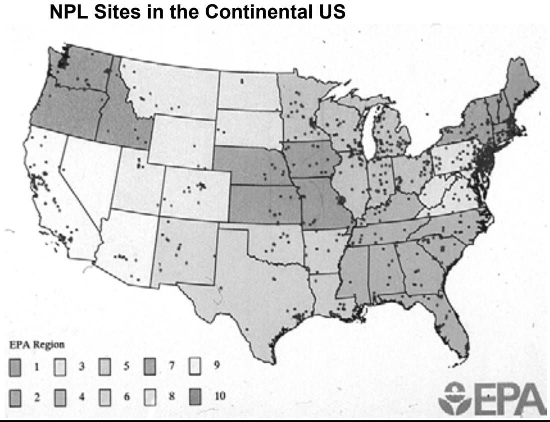
Figure 26.1 Relationship between industrial activities and environmental releases.

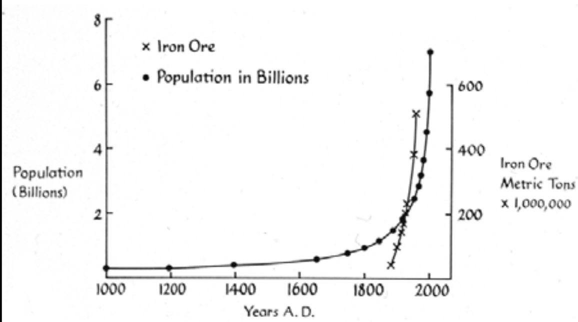
Spatial

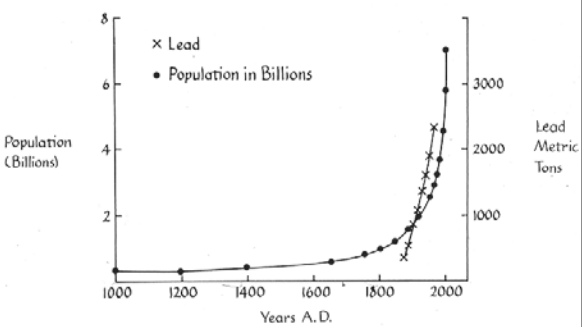
Temporal		
	Local *Short-lived*	Global *Short-lived*
	Local *Persistent*	Global *Persistent*

Figure 26.2 Patterns in environmental fate of trace pollutants.

NPL Sites in the Continental US







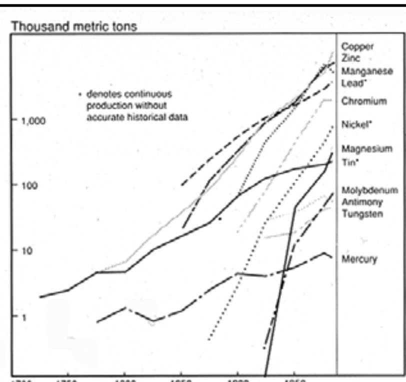


Figure 26.3 Annual worldwide production of selected metals from 1700 to 1983. Data from Schmitz 1979 and Minerals Yearbook 1984.

Domestic demand for minerals has increased at an astonishing rate. More metal and mineral products have been used in the past 50 years than were used in the entire previous history of the world.

Examples of Occupational Diseases in Miners and/or Smelter Workers

Arsenic:	Excess lung cancer among smelter workers exposed to arsenic trioxide
Asbestos:	Mesothelioma, a rare form of lung cancer. The risk is much higher in miners who smoke, as these two carcinogens act in a synergistic manner.
	Also, "asbestosis" (pulmonary fibrosis)
Chromium:	Lung cancer with exposure to the more insoluble hexavalent Cr; not with trivalent Cr
Coal:	Pneumoconiosis and silicosis ("Miners' Asthma")
Lead:	Lead poisoning, with colic, kidney dysfunction, anemia, neurologic effects (e.g., peripheral neuropathy, insomnia, irritability, etc.)
Mercury:	Mercury poisoning, with neurologic and renal dysfunction
Nickel:	Nasal cancer in refinery workers
Uranium:	Lung cancer from its decay products, including its polonium and radon "daughters." The risk is higher among miners who smoke.

THE THIRTY-NINE DISASTERS*

Year	Mine	Place	No. of Deaths	Cause
United States				
1960	Pine Creek No. 22	West Virginia	18	Mine Fire
1961	Viking	Indiana	22	Explosion
1962	Blue Blaze No. 2	Illinois	11	Explosion
1962	Robena No. 3	Pennsylvania	37	Explosion
1963	Compass No. 2	West Virginia	22	Explosion
1963	Carbon Fuel No. 2	Utah	9	Explosion
1965	Kline No. 2a	Tennessee	5	Explosion
1965	Mars No. 2	West Virginia	7	Fire/ Explosion
1965	Dutch Creek No. 2	Colorado	9	Explosion
1966	Dora No. 2	Pennsylvania	5	Suffocation
1966	Siltix	West Virginia	7	Explosion
1968	River Queen No. 1	Kentucky	9	Explosion
1970	Finley Nos. 15-16	Kentucky	38	Explosion
1972	Buffalo Creek	West Virginia	125	Tip Slide
1972	Blacksville No. 1	West Virginia	9	Fire
1972	Itmann No. 3	West Virginia	5	Explosion
1977	P and P No. 2	Virginia	4	Explosion
1978	Moss No. 3	Virginia	5	Inundation (blackdamp)
1981	Dutch Creek No. 1	Colorado	15	Explosion
United Kingdom				
1960	Cardowan	Lanarkshire	4	Explosion
1960	Six Bells	Monmouthshire	45	Explosion
1962	Hampton Valley	Lancashire	19	Explosion
1962	Tower	Glamorganshire	9	Explosion
1965	Cambrian	Glamorgan	31	Explosion
1966	Merthyr Vale	Aberfan	144	Tip Slide
1967	Michael	Fife	9	Fire

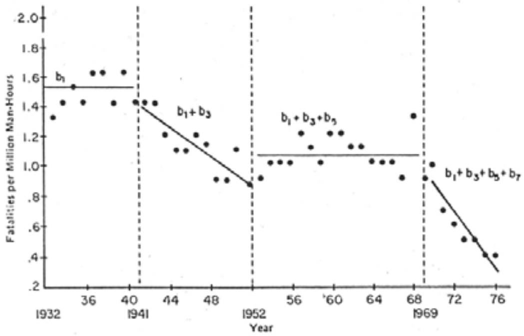
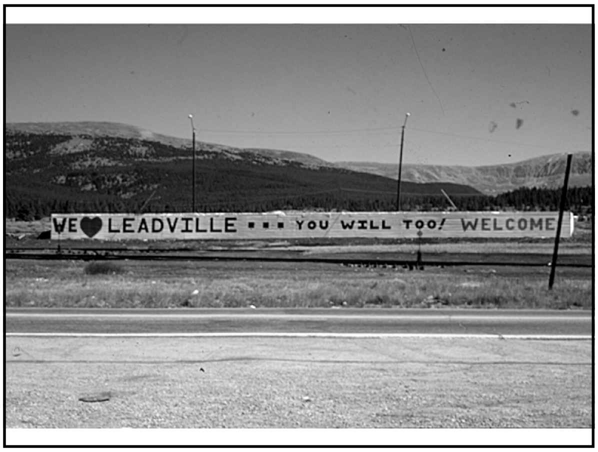
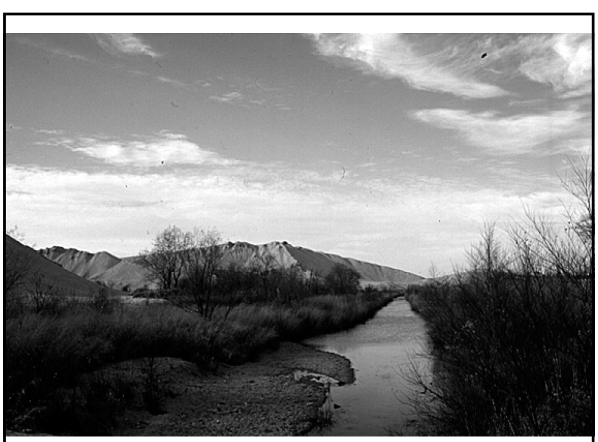
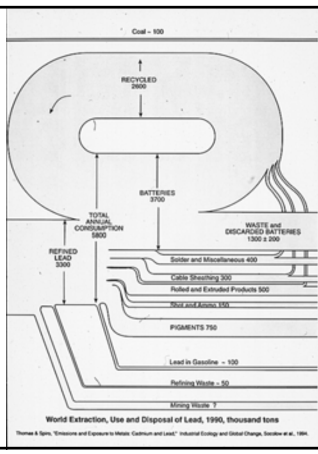


Figure 3. Multiple interrupted time-series analysis of the U.S. coal mine fatality rate (1932-76). Reprinted with permission from Lewis-Beck and Alford (1980:749).

FIGURE 1. Black lung rally on the Capitol steps, 1977. (Photograph © Earl Dotter).


WE ♥ LEADVILLE ... YOU WILL TOO! WELCOME




Coal ~ 100
RECYCLED 2600
BATTERIES 3700
TOTAL ANNUAL CONSUMPTION 5800
REFINED LEAD 3300
WASTE and DISCARDED BATTERIES 1300 ± 200
Solder and Miscellaneous 400
Cable Sheathing 300
Rolled and Extruded Products 500
Shot and Ammo 150
PIGMENTS 750
Lead in Gasoline ~ 100
Refining Waste ~ 50
Mining Waste ?
World Extraction, Use and Disposal of Lead, 1990, thousand tons
Thomas & Spiro, "Emissions and Exposure to Metals: Cadmium and Lead," Industrial Ecology and Global Change, Socolow et al., 1994.


THE DUTCH BOY PAINTER AT WORK
See to it that he is present at every painting job. His picture is a trade-mark and guaranty for pure white lead

Public Health Then and Now

75

A 'Gift of God'?: The Public Health Controversy over Leaded Gasoline during the 1920s

David Rosner. Ph. D, MSPH, and Gerald Markowitz, Ph.D.

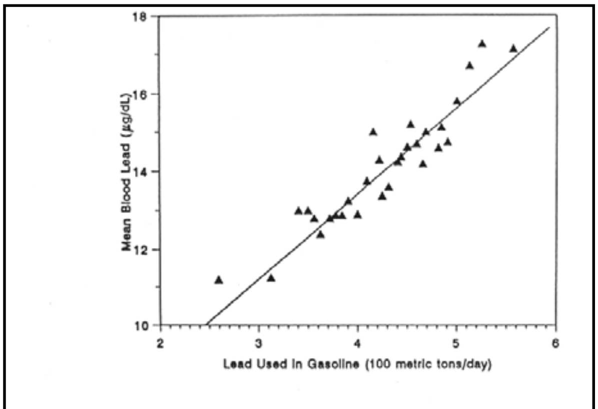
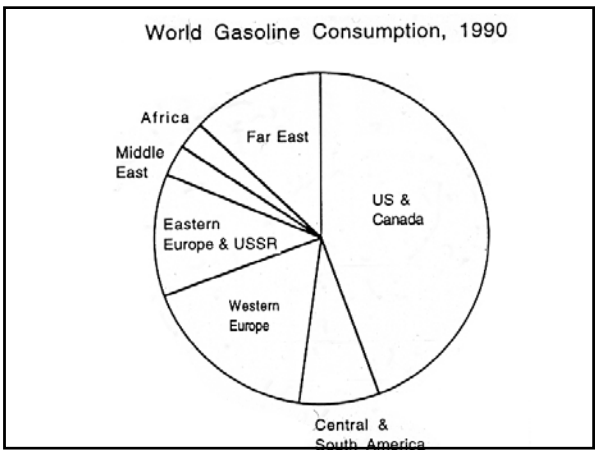
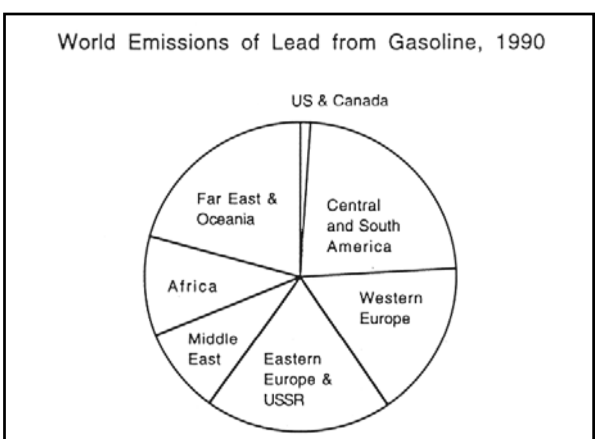
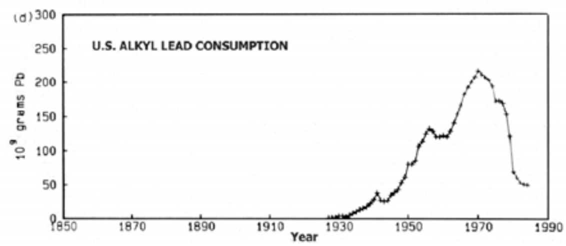


FIGURE 3-8 U.S. mean blood lead vs. lead used in gasoline. Source: Schwartz and Pitcher, 1989.







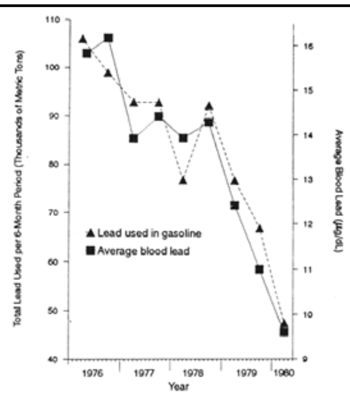
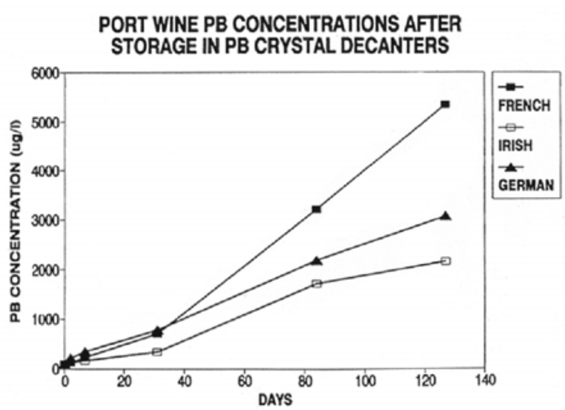


FIGURE 1-3 Lead used in gasoline production and average NHANES II blood lead (Feb. 1976-Feb. 1980). Source: Annest, 1983.



PB CONCENTRATIONS OF BEVERAGES STORED IN DECANTERS

HOME	BEVERAGE	YRS	PB (UG/L)
1	BRANDY	5	7746
2	MADEIRA	5	1402
3	SCOTCH	>3	2587
	PORT	.5-1	203
	ARMAGNAC	.5-1	472
4	BRANDY	5-10	19920
	BRANDY	5-10	21530
	BRANDY	5-10	8390
5	GIN	.5-1	13
	BRANDY	>1	68
	GRAND MAR.	>2	173
	BOURBON	1.5	17
	TEQUILA	1.5	300
	VODKA	1	11

American Journal of Diseases of Children

VOLUME 66 NOVEMBER 1943 NUMBER 5

LATE EFFECTS OF LEAD POISONING ON MENTAL DEVELOPMENT

RANDOLPH K. BYERS, M.D.
AND
ELIZABETH E. LORD, PH.D.
BOSTON

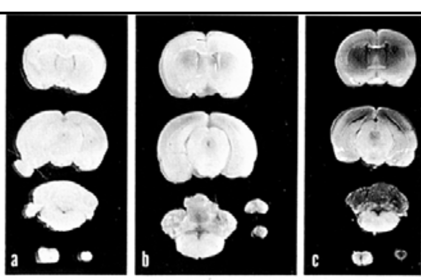


Fig. 1. a) Normal brain of 25-day-old rat 24 hours after intraperitoneal injection of Trypan Blue. No staining of the brain occurred. b) Brain of lead poisoned, 25-day-old rat one hour after the intraperitoneal injection of Trypan Blue. Some staining is visible in the striatum, occipital lobes, cerebellum, and spinal cord. c) Brain of lead poisoned, 26-day-old rat 24 hours after intraperitoneal injection of Trypan Blue. Staining of the brain is more intense and widespread.

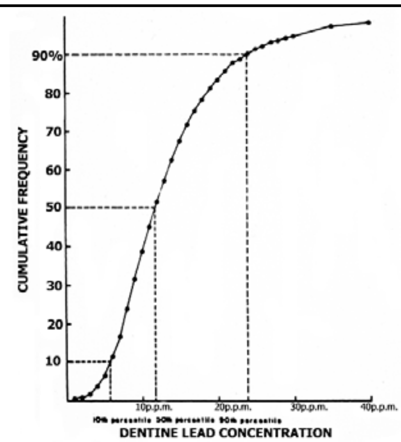
Lampert, Garro and Pentschew

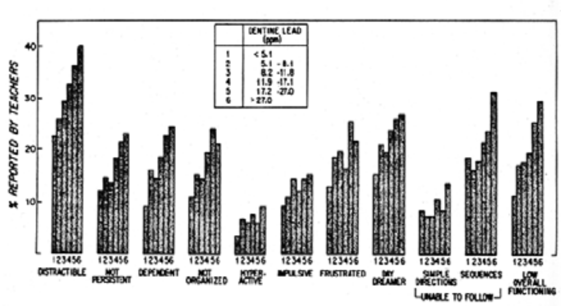
The New England
Journal of Medicine

Volume 300 MARCH 29, 1979 Number 13

DEFICITS IN PSYCHOLOGIC AND CLASSROOM PERFORMANCE OF CHILDREN WITH ELEVATED DENTINE LEAD LEVELS

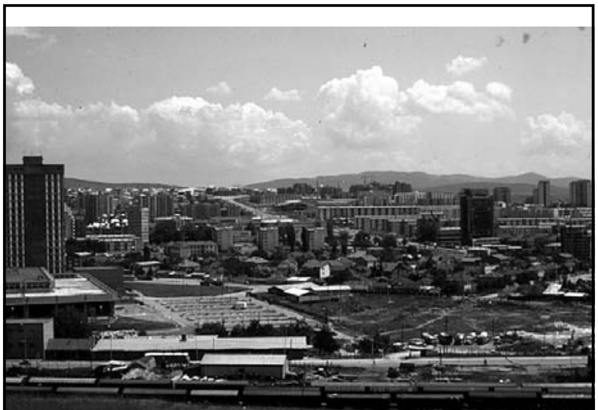
Herbert L. Needleman, M.D., Charles Gunnoe, Ed.D., Alan Leviton, M.D., Robert Reed, Ph.D., Henry Peresie, Ph.D., Cornelius Maher, Ph.D., and Peter Barrett, B.S.

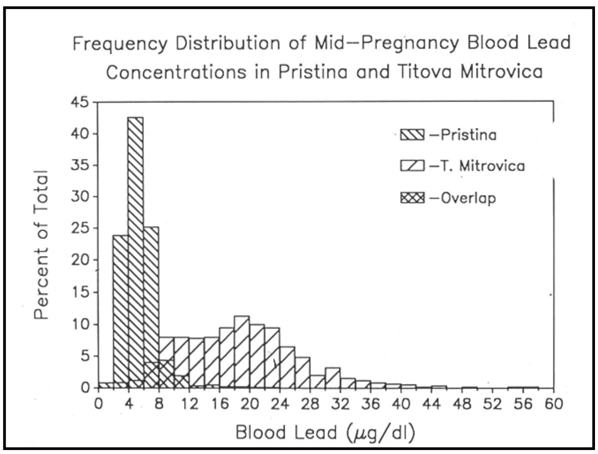




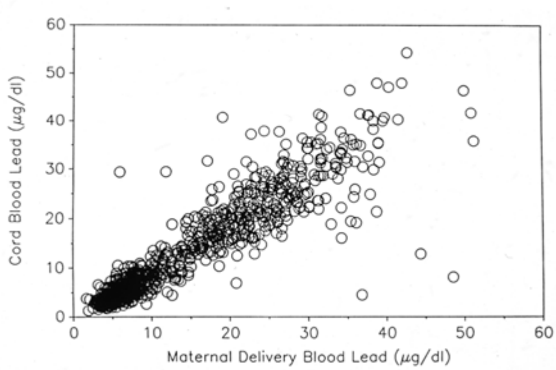
The Yugoslavia Prospective Study of Environmental Lead Exposure

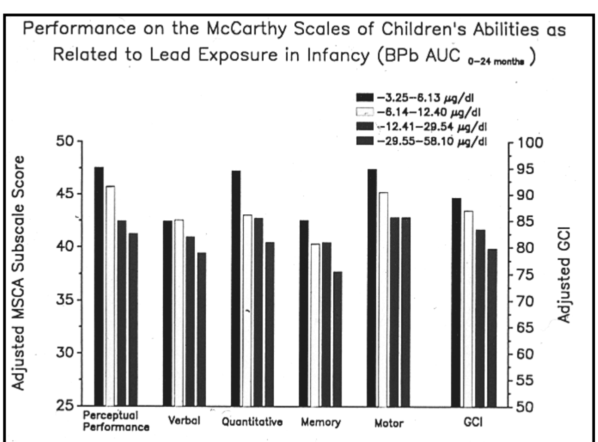
NIEHS R01 ES03460

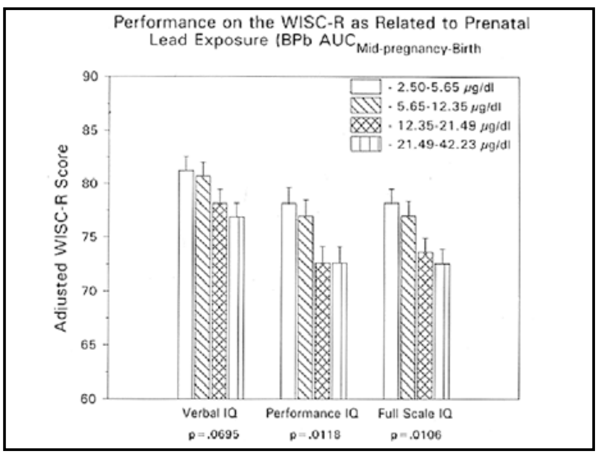




Frequency Distribution of Mid-Pregnancy Blood Lead
Concentrations in Pristina and Titova Mitrovica
Percent of Total
Blood Lead (μg/dl)
—Pristina
—T. Mitrovica
—Overlap
0 5 10 15 20 25 30 35 40 45
0 4 8 12 16 20 24 28 32 36 40 44 48 52 56 60


Cord Blood Lead (μg/dl)
Maternal Delivery Blood Lead (μg/dl)
0
10
20
30
40
50
60


Performance on the McCarthy Scales of Children's Abilities as Related to Lead Exposure in Infancy (BPb AUC 0–24 months)
−3.25−6.13 μg/dl
−6.14−12.40 μg/dl
−12.41−29.54 μg/dl
−29.55−58.10 μg/dl
Adjusted MSCA Subscale Score
Adjusted GCI
Perceptual Performance
Verbal
Quantitative
Memory
Motor
GCI
P
<.0001
.0290
.0003
.0012
.0154
<.0001


Performance on the WISC-R as Related to Prenatal Lead Exposure (BPb AUC Mid-pregnancy-Birth
- 2.50-5.65 µg/dl
- 5.65-12.35 µg/dl
- 12.35-21.49 µg/dl
- 21.49-42.23 µg/dl
Adjusted WISC-R Score
90
85
80
75
70
65
60
Verbal IQ
p=.0695
Performance IQ
p=.0118
Full Scale IQ
p=.0106

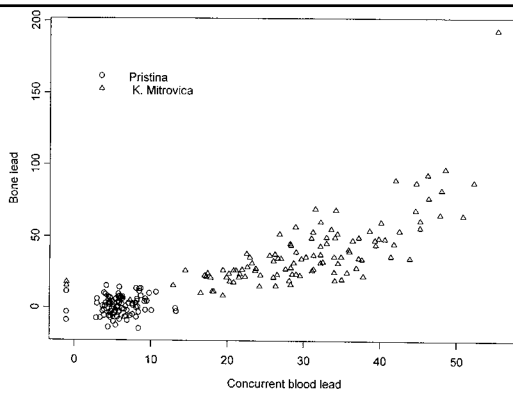
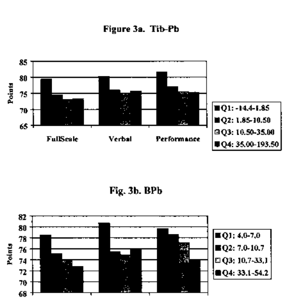


Figure 2. Associations between Concurrent Blood Lead and Bone Lead

Figure 3. Covariate-adjusted IQ Means by Quartiles of Tib-Pb and BPb[1,2]



[1] For 67% of the sample, quartile membership for Tib-Pb and BPb were identical
[2] For BPb, quartile values are hypergeometric means

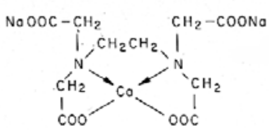
DEFINITIONS OF CHILDHOOD LEAD POISONING

Before the mid-1960s: A BPb level of below 60 $\mu g/dl$ was not considered dangerous enough for intervention.

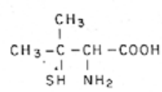
1975: CDC defines level of toxicity in children at $\geq$ 30 $\mu g/dl$.

1985: CDC sets threshold for action at a BPb level of 25 $\mu g/dl$.

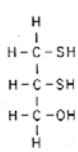
1991: Intervention level revised downwards to 10 $\mu g/dl$ largely because of the results of four ongoing prospective studies.



Calcium Disodium EDTA

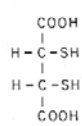


Penicillamine



2,3-Dimercaptopropanol

(BAL)

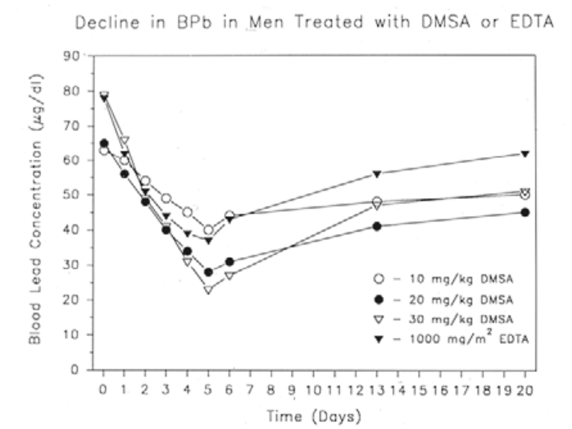


2,3-Dimercaptosuccinic acid

(DMS)

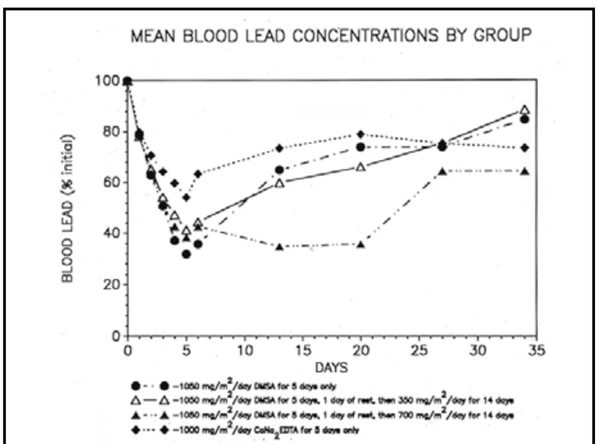
2,3-Dimercaptosuccinic Acid: A New Agent for the Treatment of Lead Poisoning[1]

J. H. GRAZIANO, J. K. LEONG and E. FRIEDHEIM[2]



Dose-response study of oral 2,3-dimercaptosuccinic acid in children with elevated blood lead concentrations

Joseph H. Graziano, PhD, Nancy J. Lolacono, MPH, and Patricia Meyer, MPH
From the Departments of Pharmacology and Pediatrics, Columbia University College of Physicians and Surgeons and Babies Hospital, New York



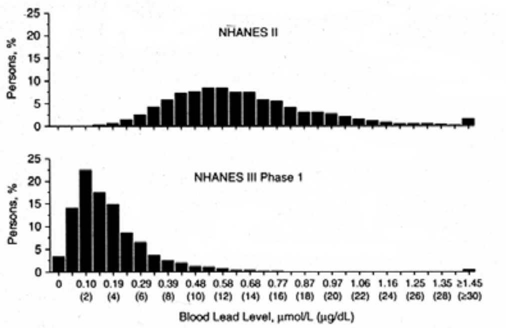


Fig 1.—Blood lead levels for persons aged 1 to 74 years: United States, second National Health and Nutrition Examination Survey (1976 to 1980, top) and phase 1 of the third National Health and Nutrition Examination Survey (1988 to 1991, bottom).