Toxin - Poison.

MEDICAL GRAND ROUNDS

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THE MAGNITUDE OF LEAD CONTAMINATION OF THE BIOSPHERE: POTENITAL DANGERS OF CHRONIC "LOW-LEVEL" LEAD EXPOSURE

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TABLE OF CONTENTS

I. INTRODUCTION TO THE PROBLEM

Over the past 20 years the clinical picture of childhood lead intoxication has shown a progressive change. Until the 1960's clinical interest focused mainly on acute lead encephalopathy in children and on occupational lead poisoning in adults.

Recent public health measures have radically reduced the incidence of severe symptomatic lead poisoning. Now it is the high level of general lead pollution of the biosphere that poses the principal threat to human health, especially to infants and children.

Early childhood screening provided a rather uniform epidemiological picture of symptomatic childhood lead poisoning.

1. Occurred in children 1 to 6 years old with the highest incidence in 1 to 3 year olds, especially blacks.

2. Children were residents of dilapidated housing of the inner city slums.

3. The major source of lead was peeling paint and lead impregnated plaster and putty.

4. Pica was considered a major contributing factor.

A. EVIDENCE OF DECREASING INCIDENCE OF SYMPTOMATIC LEAD POISONING

TABLE 1

Table 1. Incidence of symptomatic lead poisoning.[a]

Year	No. of clinical cases	No. of deaths
1959	66	2
1960	53	4
1961	48	1
1962	44	1
1963	42	3
1964	45	1
1965	32	0
1966	32	1
1967	15	1
1968	13	0
1969	19	0
1970	20	1
1971	11	0
1972	8	0

[a] Data of Klein (*4*) from Baltimore City Health Department.

(McCabe, 1979)

B. EVIDENCE OF WIDESPREAD EXCESSIVE LEAD ABSORPTION

The release of more than 600,000 tons of lead into the environment each year in the US by industry and automobiles has produced a pervasive general contamination of the biosphere. (NAS, 1980) As a result every member of our population is exposed to elevated levels of lead in the air breathed and in the food and water ingested. Evidence of the danger of this general contamination can be seen from the high levels of blood lead found in children during screening studies carried out between 1972 -1978.

a) Of the 2,485,320 children screened between 1972-1978 6.87% (170,738) had evidence of undue lead absorption according to CDC criteria. (Lin-Fu, 1980)

b) Of the 400,000 children screened nationwide in 1978, 6.48% had evidence of undue absorption, but this varied widely from city to city.

Newark	17.7%	Milwaukee	21.7%
Philadelphia	17.8%	St. Louis	19.7%

c) Nationwide in 1978, 2.4% of the screened children were in CDC classes III or IV, requiring immediate therapy even though asymptomatic. Again this varied from city to city.

Newark	6.8%	Milwaukee	8.4%
Philadelphia	5.7%	St. Louis	7.2%

Since lead has become an ubiquitous contaminant of the human environment is should be cause for great concern to the medical community. This talk will be focused in four areas.

1. The sources producing this ubiquitous lead contamination of our biosphere.

2. The magnitude of biospheric lead pollution.

3. The reasons for the increased risk for harmful effects in infants and young children.

4. Evidence indicating that intellectual and behavioral defects are subtle but serious health hazards of chronic "low-level" lead exposure.

II. LEAD POLLUTION OF THE BIOSPHERE

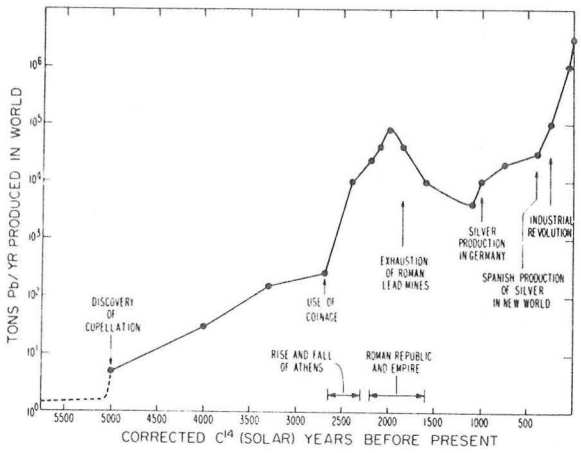
A. SOURCES OF POLLUTION

Lead in the biosphere comes from two major sources, i.e., from natural emissions and from man's many technological activities.

About 2000 tons of lead are emitted into the atmosphere each year from natural sources, mainly volcanic eruptions and wind-blown dust. (Patterson, 1980)

FROM THE EVOLUTIONARY POINT OF VIEW mankind has experienced a rather abrupt and substantial increase in exposure to lead and at present adds about 634,000 tons of lead each year into biosphere as a consequence of his technological activies. (NAS, 1980)

Fig. 1



World lead production during the last 5500 years,

(Patterson, 1980)

Lead was probably used in Egypt as early as 5000-7000 B.C. A technique for cupellation is described in detail in the old testament. For at least the past 4500 years man has been exposed to amounts of lead greater than that naturally occuring in the ecosphere. From then until Roman times man produced only a few hundred tons of lead per year.

However, during Roman times production increased to more than 60,000 tons per year and this lasted for almost 400 years. The per capita output of lead by the Romans was almost as great as Western man today. However, the effects of lead pollution until the industrial revolution in the 1750's were mainly local. (Patterson, 1980)

Greatest contamination of the ecosphere started after 1940.

TABLE 2

Lead Consumption for Selected Uses in the United States[a]

Year	Lead Consumption, Thousands of Tons		
	As White Lead	In Gasoline Additives	In Storage Batteries
1935	80	37[b]	175
1940	66	50[b]	220
1945	36	76	60
1950	36	114	398
1955	18	165	380
1960	8	164	353
1965	8	225	555
1968	6	262	513

(NAS, 1972)

Today about 2,500,000 TONS of lead are produced yearly. Forty percent is used for the storage battery industry alone. Although leaded gasoline (circa 2 gm/gallon) was introduced in 1925, only since 1950 has there been a marked increase in the production and use of leaded gasoline. Lead added to gasoline increased from 37,000 tons in 1935 to 262,000 tons in 1968.

Automotive emissions are the largest single source of atmospheric emission of lead and represent a much more mobile and widely dispersed form of lead pollution than other sources such as discarded batteries. (NAS, 1980)

By 1976 in the USA alone about 217,000 tons of particulate lead were dispersed into the ambient air. (NAS, 1980) About 95% or 190,000 tons were emitted from automobiles using leaded gasoline. In the USA from 90-98% of all airborne lead that can be traced to its source comes from the combustion of leaded gasoline.

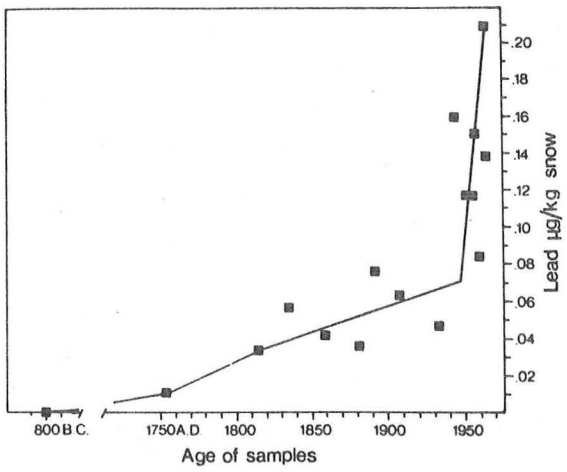
B. EVIDENCE FOR CONTAMINATION OF THE ATMOSPHERE - CHANGES IN LEAD CONCENTRATION OF AMBIENT AIR FROM PRETECHNOLOGICAL TIMES TO THE PRESENT

In order to evaluate the magnitude of environmental contamination of the biosphere it is imperative to know the environmental levels of lead prior to the relatively recent increase in production and use of lead. This chronology of contamination can be ascertained since the magnitude of atmospheric pollution is reflected in the concentrations of lead in different layers of Artic snow. (Murozumi et al, 1969)

Measures of lead in ice layers beneath Artic snow reveals that the layer that fell around 2800 years ago contained 1 ng lead/kg. This is close to the "natural" concentration (0.1 ng lead/kg) expected from natural terrestrial dusts present in the ice. (Patterson, 1980)

Lead content of Artic snow has shown a slow but continuous increase since the 18th century associated with rapid industrialization. However, the steepest increase occurred since the introduction of leaded gasoline and its widespread use because of the worldwide explosion in the number of automobiles. Today, Artic snow concentrations of lead are greater than 200 ng/kg a more than 500 fold increase above the natural levels. (Chow, 1978 in Nriagu)(Patterson, 1965, 1980)

Fig. 2



(Murozumi et al, 1969)

The total natural global emissions of lead orginating mainly from lead rich aerosols from volcanic eruptions, windblown dusts and forest fires is about 2000 tons per year. With a mean residence time of 10 days the estimated natural concentration of lead in the trophosphere is about 0.01 ng Pb/m^3. The expected natural concentration of lead over land areas inhabited by humans in prehistoric times was calculated by Patterson (1965, 1980) to be 0.04 ng/m^3.

Even this very low estimated natural concentration of lead in the atmosphere over land very likely represents an overestimation since in 1974 Chester reported that the air in the Westerly Winds of the North Atlantic had a concentration of lead of only 0.049 ng/m^3.

The concentrations of atmospheric lead in different rural and remote sites over the globe are shown in Table 3. The lowest average concentration in the USA is 8 ng Pb/m^3 found in the White Mountains of California in the Sierra Nevada range at an altitude of 2½ miles.

TABLE 3

Measured Concentrations of Lead in Air at a Variety of Rural and Remote Sites[a]

Site	Lead in Air (ng Pb/m^3)
Rural	
Chadron, Nebraska, 1973-1974	45 (9-90)
Walker Branch Watershed, Tennessee, 1976-1977	107 (63-172)[b]
Rural background, Belgium, 1972	230
Oceanic, New York Bight, 1974	130 (20-300)
Chacaltaya, Bolivia, 1974	37 (18-76)
Puerto Montt, Chile, 1975	12 (9-15)
Remote	
Novaya Zemlya, USSR, 1968-1969	0.23
Jungfraujoch, Switzerland, 1973-1974	8.7 (0.13-25)
Northern Norway, 1971-1972	4.0 (0.6-20)[c]
White Mountains, California, 1969-1970	8.0 (1.2-29)
Bermuda, 1971-1972	3.0 (0.1-20)[c]
Hawaii, 1969-1970	2.0 (0.3-13)[c]
Thule, Greenland, 1965	0.5
Antarctica, 1971	0.4
South Pole, 1974	0.049
North Atlantic, 1971-1972	1.6
North Atlantic, 1971-1972	

(NAS, 1980)

The 2500 to more than 1,000,000 fold increase over natural levels of atmospheric lead concentrations in the USA are shown in Table 4.

TABLE 4

ATMOSPHERIC LEAD CONCENTRATIONS AT DIFFERENT LOCATIONS

SITE	CONCENTRATION $\mu G/M^3$
AVERAGE URBAN	0.5 TO 10
AVERAGE RURAL	0.1 TO 1
AVERAGE RURAL - REMOTE	<0.1
MID MANHATTAN - AVERAGE	7.5
NEXT TO HIGHWAYS	5 TO 20
NEAR AUTOS IN TRAFFIC	8 TO 40
IN TUNNELS	20 TO $>$ 100

(NRIAGU, 1978)

(Nriagu, 1978)

In rural America atmospheric lead averages 0.1 to 1 μg Pb/m^3 (100 to 1000 ng Pb/m^3), a value 2500 to 25,000 times the natural level.

The average urban concentration of atmospheric lead varies from 0.5 to 10 μg Pb/m^3 or 12,500 to 250,000 times the natural level.

During traffic hours in many European and American metropolitan areas airborn lead concentration average 8 to 10 μg Pb/m^3. Short term levels as high as 50 μg Pb/m^3 have been reported along major freeways during peak traffic hours in the USA. At times modern man breaths air containing one million times more lead than pretechnological man breathed. This occurs in center city with tall buildings which produce a "canyon effect" and in tunnels where the levels are as high as 100 $\mu g/m^3$.

Automotive exhausts account for more than 95% of the atmospheric lead contaminations in US cities. Evidence that the same is true in European cities has been reported in 1975 by Janssens and Dams.

Despite the awe inspiring magnitude of lead contamination of city air, this may well represent an underestimation of the lead pollution to which modern man is subjected. Most measurements of atmospheric lead are made from samples collected by filters. These filters do not trap volatile organic lead compounds. According to Nriagu (1978), organic lead may constitute as much as 1 to 15% of the total lead in some urban atmospheres. New data suggest the magnitude may even be higher.

Recently Mok and Smythe (1978) reported that air at street level 5 meters from a busy highway contained 2.8 $\mu g/m^3$ of particulate lead and 18.2 $\mu g/m^3$ of "free vapor" lead. This supports an earlier study of Robinson et al (1975) in which they reported very high levels of vapor lead (up to 200 $\mu g/m^3$) in some urban samples. Under unusual circumstances organoleads concentrations of over 50% have been recorded in the vicinity of filling stations. (Jones, 1981)

C. EFFECT OF INCREASING LEAD CONTAMINATION OF THE ATMOSPHERE ON OTHER PARTS OF THE BIOPSHERE

1. EFFECT OF ATMOSPHERIC LEAD FALLOUT ON SOIL AND CITY DUST AND DIRT

The importance of airborn lead contamination of dust and soil arising from leaded gasoline is evident from the studies in 1979 of Schmitt and associates who compared the surface soil and dust contamination in 3 British Columbia cities. The lead accumulations were largely attributed to dustfall from a nearby large smelter in Trail and to automobile traffic in Nelson and

Vancouver. Mean concentration of lead in the soil and dust of the small rural city of Nelson was low (192 ppm); in the areas of Vancouver with heavy traffic the lead concentrations were similar to those found within 1.6 Km of the large smelter at Trail (1545 and 1320 ppm respectively).

In Vancouver soil was collected from boulevards, roadside gutters and vacant lots adjacent to main streets in the downtown residential and commercial districts, where children often come in contact with dirt, dust, and soil.

TABLE 5

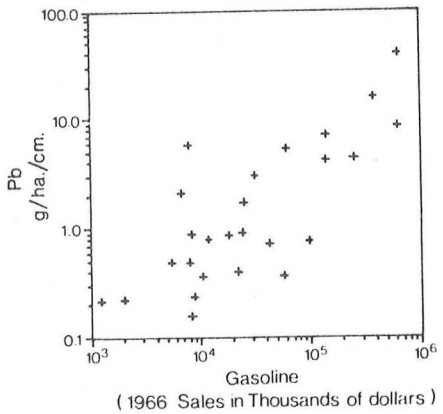
Table I—Lead concentration of surface soil in Trail, Nelson and Vancouver, BC in 1977

City	No. of samples	Concentration (parts per million [ppm]) Median	Mean	Standard error	75th percentile	*t* value*
Trail	153	800	1320	109	1687	—
Nelson	55	83	192	39	253	9.74
Vancouver (selected areas)	37	1240	1545	174	2189	−1.09
Total	245	620	1101	80	1490	—

*Student's *t* statistic from a comparison of the Trail mean with the means of Nelson and Vancouver.

Aside from fallout in areas near smelters, the magnitude of transfer of lead from the atmosphere to soil and city dust and dirt bears a direct relationship to the density of automotive traffic.

Fig. 3



Average gram of lead deposited on one hectare

(Chow, 1978)

While a major factor in symptomatic lead poisoning in city children is residence in old deteriorating housing with flaking, powdering lead paint and putty, the contamination of city dust, dirt and soil by airborn lead is a significant source of the lead burden in young city children. This is supported by the data of Billick and Grey (1978) and Kennedy (1978) who found no source of lead paint exposure in up to 45% of children with elevated blood lead levels.

Street dust frequently contains high concentrations of lead, especially in areas with heavy traffic. Levels in excess of 5000 ppm are not uncommon and levels between 20,000 and 60,000 ppm have been reported.

Swallowing of lead contaminated dust may well account for a major part of the higher mean lead content of city children compared to rural children and for the rather large percent (25%) of city children whose blood lead content was between 40-60 µg/dl. (Nriagu, 1978)

The magnitude of atmospheric lead fallout into city dust and dirt is seen in Table 6.

TABLE 6

LEAD FALLOUT IN STREET DUST

LOCATION	LEAD LEVEL IN PPM
NEW YORK CITY - HEAVY TRAFFIC	20,000
CHICAGO - EXPRESSWAY	4,470
77 CITIES - MIDWEST - RESIDENTIAL	1,636
77 CITIES - MIDWEST - COMMERCIAL	2,413
8 CITIES - INDIANA - RESIDENTIAL	290 - 9972
8 CITIES - INDIANA - COMMERCIAL	942 - 6597
TORONTO - COMMERICAL	67,000

(NAS, 1980)

2. EFFECTS OF ATMOSPHERIC LEAD FALLOUT ON THE CONCENTRATION OF LEAD IN DRINKING WATER

The estimated prehistoric concentration of lead in fresh water was 20 ng/l whereas marine water's natural concentration was only 0.5 ng/l. (Patterson, 1980)

The EPA and the WHO have set 50 µg Pb/l as the mandatory upper limit for lead concentrations in public water (2500 x natural levels). The median concentration of lead in drinking water from 100 largest US cities was 3.7 µg/l, a value 185 times the natural concentration in fresh water.

Studies have been made of the percentage of drinking water exceeding the 50 µg/l manditory limit in two large US cities. In Seattle, 24% (Dangle, 1975) and in Boston 65% of water sources (Greathouse et al, 1976) exceeded this value. (in Mahaffey, 1978)

Where there is aggressive water (low pH, low minerals) coupled with lead pipes, lead solder joints or lead lined storage tanks very high levels of lead in water are found. The lead concentration of soft water delivered through lead pipes averaged 1000 µg/l.

3. EFFECTS OF ATMOSPHERIC LEAD FALLOUT ON THE CONCENTRATION OF LEAD IN FOOD.

Atmospheric lead fallout can increase the lead content of food in at least 4 different ways:

1. biological uptake from contaminated soil into plants which are eaten by man.
2. direct deposition of airborn lead onto plants.
3. absorption of lead from cooking water contaminated by fallout.
4. direct depositions on food prepared or in the process of preparation.

In addition, lead can be added to food in several other ways; 1) from dust and/or solder on processing machinery, 2) from solder used to seal cans, 3) from lead leached from storage containers with soldered seams.

According to the NAS (1980) an increasing number of reliable recent reports indicate that most of the lead content of typical vegetation (90 to 99%) originates from recent atmospheric pollution. Settle and Patterson (1979) reported that the diet of modern man (300 µg/day) contains 100 times the lead content of pretechnological man (3 µg/day).

In general canned food contains about 2 times as much lead as fresh foods but this varies widely and very high levels (greater than 400 µg/L) have been reported in some baby foods. However, Patterson has shown that the lead content of tuna increases 4000 fold by the canning process but was contaminated only 16 fold by pollution in the ocean.

Lead content of many foods grown in urban gardens especially leafy vegetables have a much higher lead content than food grown in rural areas.

Schutz (1979) reported that the lead content of foods eaten in Sweden (30 µg/day) is an order of magnitude less than what Americans ingest (300 µg/day). Schutz's reconfirmed data revealed that Swedish adults ingest 10 times lower lead than consummed in the USA. This is supported by other data which revealed 10 times less lead in the skeletons of modern Swedes compared to Americans. (NAS, 1980)

A summary of the daily exposure of pretechnological "natural" man and modern man to concentrations of lead in air, water and food is depicted in Table 7.

TABLE 7

Inventory of Estimated Average Daily Pb Absorbed into Blood in Adult Humans (ng Pb/day)

Source	Prehistoric Natural	Contemporary Urban American
Air	0.3	6,400
Water	<2.0	1,500
Food	<210.0	21,000
TOTAL	<210.0	29,000

Natural

Air	0.04 ng Pb/m^3	× 20.0 m^3/day	× 0.4	=	0.3 ng Pb/day
Water	<20.0 ng Pb/kg	× 1.0 kg/day	× 0.1	=	<2.0 ng Pb/day
Food	<2.0 ng Pb/g	× 1.5 kg/day	× 0.07	=	<210.0 ng Pb/day
TOTAL					<210.0 ng Pb/day

Contemporary

Air	800 ng Pb/m^3	× 20.0 m^3/day	× 0.4	=	6,400 ng Pb/day
Water	15,000 ng Pb/kg	× 1.0 kg/day	× 0.1	=	1,500 ng Pb/day
Food	200 ng Pb/g	× 1.5 kg/day	× 0.07	=	21,000 ng Pb/day
TOTAL					29,000 ng Pb/day

III. EVIDENCE FOR INCREASED HUMAN BODY BURDEN OF LEAD SECONDARY TO POLLUTION OF THE BIOSPHERE

A. SUPPORTING DATA FROM CHANGES IN BLOOD LEAD LEVELS

In 1965 Patterson on the basis of geophysical considerations computed the "natural" blood lead levels of ancient pretechnological man to be 0.2 µg/dl or 100 to 200 times less than the concentration (20 to 40 µg/dl) frequently found today. Again, Patterson's calculations have been confirmed. This time by the report of Hecker et al in 1974 that the Yanomamö Indians, a remote "unacculturated" population living at the source of the Orinoco River in southern Venezuela had a mean blood lead level of 0.87 µg/dl. According to Ericson et al, these extant primitives nevertheless live in an ecosystem remote but partially con-

taminated by industrial aerosols. Consequently Patterson's estimation of "natural" blood lead levels of 0.2 µg/dl is certainly in the correct order of magnitude and not an underestimation.

The average blood level of present day Nepalese adults and children living at the foothills of the Himalayas was found by Piomelli to be 3.4 µg/dl. This is a remote but not "unacculturated" population. By contrast less than 0.15% of New York City school children (3 of 2044) had blood lead levels of 3 µg/dl or less.

The maximum "safe limit" for blood lead according to recent CDC and EPA criteria is 30 µg/dl. This is a concentration about 9 fold greater than present day Nepalese, 35 time greater than present day Yanomamö Indians and 150 fold greater than prehistoric man.

Recent nationwide surveys have shown that blood lead levels greater than 40 µg/dl clearly indicating undue lead absorption were found in about 25% of asymptomatic young preschool children living in large metropolitan areas. (Nriagu, 1980)

TABLE 8

Incidence of overexposure to lead in the USA.[a]

Location	Children 0–5 yr (%)	Adult	
		Females (%)	Males (%)
Baltimore	25.3–31.5	–	–
Camden, New Jersey	–	1.8	–
Chicago	2.0	–	–
Illinois, 12 cities	11.4–31.3	–	–
New Haven	23.7–29.8	–	–
Newark	38.9	–	–
New York	20.2–45.5	–	–
Norfolk, Virginia	22.7	–	–
Oakland		1.9	5.5
Philadelphia	34.0	0.7	2.3–4.5
Washington	22.0–39.2	–	–
Cincinnati, various occupations	–	–	2.9–6.7
Los Angeles, various occupations	–	3.3–4.4	0.6–5.2
Residents near a roadway	–	1.8	–
Average	24.6	2.2	2.8

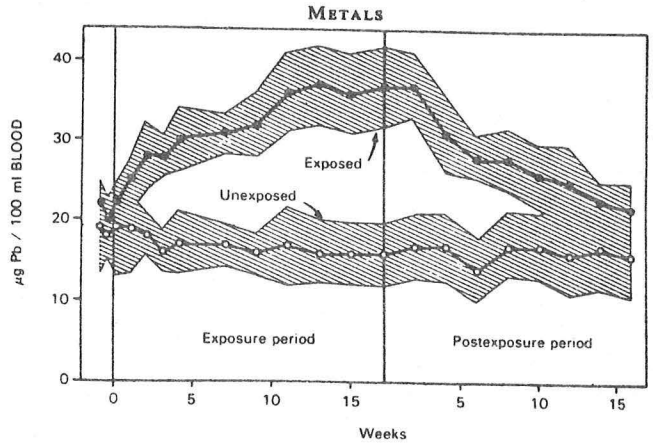
a. The table shows the percentage of the various populations having blood lead levels greater than 400 µg/l, the level believed to be indicative of overexposure.

(Nriagu, 1980)

According to calculations of the EPA each 1 $\mu g/m^3$ increase in atmospheric lead concentration results in a 2 µg/dl increase in blood lead levels. However, the recent studies of Rabinowitz et al (1977) indicate that for each 1 $\mu g/m^3$ increase in ambient air lead concentration, blood lead increases about 3.5 µg/dl. Of the atmospheric lead inhaled about 40% is absorbed. The EPA has adopted 1.5 $\mu gPb/m^3$ as the national standard. This is more than 37,500 times the pretechnological uncontaminated air levels.

The effect of increasing the lead pollution of ambient air on blood lead levels is seen in Figure 4.

Fig. 4



Effect of continuous air exposure to 10.9 µg Pb/M^3 on the concentration of lead in the blood of human volunteers. (From Cole, J. F., and Lynam, D. R.: ILZRO's research to define lead's impact on man. *Proceedings of the International Symposium, Environmental Health Aspects of Lead*, Amsterdam, 1972. Luxembourg, Commission of the European Communities, 1973.)

The Conservation Society of England reported that 32-69% of blood lead comes from inhaled air. The closer to urban traffic and the greater the urban traffic the higher were the blood lead concentrations of 5228 children surveyed by Caprio et al. (1974)

TABLE 9

Table 4.—Percentage of Lead Exposure Groups Affected by AWVDs

AWVD	Blood-Lead Levels: < 40µg/100 ml	40µg to 59µg/100 ml	60µg/100 ml and over
< 24,000	58.3	36.6	5.1
≥ 24,000	37.9	51.3	10.8
City-wide expected values	65.1	29.9	5.0

Caprio et al (1974) reported that in Newark, N.J. 62% of school children residing in an area of high AWVD (average weekly vehicle density) exceeding 24,000 had blood lead levels in excess of 40 µg/dl. The number of school children with blood lead levels of 60 µg/dl or over doubled to 10.8% in residential areas exposed to AWVD exceeding 24,000. This level (60 µg/dl) found in these 10.8% of city children living near heavy traffic is 18 times that of Nepalese children, 70 times that of the Yamomamö Indians and 300 times that of "natural" man.

B. SUPPORTING DATA FROM CHANGES IN THE LEAD CONTENT OF HUMAN BONES

The massive changes in blood lead levels in present day Americans compared to other cultures and to ancient man indicate a much greater exposure to lead and an anticipated elevation of body burden of lead. However, blood lead levels reflect only recent exposure since the residence time of lead in blood after exposure is only about 36 days. The bone is a much better indicator of life long exposure since bone lead represents more than 95% of body lead and the residence time of lead in bone after exposure averages 10,000 days.

Ericson, Shirahata, and Patterson (1979) measured the skeletal concentration of lead and the Pb/Ca and Ba/Ca ratios in the bones of ancient Peruvians who lived 1400-2000 years ago. These data indicate that compared to ancient Peruvians there has been about a 600 fold increase in bone lead and therefore in body burden of lead in modern Americans! (Patterson, 1980)

TABLE 10

Predicted and Measured Ba/Ca and Pb/Ca Ratios in Ancient and Modern Human Skeletons

Material	Atomic Elemental Ratios	
	Ba/Ca	Pb/Ca
Average crustal abundance	3×10^{-3}	6.4×10^{-5}
Predicted in human bones	3×10^{-6}	3×10^{-8}
Measured, Peruvian bones	2.5×10^{-6}	6×10^{-8}
Measured, British bones	7×10^{-6}	2.1×10^{-5}
Measured, American bones	2.9×10^{-6}	3.5×10^{-5}

SOURCE: Data from Ericson et al. (1979).

The effects of this contamination of the biosphere with urban contamination far out stuffing rural contamination can been seen from a comparison of the lead organ concentrations of wild city and rural rats (Mouw et al, 1975). It should be noted that brain concentration of lead was more than 5 fold increased in city rats compared to rural rats.

TABLE 11

—Lead Concentrations (Both Sexes) (μg/gm Wet Weight)*					
	Urban		Rural†		
Sample Source	No. of Samples	Mean ± SE	No. of Samples	Mean ± SE	Significant Differences, *P* Value
Blood	39	0.55 ± 0.04	28	0.17 ± 0.02	.001
Liver	21	3.34 ± 0.45	19	0.44 ± 0.09	.001
Kidney	25	22.7 ± 2.8	20	1.14 ± 0.28	.001
Lung	23	1.24 ± 0.20	19	0.24 ± 0.03	.001
Brain	22	1.11 ± 0.17	19	0.21 ± 0.06	.001
Bone	24	200.00 ± 19	18	10.3 ± 1.9	.001
Feces	19	178.00 ± 30	19	46.6 ± 6.0	.001

Data are from 41 urban rats (26 males and 15 females, body weight =382 ± 13 gm) and 28 rural rats (20 males and eight females, body weight =322 ± 14 gm).

(Mouw et al, 1975)

IV. INCREASED RISK OF HARMFUL EFFECTS FOR VERY YOUNG CHILDREN

Clinically most non-industrial lead toxicity occurs among children between the ages of 1 and 6 years with the highest incidence between 2 to 3 years of age. Young children are at a much greater risk for increase body burden of lead and for a greater incidence of elevated blood lead levels from any given level of pollution of the biosphere when compared to adults for at least four reasons:

A. INCREASED VENTILATORY EXCHANGE - young children (age 1-3 years) not only have a greater (2 times) ventilatory exchange per kg body weight than adults (Knelson, 1974) but also are exposed to higher concentrations of airborn lead generated by combustion of leaded gasoline because of the falling concentrations of lead in air as distance from the street increases from 2 to 8 feet (Nriagu, 1978). In addition, young children often are exposed to high particulate concentrations of lead from dust raised during their play and by foot or vehicular traffic. During reaerosolization particulate concentrations fall rapidly with height so that older children and adults are exposed to considerably lower lead concentrations than 1 to 6 year old children.

TABLE 12

ATMOSPHERIC LEAD CONCENTRATIONS IMMEDIATELY ABOVE STREET

FEET ABOVE STREET	CONCENTRATION μG/M^3
TWO	14.3
THREE	9.0
FIVE	8.3
EIGHT	2.8

(NRIAGU, 1978)

B. INCREASED GASTROINTESTINAL ABSORPTION OF FOOD LEAD

The studies of Kehoe and of Rabinowitz et al showed that adults absorb on the average 7 to 10% of ingested food lead. In contrast Alexander et al in 1974 and of Zeigler et al in 1978 reported that infants and young children absorb 40 to 50% of ingested food lead.

The average infant from birth to 2 years ingests about 100 µgPb/day in food and water and absorbs about 40-50 µg/day. A child from 2 to 3 years of age ingests about 150 µg/day and absorbs about 60-75 µg/day from food and water. In contrast, adults ingest about 300 µg/day but absorb only 21-30 µg/day.

In addition, several studies have shown that iron deficiency and low dietary calcium intake both result in augmented absorption and retention of dietary lead. Numerous nutritional surveys have confirmed the high incidence of both iron deficiency and low calcium intake in children who reside in the inner city of large metropolitan area. (Mahaffey, 1980, Zeigler et al, Sorrell et al, Mahaffey & Rader, 1980, Watson et al).

C. INCREASED INGESTION OF HOUSEHOLD AND STREET DUST AND DIRT -

Mouthing of objects and oral exploration is a part of normal development in young children. Recently this normal mouthing activity has been rediscovered as an extremely important contributing cause to the elevated blood lead levels of young urban children. The exaggerated mouthing practices of the very young include finger licking, thumb sucking and the mouthing of toys and pets. This is an efficient way to transfer lead laden dust and dirt from the environment to the mouth. Since urban dust contains lead in concentrations between 1000 and 10,000 µg/gm, the normal hand-to-mouth and mouthing activity of children results in the ingestion of considerable quantities of lead. Ingestion of only 100 mg of dust or dirt containing 3000 ppm of lead would add an additional external dose of 300 µg of lead of which 120-150 µg would be absorbed.

Pica, defined as the repeated ingestion of non-food substances such as clay, dirt, laundry starch, paint, etc., was at one time considered mainly to be associated with lower range IQ levels and to be abnormal. While pica remains an important cause of very high lead levels associated with clinical lead poisoning in young children, it does not seem to be an important contributing factor to blood lead levels in the range of 45 µg/dl. (Mahaffey, 1978 in Nriagu)

Pica is more likely related to cultural experience than to low IQ. It is far more prevalent in both Blacks and American Indians than in Caucasions. This is supported by the data indicating that compared to mothers who do not have children with pica, mothers of children with pica 1) more frequently have pica themselves, 2) often have taught pica to their children, 3) have other children with pica. Moreover, the Committee on Toxicology of the NAS reported in 1975 that 50% of all children ages 1 to 3 years have pica. (Mahaffey, 1978)

The mouthing activity of children with high (40 to 70 µg/dl) and low (less than 29 µg/dl) blood lead levels were compared by Charney et al. (1980)

TABLE 13

MOUTHING HABITS IN CHILDREN

GROUP	HIGH LEAD (N=49)	LOW LEAD (N=50)	P VALUES
HABITUAL FINGER SUCKING	74%	50%	.04
MOUTHING TOYS	82%	58%	.007
CHEWING PENCILS	50%	24%	.001
SOIL Pb - µG/GM			
MEAN	1563	1008	.04
MEDIAN	1502	633	

(CHARNEY, et al 1980)

(Charney et al, 1980)

The studies of Charney et al in 1980 also compared the household dust and hand lead levels in young children with high blood lead levels (40 to 70 µg/dl) to those with low blood lead levels (29 µg/dl or less). The house dust and hand lead levels were significantly higher in the high blood lead level group.

TABLE 14

Household Dust Lead

Household Dust Values (μg/sample)	High PbB Group (n = 49)	Low PbB Group (n = 50)	*P* Values
Mean	265 (± 288)	123 (± 160)	
Median	149	55	≤.01
Combined group median (93 μg)			
Above median*	32	17	
Below median	17	33	.005

*$\chi^2 = 9.7$.

TABLE 15

Hand Lead Level

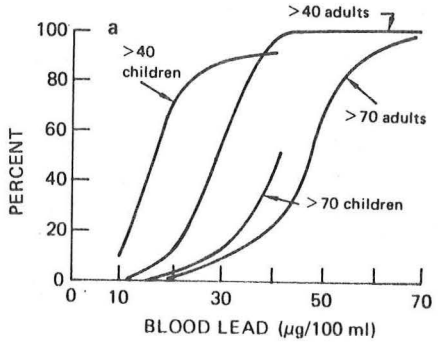
Hand Pb Level (μg/sample)	High PbB Group (n = 49)	Low PbB Group (n = 50)	*P* Values
Mean	49 ± 69	21 ± 28	.01
Median	30	12	
Combined group median (20 μg/sample)			
Above median*	31	16	
Below median	18	34	.005

*$\chi^2 = 9.7$.

(Charney et al, 1980)

D. BIOCHEMICAL HYPERSENSITIVITY TO LEAD - Young children appear to show biochemical alterations at lower blood lead levels than adults. One example of this is seen in a comparison of the dose-response curve for effects of lead on heme synthesis in adults and children. The percentage of each population with 40% and 70% inhibition of activity of delta aminolevulinic acid dehydratase (ALAD) in relation to blood lead levels is shown in Figure 5.

Fig. 5



Dose-response curves for effects of lead on heme synthesis in adults and children. A. Percentage of population with 40 percent and 70 percent inhibition of ALAD in relation to lead in blood.

(NAS, 1980)

V. POTENTIAL DANGER OF LOW LEVEL LEAD EXPOSURE ON INTELLIGENCE, BEHAVIOR AND LEARNING

According to Piomelli and others while lead poisoning threatens almost all people exposed to our lead polluted environment, it is a greater hazard to young children who are both more exposed and more sensitive to lead. Biochemical and morphological abnormalities can be produced by giving newborn animals amounts of lead which do not cause obvious toxicity. The fetus and young children are at greatest risk since the developing brain is especially vulnerable to toxins. Lead is a known neurotoxin capable of causing encephalopathy, at times fatal, especially in young children.

Although an increase in body burden of lead is not necessarily identical to an adverse health effect, there are however, increasing data in both animal and human studies indicating that "low-level" lead exposure while clinically asymptomatic may be producing significant subtle yet deleterious neurophysiological effects and behavioral effects which may require especially sophisticated experiments to document. Some of these studies will be reviewed.

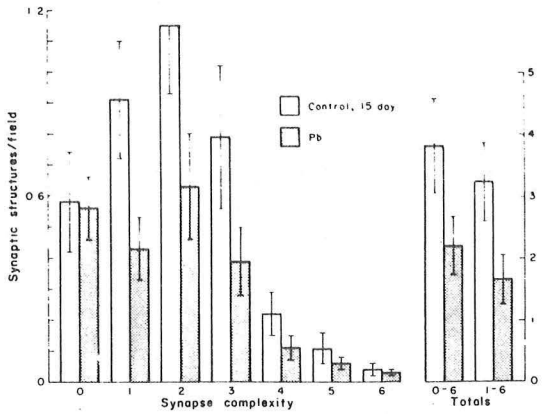
A. ANIMAL STUDIES ON BRAIN DEVELOPMENT, LEARNING AND BEHAVIOR.

Evidence is now abundant from a growing number of animal studies that clearly indicates that prenatal or perinatal exposure to lead at blood lead concentrations commonly encountered (32-36 µg/dl) in human populations not only produces impairment of neurological development and learning ability but also in behavioral abnormalities in rats, mice, dogs, sheep and monkeys.

a) Recent studies of Bull et al, 1979 showed rat pups exposed *in utero* to maternal blood lead levels of 32 µg/dl produced delays in maturation of the cerebral cortex associated with delayed expression of exploratory and locomotive behavior.

Both McCauley, Bull et al (1979) and Averill et al (1980) reported impaired cerebral synatogenesis after prenatal exposure to lead. In McCauley's studies 15 day old rat pups exposed *in utero* to lead levels in maternal blood of only 32-36 µg/dl had signficantly ($P=0.001$) reduced synaptic densities in the cerebral cortex as determined by ethanol-phosphotungstic acid staining. Brain lead averaged 0.25 µg/gm a value similar to that found in rural rats (0.21 µg/gm) and 5 times less than found in city rats (1.11 µg/gm).

TABLE 16



Pb-induced depression of synaptic figures in the 15 day old rat cerebral cortex. Level of complexity was judged by the number of presynaptic dense projections. Results of 7 control and 7 Pb litters ± S.E.M.

These data demonstrate that delays in brain development and abnormal behavior can be produced in neonatal rats at lead exposures frequently found in human newborn populations.

In 1974 Carlson et al reported impaired learning in lambs secondary to in utero exposure to lead. In those studies pregnant ewes were given lead sufficient to maintain mean blood lead concentrations of 34 µg/dl and 18 µg/dl throughout gestation. Lambs from these groups at age 2 to 4 weeks had blood lead levels of 24 and 17 µg/dl respectively, whereas control lambs had levels of 6 µg/dl.

Between 10 to 15 months of age tests revealed that the lambs prenatally exposed to maternal blood lead of 34 µg/dl required significantly more days to learn visual discrimination than controls or those exposed to 18 µg/dl in maternal blood. In some instances visual discrimination could not be learned at all by those exposed to maternal blood lead levels of 34 µg/dl.

Finally, neurochemical studies in animals indicate that lead at low levels is a potent neurotoxin especially in the perinatal human or animal when the blood brain barrier permeability and neuronal development enhance many toxins. According to Silbergeld and Hruska, (1980) neurotoxic effects at concentrations as low as 0.02 ppm (2 µg/100 dl) affect the biochemical balance of transmission and inhibition of nerve impulses. They caution against assuming the existence of any "safe" levels of lead exposure and raise concern that neurons may be irreversibly damaged by any exposure to lead. According to the 1980 report of the NAS "the evidence that small amounts of lead interfere with neurochemical functions is already persuasive and growing."

B. STUDIES IN HUMANS

Clinical symptomatic lead poisoning has long been known to effect the central and peripheral nervous systems of children and adults and to be followed by behavioral changes and impaired learning ability. The most critical unresolved issue is whether psycho-neurological, behavioral and learning abnormalities or defects occur at body burdens of lead typical of the range observed in urban areas.

There is definitive evidence from both human and animal studies that lead crosses the placenta and accumlates in fetal tissues starting after the 12th-14th week of pregnancy in humans (Jaworski, 1979, NAS 1980, Baltrop and Burland, 1969). Fetal blood lead levels correspond closely to maternal blood levels. The distribution of lead amongst fetal tissues is similar to its distribution after birth except that a proportionately much greater fraction of total body burden is concentrated in the brain of the fetus (Drill, 1979, NIOSH, 1978, NAS, 1980).

Immediately relevant to the studies on the deleterious effects of perinatal lead exposure in animals are the studies of Beattie, Moore, et al (1975) and Moore (1980) who found a strong association between drinking household water with high levels of lead during pregnancy in Glasgow, and unexplained mental retardation in 77 children.

The probability of mental retardation was significantly increased when water lead in the home exceeded 800 µg/l. Under such circumstances the child was at least 1.7 times (and probably much greater) more likely to be mentally retarded than a child whose exposure to water lead was unknown. (Moore, 1980)

Blood lead measured in these children a few days after birth showed levels of 25.4 µg/dl in retarded vs. 17.8 µg/dl in a carefully matched control group. In this case the childrens' blood lead level cannot be attributed to their behavior and the inference that lead was a causal factor in their impaired intellectual level is strongly supported.

According to Cowan and Levitan, (1980), Moore's data indicate a progresively increasing relative risk of mental retardation with increasing blood lead levels found shortly after birth. Increase in blood lead from 20 to 41 µg/dl near birth increased the relative risk of mental retardation 6 fold.

Fig. 6

EPIDEMIOLOGIC CONSIDERATIONS

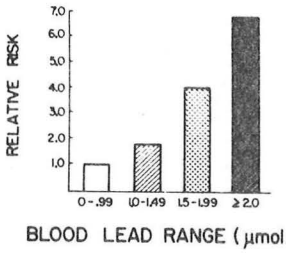


FIG. 3. Relative risk of mental retardation by blood lead level. Relative to a risk of 1.0 for children with blood lead levels < 1 μ mole/liter. (Adapted from Moore et al., ref. 27.)

(Adapted from Moore, in Cowan & Levitan, 1980)

There are many studies purporting to show and some failing to show effects of childhood exposure to lead on neurophysiologic development, on behavior and on learning. Unfortunately too many of these studies are poorly conceived, inadequately controlled and uncritically interpreted. Two recent reviews critically examine most of these studies (Repko and Corum, 1979: Rutter, 1980)

The problem of attempting to determine whether or not cognitive and neurological deficits and deviant behavior result from early and/or long term lead exposure at levels unassociated with previously recognized overt disease and at blood lead levels previously thought to be "safe" requires, after appropriate control of all confounding variables, the careful consideration of at least 3 major factors: 1) definition of what constitutes a "normal" body burden of lead, and selection of appropriate controls, 2) appropriate laboratory assessment of lead exposure, and 3) ability to measure outcome of exposure.

a) Definition of a "normal" body burden of lead is difficult as a consequence of the ubiquitous and inescapbable nature of lead in our contaminated environment. It may not be possible to identify a group of children and newborn truly unexposed to lead. What is normal if all are exposed? It seems likely that only degrees of exposure differ. Under these circumstances it is best to classify exposures into a number of categories according to lead levels and then to compare different degrees of exposure rather than be mislead into thinking that the controls are exposure free.

b) The method of determining exposure used in any study is critically important. The relevant lead exposure in assessing neurobehavioral and learning deficits is likely to have been several years prior to measurement, at an age when the CNS is

presumed to be more susceptable to environmental toxins. Blood lead levels are not an appropriate measure of exposure which occurred years previously since the residence time of lead in blood is only 36 days. Blood lead does not measure any exposure rather it measures only recent exposure.

By contrast during formation and calcification, deciduous teeth are reliable indicators of cumulative lead exposure. Dentine lead levels quantitate cummulative exposure since dentine is a permanent storage system for lead. No loss occurs over time even with chelation therapy and dentine lead levels are dose related. The magnitude, time course, and duration of lead absorption are better recorded by dentine lead than any other readily available measurement and hence is the best estimate of meaningful body burden.

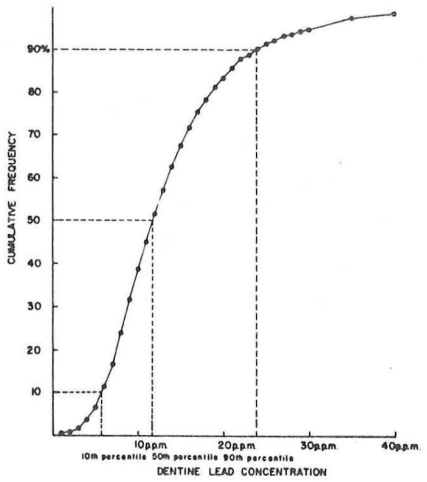
Effects of subclinical lead exposure on neurobehavioral and learning ability of young children produced confusing results because data indicating present exposure (blood lead levels) were used rather than using an accurate retrospective measure of exposure (dentine or tooth lead).

c) The sensitivity of the tools used to measure the outcome of exposure also is of critical importance. If the ill effects of "low dose" lead exposure in children are subtle intellectual and behavioral defects, then the tools used to evaluate neurobehavioral and learning deficits must be sensitive enough to detect small differences. In some previously mentioned negative studies attempts were made to weigh mice on scales built for elephants.

The most sophisticated and notable of all recent studies which was carefully controlled for confounding variables and which paid appropriate attention to the three major prerequisites for adequate testing just discussed were the studies of Needleman and associates in 1979.

Needleman and his associates measured the dentine lead concentration of recently shed deciduous teeth of 2335 (70%) of 3329 asymptomatic children attending first and second grades in Chelsea and Somerville, Massachusetts. Subjects whose dentine lead levels fell in the highest 10th percentile (greater than 24 ppm) and the lowest 10th percentile (less than 6 ppm) were classified as high dentine and low dentine lead groups. None had past or present evidence of clinical lead intoxication. These children then underwent extensive neuropsychologic evaluation. 135 children were in the low dentine lead group and 86 were childen in the high dentine lead group.

Fig. 7



Cumulative Frequency Distribution of Dentine Lead Concentrations (3221 Specimens).

In addition, day to day classroom behavior of all 2146 children whose teeth were analyzed for lead were evaluated by their teachers in blind fashion (in regard to dentine lead levels) by a forced item choice behavioral questionnaire.

THE FREQUENCY OF NON-ADAPTIVE CLASSROOM BEHAVIOR INCREASED IN A DOSE RELATED FASHION TO PROGRESSIVE INCREASES IN DENTINE LEAD.

Fig. 8

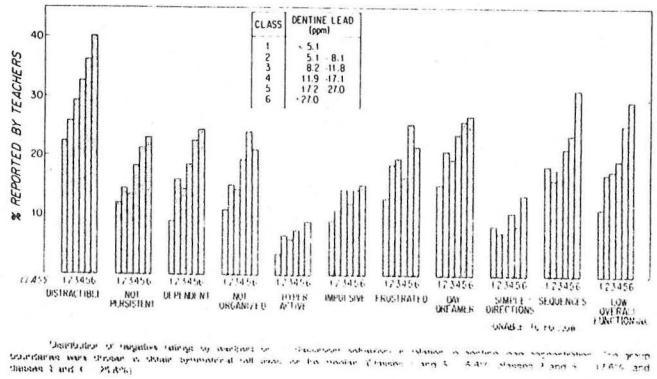


TABLE 17

Comparison of High and Low Lead Subjects on Teachers' Behavioral Rating Scale The Numbers Show the Per Cent of Students in Each Group Receiving a Negative Response

Item	Low Lead (%)	High Lead (%)	P Value
Distractible	14	36	0.003
Not persistent	9	21	0.05
Dependent	10	23	0.05
Disorganized	10	20	0.14
Hyperactive	6	16	0.08
Impulsive	9	25	0.01
Easily frustrated	11	25	0.04
Daydreamer	15	34	0.01
Does not follow:			
Simple directions	4	14	0.05
Sequence of directions	12	34	0.003
Low overall functioning	8	26	0.003
Sum score (mean)	9.5	8.2	0.02*

*Analysis of covariance.

MOST IMPORTANT IS THE FACT THAT TESTING REVEALED SIGNIFICANT DIFFERENCES BETWEEN LOW DENTINE AND HIGH DENTINE LEAD GROUPS IN THE FULL SCALE WECHSLER IQ TEST, IN THE VERBAL IQ TEST, IN THE SEASHORE RHYTHM TEST AND IN REACTION TIME TESTS.

TABLE 18

LEAD AND NEUROPSYCHOLOGICAL DEFICIT

TABLE 5. *Outcome measures*

	High lead ($\bar{x}$)	Low lead ($\bar{x}$)	*p*-Value (2 tail)
Full-scale IQ (WISC-R)[a]	102.1	106.6	.03
Verbal IQ	99.3	103.9	.03
Performance IQ	104.9	108.7	.08
Seashore rhythm test	19.4	21.6	.002
Token test	23.6	24.8	.09
Sentence-repetition test	11.3	12.6	.04
Reaction time[b]			
Block 1 (3 sec)	.37 ± .09	.35 ± .08	.32
Block 2 (12 sec)	.47 ± .12	.41 ± .09	.001
Block 3 (12 sec)	.48 ± .11	.41 ± .09	.001
Block 4 (3 sec)	.41 ± .12	.38 ± .1	.01

[a] Wechsler intelligence scale for children.
[b] Under varying intervals of delay. High-lead and low-lead results are expressed (in sec) as $\bar{x} \pm$ SD.

Needleman in collaboration with Burchfiel, Duffy and Bartels (1980) utilized the most sophisticated of electroencephalographic techniques to determine whether any differences in electrophysiologic brain behavior existed between the high dentine lead and low dentine lead groups. Nineteen high lead and 22 low lead children underwent quantitative electroencephalographic studies. Computer assisted spectral analysis of EEG data revealed significant differences between groups.

The high dentine lead group had consistently higher percentages of slow frequency delta (0.5-3.5 Hz) activity recorded from a series of adjacent electrodes covering the central, parietal and occipital regions of the head bilaterally.

Fig. 9

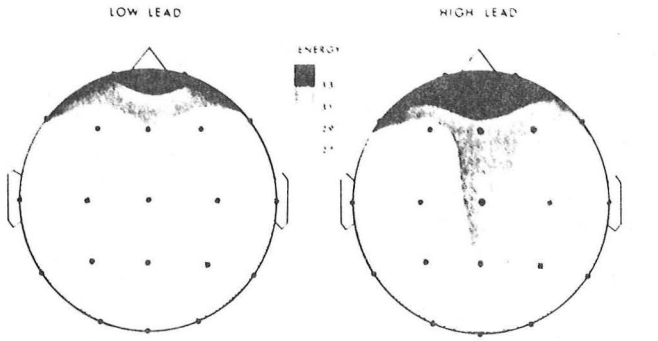


FIG. 2. Topography of delta EEG spectral energy in high and low lead children. EEG recorded while subjects were relaxed, alert with eyes closed. Delta energy was calculated as the percentage of total EEG spectral energy in the frequency range 0.5–3.5 Hz. This was done for each electrode derivation from the mean EEG spectrum of the high and low lead group, respectively, and topographical maps of delta energy were constructed by linear interpolation based upon the values of the nearest three electrode points (11).

The second pattern of differences in the eyes closed EEG was a decrease in the percentage of alpha (8-12 Hz) activity in the occipital lobes and in the midline central-parietal region of the high dentine lead subjects compared to low dentine lead subjects.

These EEG data coupled with the neurobehavioral and learning deficits in these "asymptomatic" normal school children indicate that levels of lead absorption resulting from "ordinary" everyday environmental lead exposure can significantly and in a deleterious way affect CNS functions of "normal" children.

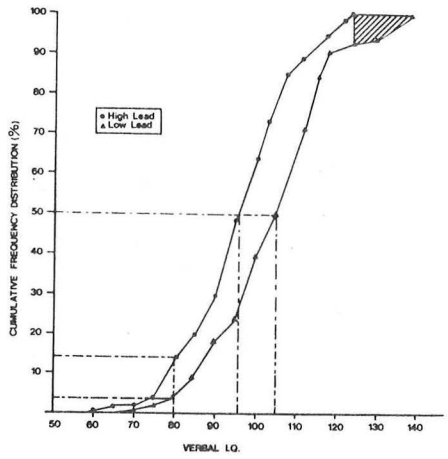
Although some critics conclude that evidence of only slight congnitive impairment seems to occur, i.e., less than 4-5 IQ points, Rutter has pointed out that a reduction in IQ of 5 points for the general population would produce a doubling of the number of mentally retarded children with an IQ under 70.

The data of Needleman et al (1982) on the actual cumulative frequency distributions of verbal IQ scores disclose that a shift of 4 points in the mean of a normal distribution has dramatic effects on the properties of the tails of the distribution.

1. Children with elevated dentine lead levels were 3 times more likely to have a verbal IQ level below 80 than children with low dentine lead levels.

2. Equally disturbing is the evidence that although 5% of those with low dentine lead levels had IQ scores in the superior range (greater than 125), no child with elevated dentine lead levels scored in this range.

Fig. 10



Cumulative Frequency Distribution of Verbal IQ Scores in Subjects with Low or High Levels of Lead.

(Needleman and Leviton, 1982)

The evidence presented today from epidemiological studies, from neurochemical and neurophysiological investigations, and from neurobehavioral and learning studies in both animals and young children taken as a whole provide strong evidence that lead at body burdens that do not seem to produce overt easily recognized clinical disease is a primary etiological factor in the impaired intellectual development and behavioral problems of some children. The weight of evidence presently available strongly suggests that not just some but perhaps almost all children with more typical ordinary present day exposure to lead may be at risk of potential deleterious effects on behavior and learning ability.

In Repko and Corum's extensive review they concluded by stating "objective evaluation of the literature clearly suggests a sequelae of lead intoxication which is continuous and pervasive. The clinical and neurobehavioral sequelae are not indicative of separate and independent functional impairments but rather a continuum of dysfunction experienced by all exposed individuals. Moreover, the effects on neurological and behavioral systems appear to begin at levels heretofore regarded as safe and acceptable."

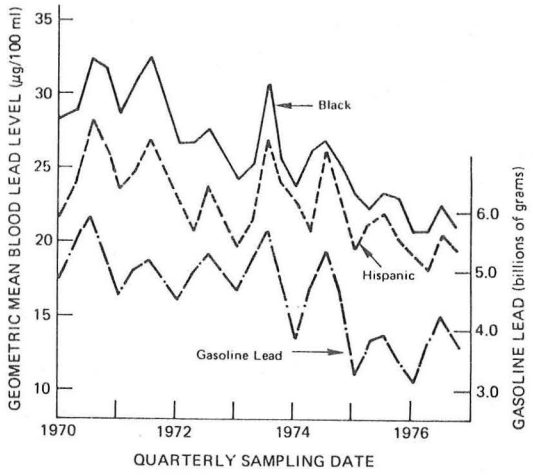
VI. SOME RAYS OF HOPE FROM RECENT CHANGES IN FEDERAL REGULATORY ACTIVITY

Ongoing federal regulations regarding ambient air quality standards for lead, emission standards for industrial sources of lead and especially limitations on lead content of gasoline already have made a significant impact on the level of lead contamination to which we are all exposed.

Since 1974 the use by new automobiles of lead free gasoline (0.05 gm/gal.) which was introduced not to protect humans but to protect catalytic converters coupled with a 6 year progressive reduction in the lead content of leaded gasoline (from 2.04 to 0.5 gm/gal.) has produced a significant decrease in lead emission into the atmosphere.

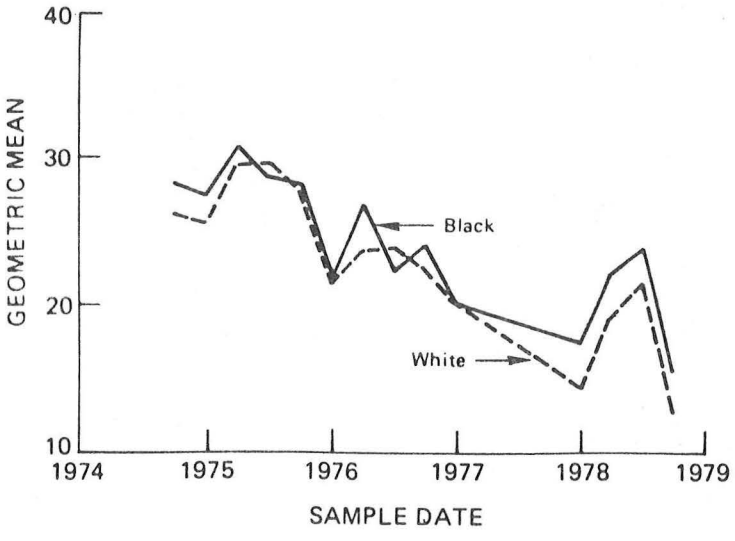
According to a recent CDC report the amount of lead added to the environment from gasoline dropped from 190,000 tons in the USA in 1976 to 90,000 tons in 1980, a 53% reduction. In some areas this has been associated with reduced blood lead levels in children.

Fig. 11



(NAS, 1980)

Fig. 12



(NAS, 1980)

H-2 Wednesday, May 26, 1982 The Dallas Morning News 17-A

EPA allowing more lead in gasoline

By Robert D. Hershey Jr.
New York Times News Service

WASHINGTON — The Environmental Protection Agency has been telling oil companies that they are free to increase the lead content of regular gasoline nearly 10 percent above the published limit.

The agency has limited large refiners officially to 0.5 grams of lead per gallon of gasoline since 1980. While environmentalists and many companies assumed the level could not be exceeded even slightly, the government has interpreted the limit to be almost 0.55.

Lead has been long identified as a risk to human health, causing birth defects and mental retardation in children, among other problems.

"An average lead level of 0.549 gram per gallon would not lead to an enforcement action by EPA," an agency enforcement official, Richard Kozlowski, said in a letter late last month to Mobil Oil Corp. The letter was disclosed Tuesday by the Center for Auto Safety, a Ralph Nader Group.

In the fall of 1980, the EPA gave the sale information to several other companies, either orally or in writing, Kozlowski said.

The Center for Auto Safety said Tuesday it was "shocked and outraged" to learn of what it called a secret relaxation of the limits and said an additional 10,000 tons of lead will be emitted from car exhausts in the next year if it is left unchanged.

But the EPA said there had been no change in policy. Responding to an inquiry, Koslowski said that when the regulations went into effect in October 1980 it was unclear how to interpret the 0.5 gram per gallon standard. He said the agency moved promptly, however, to decide that the figure should be taken to mean as much as 0.549 grams was permissible and that companies that asked for guidance have been so informed.

"The question was how you rounded point-5," Koslowski said.

18 THE WALL STREET JOURNAL, Thursday, May 27, 1982

Toxic Tragedy: Lead Poisoning Takes Continuing Toll on Children

Herbert Needleman jarred the medical community anew, however. It found that a group of Boston schoolchildren with lead presence as low as 30 micrograms per 100 milliliters of blood (at the time, not considered a dangerous level) had mean IQ levels four points lower than a group with little or no lead. Their classroom attentiveness and ability to follow directions also suffered by comparison, according to teachers.

Dr. Needleman's is one of the voices raised against the diminished federal effort. "If the cuts continue we're going to see an increase in the number of lead-related deaths," he warns.

There may be a federal pullback on another front, too. The Environmental Protection Agency has for seven years been forcing oil refiners to gradually decrease lead levels in gasoline. Now, at the behest of a White House regulatory-relief task force, EPA is considering easing lead restrictions. An agency spokesman says a decision isn't expected for several months, as comments from industry, favoring proposed changes, are weighed against those of health officials, vehemently opposing them. The latter say that any progress with lead-poisoning in recent years is due mainly to lead reductions in "regular" gasoline and the requirement that new cars use only unleaded gasoline. Indeed, the mean level of lead in children's blood has gone down 25.6% since the EPA curbs began.

Impaired Mental Capacities

While few lives are lost to lead poisoning, compared with a decade back, many more are seriously damaged than was once thought. The study, by the National Center for Health Satistics, found "at least 50% more" children than expected with chronically high lead levels, says Vernon Houk, the U.S. Center for Disease Control's environmental health services director. The big casualty: mental capacities.

"Lead won't kill these kids," says Devra Lee Davis, director of the Environmental Law Institute in Washington. "It will make them a little dumber, make life a little harder. And for many of them, life's already tough. Elevated levels of lead just drains the quality of their lives."

In view of the ubiquitous and unescapable lead contamination of our biosphere measures already in motion to reduce exposure to lead in air and in atmospheric fallout must be made even more stringent and the timetables to achieve these reductions must be accelerated.

For as one of this country's wisest founders, Benjamin Franklin, lamented in his famous letter on lead poisoning -

"how long must a useful truth be known and exist
before it is generally receiv'd and practis'd upon."

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APPENDIX I

Advantages and disadvantages of direct measures of lead exposure

Method	Advantages	Disadvantages
Blood lead	Useful for screening programs	Measures only recent exposure; variable laboratory measurements; affected by physiologic state (e.g., infections, acidosis); collection procedures may be difficult in young children
EP[a]	Useful for screening; earlier indicator of exposure than blood lead; unaffected by contamination with environmental lead; can also be used as indicator of pre-anemia state	Levels increased in iron deficiency as well as with lead exposure; measures only recent exposure
Examination of red cells for basophilic stippling	—	Not always found in chronic clinical lead poisoning; relatively insensitive to lesser degrees of lead exposure
Inhibition of δ-ALAD[b] activity, as assayed *in vitro* in circulating erythrocytes	Measurable effects at fairly low levels of lead exposure (20 μg/dl)	Limited availability of test
Coproporphyrin excretion in urine	Measurable effects at fairly low levels of lead exposure (40 μg/dl)	Measures only recent exposure
δ-ALA excretion in urine	Measurable effects at fairly low levels of lead exposure (40 μg/dl)	Measures only recent exposure
Calcium disodium EDTA[c] mobilization test	Provides measure of mobile or potentially toxic fraction of total body lead burden	Potentially dangerous if blood lead level > 70 μg/dl; difficult to collect specimens in young children
X-ray of long bone	Measure of past exposure	Not useful for measurement of recent exposure; "lead lines" seldom seen in children < 24 months of age; high false negative rate; radiation exposure
Flat plate of abdomen	—	Shows ingested foreign material only within preceding 24–36 hr; high false negative rate; radiation exposure
ZPP[d]	Measures average exposure over approximately the prior 4 months; less variable under conditions of variable exposure than blood lead	Measures only relatively recent exposure
Dentine lead	Quantitates cumulative exposure; no loss of lead over time; dose-related measure of exposure; not affected by chelation; easily sampled in young children; can accurately measure low level exposures	Unable to determine precise age at exposure
Hair	Easily sampled	Uncertain whether hair reflects a body metal burden; easily contaminated by environmental lead

[a] Erythrocyte protoporphyrin.
[b] δ-Aminolevulinic acid dehydratase.
[c] Ethylene diaminetetraacetate.
[d] Zinc protoporphyrin.

APPENDIX II

Risk Classifications for Asymptomatic Children[26]

BLOOD LEAD (μg/dl whole blood)	ERYTHROCYTE PROTOPORPHYRIN (μg/dl whole blood)			
	≤ 49	50–109	110–249	≥ 250
Not done	I	*	*	*
≤ 29	I	Ia	Ia	EPP+
30–49	Ib	II	III	III
50–69	**	III	III	IV
≥ 70	**	**	IV	IV

EPP+ = Erythropoietic protoporphyria—Although rarely iron deficiency may cause EP elevations to 300 μg/dl.
* = Blood lead necessary to estimate risk.
** = Combination of results not generally observed in practice; if observed, retest with venous blood immediately.

NOTE: Diagnostic evaluation should be provided more urgently than the classification would otherwise indicate in the following cases:

1. Children with any symptoms compatible with lead poisoning.
2. Children under 36 months of age.
3. Children whose blood lead and EP values place them in the upper part of a particular class.

It must be emphasized the suggested guidelines refer to the interpretation of screening results, but the final diagnosis and disposition rest on a more complete medical and laboratory examination of the individual child.

Criteria for management of children with lead poisoning

Criteria	Treatment	Environmental action needed
Lead encephalopathy Neurologic symptoms of lead intoxication	Intensive chelation (dimercaprol plus CaNaEDTA every 4 hours)	Child kept in hospital until corrective measures are taken
Lead intoxication Blood lead level >60 μg/dl and/or other symptoms of lead toxicity	Dimercaprol and CaNaEDTA alternated every 8 hours	Same as for lead encephalopathy
Increased body burden of lead Blood lead level between 30 and 59 μg/dl and:		
■ Strongly positive CaNaEDTA test	CaNaEDTA for 5 days	Investigation of home for source of lead and corrective measures
■ Mildly positive CaNaEDTA test	Penicillamine daily	
■ Barely positive CaNaEDTA test	Follow-up only	

LEAD POISONING: TREATMENT PRIORITY OUTLINE

Class		Treatment
IV	Blood lead ≥ 70 mcg/dl Erythrocyte protoporphyrin ≥ 250 mcg/dl	Immediate hospitalization (a) BL ≥ 100: CaEDTA + BAL (5-day course) (b) BL 70-99 ☹ symptoms: CaEDTA + BAL (5-day course) (c) BL 70-99 ☺ symptoms: CaEDTA + BAL for first 48 hours only
III	Blood lead 50-69 EP 110-249	If asymptomatic: CaEDTA mobilization test If positive: CaEDTA for full course
II	Blood lead 30-49 EP 50-109	Terminate exposure, treat iron deficiency and other nutritional problems
I	Blood lead < 30 EP < 50	Normal

Modifying factors:

1. Age—12 to 30 months → peak toxicity (older children tolerate better)
2. Season—summertime peak of encephalopathy
3. Intercurrent illness—fever, dehydration
4. All cases, if *symptomatic*, will be treated *without* mobilization test.

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