

Testimony on

AIR QUALITY STANDARD FOR Lead. Environmental Health Aspects.

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REFERENCES

Patterson, C. C.

Contaminated and natural lead environments of man
Arch Environ Health 11:344-360 (1965)

Goldsmith, J. R., and Hexter, A. C.

Respiratory exposure to lead: epidemiological and experimental dose-response relationships.

Science 158 (3797): 132-134 (1967)

Schroeder, H. A., and Tipton, I. H.

The human body burden of lead
Arch Environ Health 17:965-978 (1968)

Epstein, S. S., and Mantel, N.

Carcinogenicity of tetraethyl lead.
Experientia 24:580 (1968)

Ferm, V. H.

The syneratogenic effect of lead and cadmium
Experientia 25:56 (1969)

Roe, F.J.C., and Lancaster, M.C.

Natural metallic and other substances as carcinogens
Brit. Med. Bull. 20:127 (1964)

Muro, L. A.

Chromosome damage in experimental lead poisoning
Arch Path 87:660 (1969)

Hernberg, S., Nikkanen, J.

Enzyme inhibition by lead under normal urban conditions
Lancet 1:63-64 (1960)

Health aspects of human exposure to environmental lead.

The toxicity of lead is well documented 1) from occupational exposure in years past before industrial hygiene standards were enforced, and 2) from acute lead poisoning observed in children who have eaten old, peeled, lead paint.

The principal target of this type of toxicity is the brain; clinical damage is ordinarily associated with blood levels of 80-100 mcg/100 ml blood, or higher.

These observations are based on many clinical studies. However, we are still painfully ignorant of many aspects of the biological effects of lead. For example, we know essentially nothing of the mechanism by which lead damages nerve cells in the brain. We can then only speculate about the possible low-level chronic damage that may be connected with blood levels in the range of 15-30 mcg/100 ml. We do know that most urban residents carry this burden for most of their lives.

The distribution of lead within the body is also not well known. We do know that lead tends to follow calcium into the bones, where it is presumably inert except at very high levels. We can assume that such lead may be mobilized back into the body circulation during periods of stress and calcium demand (for example milk production during the nursing period) but we have no definite knowledge of the clinical consequences.

Higher levels of lead are also associated with changes in the red blood cells -- for example "stippling" changes, which are helpful in the diagnosis of lead poisoning.

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Besides these clinical observations in human disease connected with lead poisoning, several studies have shown that lead (or tetra-ethyl lead) can have other forms of toxicity in experimental situations, when tested in animals. The most alarming of these is cancer of the kidney; in addition there have been isolated (but uncontradicted) reports of leukemia, of congenital malformations, and of chromosome breakage. All of these studies used very high doses of lead, as is necessary and customary in animal experimentation. If the expected damage in man were merely proportional to the (reduced) dose in common experience, the result would still be quite alarming. We do not know how to choose between this speculation, and the alternative one, that the lower doses are absolutely harmless.

Henry Schroeder has reported long-term feeding experiments on rats at low doses of lead, and found a definite but non-specific reduction in life span and vitality. In many ways, his findings may be the most alarming of all in assessing the chronic toxicity of our exposure in present-day urban environments, and they deserve to be repeated in a variety of circumstances and by other investigators and with other animals. In a further study of his own, he failed to find these effects of lead, in diets that were also enriched with chromium, a trace metal that he believes is generally deficient in our highly refined diets.

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Most recently, S. Hernberg and J. Nikkanen reported that commonly occurring blood levels of lead, around 15 mcg/100 ml, in "healthy" subjects were associated with a reduced activity of an enzyme, "ALADH" in the red cells. The health significance of this alteration is not known, nor do we know (as yet) what may be happening with respect to many other enzymes and biological functions at such levels of lead -- functions known to be impaired at higher levels.

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A substantial percentage of the population, especially those of African ancestry or from the Mediterranean basin, are known to carry genes for unusual forms of red cell hemoglobin or of another enzyme, G6PDH. (The distribution of these genes among Africans and among Italians and Greeks is believed to be related to an advantage in coping with malaria, which was highly prevalent in their ancestral homes in earlier generations). These mutations render these individuals more vulnerable to various kinds of chemical damage to the blood; their sensitivity to lead has simply not been studied, but it would be surprising if it were not influenced. But for that matter, we really do not know how lead is absorbed from the gut and the lungs into the blood stream, nor how it is combined when it is in the blood. This is vitally important, for it is quite likely that total blood levels are a sum of several parts -- some complexes that may be detoxicated complexes of lead, some that may reflect its attack on vital body functions.

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We then must attempt to make reasonable policies with an unreasonable poverty of information. All we know for certain is that lead burdens about three times higher than seen in most urbanites are dangerous threats to life, a narrow margin for which no other example is obvious. Just because intermediate levels of lead are so prevalent, it is impossible to draw firm conclusions about their health effects on the general population. We do know that the life expectancy trends in industrialized countries have reached a plateau, and sometimes a small downturn, in recent years; and most authorities attribute at least part of this to environmental pollutions.

The economic advantage of the use of lead additives averages out to about \$5 per capita per year; most of it is achieved with the first 20% of the amount of lead actually used. Our problem is to balance the unknown, but potentially enormous, long-term health hazards against the calculable short-term economic advantages. (I believe these will be fairly short-term for two reasons: 1) that the petroleum industry will find other economical routes to efficient octane values, and 2) that our uncertainties about the health impact of lead will be resolved with further research.)

Several approaches towards equitable resolution might be considered. For example, the lead industry might be directly compelled, or indirectly taxed, to purchase an insurance policy to cover health damages that might be claimed in future by many citizens. At present, the gamble that lead additives are harmless is advocated by the industry, but they will lose very little if they were touting the wrong bet. These kinds of measures would require contentious and elaborate new laws. A stringent air quality standard is the one effective method of protecting the public interest in the face of these uncertainties.