

Conclusions of the Committee on Human Health Consequences of Lead Exposure from Automobile Emissions*

by Hans L. Falk

In 1975, Dr. David P. Rall, Chairman of the DHEW Committee to Coordinate Toxicology and Related Programs asked that a committee of departmental experts be formed to examine and report on human health consequences of lead exposure from automobile emissions. The committee submitted its report in July 1976, and the report was circulated to experts and organizations in and outside Government for comment.

The following pages contain a summary of the conclusions of the Committee. This is followed by a series of papers prepared by members of the committee and modified, where necessary, by comments received from reviewers.

The Committee desires to record its thanks to the individual reviewers who devoted much time and thought to the consideration of the documents. Their work has added considerably to its value.

In accordance with its mandate, the Committee focused its attention on the specific subject of the health consequences resulting from lead exposure, and the contribution of automobile emissions to such exposure. It did not therefore address itself to a consideration of the economic or other consequences resulting from a phase-out of lead in gasoline, and apart from making general comments on the chemical emissions from alternative antiknock compounds, it felt that there was not enough information available to evaluate the health consequences of these replacements for lead.

Apart from a general consideration of possible ultimate effects on health, the Committee did not examine in detail the broader ecological effects of the increasing global lead contamination of the earth's surface. Concentrations of lead in the polar ice caps and a consideration of the time-frame in which these have been deposited gives an indication of the amounts of lead which have been or are being deposited in areas far distant from the sources of emission, and assists in calculations of the amounts being deposited in the oceans. Lead particles from industrial sources, from lead added to gasoline, and from the natural lead content of other fuels are deposited and some will be conveyed by aquatic runoff to accumulate in river, lake, and ocean sediments. In sharp contrast to the ease with which certain other toxic elements, such as mercury, are conveyed upwards to man via aquatic food chains, the amounts of lead so conveyed are small except in the case of molluscs and crustaceans. Marked localized increases in the lead content of these have been found in the vicinity of lead smelters and lead industries. It is unlikely that lead from gasoline has had a sufficient effect on the lead content of the sea to affect man. In the soil, lead concentrates in the surface layers, and little is removed by leaching. The amounts absorbed by the root fibrils of plants are usually small with comparatively little translocated upwards. Only when the amounts in soil are very large does the lead content of plant tissue increase markedly (1-4).

The report is designed to provide a balanced assessment of the adverse health effects due to lead exposure and the various factors affecting lead toxicity, in order to assist in assessing the relative

*Members of the Committee were: Dr. Hans L. Falk (Chairman), Dr. Terri Damstra, Dr. Kathryn Mahaffey, Dr. Warren T. Piver, and Dr. Herbert S. Posner.

importance of the lead contribution from automobile emissions as compared with other sources of human intake such as the diet. Many reviews have been published on the subject; where certain aspects have already been assessed in detail, the evidence is summarized briefly in order to allow more attention to be devoted to those newer aspects where a definitive assessment is still needed.

A proportion of the lead in gasoline is retained in the engine and exhaust systems; the remainder is emitted in particulate form. The finer particles remain airborne for varying lengths of time; the larger particles are deposited more quickly. They contaminate paved surfaces, soil, vegetation, and sediments resulting from aquatic run-off. As a constituent of dirt and dusts, they may be transported by wind and other agents and contaminate both indoor and outdoor places where people live and children play. The tetraethyl- or tetramethyllead in gasoline is largely converted in inorganic lead during combustion in the engine. The amounts of organic lead emitted or evaporating to contaminate the ambient air of highways or the forecourts of filling stations are small and rapidly disperse (2, 4).

About ten years ago, typical average lead levels in the atmosphere ranged from $11.3 \mu\text{g}/\text{m}^3$ or higher near busy expressways, to $1\text{--}4 \mu\text{g}/\text{m}^3$ in the central areas of many cities, $0.01\text{--}0.5 \mu\text{g}/\text{m}^3$ in suburban and rural areas, and as low as $0.02 \mu\text{g}/\text{m}^3$ in remote areas (2). Indoor values are usually considerably lower than outdoor values (5). There are indications that such levels in the environment are now falling and are likely to decline further as the result of increasing use of lead-free gasoline. Recent work has suggested that in adults approximately 35% of inhaled lead is retained in the lungs, and it has been calculated that continuous exposure to $1 \mu\text{g Pb}/\text{m}^3$ of air from engine exhausts would result in an increase of approximately $1 \mu\text{g Pb}/100 \text{ ml}$ blood (6). Other attempts at deducing such a relationship have yielded results mostly varying from $1.5 \mu\text{g Pb}/100 \text{ ml}$ blood for each $1 \mu\text{g Pb}/\text{m}^3$ air down to an insignificant amount (7-11).

These figures suggest that the increased blood lead levels which would result from the airborne lead to which most urban dwellers are exposed might be in the region of $1\text{--}2 \mu\text{g Pb}/100 \text{ ml}$ blood. Population studies have shown that urban blood lead levels are significantly higher than suburban, yet the evidence of a correlation between airborne lead and blood lead levels in such areas is inconclusive (9). Confounding factors may include the constantly varying, hour to hour, exposures of individuals, the added effects of lead dusts, individual differences in lead metabolism, and possible differences in dietetic and social factors.

Blood lead relationships do not, however, give a satisfactory indication of potential risks. In adults most of the lead ingested is eliminated in the feces and urine. On an average only about $10 \mu\text{g}/\text{day}$, less than 4% of the average daily intake, is stored (12). Of the total body burden 95% is stored in bone, more than 70% in dense bone (13). It is the lead in the more vascular cancellous bone and in the soft tissues (including the erythrocytes, kidney, and brain) which may be more available to cause toxic reactions either *in situ* or as a result of mobilization. In children skeletal lead levels are lower, though increasing with age, but the skeletal system is more vascular, and therefore the proportion of available lead is likely to be greater.

Lead in dirt and dusts is likely to make an important contribution to the lead intake of some children, either as a result of direct "mouthing" habits (pica) or by the ingestion of lead on the hands. The inhalation of dust from the floor or ground, as for instance from children playing on contaminated carpets or on contaminated pavements during dry, windy weather, may also make some contribution.

Lead from automobile exhausts deposited on vegetation is largely in the vicinity of roads, but the finer particles may travel long distances before being deposited on soil and vegetation (1, 14-18). Fortunately, with most vegetables and fruits the outer portion, more liable to be contaminated, is either discarded or washed or constitutes a small proportion of the total weight. Similarly, the amount of lead from automotive emissions deposited on the soil and ultimately reaching man's food either in fruit or vegetables or in meat and fish (including shellfish) would be small.

For comparative purposes the committee surveyed the lead intake in the diet. Although only some 10% of the lead in food is absorbed, this constitutes the major intake from nonindustrial sources. Apart from cases of accidental or unintentional contamination of foods such as lead in canned infant foods, in wines, or in shellfish taken from contaminated coastal areas, the total intake from food is difficult to reduce.

The Committee also surveyed the general toxicologic properties of lead in the human body. The evidence indicates that children are more susceptible to lead poisoning than adults, their lead intake and absorption per kilogram body weight is likely to be higher, and they are more liable than adults to suffer from lead encephalopathy with its resulting permanent impairment of the nervous system. Possible effects of lead as an etiological agent in neurological disease, in peripheral neuropathy, in behavioral aberrations, its possible effects on reproduction and postnatal developments, on

chromosomes and on the immune, endocrine, and renal systems were each considered. At levels of intake not associated with undue or unusual exposure to lead, certain biochemical changes are observed although their relationship to harmful toxic effects is unclear. More information is needed to define subclinical effects, evaluate their significance, and determine threshold limits separating subclinical effects from levels resulting in specific symptoms and symptom complexes. There is insufficient knowledge of the long-term health effects of low-level lead exposure.

Difficulties have been experienced in finding satisfactory biological indices of lead hazard, and the results obtained by the use of different indices are frequently conflicting. There is no satisfactory index of tissue levels in many organs such as the brain or of the total body burden. The latter would be of little use without an indication of the chelatable fraction. Blood lead values are the most frequently used index, but they have many drawbacks, since they are influenced by recent transitory exposures and by the hematocrit level. Inhibition of δ -aminolevulinic acid dehydrase (ALAD) has recently been used rather extensively, but it is influenced by factors other than lead and is not a good index at the lower levels of lead exposure. Among other indices, serum or plasma lead levels have been rarely used but should provide a good measure of the available circulating lead, though they will fluctuate rapidly with variations in intake. Free erythrocyte protoporphyrin (FEP) has been advanced recently as a more stable index of available circulating lead and is less influenced by transitory changes. More work is needed to determine the best available indices for routine screening and research purposes and to determine the correlations between the results derived from various indices and the factors influencing them.

Considerable ambiguity has frequently arisen as a result of looseness in the use of the word "normal" in defining average values or ranges in different populations, and also because of inherent ambiguities in the word as it is used in a variety of concepts.

Recommendations

Lead emission from automobile exhaust is one of the sources of a toxic element which can be eliminated. While the current contribution may not of itself present an obvious immediate serious hazard, it is one source of exposure which can and sometimes does contribute to urgent and serious health consequences in individuals.

Even if current levels of lead exposure for most

children are too low at the time of writing to have a clear-cut adverse effect, it may simply be a matter of time until a critical level is reached for future generations. Symptoms and disease states might be facilitated because of the build-up of lead in many environments. The general ecologic undesirability of contributing unnecessarily to global surface contamination with such a potentially toxic substance is apparent.

The Committee therefore recommends the phasing out of lead in gasoline by reducing its content as has been proposed in the U. S. by EPA and by many other developed countries. It agrees with the current requirements which ensure that all new vehicles are designed to run on lead-free gasoline and constructed so that leaded fuel cannot be used in them. However, many existing vehicles requiring leaded fuels will continue in use for some years, and the Committee recommends that gasoline for these vehicles should have the minimum amount of lead consistent with efficient functioning, and that such vehicles be fitted with a filtration or other device to prevent further contamination of the environment.

Any replacement of lead by other fuel additives or by engineering modifications to the engine or exhaust system should have a reduction in adverse health effects as its primary consideration.

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