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Sources of Lead in the Urban Environment

Severe lead poisoning, often producing encephalopathy with significant residual damage to the central nervous system, was diagnosed among children living in inner-cities of the United States well into the late 1960s.[1] Within these urban "lead belts," leaded paint was frequently the vehicle producing the observed severe pediatric cases of lead toxicity.[2] Through the combined efforts of public health agencies, regulation on quantities of lead permitted in paint, as well as dedicated actions of individual physicians and parents, there has been a substantial decline in the prevalence of pediatric lead encephalopathy. Following recognition that a high percentage of children surviving lead-encephalopathy had significant residual central nervous system damage, a major shift in focus from case-finding to prevention occurred in the 1960s. Results of the childhood lead screening programs indicated that repeated ingestion of high-lead paint chips probably accounted for the overtly encephalopathic cases; however, eating paint chips was questionable as a principal cause for mild to moderate cases of lead toxicity.[2]

During the 1970s non-paint sources of lead were more carefully scrutinized. In addition, understanding of the clinical implications of relatively lower level lead exposure greatly expanded.[3] What evolved was recognition that blood lead levels* previously considered typical for urban areas, e.g., 30–50 ug/dl, were associated with adverse effects on central nervous system function,[3] heme biosynthesis,[4] and the kidney.[5,6] It also became evident that in relatively non-urbanized areas of the United States (e.g., Iowa, Wisconsin) children had lower blood lead concentrations. To investigate typical levels of lead exposure for the United States population, measurement of blood lead was included in the second National Health and Nutrition Examination Survey (NHANES II). In contrast to smaller scale studies of specified at-risk populations, NHANES II established the distribution of blood lead levels for the United States population during the period 1976–1980.[7] It demonstrated clearly that urbanization was associated with an increased prevalance of elevated blood lead levels.[8] At the mid-point of the survey (March 1, 1978), 11.6 ± 1.9 per cent of children, ages six months through five years, living in central cities of urban areas with a population of ≥1,000,000 persons had blood lead concentrations ≥30 ug/dl. In contrast, the prevalence of these elevated blood lead concentrations among comparably aged children living in rural areas was far lower, 2.1 ± 0.9 per cent.[8] By comparison, low blood lead levels (<10 ug/dl whole blood) occurred in only 7.1 per cent of children six months through five years of age living in the urban areas of ≥1,000,000 persons, but in rural areas lower blood lead concentrations were much more common, 18.2 per cent.[9]

A continuing controversy surrounds sources of lead to urban residents. Because lead poisoning has frequently been associated with paint, some have assumed that leaded paint is the vehicle for mild to moderate cases of lead toxicity, but others have questioned this assumption. The investigation of lead concentrations in inner-city garden soils, conducted by Mielke, *et al*,[10] and reported in this issue of the *Journal*, sheds new light on the childhood lead exposure problem. Previous site-specific measurements of lead in soil and dust samples collected near lead smelters or heavily

*Although blood lead concentration is not a perfect index of body burden of lead in episodic high-dose lead exposures, it is a much more useful indicator of lead exposure in lower-dose, sustained lead exposures. Of added importance, blood lead has been the indicator of internal lead dose associated with health effects of lead.

traveled freeways have shown the potential for contamination by specific sources.[11] The fractional environmental burden of lead from paint, leaded gasoline, and industrial emissions as lead sources will vary from location to location. The particular significance of the research by Mielke and his colleagues is their demonstration for an entire major urban area that garden soils are consistently and heavily contaminated with lead. In their study, 10 per cent of the soil samples from vegetable gardens collected within a 30-mile radius of downtown Baltimore, Maryland had a lead concentration exceeding 778 ppm. Using cartography and a recently perfected statistical technique termed multi-response permutation procedures, Mielke, *et al,* have determined that the probability of this clustering of high lead soils in the urban area occurring by chance alone is extraordinarily low. It is significant that these garden soil samples had been mixed to a depth of 20–30 cm. Experience with dust and soil samples indicates that soils mixed to this depth typically have far lower lead concentrations than do urban dusts which often contain lead in concentrations of thousands of parts per million.[12] As established by Mielke, *et al,* the urban clustering of high-lead soils cannot be explained simply as lead from paint leaching into soils. Rather a more general urban pattern occurs that is consistent with use of leaded gasoline and traffic density.[10]

Lead from the environment is transferred to people by a variety of routes. In addition to lead ingested from foods grown in these urban gardens, soil and dust are also ingested by children during normal developmental processes (mouthing of hands, toys, and other objects; creeping, etc.). Detailed reviews of the potential for dust as a contributor to the childhood lead problem have been provided by Charney,[13] Roels, *et al,*[14] Duggan,[15] Landrigan, *et al,*[16] and others. Foods grown in garden soils in urban areas can be contaminated by lead either from the soil or from atmospheric deposition of lead on the surface of the plants.[17] In addition, the extent of general contamination of the food supply by lead found in dust and dirt is becoming increasingly evident.[17] Wolnik, *et al,*[18] analyzed over 1,600 samples of raw agricultural commodities collected from normal agricultural areas remote from road-ways or primary metal smelters, and found concentrations of lead that were lower than any previously reported analysis of food samples typically available to Americans.

The maximal permissible intakes of lead in the range of 100–150 ug Pb/day from all sources for infants and young children that I published in 1977 were based on lead-induced health effects recognized in the early to mid-1970s.[19] As our understanding of the range of health effects associated with lower levels of lead exposure has increased, the importance of multiple routes of lead exposure likewise has increased. As environmental lead pollution is brought under control, human lead exposure likewise decreases. For example, following reduction in lead sources during the 1970s a 37 per cent decline in blood lead levels was observed between 1976 and 1980 in the NHANES II data. Although a number of potential factors responsible for the decrease were considered, the most probable explanation is reduced atmospheric pollution from leaded gasoline.[20] These changes reflect public health gains achieved in the 1970s. Because of the persistence of environmental lead, population screening for pediatric lead exposure, especially in urban areas, must be continued and measures to control atmospheric lead pollution must not be relaxed.

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