LEAD IN SOIL;

RECOMMENDED MAXIMUM PERMISSIBLE LEVELS

NEW JERSEY STATE DEPARTMENT OF HEALTH

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Lead in Soil: Recommended Maximum Permissible Levels

Introduction

Environmental exposure to lead has long been recognized as a public health problem particularly among children. The vulnerability of the age group one to five years to soil lead is enhanced because of their hand to mouth activities, pica and a high rate of intestinal absorption. Excessive concentration of lead in soil has been shown to increase lead levels in children.[1-7] As a result, there has been an increasing awareness for the need to monitor lead levels in soil and to control soil lead contamination by maintaining a "safe" level. Given the widespread presence of lead in urban soil, reduction of lead to background uncontaminated levels is not possible.[8] The major focus of this report is to propose a "safe" or permissible level of lead in soil in highly urbanized areas, below which potential adverse health effects will be minimized.

Background

Environmental Assessment

Soil lead contamination has been attributed to various sources[9]. Flaking lead paint, particularly in and around houses or buildings has been considered as a major source of contamination. Air-borne lead particles deposited in soil is another important source. Emissions from industries, incinerators and similar sources and from vehicular traffic using leaded gasoline contribute to soil lead content. Urban environments receive a higher deposition of lead from vehicular emissions than rural areas. Furthermore, lead concentrations in urban soils are not evenly distributed[3].

In general, lead tends to remain at the surface soil and concentrations are lower at deeper layers. Lead-contaminated soil and dust have been identified as important sources of exposure for children especially in an urban setting[4]. Wide variations in soil lead levels have been observed. Studies have reported values ranging from less than 100 ppm to well over 11,000 ppm[8]. In a recent study in Baltimore, the lead levels in garden soil samples ranged from 1.0 ppm to over 10,000 ppm with a median of 100 ppm[3]. Spittler and his co-workers did a similar study on garden soil in Boston[10]. Soil lead levels were higher in inner-cities and near roadways. Also, front yards of homes facing roadways had higher lead contamination than backyards. Automobile and industrial emissions have been found to be mainly responsible for increase in urban soil lead levels. From limited sampling done to date, median values of lead in surface soil samples from different areas in New Jersey varied from 4 ppm to 1245 ppm[11]. The overall median levels were 238 ppm and 73 ppm for suspected contaminated and control sites respectively. Newark had the highest median of 1245 ppm followed by Jersey City (684 ppm), Secaucus (495 ppm) and other towns with levels below 400 ppm. Samples from areas in Princeton and Flemington were below 100 ppm. As observed in earlier studies, front yards of homes in Newark had a higher level (1755 ppm) than backyard (1060 ppm).

The soil samples were prepared with an acid digestion procedure (EPA Method 3050) for analysis by flame atomic absorption spectroscopy (AAS) initially and if negative, furnance AAS was used. A dried and pulverised sample is digested in nitric acid and hydrogen peroxide. The digestate is then refluxed with either nitric acid for the furnace analysis or hydrochloric acid for the flame analysis.

Health Effects

Severe lead toxicity often causes encephalopathy. Prevention of this serious sequelae of lead poisoning was a major focus in the 1960s[12]. During the 1970s, recognition of chronic exposure of lead and its cumulative effect shifted the emphasis to the understanding of the adverse effects of low levels of lead intoxication. The study by Needleman et al showed a positive relationship of lead in shed milk teeth with poor ratings from classroom behavior[13]. These findings supported the 'no threshold' view and also indicated the need for more attention to be given to cumulative adverse effects of lead at low levels of intake. A recent study in Boston[14] emphasized this view with its findings on fetal lead exposure associated to retardation of mental development.

The blood lead concentration has been generally accepted as the best measure of the external dose of lead[8], although it is not considered as a reliable index of past absorption or of toxicity per se. However, Needleman et al had observed that children with higher tooth lead levels tended to have had higher blood lead levels previously (4 or 5 years prior to tooth shedding).

In recent years, progress has been made in achieving the goal to remove lead from the environment of children before it enters their bodies. The Second National Health and Nutrition Examination Survey (NHANES-II) has established average blood lead levels for the US population[15]. These data demonstrated that urbanization was associated with an increased blood lead level. Lead levels in blacks were on an average 6 ug/dl higher than those in whites. The lowest blood lead associated with adverse biological effects has been observed to be 10 ug/dl.[16] ALAD (delta-aminolevulinic acid dehydratase) inhibition is associated with this low level. More serious conditions such as anaemia and neurologic effects occur at

Leaded gasoline makes a substantial contribution to soil and dust lead levels.[17] The reduction of lead in gasoline and removal of lead in paint for residential areas have been primarily responsible for a decline in the average blood lead levels in children on a national basis. In areas with very high concentrations of lead in soil and dust, large scale cleanup operations of soil or relocation of the population will be the ideal remedial actions to protect children from undue lead exposure. Such responsibilities for regulating lead exposure include the setting up of acceptable levels of lead in soil by government agencies.

Material and Method

This report focuses on the method and the computation involved in deriving a suggested permissible level of lead in soil, below which potential hazardous health effects will be minimized. Firstly, a review of the existing literature showed that several studies have investigated the relationship of exposure to lead in soil and dust and the amount of lead absorbed by humans, especially children. The data from these studies led to the finding that lead in soil is positively correlated with blood lead in children. From several studies, U.S. Environmental Protection Agency (EPA) estimated the blood lead slope as ranging from 0.6 to 6.8 ug/dl per 1,000 ug/g of soil lead concentration[18]. Available data on the estimates of the amount of soil ingested by children showed one hundred fold variation and were not considered useful in deriving a "safe" soil level.[19,20,21].

Duggan did an assessment of the relationship of blood lead and lead in soil/dust, based on twenty-one samples out of nine studies, which had data permitting a quantitative estimation of the blood lead slope[22]. His estimate was an increment of about 5 ug/dl in the blood lead level of children for an increment of 1,000 ppm of lead in soil. These studies varied a great deal in the type of soil and the study population. Soil or dust source included various types such as boot tray dust, house dust, outdoor dust, playground dust and soil. Most of these studies were on children under five years of age, a few on older children up to fourteen years, and one on a mixed population of adults and children.

The blood lead slopes, computed by Duggan for all twenty-one samples, were available, ranging from 0.6 ug/dl to 65 ug/dl per 1,000 ppm of lead in soil. The analysis for our present study was based on data on these slopes.

As our objective is to derive an acceptable level for lead in soil, of Duggan's twenty-one slope estimates, eight were selected as they were based on soil alone as the source and only on children under twelve years.

Results and Discussion

Table 1 shows the slopes ranging from 0.6 to 65 ug/dl per 1,000 ppm of these eight studies. As lead levels in blood are known to be distributed lognormally, and the range for slopes (0.6-65.0 ug/dl) is very wide, analysis was done on log transformations of the slopes. The mean of the logs is 0.5321 with a standard error of 0.2435. Transforming back, the geometric mean and the geometric standard error of the slopes is 3.41 $\pm$ 1.75 ug/dl. Applying the "worst case" or upper limit analysis to the logs, the one-tailed 95% upper confidence limit equals 0.5321 +1.65 x .2435=0.9339.[23,24] Transforming back, anti log 0.9339 = 8.5877 ug/dl per 1,000 ppm of lead in soil. This slope corresponds to the worst case situation.

Using the slope 8.59 ug/dl, soil concentrations have been calculated for different amounts of blood lead contributed from soil, as shown in Table 2. Having computed the soil concentration for different amounts of blood lead contributed through soil, the next important consideration is the choice of a permissible amount of blood lead from soil. The soil lead concentration corresponding to this blood lead level would be the suggested permissible level. Keeping in mind the background level of blood lead for children under twelve years, the ideal situation would be to have no increment in blood lead level contributed from soil. This stringent condition demands a zero level concentration of lead in soil. Looking at estimates of soil lead levels available from various studies in the U.S.A. and elsewhere, one can realize that to bring down the lead concentration to zero would be an impratical task. As shown in Table 2, even for 1 ug/dl of blood lead from soil, the soil concentration has to be around 100 ppm. If 5 ug/dl of blood lead is chosen as a tolerable level, the corresponding soil concentration is 582 ppm, rounded off to a figure of 600 ppm. With a suggested permissible level of 600 ppm, it can be stated with reasonable certainty that this soil concentration will contribute no more then 5 ug/dl to blood lead for children under 12 years.

This suggested level of 600 ppm lies within the range given by the Center for Disease Control in the following statement:[25]

"In general, lead in soil and dust appears to be responsible for blood lead levels in children increasing above background levels when the concentration in the soil or dust exceeds 500-1000 ppm."

Furthermore, it is important to keep in mind that exposure of children to lead contaminanted soil or dust is enhanced when they play on non-grassy surfaces than on grass covered area[26], a scenario similar to the vulnerability of children exhibiting mouthing behavior.

In conclusion, maximum permissible levels of lead in soil have been recommended by the New Jersey State Department of Health, based on the dose-response relationship of lead in soil and blood lead in children as follows:

1. A maximum permissible level of 250 ppm of lead in soil is recommended in areas without grass cover and repeatedly used by children below five years of age among whom mouthing objects is highly prevalent. This level may add at the most about 2 ug/dl to the blood lead level of children.

2. A maximum permissible level of 600 ppm of lead in soil is recommended in areas repeatedly used by children below 12 years of age. This level may add at the most 5 ug/dl to blood lead level of children.

3. A maximum permissible level of 1,000 ppm of lead in soil is recommended in areas such as industrial parks or along streets and highways or in other areas infrequented by children.

The Department of Health also recommends that municipalities should consider the passage of local ordinances prohibiting the development of residential areas in lead-contaminated soil unless the lead soil concentration is reduced to the appropriate maximum permissible level.

Summary

Lead in soil has been recognized as a public health problem, particularly among children. In recent years, attention has been directed to cumulative adverse effects of lead at low levels of intake. Lead-contaminated soil and dust have been

identified as important contributors to blood lead levels. Based on available data on blood lead and lead in soil/dust, an approach has been developed to suggest a permissible level of lead in soil, below which there will be reasonable certainty that adverse health effects will not occur. An acceptable level of 600 ppm of lead in soil suggested as a "safe" level would contribute no more than 5 ug/dl to total blood lead of children under twelve years of age. Maximum permissible levels of lead in soil have been recommended based on the dose-response relationship of lead in soil and blood lead in children.

References

1. Lin-Fu, J.S. 1973. Vulnerability of children to lead exposure and toxicity (First of Two Parts). N Engl J Med 289:1229-1233.

2. Lin-Fu, J.S. 1973. Vulnerability of children to lead exposure and toxicity (Second of Two Parts). N Engl J Med 289:1289-1293.

3. Mielke, H.W.; Anderson, J.C.; Berry, K.J.; Mielke, P.W.; Chaney, R.L.; Leech, M: 1983. Lead concentrations in inner-city soils as a factor in the child lead problem. Am J Public Health 73:1366-1369.

4. Duggan, M.J.; Williams, S. 1977 Lead-in-Dust in City Streets. The science of the total environment, Amsterdam: Elsevier Scientific Publishing Company, 7:91-97.

5. Brunekreef, B.; Veenstra, S.J.; Biersteker, K.; Boley, J.S.M. 1981. The Arnhem lead study 1. Lead uptake by 1-to 3- year-old children living in the vicinity of a secondary lead smelter in Arnhem, The Netherlands. Environ Research 25: 441-448.

6. Roels, H.A; Buchet, J.P.; Lauwerys, R.R.; Bruaux, P.; Claeys-Thoreau, F.; Lafontaine,A.; Verduyn, G. 1980 Exposure to lead by the oral and the pulmonary routes of children living in the vicinity of a primary lead smelter. Environ Research 22: 81-94.

7. Schmitt, N.; Philion, J.J.; Larsen, A.A.; Harnadek, M.; Lynch, A.J. 1979. Surface soil as a potential source of lead exposure for young children. CMA Journal 121:1474-1478.

8. National Academy of Sciences. 1980. Lead in the human environment. Report prepared by the Committee on Lead in the Human Environment, National Research Council. National Academy Press, Washington, D.C.

9. American Academy of Pediatrics. 1985. A Statement on childhood lead poisoning. Draft prepared by Committee on Environmental Hazards.

10. Spittler, T.M.; Feder, W.A. 1979. A study of soil contamination and plant lead uptake in Boston urban gardens. Commun. in Soil Science and Plant Analysis 10:1195-1210.

11. New Jersey State Department of Health, Division of Occupational and Environmental Health. 1985. Unpublished Data on Lead in Soil.

12. Mahaffey, K.R. 1983. Sources of lead in the urban environment. Am J Public Health 73:1357-1358.

13. Needleman, H.L.; Gunnoe, C.; Leviton, A.; Reed, R.; Peresie, H.; Moher, G.; Barrett, P. 1979. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. N Engl J Med 300:689-695.

14. Bellinger, D.; Leviton, A.; Waternaux, C.; Needleman, H.; Rabinowitz, M. 1987. Longitudinal analysis of prenatal and postnatal lead exposure and early cognitive development. N Engl J Med 316: 1037-43.

15. Mahaffey, K.R.; Annest, J.L.; Roberts, J.; Murphy, R.S. 1982. National estimates of blood lead levels: United States, 1976-1980 Association with selected demographic and socioeconomic factors. N Engl J Med 307:573-579.

16 Minnesota Department of Health, Division of Maternal and Child Health Services 1984. Lead exposure and the health effects on children. Report to the Minnesota Legislature.

17. Caprio, R.J.; Margulis, H.L.; Joselow, M.M. 1974. Lead absorption in children and its relationship to urban traffic densities. Arch Environ Health 28: 195-197.

18. U.S. Environmental Protection Agency, Office of Research and Development 1983. Air Quality Criteria for Lead Volume I of IV. Research Triangle ParK NC: U.S. Environmental Protection Agency; EPA Report No. EPA-600/8-83-028A.

19. Binder, S.; Sokal, D., and Maughan, D. 1986. Estimating soil ingestion: The use of tracer elements in estimating the amount of soil ingested by young children. Arch Environ Health 41: 341-345.

20. Clausing, P.; Brunekreef, B.; and van Wijnen, J.H. 1987. A method for estimating soil ingestion by children. Int Arch Occup Environ Health 59:73-82.

21. Hawley, J.K. 1985. Assessment of health risk from exposure to contaminated soil. Risk Analysis 5: 289-302.

22. Duggan, M.J. 1980. Lead in urban dust: An assessment. Water, Air and Soil Pollution, Boston: D. Reidel Publishing Company, 309-321.

23. American Industrial Health Council. 1985. Proposals to improve scientific risk assessment for chemical carcinogens: Dose response evaluation and characterization of risk. Scientific working paper by American Industrial Health Council, Washington, D.C. 33-38.

24. Wilson, R.; Crouch, E. 1982. Risk/benefit analysis. Cambridge, MA: Ballinger Publishing Company, 61-63.

25. Centers for Disease Control. 1985. Preventing lead poisoning in young children. U.S. Department of Health and Human Services, Atlanta, Georgia.

26. Lewis and Clark County Health Dept.; Montana Dept. of Health and Environmental Sciences; Center for Environmental Health, Centers for Disease Control; Public Health Service, U.S. Dept. of Health and Human Services; U.S. Environmental Protection Agency. 1986. East Helena, Montana child lead study, summer 1983. Final Report, 36.

TABLE I

Data Relating to Lead in Blood with Lead in Soil*

Author and Reference	Number of Persons in Study	Age of Persons (yr)	Slope (ug/dl/1,000 ppm)
Angle et al (i)	153	2-5	65.0
Angle et al (ii)	25	10-12	15.0
Barltrop et al	82	2	0.6
Galke et al (i)	187	up to 5	3.3
Galke et al (ii)	187	up to 5	1.6
Shellshear et al	68	1-5	3.9
Yankel et al (i)	1,149	1-9	0.6
Yankel et al (ii)	1,149	2-3	2.5

*Source: Duggan MJ. Lead in Urban Dust: An Assessment. Water, Air and Soil Pollution, Boston: D. Reidel Publishing Company, 1980; 309-321.

TABLE 2

Lead Concentration in Soil by Blood Lead Contribution from Soil

Blood Lead from Soil* (ug/dl)	Soil Concentration (ppm) at 95% Upper Confidence Limit of 8.59 ug/dl 1,000 ppm
1	116
5	582
10	1,164
15	1,746
20	2,328
25	2,910

*In addition to background level