

◆ Health Effects Review ◆

Volume 1 Issue 6

May 1996

ENVIRONMENTAL LEAD

INTRODUCTION

Lead has been widely used for many centuries, and its adverse health effects on workers were recognized as long ago as the second century BC by Greek physicians. In the 1800s, Australian physicians identified lead-based paint as a cause of lead poisoning in children. In recent years, researchers have observed more subtle health effects of lead exposures below those causing frank poisoning.¹ In 1991, the U.S. Centers for Disease Control and Prevention lowered the blood lead level guideline for the prevention of health effects in children from 25 to 10 ug/dl, and the same change was made by Health Canada in 1994.²

Over the past 40 years, many countries have taken regulatory actions to reduce environmental and occupational exposures to lead, including the elimination of lead additives, such as tetraethyl lead, in gasoline, and restrictions on lead use in food cans, drinking water plumbing, glass or ceramic utensils and house paint.³ Limiting the use of lead in fuels has proven a remarkably effective way to reduce lead pollution. USEPA⁴ found a 94% reduction in lead emissions to air between 1978 and 1987, most of which was attributed to the phase-out of lead in gasoline. Two recent studies from Spain and Switzerland have shown blood lead level decreases of approximately 50% shortly (5-10 years) after restrictions are placed on lead content of gasoline.^{5,6}

As summarized by Silbergeld,³ concerted international efforts may achieve more efficient and rapid reduction of lead exposure risks. Lead emissions can cross national boundaries, and increasing emissions from leaded fuel use in developing countries increases the global loading of lead to the environment, while also putting the citizens of developing countries at great risk. Lead-containing products,

such as lead-soldered food cans, are part of the international trade market, and scientists have suggested that the most effective means of reducing risk from that exposure is an international agreement to eliminate lead solder use. The Organization for Economic Cooperation and Development (OECD) began an international chemical risk reduction project in 1990, with lead as one of the top five candidates for reduction efforts. The U.S. has proposed a set of lead reduction efforts, and has the support of Nordic and European Union countries; however, several member countries, including Canada, have argued for voluntary actions by the lead industry

instead of regulatory action by member countries. A proposal for some international action was to be brought to the OECD Ministerial Council in 1996. In addition, the U.N. Commission on Sustainable Development sponsored a conference in 1995 to discuss the problems caused by lead in gasoline, which engendered some bilateral projects between the U.S. and several developing nations.

Despite these efforts, extraction and production of lead continues to increase. Silbergeld³ found that primary and secondary lead production actually increased worldwide from about 2 million tons in 1960 to 5.4 million tons in 1993.

LEAD IN THE GREAT LAKES REGION

Lead enters the Great Lakes from the atmosphere and tributary waters. People may be exposed to lead directly from the Great Lakes in drinking water. However, lead in the Great Lakes does not appear to be the major source for lead exposure for people residing in the Great Lakes basin, since lead levels in drinking water from the Great Lakes rarely exceed water quality guidelines (5 ug/l in U.S., 10 ug/l in Canada).

Nriagu et al.⁷ studied changes in metal concentrations in the Great Lakes, and found lead in concentrations ranging up to 32 ng/l (or ppt); the authors note that the concentrations are lower than those in the open ocean. The average concentrations ranged from 3.2 ng/l in Lake Superior to 9.9 ng/l in Lake Ontario. The lead concentration distribution was found to be related to the presence of loading sources such as industrialized areas. Estimated atmospheric input of lead ranged from 820 ug/m²/yr in Lake Ontario to 3760 ug/m²/yr in Lake Erie, while calculated residence times for lead were lower than those for other metals (0.02 to 0.57 years). The authors conclude that a large fraction of lead in atmospheric aerosols is soluble in water and likely to be quickly hydrolyzed or scavenged by particles in the water, resulting in a fairly rapid turnover of lead in the lakes.

Researchers in Windsor⁸ summarized data from 1975-1990 on lead in drinking water sources in six Great Lakes basin cities, and found that mean "raw" water lead concentrations were below the detection limits in Gary, Indiana and two Ontario cities, Thunder Bay and Toronto. The maximum levels measured in treated drinking water during 1989 and 1990 were below detection limits in all six cities but Rochester, New York, where a maximum of 10 ug/l was found in 1990. In previous years, lead concentrations in treated drinking water only exceeded the U.S. drinking water standard (50 ug/dl at that time) in one city (Milwaukee, Wisconsin) where water samples were drawn from the distribution network and not directly from the treatment plant.

Bernier et al.⁹ cited preliminary results from a cohort study of Great Lakes fish-eaters that show a small increase in lead levels among fish-eaters (mean 3.7 ug/dl) compared to non fish-eaters (mean 3.0 ug/dl). However, Rice,¹⁰ in a recent review of neurotoxic pollutants in the Great Lakes, concluded that Great Lakes fish do not provide an elevated intake of lead compared to other foods.

Environmental lead exposures tend to be higher in urban centers. Silbergeld³ summarized data from the U.S. EPA showing median air lead concentrations that range from 1.075 ug/m³ in urban areas to 0.33 and 0.10 ug/m³ in suburban and rural areas, respectively. In Northern Ontario, air lead levels in 1992 ranged from non-detect to 0.04 ug/m³ (mean 0.015 ug/m³).²

In the U.S. in 1989-91, Silbergeld³ reported median blood lead levels (in ug/dl) to be 3.6 and 2.1 for male and female white persons, 4.7 and 2.8 for male and female black persons, and 4.0 and 2.3 male and female Mexican-Americans.³ A report of lead surveillance in the U.S. adults shows that the total number of people with lead levels exceeding 25 ug/dl decreased 10% between 1994 and 1995.¹¹ In Canada, blood lead concentrations have been lower than in the U.S. A survey of 395 children in Northern Ontario found an average blood lead level of 3.91 ug/dl (4.38 and 3.45 ug/dl for boys and girls, respectively).² However, according to Flegal and Smith¹², the natural background blood lead level in humans is approximately 0.016 ug/dl, which is approximately 600-fold lower than the level considered acceptable in children (10 ug/dl).

A monthly review and summary of the scientific literature on human health effects and environmental pollutants, with an emphasis on pollutants of the Great Lakes ecosystem. Prepared under the direction of the Health Professionals Task Force of the International Joint Commission. This does not represent the official position of the International Joint Commission.

*Health Professionals Task Force
Secretary: Jim Houston
International Joint Commission
Canada Section
100 Metcalfe Street
Ottawa, Ontario K1P 5M1
phone (613) 995-0230
fax (613) 993-5583*

HEALTH EFFECTS

Lead has been associated with toxic effects in numerous systems in the body. As summarized by the USEPA,⁴ the major low-level effects of concern include (1) heme biosynthesis and related functions; (2) neurological development and function, (3) reproduction and physical development, (4) kidney function, and (5) cardiovascular function. Some recent studies include:

* A recent study suggests that men with blood lead levels of 40 ug/l (the U.S. worker protection criteria) or more have reduced sperm count and total number when compared to men with blood lead levels of 15 ug/dl or lower.¹³

* New results from the Normative Aging Study in Boston indicate that low-level exposure to lead may impair renal function in middle-aged and older men at blood lead levels below 10, 25 and 40 ug/dl.¹⁴ Another report from this cohort indicates that higher blood lead levels were associated with increased diastolic blood pressure, though not in men on anti-hypertensive drugs.¹⁵

* A recent study compared peripheral nerve function in lead-exposed workers (mean blood lead level of 36.9 ug/dl) to those in a referent group (mean blood lead level of 10.5 ug/dl), and found decreased function to be associated with higher blood lead levels (above 40 ug/dl).¹⁶

The greatest cause for concern appears to be the findings of adverse effects of low-level lead exposure on children's development. Rice¹ recently reviewed human and animal studies of lead's behavioral effects, and found increased distractibility, inability to inhibit inappropriate responding, perseveration and inability to change response strategy to be behavioral changes observed in many studies of low level lead exposure to young humans and animals. In humans, reduced IQ has been associated with lead exposure in early life, while in animal studies, deficits have been observed on a wide variety of tests that assess attention, learning or memory.

A recent report¹⁷ finds that lead exposure in early childhood results in continued developmental deficits in older children. A cohort of children (375 in this report), now 11-13 years of age, who live near a lead smelter in Port Pirie, Australia has been followed since birth. The geometric mean blood lead level in the cohort was 8.3 ug/dl at birth (umbilical cord blood), 21.2 ug/dl at 2 years, 11.6 ug/dl at 7 years, and 7.9 ug/dl in this study. There was a consistent inverse relation between blood lead level and scores for all IQ scales, unadjusted for covariates. With adjustment for confounders such as gender and socioeconomic measures, the association of IQ with maternal or cord blood lead became insignificant, but the inverse associations remained significant or marginally significant with blood lead measurements at 15 months to 7 years of age. It is estimated that the mean score for full scale IQ declined by 3.0 points for a doubling (10-20 ug/dl) of lifetime average blood lead concentration at age 11-13. These findings are in agreement with those of earlier studies; however, the authors note that their estimate of "effect" was a little higher than most, but not all, earlier studies.

Another recent study in Massachusetts¹⁸ found a dose-response relationship between hair lead levels and a number of Boston Teacher's Rating scales in first grade children, with no evidence of a "safe" threshold level. In a meta-analysis of studies on low-level lead exposure effects on children, Schwartz¹⁹ found no apparent threshold for the negative association between IQ and lead level. For a 10 ug/dl increase in lead level, the predicted IQ reduction from studies with a mean blood lead level of less than 15 ug/dl was 3.23 points, compared with a reduction of 2.32 IQ points in studies with a mean blood lead level above 15 ug/dl.

Interestingly, researchers at the University of Pittsburgh¹⁹ found significantly (p=0.03) poorer cognitive function to be associated with higher blood lead levels in older women (>65 years) from a rural community (mean lead level 4.5 ug/dl). The association was not found for the women living in an urban community.

Though lead is toxic to many organs and systems, there may be common cellular mechanisms of toxicity. Lead is known to interfere with the body's use of calcium and zinc (all are divalent cations). Calcium is central to many cellular processes, and zinc is a component of a number of important enzymes, including an enzyme needed for hemoglobin production.¹² Tong et al.¹⁷ also propose a mechanism for observed developmental deficits in lead-exposed children that is based on findings in animal studies that lead alters the release processes for neurotransmitters such as dopamine, norepinephrine or acetylcholine by interfering with calcium metabolism or nerve synapse function.

NEED FOR CONTINUED REDUCTION IN LEAD EXPOSURE

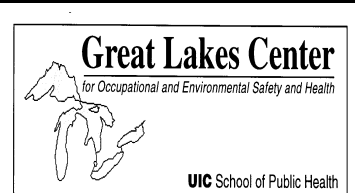
Clearly, lead remains as a threat to public health on a global basis as well as in the Great Lakes basin, though the primary exposure source may not be the Great Lakes themselves. In the U.S. and Canada, blood lead levels have declined, largely as a result of regulatory limits on the use of lead in gasoline. However, remaining sources of environmental lead include

(1) lead-based paint, (2) lead contamination of soils, and (3) industrial point sources of lead which can raise lead levels in the local area. Based on recent research findings, it appears that there may be no "safe" threshold of lead exposure to protect developing children from developmental harm.^{18,19}

As summarized by Proctor et al.,¹⁵ a shift in the distribution of neurobehavioral or intelligence test scores can result in a dramatic societal change. A 3-point reduction in average IQ score may bring about a large public health "cost" to our society. The economic benefit of

reducing lead exposure has been assessed by Salkever,²¹ who used updated information on the economic effects of reduced educational achievement to reevaluate an earlier analysis that found a total economic benefit of \$6.937 billion for a 1 ug/dl reduction in blood lead for one year's cohort of children. Of that total amount, \$5.060 billion was determined to be the benefit of avoiding earnings losses for those children. With updated economic information, it was found that that estimate is increased by at least 50%, or an additional \$2.5 billion for a 1 ug/dl lead reduction in a single birth cohort.

As Hryhorczuk²² emphasizes the need for international action to reduce sources of lead exposure on a global basis, including leaded gasoline, paint, food cans, glass and ceramics, cosmetics and wastes such as lead acid batteries.



PREPARED BY:

Great Lakes Center for
Occupational and Environmental
Safety and Health
School of Public Health
University of Illinois at Chicago
2121 West Taylor Street
Chicago, Illinois 60612-7260
(312) 996-7887
email: mross1@uic.edu

Project Coordinator:
Mary A. Ross, MA
Senior Science Advisor:



World Health Organization
Collaborating Centre for Occupational
and Environmental Health

REFERENCES:

- 1) Rice DC. 1996. Environ Health Perspect. 104(Suppl 2):337-351.
- 2) Smith LF, Rea E. 1995. Can J Public Health 86(6):373-376.
- 3) Silbergeld E. 1995. Int J Occup Environ Health 1(4):336-348.
- 4) USEPA. 1990. Review of the national ambient air quality standards for lead: staff paper. EPA-450/2-89-022. USEPA, OAQPS, Research Triangle Park NC 27711
- 5) Schuhmacher M, Belles M, Rico A, Comingo JL, Corbella J. 1996. Sci Tot Environ 184:203-209.
- 6) Wietlisbach V, Rickenbach M, Berode M, Guillemin M. 1995. Environ Res 68:82-90.
- 7) Nriagu JO, Lawson G, Wong HKT, Cheam V. 1996 Environ Sci Technol 30:178-187.
- 8) Henshaw PF, Bewtra JK, Biswas N. 1993. J Great Lakes Res 19(3):521-532.
- 9) Bernier J, Brousseau P, Krzystyniak K et al. 1995 Environ Health Perspect 104(Suppl 9):23-34.
- 10) Rice D. 1995. Environ Health Perspect. 103(Suppl 9):71-87.
- 11) MMWR. 1996 (Apr 26) 45(16):333-4.
- 12) Flegal AR, Smith DR. 1995. Rev Environ Contam Toxicol 143:1-45.
- 13) Alexander BH, Checkoway H, et al. 1996. Occ Environ Med 53:411-416.
- 14) Kim R, Rotnitzky A, Sparrow D, et al. 1996. JAMA 275(15):1177-1181.
- 15) Proctor SP, Rotnitzky A, Sparrow D et al. 1996. Am J Epidemiol 143(3):528-536.
- 16) Chia SE, Chia HP, Ong CN, Jeyaratnam J. 1996. Occup Med 46(1):59-64.
- 17) Tong S, Baghurst P, McMichael A, et al. BMJ 312:1569-1575.
- 18) Tuthill, RW. 1996. Arch Environ Health 51(3):214-220.
- 19) Schwartz J. 1994. Environ Res 65:42-55.
- 20) Muldoon SB, Cauley JA, et al. 1996. Neuroepidemiol 15:62-72.
- 21) Salkever DS. 1995. Environ Res 70:1-6.
- 22) Hryhorczuk DO. 1995. Int J Occup Environ Health 1(4):366-7.