

## Case Study #1: Standard Setting for Lead - The Cautionary Tale

<b>8.1 INTRODUCTION</b> .....	<b>227</b>
<b>8.2 EXPOSURE</b> .....	<b>228</b>
8.2.1 <i>Uses, Sources, Media and Routes of Exposure</i> .....	228
8.2.2 <i>Blood-Lead Surveys in Canadian Children</i> .....	231
8.2.3 <i>Risk Factors for Children</i> .....	232
<b>8.3 HEALTH CONCERNS</b> .....	<b>234</b>
8.3.1 <i>Introduction</i> .....	234
8.3.2 <i>Lowering the “intervention” level</i> .....	234
8.3.3 <i>A Systemic Poison</i> .....	236
8.3.4 <i>Approaches to Studying the Neurotoxicology of Lead</i> .....	238
8.3.4.2 <i>Prospective and Longitudinal Studies</i> .....	241
8.3.5 <i>Lead and Behaviour</i> .....	242
8.3.6 <i>Summary</i> .....	243
<b>8.4 THE REGULATORY RESPONSE</b> .....	<b>244</b>
8.4.1 <i>Lead in Gasoline</i> .....	245
8.4.1.1 <i>Regulation of Lead in Gasoline in the United States</i> .....	248
8.4.1.2 <i>Regulation of Lead in Gasoline in Canada</i> .....	251
8.4.2 <i>Smelters and Soil</i> .....	255
8.4.3 <i>Ontario’s Multi-Media Approach</i> .....	256
8.4.4 <i>Lead in Drinking Water</i> .....	259
8.4.5 <i>Lead in Food</i> .....	259
8.4.6 <i>Lead in Consumer Products</i> .....	261
8.4.6.1 <i>Introduction</i> .....	261
8.4.6.2 <i>Lead in Ceramics, Glassware and Kettles</i> .....	261
8.4.6.3 <i>Lead in Paint</i> .....	262
8.4.6.4 <i>New and Unexpected Sources</i> .....	264
8.4.6.5 <i>Health Canada’s Lead Reduction Strategy</i> .....	267
8.4.7 <i>The OECD Declaration of Risk Reduction for Lead</i> .....	270
8.4.8 <i>Blood-Lead Testing and Follow-Up</i> .....	271
8.4.8.1 <i>Approaches in the United States</i> .....	271
8.4.8.2 <i>Canadian Comparisons</i> .....	272
8.4.8.3 <i>Pediatric Management of Lead Toxicity in Canada</i> .....	273
<b>8.5 CONCLUSIONS AND LESSONS LEARNED</b> .....	<b>273</b>
<b>8.6 RECOMMENDATIONS</b> .....	<b>277</b>
<b>8.7 REFERENCES CITED</b> .....	<b>278</b>

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### 8.1 INTRODUCTION

Medical and scientific understanding of the health effects in children of lead poisoning is extraordinarily detailed. This knowledge arises from a large number of studies investigating health effects in children exposed to environmental lead contamination in industrialized countries. Health effects are asymptomatic or sub-clinical, are both cognitive and behavioural, and include developmental delays, deficits in intellectual performance and neurobehavioural functioning, decreased stature, diminished hearing acuity and reduced attention span. The effects of low-level lead poisoning in children may be irreversible and there may be no threshold for health effects.<sup>1</sup> Numerous risk factors predispose children to both higher exposure and greater vulnerability to the hazards of lead.

Human activity, primarily during the 20th Century, has created global contamination with this persistent neurotoxin. A gradient is apparent that closely follows traffic patterns; contamination is highest in urban areas and along motor ways and decreases with distance as traffic concentration subsides. This pattern is an historical though still relevant one in most industrialized countries and is being repeated throughout the developing world where lead is frequently still allowed in gasoline.

Additional lead exposure from numerous sources, including food and water but especially lead in paint, has coincided with lead from gasoline. In some communities, child lead burdens also have been greatly increased by point sources of industrial lead contamination. New and unexpected sources of lead in consumer products have arisen frequently in recent years such as lead in imported crayons, plastic mini-blinds, some candle wicks, and a range of children's toys and clothing/accessories.

The regulatory response to controlling lead in the environment and consumer products is the cautionary tale. An early warning in 1904 from an Australian doctor about the hazard to children from lead in paint and the need for regulation was greeted with derision by both medical professionals and those with commercial interests in lead compounds. By the 1920s, the Australian Medical Congress passed a resolution seeking a ban on lead in paint. The regulation of lead in paint did not begin in North America until 50 years later. Also in the 1920s, warnings about the public health consequences of allowing lead in gasoline were largely dismissed. Again, it was not until nearly 50 years later, when worldwide automotive lead pollution had reached 350,000 tons/year, that regulating the level of lead in gasoline was contemplated. In North America, it was to be a twenty year battle that was not decided in favour of banning lead in gasoline until scientists were able to clearly show that millions of children were already affected.

Canadian regulation of lead has followed and consistently lagged behind or been less stringent than regulatory action taken in the United States. Changes to lead standards in Ontario that were proposed (but not entirely implemented) in the early 1990s provide examples of a number of positive aspects of environmental standard setting as well as ongoing shortcomings. In all the categories reviewed herein, with the exception of lead in gasoline, current regulations of lead, primarily by the federal government, are as yet non-existent (in the case of many consumer products) or they are either ambiguous half-measures or dangerously out of date.

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<sup>1</sup> The discussion in this case study of the health effects of lead and the regulatory response relies upon the reader having access to the discussion of epidemiology and causation in Chapter 4.

In response to the seemingly unending and unexpected number of consumer products containing lead, (crayons, mini-blinds, children's toys, etc.) "regulation" by the federal government generally takes the form of "consumer advisories" but only after a problem has been discovered usually as a result of mandatory blood-lead testing programs in the United States. These "advisories" are used even for products capable of causing serious and acute lead poisoning. No regulatory powers exist to require consumer product recalls.

Overwhelmingly, the public policy approach in Canada is one of accommodation by seeking voluntary measures from those with commercial interests in lead-containing products. However, a regulation for children's products proposed in early 1999, would, if implemented, reverse a three decade trend of delayed, reactive and in some cases inadequate regulatory responses and instead make Canada a world leader in *preventing* childhood lead exposure. It remains to be seen whether this possible sea-change in the Canadian regulatory approach to controlling lead in children's environments will pass muster with those who ensure regulatory action does not conflict with other commitments to international trade agreements. Previous behaviour of Canadian negotiators on the international scene is inconsistent with this promised new approach to domestic regulation.

Further comparisons between Canada and the U.S. are noteworthy with respect to blood-lead testing and the pediatric management of lead toxicity in Canada. The case study concludes with a series of health and policy-related recommendations.

## 8.2 EXPOSURE

### 8.2.1 *Uses, Sources, Media and Routes of Exposure*

Human use of lead dates back 6000 years to ancient Egypt and medical historians are fairly certain that widespread lead poisoning of the upper class contributed to the fall of the Roman Empire. Despite having long known that lead is poisonous, its plentiful availability and physical and chemical characteristics have given rise to many applications. Ancient lead uses were similar to many modern applications including using lead to obtain bright whites and yellows in pottery glazes, for solder, weights, plummets, sinkers, caulking, writing tools, coins, trinkets and trademarks.<sup>2</sup>

Figure 8.1 shows the historical trend. The inset graph in Figure 8.1 shows the extent of global lead contamination wrought by human use of lead. Lead in the polar ice caps began to rise at the beginning of the Industrial Revolution and then increased exponentially after lead was added to gasoline in the 1920s. Lead contamination during the 20th century has increased environmental lead levels so much that they are now hundreds to thousands of times higher than natural background levels.<sup>3</sup>

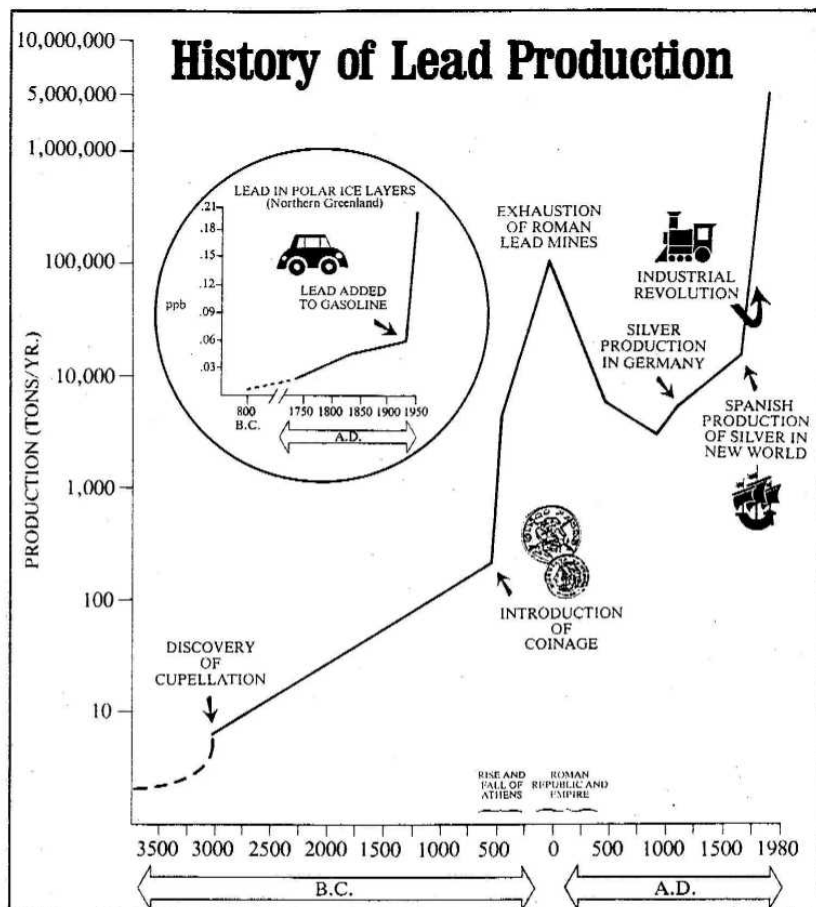
Global lead contamination has been achieved primarily via dispersal of leaded gasoline emissions. A gradient of contamination has been identified that closely follows traffic patterns; contamination is

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<sup>2</sup> Nriagu, J.R. Saturnine Gout Among Roman Aristocrats: Did Lead Poisoning Contribute to the Fall of the Empire?, *New Engl. J. of Med.*, 11 (1983), pp. 660-663.

<sup>3</sup> See: Settle, D.M. and C.C. Patterson. Lead in Albacore: Guide to Lead Pollution in Humans, *Science*, 207 (1980), pp. 1167-1176; and Murozumi, M., T.J. Chow and C.C. Patterson. Chemical Concentrations of Pollutant Lead Aerosols, Terrestrial Dusts and Sea Salts in Greenland and Antarctic Snow Strata, *Geochim. Cosmochim. Acta*, 33 (1969), pp. 1247-1294.

highest in urban areas and along motor ways and decreases with distance as traffic concentration subsides. With the removal of lead from gasoline (in the late 1980s in the United States, and as of January 1st 1990 in Canada) this pattern is now an historical one and lead exposure prevention activities, particularly in the United States, have been based on the assumption, at least since 1991, that the greater, or at least equally important lead risk now exists from old lead-based paints.<sup>4</sup>



**Figure 8.1. History of Lead Production.**

Estimated tons of lead produced per year worldwide since about 3000 B.C. The present level is about 5 million tons per year. Note that the scale for tons per year is logarithmic (each unit of increase is ten times larger than the preceding unit of increase). The insert shows measurements of lead in parts per billion in polar ice layers since about 1750.

(Source: Seattle, D.M. *et al.*, 1980, *op.cit.*, and Murozumi, *et al.*, 1969, *op.cit.* reproduced from Wallace and Cooper, 1986, *op.cit.*)

However, since lead is persistent and binds to soil and dust particles, roadside lead contamination remains a significant and primarily urban, exposure medium. Indeed, a recent study of lead contamination in urban areas of the United States confirms that soil represents a giant reservoir of lead and that children's blood lead levels are strongly correlated with soil-lead concentrations. The association between soil lead and blood lead was 12 orders of magnitude stronger than the association between the age of housing (the older

<sup>4</sup> See, for example, Centers for Disease Control. *Preventing Lead Poisoning in Young Children*. United States Department of Health and Human Services. (1991)

the house, the greater the chance of lead-bearing paint) and blood lead levels. The study clearly showed that the source of this lead contaminated soil is the historical use of lead in gasoline.<sup>5</sup>

During the mid-1980s, soil lead levels ranging from 150 to 3000 parts per million were typical in Canadian urban areas. Where elevated soil lead levels are found, levels of lead in street dust and house dust can be expected to be comparable and generally slightly higher than the soil lead levels.<sup>6</sup> By way of comparison, the soil removal guideline in Ontario for residential soil is 200 parts per million (but only for the clean-up of contaminated industrial lands). Suburban and rural soil lead levels are lower and overall, it is reasonable to expect that the urban levels recorded in the 1980s are higher than would be found now. The removal of lead from gasoline in 1990 means that soil lead levels will very gradually decrease over time.

In “developing” nations, massive lead contamination is occurring from the continued use of leaded gasoline. Lead levels along roads in Nigeria approach 7000 parts per million, about 15 times higher than the level used to designate a toxic Superfund Site in the U.S. In Mexico City, half the children tested have dangerous levels of lead in their blood<sup>7</sup> and in Cairo, more than 300 infants die annually due to maternal lead exposure.<sup>8</sup>

Other sources and media contributing to lead exposure can be generally classified as either industrial or household. Industrial sources contributing to environmental burdens include industries directly involved in the mining, milling and smelting of lead ore bodies or the secondary recovery of lead from products such as batteries and cables. Copper, nickel, zinc, iron and steel production also add to environmental lead contamination as does the burning of coal, and the incineration of waste oil, garbage and sewage. Point sources of lead such as primary and secondary smelters have contributed to very high levels of localized contamination in some communities. In Canada these have included most notably Trail, British Columbia, Bathurst, New Brunswick, (primary smelters) and the South Riverdale and Niagara neighbourhoods in Toronto (secondary smelters).

Household sources and media can include old paint chips and dust, use of lead solder for plumbing or stained glass, cigarette smoke and dust, some pottery glazing and glazes, lead foil packaging, some toys and figurines, lead crystal, fishing sinkers, bullets, some folk remedies and cosmetics, some baby bottles and soothers, some imported crayons, plastic mini-blinds and home playground equipment. Finally, lead contamination of food occurs from diverse sources. Drinking water can be contaminated with lead from the external water source, old lead pipes, and via leaching of lead from soldered joints when water is left standing in lead-soldered copper pipes for several hours.<sup>9</sup>

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<sup>5</sup> Mielke, H.W. Lead in the Inner Cities: Policies to reduce children’s exposure to lead may be overlooking a major source of lead in the environment. *American Scientist*, 87 (1998), pp. 62-73.

<sup>6</sup> Nriagu, J.O. Lead Contamination of the Canadian Environment. In: *Health Effects of Lead*, M.C.B. Hotz (ed.), (Royal Society of Canada Commission on Lead in the Environment, Toronto, 1986), pp. 61-77.

<sup>7</sup> Morris, David, *The Ethyl Corporation: Back to the Future*. Institute for Local Self Reliance. (Sept. 9, 1997) ([www.ilsr.org](http://www.ilsr.org))

<sup>8</sup> The World Bank Group, (1996) Press release No. 96/68S at [www.worldbank.org/html/extr/gaspr.htm](http://www.worldbank.org/html/extr/gaspr.htm)

<sup>9</sup> Lead sources information summarized from numerous sources including ATSDR (Agency for Toxic Substances and Disease Registry), (1988). *The nature and extent of lead poisoning in the United States: a report to Congress*. Atlanta; (Royal Society of Canada Commission on Lead in the Environment, 1985) Final Report and other sources. Note that the use of lead to solder copper pipes has been gradually phased out in Ontario (see Section 8.4.4 below).

Within the huge array of potential sources and media contributing to lead exposure, it is important to sort out the relative contribution of each and the routes of exposure for children. Exposure will vary depending on location, age of housing, and individual social and household circumstances. The relative contribution of different routes of exposure has also changed significantly over time. For example, in Canada two very different kinds of high level exposure were being identified in the late 1960s: lead in paint and lead from industrial point sources. In both circumstances, very high levels of lead caused serious cases of clinical lead poisoning. On the one hand, lead poisoning was found to be occurring when children chewed on furniture or toys covered with paint containing high lead levels; on the other, industrial point sources created high lead levels in soil and house dust which greatly increased children's lead burden. In contrast, in the population at large, investigations during the 1970s and 80s revealed a broad array of lead sources. The single largest source was identified as the environmental dispersal of lead from gasoline which was responsible for 70 to 90% of environmental contamination when it was still used in gasoline.

In general, exposure occurs via four basic media: food, dust or dirt/soil, air and water and via two routes, inhalation and ingestion. Baseline exposure from these four media and exposure routes can be calculated for the population at large. While this framework for viewing exposure is relatively comprehensive, a fifth category needs to be added: lead from consumer products which are increasingly a new and significant source of lead exposure. Exposure from products such as plastic mini-blinds or children's toys are an interior source of lead via direct contact with lead-contaminated dust. Such exposure might be caught under the exposure category of house dust but only if appropriately recognized during a risk assessment. As discussed in Section 8.4.6.4 below, Health Canada's risk assessment of lead in plastic mini-blinds overlooked this fact and, along with additional errors, significantly underestimated the house dust exposure pathway. Finally, transplacental exposure is highly relevant for lead exposure, as is breast milk, although to a lesser extent.

Children are generally more highly exposed to lead than adults. Urban dwellers tend to be more highly exposed than those in rural areas, again because of the historical contamination of the environment from the use of leaded gasoline. However, blood-lead levels in remote and urban populations are no longer reflecting this gradient<sup>10</sup> likely due to the passage of time since gasoline lead phase-out. Additional exposure can arise from occupational circumstances, hobbies or other lifestyle factors. Then there are the special circumstances of point sources, which can often be "hotspots" of contamination such as lead industries or otherwise contaminated sites that can affect adjacent communities and increase their lead exposure above the baseline expected in the population at large.

Actual exposure data for Ontario children was estimated in 1993 as part of Ontario's Multi-Media Approach to revising standards for lead (discussed further in 8.4.3 below). The exposure assessment component of the study found that approximately 24% of exposure results from food; 64% results from soil; 11% from drinking water; and less than 1% from direct inhalation.<sup>11</sup> Consumer products as a lead exposure source were not factored into the calculations.

### **8.2.2 Blood-Lead Surveys in Canadian Children**

Surveys of blood-lead levels in Canada have been limited to studies around known sources of industrial

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<sup>10</sup> Smith, L. and E. Rea. Low blood lead levels in Northern Ontario - what now? *Can. J. Public Health*, 86 (1995), pp. 373-376.

<sup>11</sup> Ontario Ministry of Environment and Energy. *Rationale for the Development of Soil, Drinking Water, and Air Quality Criteria for Lead*. Hazardous Contaminants Branch. (October, 1993)

contamination, the Ontario-wide survey conducted in 1984 and additional community-focused surveys in southern and northern Ontario during the 1980s. As well, community surveys were conducted in Vancouver, Alberta, and Quebec in the late 1980s. There was also a national survey of blood-lead levels conducted in 1978 as part of the Canada Health Survey. However, as noted in reviews by both Environment Canada and Health Canada, the findings are not considered reliable. The overall distribution was not log normal as would be expected in such a survey and 27% of observations were at or less than 1  $\mu\text{g}/\text{dL}$  (microgram/decilitre),<sup>12</sup> an unusual characteristic which, experts have noted, casts doubt on the entire study.<sup>13</sup>

In 1994, a federal-provincial committee on environmental and occupational health cautiously estimated that as many as 66,285 urban children in Canada may have blood-lead levels greater than 10  $\mu\text{g}/\text{dL}$ , the level where lead can begin to cause health effects in young children. This report also noted that blood-lead levels have been steadily decreasing since the 1970s.<sup>14</sup> It is reasonable to expect, with the elimination in 1990 of lead from gasoline, that this decrease has continued during the 1990s. A corresponding decrease in blood-lead levels and the number of children with elevated blood-lead levels is also likely. Limited data indicate that mean blood-lead levels in both urban and rural children in Ontario are approximately 3  $\mu\text{g}/\text{dL}$ .<sup>15</sup>

### 8.2.3 Risk Factors for Children

Young children (six years of age and younger) are at greater risk from lead exposure than are older children or adults for several reasons related to behaviour and physiology. Play patterns, hand-to-mouth activity, and occasionally *pica* (eating soil and non-food items), bring young children into greater contact with materials that may be contaminated with lead. Physiological characteristics in children that create greater lead exposure include: a higher degree of gastrointestinal absorption of lead; higher dietary intake per unit body weight; higher respiratory volume relative to body size; and differences in distribution of lead in the body.<sup>16</sup> Adults store 99% of absorbed lead in bones and teeth whereas children store only 70% with the balance remaining in circulation and available to soft tissues, especially the brain. Fetuses *in utero* are also at risk from lead exposure since lead readily crosses the placenta. Since lead stored in bone

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<sup>12</sup> A deliberate choice was made in this case study to avoid metric nomenclature in describing blood-lead levels. Since the majority of scientific literature on lead reports blood lead measures in micrograms per decilitre, stepping away from Canadian conventions has been done for convenience in making easy comparisons.

<sup>13</sup> Environment Canada. Socio-Economic Impact Analysis of Lead Phase-Down Control Options, Environmental Protection Service, (1984), p. 16; Health Canada. Blood Lead Intervention Levels and Strategies: Update of Evidence for Low-Level Effects of Lead and Blood Lead Intervention Levels and Strategies--Final Report of the Working Group. Federal-Provincial Committee on Environmental and Occupational Health. Environmental Health Directorate. (September, 1994), pp. 22-30; and, Statistics Canada. The Health of Canadians, Report of the Canada Health Survey, Supply and Services Canada, Cat. No. 82-538E. (Ottawa, 1981)

<sup>14</sup> Health Canada. Blood Lead Intervention Levels and Strategies: Update of Evidence for Low-Level Effects of Lead and Blood Lead Intervention Levels and Strategies--Final Report of the Working Group. Federal-Provincial Committee on Environmental and Occupational Health. Environmental Health Directorate. (September, 1994), pp. iii and p.24.

<sup>15</sup> Personal communication with Dr. Lesbia Smith, Ontario Ministry of Health. (May, 1999)

<sup>16</sup> Bellinger, D. Developmental Effects of Lead. Childhood Lead Poisoning: What's New, What's Sadly Not. In: *Proceedings of the 1998 Children at Risk Conference Environmental Health Issues in the Great Lakes Region*. (Chicago, July 8-9, 1998)

is known to go into circulation during pregnancy and lactation,<sup>17</sup> total female body burden and ongoing exposure (via placenta) in pregnant women constitutes an endogenous source of fetal lead exposure.

Nutritional status also significantly affects lead uptake and toxicity. Mushak and Crocetti<sup>18</sup> have reported on the large body of literature showing that deficiencies or alterations in essential nutrients like calcium, iron, phosphorus and zinc will enhance lead exposure and increase the degree of lead toxicity associated with such exposure. These investigators have further reported<sup>19</sup> on the socio-economic and demographic status of those most affected. In the United States, low income African-American children and African-American women of child-bearing age residing in densely populated urban areas have the highest elevations of blood-lead levels. Black male children are especially impacted. The study also reports that nutritional status in these population groups is sub-optimal particularly for calcium and iron. Both of these nutrients have a strong, inverse interactive effect on lead absorption and toxicity.

In Ontario, the first major study to determine blood lead levels and risk factors in Ontario children (conducted in 1984)<sup>20</sup> indicated that children who were younger, male, and from families of lower socio-economic status had higher blood lead levels. Higher levels were also strongly associated with several features of children's homes (apartment dwelling, fireplace, radiator heating systems, lack of an air filter, linoleum flooring and peeling paint in children's rooms or play areas) and neighbourhoods, (local traffic density, proximity to gasoline stations and industry) as well as local environmental levels (air and soil) of lead. The majority of these risk factors reflected the presence of lead in gasoline. The contribution of diet (and nutritional status) to childhood lead intake was not adequately addressed in this study, although it is a recognized risk factor.

Another recognized risk factor, socio-economic status, is of increasing importance in Ontario as child poverty levels increase.<sup>21</sup> Numerous population studies have confirmed the relationship between lead levels and social indicators of disadvantage.<sup>22</sup> For example, lack of suitable play areas, missed meals, and sub-optimal nutrition, poor hygiene, substandard housing (making hygienic conditions more difficult to achieve), inner city location of housing, etc., are all factors that will increase lead exposure in children.

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<sup>17</sup> Agency for Toxic Substances and Disease Registry (ATSDR). *The Nature and Extent of Lead Poisoning in Children in the United States: a report to Congress*. (1988), pp. 15, I-46, III-4 - III-13 and multiple references therein.

<sup>18</sup> Mushak, P. and A.F. Crocetti. Lead and Nutrition: Part I. Biological interactions of lead with nutrients. *Nutrition Today*. 31 (1996), pp. 12-17.

<sup>19</sup> Mushak, P. and A.F. Crocetti. Lead and Nutrition: Part II. Some Potential Impacts of Lead-Nutrient Interactions in U.S. Populations at Risk *Nutrition Today*. 31 (1996), pp. 115-122.

<sup>20</sup> Duncan, C.E., et.al. *Blood Lead and Associated Risk Factors in Ontario Children, 1984*. Ontario Ministry of Health, Ministry of Labour and Ministry of the Environment. (1985)

<sup>21</sup> In 1989, one in ten children in Ontario lived in poverty. Today it is one in five. Armine Yalnizyan. *The Growing Gap*, Centre for Social Justice. ( Toronto, 1998)

<sup>22</sup> The fact was reported in: Ontario Ministry of Environment. *Scientific Criteria Document for Multimedia Environmental Standard Development - Lead*. (1994), p. 131; and confirmed by additional studies since, e.g., Mushak and Crocetti, (1996), *op.cit.*



## 8.3 HEALTH CONCERNS

### 8.3.1 Introduction

People have long known that lead is toxic. Needleman<sup>23</sup> has reported on historical findings of the neurotoxic and other physiologic effects of lead exposure. For example, Dioscorides noticed in the second century B.C. that “lead makes the mind give way” and Benjamin Franklin noted “dry gripes” (colic) and “dangles” (wrist drop) in typesetters and painters. Recent research reveals that lead poisoning likely contributed to the demise of the 1845 Franklin expedition that sought a northwest passage through the Canadian Arctic. High lead content in the ship’s foodstuffs as well as poorly soldered cans likely contributed to both declining health and food spoilage, but also to impaired judgement of those on board ship.<sup>24</sup>

Extensive literature documents the effects of lead poisoning in occupational settings. Symptoms of clinical lead poisoning in adults include colic, anemia and encephalopathy with multiple effects seen in both the central and peripheral nervous systems.

The modern focus on childhood lead poisoning began around 1900 with medical reports of lead poisoning from children eating paint. Symptoms included wrist drop, foot drop, persistent vomiting, colic, encephalopathy, convulsions, anemia and in some cases, death. Until at least the 1960s, most medical professionals considered lead poisoning to be a disease typified by the above symptoms and generally related to a single acute exposure, primarily by ingestion of lead-bearing paint. Also in the 1960s and 1970s, similar, but generally less severe, symptoms were found in children living in communities polluted by industrial point sources of lead such as primary or secondary lead smelters.<sup>25</sup>

As investigations and related regulatory action occurred for these individual sources, the focus also broadened to addressing subclinical or asymptomatic health effects of lead from a multiplicity of low-level, chronic exposures.

### 8.3.2 Lowering the “intervention” level

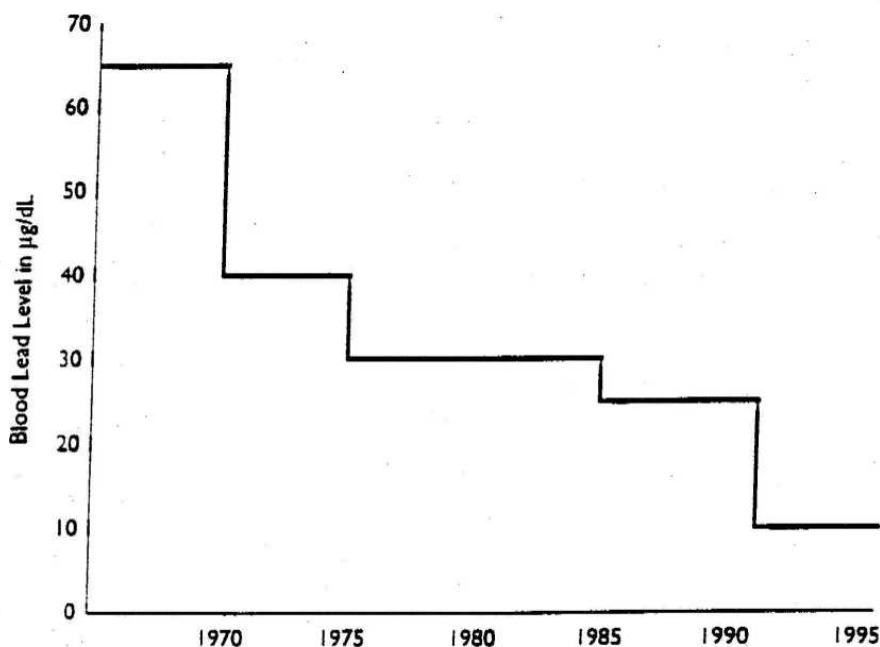
As this research progressed, an increasing range of health effects were shown to be associated with lower and lower blood-lead levels. In the 1950s and 1960s a blood-lead level of 60  $\mu\text{g}/\text{dL}$  in children was considered an “intervention” level, or a point at which steps should be taken to reduce lead exposure. This level was derived primarily from occupational exposure studies and there was limited recognition of differences in susceptibility between adults and children. The United States Centers for Disease Control (CDC) reduced this intervention level to 30  $\mu\text{g}/\text{dL}$  in 1978, then to 25  $\mu\text{g}/\text{dL}$  in 1985 and further to 10

<sup>23</sup> Needleman, H.L., The Current Status of Childhood Lead Toxicity, in *Advances in Pediatrics*. 40 (1993), pp. 125-139.

<sup>24</sup> Owen, J., *Frozen in Time: Unlocking the Secrets of the Franklin Expedition*. (Western Producer Prairie Books, Saskatoon, Sask., 1989)

<sup>25</sup> Summaries of numerous smelter investigations can be found in: Royal Society of Canada Commission on Lead in the Environment. (1985) *Lead in the Canadian Environment: Science and Regulation, Final Report*, Section VIII; USEPA, *Air Quality Criteria for Lead*, Volume I summaries, Volume II, Section 7C, Volume III, sections 11.4 and 11.5(1986); and Millstone, E., *Lead and Public Health*. (Earthscan Publications Ltd., London, 1997), Chapter 3.

$\mu\text{g/dL}$  in 1991<sup>26</sup> (see Figure 8.2). The drop to  $10 \mu\text{g/dL}$  would have occurred earlier but for technical and practical limitations in screening programs. By at least 1987, the CDC stated that it considered  $10\text{--}15 \mu\text{g/dL}$  to be the level where lead-induced health effects occur in children<sup>27</sup> and many medical professionals, including at the CDC, were speculating that there is probably no threshold for lead-induced effects in children.<sup>28</sup> The problem was the use, at that time, of the Erythrocyte Protoporphyrin (EP)<sup>29</sup> test as a screening tool. When blood lead levels are below  $25 \mu\text{g/dL}$ , the EP test is meaningless; no relationship exists between EP and blood lead. The inability to do mass screening with the EP test delayed the reduction of the blood lead intervention level until the CDC could develop mass screening techniques for blood lead. This delay in dropping the intervention level was misrepresented and misused by the Canadian government in deliberations over the phase-down of the Canadian standard for lead in gasoline (see section 8.4.1.2 below).



**Figure 8.2. Lead and Public Health.**

(Source: adapted from Centers for Disease Control, 1991, *op.cit.*, and Millstone, 1997, *op.cit.*)

The evidence behind the steady downward progression of the blood-lead intervention level is a huge body

<sup>26</sup> Preventing Lead Poisoning in Young Children, A Statement by the Centers for Disease Control, United, States Department of Health and Human Services. (October, 1991)

<sup>27</sup> Vernon Houk, Director of the Center for Environmental Health, Centers for Disease Control, Atlanta, Georgia, personal communication with Kathy Cooper, Canadian Environmental Law Association (October 9, 1987) as cited in: Canadian Coalition for Lead-free Gasoline, *Lead in 1988: More Urgent Than Ever*. A brief presented to the Hon. Tom McMillan, Minister of the Environment, and the Hon. Jake Epp, Minister of National Health and Welfare. (June 15, 1988)

<sup>28</sup> More recent studies continue to confirm this hypothesis. David Bellinger, an expert on the neurotoxicity of lead notes that  $10 \mu\text{g/dL}$  has no particular biological significance. Deficits in IQ have been identified in children whose blood-lead levels were never recorded above  $8 \mu\text{g/dL}$  from 0-10 years. Any amount of lead seems to have deleterious effects. See discussion of cross-sectional and longitudinal studies below.

<sup>29</sup> A substance in red blood cells that increases as blood lead increases.

of scientific literature. Lead is the most extensively studied pollutant. As Millstone<sup>30</sup> points out, debate during the last 30 years has not been about whether lead is poisonous but about:

- \* the levels at which adverse effects can be detected;
- \* whether or not there are thresholds below which those effects cease to occur; and
- \* whether or not the results of over-exposure persist or are merely transient.

Evidence of health impacts at lower and lower levels of exposure continues to emerge, the reliability of the evidence has steadily improved and no evidence yet contradicts the suspicion that there may be no threshold below which lead does not exert a toxic effect. Nor is there any evidence to suggest that lead provides any essential or useful biochemical function in humans.

Finally, it is worth noting how tiny an amount of lead these blood lead levels actually represent. For example, it has been calculated by the World Health Organization that a child's lead exposure of 3.7  $\mu\text{g}/\text{kg}/\text{day}$  will result in a blood lead level of 10  $\mu\text{g}/\text{dL}$ . The actual exposure would be 0.06 mg<sup>31</sup> or perhaps the amount of pure lead that could fit on the head of a pin. It is no wonder that extreme lead poisoning can result from childhood exposure to dust and flakes of old lead-bearing paint which can typically contain 20% or even as much as 50% pure lead. For more typical exposure situations, it is easy to see how very low levels of lead contamination from a multiplicity of sources can approach or exceed the amount needed to contribute to a blood lead level of 10  $\mu\text{g}/\text{dL}$ .

### 8.3.3 *A Systemic Poison*

Lead can exert deleterious effects on all major systems of the body including the central and peripheral nervous system, the cardiovascular system, the liver, the kidneys, the gastrointestinal system, the reproductive system and the endocrine system. Inconclusive evidence suggests lead is a probable human carcinogen and may be a teratogen.

Although the huge body of literature on the toxic effects of low level lead exposure focuses on effects on humans, and children in particular, early and ongoing studies on animals provided a warning bell that went unheeded.<sup>32</sup>

In studies of the effects of lead on humans, toxicity begins with lead-caused reductions in heme, the vital substance in the hemoglobin of red blood cells. Figure 8.3 is adapted from an exhaustive review conducted in 1986 by the United States EPA<sup>33</sup> addressing the multi-organ impact of reductions of the heme body pool caused by lead. That review described the cascade of effects seen in multiple physiological processes in many organs and tissues.

The health effects at low level lead exposure (i.e., below 20  $\mu\text{g}/\text{dL}$  and even below 10  $\mu\text{g}/\text{dL}$ ) can be viewed in two broad categories: developmental and neurological. In the area of developmental effects, studies have shown conclusively that lead exposure *in utero* can adversely affect the fetus in terms of

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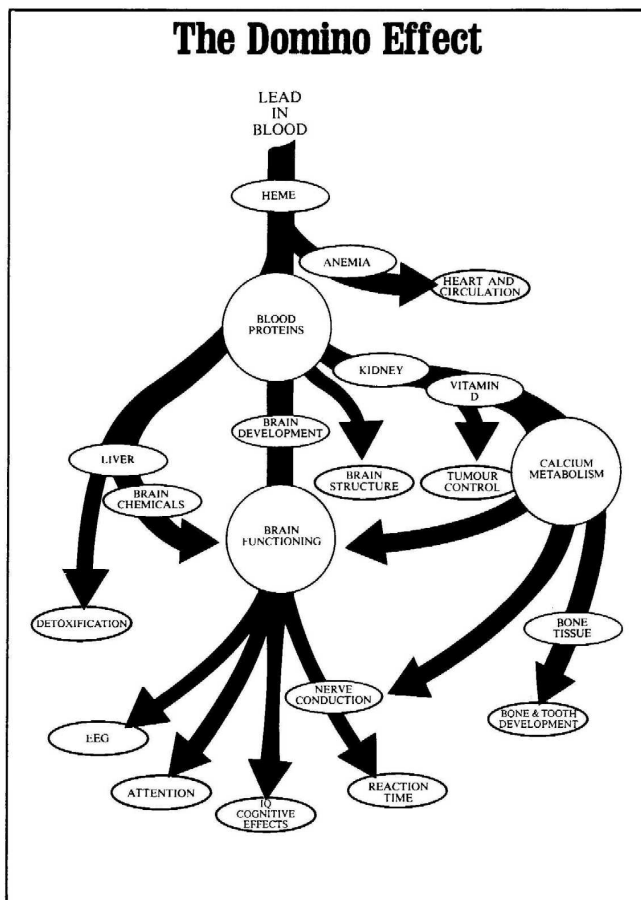
<sup>30</sup> Millstone, Eric. *Lead and Public Health*. (Earthscan Publications Ltd, London, 1997), p.5.

<sup>31</sup> Calculated on the basis of an exposure level of 3.7  $\mu\text{g}/\text{kg}/\text{day}$  for a 13 kg child over 14 days.

<sup>32</sup> See detailed review of the biological effects of lead exposure from animal studies in: United States Environmental Protection Agency. *Air Quality Criteria for Lead*. Vol. IV of IV, Section 12. Pp. 12-1 - 12-370. (1986) EPA-600/8-83/028dF.

<sup>33</sup> *Ibid*, Sections 12 and 13, and Figure 13-4, (1986), pp. 13-31.

reduced gestational age, lower birth weight and smaller head circumference.<sup>34</sup> Lead has also been shown



**Figure 8.3. The “Domino” Effect.**  
 The many impacts of lead-caused reductions in heme.  
 (Source: Adapted from U.S. Environmental Protection Agency, 1984, *op.cit.*, and reproduced from Wallace and Cooper, 1986, *op.cit.*)

<sup>34</sup> Mushak, P., *et.al.* Prenatal and postnatal effects of low-level lead exposure: Integrated summary of a report to the U.S. Congress on childhood lead poisoning, *Environ. Res.* 50 (1989), pp. 11-26; and National Academy of Sciences, National Research Council. *Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations.* (National Academy Press, Washington, D.C. 1993), pp. 31-98; see also Needleman, H.L. and D. Bellinger. The Health Effects of Low Level Exposure to Lead, *Annu. Rev. Publ. Health.* 12 (1991), pp. 111-140.

to contribute to reduced stature in children<sup>35</sup> and to negatively affect children's hearing.<sup>36</sup>

Two areas of neurological effects are apparent: cognitive and behavioural. Cognitive effects have been most extensively documented while the effects of lead on behaviour have received less attention and are less easily studied and quantified. Despite the fact that lead can affect many organs and systems in the body, the health concerns for (and controversy over) low level lead exposure in children are primarily in the area of neurotoxicity.

### 8.3.4 Approaches to Studying the Neurotoxicology of Lead

Different approaches exist in the literature to summarize the many studies showing the neurological effects in children of low level lead. Millstone<sup>37</sup>, following Yule and Rutter<sup>38</sup>, provides a very useful review that categorizes the different studies undertaken over the last 20 to 30 years indicating how evidence has emerged of effects at progressively lower levels, how the reliability of the evidence has progressively strengthened and how statistical analyses, particularly meta-analyses, have confirmed the results of numerous studies that only approached or barely achieved statistical significance. Throughout the 1970s, 80s and 90s, evidence of neurotoxic effects from both animal and human studies continued to grow. As is typically the case with the blunt tool of environmental epidemiology, demonstrating significant or causal effects was difficult. Study results were sometimes equivocal, i.e., suggestive of an effect but only approaching statistical significance. Individual studies were rarely definitive, and results were contested, often by those representing or who had worked for the lead industry. Nevertheless, with the increasing number and sensitivity of studies and improvements in statistical analysis, the body of evidence is now profound.

Six different approaches to studying neurotoxic effects that have been used (roughly in temporal sequence) are:

1. clinical studies of children with high lead levels;
2. studies of "mentally retarded" or behaviourally deviant children;
3. chelation studies;
4. smelter studies;
5. general population cross-sectional studies; and
6. general population prospective/longitudinal studies.<sup>39</sup>

<sup>35</sup> Schwartz, J., *et.al.* Relationship between childhood blood lead levels and stature, *Pediatrics*. 77 (1986), pp. 281-288.

<sup>36</sup> Robinson, G.S., *et.al.* Effects of low to moderate lead exposure on brainstem auditory evoked potentials in children. In: *World Health Organization Regional Office for Europe - Environmental Health Document 3*. (Copenhagen: WHO, 1985), pp. 177-182; Robinson, G.S., *et.al.* Effects of environmental lead exposure on the developing auditory evoked potential and pure tone hearing evaluations in young children. In: *Heavy Metals in the Environment: International Conference, New Orleans*, S.E. Lindberg and T.C. Hutchinson (eds.) (1987), pp. 223-225; and Schwartz, J. and D.A. Otto. Blood lead, hearing thresholds, and neurobehavioral development in children and youth, *Arch. Environ. Health*. 42 (1987), pp. 153-160.

<sup>37</sup> Millstone, Eric. (1997) *op. cit.* Chapter 3.

<sup>38</sup> Yule, W. and M. Rutter. Effects of Lead on Children's Behavior and Cognitive Performance. Kathryn R. Mahaffey (ed.) *Dietary and environmental lead: human health effects.* (Elsevier, Amsterdam, 1985)

<sup>39</sup> after Millstone (1997), *op.cit.* The unfortunate and somewhat archaic choice of the term "mentally retarded" appears to have been chosen by Millstone to reflect the terminology in use when these studies would have been conducted, likely in the 1970s or earlier.

Clinical studies, generally from the 1940s or earlier, of children with high lead levels and obvious symptoms of lead poisoning provided the first indication of lower intelligence and school or behavioural problems. These early clinical studies were unreliable for a variety of reasons including small sample size and inadequate statistical control of confounding factors. Studies of “mentally retarded” or behaviourally deviant children had similar and more serious weaknesses making any possible effect of lead impossible to isolate. Therapeutic chelation studies have been evaluated to determine if chelation therapy (a clinical treatment that extracts lead from patients with high blood lead levels) results in improved performance. Studies have found children to perform better in IQ tests following chelation therapy that lowered their blood lead levels. However, one study noted that optimal nutrition was essential to these results since chelation therapy can leach from the body other essential minerals such as zinc, iron, and manganese. For this reason and other limitations of these study findings, chelation therapy is a valuable part of the clinical response to treating lead poisoning but it is less useful in the research strategy to address the neurotoxicology of low level lead.

The fourth approach has been to study neurological effects in children living in communities adjacent to primary and secondary lead smelters.<sup>40</sup> Such studies were begun during the 1960s and 1970s as health effects and/or poisonings were reported and as concern about the effects of sub-clinical lead poisoning grew. Methodological problems such as the confounding influence of gasoline lead emissions and other variables related to both lead exposure and factors affecting neurological development made many of these studies inconclusive. Some revealed lower intelligence performance in the 40 to 60  $\mu\text{g}/\text{dL}$  range, levels considered greatly elevated now but which were considered acceptable when many of these studies were done. One of the more robust studies undertaken near a lead-zinc smelter in Greece showed a consistent decrease in IQ test performance with increasing blood-lead levels; the effect was only noticeable above the 25  $\mu\text{g}/\text{dL}$  blood-lead level.<sup>41</sup>

The above four approaches contributed to the understanding of the study conditions necessary to isolate the neurotoxicological effects of lead. The studies in the final two approaches - cross-sectional and prospective/longitudinal - applied these lessons including: 1) the need for sufficiently large sample sizes (to detect marginal effects); 2) the care needed to estimate and adjust for confounding factors (i.e., the complex and varied physical and social characteristics of a child’s background); and 3) the need to address the issue of causality. The first and second issues were, over time, addressed in cross-sectional studies; the third was addressed to a certain extent in cross-sectional studies but definitively addressed in five prospective or longitudinal general population studies.<sup>42</sup>

#### **8.3.4.1 Cross-sectional studies and meta-analysis**<sup>43</sup>

Between 1972 and 1990, 27 cross-sectional studies correlated body lead burdens (either through lead content of shed teeth or blood or both) and neuropsychological functioning at a single point in time. One of the most influential was Needleman’s study of tooth lead as a measure of past exposure. The study found, after control of covariates, that the children with higher tooth lead levels had IQ scores six points

<sup>40</sup> Note that Millstone’s distinction between his 4th category of “smelter studies” and his 5th category of “cross-sectional studies” is somewhat arbitrary since some of the smelter studies are also “cross-sectional” studies. The six categories do however provide a useful way of dividing up a huge body of scientific inquiry in particular by describing the roughly chronological flow of research towards the current, and enormous, body of literature documenting the neurological effects of lead in children.

<sup>41</sup> Hatzakis, A. *et.al.* Psychometric Intelligence Deficits in Lead-exposed Children, in Smith, M.A. *et.al.* (eds.) *Lead Exposure and Child Development*. (Kluwer Academic Publishers, Dordrecht, 1988), pp.211-223.

<sup>42</sup> Millstone (1997), *op. cit.*, p. 37.

<sup>43</sup> As reviewed in Millstone (1997), *op. cit.*

lower than their low-lead counterparts.<sup>44</sup> For many years thereafter, much ink and angst were spent by Needleman and his detractors in defending and discounting the results of this study.

The study had its shortcomings but it was joined by 26 others, some of which found comparable results, (i.e., statistically significant association between very low blood lead levels and neurotoxic effects), some which found no association, and others which were equivocal. Although these studies rarely showed a definitive relationship between lead and neurotoxic effects, more studies tended towards demonstrating positive associations (i.e., statistically significant associations) than not. Moreover, the methods for both evaluating IQ and also identifying and appropriately controlling for confounding variables were hotly debated. By the end of the 1980s, the sum of evidence, although not fully conclusive, (but supported by an increasing body of similar evidence from animal studies of the neurologic insult of lead), raised enough concern for the Centers for Disease Control to state that lead causes neurotoxic effects in children at blood-lead levels of 10 to 15  $\mu\text{g}/\text{dL}$ . By 1990, epidemiological data were enhanced by two factors: 1) Needleman and Gatsonis' meta-analysis of 12 previous studies; and 2) the first published findings from longitudinal studies which began to show the causal link between lead and neurotoxic effects.

Needleman and Gatsonis<sup>45</sup> identified, among the 27 studies noted above, 12 studies which were appropriately similar and/or otherwise adequately conducted to include in a meta-analysis. They separated the studies into those that were based on tooth lead and those based on blood lead. On completion, the combined P value for the blood lead studies was less than 0.0001 (i.e., a less than 1 chance in 10,000 that the effect occurred randomly).

The application of meta-analysis to the findings on lead neurotoxicity was a profound advance. Previously, reviewers of the many studies could only make educated judgements based on patterns of results and conclude that the results were collectively generally tending in the direction of showing an effect but still equivocal. Meta-analysis quantitatively summarizes or pools the results of many studies to form, in essence, one large study with greater statistical power (ability to detect any true associations between lead and IQ) than any of the individual studies on their own.<sup>46</sup>

Two more meta-analyses were conducted on cross-sectional studies. The first<sup>47</sup> did not find a strong association between neurobehavioural effects and environmental lead exposure although within the range of tests conducted there were stronger and more consistent associations for certain tests including, for example, disruption of visual-motor integration. This meta-analysis did not confirm or refute neurobehavioural effects of lead-exposed children although the results did tend in the direction of an effect.

The final meta-analysis, conducted by the International Programme on Chemical Safety of the World Health Organization<sup>48</sup> confirmed the findings of Needleman and Gatsonis. It included several of the studies used in the second meta-analysis described above, as well as three additional studies. The meta-

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<sup>44</sup> Needleman, H.L., C. Gunnoe, A. Leviton, *et.al.*, Deficits in psychological and classroom performance of children with elevated dentine lead levels. *N. Engl. J. Med.* 300 (1979), pp. 689-695.

<sup>45</sup> Needleman, H.L. and C. Gatsonis. Low level lead exposure and the IQ of children. *JAMA.* 263 (1990), pp. 673-678.

<sup>46</sup> Fletcher, R.H., *et.al.* *Clinical Epidemiology: the essentials.* (Williams and Wilkins, Baltimore, 1988)

<sup>47</sup> Winneke, G. *et.al.* Results from the European Multicentre Study on Lead Neurotoxicity in Children: Implications for a Risk Assessment. *Neurotoxicology and Teratology.* 12 (1990), pp. 553-559.

<sup>48</sup> World Health Organization's International Programme on Chemical Safety. *Inorganic Lead.* Environmental Health Criteria 165. (Geneva, 1995)

analysis found a statistically significant correlation between IQ deficits and blood lead, specifically, that an increase in blood lead levels from 10  $\mu\text{g}/\text{dL}$  to 20  $\mu\text{g}/\text{dL}$  correlates to a drop of approximately 2 IQ points.

Despite the equivocal results of the second meta-analysis, all three of these meta-analyses strengthened the conclusion that blood lead is associated with poor performance in neuropsychological tests. All studies were cross-sectional so the direction of causation remained unproven. For example, the studies could not say whether a child with lower intelligence had a higher blood-lead level because he/she was less intelligent or because the higher blood-lead level caused the lower intelligence. However, as Needleman and Gatsonis point out, two key criteria for demonstrating causality *were* evident in the studies, including: 1) the supposition that lead is the causal factor is supported by animal studies showing the biochemical mechanisms at work that can explain the adverse effects; and 2) the adverse effects of lead are consistently found in many studies under many different circumstances. Further, in all of the many studies undertaken, no evidence points to another confounding variable or set of variables.<sup>49</sup>

### 8.3.4.2 Prospective and Longitudinal Studies

Throughout the 1980s, alongside (and following on the earlier) cross-sectional studies, five separate studies were begun that would track children from *in utero* (via maternal and cord blood samples) through successive developmental stages and record both blood-lead levels and results of neurobehavioural tests. The five studies were conducted in Cleveland<sup>50</sup> and Cincinnati<sup>51</sup> in Ohio, Boston,<sup>52</sup> in Massachusetts, and Port Pirie<sup>53</sup> and Sydney<sup>54</sup> in Australia.

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<sup>49</sup> Needleman and Gatsonis (1990), *op. cit.*

<sup>50</sup> Ernhart, C.B. A critical review of low-level prenatal lead exposure in the human: 2. Effects on the developing child. *Reproductive Toxicology*. 6 (1992); and Greene, T. and C.B. Ernhart. Dentine Lead and Intelligence Prior to School Entry: A Statistical Sensitivity Analysis, *Journal of Clinical Epidemiology*. 46 (1993), pp. 323-339.

<sup>51</sup> Deitrich, K.N. *et al.*, Lead exposure and neurobehavioral development in late infancy, *Environmental Health Perspectives*. 89 (1990); Deitrich, K.N. *et al.*, Lead exposure and the cognitive development of urban pre-school children: the Cincinnati lead study cohort at age 4 years, *Neurotoxicology and Teratology*. 13 (1991), pp. 203-211; Deitrich, K.N. *et al.*, Lead exposure and the central auditory processing abilities and cognitive development of urban preschool children: the Cincinnati lead study cohort at age 5 years, *Neurotoxicology and Teratology*. 14(1) (1992), pp.51,56; Deitrich, K.N. *et al.*, Lead exposure and the motor developmental status of urban six-year-old children in the Cincinnati prospective study. *Pediatrics*. 91 (1993), pp. 301-307; Deitrich, K.N. *et al.*, The Developmental Consequences of Low to Moderate Prenatal and Postnatal Lead-Exposure - Intellectual Attainment in the Cincinnati Lead Study Cohort Following School Entry, *Neurotoxicology and Teratology*. 15(1) (1993), pp. 37-44.

<sup>52</sup> Bellinger, D., *et al.*, Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development, *New Engl. J. Med.* 316(17) (1987), pp. 1037-1043; Bellinger, D., *et al.*, Antecedents and correlates of improved cognitive performance in children exposed *in utero* to low levels of lead, *Environmental Health Perspectives*. 89 (1990), pp. 5-11; Bellinger, D., *et al.*, Low-Level Lead Exposure and Children's Cognitive Function in the Preschool Years, *Pediatrics*. 87 (1991), pp. 219-227; Bellinger, D., *et al.*, 1993. Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study, *Pediatrics*. 90 (1993), pp. 855-861.

<sup>53</sup> McMichael, A.J., *et al.* Port Pirie cohort study: environmental exposure to lead and children's abilities at the age of four years', *New Engl. J. Med.* 319 (1988), pp. 468-475; Baghurst, P.A., *et al.*, Environmental exposure to lead and children's intelligence at the age of seven years', *New Engl. J. Med.* 327 (1992), pp. 1279-1284; Wigg, N.R., *et al.*, Port Pirie cohort study: childhood blood lead and neuropsychological development at age two years, *Journal of Epidemiology and Community Health*. 42 (1988), pp. 213-9; Tong, S., *et al.*,



Children in these five studies were given a range of age-appropriate neuropsychological tests during infancy and at 2, 4, 7, and in one case 12 years of age. Most, but not all of these test results found significant associations between blood lead and poorer performance in tests of cognitive ability, motor skills, and other indicators of neurological development. The Boston study confirmed the suspicion that children are especially vulnerable to the effects of lead at 24 months of age. The Port Pirie study found adverse effects throughout, at ages 2, 4, 7 and 12. Despite the fact that blood lead levels had dropped significantly in this older age group, the study showed that blood lead levels in early childhood correlated to adverse effects later in life, (independent of the blood lead level found at the later stage), contradicting a claim often made by the lead industry that adverse health effects in childhood are unlikely to be permanent. The Port Pirie study also indicated that no clear threshold existed below which adverse effects did not occur.

Taken together, these five studies provide powerful evidence of the causal relationship between lead and adverse neurological effects in children. A meta-analysis conducted on four of these studies by the World Health Organization<sup>55</sup> reached the same conclusion as the meta-analysis noted above on the cross-sectional studies, namely that, an increase in blood-lead levels from 10  $\mu\text{g}/\text{dL}$  to 20  $\mu\text{g}/\text{dL}$  results in an IQ deficit of approximately 2 points.

### 8.3.5 *Lead and Behaviour*

The foregoing review provides a sense of the long passage of time during which the large body of evidence has accumulated to demonstrate the causal connection between low level lead exposure and cognitive effects, generally measured as IQ deficits. Alongside this research and generally more recently in this long saga, investigators have increasingly noted and tried to isolate effects of lead on behaviour. In general, effects have been seen or suspected in the areas of attention, activity level, sleep patterns, aggression, depression, low self-esteem, criminality and other negative behaviours. Such effects are the least well studied generally due to a lack of reliable, validated tools or measurement techniques that can provide reproducible measures of these effects.<sup>56</sup> One such tool is the Child Behaviour Check List (CBCL). In an investigation of young children, those with higher lead exposure (those with two consecutive blood lead levels of 15  $\mu\text{g}/\text{dL}$  or higher) had significantly higher overall CBCL scores including such variables as higher rates of sleep problems, somatic problems, hyperactivity and aggression. The authors conclude that this study provides further evidence of the detrimental effect of lead on child behaviour at levels typical of present-day exposure<sup>57</sup> (typical of inner-city areas of the United States and likely some inner city areas of Canada).

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Lifetime exposure to environmental lead and children's intelligence at 11-13 years: the Port Pirie cohort study, *British Medical Journal*. 313 (1996), pp. 1569-1575.

<sup>54</sup> Cooney, G.H., *et.al.*, Low-level exposure to lead: the Sydney lead study, *Developmental Medicine and Child Neurology*. 31(1989), pp .643-644; World Health Organization's International Programme on Chemical Safety. *Inorganic Lead*, Environmental Health Criteria 165. (Geneva, 1995), p. 181, Table 21.

<sup>55</sup> World Health Organization's International Programme on Chemical Safety. *Inorganic Lead*, Environmental Health Criteria 165. (Geneva, 1995)

<sup>56</sup> Shannon, M.W., Director, Pediatric Environmental Health Center, Children's Hospital, Boston, Harvard Medical School in a presentation at Pediatric Environmental Health: putting it into practice. June 4-7, 1999. San Francisco.

<sup>57</sup> Sciarillo, W.G., G. Alexander, and K.P. Farrell, Lead Exposure and Child Behavior, *Am. J. Public Health*. 82(10) (October 1992), pp. 1356-60.

It remains unclear whether behavioural effects are a primary result of lead toxicity or whether they are secondary to cognitive effects. In a recent retrospective cohort study of young boys, Needleman has found lead exposure to be associated with increased risk for antisocial and delinquent behaviour with the effects following a developmental course.<sup>58</sup> Finally, throughout the entire range of literature on the physiological, cognitive and behavioural effects of lead, effects seem to be more serious in boys than in girls.

### 8.3.6 Summary

To summarize, the adverse neurological and neurobehavioural effects of low level lead exposure include a variety of measured and observed effects including:

- deficits in IQ or deficits in comparable/age appropriate tests<sup>59</sup> of intellectual functioning;
- deficits in speech and language processing;
- deficits in perceptual-motor function and integration;
- deficits in reaction time;
- reduced attention span;
- non-adaptive classroom behaviour;
- deficits in reading, spelling and mathematics scores;
- poorer handwriting;
- significant increase in the risk for learning disabilities, as measured by the need for remedial education in reading, speech and math;
- sevenfold increased risk of failure to complete high school;
- sixfold increased risk for reading disability;
- poorer vocabulary and grammatical reasoning scores; and
- poorer hand-eye coordination.<sup>60</sup>

However, despite all that is currently understood about the effects of lead, we remain limited in a clear diagnosis of lead toxicity at low exposure levels. Effects are variable and do not have a consistent behavioural signature. As Bellinger notes:

*There currently is no particular constellation of neuropsychological findings that can be used in the diagnostic sense. Some studies indicate that verbal abilities are most impacted by lead, while others indicate that the visual and spatial abilities are most affected. The most consistent finding is the reduction in the ability to sustain attention.*<sup>61</sup>

On a population basis, Needleman and Bellinger<sup>62</sup> postulate that there is probably an overall downward

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<sup>58</sup> Needleman, H.L., *et al.*, Bone Lead Levels and Delinquent Behavior, *JAMA*. 275(5) (1996), pp. 363-369.

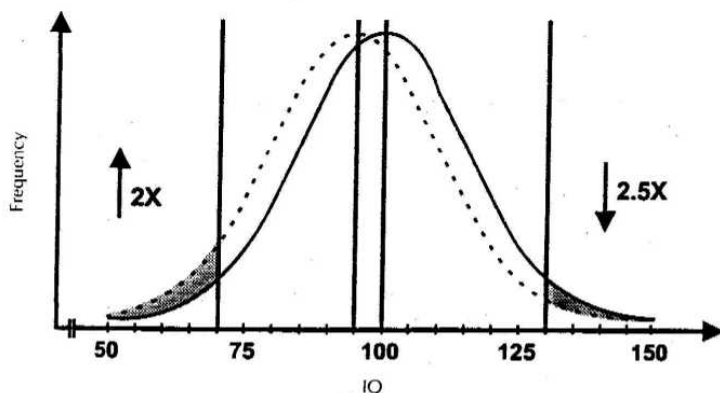
<sup>59</sup> For example, in infants the Bayley Scales of Infant Development, also known as the Bayley Mental Development Index (MDI) are used; at 24 months investigators apply the McCarthy Scales of Children's Abilities and measure the General Cognitive Index (GCI). For older children tests include the Wechsler Revised Intelligence Scale for Children (WISC-R), the Kaufman Test of Educational Achievement (K-TEA) and the Kaufman Assessment Battery for Children (K-ABC).

<sup>60</sup> Summarized from Needleman, H.L. and D. Bellinger. The Health Effects of Low Level Exposure to Lead, *Annu. Rev. Publ. Health*. 12 (1991), pp. 111-140.

<sup>61</sup> Bellinger, D. Developmental Effects of Lead. Childhood Lead Poisoning: What's New, What's Sadly Not. In: *Proceedings of the 1998 Children at Risk Conference Environmental Health Issues in the Great Lakes Region*. (Chicago, July 8-9, 1998)

<sup>62</sup> Needleman, H. and D. Bellinger. The Health Effects of Low Level Exposure to Lead. *Annu. Rev. Publ.*

shift in intelligence. On the basis of blood-lead levels prevalent in the U.S. in 1990, (slightly higher but comparable to Canadian levels), they conclude that lead exposure may prevent about 5% of the population from achieving truly superior function and at the lower end of the scale, greater intellectual damage may be occurring in many more children than would otherwise occur without exposure to lead. A slightly different way of looking at the same data is provided in Figure 8.4 representing the lead-caused downward drop in IQ across the population. The overall downward shift would not affect most individuals but would result in a 2.5-fold decrease in very gifted (IQ above 130) people and a 2-fold increase in people with an IQ below 70, the level considered to be in the range of “mental retardation.”



**Figure 8.4. Normal Distribution of IQ.**

Normal Distribution of IQ with a mean of 100 (solid curve) and decreased by five points (dashed curve). The filled area on the right tail of the distribution represents a decrease in the number of individuals with IQ's greater than 130 by a factor of 2.5, while the filled area on the left tail represents a doubling of the number of persons with IQ's less than 70 (defined clinically as mental retardation).

(Source: Rice, D.C. Issues in Development Neurotoxicology: Interpretation and Implications of the Data, *CJPH*. 89 (Suppl 1) (1998) 531-536, as adapted from Weiss, B. Risk Assessment: the Insidious Nature of Neurotoxicity and the Aging Brain. *Neurotoxicol.* (1990) 11-305.)

## 8.4 THE REGULATORY RESPONSE

Enormous vested interests have been involved whenever concerns have been expressed about the need to control environmental lead contamination either around point sources such as primary and secondary smelters or from the mobile source of lead in gasoline. Regulatory action has been led by the United States and some European countries with Canada consistently lagging behind. The United Kingdom probably has been the worst example,<sup>63</sup> in developed countries, of recalcitrance in the face of overwhelming evidence of a problem and the need for stronger regulatory and public health responses. Not to be outdone however, the Canadian government regulatory response to lead in consumer products has been very slow, ambiguous and is now increasingly and even dangerously out of date (see Section 8.4.6 below). Regulation of lead in developing countries is appallingly lax and the consequences are sobering.

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*Health*. 12 (1991), pp. 111-140.

<sup>63</sup> See detailed critique in Millstone, E. (1997), *Lead and Public Health*, *op.cit.*

*In developing countries, all urban children under 2 years of age and more than 80 percent of those between the ages of 3 and 5 are suspected to have blood lead levels exceeding standards set by the World Health Organization. About 15 to 18 million children in these countries may suffer permanent brain damage due to lead poisoning.*<sup>64</sup>

### 8.4.1 Lead in Gasoline

From the beginning in the 1920s the use of lead in gasoline has been a controversial public health issue. Little was known about the public health consequences of widespread dispersal of a substance known to be highly toxic. Concerns raised by labour and public health advocates and experts were effectively dismissed by the lack of any definitive proof of harm that might arise from widespread environmental dispersal and levels of exposure that would be far lower than anything seen in occupational exposure settings.

The story of how the environmental consequences of adding lead to gasoline were initially evaluated and subsequently studied is worth reviewing in some detail. Rosner and Markowitz<sup>65</sup> provide an excellent review of the public health controversy in the 1920s and Nriagu<sup>66</sup> continues the story to the 1960s to show how, with the results of influential new and independent studies, the lead industry's self-serving hold on the scientific investigation of the issue was pivotally moved to the arena of more independent science.<sup>67</sup>

The proposal to add lead to gasoline set off a storm of controversy involving issues that will sound similar to any debate around the use of a known toxin at levels below which science can show or has shown environmental or human health effects.

In response to demands for research into possible health risks, an agreement was made in 1923 between the U.S. government and General Motors (GM) Research Corporation whereby GM would pay for a study done by the U.S. Bureau of Mines. GM obtained clauses in the agreement that would bar press and progress reports during the study. The word "lead" was omitted from correspondence and the trade name "ethyl" used instead. By the end of the study Ethyl Corporation, formed by GM and Dupont to produce "ethyl" gasoline (short for tetra ethyl lead or TEL, the gasoline additive), had negotiated exclusive rights for comment, criticism, and approval of any of the research generated by the study before it was released. Not surprisingly, the study found no problems with the use of lead in gasoline.

In 1924, at the same time that the U.S. Bureau of Mines began to assure the American public of the safety of leaded gasoline, a disastrous series of poisonings occurred in the Standard Oil Company's experimental laboratories. Over the space of four days, five out of 49 TEL workers died, and 35 were severely poisoned. As a result, scientists, public health experts, and labour activists across the U.S. attacked the industry-funded study that had absolved lead in gasoline, claiming that it was inadequate and biased. They called for regulatory action banning the use of lead in gasoline.

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<sup>64</sup> World Bank Group (1996) Press Release No. 96/68S at [www.worldbank.org/html/extr/gaspr.htm](http://www.worldbank.org/html/extr/gaspr.htm)

<sup>65</sup> Rosner, D. and G. Markowitz. A Gift of God?: The Public Health Controversy over Leaded Gasoline during the 1920s, *American Journal of Public Health*. 75(4) (1985), pp. 344-352. See also: Wallace, B. and K. Cooper. *The Citizen's Guide to Lead: Uncovering a Hidden Health Hazard*. (NC Press, Toronto, 1986), Chapter 10.

<sup>66</sup> Nriagu, J. Review: Clair Patterson and Robert Kehoe's Paradigm of "Show Me the Data" on Environmental Lead Poisoning, *Environmental Research*, Section A. 73 (1998), pp. 71-78.

<sup>67</sup> Note that this and subsequent references in this section to the "lead industry" refers to the full range of industries commercially involved in the use of lead and lead-bearing products.

The Surgeon General of the U.S. Public Health Service then convened a conference in May of 1925. However, at the conference, in the face of insufficient information about the possible long-term implications of the use of leaded gasoline, Robert Kehoe, on behalf of Ethyl Corporation, established a highly effective strategy that has been used by industry to this day and which the lead industry and Robert Kehoe in particular used very effectively for the next forty years. First, he separated the occupational lead hazard from the public health concern by insisting that worker training and other safe industrial practices could fully address the workplace lead poisoning. Second, he also insisted that:

*if it is shown as a result of this discussion--that an actual hazard exists from exhaust gases from motors, that an actual danger to the public is had as a result of the treatment of gasoline with lead, it will be discontinued from that moment.*<sup>68</sup>

Kehoe's insistence that if actual harm could be shown, leaded gasoline would be discontinued sounded scientifically valid; industry appeared entirely reasonable, even magnanimous.

More long term studies were recommended to resolve the issues raised at the May 1925 conference. Instead, a Blue Ribbon Committee was set up and instructed to conduct another study and provide answers within seven months. Under such time pressures the committee could only recommend ongoing study to detect long-term effects:

*...the committee feels that the investigation begun under their direction must not be allowed to lapse...It should be possible to follow closely the outcome of a more extended use of this fuel and to determine whether or not it may constitute a menace to the health of the general public after prolonged use or other conditions not now foreseen... The vast increase in the number of automobiles throughout the country makes the study of all such questions a matter of real importance from the standpoint of public health and the committee urges strongly that a suitable appropriation be requested from Congress for the continuance of these investigations by the Surgeon General of the Public Health Service.*<sup>69</sup>

These recommendations went unheeded and further investigations were conducted by the industry itself. In fact, the Public Health Service took Robert Kehoe's advice that since there was no apparent evidence of any immediate public health threat and since such studies would be very expensive, further research should be conducted and paid for by the industry itself. Kehoe's approach has been called "show me the data"<sup>70</sup> and it was highly effective. To maintain its unregulated position and hold off any future attempts at regulation, the industry made sure that it stayed in control of research and data collection. As Nriagu points out, over time, the effect of "show me the data" was to enshrine a system of industry self-regulation and to enable two possible, self-serving outcomes. First, if no health risk became apparent, the insistence on proof would be verified as the correct course of action. Alternatively, if health hazards did become apparent, they would be very difficult to prove. In a world of imperfect information, uncertainty could always be found, especially if the industry controlled most of the information.

"Show me the data" is a strategy that has been used effectively by the tobacco industry as well as industries with commercial interests in mercury, asbestos, pesticides, etc. It is inevitably combined with vigorous arguments about the easily quantified and often very large economic benefits of any particular industrial activity weighed against the far more difficult to assess potential health costs that could be indirectly felt by society at large and which are often separated in time and space from the economic activity and difficult to prove. "Show me the data" places an onerous burden of proof on the public according to a scale defined by industry and within a scientific environment in which the industry can

<sup>68</sup> As quoted in Nriagu (1998), *op.cit.*

<sup>69</sup> As quoted in Rosner and Markowitz (1985), *op.cit.* p.350.

<sup>70</sup> By Nriagu (1998), *op.cit.*

constantly raise issues of uncertainty and lack of proof. The reverse of the argument also serves the industry. When a lack of data exists and a report or conference, like the Surgeon General's conference of 1925, cannot show reasons to stop the use of lead additives, a false sense of safety is implied. Under circumstances of such misplaced trust, the industry can respond to questions about safety with the response that they do not know nor does anyone know for sure whether the product is safe. Nriagu summarizes:

*The Kehoe Paradigm thus was a bifocal proposition with either angle favoring an industry that opposes regulation and is committed to defending its position using the weight of scientific and medical evidence.*<sup>71</sup>

And so it went, virtually unchallenged for forty years until Clair Patterson exposed the lead industry's lack of scientific objectivity and, by "showing them the data", undermined most of the industry's theoretical framework, a framework for which Robert Kehoe was largely responsible. At Senate Hearings in 1966, Patterson stated:

*It is clear, from the history of development of the lead pollution problem in the United States that responsible and regulatory persons and organizations concerned in this matter have failed to distinguish between scientific activity and the utilization of observations for a material purpose. [such utilization] is not science... it is the defense and promotion of industrial activity. This utilization is not done objectively. It is done subjectively ... It is not just a mistake for public health agencies to cooperate and collaborate with industries investigating and deciding whether public health is endangered--it is a direct abrogation and violation of the duties and responsibilities of those public health organizations. In the past, these bodies have acted as though their own activities and those of lead industries in health matters were science, and they could be considered objectively in that sense.*<sup>72</sup>

Patterson's data were from his geochemical studies that revealed body burdens of lead in Americans to be at least 100 times above natural background values. By "showing the data" Patterson undermined the foundation of Kehoe's entire theoretical construct concerning the threshold of lead exposure and lead toxicity. Kehoe had determined from his occupational exposure studies that "safe" workplace levels for lead in air were 100  $\mu\text{g}/\text{m}^3$ . He then concluded that, since ambient air lead concentrations were about 1  $\mu\text{g}/\text{m}^3$ , exposure to the public was 100 times lower than his scientifically derived "safe" level. However, when Patterson compared ambient levels (in the U.S.) to his biochemical data (of lead levels in remote locations indicative of actual background atmospheric lead levels), he showed that human activity was responsible for a 2000-fold increase in ambient levels. Patterson concluded that the average U.S. resident was being subjected to severe chronic lead insult.

Patterson also challenged Kehoe's notion of the "toxic limit" or point at which clinical lead poisoning could be observed. Kehoe had, within the "show me the data" approach, insisted that lead poisoning be rigidly defined as being manifest only with the appearance of clinical symptoms; hence his insistence that lead toxicity in children manifests at blood lead levels of 80  $\mu\text{g}/\text{dL}$ . This insistence was reinforced by the refusal to accept uncertain results; a convenient approach since the more subtle or sub-clinical effects of

<sup>71</sup> Nriagu (1998). *op.cit.*, p. 75. Nriagu refers to the notion of "show me the data" as the Kehoe Paradigm following the original and more detailed review of Kehoe's influence in Loeb, A.P. Birth of the Kehoe Rule: Implications of the Surgeon General's Review of Tetraethyl Lead, (1997), pp. 1925-26. Unpublished manuscript; presented at Annual Meeting, Am. Soc. Environmental History, Baltimore, MD. (March 7, 1997); and Loeb, A.P. The hazards of gasoline lead additives, in: *The Car, Its Fuel, Air Pollution and Regulation*. (1994) In press.

<sup>72</sup> Muskie Hearings. Hearings before a sub-committee on air and water pollution of the committee on public works of the United States Senate, 59th Congress, (June 7-15, 1966), pp. 113-343. U.S. Government Printing Office, Washington, DC as cited in Nriagu (1998), *op.cit.*, p.76.

lead are difficult to recognize and/or can be confused with other more mundane conditions such as colic, dizziness, headaches, etc. Over time this difficulty intensified as later research revealed increasingly subtle health effects at lower and lower exposure levels that do not manifest as clinical symptoms at all. But the debate began with Patterson's speculation that classical lead poisoning was likely on the extreme end of a continuum of health effects that had yet to be identified or clarified.

Since Patterson's influential challenge to the lead industry's hold on the science of environmental lead contamination and the health effects of lead, literally thousands of scientific reports on the chemistry, environmental fate and health effects of lead have been published. Throughout, the lead industry has effectively persisted with the Kehoe approach of insisting on absolute proof of harm, funding and/or advancing dubious research, and emphasizing the many issues of uncertainty in this highly complex arena of scientific investigation as a means of undermining regulatory action on the industry.

The regulatory response to all of this scientific debate and controversy occurred first and most dramatically in the United States. The situations in both the United States and Canada are worthy of review since actions in the United States both influenced and at key junctures were inappropriately ignored by regulators and investigators in Canada.

#### 8.4.1.1 Regulation of Lead in Gasoline in the United States

With the introduction in the early 1970s of catalytic converters, (to control other air pollutants and which are intolerant of lead), unleaded gasoline was made available in the United States. By the late 1970s the U.S. was regulating the lead content of the total gasoline "pool." As a result of increasing controversy and concern about lead contamination and the known contribution from gasoline, the Natural Resources Defence Council in Washington sued the USEPA in 1976 and forced it, after a protracted three year battle, to reduce the limit of lead in gasoline to 0.29 g/L; this standard was to take effect in 1982.

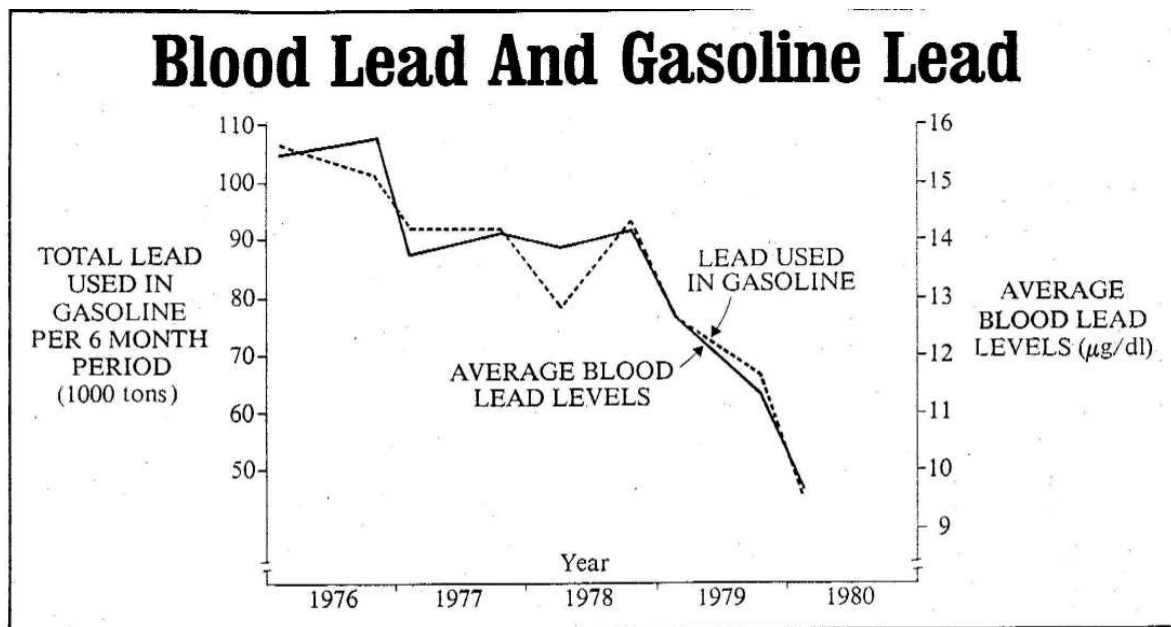
Throughout the 1970s and early 1980s as the overall fleet gradually changed and cars requiring lead-free gasoline increasingly dominated the roads, the use of leaded gasoline declined steadily. While the industry was likely aware of declining overall sales, further evidence of this decline in the use of leaded gasoline came from an unexpected source: records of blood lead levels.

The second National Health and Nutrition Examination Survey or NHANES-II revealed a steady drop in American's blood lead levels from 1976 to 1980.<sup>73</sup> On first review of these data, scientists at the Centers for Disease Control (CDC) thought they had a laboratory contamination problem early on in the study.<sup>74</sup> Upon systematic re-examination of the blood-lead samples and careful review of associated variables such as race, sex, age, season, region of the country, etc., CDC investigators eventually looked at lead exposure sources and zeroed in on the decreased use of leaded gasoline over the time period of blood lead sampling. The result is a now famous graph (Figure 8.5) showing a near lock-step association (with a remarkable correlation coefficient of 0.95) between declining blood-lead levels and declining use of leaded gasoline. Blood lead levels even matched seasonal variations; increasing in summer when more gasoline is used. Joel Schwartz, an EPA scientist also reviewed the data using slightly different

<sup>73</sup> Centers for Disease Control. Blood-Lead Levels in the U.S. Population, *Morbidity and Mortality Weekly Report* 31, No. 10 (March 19, 1982), pp. 132-134; and eventually reported in: U.S. Department of Health and Human Services/Public Health Service (1984). National Health and Nutrition Examination Survey Series II, No. 233, Blood Lead Levels for Persons Ages 6 Months - 74 Years: United States, (1976-1980). DHHS Publication No. (PHS), pp. 84-1683.

<sup>74</sup> See discussion in Savan, B. *Science Under Siege*. (CBC Enterprises, Toronto, 1988), Chapter 3, pp. 60-68.

calculations and reached similarly categorical conclusions about the association between blood lead and gasoline lead.<sup>75</sup>



**Figure 8.5. Blood Lead and Gasoline Lead.**

Trends in the average blood lead levels of people living in the United States (February 1976 – February 1980) and lead used in gasoline production. Note: One ton equals about 0.98 metric tonnes.

(Source: Adapted from U.S. Environmental Protection Agency, 1985, *op.cit.* and reproduced from Wallace and Cooper, 1986, *op.cit.*)

Scientists from Ethyl Corporation and Dupont, both large producers at the time of gasoline lead additives, then weighed in and drew quite different conclusions from the NHANES-II data finding no association between the two variables.<sup>76</sup> To address the conflicting results the EPA convened the NHANES-II Time Trend Analysis Review Group, expert statisticians who ultimately supported the EPA and CDC findings and panned the industry study.<sup>77</sup>

Meanwhile, under requirements mandated by the U.S. *Clean Air Act*, the US EPA was undertaking the research and analysis necessary to revise the Air Quality Criteria for lead. The result, available in draft form in 1984, and finalized in 1986, was an enormous four volume set of documents fully describing the properties, environmental sources, pathways, metabolism, and health effects of lead, in particular documenting the emerging, but still very controversial evidence of effects at very low levels of exposure.<sup>78</sup> It showed that leaded gasoline was responsible for 80% of environmental lead contamination

<sup>75</sup> Ibid.

<sup>76</sup> Lynam, D.R., G.A. Hughmark, B.F. Fort Jr. and C.A. Hall. Blood Lead Concentrations and Gasoline Lead Usage (1983), and Pierrard, J.M., C.G. Pfeifer and R.D. Snee, Assessment of Blood Lead Levels in the USA from NHANES data; both papers presented at the *International Conference on Heavy Metals in the Environment*. (Heidelberg, Germany, 1( September, 1993)

<sup>77</sup> J. Rosenblatt, H.Smith, R.Royall, R.Little, and J.R.Landis. *Report of the NHANES-II Time Trend Analysis Review Group*, USEPA, (North Carolina, June 15, 1983), pp.12 and 14.

<sup>78</sup> U.S. Environmental Protection Agency. *Air Quality Criteria for Lead, Volumes I - IV*. Environmental Criteria



and at least 50%, and probably more, of the lead found in human blood. Also at EPA, Joel Schwartz, noted above, got back to his primary task which was to evaluate the environmental costs of *increasing* lead in gasoline which, true to the spirit of those Reagan years, was being considered as part of deregulating the industry. However, instead of providing a rationale for deregulation, Schwartz's cost-benefit analysis predicted significant cost savings for children's health care and remedial education if the U.S. undertook a dramatic 90% reduction of the amount of lead allowed in gasoline.<sup>79</sup> The study also found that consumers would save millions of dollars in reduced car maintenance costs and improved fuel economy. Benefits outweighed costs by a factor of 4 to 1. In addition, the study predicted that if the preliminary evidence that lead has a detrimental effect on blood pressure (in adults) were shown to be reliable, the anticipated health care savings would increase further by over \$38 billion between 1985 and 1992.<sup>80</sup>

The combined effect of the NHANES Review Group report in 1983, the draft Air Quality Criteria documents and draft results from the Cost-Benefit analysis, (also available in 1984),<sup>81</sup> and a pre-election year resulted in the announcement in July of 1984 of a new regulation to reduce the level of lead in gasoline by 91% (from 0.29 g/L to 0.026 g/L) to take effect in January of 1986.

The NHANES-II data showed that average blood-lead levels in American children were in the range now considered, and then suspected, to cause health effects. Rural children (age six months to two years) had an average blood lead level of 13.9  $\mu\text{g/dL}$ , their urban counterparts averaged 18  $\mu\text{g/dL}$ , and poor, urban black children were the worst affected, with nearly 20% at levels at or above 30  $\mu\text{g/dL}$ .<sup>82</sup> However, NHANES-II had also shown that blood-lead levels were dropping. The data were current to 1980 and despite the thoroughness and value of the Air Quality Criteria for Lead documents and the Cost-Benefit analysis of gasoline lead phase-down, neither of these studies provided comprehensive data on the current extent (i.e., by 1986) of lead insult on American children. In response, Congress commissioned another landmark study by the Agency for Toxic Substances and Disease Registry (ATSDR) which was published in 1988. Although the ATSDR Report followed on the decision to dramatically drop the level of lead in U.S. gasoline, it is worth review here since it had a significant effect on regulatory action on lead in gasoline in Canada.

The ATSDR Report,<sup>83</sup> *The Nature and Extent of Lead Poisoning in the United States*, made two crucial contributions to the lead debate. First, like the Air Quality Criteria documents, it provided yet another

and Assessment Office. (Research Triangle Park, North Carolina, 1986) EPA-600/8-83/028dF.

<sup>79</sup> The 90% reduction arose from research conducted for the cost-benefit analysis which confirmed that only a very small amount of lead was necessary for the few vehicles that still required lead as a valve lubricant. The historical use of lead additives was to boost octane in the gasoline. The 90% reduction would eliminate the octane boosting capability so refineries would have to employ alternative refining techniques; this (and the loss of lead additive sales) would be the major cost to industry of a lead phase-down.

<sup>80</sup> Schwartz, J., H.Pitcher, R.Levin, B.Ostro, and A.L.Nichols. *Costs and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis*. EPA-230-05-85-006. Office of Policy Analysis, USEPA,(Washington, DC. February, 1985).

<sup>81</sup> Both the Air Quality Criteria documents and the Cost-Benefit Analysis were the subject of extensive and thorough peer review prior to their final publication in 1986 and 1985, respectively.

<sup>82</sup> Mahaffey, K.R., *et.al.*, National estimates of blood levels: United States, 1976-1980: associated with selected demographic and socio-economic factors. *New Engl. J. Med.* 307 (1982), pp. 573-579. Note that the CDC "intervention level" was still 30  $\mu\text{g/dL}$  at the time of these studies.

<sup>83</sup> Agency for Toxic Substances and Diseases Registry. *The Nature and Extent of Lead Poisoning in Children in the United States: A report to Congress*. Department of Health and Human Services. (Atlanta, 1988)

comprehensive and fully up to date review of the worldwide investigation into the low level effects of lead. It confirmed and consolidated the emerging international scientific consensus that blood lead levels between 10 and 15  $\mu\text{g}/\text{dL}$  in children could cause adverse health effects. Second, this report concluded that about 2.4 million children (17% of those living in the Standard Metropolitan Statistical Areas - SMSAs, i.e., urban American children) had blood-lead levels above 15  $\mu\text{g}/\text{dL}$  in 1984. Huge numbers of children from all socio-economic backgrounds were affected but the report also confirmed that poor, urban, black children, especially males were disproportionately affected; they had higher average blood lead levels and many more children were affected than were white children. The report also found that about 4 million women of child-bearing age and over 400,000 fetuses likely had lead exposures that could result in blood-lead levels above 10  $\mu\text{g}/\text{dL}$ . The report also indirectly confirmed what the CDC suspected; that during the mid-1980s, average blood-lead levels in American children still hovered around 10  $\mu\text{g}/\text{dL}$ . The rest of this story continues with the decision in 1991 to lower the “intervention level” to 10  $\mu\text{g}/\text{dL}$  and to undertake blood-lead screening of every child in the country (see discussion of Blood-Lead Testing and Follow-Up in Section 8.4.8 below).

#### 8.4.1.2 Regulation of Lead in Gasoline in Canada

In the 1970s in Canada, environmental and medical groups were also agitating for regulatory controls on leaded gasoline. The lead industry vigorously opposed regulation. The Canadian government response in 1976 was to set a limit of 0.77 g/L, a level that was higher than most other industrialized countries. By 1985 this standard remained unchanged and was the most lenient in the industrialized world.<sup>84</sup>

In the early 1980s, officials at Health Canada and Environment Canada were aware of the mounting medical evidence and also knew that the U.S. lead standard was scheduled to be substantially lower (0.29 g/L) than Canada’s standard by 1982. In December of 1983, Environment Canada announced its intention to reduce the Canadian standard to 0.15 g/L. The lead industry lobby went into overdrive. It claimed that the reduction would eliminate 1950 jobs, cost more than \$3 billion over 20 years and add 5.4 cents to the price of a litre of gasoline.<sup>85</sup>

The federal cabinet received an avalanche of correspondence and reports from the International Lead Zinc Research Organization (ILZRO) as did Provincial governments and opposition parties in five Provinces with lead industries. Many Provincial politicians agreed to lobby their federal counterparts on the lead industry’s behalf. Environment Canada did not have the manpower to respond and by the time a study was published (in February of 1984) it appeared that political orders had been sent to ensure it was not particularly useful. The *Socio-Economic Impact Analysis (SEIA) of Lead Phase-Down Control Options* was a minimalist piece of work by comparison to the rigour and detail of the studies occurring in the U.S. While the EPA was conducting its cost-benefit analysis of a 90% lead phase-down, Environment Canada stated in the SEIA that:

*It is not possible to assign a monetary value to human health and therefore a benefit-cost analysis could not be undertaken.*<sup>86</sup>

The SEIA was used to justify a move to the U.S. standard of 0.29 g/L but not until 1987, five years after the U.S. would go to that level. In stark contrast, within months of making this decision in Canada, the

<sup>84</sup> Wallace, B and K.Cooper. *The Citizen’s Guide to Lead: Uncovering a Hidden Health Hazard*. (NC Press, Toronto, 1986), p. 110.

<sup>85</sup> Ferguson, J., Lead industry lobby earns reputation for toughness, *Globe and Mail*. (Nov. 6, 1984), p.M-2.

<sup>86</sup> Environment Canada. *Socio-Economic Impact Analysis of Lead Phase-Down Control Options*, Environmental Protection Service. (February, 1984), p.xi.

U.S. announced its 90% reduction from that level (0.29 g/L to 0.026 g/L) to take effect in January of 1986. To address the increasing controversy over control of lead in gasoline, Environment Canada approached the Royal Society of Canada. After several months of negotiation and during the lead-up to a federal election, the choice was made to have the Royal Society of Canada conduct an inquiry to review all aspects of lead in the Canadian environment. An interim report concerning lead in gasoline was requested by September of 1985 and a full report by September of 1986. It is a well known Canadian political tradition to duck controversy by deferring to an “independent” review. Both the interim and final reports of the Commission were widely criticized as a whitewash.

The most scathing criticism came from four American scientists, all experts in the field of lead toxicology. Their collaborative rebuttal to the Commission’s interim and final reports is contained as an appendix to the Commission’s final report.<sup>87</sup> The following excerpt from the cover letter<sup>88</sup> to the rebuttal summarizes some of their concerns:

*We find that the report ignores many important scientific findings, fails to recognize the significance of the accumulated toxicological data, and adopts a policy of placing the burden of proof of adverse effects on Canadian citizens themselves. It shunts aside the most compelling priority, prevention. Instead of seeking to protect public health by recommending adequate safety margins, the report indicates that lead toxicity is not a significant problem until clinically overt disease becomes evident. To those of us who are familiar with the signs, symptoms and clinical course of lead toxicity in both children and adults, the Commission’s archaic position yields a policy that irreversible consequences are the basis upon which exposure standards are erected. The Commission, however, should be guided by the knowledge that, once children develop overt symptoms, irreversible damage to the central nervous system is probably already present.*

*Statements in the document, that adverse health effects of lead have not yet been established at blood lead values between 30-50  $\mu\text{g/dL}$  and below 30  $\mu\text{g/dL}$ , are applied to areas where substantial evidence of adverse health effects have been demonstrated in humans. This thesis, developed by the Commission, fails to reflect the current judgement of the scientific community; and, even where appropriate, the Commission suggests that evidence of a clearly recognized health hazard to children and the fetus should not be translated into policy, if the evidence is not beyond any “shadow of a doubt.”*

*The Commission’s report seems to us a disservice to Canadian citizens and a retreat from informed regulation. Those of us who participated in the review process perceive insufficient evidence that our efforts were weighed seriously, thoughtfully, and impartially. It appears that we were used to provide a simulacrum of objectivity.*

Similarly marked differences between the two countries are apparent in discussions of blood lead levels. An average blood-lead level in Ontario’s child population of 10  $\mu\text{g/dL}$  was described in 1984 by Ontario government agencies as “relatively low”<sup>89</sup> while in the U.S. such a level formed part of the basis for

<sup>87</sup> Needleman, H., J.F. Rosen, J. Schwartz and B. Weiss. Rebuttal to the Interim Report Issued by the Commission on Lead in the Environment: Lead in Gasoline - A Review of the Canadian Policy Issue, attached to a letter to F.Kenneth Hare. (January 3, 1986); and Rosen, J.F., et.al., Dissenting Opinion by U.S. Researchers, Appendix XI, Lead in the Canadian Environment: Science and Regulation, Final Report. The Royal Society of Canada Commission on Lead in the Environment. (September, 1986)

<sup>88</sup> Rosen, J.F., et.al., (1986), *op.cit.*, cover letter to dissenting opinion, contained in Appendix XI to Royal Society of Canada Commission on Lead in the Environment, Final Report.

<sup>89</sup> Duncan, C., R.A. Kusiak, J. O’Heany, L.F. Smith, L. Spielberg and J. Smith. *Blood Lead and Associated Risk Factors in Ontario Children, 1984*. Summary and Conclusions of Technical Working Group Report, Ontario Ministries of Health, Environment and Labour. (1984), p.20. This report identified urban Ontario

justifying the need to “reduce the amount of lead in gasoline as quickly as possible.”<sup>90</sup>

The Royal Society’s Lead Commission interim report on lead in gasoline endorsed the Canadian government’s go-slow position to reduce gasoline lead from 0.77 g/L to 0.29 g/L in 1987 concluding that: *The reductions in blood-lead levels achieved by these 1987 regulations will, in the opinion of the Commissioners, be sufficient to protect almost all segments of the Canadian population against the known harmful effects of lead exposure. There may be exceptions in certain urban hotspots, and among the industrially-exposed labour force.*<sup>91</sup> (emphasis added)

No evidence was provided by the Commission to justify this statement. Although the Commission was aware of the fact that 4.2% of children in Ontario had blood-lead levels above 20 µg/dL in 1985, (excluding children in “hotspot” areas near urban lead industries), the Commission did not provide any evidence to show that this percentage would decrease or be eliminated as a result of the 1987 regulation. The impression is also given that children in “hotspot” areas need not be included in decisions about lead in gasoline despite the fact that such children were consistently found to have higher average blood-lead levels than in the general population. Moreover, this argument could be made conveniently since the Commission completely ignored the U.S. cost-benefit analysis and omitted any substantive discussion of the well-established relationship between gasoline lead and blood-lead levels. Since it was well established that gasoline lead contributes at least 50% of the lead in every child’s blood, lead exposure in urban hotspot areas was additional to this already high baseline exposure from gasoline.

Following on the Royal Society’s interim report, Environment Minister Tom McMillan announced in March of 1986 that Canada would match the U.S. standard of 0.026 g/L by the end of 1992, *seven years* after the U.S. move to this standard. The following September, the Royal Society Lead Commission’s final report endorsed the phase-down timeline.

Public interest organizations, notably the Learning Disabilities Association of Canada (LDAC) have long been active on the lead issue. In 1982, in its first of many briefs to the federal government, the LDAC provided a review of the recent health evidence and urged swift regulatory action to reduce and ultimately eliminate children’s lead exposure.<sup>92</sup>

During 1985, the LDAC continued this pioneering advocacy work by joining with citizens’ groups from the two smelter neighbourhoods in Toronto (Niagara and South Riverdale - see Smelters and Soil, Section 8.4.2 below) and other national environmental and children’s health groups to form the Canadian Coalition for Lead-Free Gasoline. The Coalition was a vocal critic of the Royal Society’s two reports and the federal government’s decision to delay, for seven years, the move to the U.S. gasoline lead standard. These criticisms were echoed by many others including the Water Quality Board of the International Joint

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children to have an average blood-lead level of about 12 µg/dl and the average for all children to be about 10 µg/dl. By comparison, the average blood-lead level in U.S. urban children in 1983 was estimated to be the same level, about 10 µg/dl, by J.Pirkle, U.S. Centers for Disease Control, via personal communication with K. Cooper, Toronto. (March, 1986)

<sup>90</sup> Schwartz, J., H. Pitcher, R. Levin, B. Ostro, and A.L. Nichols. *Costs and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis*. EPA-230-05-85-006. Office of Policy Analysis, USEPA, (Washington, D.C., February, 1985), p. E-2.

<sup>91</sup> Royal Society of Canada’s Commission on Lead in the Environment. *Lead in Gasoline: A Review of the Canadian Policy Issue*, (1985), p.xiv.

<sup>92</sup> The Canadian Association for Children and Adults with Learning Disabilities (name now changed to Learning Disabilities Association of Canada). *The Effects of Low Level Lead Exposure on the Brain, Learning and Behaviour: A Brief to Support the Phase-Down of Lead in Motor Gasoline in Canada*. (November 23, 1982)

Commission<sup>93</sup> and the Canadian Medical Association,<sup>94</sup> both of which echoed the Coalition's call for a swifter phase-down of lead from gasoline.

In 1988, in a brief<sup>95</sup> to the federal ministers of Environment and Health and Welfare, the Coalition summarized the most recent health studies (including some of the earliest findings of the five longitudinal studies described in Section 8.3.4.2 above). The Coalition also showed that children's average blood-lead levels in the two countries were similar and estimated, conservatively, that half a million pre-school children in Canada had blood-lead levels of 10  $\mu\text{g}/\text{dL}$  or higher.<sup>96</sup> The Coalition also verified that the industry could phase-down to the U.S. level in 18 months, if necessary, preventing 5000 tonnes of lead from entering the Canadian environment.

The federal government persisted, well into 1988, in stating that a blood-lead level of 25  $\mu\text{g}/\text{dL}$  or higher was the concern level, citing the CDC's "intervention level" (set in 1985) and the Royal Society Lead Commission's report. However, as noted in section 8.4.1.1 above, despite setting the intervention level at 25  $\mu\text{g}/\text{dL}$  in 1985, the CDC was poised to lower the intervention level as soon as improvements were available in mass screening for blood-lead. Upon hearing how the Canadian government was referring to the CDC "intervention level", Vernon Houk, Director of the CDC's Center for Environmental Health told the Coalition: "That is a serious misuse of our data. The CDC is very concerned about children's blood-lead levels in the 10 to 15  $\mu\text{g}/\text{dL}$  range."<sup>97</sup>

The federal government continued to ignore the data on comparable blood-lead levels in the two countries and to refer to out of date information about health effects until the ATSDR report (discussed in Section 8.4.1.1 above) was published in August of 1988. The federal government briefly switched tactics stating that, even though the evidence now showed effects at lower blood lead levels, Canadian children likely had an average blood-lead level of about 4 to 6  $\mu\text{g}/\text{dL}$ , not the average level of 7 to 8  $\mu\text{g}/\text{dL}$  estimated by the Coalition. The Coalition disagreed with this lower estimate but was able to respond that if the average was as low as Health Canada thought, it would translate into 60,000 to 100,000 pre-school children with blood-lead levels above 10  $\mu\text{g}/\text{dL}$  (instead of the half a million children estimated by the Coalition).<sup>98</sup> Health Canada was then in the position of defending its estimated average blood-lead level of 4 to 6  $\mu\text{g}/\text{dL}$  (thereby placing *only* 60,000 to 100,000 children above 10  $\mu\text{g}/\text{dL}$ ) as being somehow more acceptable.

Throughout the Coalition's campaign, there were apparently thousands of public letters of protest to the Environment and Health Ministers' offices from across Canada. With the publication of the ATSDR report and the Coalition's estimates of the number of children affected (regardless of whose estimated

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<sup>93</sup> Water Quality Board of the International Joint Commission. *Report on Great Lakes Water Quality*. Presented at Toledo, Ohio, IJC Meeting. (November, 1987)

<sup>94</sup> Canadian Medical Association, Policy Summary (1987). From the 1987 Annual General Meeting: "The CMA regrets the recommendation of the Royal Society of Canada to postpone the reduction of lead in gasoline to 0.026 g/L and requests the federal government to make the move in 1990 as previously recommended."

<sup>95</sup> Canadian Coalition for Lead-free Gasoline. *Lead in 1988: More Urgent Than Ever*. Brief presented to the Hon. Tom McMillan, Minister of the Environment, and the Hon. Jake Epp, Minister of National Health and Welfare. (June 15, 1988)

<sup>96</sup> *Ibid.* This estimate assumed a mean blood-lead level of 7 - 8  $\mu\text{g}/\text{dL}$ , a geometric standard deviation of 1.42 - from the Ontario Blood-Lead Survey of 1984, in a log normal distribution.

<sup>97</sup> Personal communication with K. Cooper, Canadian Environmental Law Association. (October 1987)

<sup>98</sup> Canadian Coalition for Lead-free Gasoline (1988), *op.cit.*, p.28; and Blount, J. Report fuels demand for leaded gas ban, *Globe and Mail*. (August 16, 1988), p. A9.

average blood-lead level was considered) news coverage was extensive, followed by editorial columns in newspapers across Canada supportive of the Coalition's position. It was also an election year. Shortly thereafter, the federal government announced a total phase-out, or ban, on leaded gasoline, to take effect on January 1st, 1990.

#### 8.4.2 *Smelters and Soil*

Lead emissions from the primary smelting of lead ore and the secondary smelting of lead products (car batteries, cables, etc.) have contributed to localized contamination of air, soil, and street and house dust in communities around the world including several across Canada. The federal and provincial governments have responded with various regulatory limits, including federal limits of air emissions from secondary lead smelters and industrial effluent emissions to water. Provincial limits (varying from province to province) have been placed on stack emissions, ambient air, dustfall, unwashed plant foliage, marine and freshwater effluents, sewer discharges, sewage sludge, irrigation water, livestock watering, and soil.<sup>99</sup>

To illustrate central issues in these diverse circumstances, this section focuses on the controversy and control strategies surrounding two secondary lead smelters in Toronto. The story is fairly typical of the situations that have occurred in most communities where lead industries have been located: initial stages of intense denial of responsibility by the industry; inadequate and at times, arguably negligent, initial and even protracted responses from regulatory agencies; enormous controversy around studies to assess effects, control emissions and decide on clean-up strategies; protracted and acrimonious disputes throughout the time that scientists continued to reveal increasing evidence of harm at lower and lower exposure levels; repeated blood-lead testing; legal actions; extensive soil removal or remediation strategies; and, eventual and steady reduction in emissions via regulatory controls, improvements in pollution control technology and cleaner operations.

All of the above conditions were part of the story of the two Toronto secondary smelters.<sup>100</sup> For over 40 years, these smelters released lead into the South Riverdale and Niagara neighbourhoods in Toronto. Several cases of acute lead poisoning occurred in the 1960s and children continued to be hospitalized into the early 1970s. Until residents began pressuring the government for action in the early 1970s, very little was known about the excessive lead contamination in these neighbourhoods.

The Ontario government did not test for lead in either neighbourhood until 1972 when a test conducted in response to a complaint about noise and heavy dustfall revealed astronomically high levels of lead ranging from 22% (220,000 ppm) to 43.3% (433,000 ppm) in dust on a backyard barbecue and picnic table. (Note that ore containing 5% lead is considered minable.) The battery crushing operation at the smelter was ordered shut down and subsequent tests revealing lead levels of 2% to 7% were considered, by the Ministry, to be acceptable and not a hazard to residents. Blood-lead testing showed many area residents with levels over 40  $\mu\text{g}/\text{dL}$  but government officials stated that only levels of 80  $\mu\text{g}/\text{dL}$  or higher were cause for concern.

Independent testing by University of Toronto scientists confirmed excessive localized contamination

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<sup>99</sup> See summary chapter and appendix in Wallace, B. and K. Cooper. (1986) *The Citizen's Guide to Lead, op.cit.*, Chapter 13 and Appendix J.

<sup>100</sup> This account summarized from Wallace, B. and K. Cooper. (1986) *The Citizen's Guide to Lead, op.cit.*, pp. 73-75; and Wallace, B. and K. Cooper. *Lead, People and the Environment*, A report prepared for the Niagara Neighbourhood Association. (October, 1985), Section C, pp. 77-142.

around the Niagara Neighbourhood smelter and for the first time, revealed the even larger problem around the South Riverdale smelter. For several years, the Ministry of the Environment refused to accept that there was a problem. The lead industries involved threatened media and concerned public officials with legal action. Eventually, several large investigations were conducted, lead standards for air emissions were either established or revised downward and the matter went to a public hearing in 1976 to determine, among other things, the extent of soil clean-up required.

The lead standards set during this controversy were based on information then available, that is, they were based on information about the amount of lead in human diets, absorption rates for lead in food and air, and the amount of lead that could be safely consumed on the basis of what was then known or agreed upon concerning health effects. The standards were set in the mid-1970s so by the early to mid-1980s, all of this information was out of date or shown to be incorrect. Nevertheless, during the 1970s, standards (or in most cases largely unenforceable guidelines) were set for lead in ambient air, dustfall, unwashed plant foliage, and soil. The most important and controversial limits were for lead in dustfall and lead in soil.

The various investigations of the two smelters revealed that dust and soil contamination in the two neighbourhoods resulted from excessive dust fallout that could be attributed in large measure to routine fugitive emissions (from the yard operations, out of broken windows, etc.) rather than entirely from stack emissions. The dustfall guideline of 100 milligrams of lead per square metre over a 30 day period ( $\text{mg}/\text{m}^2/30$  days) was set on the basis of limited information. However, at this dustfall level it was assumed that soil-lead accumulation would increase only 300 ppm over twenty years. In fact, soil-lead levels increased by more than 300 ppm in *one* year in a backyard near one of the smelters. It is possible that this increase was a reflection of the many exceedances of the guideline. However, it confirmed that the existence of an unenforceable guideline did not prevent dramatic increases in soil lead levels.

At the public hearing held in 1976, arguments were made for a soil removal guideline of 1000 ppm. However, the Hearing Board agreed with the arguments of some government officials and the industry concerning lack of definitive proof of effects and went with a higher level of 2600 ppm. This level was chosen on the basis of one small study on rats, eventually published in 1980. Large scale soil removal operations were conducted in the two neighbourhoods. During the early 1980s, the soil removal guideline was revised down to 500 ppm and another large scale soil removal operation occurred, this time coupled with intensive house dust cleaning as well. The two industries involved managed to avoid paying for more than a third of the first soil removal operation and none of the second soil removal and house dust cleaning operations although the total price tag for both was well over \$20 million.

Further reductions in lead standards were recommended in Ontario in the early 1990s and are discussed in the next section.

### **8.4.3 Ontario's Multi-Media Approach**

After nearly 20 years of experience dealing with controversy surrounding the two Toronto lead smelters, confronting the steady increase in scientific information about health effects at lower and lower exposure levels, and being leaders in Canada in the surveying of children's blood-lead levels, the Ontario government, through the Ministry of Environment (MOE) undertook a review of its standards and guidelines for lead in soil, water and air.

In 1993 the Ministry published its rationale<sup>101</sup> for revising these three criteria and in 1994 published the

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<sup>101</sup> Ontario Ministry of Environment and Energy. *Rationale for the Development of Soil, Drinking Water, and Air*

companion scientific criteria document<sup>102</sup> outlining the multi-media approach applied to the development of the revised lead standards. The multi-media approach was, in many respects, an impressive step forward. As discussed in Chapter 4, the following summary is an example of the application of the two-step process of risk assessment and risk management in deriving a final set of multi-media standards.

The MOE documents summarized the international scientific consensus on children's health effects at and possibly below blood lead levels of 10  $\mu\text{g}/\text{dL}$ . At the time, an estimated 18,000 children in Ontario had blood lead levels above 10  $\mu\text{g}/\text{dL}$ . The study made it clear that, in the preceding decade, even though lead exposure had both dropped and sources had changed, there was still cause for concern and regulatory standards were in need of revision. Lead in soil was seen to be an exposure medium of particular importance.

The study proceeded by first deriving an Intake of Concern (for the population),  $\text{IOC}_{\text{pop}}$ , for lead. The  $\text{IOC}_{\text{pop}}$  of 1.85  $\mu\text{g}/\text{kg}/\text{day}$  was calculated by halving a daily lead intake that roughly corresponds to that which would result in a blood lead level of 10  $\mu\text{g}/\text{dL}$  (3.7  $\mu\text{g}/\text{kg}/\text{day}$ ). Applying a factor of two was intended to account for variability in the population and uncertainty. Hence, the  $\text{IOC}_{\text{pop}}$  is intended to be preventative to ensure that on an individual basis, children's blood lead levels do not exceed 10  $\mu\text{g}/\text{dL}$ .

To determine soil, drinking water and air limits, the multi-media approach was structured to consider all potential sources of exposure simultaneously. The four media or exposure pathways considered were food, water, soil and air. Consumer products were not included in the analysis - a potentially significant shortcoming (see Section 8.4.6 below). The proposed limits for soil, drinking water and air (food limits are not within provincial jurisdiction) were health-based criteria derived using calculations that took into account the lead exposure that could be expected from each of the four media and the particular susceptibilities of children. For example, the proposals recommended revising the soil removal guideline (for decommissioning or clean-up of contaminated lands to a standard considered acceptable for residential redevelopment) from 500 ppm to 200 ppm. The lower level was derived by making several calculations:

- 1) an allocation factor of 64% (calculated as the amount of a child's lead intake that can be generally attributable to ingestion of soil or dust, based on typical soil lead concentrations of 150 ppm and house-dust lead concentrations of 200 ppm and considering child-specific lead absorption rates);
- 2) a soil/dust consumption rate of 80 mg/day;
- 3) assuming an average body weight of 13 kg (average weight of a child in the relevant age range); and
- 4) applying the above three calculations to the  $\text{IOC}_{\text{pop}}$  of 1.85  $\mu\text{g}/\text{kg}/\text{day}$  (noted above).

The result was a health-based soil limit of 192 ppm which was rounded up to 200 ppm. In summary, this limit would mean that if a child weighing 13 kg ingests 0.08 grams of soil or dust each day, then a soil lead concentration of 200 ppm would limit the child's lead intake to less than 1.2  $\mu\text{g}/\text{kg}/\text{day}$ , or 64% of the  $\text{IOC}_{\text{pop}}$  of 1.85  $\mu\text{g}/\text{kg}/\text{day}$ . Note that this level was then proposed to be used only for industrial site decommissioning and clean-up since it is recognized that soil lead levels in urban areas can be much higher than 200 ppm. Hence, the revised soil removal limit will do nothing to reduce exposure in areas along busy roads or in central city areas where soil levels are often much higher than 200 ppm.

Health-based levels were derived for each of soil, drinking water and air. For drinking water, the health-based limit was 4.5  $\mu\text{g}/\text{L}$ . However, the existing Ontario Drinking Water Objective (ODWO) of 10  $\mu\text{g}/\text{L}$  was proposed due to the high cost of achieving the lower health-based limit. Continuation with the

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*Quality Criteria for Lead.* Hazardous Contaminants Branch. (October, 1993)

<sup>102</sup> Ontario Ministry of Environment and Energy. Scientific Criteria Document for Multimedia Environmental Standards Development - Lead. (March 1994)



existing ODWO for lead was estimated to result in an approximate increase of lead exposure of 11% over the limit defined by the  $IOC_{pop}$ . It was also concluded that the practice of flushing standing water from pipes fairly consistently ensures that drinking water lead levels are below the  $10 \mu\text{g}/\text{dL}$  limit. Similarly, the health-based criteria for the 30 day average ambient air quality criteria (AAQC) was  $0.05 \mu\text{g}/\text{m}^3$ . However, the recommended limit was  $0.7 \mu\text{g}/\text{m}^3$  to take into account what was considered technically and economically achievable by a model secondary lead smelter. Again, a calculation was made finding that this increase above the health-based criteria could result in an approximate additional increase of 14% in total lead exposure over the limit defined by the  $IOC_{pop}$ .

The multi-media approach is laudable in many respects and well-supported recommendations were made to revise air and soil standards. The study also recommended prudently that standards, guidelines and objectives proposed in the report should be reviewed as new information becomes available. Numerous recommendations were made concerning means of avoiding exposure from lead in paint and consumer products. However, several problems are evident in the approach.

First, the use of soil and house dust lead levels typical of suburban environments (150 to 200 ppm) in these calculations is problematic. Children living in homes with higher levels of lead in soil and house dust, as can be expected in many inner city locations, are more highly exposed (especially if deteriorating paint is present in older dwellings) and multi-media calculations that rely on these lower levels of lead in soil/dust will underestimate exposure for inner city children. This underestimate will arise both because of historical leaded gasoline contamination and older dwellings with leaded paint. Point source locations (i.e., communities adjacent to lead-emitting operations) will have significantly higher soil and dust lead levels. This latter fact was recognized but not accounted for in the multi-media study's estimated daily intake calculations for the population at large.

Second, the decision to stay with the existing ODWO instead of using the health-based limit is dependent upon regular flushing of standing water from pipes. The encouragement of this practice occurred via awareness campaigns that followed on media attention when the problem was identified in the late 1980s. It is debatable whether, many years later, there is sufficient follow-through and assurance that flushing of pipes is actually occurring in homes, schools and daycare centres (lead in drinking water is discussed further in the next section).

Similarly, it is debatable whether there is general public awareness about the content of the many recommendations in the multi-media study regarding lead in consumer products. Moreover, the multi-media study did not, and understandably could not, account for new and unexpected sources in consumer products (such as lead in crayons, mini-blinds, and the large range of children's toys and accessories recently discovered by Greenpeace and others - see discussion in Section 8.4.6 below).

The multi-media exposure assessment showed that the total exposure of the Ontario population had declined dramatically over the previous decade. Daily lead intake from all major pathways for young children was estimated to be  $1.9 \mu\text{g}/\text{kg}/\text{day}$ . Note that this level is essentially the same as the  $IOC_{pop}$  of  $1.85 \mu\text{g}/\text{kg}/\text{day}$  used in the multi-media analysis. Recognizing that this level of exposure leaves a very small safety margin for typical urban children, the study notes that the  $IOC_{pop}$  could easily be exceeded under higher exposure scenarios.

The multi-media approach and the resultant proposals for regulatory limits did not leave very much of a safety margin for known sources of additional exposure such as deteriorating lead-bearing paint or the various consumer products noted in the study including lead-bearing ceramic dishes, hobbyist materials, and lead shot and fishing sinkers. The study only includes detailed (and appropriate) recommendations for avoiding lead from these additional, known (at the time) sources. Nor could the study and resultant standards provide much of a safety margin for these additional sources when the baseline exposures from

the four media covered are already so high due to historical circumstances.

The fact remains however that an approach that attempts to cover all media is destined to become out of date relatively quickly when new and unexpected sources of lead (not even foreshadowed in the multi-media report) continue to arise and the baseline level of exposure will be a problem for many years to come. This baseline level is not only the result of historical contamination from leaded gasoline emissions and contamination around point sources but, as the multi-media study points out, there are an estimated 2,056,850 homes in Ontario built before 1970 which may be affected by lead-based paint. If poverty levels continue to increase, this exposure source could well become as serious a problem in the future as has been and continues to be experienced in many inner city areas of the U.S.

#### **8.4.4 Lead in Drinking Water**

Lead in drinking water is not regulated by the federal government although guidelines have been set since the 1970s. A guideline of 50  $\mu\text{g}/\text{L}$  (equivalent to 50 ppb) was set in the early 1970s and was revised down to 10  $\mu\text{g}/\text{L}$  in the late 1980s following on media publicity around testing done by the Canadian Broadcasting Corporation (CBC) that revealed drinking water in some schools with lead levels above the 50  $\mu\text{g}/\text{L}$  guideline.<sup>103</sup> It was clear that lead levels were higher in first draw water that had been standing in contact with lead-soldered pipes for longer than six hours. Flushed samples revealed much lower levels. The federal and Ontario governments responded by lowering the drinking water guidelines for lead to 10  $\mu\text{g}/\text{dL}$ . Unfortunately, the federal guideline is for flushed samples instead of “first draw” or standing water samples thus ignoring the key exposure source which results from water standing in contact with lead-soldered pipes. In Ontario, the *Building Code*<sup>104</sup> was amended to prohibit the use of lead solder for potable water supplies. However, by not banning lead solder altogether, as had been done in the U.S. in response to the same problem, lead solder continues to be available and can be misused by unlicensed plumbers or plumbers who may chose to ignore the rules (or homeowner “do-it-yourselfers” who are unaware of the rules).

Lead levels in drinking water sampled in Ontario appear to be relatively low and generally within the guideline although levels are consistently higher in the first draw samples than in flushed samples, with many more exceedances of the guideline in the former case than in the latter.<sup>105</sup> Higher lead concentrations in the first draw or standing water samples were associated with low pH, low alkalinity, lead pipes or lead-soldered copper plumbing. However, elevated levels were also recorded in some flushed samples when water was “non-aggressive” (i.e., it did not have a high or low pH) but did have lead service connections. The result of these surveys reinforces the need for routine educational activities to remind parents, and other child caregivers of the measures that can be taken to ensure that first draw or standing water is not consumed by young children.

#### **8.4.5 Lead in Food**

As noted in the discussion of lead in gasoline, when the detailed investigations of the early 1980s were conducted, primarily in the U.S., it became clear that lead in gasoline contributed at least 50% of the lead contamination of food. It was also clear that alongside dust/soil, food was one of the most significant

<sup>103</sup> Ontario Ministry of Education, undated. Report on the Survey of Drinking Water in Ontario Schools for Lead.

<sup>104</sup> *Building Code Act*, R.S.O. 1992, c. 23.

<sup>105</sup> Ontario Ministry of the Environment. (1994), *op.cit.* Appendix D, Section D-2.2.2.

sources of human lead exposure. Lead-soldering of food cans was found to increase the lead contamination of food by as much as 5 to 10 times.

In Canada, the *Food and Drugs Act*<sup>106</sup> governs the advertisement and sale of food, drugs, cosmetics and therapeutic devices. It was adopted to prevent public deception regarding these products and to prevent injury to the health of the purchaser or user. The Act prohibits the sale of any food that has in or upon it any poisonous or harmful substance, is unfit for human consumption or is adulterated.

With the exception of partially assisting with the eventual voluntary phase-out of lead-soldered cans towards the end of the 1980s, regulation of lead in food in Canada has been a largely meaningless exercise. A regulation was enacted in 1968 setting maximum lead levels for a range of foodstuffs.<sup>107</sup> The list of foods was not particularly representative of the foods consumed in Canada. As already noted, the information about health effects of lead exposure in the 1960s included a blood-lead concern level of 80  $\mu\text{g}/\text{dL}$ . The regulated levels were based on then-current health effect information and they were extremely high (generally one to three orders of magnitude higher than actual lead levels found in food). Hence, assurances that regulated levels were not being exceeded would have been virtually meaningless.

Where the regulated levels were closer to lead levels that would actually have been found in food, they were established to address two contamination sources: the use of lead arsenate as a pesticide on apples (phased-out during the 1970s) and lead soldering of cans. In these cases, the regulated levels were generally not much different than the levels that could be found in apple products or lead-soldered canned food. As noted, it was well established that lead-soldering could increase the lead content of food by 5 to 10 times and represented a significant contribution to dietary sources of lead.

The federal government response in 1979 to this information was to revise the lead in food regulations in a minimal fashion by revoking the limits for a range of irrelevant foodstuffs (e.g., lead in a range of baking ingredients, etc.) and to re-publish the revised regulations for a more representative range of foods.<sup>108</sup> However, it was an even smaller list than had been prepared in 1968 because, although not explicitly stated, the revised list was intended to address foods that are generally found in cans, and, at the time, often in lead-soldered cans. For those foods that had been on the 1968 list, the 1979 regulated levels were unchanged from those set in 1968. Additional foodstuffs on the 1979 list included evaporated milk, condensed milk and concentrated infant formula, all of which were more likely or exclusively to be fed to children and which were often packaged in lead-soldered cans. Still another addition was made in 1986 when lead limits were placed on tomato paste and sauce and whole tomatoes, and again it was unstated but implied that these were canned products.<sup>109</sup> The regulatory limit for all of these products was, again, not much different than the lead level that was typically found in these foods if they were in lead-soldered cans. Indeed, testing by Health Canada in 1988 confirmed that infant formula in lead-soldered cans was routinely at or significantly above the regulatory limit.<sup>110</sup> With increased publicity throughout the 1980s about the health effects of lead and the significant contribution of lead-soldered cans to dietary intake, food manufacturers responded to negative publicity and public pressure and took the initiative to

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<sup>106</sup> *Food and Drugs Act*, R.S.C. 1985, c. F-27.

<sup>107</sup> *Food and Drug Regulations*, C. R. C., c. 870, B. 15.001.

<sup>108</sup> SOR/79-249.

<sup>109</sup> SOR 86/258.

<sup>110</sup> Dabeka, R.W., Food Research Division, Bureau of Chemical Safety, Health and Welfare Canada. Graphite Furnace Atomic Absorption Spectrometric Determination of Lead and Cadmium in Canadian Infant Formulas and Calculation of Dietary Intakes of Lead and Cadmium by Infants. Presentation made at the Third Chemical Congress of North America. (June 8, 1988) and for Release at Press Conference.

gradually phase out lead-soldering of cans. Regulatory action by the federal government had little to nothing to do with this change and regulatory standards for lead in food were never, and still are not, protective of children's health.

## **8.4.6 Lead in Consumer Products**

### **8.4.6.1 Introduction**

Modern lead usage in a wide variety of consumer products continues to occur for the same reasons lead has always been used: it is cheap, plentiful, malleable, strong, and it can produce bright colours and durable surfaces. Regulatory controls in Canada and other developed countries have been placed on the lead content of consumer products alongside the controls discussed thus far.

As discussed in Chapter 7, consumer products are regulated by Health Canada under authority of the *Hazardous Products Act*.<sup>111</sup> The application of this Act to consumer products is both limited and unclear. It is difficult to determine whether a particular children's product is regulated under the Act and its regulations, and if so, whether it is in compliance with the regulatory stipulations. Product inspection is also problematic. Recent discoveries of many unexpected and very hazardous sources of lead in consumer products (as discussed in Section 8.4.6.4 below) belies any assumptions that the public may have concerning product safety. Many people reasonably but mistakenly assume that if a product is on the shelf, especially if it is intended for children, that it has been tested in some way to determine whether it is safe; such testing does not occur and *may* only occur after a problem has been detected (see Section 8.4.6.4 below and Chapter 7).

Also discussed in Chapter 7 is the fact that Health Canada has no power to recall hazardous products from store shelves and instead, for both regulated and unregulated products, issues public advisories and warnings.<sup>112</sup>

The earliest regulations for lead in consumer products in Canada were established for lead in Ceramics, Glassware and Kettles (Section 8.4.6.2) and lead in paint (Section 8.4.6.3). The 1990s have seen a wide variety of new and unexpected sources of lead in products intended for children.<sup>113</sup> These are discussed in detail in Section 8.4.6.4 followed by a review of Health Canada's seemingly stalled "Lead Reduction Strategy."

### **8.4.6.2 Lead in Ceramics, Glassware and Kettles**

Regulations governing the amount of lead that can leach from glazed ceramics<sup>114</sup> and from kettles with

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<sup>111</sup> *Hazardous Products Act*, R.S.C. 1985, c. H-3.

<sup>112</sup> Under authority of the *Department of Health Act*, R.S.C. 1996, c. H-3.2.

<sup>113</sup> Not discussed in detail here is the fact that the federal government recently banned the use of lead shot for hunting waterfowl. It is noteworthy that the current Ontario government opposed this federal action. Lead shot can still be used for upland game. Moreover, the fact that lead is readily available (e.g., in scrap yards, from tire balancing in car garages, etc.) contributes to the fact that hunters can and do readily make their own shot making it possible for them to ignore/neglect restrictions on using lead shot for waterfowl.

<sup>114</sup> Hazardous Products (Glazed Ceramics) Regulations, C.R.C., c. 925.

lead-soldered seams<sup>115</sup> were also set in the 1970s and on the basis of health information current at that time. The requirement that ceramics could not release more than 7 ppm of lead, (into a 4% acetic acid solution when allowed to stand for 18 hours at room temperature), which remained until 1998, was as out of date and inadequate as the lead in food regulations, and for the same reasons. Similarly, the limit on the amount of lead that can leach from lead-soldered seams in kettles (0.05 ppm or 50 parts per billion) is not protective. For comparison, the lead in drinking water standard in Ontario is now 10 µg/L (or 10 parts per billion) and, as noted in Sections 8.4.3 and 8.4.4 above, the health-based limit is even lower.

Kettles are apparently no longer soldered with lead<sup>116</sup> but some ceramics, mostly imported from Latin America or China, can contain lead. Leaching of lead from such products can be significant; some products have caused serious cases of acute lead poisoning. Revisions<sup>117</sup> in 1998 brought the *Hazardous Products Act* regulations in line with rules in the U.S. and broadened the regulations to apply to a range of ceramic and glassware products. The amount of lead allowed to leach from these products varies depending on the use. For example, permitted levels are lower for containers like pitchers and cups that would be used to hold or store liquids than for plates or small bowls. The test for lead involves determining how much lead can leach from the surface after it has been in contact with a 4% acetic acid solution (comparable to the acidity of vinegar) for 24 hours at room temperature. The higher the acidity of the food and the longer the storage time, the greater is the amount of lead that could leach into the food.

The new regulations place similar leachability requirements on lead contained in decorative patterns on the exterior surface of cups or glasses and require that such patterns be located a minimum of 20 millimetres below the rim.

While the harmonization with U.S. standards is of assistance to the industries producing and importing ceramics and glassware, the revised lead levels can potentially permit significant dietary lead intake. For example, the new regulation stipulates that a glazed pitcher cannot leach more than 0.5 mg/L of lead into a 4% acetic acid solution if left for a 24 hour period. This regulatory level is equivalent to 500 parts per billion. If a child were regularly exposed to acidic foods (such as orange juice) contained, and especially stored in lead-glazed ceramics that approached this regulatory limit, dietary lead intake would be extremely high. While the likelihood of this kind of routine exposure is low, these revised standards are not particularly protective of young children.

### 8.4.6.3 Lead in Paint

Lead in paint regulations in Canada are similarly outdated and proposed revisions are both problematic and have languished in the “proposal” stage for almost three years. Canadian regulations for lead in paint were set in 1970 placing a limit of 5000 ppm of lead in paint used on furniture, toys and other articles intended for children. In 1973 this limit was also applied to paint or decorative coatings on pencils and artists’ brushes. In 1978, the 5000 ppm limit was extended again to cover interior paints, and furniture or household products used in non-commercial/industrial buildings.<sup>118</sup> Paint with a higher lead content could continue to be used on the exterior surfaces of all buildings, and on the interiors and contained

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<sup>115</sup> Hazardous Products (Kettles) Regulations, C.R.C., c. 927.

<sup>116</sup> This assumption is apparently no longer valid with the recent discovery, primarily in the Toronto area, of lead-soldered kettles imported from Turkey capable of leaching extremely high levels of lead.

<sup>117</sup> SOR/98-176.

<sup>118</sup> Hazardous Products (Liquid Coating Materials) Regulations, C.R.C., c. 928.

furniture of industrial/commercial premises, as well as all other buildings that children do not, or are unlikely to frequent, provided that the paint is labelled appropriately as containing lead. The Canadian limit of 5000 ppm was extended again, in 1985, to carriages and strollers<sup>119</sup> and in 1988, to cribs and cradles.<sup>120</sup>

In contrast to this complicated situation, the U.S. revised its regulation<sup>121</sup> for the lead content of both interior and exterior paints from 5000 ppm to 600 ppm in 1978, a level that remains nearly eight times lower than the Canadian regulation. Since at least the mid-1980s, children's health advocacy groups in Canada have been urging an updated regulation to match the U.S. standard and in particular, an end to the distinction between paint used in buildings that may or may not be "frequented by children."

By 1990, Health Canada had briefly considered harmonizing with the U.S. standard but chose instead a voluntary arrangement with paint manufacturers to limit lead in paint and coatings to the U.S. standard of 600 ppm. The result has been that most paint in Canada is likely to be within the 600 ppm limit since much of it is imported from manufacturers in the U.S. However, a 1991 study addressing lead in paint made two significant findings: eight percent of consumer respondents had used exterior paint, (for which there was/is no limit for lead content), on interior surfaces; and one manufacturer used lead pigments in an exterior stain for wood at a level of 20% or 200,000 ppm, while another had used lead pigments in exterior paint at a level of 5%, or 50,000 ppm.<sup>122</sup>

In 1997, in an effort to please all stakeholders, Health Canada proposed a new regulation that would implement the 600 ppm limit but only for the interior and exterior walls of buildings frequented by children, as well as for furniture and household products in such buildings.<sup>123</sup> For other surfaces, including the exterior and interior surfaces, and contained furniture, of buildings not frequented by children, the proposed regulation only sets out labelling requirements to warn of lead if it is present, with no upper limit on the amount of lead that can be used. This complicated compromise was apparently intended to appease municipalities that have large stockpiles of high lead content paint and that want to be able to either use the paint or to direct it to the paint recycling industry.

Several national organizations concerned about children's health and welfare strongly objected to the proposed regulation on several grounds. The notion that furniture and buildings will remain unfrequented by children is dubious at best. Nor does this notion address exposure risks for pregnant women or indeed, women of child-bearing age. Occupancy and use of buildings can change regularly and significantly over time. Child care centres for example are always looking for inexpensive locations to keep down overhead costs. In reply to such concerns, Health Canada's Product Safety Bureau stated that "the potential health risks from these scenarios would have to be addressed by the property owners or occupational health and safety authorities depending on the particular situation." This approach seems a very poor way of ensuring health protection; the likelihood of records being kept on the type of paint used seems very remote.<sup>124</sup>

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<sup>119</sup> Carriages and Strollers Regulations, SOR/85 - 379.

<sup>120</sup> Cribs and Cradles Regulations, SOR/86-962.

<sup>121</sup> 16 C.F.R. s.1303 (1998) (Cornell Law, <http://www.law.cornell.edu/cfr/16p1303.htm>).

<sup>122</sup> ABT Associates of Canada. Evaluation of the Regulations for Lead and Mercury Content in Paints: Project Report to Health Canada.(1991)

<sup>123</sup> Notice (Proposed Regulatory Text), *C.Gaz.* 1997.I.1759. Notice (Order), *C.Gaz.* 1997.I.1755.

<sup>124</sup> Personal communication with Barbara McElgunn, Learning Disabilities Association of Canada (June, 1998)

While the proposed regulation will finally put the 600 ppm limit into Canadian law, it does so ambiguously by allowing continued inappropriate uses to be “controlled” by labelling requirements. Hence, the proposed regulation, still not promulgated as of February 2000, focuses primarily on labelling requirements for the continued use of lead rather than taking the regulatory action chosen in the U.S. over 24 years ago.

#### 8.4.6.4 New and Unexpected Sources

With all that is known about lead contamination and health effects in children it is surprising that new exposure sources seem to continually arise. It is less surprising given that these new sources arise almost consistently in consumer products that are imported from developing countries with weak or non-existent laws concerning product safety and environmental or occupational exposure to toxic substances.

With the increasing economic globalization and free trade agreements of recent years, a related agenda has become apparent. Throughout the industrialized world, trade agreements have been accompanied by governmental reluctance or refusal to pass new legislation or regulations and increasing movement of production facilities to developing countries including virtually unregulated “export processing zones.” Governments are increasingly opting for voluntary arrangements and, in many cases, they are also agreeing to roll back existing environmental, occupational or product safety controls in the face of corporate lobbying as well as resistance and/or refusal to accept regulatory controls on industrial operations or products.<sup>125</sup> In this context, the use of lead in many developing countries is poorly regulated in both occupational and environmental settings. Several recent examples are discussed here.

##### *Lead in Crayons*

The first is the discovery in 1994 of high lead content in crayons imported from China, and labelled “non-toxic.” The discovery occurred due to routine screening for blood-lead levels in U.S. children. An eleven month old child was found to have a blood lead level of 43  $\mu\text{g}/\text{dL}$ . Follow-up investigations revealed high levels of lead (800 ppm) in orange crayons. The discovery led to additional testing, findings of other crayons from China with high lead levels, and subsequent port seizures and a federal recall of over a dozen brands of crayons.<sup>126</sup> In Canada, products were not recalled. Indeed, as noted above, no authority exists under the *Hazardous Products Act* to recall products. Health Canada only issued a warning to parents and caregivers of children.

##### *Lead in Plastic Mini-blinds*

A more recent and much larger exposure source was the discovery of lead in plastic mini-blinds imported from China, Taiwan, Mexico and Indonesia. The Arizona Department of Health Services (ADHS), the same agency that discovered the lead-bearing crayons, investigated a case in 1995 of a one-year-old boy with a blood lead level of 37  $\mu\text{g}/\text{dL}$ . Although not discovered via the U.S. practice of routine blood-lead screening, the mini-blinds discovery did occur as a result of mandatory reporting of elevated blood-lead levels to public health authorities. The ADHS investigation eventually isolated the source to a plastic mini-blind within reach of the child’s crib. The ADHS issued a lead warning about the blinds.<sup>127</sup> The

<sup>125</sup> See for example: Swenarchuk, M. and P. Muldoon, De-regulation and Self-regulation, A Public Interest Perspective, presented at a workshop on De-regulation, Self-regulation and Compliance in Administrative Law. March 1996, Canadian Environmental Law Association.

<sup>126</sup> Arreola, P., *et al.* Lead-Tainted Crayons From China Part I: Secondary Prevention in Arizona. *Environmental Health*. (March, 1996), pp.6-15.

<sup>127</sup> Arizona Department of Health Services. *Miniblind Lead Warning Issued*, News Release. (December 7, 1995)

initial response from the U.S. Consumer Product Safety Commission was to discount the concerns.<sup>128</sup> Soon after, similar findings arose in North Carolina of high lead levels on mini-blinds, associated with elevated blood lead levels in a child in a daycare centre. The Window Covering Safety Council in the United States denied any association between the blinds and elevated blood lead levels.<sup>129</sup>

Further testing by the U.S. Consumer Products Safety Commission confirmed that the blinds deteriorated in sunlight causing a layer of lead-bearing dust to form on the surface of the blinds. A national advisory was issued recommending that the blinds be removed from homes with children under six years of age.<sup>130</sup> Over 25 million blinds had been sold in the U.S. and over 8 million had been sold in Canada.

The response in Canada was to issue a similar consumer advisory based on a risk assessment<sup>131</sup> that contained a significant error. In calculating the likely exposure in house dust, the risk assessment calculations incorrectly used the *average*<sup>132</sup> level of lead in the dust on the blinds as being representative of the 90th percentile. In so doing, the entire risk assessment greatly underestimates the potential exposure to lead from the most significant pathway - lead in dust. Nor does the risk assessment consider the likelihood of redistribution of lead dust from the blinds into house dust via cleaning activities, especially dry dusting. Even with these significant limitations, the study concluded that the blinds posed a hazard to young children. Neither the U.S. or Canada evaluated nor, initially, warned about the risks of these blinds to pregnant women. By issuing only an advisory, instead of a product recall, and limiting it to homes with young children, it is very likely that many of these mini-blinds were not removed and continue to represent a lead source to interior spaces that could eventually be “frequented by children.” Such blinds are also routinely found in second-hand and thrift stores or garage sales.

Follow-up investigations undertaken by the North Carolina Department of Health indicate that despite the U.S. Consumer Product Safety Commission’s (CPSC) 1996 hazard warning, mini-blinds remain a significant source of lead exposure for young children. 1998 investigations revealed dust lead levels from mini-blinds as high as 77,213  $\mu\text{g}/\text{ft}^2$  in the homes of children suffering from lead poisoning. The North Carolina Department of Health and Human Services, citing its concern that the CPSC actions have failed to protect young children, has called for a product recall to eliminate the continuing lead poisoning hazard posed by millions of mini-blinds that remain in windows across the U.S. Routine samples indicate that young children are being exposed to levels of ingested lead that on average exceed the U.S. windowsill standards (for old lead-bearing paint) by more than two orders of magnitude.<sup>133</sup>

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<sup>128</sup> Letter from Dr. Jack Dillenberg, Director, Arizona Department of Health Services to Robert G. Poth, Director, Division of Regulatory Management, U.S. Consumer Product Safety Commission. (May 1, 1996)

<sup>129</sup> Window Covering Safety Council. Mini Blinds Pose No Lead Poisoning Danger to Children: North Carolina Health Officials may have relied on discredited study. News Release. (date illegible, likely March or April of 1996)

<sup>130</sup> U.S. Consumer Product Saety Commission. *CPSC Finds Lead Poisoning Hazard for Young Children in Imported Vinyl Miniblinds*. News Release, Office of Information and Public Affairs. (June 26, 1996)

<sup>131</sup> Wood, G., Bureau of Chemical Hazards, Environmental Health Directorate, Health Canada. *Risk Assessment for Lead in Dust from PVC Mini-Blinds*. (July 5, 1996)

<sup>132</sup> The dust-lead information originated from the analyses done by the U.S. Consumer Product Safety Commission and reported in: Health Sciences Laboratory Mini-Blind Study Surface Lead Determination (May 30, 1996), obtained from the US CPSC Office of Compliance, Division of Regulatory Management (June 7, 1996).

<sup>133</sup> Letter from Ed Norman, Children’s Environmental Health Branch, North Carolina Department of Environment and Natural Resources, to the Honourable Ann Brown, U.S. Consumer Product Safety Commission. (October 15, 1997); memorandum from Kenneth Rudd, North Carolina Department of Health and Human Services, to Ed Norman, North Carolina Department of Health and Human Services. (June 15, 1998); and



***Lead in Children's Products***

As discussed in Chapter 7, toys, equipment and other products for use by a child in learning or play that contain a toxic substance are restricted products and can be regulated under the *Hazardous Products Act*. Plastic products are also regulated under the Act but only toys, equipment, or other products used by children under three years of age. In the course of this report's investigation, after several conversations with officials from Health Canada and Justice Canada, it was finally apparent that plastic children's products that are, or are likely to be used, by children of three years of age or older, are not regulated at all under the *Hazardous Products Act*.<sup>134</sup> The *Food and Drug Regulations*, under the *Food and Drugs Act*, having to do with toxic substances released from plastics in food packaging may also come into play but it is far from clear whether or how the standard for food packaging is applied to the plastics found in children's toys.

When the mini-blinds discovery was made, Greenpeace and others had long been warning about the other hazards of polyvinyl chloride (PVC) plastics, most notably the fact that they create dioxin when they are burned, for example in a garbage incinerator or the Plastimet fire in Hamilton. The discovery of lead in mini-blinds made Greenpeace suspect similar contamination in children's products made with PVC plastics. In 1997, Greenpeace investigated the lead and cadmium content of a range of plastic children's products.

The Greenpeace study revealed alarmingly high levels of both lead and cadmium in a variety of children's products that are readily available and commonly used across Canada and the U.S.<sup>135</sup> Products tested include plastic backpacks, rain clothes, assorted toys and toy cables (on headphones, toy phones, etc.). While Health Canada has proposed, as a guideline, a maximum total lead content in children's products of 15 ppm,<sup>136</sup> lead levels as high as 18,750 ppm were found in the products tested. Further tests into the level of extractable lead in these products, as well as the release of lead-containing dust from the products revealed exceedances of the daily ingestion limits set by the European Union (0.7 µgrams) and the U.S. Consumer Product Safety Commission (15 µgrams). High levels of lead and cadmium in children's products were confirmed by Greenpeace in a further, 1998 study.<sup>137</sup>

[Greenpeace has also revealed similarly dangerous levels of toxins called phthalates in these products. These plastic additives leach out of products when sucked or chewed by children. There is no regulatory limit for phthalate levels in children's plastic products in Canada.]

Four more new sources of lead in children's products surfaced in 1998. In March, Health Canada sent a mass mail-out to the figurine industry, requesting that lead not be used in the manufacture of "role-play figurines," commonly manufactured with 75% lead (or 750,000 ppm). In April, Health Canada issued a

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letter from A. Dennis McBride, State Health Director, North Carolina Department of Health and Human Services, to the Honourable Ann Brown, U.S. Consumer Product Safety Commission. (July 15, 1998)

<sup>134</sup> Personal communication with Louise McGuier-Wellington, Justice Canada. (May 18, 1999)

<sup>135</sup> Di Gangi, J. Lead and Cadmium in Vinyl Children's Products: A Greenpeace Exposé; Greenpeace Canada Briefing. (Oct. 9, 1997) Vinyl Children's Products Pose Lead and Cadmium Hazard.

<sup>136</sup> Health Canada, Strategy for Reducing Lead in Children's and Other Consumer Products, Discussion Paper, Draft II. (August, 1997)

<sup>137</sup> Greenpeace Release. (Nov. 1998), Greenpeace testing results for lead and cadmium in PVC plastic (vinyl) consumer products bought in Canada in late October, 1998; and Greenpeace Media Release. (Nov. 16, 1998). Leading child health and environmental organizations urge removal of hazardous vinyl children's products from sale.

consumer warning about lead exposure from *Kids Klub Necklace with Pendant*. The pendant was almost pure lead and caused lead poisoning in a child in Calgary. The pendant was capable of leaching (during mouthing or ingesting) 1022 ppm of lead. In June, Health Canada, with the manufacturer, recommended a recall of *GapKids* anoraks due to high lead content in the paint on the zippers. Again in October, under pressure from Health Canada, *Universal Studios* issued a news release concerning the lead content of “promotional” toys distributed with its video, “The Battle for Mount Olympus.” Two pendants, a ring and a sword, on the “promotional” necklace were almost pure lead: 72% and 73% respectively. Tests showed they were capable of leaching 104 ppm and 252 ppm respectively. For all of these examples note that the current (and under review - see Section 8.4.6.5 below) level of leachable lead considered acceptable for children’s products is 90 ppm.<sup>138</sup>

#### ***And Still More Sources***

In addition to the Turkish kettles noted in Section 8.4.6.2 above, concerns have recently been raised about lead in calcium supplements and in the wicks of some candles.

Ironically, calcium supplements are known to decrease gastrointestinal lead absorption<sup>139</sup> and are therefore heavily marketed and routinely recommended for children and pregnant women at risk of excess lead exposure. They are routinely recommended for all women approaching or after menopause to prevent osteoporosis. However, on the basis of their findings of high lead levels in calcium dietary supplements (including antacids), the Natural Resources Defense Council (NRDC), along with five other national organizations in the U.S., petitioned the U.S. Food and Drug Administration for a rule limiting lead in these products.<sup>140</sup> The NRDC study found both high lead levels in these products and evidence of available technology and production methods to minimize this lead content.

The finding of lead in candle wicks releasing potentially dangerous levels of lead into the air occurred in late 1999 by long-time heavy metals expert Jerome Nriagu.<sup>141</sup> His study showed lead emission rates in excess of the ambient air quality criterion set by the Environmental Protection Agency for outdoor air.

#### **8.4.6.5 Health Canada’s Lead Reduction Strategy**

Health Canada began, in 1997, to develop a national strategy on lead reduction. Three years later this strategy remains in draft form. Although the strategy is apparently being expanded to cover more products, it is unavailable for public review.<sup>142</sup> The lofty rationale for the strategy was to address the fact that the historical approach of reducing lead exposure on a product-by-product basis has resulted in an

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<sup>138</sup> Subramanian, M., Acting Director, Product Safety Bureau, Health Canada. Undated speech apparently delivered in February, 1999; and Health Canada (1998) News Release: Warning. Potential Lead Exposure From Kids Klub Necklace with Pendant. (April 22, 1998) [Http://www.hc-sc.gc.ca/main/hc/web/english/archives/warnings/98\\_23e.htm](http://www.hc-sc.gc.ca/main/hc/web/english/archives/warnings/98_23e.htm)

<sup>139</sup> Bruening, K, *et.al.* Dietary Calcium Intakes of Urban Children at Risk of Lead Poisoning, *Environmental Health Perspectives* 107(6) 431-435 (1999).

<sup>140</sup> Natural Resources Defense Council, Alliance to End Childhood Lead Poisoning, Physicians for Social Responsibility, Public Citizen, Sierra Club and the American Public Health Association, Citizens’ Petition to Initiate Rulemaking: Lead in Calcium Supplements. Available May 17, 1999 at [www.nrdc.org/nrdcpro/petit/calpet.htm](http://www.nrdc.org/nrdcpro/petit/calpet.htm) and [www.nrdc.org/nrdcpro/petit/appendix.html](http://www.nrdc.org/nrdcpro/petit/appendix.html)

<sup>141</sup> Some candles with lead wicks emit lead into the air, University of Michigan News Release, October 6, 1999. Available at: [www.umich.edu/~newsinfo/Releases/1999/Oct99/r100699.html](http://www.umich.edu/~newsinfo/Releases/1999/Oct99/r100699.html)

<sup>142</sup> Personal communication, Jonathan Williams, Product Safety Bureau, Health Canada, March 27, 2000.

ongoing situation of products being available that contain excessive amounts of lead. Instead, Health Canada concluded that preventive measures that avoid crisis situations are necessary. Other stated goals of the strategy are to enable industry to phase out the use of lead in non-essential applications and to provide industry with guidance to set quality control, purchase raw materials, etc. As well, the strategy is intended to create public confidence in the products and the marketplace.

A range of guidelines for achieving these objectives were outlined when the strategy was launched for consultation in 1997. These include: 1) the elimination of lead from all non-essential production applications; 2) lead should not be added during production to any product subject to this strategy; 3) the total lead content should not exceed 15 ppm which will be used to determine compliance with the strategy and the above two guidelines; 4) use of children's and other consumer products by children should not increase the overall body lead burden of the user; 5) industry (manufacturers, importers, retailers, and distributors) is responsible for voluntary compliance with the strategy; and 6) all products subject to the Health Canada strategy and guidelines will be in compliance by the year 2001.

Despite the appropriate (and long overdue) desire to put in place preventive measures, the approach was presented as overwhelmingly a voluntary one with the use of regulatory controls via the *Hazardous Products Act* seen as a last resort after problems are identified and exposure has occurred thus missing the stated intention of a more preventative approach. Consultation was fairly extensive on this strategy and plans were to finalize it in March of 1998. Nothing has yet emerged from the department in either further draft or final form. In early 1999 this absence appeared to indicate an intention to follow a stronger, regulatory approach. Dr. Mani Subramanian, Acting Director of Health Canada's Product Safety Bureau, indicated in a speech that Health Canada was instead proposing to put this strategy, essentially a set of unenforceable guidelines, into a regulation under the *Hazardous Products Act*.

If Dr. Subramanian's comments are to be taken literally Health Canada could become a world leader in implementing a binding regulation that will limit children's exposure to lead from consumer products. Following completion of consultations regarding the Lead Reduction Strategy, Health Canada stated that it was finalizing a fifth and final draft which would include a draft regulation under the *Hazardous Products Act* that would set lead limits in consumer products that are intended for use by children (up to 96 months of age). A variety of products including toys and playthings, children's jewelry, and carriages and strollers would be included. The regulation would prohibit the intentional addition of lead to such products, which would be deemed to have occurred when the total lead content in a product exceeds 65 ppm on a mass basis. Following this determination, two actions would be possible. When the migratable fraction of lead in the product exceeds 90 ppm, the product would be recalled. Should this fraction be less than or equal to 90 ppm, the distributor would be given a 12 month grace period to reduce the total lead quantity to 65 ppm or less. Failing this, the product would be pulled from store shelves.

Health Canada is also considering the inclusion of a special requirement for products that are intended to be mouthed by children. Should the migratable fraction of the lead in these products **exceed 30 ppm, or** if the total lead content of the product exceeds 65 ppm, the product would be pulled off the market.

Once in place, Health Canada states that it has plans to supplement this regulation with another regarding the remaining consumer products with which children are likely to interact, such as window and floor coverings, and furniture.<sup>143</sup>

By way of comparison, in the U.S., consumer products are regulated under the *Federal Hazardous*

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<sup>143</sup> Subramanian, M., Acting Director, Product Safety Bureau, Health Canada. Undated speech apparently delivered in February, 1999.

*Substances Act*.<sup>144</sup> A “hazardous substance” includes any substance or mixture of substances that are toxic,<sup>145</sup> if such substance or mixture of substances may cause substantial personal injury or substantial illness during or as a proximate result of any customary or reasonably foreseeable handling or use, including reasonably foreseeable ingestion by children. Household products that expose children to hazardous quantities of lead under reasonably foreseeable conditions of handling or use are therefore “hazardous substances.” A household product that is not intended for children but which creates a risk of injury due to lead content requires precautionary labeling under the Act. Toys and other products that are intended for use by children and which bear or contain a hazardous amount of lead that is accessible to children for ingestion are banned hazardous substances.<sup>146</sup> The Consumer Product Safety Commission evaluates the potential hazard associated with products that contain lead on a case-by-case basis. The Commission considers a number of factors including the total amount of lead contained in a product, the bioavailability of the lead, the accessibility of the lead to children, the age and foreseeable behaviour of the children exposed to the product, the foreseeable duration of the exposure, and the marketing, patterns of use, and life cycle of the product.<sup>147</sup>

On January 15, 1998, the Commission issued a guidance to the manufacturers of consumer products, requesting that they eliminate the use of all lead that may be accessible to children from products used in or around households, schools, or in recreation and not just restrict themselves to avoiding hazardous quantities. The Commission further recommended that importers, distributors and retailers obtain assurances from manufacturers that products do not contain lead that may be accessible to children, before purchasing such products. The Guidance is non-binding and is meant to supplement the *Hazardous Substances Act*.<sup>148</sup>

The retreat from regulatory approaches to controlling toxic substances, even those as well understood as lead, has been a consistent pattern throughout the 1990s with the increasing expansion of economic globalization. However, if Health Canada does in fact implement the strategy outlined in Dr. Subramanian’s speech, it would represent a significant and important change in the regulation of toxic substances. It would be a technology-forcing measure in that it could only be achieved by removing lead from the production process. The toy industry is apparently opposed to the proposals.

Upon becoming aware of Dr. Subramanian’s speech in early June of 1999, Greenpeace and other children’s health organizations jumped on it lauding the new approach as a great step forward. However, news headlines stating “Tough limits on lead help our children: Ottawa to take ‘strong stand’ on kid’s products”<sup>149</sup> may have served the purpose of legitimizing more delay by giving the false impression that Health Canada was taking strong regulatory action. To the contrary, yet another source of lead appeared in late 1999 (the lead in candle wicks noted above) and Health Canada, as of mid-March 2000, has yet to issue even an advisory on these candles or if it has there is nothing on this matter posted to the Health

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<sup>144</sup> 15 U.S.C. ss.1261-1278 (1998) (Cornell Law, <http://www.law.cornell.edu/uscode/15/1261.html>).

<sup>145</sup> Meaning any substance that has the capacity to produce personal injury or illness to man through ingestion, inhalation or absorption through any body surface. 15 U.S.C. s.1261 (1998) (Cornell Law, <http://www.law.cornell.edu/uscode/15/1261.html>).

<sup>146</sup> 15 U.S.C. s.1261 (1998) (Cornell Law, <http://www.law.cornell.edu/uscode/15/1261.html>); and Notice of Approval of Guidance Documents on Lead in Consumer Products, 63 Fed. Reg. 3310 (1998) [hereinafter *Guidance Documents on Lead*].

<sup>147</sup> Ibid.

<sup>148</sup> Ibid.

<sup>149</sup> “Tough limits on lead help our children: Ottawa to take ‘strong stand’ on kid’s products.” *Toronto Star*, May 14, 1999.

Canada website. More recently, Health Canada officials state that the Lead Reduction Strategy is delayed because it has been expanded to a broader range of products. This expansion has required more work due to the need to justify regulatory limits with “sound science.”<sup>150</sup> It remains to be seen whether the Canadian toy industry will continue to oppose regulations limiting the amount of lead to which children can be exposed. It also remains to be seen whether the proposal outlined in Dr. Subramanian’s speech will pass muster with other government departments concerned with ensuring that regulations in Canada do not create “non-tariff barriers to trade.” The regulatory proposal has yet to be reviewed by the Department of Foreign Affairs and International Trade but Health Canada does not expect such a review to influence regulatory actions that it intends to take under the *Hazardous Products Act*.<sup>151</sup> This optimism may be misplaced if the past is an indication of the future since tough action on lead at the international level has been opposed and weakened in the past by the actions of Canadian negotiators.

#### **8.4.7 The OECD Declaration of Risk Reduction for Lead**

Governments and regulatory agencies in various countries, including Canada, have responded in various ways to the recent discoveries as well as to ongoing lead hazards. Internationally, efforts aimed at controlling the risks associated with lead exposure have included a 1995 Organisation for Economic Co-operation and Development (OECD) agreement. Proposed by the U.S. and the European Commission and backed by a majority of OECD members, it called for a phase-out of lead from gasoline, the virtual elimination of lead in products intended for children, an end to the use of lead solder in food and drink cans and reduced exposure to lead in paint, ceramics and crystalware. The agreement, which would have done much to reduce lead exposure, was blocked by Canada and Australia, which favoured voluntary industry actions.<sup>152</sup> The following year the OECD Declaration of Risk Reduction for Lead was adopted by the 26 OECD Environment Ministers, as well as the Environment Commissioner of the European Community. It commits signatory countries to strengthen their efforts to reduce the risks associated with exposure to all major sources of lead. The apparent difference between this agreement and its doomed predecessor is the new qualifier that states ...reduce lead exposure when they deem it to be *appropriate* (emphasis added) to do so through:

- (i) phasing down the use of lead in gasoline;
- (ii) eliminating exposure of children to lead in toys and other products with which they may come in contact;
- (iii) phasing-down the use of lead in paint and rust proofing agents;
- (iv) eliminating human exposure to lead from food and beverage containers;
- (v) restricting use of lead shot in wetlands; and
- (vi) other actions which address risk of exposure for water, air and the workplace.<sup>153</sup>

The inclusion of the subjective test of “appropriateness” greatly diminishes the force of the agreement, permitting signatories to follow a lead reduction path and timetable of their own choosing.

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<sup>150</sup> Personal communication, Jonathan Williams, Product Safety Bureau, Health Canada, March 27, 2000.

<sup>151</sup> Ibid.

<sup>152</sup> Pearce, F. Lead trickles through European loophole...while industry blocks international ban, *New Scientist*. (July 15, 1995)

<sup>153</sup> Intergovernmental Forum on Chemical Safety. [Proceedings] of Forum II: Second Session of the Intergovernmental Forum on Chemical Safety. Thematic Session on Partnership: Lead Risk Reduction. (February, 1997).

### 8.4.8 Blood-Lead Testing and Follow-Up

#### 8.4.8.1 Approaches in the United States

With the decision by the U.S. Centers for Disease Control (CDC) in 1991 to lower the “intervention level” to 10  $\mu\text{g}/\text{dL}$ , the U.S. also embarked on a “Strategic Plan for the Elimination of Childhood Lead Poisoning” which included, among other things, a move to phase-in mass screening for blood-lead levels in all children beginning with those at highest risk. Emphasis also shifted and expanded from the historical contamination of soil and dust by leaded gasoline to include preventing exposure to lead in paint in older housing - the latter still being the major cause of high-dose lead poisoning in the U.S.<sup>154</sup>

The 1991 Statement by the CDC estimated that about 15% of U.S. children under the age of six had blood-lead levels above 10  $\mu\text{g}/\text{dL}$  (which translated into about 250,000 children). In fact, when the NHANES-III<sup>155</sup> data for 1988-91 were published, the numbers were higher: about 1.7 million children were still over 10  $\mu\text{g}/\text{dL}$ .<sup>156</sup> However, the overall average had dropped dramatically from the late 1970s. The NHANES-II data had revealed an average blood-lead level in children (in the late 1970s) of 12.8  $\mu\text{g}/\text{dL}$  while the NHANES-III average was 2.8  $\mu\text{g}/\text{dL}$ . Exposure remained the most serious for low income, black, male, inner city children.

*Blood-lead levels were consistently higher for younger children than for older children, for older adults than younger adults, for males than for females, for blacks than for whites, and for central-city residents than for non-central-city residents. Other correlates of higher blood-lead level included low income, low educational attainment, and residence in the Northeast region of the United States.*<sup>157</sup>

It stands to reason that the U.S. experience with childhood lead poisoning is worse than the experience in Canada for two key reasons. First, there were far more cars using leaded gasoline. When the use of leaded gasoline reached a maximum in the U.S. in the early 1970s, worldwide automotive emissions of lead to the environment stood at 350,000 tons. Over 270,000 tons of that total was emitted in the U.S.<sup>158</sup> As previously noted, this historical lead burden, mostly in inner city soil, represents a huge reservoir of lead which will continue to be an exposure source for decades into the future. Second, there are far more homes and especially tenement apartment buildings in the U.S. where lead paint has deteriorated and continues to deteriorate due to both more extreme and more widespread levels of poverty. For both of these reasons, the choice in the U.S. to conduct mass screening for blood-lead levels combined with detailed follow-up protocols is clearly necessary. The screening and follow-up protocols (including mandatory reporting of high blood-lead levels to public health agencies) have provided the additional and unexpected result of identifying new exposure sources in various consumer products.

<sup>154</sup> Centers for Disease Control. *Preventing Lead Poisoning in Young Children. A Statement by the Centers for Disease Control*, U.S. Department of Health and Human Services, Public Health Service. (October, 1991)

<sup>155</sup> NHANES stands for the National Health and Nutrition Examination Survey, conducted three times in the U.S. since the 1970s. The NHANES-II survey is discussed above in Section 8.4.1.1.

<sup>156</sup> Pirkle, J.L., *et.al.*, The decline in blood-lead levels in the United States. *Journal of the Amer. Med. Assoc.* 272(4)(1994), pp. 294-291.

<sup>157</sup> Brody, D.J., *et.al.*, Blood-lead levels in the U.S. population, *Journal of the Amer. Med. Assoc.* 272(4) (1994), pp. 277-282.

<sup>158</sup> Nriagu, J.O. Clair Patterson and Robert Kehoe’s Paradigm of “Show Me the Data” on Environmental Lead Poisoning, *Environmental Research* (Section A, 78)(1998), pp. 71-78.

### 8.4.8.2 Canadian Comparisons

The Canadian situation with respect to these two main sources (gasoline and paint) is worth comparing to the U.S. experience. Total automotive lead emissions in Canada were obviously far lower. But the pattern of urban concentration and population distribution in Canada is mainly a linear expanse along the Canada-U.S. border. During the 1980s, urban lead exposure and children's blood-lead levels in Canada were comparable to the U.S. situation and the need to eliminate lead from gasoline was just as urgent. Lead in paint has, historically, been a different situation in the two countries. In Canada, there appears to have not been as serious a problem (although this statement should be qualified since there has been very little testing for lead paint contamination and it is possible that pediatric diagnosis and follow-up of elevated blood lead levels from lead in paint could have been missed - as discussed further below). Since the paint used during the 20th Century (up until at least the 1960s) has been equally loaded with lead, the difference between the U.S. and Canada can reasonably be attributed to two general factors: the much lower prevalence of old, tenement-style housing in Canada and the social safety net that has ensured much lower levels of overall, and especially childhood poverty in Canada.

Three important factors may change this situation for Canadian children. First, Canada's social safety net has been considerably weakened in recent years, and child poverty has risen dramatically. With increased poverty in children living in older dwellings we can reasonably expect both increased deterioration of lead-bearing paint combined with sub-optimal nutrition. The latter is well known to increase the absorption of lead to which children are exposed. Second, estimates of the number of homes with lead-bearing paint are extremely high: over two million homes in Ontario alone. (While deteriorating paint is a serious problem in the context of increasing poverty, it is of course also an issue for any household where renovations and/or paint removal is conducted.) Third, as discussed in Section 8.4.6.3, Canadian regulation of lead in paint is woefully out of date. The allowable level of lead in paint is eight times higher than in the U.S. (although the industry says that it voluntarily follows the U.S. standard) and more important, the Canadian regulation contains dangerous and irrational loopholes such that it places no limit whatsoever on the lead content of exterior paints or on paints used on the interior and exterior surfaces of commercial buildings or on furniture used in such buildings.

As discussed in Section 8.2.2, blood-lead surveys in Canadian children have been relatively limited. The Ontario blood-lead survey of 1984, combined with other community-specific surveys across the country, and the joint federal-provincial review conducted in 1994, confirmed that during the 1980s, Canadian children were, on average, similarly exposed to lead as U.S. children and that blood-lead levels dropped substantially in a lock-step fashion with the phase-down and phase-out of lead from gasoline. Incidences of lead poisoning from lead paint or the more recent exposures from consumer products have been extremely rare in Canada. However, a recent study investigating records from the 1980s of Montreal children with high blood-lead levels reveals some disturbing findings.

The study<sup>159</sup> traced medical records of children with high blood-lead levels in Montreal during the 1980s. With no central reporting or coordination of such data, the study team sought out laboratory records of blood-lead tests. Given the limited means of obtaining data, these investigators suspected that the small sample they studied likely represented a small fraction of all Montreal children exposed to lead during the time period of the study. Moreover, since the signs and symptoms of lead effects are subtle and non-specific, the study team assumed that clinical suspicion alone would likely have led to a limited use of blood-lead testing by clinicians. For the twelve cases reviewed, *pica* and iron deficiency were the most

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<sup>159</sup> Valiquette, L. and T. Kosatsky. Portrait of Montreal Children with High Blood Lead Levels Identified Through Community-wide Review of Laboratory Records, *Chronic Diseases in Canada*. 16(2) (1995) [www.hc-sc.gc.ca/main/lcdc/web/publicat/cdic/cdic162/cd162a\\_e/htm](http://www.hc-sc.gc.ca/main/lcdc/web/publicat/cdic/cdic162/cd162a_e/htm)

frequently cited pre-disposing factors to findings of high blood-lead levels, (at the time, “high” was a level above 25  $\mu\text{g}/\text{dL}$ ), with paint being the main exposure source identified. The study assessed the management of pediatric lead poisoning by the local health care system and public health authorities.

Key findings included evidence of deficiencies in individual and community follow-up of cases of children with lead poisoning, and deficiencies in offering potential solutions. In five of twelve cases, there was no evidence that blood-lead levels were followed up (including a child with a level of 94  $\mu\text{g}/\text{dL}$ ). In four of the twelve cases, there was no indication that exposure sources had been evaluated or eliminated. This follow-up may have occurred but there is no evidence that it was done, including no evidence of assessing risk to siblings, an important step in the management of childhood lead poisoning. The study concluded that the collaboration of clinicians is crucial for reporting and individual case follow-up and that exchange between all groups concerned (laboratories, clinicians and public health authorities) would improve the medical and environmental management of identified cases.

### 8.4.8.3 Pediatric Management of Lead Toxicity in Canada

Given the continuing exposure of Canadian children to lead from a diversity of sources, the inadequate regulatory responses by the federal government, and the very small safety margin between average blood-lead levels and the level where health effects are known to begin,<sup>160</sup> it would seem prudent to heed the findings of this Montreal study. While it does not appear necessary to follow the U.S. strategy of mass screening, various aspects of the pediatric management of lead exposure and toxicity are worth addressing. First, it seems necessary to ensure that pediatricians and family doctors are aware of the minimal safety margin for all children and the fact that historical environmental contamination will work against any change in this small safety margin for many years and perhaps decades into the future. The steady stream of new and unregulated sources increases the need for greater pediatric awareness of exposure sources. Second, the subtle and non-specific nature of the health effects should be part of pediatric training. Since obvious clinical symptoms do not arise until blood-lead levels are well above the onset of neuropsychological effects, doctors also need to be aware of the diversity of exposure sources and factors that pre-dispose children to greater exposure including inner city location, older housing, poverty, etc. Third, greater coordination among doctors and public health agencies would be useful including mandatory reporting to public health agencies of blood-lead findings above 10  $\mu\text{g}/\text{dL}$  and mandatory follow-up to investigate and eliminate exposure sources, ensure sibling follow-up, etc. Fourth, preventive approaches could include the mandatory distribution of educational materials to all pregnant and new mothers about the dangers of lead, the range of exposure sources and the means of avoiding exposure including both physical avoidance and optimal nutrition to reduce absorption of lead that is ingested.

## 8.5 CONCLUSIONS AND LESSONS LEARNED

The regulation of lead has been, and continues to be, a protracted and reactive approach.

Multiple sources and exposure pathways continue to exist including the large reservoir of lead in urban and roadside soil and dust from 60 years of leaded gasoline use and the ongoing, unregulated and often very dangerous discoveries of lead in mostly imported consumer products. Numerous risk factors make children highly susceptible to the dangers of lead and risk factors for poor children are increasing

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<sup>160</sup> Including recognition that there may be no threshold below which lead does not begin to harm a child’s developing nervous system.



alongside increasing rates of child poverty.

However, the control of lead is, on the one hand, a success story. Through a combination of both regulation and voluntary action by industry, children's lead exposure has dropped dramatically. On the other hand, appropriate action was delayed until health-effect levels of exposure occurred in huge numbers of children. For a persistent toxin and with new exposure sources continuing to arise, there remains a very small safety margin for all children. The history of standard setting for lead illustrates many of the central problems with risk assessment and risk management for regulating toxic substances.

Early warnings in the 1920s about the potential danger of lead in gasoline could not be proven. In the absence of proof of harm, safety was assumed. The need for more long-term study was agreed, except that the lead industry maintained almost exclusive control of further research for the next forty years. When independent scientists began to question industry-funded or generated science of both the environmental fate and human health effects of lead and to call for regulation, the lead industry objected. For twenty more years, regulatory action was consistently delayed in the face of scientific uncertainty as to exposure and health effects and lead became one of the most extensively studied pollutants in the world.

This huge body of scientific literature now provides powerful evidence of the causal relationship between lead and adverse neuropsychological effects in children. A key finding, verified in two powerful meta-analyses is that an increase in blood-lead levels from 10  $\mu\text{g}/\text{dL}$  to 20  $\mu\text{g}/\text{dL}$  results in an IQ deficit of approximately 2 points.

In summary, very low levels of lead can cause adverse neurological and neurobehavioural effects in young children. Numerous studies have revealed a wide variety of measured and observed effects at blood-lead levels of 10 to 15  $\mu\text{g}/\text{dL}$ . Some effects appear below 10  $\mu\text{g}/\text{dL}$  with no apparent threshold. Effects include:

- deficits in IQ or deficits in comparable/age appropriate tests of intellectual functioning;
- deficits in speech and language processing;
- deficits in perceptual-motor function and integration;
- deficits in reaction time;
- reduced attention span;
- non-adaptive classroom behaviour;
- deficits in reading, spelling and mathematics scores;
- poorer handwriting;
- significant increase in the risk for learning disabilities, as measured by the need for remedial education in reading, speech and math;
- sevenfold increased risk of failure to complete high school;
- sixfold increased risk for reading disability;
- poorer vocabulary and grammatical reasoning scores;
- poorer hand-eye coordination; and
- increased risk for antisocial and delinquent behaviour with the effects following a developmental course.

The effects of lead also seem to be more serious in boys than in girls. However, despite all that is currently understood about the effects of lead, we remain limited in a clear diagnosis of lead toxicity at low exposure levels. Effects are variable and do not have a consistent behavioural signature. On a population basis, experts postulate that there is probably an overall downward shift in intelligence. On the basis of blood-lead levels prevalent in the U.S. in 1990, (slightly higher but comparable to Canadian

levels), they conclude the possible consequence that lead exposure may prevent about 5% of the population from achieving truly superior function and at the other end of the scale, intellectual damage may be occurring in many more children than would otherwise occur without exposure to lead.

It took a long time, an enormous amount of money and a lot of acute and chronic lead poisoning of children to reach the above conclusions. Although early animal studies and preliminary studies of lead-exposed children raised alarm about the neuropsychological toxicity of low-level lead exposure, the lead industry insisted on proof of harm. To a large degree, regulatory agencies did the same, particularly in Canada, by applying a regulatory framework founded upon risk assessment and risk management. Hence, global lead contamination via automobile emissions, industrial point source emissions, poisonings from lead in paint (and many other sources) enabled the above conclusions as to health impacts to be verified by assessing actual effects in huge numbers of children.

The initial round of regulatory actions in the 1960s and 70s followed the typical chemical-by-chemical approach. Indeed, for most chemicals, including lead, the subdivision went further and regulatory controls have been placed on lead in occupational settings, paint, gasoline, air, soil, drinking water, consumer products and other areas. All of these limits quickly became obsolete as scientists continued to reveal health effects at lower and lower levels of exposure. However, the science was highly complex particularly in the two areas where uncertainty is always a problem in risk assessment, i.e., in assessments of exposure and dose-response relationships. Early findings were rarely definitive although the majority of findings tended in the direction of adverse effects if they did not show them conclusively. Industry-funded research rarely found health effects.

Decisions during the 1980s as to whether regulatory limits should be further reduced were mired in this scientific debate with the lead industry insisting on the innocence of its products (or “innocence” at debatably high exposure levels). The regulatory debates were (and continue to be) typical of those for many pollutants with industry insisting that the highest possible standard of scientific proof be applied to the assessment of hazardous pollutants or products. Meanwhile, average blood-lead levels in children remained near the health effect level (at or above 10  $\mu\text{g}/\text{dL}$ ). By the time the Canadian government finally agreed (in 1988) to phase-out lead from gasoline, (overwhelmingly the largest source of environmental lead contamination), it was estimated that nearly half a million children had blood-lead levels at or above 10  $\mu\text{g}/\text{dL}$ . Other estimates were lower at 60,000 to 100,000 children. For two decades Canadian limits on lead in gasoline had consistently been among the highest in the industrialized world. To finally change this lax regulatory attitude took a combination of extensive public pressure and media exposure, the publication of yet another authoritative compendium of the hazards of lead exposure, and pre-election sensitivity. If the above combination of factors had not been the case, it is extremely unlikely the federal government would have advanced its go-slow approach to gasoline lead phase-down and phase-out.

The approach of bowing to industry pressure and waiting for definitive proof of harm before taking regulatory action has not been limited to lead in gasoline. Canadian regulations for lead in paint are 24 years behind those in the United States. The U.S. standard of 600 parts per million applies to all interior and exterior paints. In Canada, although industry says it has voluntarily moved to the U.S. regulation, the legal limit remains eight times higher, at 5000 parts per million, and does not apply to exterior paint. Similar half-measures have been applied to controlling lead in drinking water. While the U.S. banned lead solder, the Ontario *Building Code* only bans its use for incoming water pipes. The federal government’s regulation of lead in food was and remains an utterly meaningless exercise.

New and unexpected sources of lead continue to arise. Since 1994 dangerously high lead levels have been discovered in crayons imported from China, plastic mini-blinds, and children’s toys, clothing and accessories. With increasing globalization and deregulation in the 1990s, regulatory actions in many countries, including the U.S. and Canada, have been even slower to materialize. Instead, in Canada they

have been replaced with voluntary codes of conduct (paint) or simply consumer warnings (mini-blinds). After-the-fact product recalls (as occurred with the crayons imported from China), are within the legislative mandate of the Consumer Product Safety Commission in the U.S. but in Canada, the *Hazardous Products Act* does not provide for the authority to require product recalls.

However, if previous statements are to be believed, if international trade obligations do not trump attempts at domestic regulation, and if the toy industry does not continue to oppose regulating lead out of its products, Canada may be poised to implement a regulation under the *Hazardous Products Act* concerning lead in children's products that would make Canada a world leader in the control of lead in children's products. This regulation is not likely to be in place until at least 2001.

Average blood-lead levels in Canadian children have dropped steadily since the early 1980s in parallel with the increased use of unleaded gasoline and the stepwise reduction of the lead content of leaded gasoline. However, a joint federal-provincial study in 1994 cautiously estimated that over 66,000 Canadian children still had blood-lead levels in the range known to cause health effects (i.e., above 10  $\mu\text{g/dL}$ ). This number is no doubt lower now but the evidence is clear that global contamination has created average blood-lead levels in children (in both urban and remote areas) that are close to the health effect level. Little to no safety margin exists for all children, making new and unexpected sources of lead of significant concern. Again, a central flaw in risk assessment and risk management is illustrated by the current situation, i.e., the inability to ensure preventative or precautionary levels of exposure to toxic substances when powerful vested interests can consistently and repeatedly undermine preventative regulatory action. The pediatric detection and management of lead exposure and toxicity should therefore remain an issue of concern and greater awareness.

The lessons learned from lead include the following:

1. Regulatory action on dangerous substances must include a precautionary and preventative approach. The regulatory history and ongoing approach to the regulation of lead shows that this lesson has not been learned. The phase-out of highly dangerous substances has rarely been required. For lead in gasoline, phase-out occurred only after reaching health effect levels in huge numbers of children.
2. Independent research into pollutants is essential - both the agenda for research and the actual conduct of the work. When industry has exclusive control of the research into its own pollutants, the results cannot be considered independent.
3. Lack of proof of harm must not be considered proof of safety. Given the inherent complexity and inevitable uncertainty surrounding the investigation of the effect of toxic chemicals, the scientific standard of proof is far too high. For lead in gasoline, insistence on this standard of proof amounted to an approach of delaying regulatory action while conducting an enormous, uncontrolled experiment on children.
4. The insistence on "sound science" to set regulatory standards and in particular the insistence on solid exposure data and dose-response information sets up a "Catch-22" situation. The paradox is evident in the history of the scientific inquiry and regulation of lead. Ontario's multi-media standards for lead are an example. In the early 1990s, this multi-media risk assessment and risk management exercise was able to establish very credible, scientifically defensible regulatory standards given the extensive scientific information available about lead exposure and dose-response relationships. However, given the high baseline exposure created by historical circumstances of widespread environmental lead contamination, the standards were set at levels that provided for almost no safety margin between actual exposure levels and the levels permitted in the standards. For most pollutants however, levels of environmental contamination are much lower, as was the case with lead in the 1920s. For other pollutants, scientific

uncertainty about both exposure and dose-response relationships is much higher, again as was the case for lead in the 1960s and 70s. Ontario's multi-media standards were so scientifically credible because of the huge battle that occurred over the need to regulate and the fact that 30 years worth of "sound science" created a body of evidence demonstrating irrefutable harm. With plenty of data on lead, the situation was created whereby regulators could come up with scientifically defensible standards that provide almost no safety margin. Where data are limited, i.e., in the majority of cases, regulatory limits will continue to face opposition. This "Catch-22" situation is created by regulatory regimes that do not require precautionary or preventative approaches including wide margins of safety. Essentially, the lead industry and others opposing a more preventative and precautionary approach oppose the use of prevention or the application of safety margins as "unsound science." The result is uncontrolled experimentation on children.

5. Regulation by Health Canada of lead in consumer products is a sorry tale and it remains a hollow effort although stated commitments hold considerable promise. However, at present, the public cannot assume that products offered for sale have been tested for hazards like lead or that related regulatory controls are in place to ensure children's safety.

## 8.6 RECOMMENDATIONS

1. There is a need for routine provision of audience-appropriate educational materials about lead to health care professionals, social workers, teachers, parents, caregivers of children, women of child-bearing age and pregnant women. Such educational materials need to provide information about the multiple exposure sources and pathways (historical and current), the multiple risk factors for children, the health effects of low-level lead exposure, the means of avoiding exposure, and nutritional factors that can reduce uptake of lead.

2. There should be ongoing education of clinical health professionals, including family physicians, pediatricians, nurse practitioners and midwives, regarding clinical issues of low level lead exposure including taking an exposure history to detect sources of exposure and health effects.

3. All risk assessments conducted by Health Canada for consumer products should be subject to rigorous external peer review.

4. Health Canada should immediately adopt the lead in paint standard of 600 parts per million adopted in the United States 24 years ago. This regulation must be applied to all paints.

5. The *Hazardous Products Act* requires amendment to provide for the power to recall products. It also requires amendment to eliminate all reference in the Act or its regulations to the dubiously useful and unsupportable notion of allowing hazardous or toxic exposure so long as it does not occur in areas "frequented by children."

6. Health Canada's stated commitment to regulate the lead content of consumer products such that there be no intentional addition of lead to children's products is long-overdue and should be implemented immediately.

7. As part of developing a Materials Use Policy that incorporates a precautionary and preventative approach to avoiding the use of persistent pollutants, Health Canada should mandate the phase-down and phase-out of lead in all consumer products with the exception of a very few controlled and currently non-replaceable uses such as X-ray shielding and lead-acid batteries.

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