

# Learning disabilities and the environment: What we know – and how our policies are failing children

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Learning disabilities and related attention deficit disorders affect an estimated 10% to 15% of children. The consequences of these and other neurological, developmental and behavioural disorders are life-long, often serious for both the child and his or her family, and costly for society. The role of toxic chemicals in the etiologies of these disorders has been largely ignored, although the evidence from both experimental animal and clinical research from the few neurotoxic chemicals that have been studied to date is compelling (1), and the possibilities for prevention are enormous.

An example of the costs of subtle deficits due to exposure to lead was demonstrated in a groundbreaking economic benefits analysis by Schwartz (1994) (2) based on calculations of the costs of lead-related reductions in intelligence quotient on years of schooling and earnings, and cardiovascular effects. The societal benefits of reducing blood lead concentrations in the population by just 1 µg/dL were estimated at \$17.2 billion/year to the American economy. Schwartz (2) noted that these benefit estimates are low, as other known effects of lead – on behaviour, attention, hearing, balance and reduced stature – have not been assigned a monetary value (2). This benefit was revised upward in a subsequent economic analysis (3), based primarily on labour market changes and more recent data on the relationship of intelligence quotient with educational attainment and projected earnings gains.

Worldwide, there is growing attention to the differential vulnerability of children to environmental toxicants. Since the mid 1990s, increasing concern, legislation and policy initiatives in the United States, and a joint declaration (4) have brought children's health and development into the forefront of the environmental agenda. Canada signed the 1997 Declaration of the Environmental Leaders of the Eight on Children's Environmental

Health that pledged action on the following issues: risk assessment and standard-setting that take into account the specific exposure pathways and dose-response characteristics of children; children's exposures to lead; clean water and water standards; air quality (including environmental tobacco smoke); and emerging threats to children's health from endocrine-disrupting chemicals (such as polychlorinated biphenyls and dioxins that have been shown to have neurotoxic effects, and to alter thyroid function). Thyroid hormone is critical to most processes involved in brain development – regulating neurite outgrowth, cellular migration, synaptogenesis, myelogenesis and the development of major neurotransmitter systems (5). Despite the above pledges, the effects of toxic exposures on child health and development are receiving little attention in Canada in research or by other federal programs investigating the determinants of health and development. In addition, there are gaps in regulatory programs and policies that need to be revised to protect children.

By contrast, a 1997 executive order from the White House (6) acknowledged that children may suffer disproportionately from environmental health risks, and directed all American federal regulatory agencies to ensure that their policies, programs and standards address these risks. The executive order also established a high level interagency task force to recommend federal strategies and research. The above actions have generated a number of new initiatives in the United States: eight centres for children's environmental health and disease prevention research, and announced this year, an additional four more centres on neurodevelopmental effects; a new Office of Children's Health Protection at the Environmental Protection Agency (EPA); and a major proposed study, A Prospective Longitudinal Study of

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Environmental Effects on Children's Development, that will involve thousands of pregnancies from American intake sites.

The need for new approaches to government standard-setting and premarket safety evaluations to protect children was addressed in a five-year United States National Academy of Sciences (NAS) report, *Pesticides in the Diets of Infants and Children* (1993) (7). Among other findings, the report stated: "The data strongly suggest that exposure to neurotoxic compounds at levels believed to be safe for adults could result in permanent loss of brain function if it occurred during the prenatal and early childhood period of brain development". Many toxic agents are known to damage the developing, and unprotected, brain by interfering with those processes undergoing development at the time of the exposure (Rodier, 1995) (8). It is clear that even subtle structural or neurochemical defects can nonetheless have devastating functional consequences.

### TOXICITY TESTING

The NAS report made several recommendations for changes in risk assessments and standard-setting to protect children. To assess risk, regulators need adequate toxicity data to establish a No Adverse Effect Level (NOAEL) and adequate exposure data that takes into account both aggregate exposures (from all sources of the chemical, eg, water, food, carpets) and cumulative exposures (from several chemicals with a similar mode of action). The NAS Committee stated that the need for developmental neurotoxicity testing, which is not a core data requirement for pesticides, was of particular importance, as is the need to assess the potential for toxicity to the developing immune and reproductive systems. In assessing risks to children, the NAS recommended that an additional uncertainty factor be applied to the animal data to take into account toxicity and/or exposure data gaps. This action was mandated by Congress in the 1996 United States Food Quality Protection Act, requiring reassessments of pesticides. New EPA requirements for neurodevelopmental data have led to some new regulations and bans on the major uses of two common pesticides.

In 1989, The Learning Disabilities Association of Canada (LDAC) and LDA America adopted resolutions titled, "The Need for Federally Mandated Developmental Neurotoxicity Testing to Protect Human Health: Central Nervous System Development" ([www.ldac-taac.ca](http://www.ldac-taac.ca)). Canada's Minister of Health assured LDAC in 1990 that new guidelines would be issued in that year, which has not happened. However, the Pest Management Regulatory Agency is harmonizing its re-evaluation process with the USEPA and requiring developmental neurotoxicity testing for two classes of pesticides that act on the nervous systems of pests. However, this leaves risk assessments for other pesticides, food additives and colours, drugs, cosmetics, and high volume neurotoxic chemicals without these data. For example, an organic form of a known neurotoxi-

cant, manganese, methylcyclopentadienyl manganese tricarbonyl (MMT), was approved, and reassessed as being safe for use in Canadian gasoline without developmental neurotoxicity data. Manganese exposure produces effects on neurotransmitter systems in developing animals, but not in adult animals (9), and in humans, manganese toxicity produces neuropsychiatric disorders and symptoms similar to Parkinson's disease (10). In the United States, the USEPA refused Ethyl Corporation's petition to market MMT, based on unresolved health concerns; however, this was overturned in a narrow court decision that found that EPA could not ban a fuel additive based on health effects alone under the Clean Air Act.

### EXPOSURE

Because children are smaller, they receive a more concentrated dose of a toxicant than adults. The fetus and the infant have immature detoxification systems, and the blood-brain barrier is not yet formed. Children also consume more of fewer foods, so a child might receive a higher exposure to a chemical contained on or in a favourite food during many meals every day. They play and breathe closer to the floor where contaminants accumulate in air and dust. Compared with adults, children consume more food and water, breathe more air on a mg/kg body weight basis and tend to absorb more toxicants.

The NAS report found that infants would consume up to seven times the amount of water on a mg/kg body weight basis than that consumed by adults. Water can be a source of exposure to toxicants for children, especially to children living in areas where groundwater is contaminated with pesticide and nitrate runoff. However, in Canada there is no federal legislation that sets enforceable standards for contaminants in drinking water; rather, the federal government, with the provinces, establishes 'guidelines' or nonenforceable limits for these chemicals and regulation is left to the provinces. Unfortunately, no systematic chemical analyses with reporting and enforcement of drinking water guidelines are mandated for populations in Canada by any department, or level of government. In a survey of well water (Ontario, 1989 [unpublished data]), atrazine, a triazine herbicide, was found in one sample at 210 parts per billion (ppb), 40 times the Canadian Maximum Acceptable Concentration (MAC) of 5 ppb. A five-year study of the effects of environmental groundwater concentrations of pesticides (aldicarb and atrazine) and nitrates on the immune, hormonal and nervous systems of mice found effects, replicated many times, in altered thyroid levels, immune system suppression, and increased aggressiveness (11).

Do drinking water guidelines, or MACs, protect children? The rationale for the MAC for each chemical in drinking water is published by Health Canada, and calculated on an adult weight and consumption pattern. For example, consider atrazine with a MAC of 0.005 mg/L or 5 ppb, and recalculate the MAC based on a 7 kg child consuming 1 L/day, instead of a 70 kg adult consuming

1.5 L/day, the MAC would need to be close to an order of magnitude lower at 0.0007 mg/L or 0.7 ppb.

## RISK MANAGEMENT

Research informs both clinical management and regulatory policy. In the 1960s, a blood lead level of 60 µg/dL was considered to be toxic. Since the 1970s, epidemiological research has altered the perception of lead's hazards, lead was banned as a gasoline additive, and the concern for lead toxicity is now 10 µg/dL. There may be no threshold for lead's effects – a recent study by Lanphear et al (12) linked levels as low as 2.5 µg/dL with neurodevelopmental effects.

Unlike mandated programs in the United States, there are virtually no lead screening programs for children at risk in Canada. A study by Valiquette and Kosatsky (13) investigated laboratory records to review care received by children in Montreal in the 1980s with elevated blood lead levels, greater than 25 µg/dL, and found inadequate follow-up, little reporting to public health authorities or management of the source of lead, which in most cases was lead-containing paint (13). There is a voluntary agreement in Canada with the paint industry that should limit lead in paint to the United States 1977 regulated limit of 600 ppm, but Canada's regulation under the Hazardous Products Act still permits more than eight times the 600 ppm limit for indoor paint, with no limit on outdoor paint. LDAC has urged that this regulation be updated for more than 10 years. A strategy to restrict the lead content of consumer products that was supposed to be issued for comment in 1998 is still in limbo. The risk assessment for lead is complete, but the snail's pace approach to regulation shows that there is a need for enhanced risk management in Canada.

## REFERENCES

- Weiss B. Vulnerability of children and the developing brain to neurotoxic hazards. *Environ Health Perspect* 2000;108(Suppl 3):375-81.
- Schwartz J. Societal benefits of reducing lead exposure. *Environ Res* 1994;66:105-124.
- Salkever DS. Updated estimates of earnings benefits from reduced exposure of children to environmental lead. *Environ Res* 1995;70:1-6.
- 1997 Declaration of the Environment Leaders of the Eight on Children's Environmental Health, Miami, Florida, May 6, 1997.
- Porterfield SP. Thyroidal dysfunction and environmental chemicals – Potential impact on brain development. *Environ Health Perspect* 2000;108(Suppl 3):433-7.
- The White House. Protection of Children from Environmental Health Risks and Safety Risks. April 21, 1997.
- National Research Council. Pesticides in the Diets of Infants and Children. Washington: National Academy Press, 1993.
- Rodier PM. The developing brain as a target of toxicity. *Environ Health Perspect* 1995;103(Suppl 6):73-6.
- Shukla GS, Dubey MP, Chandra SV. Manganese-induced biochemical changes in growing versus adult rats. *Arch Environ Contam Toxicol* 1980;9:383-391.
- Frumkin H, Solomon G. Manganese in the U.S. gasoline supply. *Am J Ind Med* 1997;31:107-15.
- Porter WP, Jeager JW, Carlson IH. Endocrine, immune and behavioral effects of aldicarb (carbamate), atrazine, (triazine) and nitrate (fertilizer) mixtures at groundwater concentrations. *Toxicol Ind Health* 1999;15:133-50.
- Lanphear BP, Dietrich K, Auginger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 µg/dL in US children and adolescents. *Public Health Rep* 2000;115:521-9.
- Valiquette L, Kosatsky T. Portrait of Montreal children with high blood lead levels identified through community-wide review of laboratory records. *Chronic Dis Can* 1995;Spring 16:66-72.

## RECOMMENDED READING

- National Institutes of Health. Developing brain and environmental/critical windows of exposure in children. *Environ Health Perspect* 2000;108(Suppl 3):373-600. <<http://ehis.niehs.nih.gov/>> (Version current at Dec 2, 2001).
- Greater Boston Physicians for Social Responsibility. In Harm's Way: Toxic Threats to Child Development. May 2000. <<http://www.igc.org/psr/>> (Version current at Dec 2, 2001).
- The American Academy of Pediatrics: Handbook of Pediatric Environmental Health, 2000. <<http://www.aap.org/pubserv/>> (Version current at Dec 2, 2001).
- J Routh Reigart, James R Roberts. Recognition and Management of Pesticide Poisonings, 5th edn. USEPA Office of Pesticide Programs, 1999. <<http://www.epa.gov/oppfead1/safety/healthcare/handbook/handbook.htm>> (Version current at Dec 2, 2001).
- USEPA Office of children's Health Protection – OCHP. <<http://www.epa.gov/children>> (Version current at Dec 2, 2001).
- International Joint Commission. Environmental Health in Family Medicine: six modules based on clinical cases; <<http://www.ijc.org/boards/hptf/modules/content.html>> (Version current at Dec 2, 2001).