Chapter 2: Relationship Between Children's Health and Environmental Contaminants

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2.1 INTRODUCTION

In contrast to adults, children represent a particularly vulnerable and sensitive group because their bodies and physiological systems are still undergoing substantial growth and development. In addition, children are often more exposed to environmental health risks because of their particular behaviours and activity at each developmental stage. They are also characteristically more often involuntarily exposed to environmental chemicals.

It has been only relatively recently that research has focused on better describing the ways in which children might be quantitatively and qualitatively more exposed and more susceptible to the effects from environmental contaminants. This may not be the case for all contaminants, in all circumstances, but we attempt here to describe the broad scope of the problem. While this knowledge is far from being complete, we do recognize that children's unique susceptibility can stem from multiple factors.

This chapter provides an overview of aspects of health effects in children from exposure to environmental contaminants. The summary is presented in several parts: 1) the factors that influence children's susceptibility to environmental contaminants, 2) the developmental time frames during which impacts of contaminants have been observed; 3) physiological and developmental reasons children and infants are most sensitive to health impacts from exposure to environmental contaminants; 4) the specific risk factors and routes of exposure that are unique to children and infants; 5) the known effects of specific categories of contaminants on childhood health and development; and lastly, 6) some child health problems that have been linked to exposure to environmental contaminants. We conclude this chapter by highlighting areas where there are gaps in information on children's environmental health. We also underscore several issues of concern that have been identified here and by others in the field of environmental health.

2.2 FACTORS INFLUENCING CHILDREN'S EXPOSURE, UPTAKE AND SUSCEPTIBILITY TO ENVIRONMENTAL CONTAMINANTS

2.2.1 Greater Exposure

- Canadian children (less than 12 years old) spend more than 70% of their time indoors at home, 10% at school, and 8.5% outdoors.[1] Therefore, not only the home environment, but also the school, and recreational facilities, especially swimming pools and hockey arenas, must be considered as places of exposure to different toxins.

- Children and youths in Canada also spend more of their time outdoors than do adults.[2] They are also typically more active when they are outdoors. Evidence shows that exercise enhances uptake of air

[1] Chance, G. and E. Harmsen. Children are different: Environmental contaminants and Children's Health. *Can J Pub Health.* 89 (*Suppl* 1) (1998),S9-13.; and Leech, J.A., K. Wilby, E. McMullen and K. Laporte. The Canadian Human Activity Pattern Survey: Report of methods and population surveyed. *Chronic Dis. Can.* 17(3/4) (1996), 118-23.

[2] Leech, J.A., *et.al.* 1996, *op.cit.*

pollutants[3] and a child's greater exploratory behaviour also translates to greater exposure to contaminants present in soil.

- There is a vertical gradient in concentrations of some contaminants. For example, pesticides are often found in higher concentrations near the floor, closer to a child's breathing zone and to where they play. The young are closer to the ground (both indoors and outdoors). The infant crawling phase and overall smaller size of infants and toddlers means that they come into contact more frequently with soil, lawns, carpets and floor surfaces, all of which may harbour chemical contaminants.[4]

- Children, especially infants, exhibit significant exploratory behaviour, including greater hand-to-mouth activity, eating of non-food items (pica) and putting items in their mouths, ∴ they are more often directly transferring and ingesting residues from indoor dust, soil and products, such as toys.[5]

- Children and infants have particular food preferences and/or are nourished by certain kinds of foods (e.g. breast milk, fruits and vegetables) that are typically (or potentially) higher in contaminant levels.[6]

- Children are not sufficiently cognitively developed to allow for avoiding environmental exposures of their own accord. That is, they are passively exposed and have no say in their exposures and are dependent on the supervision of adults. For example, even though warning signs may be used when pesticide is applied on lawns or in parks, this is of little effect with young children who can't yet read and/or may not understand the danger or need to avoid exposure.

2.2.2 Greater Uptake

- Because children are smaller, they have a surface area to volume ratio that is about three times that for adults. Therefore, they have greater relative uptake of contaminants by all routes.

- Kilogram for kilogram, a child's body absorbs more contaminants that come into contact with the skin, than does the adult body.

- Children breathe in and out more rapidly than adults. Every minute they exchange more air per kilogram of body weight than adults, ∴ they have relatively higher intake of air and air pollutants. Again, smaller lungs also mean a higher surface area to volume ratio, hence greater relative absorption of contaminants via inhalation.

- Relative to body weight children eat more food and drink more water, ∴they will take in greater

[3] Reiser, Karen. General principles of susceptibility. In: *Environmental Medicine*. Brooks, Stuart M. *et.al.* (eds). (St. Louis: Mosby, 1995), pp. 351-360.

[4] Chance, G. and E. Harmsen, 1998, *op.cit.*; and Fenske, R.A. Differences in exposure potential for adults and children following residential pesticide applications. In: *Similarities & Differences Between Children & Adults: Implications for Risk Assessment*. P.S. Guzelian, C.J. Henry & S.S. Olin (Eds.) Washington: ILSI Press. (1992), pp. 214-25.

[5] Calabrese E.J., E.J. Stanek, R.C. James and S.M. Roberts. Soil ingestion: a concern for acute toxicity in children. *Environmental Health Perspectives*. 105 (1997), 1354-8.

[6] National Research Council. *Pesticides in the Diets of Infants and Children*. (Washington: National Academy Press, 1993.)

relative amounts of contaminants or residues present in their foodstuffs and from water sources.[7]

2.2.3 Specific System/Organ Susceptibility

- *In utero* exposure of the fetus to environmental contaminants, many of which are able to cross the placenta, may alter the course of development, even at very subtle exposure levels. The prenatal stages of development of major organs, body structures, nervous and reproductive systems, represent especially sensitive times.

- Postnatally, certain organ systems and body structures are also more vulnerable to effects from exposure because they undergo continued differentiation before adulthood.

- The brain, nervous system and lungs undergo extensive growth after birth and are particularly sensitive throughout much of childhood as a result. At the same time, the fact that several other body systems are immature in the infant and child, renders them particularly ill-equipped to handle toxic contaminants. The digestive tract and skin are extremely permeable and the developing lungs present large surface areas through which chemicals may be easily absorbed. The physiological mechanisms that normally help protect the body from chemicals that do invade it, such as the immune, excretory and de-toxifying systems are also underdeveloped in the earliest stages of life.

- On the whole, children are typically *more* susceptible to the biological effects of contaminants because of their immature systems and physiology. However, there are instances where the young are less susceptible depending on the particular chemical or substance. The effect of a larger relative volume of extracellular water and the reduced number of binding sites on target cells may effectively reduce the concentration and toxicity of substances in the young.[8] However, researchers are quick to point out that there is no systematic model for predicting when particular chemicals will be more or less toxic to infants and children, especially from adult or animal-derived data alone.

2.3 DEVELOPMENTAL STAGES OF CHILDREN

Growth and development is a complex process. At each stage in a child's developmental path, there are unique biological processes that occur, often accompanied by changing behaviour patterns. As a result, there are specific differences in exposure, absorption, metabolism, target organ or system susceptibility and excretion at each stage. This means that there may be many different adverse health outcomes from toxins depending on the timing of exposure in a child's life. In this section, we briefly outline the stages in human growth and development and the potential for exposure and effects on children at each stage.

The recognized stages of human growth and development, their timing, and the significant biological processes that may be altered by exposure to environmental contaminants are presented below in Table 2.1.

[7] *Ibid.*

[8] *Ibid. p.* 41

Table 2.1 Stages of development in the young, focusing on specific biological processes that increase children's susceptibility to adverse effects from toxic exposures.

STAGE	TIMING	SENSITIVE BIOLOGICAL PROCESSES
Prenatal/In utero → Embryonic → Fetal	= implantation to 8 weeks = 9 weeks to birth	• The **most** vulnerable periods according to different body systems are:[9] CNS, brain - weeks 3 through 5+ Heart - weeks 3.5 through 5.5 Eyes - weeks 3.5 through 10? Limbs - weeks 3.5 through 7 Ears - weeks 3.5 through 10? Teeth - weeks 6 to 12 Palate - weeks 6 through 12 External genitalia - 7 through 15
Infancy → neonatal → postneonatal	= birth to 1st month = 1 to 12 months postnatal	• Brain is 10% of adult volume at birth, ∴ undergoes considerable growth postnatally (until about age ten) • Myelination of nerves (until adolescence) • Rapid skeletal growth
Toddlers	= 1 to 3 years	• Brain growth, myelination • Rapid skeletal growth
Childhood	= 3 to 10 years	• Brain growth, myelination • Steady skeletal growth
Adolescence	= 10 to 18 years	• Maturation of reproductive organs • Adolescent skeletal growth spurt • Cognitive maturation

2.3.1 Pre-conception

Exposures of parents before they reproduce, are an important, yet less direct avenue of effects on their future offspring. We will not deal here with the vast and complex area of research concerning effects on the ability to conceive due to exposure to environmental contaminants. Instead we highlight two areas of pre-conceptional exposures that have more direct implications for children's health. The clearest example concerns the effects from previous maternal exposure to persistent toxins that have been stored but may be mobilized during pregnancy as a result of physiological changes.

Lead is stored in bone. With liberation of maternal calcium to provide for fetal skeletal development, mothers who were exposed to and stored lead throughout life, may transfer lead across the placenta and therefore give birth to infants with congenital lead poisoning.[10] PCBs (and other lipophilic organochlorines) are stored in maternal fat. Similarly, with metabolic changes during pregnancy, PCBs may be liberated from fat stores and cross the placenta thereby reaching the developing embryo and fetus.

[9] Rathus, S.A. *Understanding Child Development*. (Holt, Rinehart & Winston, 1988.)

[10] Bearer, C. Developmental Toxicology. In: *Environmental Medicine*. Brooks, Stuart M. *et.al.* (Eds). (St. Louis: Mosby , 1995), pp. 115-128.

When parents are exposed to environmental contaminants, there is the possibility of damage[11] occurring to their cellular DNA. Genetic mutations will only be inherited by offspring if they occur in the gametes (ova or sperm), since it is the DNA from both mother's and father's gametes that eventually combines to form the genetic material of the offspring. Gametes are most susceptible when they are active. In the case of the male, sperm are normally produced continually[12] from puberty throughout adult life, therefore there is the potential for genotoxic effects to occur if the father is exposed to contaminants in adulthood. Exposures that might affect the sperm's genetic material usually occur within a more limited period of time before conception. The length of time during which a contaminant may produce genetic effects on sperm varies depending on the specific contaminant and how the body handles that substance.[13] The exact nature of the genotoxic effects transmitted via the father is not well understood or studied.[14]

In contrast, women make their lifetime supply of eggs while still a fetus and the eggs are only active, and therefore susceptible, during this time.[15] It is therefore, when the mother is a fetus, (i.e. exposures when the grandmother was pregnant) that could potentially result in her offspring inheriting defective genetic material.[16]

2.3.2 In Utero

The nine months from conception to birth represent the most vulnerable in an individual's existence with respect to environmental exposures. It is during this time that exposure may result in physical abnormalities. Table 2.1 above lists the most vulnerable milestones in prenatal development according to formation times of major organs and body structures. The prenatal period is broadly divided into two stages, embryonic and fetal, which are characterized by differences in pattern and rate of development.

a) Embryonic period (weeks 1 to 8).
The first 8 weeks after fertilization are described as the embryonic period, during which time

[11] These genotoxic effects include mutations in hereditary material (nuclear DNA).

[12] Sperm mature in the human testes over a period of about 72 days. (Rathus, S.A., J.S. Nevid and L. Fichner-Rathus. *Human Sexuality in a World of Diversity.* 3rd Edition. (Boston: Allyn & Bacon, 1997).

[13] For example, solvents are usually cleared rapidly from the body and therefore only exposures immediately prior to conception would result in genotoxic effects on sperm. Genotoxic effects from other contaminants such as radiation and some pesticides appear to be associated with exposures that occur several months or years prior to conception. For example, a study by Gardner and colleagues (1990) attributed cancer risk in children to the occupational exposure of their fathers to radiation. (Gardner, M.J., M.P. Snee, A.J. Hall, C.A. Powell, S. Downes and J.D. Terrell. Results of a case-control study of leukemia and lymphoma among young people near Sellafield nuclear power plant in West Cumbria. *British Medical Journal.* 300 (1990), 429-434.) The banned pesticide dibromochloropropane (DBCP) was found to produce spermatoxicity and testicular tissue damage that affected fertility of these men for many years. (Eaton, M., M. Schenker, D. Whorton, S. Samuels, C. Perkins and J. Overstreet. Seven-year follow-up of workers exposed to 1,2-dibromo-3-chloropropane. *J. Occup Med.* 28 (1986), 1145-1150).

[14] Friedler, Gladys. Developmental toxicology: Male-mediated effects. In: *Occupational and Environmental Reproductive Hazards: A Guide for Clinicians*. Maureen Paul (Ed.) Baltimore: Williams & Wilkins. (1993), pp. 52-59.

[15] The human female is born with about 2 million ova in immature form. Only several hundred thousand of these actually last into puberty and of these, roughly only 400 will ripen and be released by the ovaries during a woman's reproductive years. (Rathus *et.al.* 1997, *op.cit.*)

[16] Bearer, C. Developmental Toxicology. In: *Environmental Medicine*. Brooks, Stuart M. *et.al.* (eds). (St. Louis: Mosby , 1995), pp. 115-128.

morphological changes are most numerous and rapid. The newly formed embryo is usually not susceptible to teratogens[17] in the 1st two weeks of its existence. Greatest susceptibility occurs thereafter however, when the organ systems and body structures are rapidly developing. Therefore *major anatomical abnormalities* can occur in weeks three through seven. This marks the earliest of various "*critical periods*" in development. That is, times where if the developmental processes are interfered with, they may express irreversible changes. For example, the critical period for sexual differentiation begins at six weeks and continues until week twelve. At this point, the embryo's reproductive structures become either male or female depending on the genetic make-up of the individual. If the embryo or fetus is exposed to abnormal levels of hormones during this critical period, there may be permanent effects on reproductive development. The children of women who took Diethylstilbestrol, a synthetic estrogen prescribed to prevent spontaneous abortion, had a higher incidence of abnormalities of their reproductive organs as well as other health problems (e.g. vaginal cancer) that did not manifest until much later in life.

b) Fetal period (weeks 9 through 40).
During the fetal period elaboration of the body structures formed in the embryo occurs, with development of only a few new features.[18] Although the fetus is therefore somewhat less vulnerable after the embryonic period, important *physiological defects* and *minor anatomical abnormalities* may still occur during early fetal development.

Generally, early prenatal or first trimester (weeks 1 to 12) maternal exposures are of greatest concern in their potential to affect fetal *anatomical* development. These are times during which there is spectacular proliferation of cells and marked tissue differentiation. These *critical periods* of cell proliferation and tissue differentiation are most susceptible to alteration by environmental contaminants because they represent times of considerable DNA synthesis. Since many women may not know they are pregnant in the first month or two, there is also a concern that they may inadvertently be exposed at one of the most critical times in their offspring's development.

2.3.3 Infancy

While most of the development of body structures and the fastest rate of growth of the individual occurs prenatally, there is still substantial and rapid growth of body structures that occurs immediately postnatally, especially during the first year of life. Infancy represents the period of life from birth to about 12 months and there are several important differences between infant and adult physiology for this critical period. The first portals of entry for contaminants, namely, lungs, digestive tract and skin form less effective barriers to entry of toxic substances. The skin[19] and gastrointestinal tract[20] of the newborn are particularly permeable and therefore will more readily absorb substances.

Children's lungs are not fully developed at birth, and the surface area of the alveoli for gas exchange

[17] The word teratogen derives from the Greek, teras, meaning "monster". Teratogens are environmental influences or agents that may cause damage to the embryo or fetus.

[18] O'Rahilly, R. and F. Müller. 4.5. Developmental morphology of the embryo and fetus. In: *Cambridge Encyclopedia of Human Growth & Development*. Ulijaszek, S.J. *et.al.* (Eds). (Cambridge: Cambridge University Press, 1998), pp. 161-3.

[19] Bearer (1995, *op.cit.*) provides an example where babies had hexachlorobenzene poisoning (with neurotoxic effects) from skin contact with this compound.

[20] The G.I. tract does have higher pH in the first 8 hours postnatally and this may inhibit absorption of some substances.

grows rapidly from 3 m^2 at birth, to 75 m^2 by adulthood. Bronchiolar branching is complete early in fetal life. But alveolar development and cellular differentiation continues to age 8. Alveoli are ∴sensitive to effects until this age. Lungs grow in size and have a large absorptive surface area allowing for significant absorption of inhaled contaminants.[21]

Other tissues that undergo significant growth postnatally include the immune, brain and nervous systems. In the immune system, thymus[22] tissue decreases, whereas lymphoid tissue increases in the infant and toddler. The brain and nervous system are both especially vulnerable and have a broad window of susceptibility that begins with fetal development and continues into childhood.[23] The blood-brain barrier, which partly protects the adult brain from toxic substances does not fully develop until about six months postnatally. Brain cells continue to be added for two years postnatally and the brain grows larger for several years into childhood.[24] The brain and nervous system are also undergoing differentiation into late childhood. Brain synapse[25] formation reaches a peak in toddlers and myelination[26] of nerves continues to adolescence. The brain's higher cognitive functions don't develop until later in childhood and adolescence. Unlike other body organs, the brain cannot readily repair cells after injury.

Several metabolic systems, (e.g. P450 system and Phase I and II enzyme systems) responsible for altering the chemistry of absorbed contaminants, are of low activity in the fetus and newborn. Renal excretion capacity[27] develops over the first 6 months of life.

2.3.4 Childhood

For simplicity's sake, childhood here includes the phases of toddlers (ages 1 to 3) and older children (4 to puberty) together, as developmental changes do not markedly distinguish these two stages of life. The changes that occur during this phase essentially represent a continuation of the growth of body structures established in infancy. For example, lungs continue alveolar development, nervous system continues myelination, the brain continues to grow and undergo synapse formation, lymphoid tissue increases. Skeletal and muscular growth continues at a steady pace.

Metabolic enzymes are more active and therefore there is somewhat better ability to handle environmental toxins, although the exact mechanisms are not well understood.[28]

Toddlers are more mobile compared to infants and typically exhibit greater hand-to-mouth activity compared to older children. These are important behavioural features which distinguish toddlers from older children. Once children reach school age, they are coming into much greater contact with the external world beyond their homes, and thereby have greater chance of exposure from a variety of

[21] Chance, G. and E. Harmsen. 1998, *op.cit.*

[22] The thymus gland is a small organ of the immune system (found near the sternum) that is the site of maturation and activation of T-cells (an important family of immune cells).

[23] Graeter L.J. and M.E. Mortenson, Kids are different: developmental variability in toxicology. *Toxicology.* 111 (1996), 15-20.

[24] Adult brain size is not achieved until about age ten.

[25] The vital connections between nerve cells.

[26] Myelin is the protective outer coating of nerves, important in nerve signal transmission.

[27] Excretion via the kidneys.

[28] Bearer, C. 1995, *op.cit.*

sources, including schools, day-care, parks, playgrounds and the homes of others. Older children are also more likely to be unsupervised and to participate (often unwittingly) in adventurous or risky behaviour.[29] For example, in 1993 a dozen Hamilton school children between the ages of 9 and 14 found liquid mercury in the laboratory facilities of an abandoned industrial plant.[30] From that small group, over 250 other children were rapidly exposed to the mercury through school chums, the majority of these children having direct contact with the mercury from touching, playing with it, spilling or pouring.

2.3.5 Adolescence

The start of adolescence varies individually, but is triggered by release of pituitary hormones under the influence of signals from the hypothalamus. The most important changes during adolescence concern the development and differentiation of reproductive tissues and structures (e.g. breasts, uterus, vagina, penis, scrotum, testicles) to their mature state, thereby making reproduction possible in late adolescence. There is rapid skeletal and muscular growth[31] in the adolescent as well, as adult body size is achieved.

Sperm production begins in the male, once the testes, prostate and seminal vesicles are sufficiently matured under the influence of pituitary hormones and testosterone (usually about age 13 or 14).

Higher brain functions, such as abstract thought, are achieved during adolescence. Myelination of nerves continues and eventually ceases during adolescence.

In general, tissues that have rapid turnover throughout life, such as blood, skin and sperm, represent vulnerable targets from environmental exposures at *any* stage in pre-adult development and over the lifetime of the individual.

Behaviourally, adolescents also have greater opportunities for exposure through occupational scenarios and are even more likely to be involved in health risk behaviours.[32]

2.3.6 Summary

There are many critical periods in children's development that represent times especially sensitive to adverse health effects from contaminant exposure. For example, the prenatal period, when major organs and body systems are forming, differentiating and growing, represents a particularly vulnerable time in life when the course of development may be altered at what might even be very low exposure levels for the pregnant woman. In early infancy there is also especial vulnerability because the brain, nervous system, lungs, reproductive system, immune system, digestive system and skin are immature and continue to differentiate and become functionally mature for varying periods of time. As a result there are different and overlapping periods of susceptibility depending on the body system under consideration. The brain and nervous system are particularly vulnerable because they have the longest window of development and their cells do not readily repair themselves after damage.

There are also accompanying behavioural changes at every stage of life which influence the exposures of

[29] Millstein, S., C. Irwin, N. Adler *et.al.* Health-risk behaviors and health concerns among young adolescents. *Pediatrics.* 3 (1992), 422-28.

[30] George, Lindsey, *et.al.* The Mercury emergency and Hamilton school children: A follow-up analysis. *CJPH*. 87 (1996), 224-6.

[31] The adolescent growth spurt.

[32] Millstein, S., C. Irwin, N. Adler *et.al.* 1992, *op.cit.*

the young. Infants are dependent on parents for the security of their environment. Toddlers are mobile and frequently put items into their mouths. Both infants, toddlers and young children spend most of their time indoors. As children get older and reach adolescence, they are more exposed to the outside world and therefore come into increasing contact with a broad range of sources of environmental contaminants.

2.4 ENVIRONMENTAL MEDIA & EXPOSURE ROUTES

2.4.1 Environmental Pathways

In general, organisms are exposed to environmental hazards via several possible pathways. Pathways trace the route that a contaminant travels from its source to the receptor (living organisms). Health Canada[33] uses a scheme that divides exposure pathways into several factors (Figure 2.1 below) including;

- contamination source
- environmental media
- points and routes of exposure
- receptor (individual child, adult or population)

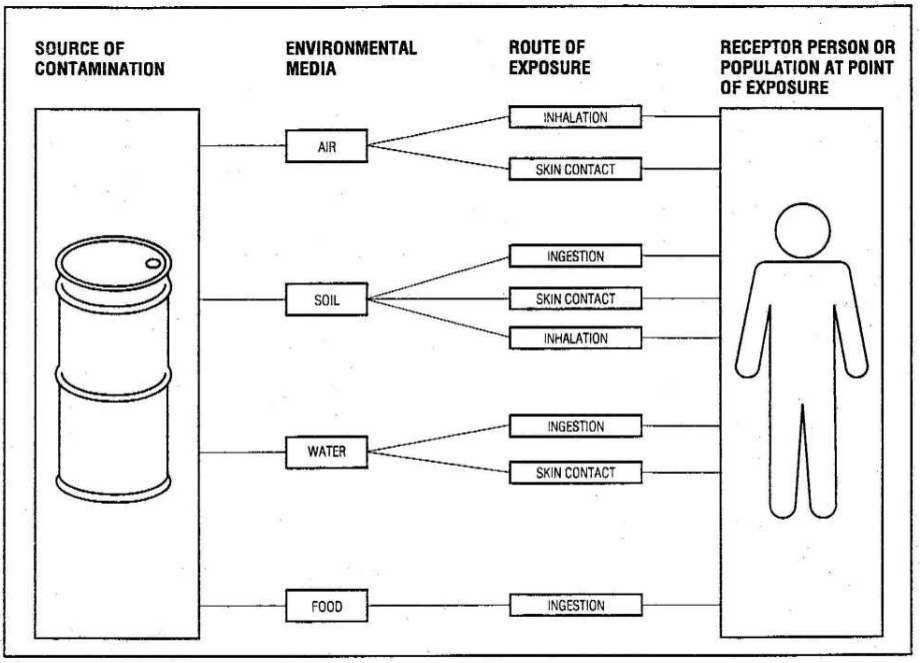

Figure 2.1. The pathways of human exposure to environmental contaminants.
(Source: Health Canada, 1995a, *op.cit.*)

Environmental media represent elements through which a contaminant travels and that ultimately are the points at which humans become exposed. Water, air, soil and food are the main environmental media. Contaminants are then transferred to, or absorbed by, the human body via three primary routes of exposure including inhalation, dermal contact (absorption), or ingestion.

The degree to which a chemical is absorbed by the body depends mainly upon the physical and chemical properties of the substance, as well as the nature of the exposure route and the exposure amount. The

[33] Health Canada. *Investigating Human Exposure to Contaminants in the Environment: A Community Handbook*. Ministry of Supply and Services, Canada. Cat. No. H49-96/1-1995E. (1995a)

main portals of entry, the lungs, digestive system and skin, may in the case of some substances, effectively inhibit absorption. If a substance passes into the body via any of these routes, it may be metabolized in the liver and excreted as a waste product in urine, feces or bile, although this does not necessarily mean that the body tissues will fully escape the harmful effects of that substance. Some chemicals are not readily excreted but become stored in the body. For example, chemicals that are fat soluble are most easily absorbed, transported through the bloodstream and to a large degree, become stored in fatty tissues of the body.

2.4.2 Children's Exposure Pathways

A fundamental reason for children's greater susceptibility to the effects of toxic contaminants is their greater exposure compared to adults. This relatively greater risk of exposure is related to, a) behavioural, b) developmental and c) physiological differences between children and adults, which have already been elaborated above. What distinguishes the child's exposure pathways is that their exploratory behaviour, hand-to-mouth activity and the fact they are closer to the ground, mean they have *greater contact* with *sources* of contamination.

Beyond the four main environmental media through which contaminants travel to people, there are additional exposure media for children that are not relevant to the adult. These include, contaminant transfer via the placenta, breast milk and non-food products, such as toys, carpets, floor surfaces, etc., which may harbour contaminants. (Figure 2.2. below illustrates a model that more accurately depicts all exposures of children.) The routes of exposures for children are also quantitatively and qualitatively different.

Underdeveloped, immature organ systems (especially the lungs, skin and gastrointestinal tract) allow greater absorption of chemical contaminants via inhalation, absorption and ingestion. As well, the smaller size of the child means that they have relatively greater intake of substances via breathing, ingestion and skin contact by comparison to their body weight.

The remainder of this section will outline the media and routes of exposure that are especially significant for children. Examples will illustrate how children have greater opportunity for exposure to several major contaminants of concern and briefly, what the main health effects may be as a result.

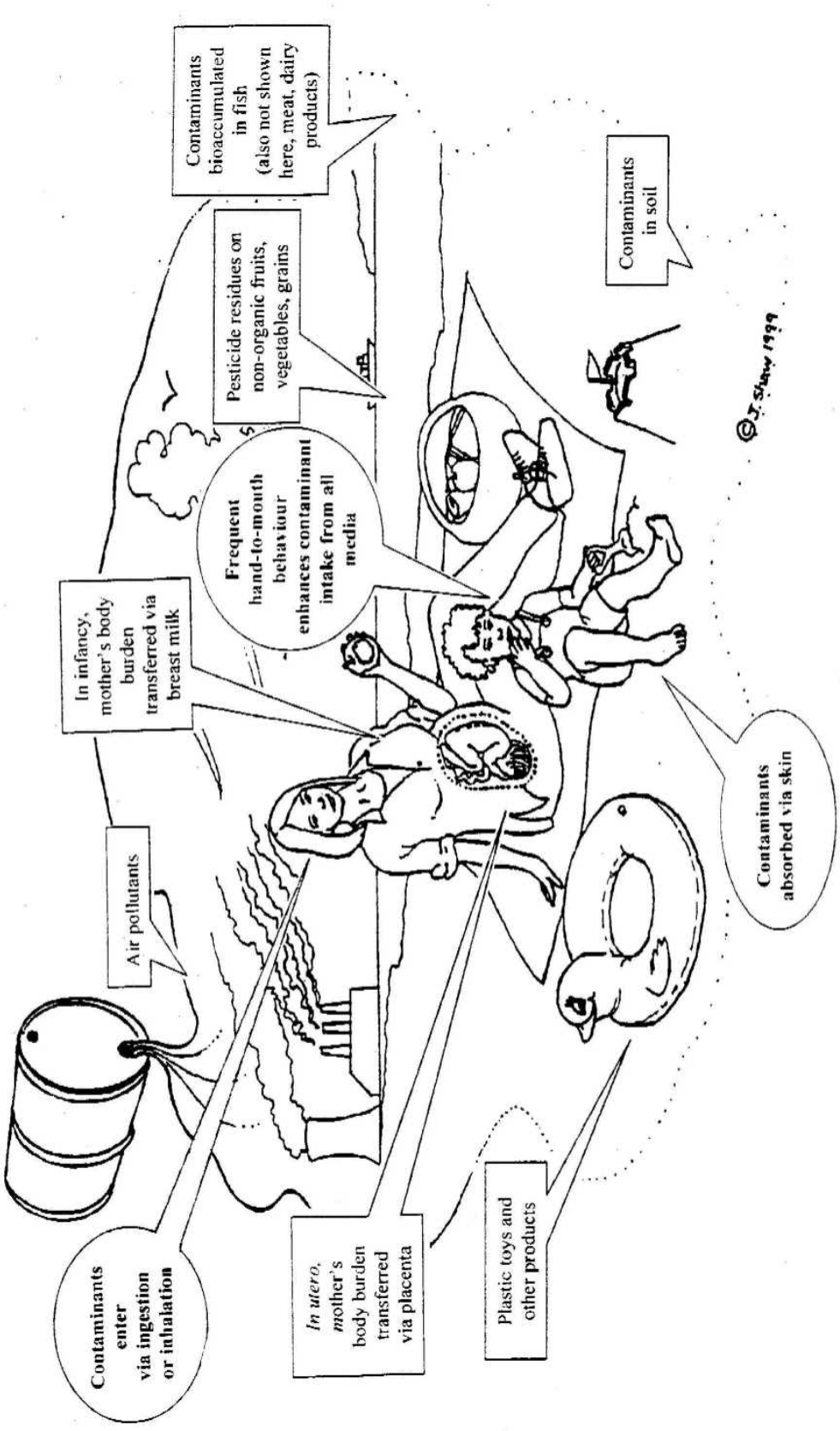

Figure 2.2. Some of the pathways of exposure to environmental contaminants for children. (Illustration by Jacqueline Shaw.)

2.4.3 Placental Transfer

a) Route of exposure: A mother's body is in fact, the first environment for an individual. Many substances can cross the placental barrier from the mother's bloodstream to the fetal bloodstream. Prenatal development may then be affected.

b) Contaminants of concern and sources: The source of fetal exposure to contaminants is in fact, the mother and her exposures. Substances that are ingested, absorbed or inhaled by the mother during pregnancy, and that stay in the bloodstream may travel across the placenta to the fetus. Compounds that the mother was exposed to during her lifetime and that became stored in her body, may also be released from maternal tissues due to physiological changes that occur during pregnancy. For example, lead stored in bone may be mobilized and the persistent fat soluble[34] compounds, including many organochlorines, like PCBs, DDT, dioxins, may also be mobilized from maternal fat stores.

c) Special concerns for children: Congenital lead poisoning has been reported in infants born to mothers who were exposed to lead as children. Infants whose mothers ate contaminated fish from Lake Michigan during pregnancy, were somewhat smaller for gestational age, had shortened gestation, smaller head circumference and were of lower birth weight.[35] Studies of newborns whose mothers body burden of PCBs and dioxins more nearly reflects that of the general population than fish eaters, suggest that while the neurological effects are not severe, higher exposure is associated with hypotonia and increased incidence of abnormally weak reflexes.[36] These studies found also that transplacental PCB exposure had a small negative effect on the neurological condition of these children at about 18 to 24 months of age but not at older ages.[37]

The main concern for placental transfer of contaminants to the fetus is that it occurs at a time when there are very sensitive developmental processes occurring that can easily be derailed, resulting in adverse effects as previously described.

2.4.4 Breast Milk

a) Route of exposure: Breast milk is the most important (and often the *only*) source of nutrition for the infant during the first few months of life. Chemicals in breast milk ultimately come from the mother's prior exposure to contaminant sources. Mothers are exposed mainly via items in their diet, such as meat, fish and dairy products. During lactation, fat stores are mobilized as a preferred[38]

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[34] The so-called lipophilic compounds.

[35] Jacobson , J.L. and S.W. Jacobson. Evidence for PCBs as neurodevelopmental toxicants in humans. *Neurotoxicology*. 18(2) (1997), 415-24; Jacobson, J.L. and S.W. Jacobson. A 4-year follow-up study of children born to consumers of Lake Michigan fish. *J. Great Lakes Res*. 19 (1993), 776-783; and Jacobson, S.W., *et.al.* The effect of intrauterine PCB exposure on visual recognition memory. *Child Dev*. 56 (1985), 853-860.

[36] Rogan W.J. *et.al.* Neonatal effects of transplacental exposure to PCBs and DDE. *J. Pediatr*. 109 (1986), 335-341; and Huisman, Marcel, *et.al.* Perinatal exposure to polychlorinated biphenyls and dioxins and its effect on neonatal neurological development. *Early Human Dev*. 41 (1995a), 111-127.

[37] Rogan, W.J. and B.C. Gladen. PCBs, DDE, and child development at 18 and 24 months. *Ann. Epidemiol*. 1 (1991), 407-413; and Huisman, Marcel, *et.al.* Neurological condition in 18-month-old children perinatally exposed to polychlorinated biphenyls and dioxins. *Early Human Dev*. 43 (1995b), 165-176.

[38] Adipose tissue stores provide approximately 60% of human milk fat, as compared to the 30% that comes from postnatal dietary intake and the 10% synthesized in the mammary gland (Schreiber, J.S. *Exposure to*

source of the fat that goes into breast milk.[39] Therefore, it is the mother's lifetime exposure, much more so than her postnatal dietary intake of these contaminants, that is the greatest determinant of their levels in breast milk.[40] Chemicals present in breast milk are then directly ingested by the nursing infant.

b) Contaminants of concern and sources: The most direct source for contaminants found in mother's milk is food, however, ultimately, these chemicals in food come from global industrial emissions that end up in air, water, soil and sediments and then from those media, find their way into the global food chain. Persistent chemicals resist degradation and become stored in the tissues of organisms. They will reach greater concentrations, as predators consume prey, bioaccumulating up the food chain.
There have been a variety of chemical contaminants identified in human milk samples globally in varying amounts.[41] These include: heavy metals such as, lead, mercury and cadmium; volatiles;[42] aromatic amines (AAs) [43]; and several from the organochlorine group of chemicals (e.g. PCBs, DDT and metabolites, other chlorinated pesticides, dioxins and furans).[44]

c) Special concerns for children: Nursing babies are at the top of the food chain and therefore, there is concern that they may receive close to an adult level dose at the beginning of their lives when they are breast-fed.[45] However, the potential for health effects from contaminants in breast milk is not simply determined.

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contaminants in breastmilk: A risk-benefit assessment. Doctoral dissertation, SUNY at Albany, School of Public Health,1992).

[39] Jensen, A.A. Transfer of chemical contaminants into human milk. In: *Chemical Contaminants in Human Milk.* Jensen, Allan Astrup & Stuart A. Slorach (eds.) (Boca Raton: CRC Press, Inc., 1991:10), pp. 9-19.

[40] *Ibid.*

[41] Sonawane, B.R. Chemical contaminants in human milk: An overview. *Environmental Health Perspectives.*103 (Suppl 6) (1995), 197-205.

[42] Pellizzari, E.D., T.D. Hartwell, B.S.H. Harris, R.D. Wadeell, D.A. Whitaker and M.D. Erickson. Purgeable organic compounds in mothers' milk. *Bull. Environ. Contam. Toxicol*. 28 (1982), 322-328.

[43] A recent study by University of Guelph and Waterloo researchers of 31 lactating women from the Guelph area found levels of from less than 0.01 to 7.44 ppb. (De Bruin L.S., J.B. Pawliszyn and P.D. Josephy. Detection of monocyclic aromatic amines, possible mammary carcinogens, in human milk. *Chem Research in Toxicology*. 12(1) (1999), 78-82.).

[44] Jensen, A.A. 1991, *op.cit.*; and Haines et.al.,. Dioxins & Furans. Chapter 6.0 In: *Persistent Environmental Contaminants and the Great Lakes Basin Population: An Exposure Assessment.* Health Canada. Minister of Public Works and Government Services, Canada. Catalogue No. H46-2/98-218E. (1998a); and Haines *et.al.*,. Polychlorinated Biphenyls. Chapter 11.0 In: *Persistent Environmental Contaminants and the Great Lakes Basin Population: An Exposure Assessment.* Health Canada. Minister of Public Works and Government Services, Canada. Cat. No. H46-2/98-218E. (1998b)

[45] The breast milk concentration of organochlorine chemicals varies with the lipid content of the milk and will rise initially with the transition from lower fat colostrum (produced in the first few days postpartum) through to higher fat, mature milk. Over time, the levels in breast milk increase and then decrease, reflecting elimination of the stored levels of these contaminants via lactation. There is an estimated 30 to 50% decrease in the mother's tissue or fluid PCB levels after five to six months of lactation (Yakushiji, Tsumoru. Contamination, clearance and transfer of PCB from human milk. *Rev. Env. Contam. Tox.* 101 (1988), 139-164). There will therefore also be a decrease in the organochlorine content of breast milk after infants of second and higher birth order.

Lead does not appear to concentrate in human milk and the minor[46] amounts found in breast milk are assumed not to present a health hazard to breastfed infants.[47] Volatiles[48] are presumed to be of low toxicity and carcinogenicity to humans. Aromatic amines (AAs),[49] recently reported in breastmilk of a sample of Ontario women, are known to produce tumours in rat mammary tissue, therefore are of some concern for increasing cancer risk in mothers and breastfed children, although there is no research to confirm this.

There has been comparatively more research on the potential health effects from exposure to organochlorine contaminants in breast milk. Long term studies of children whose mothers had measurable levels of PCBs in breast milk, have found slight effects on neuromuscular development in the first 2 years, with development progressing normally after that.[50] Others have observed cognitive and behavioural problems but indicated that prolonged breast-feeding was linked to improved memory and verbal scale test performance.[51] Health studies of breastfed Inuit infants[52] have indicated an increased incidence of ear infections and "modest" compromise to immune function.[53]

The risks associated with exposure of the infant to chemicals via breast milk are difficult to define[54] mainly because of the problem of separating the effects from prenatal (via placenta) versus postnatal (via breast milk) exposure.[55]

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[46] Small amounts of lead are found in all human body tissues. Levels in breast milk tend to be lower than those in infant formula or food. (Gulson, B.L., C.W. Jameson, K.R. Mahaffey, K.J. Mizon, N. Patison, A.J. Law, M.J. Krosch and M.A. Salter, Relationships of lead in breast milk to lead in blood, urine, and diet of the infant and mother." *Environmental Health Perspectives.* 106 (1998), 667-674; and Sinks, Thomas and Richard J. Jackson. International study finds breast milk free of significant lead contamination. *Environmental Health Perspectives*. 107(2) (1999), A58-59.)

[47] Rogan, W.J. Epidemiology of environmental chemical contaminants in breast milk. In: *Human Lactation 2: Maternal and Environmental Factors*. Margit Hamosh, Armond S. Goldman (Eds.) (New York: Plenum Pub. Corp., 1986), pp. 437-446; and Sinks & Jackson. 1999*, op.cit.*

[48] Pellizzari, E.D., *et.al.* 1982*, op.cit.*

[49] De Bruin L.S., *et.al.* 1999*, op.cit.*

[50] Rogan, W.J. *et.al.* Should the presence of carcinogens in breast milk discourage breastfeeding? *Reg. Tox. & Pharm.* 13 (1991), 228-240; and Huisman, M., *et.al.* 1995b, *op.cit.*

[51] Jacobson, J.L. and S.W. Jacobson. 1993, *op.cit.*; and Huisman, Marcel, et.al. Neurological condition in 18-month-old children perinatally exposed to polychlorinated biphenyls and dioxins. *Early Human Dev*. 43 (1995b), 165-176.

[52] Nunavik infants from Northern Quebec have had the highest known exposure to PCBs via breastmilk of any global population. Breastmilk samples from Nunavik mothers exhibited PCB levels of 111.3 µg/L, almost four times the levels for women in Southern Quebec (Dewailly, Eric, A. Nantel, J-P. Weber and F. Meyer. High levels of PCBs in Breast Milk of Inuit Women from Arctic Quebec. *Bull. Environ. Contam. Toxicol.* 43 (1989), 641-646).

[53] Ayotte, P and E. Dewailly. Health risk assessment for newborns exposed to organochlorine compounds through breastfeeding. In: J.L. Murray and R.G. Shearer (eds.) *Synopsis of Research Conducted Under the 1992/93 Northern Contaminants Program.* Environmental Studies No. 70, Northern Affairs Program. Minister of Government Services, Canada. (1993), pp. 263.

[54] Schreiber, 1992, *op.cit.*

[55] While exposure via breastmilk is considerable, it is believed that the much smaller amount that is transferred across the placenta is of greater clinical significance due to the vulnerability of the developing fetus to chemical insults (Jensen, 1991, *op.cit.*). Prenatal exposure has been characterized as largely responsible for

After repeated evaluation of the risks versus benefits of breastfeeding, it has been widely acknowledged[56] that the benefits from breast-feeding, for both infant and mother, (including psychological, nutritional, immune and health protective benefits) far outweigh the risks from exposure[57] to breast milk contaminants. Hence, virtually no health researcher or practitioner would deny that "breast is best".

2.4.5 Air

a) Route of exposure: Children inhale or come into dermal contact with contaminants from both outdoor and indoor air.

b) Contaminants of concern and sources: Ground-level ozone,[58] sulphur dioxide and acid aerosols,[59] oxides of nitrogen, particulates[60] and carbon monoxide are the main outdoor air pollutants associated with adverse effects on respiration. Other toxic substances present in ambient air, such as particles of heavy metals and other organic chemicals, often called air toxics, are of concern because of their ability to affect health in other ways including as carcinogens. Outdoor air pollutants come from coal-fired electric stations, industrial emissions, and fires, waste incineration, vehicle exhaust and residential and commercial space heating. Sources may be local or distant as there is considerable long-range transport of air pollutants in atmosphere. Ontario reports that 50% of smog measured here originates in the U.S.[61] There is a great variety of indoor air pollutants that may affect children's health such as: 1. Indoor allergens from House Dust Mites, furry or feathered pets, molds and cockroaches; 2. Gases such as formaldehyde and VOCs (Volatile Organic Compounds); and, 3. Particulates, from Environmental Tobacco Smoke (ETS)and from fireplaces or woodstoves. Many outdoor pollutants also penetrate indoors. Indoor air pollution is worse in areas with poor ventilation and dampness.

c) Special concerns for children: Children are at increased risk compared to adults because of growth and developmental factors, and because of exposure factors. Children breath faster than adults,

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"modest" deficits and "diminished potential" in exposed children (Jacobson & Jacobson, 1993, *op.cit., p.* 781).

[56] Frank, JW & J Newman. Breast-feeding in a polluted world: Uncertain risks, clear benefits. *Can Med Assoc Journal* 149(1): 33-7 (1993); and Rogan, WJ. Pollutants in breast milk. *Arch Ped Adolesc* Med 150: 981-990 (1996).

[57] It is noteworthy that there are no standards for the levels of PCBs in breast milk in Canada. There are Federal standards are for daily intake (ADI = 1ug/kg body wt.) and for blood levels. The breast milk "discretionary" level adopted by Health & Welfare Canada (50ppb), is not a regulatory standard and is not based on any scientific analysis of risk, or a known health limit or dose that is harmful to the breast-fed infant.

[58] Ground level ozone is the result of the interaction in the atmosphere in the presence of oxygen and sunlight of several precursor air pollutants, such as oxides of nitrogen (NO_x) and volatile organic compounds (VOCs).

[59] Acid aerosols are fine suspended liquid particles that are mostly sulphates derived from burning fossil fuels.

[60] Particulates refers to fine solid particles that also result from the burning of fossil fuels and emissions from industrial operations. These particles are of extremely small diameter, either less than 10 microns (PM_{10}) or less than 2.5 microns ($PM_{2.5}$) and therefore they remain suspended in air.

[61] Ontario Ministry of the Environment, *Air Quality in Ontario: A Concise Report on the State of Air Quality in the Province of Ontario, 1997*. (Toronto: Queen's Printer for Ontario, 1999). Available at: http://www.ene.gov.on.ca/envision/news/3909e.pdf

inhaling greater amounts of air pollutants relative to their body weight.[62] The lungs and airways of children are still developing, and are especially sensitive to insults from pollution. Because they spend a great deal of their time indoors, and because there are no regulations governing the quality of indoor air, children may be at greatest risk of health effects from exposure to indoor air pollution. Also, when outdoors, they tend to be more active than adults, breathing faster during play activity, and therefore increasing exposure to outdoor air pollutants. They might also be more exposed because at the time they come home from school to play in the afternoon, ozone levels are usually peaking. [63] The effects of both indoor and outdoor pollution overlap, and respiratory symptoms, including asthma, are the most recognizable response.

2.4.6 Water

a) Route of exposure: Exposure to contaminants in water is primarily through skin contact or direct ingestion of water used for drinking or bathing. It may also come from inhalation of volatilized (vapourized) substances in shower or bath water, or from chlorinated pools. Exposure may also occur via immersion in contaminated natural swimming water and therefore, through direct skin absorption.

b) Contaminants of concern and sources: Water may become contaminated from a number of sources, exposing people to various contaminants. Lead, leached from solder of older plumbing fixtures may be present in tap water. Municipal water supplies may have high levels of different substances collectively called disinfection by-products that result from chlorine treatment. Direct discharge from industrial sources may affect water used in municipal supply, or water that is used for recreational purposes. Other persistent organic chemicals, including PCBs, DDT, and dioxins, may be absorbed from water sources. Chemicals from hazardous waste sites or landfills, or irrigation runoff from pesticide treated agricultural fields, may leach into groundwater sources and therefore be a source of water contamination where well water is used.

c) Special concerns for children: Water is an underestimated source of children's exposure to contaminants.[64] Because children drink much more water per unit of body weight than adults, they are at risk of greater exposure to contaminants ingested from drinking water. Infants under age one have the highest relative water intake, consuming between about 4 to 6 times more water than adults and even older children.[65] The developing lungs and skin allow for relatively greater exposure to substances inhaled, ingested and absorbed dermally when children drink or bathe in contaminated water. Lead is a potent neurotoxicant to which children are especially vulnerable (see Case Study#1). Disinfection by-products, such as trihalomethanes (THMs), appear to be associated with an increased risk of spontaneous abortion in women.[66]

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[62] Fenske, R.A. 1992, *op.cit.*

[63] Bates, D.V. The effects of air pollution on children. *Environmental Health Perspectives* 103 (Suppl 6): 49-53. (1995)

[64] National Research Council. *Pesticides in the Diets of Infants and Children*. (Washington: National Academy Press, 1993.)

[65] *Ibid.*

[66] Waller, K. *et.al.* Trihalomethanes in drinking water and spontaneous abortion. *Epidemiology*. 9 (1998), 134-40.

2.4.7 Soil & Dust

a) Route of exposure: Exposure to contaminants in soil may come from direct dermal contact, ingestion of soil or via inhaling soil dust carried in air. Such dust particles from contaminated soil can also settle on surfaces and products, on which children mouth or chew, thereby providing another route for ingested chemicals.

b) Contaminants of concern and sources: Soil and sediments may harbour a variety of chemical contaminants to which people and especially children, may become exposed. Food grown in contaminated soil may also be a point of transfer of chemical substances from soil to humans. Sediments of natural waters may harbour contaminants that people can come in contact with through recreational activities such as swimming. Indoor "accumulation of dust, dust mites, and tracked-in soil in old carpets, sofas, and mattresses appears to be a major source of exposure to lead, pesticides, allergens, PAHs, and VOCs."[67] Disposing of lead and PCB contaminated soil from industrial sites has been an issue for large urban centres such as Toronto or Hamilton. Reclaimed industrial land is often used for subsidized housing projects, therefore children of lower income families may be at greater risk from exposure to contaminated soil.

c) Special concerns for children: Infants and small children are particularly vulnerable to exposure via direct ingestion of contaminated soil since they frequently put objects (and their hands) in their mouths. The greater exploratory behaviour of children of different ages may also bring them into more frequent contact with soil contaminants. A behaviour called pica (eating of non-food items, including soil) is common in many children and may allow for toxic exposures to soil contaminants.[68] Toddlers with pica behaviour are at greater risk of exposure to contaminants in dust.[69]

2.4.8 Food

a) Route of exposure: Contaminants found in food enter children by ingestion.

b) Contaminants of concern and sources: Food may transfer contaminants to humans in a number of different ways and from different sources. Food (including that from home gardens) grown in contaminated soil or using contaminated water will transmit chemicals to humans. Fish, (especially large, fatty species) and wildlife may be contaminated by water, sediments, or from lower organisms in the food chain. Agricultural food products may harbour residues from pesticide use. The secondary products that are made from these items, e.g. baby food, have been found to have trace measures of several different pesticides. Packaging may transfer contaminants to foods. For example, lead soldering formerly used to seal canned goods may contaminate food. Chemicals from plastic containers may readily leach into foods that are acidic (e.g. Bisphenol A; nonylphenol).

c) Special concerns for children: Anglers, immigrant families, and aboriginal communities (and their children) who all might eat greater quantities of fish caught in contaminated lakes and rivers, are those most at risk of exposure to what can be higher doses of toxins. Because of the nature of their diets and food preferences, and the relatively greater amount of food eaten per unit body weight,

[67] Roberts, J.W. and P. Dickey. Exposure of Children to pollutants in house dust and indoor air. *Rev. Env. Cont Tox.* 143 (1995), 59.

[68] Calabrese E.J., E.J. Stanek, R.C. James and S.M. Roberts. Soil ingestion: a concern for acute toxicity in children. *Environmental Health Perspectives*. 105 (1997), 1354-8.

[69] Roberts, J.W. and P. Dickey. 1995*, op.cit.*

children are again, particularly prone to exposure to contaminants in food.[70] The Guide to Eating Ontario Sport Fish[71] re-states Health Canada's advisory recommending that women of childbearing age and children under 15 avoid or limit consumption of species that are more highly contaminated. However, recent research suggests that some groups (e.g. immigrants) may not be aware of these advisories[72] and that their is wide variability in the degree to which fishers follow the recommended catching, cleaning and cooking practices in order to minimize contaminant exposure.[73]

2.4.9 Products

a) Route of exposure: Products are particularly important, both as exposure routes and as direct contamination sources. Because children are prone to putting things in their mouths, and because of their small size and crawling behaviour, they come into direct contact with many products containing toxins that can be ingested or dermally absorbed.

b) Contaminants of concern and sources:
Toys: There has been recent concern over the potential hazards to children from exposure to various toxic elements used in the manufacture of polyvinyl chloride (PVC) plastic toys. Tests of samples of plastic toys have found that many of them, from a variety of manufacturers, contain unacceptably high levels of lead and/or cadmium.[74] Some of these toys also carry lead- and cadmium-tainted dust on their surfaces when brand new and are shown to release further tainted dust under situations that simulate aging.[75] Many soft, chew toys also contain about 4 to 44% per weight phthalates, plasticising chemicals that make PVC toys soft.

c) Toys may also become *media* for transfer of environmental contaminants from other sources. A recent study examined the deposition pattern of chlorpyrifos, a semi-volatile insecticide commonly used in household and industrial applications.[76] This study demonstrated that even when this organophosphate pesticide was sprayed in apartments according to manufacturer's instructions, residues continued to be deposited on toys and other absorbent household surfaces long after[77] initial application.

d) Special concerns for children: Health Canada recently established that there is a definite unnecessary and unacceptable health risk from exposure to the most common phthalate, Di-isononyl phthalate

[70] National Research Council. 1993, *op.cit.*

[71] Ontario Ministry of Environment & Energy. *The Guide to Eating Ontario Sport Fish*. 20th Ed. (Toronto: Queen's Printer for Ontario, 1999.)

[72] Dawson, Jennifer and the Fish and Wildlife Nutrition Project. Working Paper E. Have They Been Hooked?: A Look at How Fishers Use the Guide to Eating Ontario Sport Fish. Great Lakes Health Effects Program. (1997)

[73] Sheeska, J. Working Paper D. Sheepshead patties, smoked carp and other delicacies: Preparing and Eating Sport Fish from Great Lakes Areas of Concern. Unpub. ms. Prepared for the Great Lakes Health Effects Program. Contract No. H4078-5-C385/001/SS. (1998)

[74] Greenpeace report. Vinyl Children's Products Pose Lead and Cadmium Hazard, September, 1997.

[75] Greenpeace, 1997, *op.cit.*

[76] Gurunathan, S. *et.al.* Accumulation of Chlorpyrifos on residential surfaces and toys accessible to children. *Environmental Health Perspectives*. 106 (1998), 9-16.

[77] Peak deposits were measured 36 hours after application.

(DINP), in children under one year who chew on PVC toys for extended periods of time.[78] DINP has been found to be toxic in high doses to liver, kidneys and reproductive systems in laboratory animals and may act as a weak disrupter of endocrine function.[79]The cancer risk to humans from DINP was deemed inestimable from animal models,[80] however, phthalates have been described elsewhere as probable human carcinogens based on their cancer-causing effects in mice and rats.[81] Exposure to the heavy metals from these toys is also of concern for the potential effects on neurological development as described in detail below.

DINP may be released when toys are chewed or sucked for prolonged periods. In most cases, it may be unlikely that infants are sucking on such toys for the amount of time estimated in experimental situations, however, the fact remains that babies do have a marked tendency to put things in their mouths and in many cases, these toys, like teething rings are intended to be chewed by infants. Gurunathan and colleagues[82] estimated there would be significant intake of chlorpyrifos by children from both ingestion and dermal absorption of pesticide residues on toys (but not inhalation) after a "normal" home treatment.[83]

e) Other products: Furnishings, draperies, carpets, pillows and other absorbent surfaces in the home may become reservoirs for ambient chemicals to which children may be exposed (see above viz. chlorpyrifos deposition). The materials used in the manufacture of these items may also be toxic themselves. For example, in the recent past, imported plastic mini-blinds were found to contain substantial amounts of lead.

Building materials can be hazardous to children, especially those used in older buildings, such as asbestos, lead-based paints, formaldehyde present in particle board, and foam insulation. Asbestos is a powerful carcinogen and lead is a potent neurotoxicant. Off-gassing from urea formaldehyde insulation was experienced as strongly irritating by some household occupants. Chemical agents that are used around the home may also be direct toxicants for children. Examples include household cleaning agents, make-up, shampoo, antibacterial soaps, paints, solvents, insecticides.

2.4.10 Additional Factors Influencing Exposure & Susceptibility

[78] Health Canada. *Updated: Risk assessment on di-isononyl phthalate in vinyl children's products*. Consumer Products Division, Product Safety Bureau, Environmental Health Directorate, Health Protection Branch. (November 14, 1998d), 7 pp. http://www.hc-sc.gc.ca/advisory/risk.htm.

[79] *Ibid.*; and Di Gangi, Joseph. Warning: Children at Risk. Toxic chemicals found in vinyl children's products. Report for Greenpeace, USA. (1998) http:www.greenpeacusa.org/media/publications/vinyltoys.html and Environmental News Network, Store yanks direct-to-mouth PVC toy. (Monday November 16, 1998) http://www.enn.com/news/enn-stories/1998/11/111698/toysrus.asp.

[80] Health Canada. 1998d, *op.cit.*

[81] Public Interest Research Group. Trouble in Toyland. Summary. (1998)

Di Gangi, Joseph. 1998, *op.cit.;* and Aristech Chemical Corporation. Material safety data sheet C1084E. Product code 1564; diisononyl phthalate. 1995 (as cited in Di Gangi, 1998. *op.cit.*).

[82] Gurunathan, S. *et.al.* 1998, *op.cit.*

[83] Acute exposure to chlorpyrifos appears to be associated with headaches, dizziness, muscle twitching, vomiting, blurred vision among other symptoms reported anecdotally (Anonymous. Playing with Pesticides. Environews Forum. *Environmental Health Perspectives*. 106 (1998), A10).

Introduction

A thorough understanding of the multiple determinants of children's environmental health is beyond the scope of this report. However, we acknowledge that physical environmental factors and the behavioural and developmental characteristics of the young represent only a limited portion of the correlates of children's environmental health. The full range of determinants of health and well-being is broad and interactive and includes; the social and economic environment, the physical environment, individual characteristics (such as genetic, biological, psychological and behavioural features) and community factors (such as medical, health care access and cultural factors).[84] In this report, we highlight several important reasons why children vary in their reactions or exposures to environmental contaminants.

Genetic Susceptibility

Genes are involved in the regulation of growth, development, metabolism, replication and repair, at the organ, cellular and DNA levels. There is a great degree of individual genetic or biological variability in the human population that influences the degree to which some children are adversely affected by environmental exposures. For example, the genetic anemia that results from a deficiency of the enzyme known as glucose-6-phosphate-dehydrogenase (G6PD) increases an individual's susceptibility to the toxic effects of certain oxidant chemicals.[85] However, there are also more frequently occurring genetic differences that affect susceptibility to common environmental contaminants. Of the two forms (alleles) of the gene that codes for the enzyme (delta-aminolevulinate dehydratase, ALAD) in the biosynthetic pathway of heme, it is hypothesized that one allele (ALAD-2) may bind lead more tightly, thereby rendering these individuals more susceptible to the effects from lead exposure.[86] There are commonly recognized genetic differences (polymorphisms) in the cytochrome P-450 system and other enzyme systems that de-activate pesticides suggesting that there are likely genetically-mediated differences in susceptibility to pesticide injury.[87] Children with asthma are much more susceptible to the adverse health effects from exposure to air pollution. Asthma is a condition that is likely caused to an extent by both genetic and environmental factors.

Socioeconomic and Nutritional Factors

Other children have greater response or exposure to environmental contaminants because of their social environment or economic circumstances. Children from low income households may be especially vulnerable to environmental exposures. For example, the poor housing and poor nutrition of children from low income households amplifies their exposures to pollutants like lead or pesticides.[88] Poor nutrition in the young is a factor that is implicated in altering the biological processes by which the body deals with lead. For example, greater calcium in the diet decreases the gastrointestinal absorption of lead

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[84] Evans, R.G., M.L. Barer and T.R. Marmor. (eds.) *Why are Some People Healthy and Others Not?* (New York: Walter de Gruyter Inc. 1994)

[85] Reiser, Karen. General principles of susceptibility. In: *Environmental Medicine*. Brooks, Stuart M. *et.al.* (Eds). (St. Louis: Mosby, 1995), pp. 351-360.

[86] Suk, W.A. and G.W. Collman. Genes and the Environment: Their impact on children's health. *Environmental Health Perspectives*. 106 (Suppl 3) (1998), 817-820.

[87] Rabovsky, J. Malathion metabolism. In: *Health Risk Assessment of Aerial Application of Malathion Bait.* Berkeley: California Department of Health Services, Pesticides and Environmental Toxicology Section. (1991)

[88] Chaudhuri, N. Child health, poverty and the environment: The Canadian context. *CJPH*. 89 (Suppl 1) (1998), S26-S30; and Landrigan *et.al.*, 1999. Pesticides and inner-city children: Exposures, risks, and prevention. *Environmental Health Perspectives* 107 Suppl 3: 431-437.

and therefore reduces the risk for lead poisoning.[89] Children with low iron levels are also more likely to eat excessive amounts of soil, thereby exposing them to higher levels of soil contaminants such as lead.[90] Apartment dwellers in poorer areas are more likely to be exposed to pesticides applied to control cockroach infestations. Low income neighbourhoods are also more often in closest proximity to sources of environmental contaminants such as landfills, urban industry and roadways.

Cultural Factors

Aboriginal communities, because they continue to maintain a culture that fits with their closer relationship to the land and traditional subsistence practices, are more often at risk of exposure to contaminants in the environment. For example, many First Nations communities in Canada continue to hunt wild game and fish from lakes and rivers. These food items, at higher trophic levels of the food chain, are sources of contaminants that bioaccumulate. Other groups, such as sport fishermen and immigrants may also be likely to utilize these food sources. In both cases, children may be exposed prenatally and through their diet, including breast milk.

Aboriginal children are likely the most vulnerable group of children because of the pervasiveness of other risk factors that adversely influence their health such as high infant mortality and morbidity, poverty, poor nutrition, poor housing, inadequate water supplies and sanitation, other social problems such as discrimination, suicide, lack of power, as well as the proximity of reserves to sources of environmental contaminants.[91]

2.4.11 Summary

All things considered, children are relatively more exposed to contaminants present in the main environmental media (water, air, soil and food), plus there is greater opportunity for those contaminants to enter the body via the main routes of exposure (inhalation, ingestion, or dermal contact). There are additional environmental media, not considered in the typical models for assessing exposure, through which the young may be exposed to environmental health risks. These include, placental transfer, breast milk, and products such as toys.

Children are more exposed because they are more active both indoors and out, therefore they are more likely to breathe in ambient contaminants. They exhibit more exploratory behaviour and hand-to-mouth activity and therefore, are more often accidentally ingesting or absorbing residues from objects in the environment. Being closer to the ground, they come into greater contact with contaminants present in the air, in soil and dust, and on lawns, carpets and other household items. Children's diets are typically higher in the kinds of foods that may carry contaminants, such as, breast milk, fruits and vegetables. Children's lungs, immune and nervous systems are immature for the early period of life and therefore, they are more sensitive to and less able to withstand the effects from exposure to toxins. Because of their small size, children breathe in more air, eat more food, drink more liquids and absorb more chemicals through skin on a per weight basis when compared to adults.

Genetic, social and cultural differences that influence a child's exposure to or ability to deal with

[89] Bruening, K., F.W. Kemp, N. Simone, Y. Holding, D.B. Louria and J.D. Bogden. Dietary Calcium intakes of urban children at risk of lead poisoning. *Environmental Health Perspectives*. 107 (1999), 431-435; and Mushak, P. and A.F. Crocetti. Lead & Nutrition: Part II. Some potential impacts of lead-nutrient interactions in U.S. populations at risk. *Nutrition Today*. 31(1996), 115-122.

[90] Calabrese E.J., *et.al.* 1997, *op.cit.*

[91] Chaudhuri, N. 1998, *op.cit.*

pollutants are also important factors to consider when assessing children's environmental health.

2.5 CONTAMINANTS AND THEIR KNOWN EFFECTS

2.5.1 Introduction

There is a daunting array of environmental agents,[92] to which people are exposed through various means. Many of these agents can be considered harmful to humans, especially children, however, for practical purposes, we limit ourselves here to a discussion of compounds classified as chemical and metal pollutants. This report therefore, will not include information on other very important categories of environmental contaminants that are believed to affect human health, namely; physical (e.g. electromagnetic frequency, radiation, radon, radionuclides), biological agents (e.g. molds, fungi, bacteria, etc.) and environmental tobacco smoke.

This section will present a general overview of the current state of knowledge concerning some environmental contaminants. It will focus on those that impact on human health, are particularly harmful to children and are also relevant to the Ontario and Canadian setting.

Effects, or potential effects from contaminants vary according to the type and nature of the chemical, timing of exposure, frequency and duration of exposure and exposure dose. The effects also vary according to many factors inherent to the exposed individual. For the majority of environmental chemicals, it can be said that there is much more known about human health effects from acute, high dose poisonings (i.e. from occupational, accidental exposures or intentional overexposures, i.e. suicides) than from chronic, low-level exposures. Epidemiological studies are invaluable for providing some clue to the health effects from these real-life exposures, whether occupational or environmental, in human populations.[93] More often however, the only information on chronic, low-level exposures comes from experimental studies on laboratory animals and observational data from exposed wildlife. It is important to note again, however, that environmental health researchers are documenting increasingly more subtle effects of concern from what were previously believed to be "safe" or "below threshold" exposures to many different contaminants.

2.5.2 Persistent Organic Pollutants

Persistent organic pollutants (POPs) represent a class of contaminants that includes many industrial chemicals and some pesticides. These chemicals are of major health significance because they are not easily degraded and therefore remain in the environment for a long time[94] (hence the term persistent). They are not water soluble but are soluble in fat (i.e. they are lipophilic), therefore they become stored in fatty tissues of organisms that ingest them. Because they persist and are stored in fat, they will become concentrated in organisms at increasingly higher levels of the food chain (i.e. these chemicals bioaccumulate). Humans and other mammals efficiently absorb POPs, and since they remain virtually

[92] One source estimates that of the approximately 7 million chemicals that exist, 70,000 are used currently and more than 1000 are added to the world market annually (Newill, V.A. Keynote address: significance of risk assessment in the management of environmental exposures of chemical mixtures. *Toxicol. Ind. Health.* 5 (1989), 635).

[93] For a more detailed discussion of the use of epidemiological studies as sources of data. see Chapter 4.

[94] The Great Lakes Water Quality Agreement distinguishes any pollutant as persistent if it has a half-life of greater than 8 weeks.

un-metabolized and are only minimally excreted, they are stored[95] in fatty tissues throughout the body.[96] The only normal route of elimination is by liberation from fat stores and excretion during lactation.

Specific industrial chemicals that are persistent organic pollutants include substances in the organochlorine category of POPs, such as, PCBs, dioxins and furans. (Pesticides of the organochlorine type, such as DDT, also represent POPs, but these will be considered separately in the next section.) PCBs are chemicals that had wide industrial use in transformers, electrical capacitors, and hydraulic fluids, and as flame retardants, adhesives and plasticizers. They were banned in the 1970s but are still widespread in our environment because of their marked persistence and stability. Dioxins and furans are two structurally similar families of chemicals. Although some dioxins are naturally produced (from forest fires and volcanoes), most are a by-product of chlorine and petroleum industrial processes (e.g petrochemical industry; pulp and paper bleaching process, etc.) or are produced from waste incineration. Furans are a trace contaminant of PCBs.

Health Effects

Like other organochlorines, PCBs are associated with neurodevelopmental and immune system effects in the young. They may also be cancer promoters. The most potent dioxin[97] (TCDD) has been labeled a known human carcinogen, believed to be highly carcinogenic at even low doses, and several other dioxins are described as probable human carcinogens.[98] Both PCBs and dioxin are also speculated to disrupt endocrine function in humans, based on laboratory studies in animals and tissue cultures. Scientists meeting under the auspices of the World Health Organization have recently suggested that dioxins may also have effects on neurological development, the immune system, reproductive system and growth and development in humans and other animals.[99]

Human Exposure Estimates

Because POPs bioaccumulate and because they have been widely distributed in the global environment via long-range transport, most Canadians have trace amounts of POPs in their blood and fatty tissues. By far, the greatest degree of exposure to POPs such as PCBs comes from diet (94 to 99%), particularly consumption of breast milk, fish, fatty meats and dairy items.[100] Other media through which there is some (albeit minimal) exposure include; indoor air, ambient air, house dust, soil and drinking water in decreasing order of importance.

Health Canada estimates for the average total daily intake of various POPs from all media are presented in Table 2.2 below. The general pattern is that adolescents, children, and especially breastfed infants, all have relatively much greater exposure to these contaminants than do adults. In some cases, (e.g. PCBs)

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[95] See Patton (1986, *op.cit.*) for a thorough discussion of the steps in the digestion, absorption, storage and mobilization of lipophilic chemicals in the body. Ironically it is our ability to store these contaminants in fat that allows for some degree of protection from their effects on tissues.

[96] Wolff, M.S. Lactation. In: *Occupational & Environmental Reproductive Hazards: A Guide for Clinicians*. (Baltimore: Williams & Wilkins, 1993), pp. 60-75.

[97] Dioxins represent a family of over 200 different chemicals. The most toxic dioxin is 2,3,7,8-tetra-chloro-dibenzo-p-dioxin (TCDD).

[98] McGregor, D.B., *et.al.* An IARC evaluation of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans as risk factors in human carcinogenesis. *Environmental Health Perspectives* 106 (Suppl 2) (1998), 755-760.

[99] World Health Organization. WHO experts re-evaluate health risks from dioxins. Press release WHO/45, (June 3, 1998) http://www.who.org/inf-pr-1998/en/pr98-45.html.

[100] Haines *et.al.*,. 1998b, *op.cit.*

this is almost sixty times the amount that adults receive and in other cases (e.g. dioxins and furans), the amount far exceeds the Health Canada guidelines for average daily intake. The greatest source of these extreme discrepancies in adult versus child intake is due to the exposure to contaminants present in breast milk.

Table 2.2. Estimated exposures/daily intakes for Great Lakes general population of various POPs via all media.[101]

	Breastfed	Formula					**Average Daily Lifetime Intake**	Intake Guide-lines
	0-6mos	0-6mos	7mos-4yr	5-11yr	12-19yr	20+yr		
PCBs[102]	808.18	47.96	65.60	45.93	24.73	21.83	33.00	PTDI = 1,000
Dioxins Furans[103]	57.05	12.56	9.54	4.69	2.25	1.20	2.60	TDI = 10
DDT & related cmpds[104]	701.27	222.85	226.35	121.21	58.68	43.57	69.51[105]	TDI = 20,000

In Canada, breast milk has been routinely tested for the presence of persistent toxic substances such as organochlorine pesticides and PCBs since 1967. There has been a distinct downward and leveling trend in the banned POPs (e.g. PCBs, DDT, DDE, etc.) in the Great Lakes region[106] over recent decades.[107] However, the estimates presented above indicate that exclusively breastfed infants under 6 months of age in the Great Lakes region are likely exposed to 81% of the Health Canada Provisional Tolerable Daily Intake (PTDI) for PCBs of 1µg/kg bw/day and almost *six* times the Tolerable Daily Intake (TDI)[108] of 10

[101] Haines M., *et.al.*,. 1998a, *op.cit.*; and Haines M., *et.al.*, 1998b, *op.cit.*; and Haines M., *et.al.*, DDT. Chapter 5.0 In: *Persistent Environmental Contaminants and the Great Lakes Basin Population: An Exposure Assessment*. Health Canada. Minister of Public Works and Government Services, Canada. Cat. No. H46-2/98-218E. (1998c)

[102] Figures presented in ng/kg bw/day.

[103] Figures presented in pg TEQ/kg bw/day.

[104] Figures presented in ng/kg bw/day.

[105] Estimate does not include breastfed infants.

[106] Data reported to the International Joint Commission on 12 breast milk monitoring studies from Ontario, Michigan, New York and Pennsylvania.

[107] Gobas, Frank A.P.C. *Selected Persistent Toxic Substance in Human breast Milk in the Great Lakes Basin*. Report of the International Joint Commission, (March 30, 1990), 94 pp.

[108] The TDI or Tolerable Daily Intake is an estimate of the amount of a chemical that when taken in on a daily basis,

pg TEQ[109]/kg bw/day for dioxin.[110] By comparison, the average adult 20 years of age or older takes in only 2% of the PTDI for PCBs and 12% of the TDI for dioxin.[111]

The concentration of PCBs and dioxins in breastmilk is considered an indicator of population exposure to theses contaminants by Health Canada[112] and is also relevant to determining the exposure of breastfed infants. Compared to other Ontarians and Canadians the general population in the Great Lakes basin is more exposed to PCBs. The Inuit of Northern Quebec are exceptional, however, in that their exposure is highest of all Canadians and among the highest globally.[113] Breast milk levels of dioxins and furans indicate that exposure is relatively uniform geographically for the general Canadian population.

It must be emphasized strongly that despite such high exposures at the start of one's life, breast feeding is still recommended as the optimum method of nourishing babies as the benefits of breast milk outweigh the risks from exposure to contaminants from breast milk.

Blood levels and other tissue levels of POPs have been assessed only for adults in Canada. These data come from several studies with different methodologies and study populations. Many have provided data on the levels in those who consume sport fish caught in the Great Lakes region. In general, among adults, fish consumers have relatively higher exposure to POPs compared to other controls (non-fish eaters) or the general population, although they are still less exposed compared to infants and children under age five.[114] Higher blood levels of POPs among sport fish consumers confirms the estimates of intake based on concentrations in all media.

2.5.3 Pesticides

Pesticides are chemical substances used to kill animal, insect, plant and fungal pests in agricultural and domestic applications. Spraying of pesticides, whether for crops, lawns, gardens or indoors allows for an effective route of human exposure via inhalation or ingestion. The primary route of exposure to pesticides however, is by skin absorption through direct contact with surfaces that accumulate pesticide particles.

Types

over the course of a lifetime, is presumed not to cause an appreciable risk to health. The TDI has replaced the former term, Acceptable Daily Intake (ADI) as more accurately representing our perception of the health effects from contaminants as being "tolerable" rather than "acceptable". It is important to note that international scientists recently agreed on revising the TDI for dioxins downward to a range of between 1 to 4 pg/kg/bw/day (WHO. 1998, *op.cit.*).

[109] Dioxins are of differing potency and TEQ or toxic equivalents, is a system of comparing the toxicities of different dioxins. The TEQ system expresses toxicity in terms of TCDD.

[110] Haines *et.al.*,. 1998a, *op.cit.*; and Haines *et.al.*,. 1998b, *op.cit.*

[111] *Ibid.* and Haines *et.al.*,. 1998a, *op.cit.*

[112] Health Canada. *Health-Related Indicators for the Great Lakes Basin Populations: Numbers 1 to 20*. Ministry of Public Works and Government Services, Canada. Cat. No. H46-2/98-219E. (1998a)

[113] *Ibid.*

[114] The degree to which sport fish consumers are more greatly exposed varies depending on where the fish are caught and the specific contaminant considered (Health Canada. *Persistent Environmental Contaminants and the Great Lakes Basin Population: An Exposure Assessment*. Health Canada. Minister of Public Works and Government Services, Canada. Cat. No. H46-2/98-218E. (1998b)).

Some of the pesticides that are toxic to humans and widely used, or present in the environment include, organochlorine, organophosphate and carbamate insecticides and chlorphenoxy herbicides used in weed removal. Organochlorine insecticides were banned for agricultural and commercial use in Europe and North America because of their environmental effects. However they are still used in developing countries and continue to be measured in the global environment because of their chemical stability and persistence. Until recently, Lindane (an organochlorine compound) was the active ingredient found in common medical treatments used against lice and scabies, such as Kwellada.

Health Effects

Studies on those with occupational exposure to pesticides, (i.e. farm workers, pesticide applicators, etc.) have provided considerable data on the effects in adults from acute, high dose exposure. Depending on the pesticide, exposure may be associated with irritation of skin, eyes and respiratory system. With more toxic pesticides, effects can be severe and involve the central nervous system.

Knowledge of the immediate and long term effects, from chronic, low-level exposure to pesticides is more inconsistent and controversial.[115] Of the non-occupationally exposed population, children are *potentially* the most exposed to pesticides compared to other age groups for a variety of reasons. There is a range of media and sources by which children may routinely come into contact with pesticides including: applications in their homes, yards, day care facilities, schools, parks, on family pets; via the residues in foods treated during agricultural application; and, secondarily via mother's milk. Children, because of their smaller size and greater exploratory and hand-to-mouth behaviour, are more likely to come into direct contact with and take in pesticide residues present in the environment. Lastly, physiologically speaking, children are generally more susceptible to the toxic effects of pesticides because of their immature stage of development.[116]

Organochlorine pesticides are of concern because of their ability to bioaccumulate in the environment and since they are associated with effects on neurological and behavioural development and the immune system. Observations of wildlife exposed to environmental levels of organochlorine pesticides have shown reproductive and developmental effects.

Organophosphate (OP) and carbamate insecticides are identified as "high risk" pesticides because several individual chemicals of these classes are relatively very toxic, and they often leave residues in foods that are consumed most by children.[117] Use of these pesticides during pregnancy and early infancy is believed to be associated with increased incidence of childhood cancers such as brain tumours and certain leukemias.[118] They may also have negative effects on the immune system, neurological development, reproduction and endocrine function.[119] Some pesticides may be associated with an elevated risk of various birth defects depending on timing of the exposure and the nature of the pesticide.[120] There is also

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[115] Steenland, K. Chronic neurological effects of organophosphate pesticides. *British Medical Journal*. 312 (1996), 1312-1313.

[116] City of Toronto, Public Health, Environmental Protection Office. *Pesticides: A Public Health Perspective*. (1998); and National Research Council. 1993, *op.cit.*

[117] Consumer's Union. *Worst First: High-Risk Insecticide Uses, Children's Foods and Safer Alternatives*. (Washington: Consumer's Union of U.S., Inc., September 1998)

[118] Daniels, Julie, L. Andrew, F. Olshan and D.A. Savitz. Pesticides and childhood cancers. *Environmental Health Perspectives* 105 (10) (1997), 1068-1077.

[119] City of Toronto. 1998, *op.cit.*

[120] Nurimen, Tuula. Maternal pesticide exposure and pregnancy outcome. *J. Occ. Env. Med*. 37 (1995), 935-940.

some evidence to suggest that acute exposures to pesticides may increase problems with fertility in both men and women[121] and data suggest that there is an increased risk of fetal deaths associated with pesticides, especially from the mother's exposure in agricultural activities.[122] The implications of pesticides for child health are further elaborated in Case Study #2.

Human Exposure Estimates

Recent U.S. figures using USDA and FDA food consumption and pesticide residue data, indicate that even with a fairly typical diet, there may be over a million children age 5 and under who are taking in amounts of OP pesticides beyond the EPA's adult reference dose.[123] The most recent data from monitoring for pesticide residues in the Canadian food supply revealed that 1.2% of domestic and 2% of imported fresh produce samples had levels that exceeded the Maximum Residue Levels (MRLs) set according to the Pest Control Products Act.[124]

Health Canada's Great Lakes Health Effects Program published detailed results of measures of certain priority persistent environmental contaminants in human tissues. There are several pesticides of the older organochlorine type, such as aldrin/dieldrin, hexachlorobenzene, DDT and DDE among others that have been detected in the blood, adipose tissue and breast milk of people living in the Great Lakes region. In most cases, the levels of these pesticides are on the decline and are not high enough to produce clinical symptoms, however, their persistence and bioaccumulative ability pose a concern for the health of children.[125]

2.5.4 Metals

Metal contaminants of concern to health include lead, cadmium, mercury, asbestos and aluminum, among others. Lead is a significant contaminant because of its persistence and because there has been widespread global exposure of children to lead from a variety of sources. Leaded gasoline emissions contributed to particularly high exposure in urban areas. With the removal of lead from gasoline in North America[126] this is now a historical pattern of exposure. However, because of lead's persistence and ability to bind to soil and dust particles, these represent significant continued sources of exposure in many areas.[127] Other important sources of lead exposure for children can be classified as either industrial and household. These include primary and secondary lead smelters and various industrial activities which result in lead emissions. In the home, there can be many sources of lead including old paint chips, dust, lead solder in plumbing and canned foods, cigarette smoke, and some consumer products such as toys

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[121] Curtis K.M., D.A. Savitz, C.R. Weinberg and T.E. Arbuckle. The effect of pesticide exposure on time to pregnancy. *Epidemiol*.10 (1999), 112-117.

[122] Arbuckle, T.E. and L.E. Sever. Pesticide exposures and fetal death: A review of the epidemiologic literature. *Critical Reviews in Toxicology*. 28 (1998), 229-270.

[123] Wiles, R., K. Davies and C. Campbell. *Overexposed: Organophosphate Insecticides in Children's Food. Environmental Working Group.* (January 1998) 54p. http://www.ewg.org/pub/home/reports/ops/download.pdf

[124] Eli Neidert and Glenn Havelock, CFIA. *Report on Levels and Incidences of Pesticide Residues in Selected Agricultural Food Commodities Available in Canada During 1994-1998.* (November 6, 1998.)

[125] Riedel, D., N. Tremblay & E Tompkins. *State of Knowledge Report on Environmental Contaminants and Human Health in the Great Lakes Basin*. Great Lakes Health Effects Program (Health Canada). (1997)

[126] In the late 1980s for the United States and as of January 1, 1990 for Canada.

[127] Mielke, H.W.. Lead in the inner cities: Policies to reduce children's exposures to lead may be overlooking a major source of lead in the environment. *Am Sci.* 87 (1998), 62-73.

and plastic mini-blinds[128]. Where homes have older plumbing there is also risk of lead contamination of drinking water and prepared foods. Exposure to lead via the dust pathway (whether from outdoors or from household items) may be the most important route in young children, especially those exhibiting pica and frequent hand-to-mouth behaviour.[129]

Health Effects

There is a great amount of data concerning the health effects from exposure to lead, with a good correlation between levels of exposure and concomitant health effects, especially at higher doses. Frank lead poisoning (blood lead levels above 80µg/dL) is characterized by severe effects on the nervous system, including muscle and abdominal pain, mental symptoms, paralysis, coma and death. Since lead inhibits the synthesis of hemoglobin, and increases the destruction of red blood cells, anemia may occur at blood lead levels between 20 and 40µg/dL. Blood lead levels below 20µg/dL are associated with neurocognitive effects such as IQ deficit, behavioural and learning problems.

Fortunately, with the elimination of leaded gasoline in Canada, acute lead poisoning is an extremely rare occurrence here in recent decades. However, there is continued and renewed concern for the possibility of subtle neurocognitive and developmental effects that can occur with exposure to low levels of lead. Low level lead exposure produces a syndrome of cognitive and attentional deficits; for example, a decrease in IQ, attentional problems, and poor social abilities. These cognitive and behavioural impairments lead to increases in academic failure, antisocial and extreme behaviour, and poor social adjustment, plus untold societal impacts such as, treatment costs, increased special education costs and reduced earning potential of the individual.[130]

There is no doubt that exposure to lead early in development has implications for later in life. Recently, attention is also being paid to delayed neurotoxic effects that do not manifest until adulthood, long after lead exposure has ceased. There appears to be some evidence to support previous speculation that early lead exposure may accelerate the aging process.[131]

The health effects from low-level exposure to lead in children are elaborated more thoroughly in Case Study #1.

Human Exposure Estimates

In 1991,the U.S. Centers for Disease Control revised the intervention level for blood lead in children downwards from 25 to 10µg/dL. At that time, it was recognized that there were still significant adverse health effects occurring from exposures at levels previously believed to be safe. However, there is still concern that this new level does not adequately represent a true threshold for neuro-developmental effects in children. Health Canada's Great Lakes Health Effects Program has also proposed considering blood lead in children as an important indicator for "monitoring progress or changes in human health as it relates to the Great Lakes environment."[132]

Data for blood lead levels from screening surveys in Ontario children indicate that over the decade from 1983 to 1992, there was a steady annual decline of 1.04µg/dL in blood lead levels, coinciding with the

[128] Please refer to Case Study #1 for a thorough discussion of sources of lead exposure.

[129] Roberts, J.W. and P. Dickey. 1995, *op.cit.*

[130] Rice, Deborah. Issues in Developmental Neurotoxicology: Interpretation and Implication of the Data. *CJPH*. 89 (Suppl 1) (1998), S31-36.

[131] *Ibid.*

[132] Health Canada. 1998a p.1, *op.cit.*

phase out of lead in gasoline.[133] The 1992[134] mean blood lead concentrations in Ontario children ages 1 to 5 was 3.11µg/dL which is similar to the means for U.S. (3.52µg/dL) and Britain (2.3µg/dL). While these average levels are below the current guidelines for prevention of health effects from lead, the actual distribution of blood lead levels indicates that a portion of children do have blood lead levels that are above or around the intervention level.

Health Canada[135] suggests that children are most likely to be exposed to lead from food, air and drinking water. They provide estimates[136] of daily exposures for children (ages one to four) of 1.1micrograms per kilogram of body weight (µg/kg.bw) from food, between 2 and 10µg from air and 2.9µg from drinking water.[137]

2.5.5 Air-borne Pollutants

Air-borne pollution is of concern because of the universality of human exposure. Air pollution is especially problematic in urban, industrialized areas, however, it also affects people in rural areas because of atmospheric transport of pollutants. Hence, there is the potential for large numbers of the general population, including children, to be exposed in the course of their everyday activities. There is a variety of environmental contaminants that are found in air, both indoors and outdoors.

Outdoor Air Pollutants

There are two major categories of outdoor pollutants:

1. Criteria Pollutants: The most important are the components of smog, ozone and particulates, particles less than 10 microns and 2.5 microns in size (PM_{10} and $PM_{2.5}$, respectively). Smog formation requires heat and sunlight, and smog levels are mainly a concern during the summer. Also in this group are nitrogen oxides, sulphur dioxide and carbon monoxide.

2. Air Toxics: This refers to the many chemicals that are measured at significantly lower levels than the criteria pollutants, and include chemicals such as benzene, VOCs (Volatile Organic Compounds) and PAHs (Polycyclic Aromatic Hydrocarbons). There are 40 such chemicals with regulatory limits in Ontario.

Indoor Air Pollution

The most significant indoor air pollutant in terms of health effects is unquestionably ETS (Environmental Tobacco Smoke) which will not be dealt with in this report.

Other important indoor air pollutants can be grouped into:

Biological - These include, house dust mites, moulds and allergens, from furry and feathered pets, and cockroaches. Moulds proliferate in damp conditions, and so quality of housing is an important variable.

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[133] Wang *et.al.* Decline in blood lead in Ontario children correlated to decreasing consumption of leaded gasoline, 1983-1992. *Clinical Chemistry*. 43 (1997), 1251-52.

[134] The most recent year for which results have been analysed. Wang *et.al.* (1997) *op.cit.*

[135] Health Canada. *The Health & Environment Handbook for Health Professionals*. Ministry of Supply & Services. Cat. No. H49-96/2-1995E. (1998c)

[136] These compare to daily figures for adult intake of 0.75µg/kg.bw from food, 2-10µg from air and 7.2µg from drinking water (Health Canada, 1998c, *op.cit.* Contaminant Profiles, Lead page 1).

[137] Health Canada. 1998c, *op.cit.,* Contaminant Profiles, Lead page 1)

Chemical - Numerous chemicals can be identified in indoor air, including carbon monoxide, and various volatile organic compounds. A vast array of contaminants, including heavy metals, pesticides and benzene enter the home in polluted regions in the air, on clothes and especially on footwear. The extent to which indoor air is polluted depends on the design of the building, the materials used to build, clean and furnish them and the way in which the space is ventilated, and maintained. Outdoor pollutants also penetrate into the indoor air in differing concentrations.

Health Effects

In this report, we look at the effects of outdoor air pollution, and will focus on the effects of smog. Ozone causes inflammation of the airways. The mechanism by which particulates cause harm is as yet unclear. Both ozone and particulates have been shown to have no threshold of effect, in relation to increasing hospital admissions. This demonstrates that they cause health effects even at low levels. Ozone exposure also makes asthmatics more responsive to allergens[138] and particulates are associated with an increase in infections of the respiratory tract.[139]

Exposure of children to smog air pollutants is associated with decreased lung function, increased respiratory symptoms such as sore throat and cough, and aggravation of asthmatic symptoms.[140] It has also been associated with increased hospital emergency visits and admissions, and increased school absences.[141] Smog has also been shown to lead to significant increases in mortality in the general population. A report of the Ontario Smog Plan attributes 1800 premature deaths a year in Ontario to particulates,[142] and data from Burnett and colleagues suggest that there are 5000 premature deaths a year from air pollution in 11 Canadian cities studied.[143]

These effects will not be seen to the same degree in all individuals. The very young and the elderly, those with immune and cardiorespiratory health problems, including asthma and chronic bronchitis and emphysema, smokers and people who work or are active outdoors are most affected by air pollution. Health effects from air pollution are similar for both adults and children,[144] although it is a widely held opinion that children may be at greater risk for these health problems because of greater exposure and susceptibility due to their immature lungs. Children with pre-existing respiratory disease including asthma are certainly at much greater risk from air pollution.[145]

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[138] Molfino NA, Wright SC *et.al.*, Effects of low concentrations of ozone on inhaled allergen responses in asthmatic subjects. *Lancet* 338(8761)199-203 (1991).

[139] Brunekreef B, Janssen NAH *et.al.* Air pollution from truck traffic and lung function from children living near motorways. *Epidemiology* 8,298-303 (1997).

[140] Bates DV The effects of air pollution on children. *Environmental Health Perspectives* 103 (Suppl 6):49-53 (1995).

[141] Raizenne M, *et.al.* 1996, *op.cit.*; Ontario Medical Association, 1998. *OMA Position Paper on The Health Effects of Ground-Level Ozone, Acid Aerosols and Particulate Matter*. www.oma.org/phealth/ground.htm; and Spengler J.D., P. Koutrakis, D.W. Dockery, M. Raizenne and F.E. Speizer, Health effects of acid aerosols on North American children: air pollution exposures. *Environmental Health Perspectives* May;104(5):492-9. (1996)

[142] Ontario, Ministry of Environment. Ontario smog plan: a partnership for collective action. Steering Committee Report (Jan 1998).

[143] Burnett, R.T., Cakmak, S, & Brook, J.R. The effect of the urban ambient air pollution mix on daily mortality rates in 11 Canadian cities. *Can J Pub Health* 89: 152-156 (1998).

[144] Dr. David Pengelly, personal communication, e-mail message. (April 6, 1999).

[145] *Ibid.*

There have been a number of studies that have examined the effects of air pollution specifically on children's health. A comparative study of 7 to 11-year old children from rural communities in Saskatchewan and Ontario showed that while exposure to moderate levels of ozone and sulfate (Ontario) did not produce significant differences in respiratory ailments, such exposure was associated with statistically significant decreases in lung function.[146] A combined analysis of the effects of exposure to ozone among children at summer camp provides strong evidence for decreases in forced expiratory volume with increasing levels of ozone.[147] Studies of both U.S. and Canadian children have shown that those living in areas where exposure to acidic air pollution or ozone was high had more frequent episodes of certain adverse respiratory symptoms such as bronchitis.[148] These researchers speculate that long-term exposure to acid aerosols may adversely affect lung growth, development and function.[149] Increased respiratory hospitalizations in very young children (< 2 years old) have been reported to be associated with ambient concentration of pollutants to a greater degree than adults.[150]

Recent experimental and observational studies have indicated that exposure to various pollutants may cause negative health effects beyond those already discussed. For instance high levels of air pollution have been linked to damage to DNA in alveolar macrophages[151] and nasal respiratory epithelium which may ultimately result in the development of precancerous cells in these tissues.[152] Exposure to higher levels of carbon monoxide in late pregnancy may be associated with significantly increased risk of low birth weight[153] and there is some evidence to suggest that early maternal exposure to high particulate levels carries greater odds of intrauterine growth retardation.[154] The health effects of air toxics include reproductive effects, as well as cancer. Indoor air pollutants can also contribute to respiratory problems and allergies.

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[146] Stern, B.R., M.E. Raizenne, R.T. Burnett, L. Jones, J. Kearney and C.A. Franklin. Air pollution and childhood respiratory health: Exposure to sulfate and ozone in 10 Canadian rural communities. *Environ Res*. 66: 125-142. (1994).

[147] Kinney P.L., G.D.Thurston and M. Raizenne, The effects of ambient ozone on lung function in children: a reanalysis of six summer camp studies. *Environmental Health Perspectives* Feb;104(2):170-4. (1996)

[148] Dockery D.W., J. Cunningham, A.I., Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne and F.E. Speizer, Health effects of acid aerosols on North American children: respiratory symptoms. *Environmental Health Perspectives* May;104(5):500-5 (1996); and Galizia A. and P.L. Kinney, Long-term residence in areas of high ozone: associations with respiratory health in a nationwide sample of nonsmoking young adults. *Environmental Health Perspectives* Aug, 107(8):675-9. (1999)

[149] Raizenne M., L.M. Neas, A.I. Damokosh, D.W. Dockery, J.D. Spengler P. Koutrakis J.H. Ware, and F.E. Speizer, Health effects of acid aerosols on North American children: pulmonary function. *Environmental Health Perspectives* 1996 May;104(5):506-14. (1996)

[150] Burnett R.T. *et.al.,* Effects of low ambient levels of ozone and sulfates on the frequency of hospital admissions to Ontario Hospitals. *Environ Res* 65:172-94 (1994).

[151] Macrophages are cells that play an important role in the immune response by presenting foreign cells to lymphocytes for antibody production and, in the case of the alveolar types, by actively consuming microbes themselves.

[152] Bermudez E; Ferng SF; Castro CE; Mustafa MG. DNA strand breaks caused by exposure to ozone and nitrogen dioxide. *Environ Res*, Jul, 81(1):72-80. (1999); and, Calderon-Garciduenas L., L. Wen-Wang, Y.J. Zhang, A. Rodriguez-Alcaraz, N. Osnaya, A.Villarreal-Calderon and R.M. Santella, 8-hydroxy-2'-deoxyguanosine, a major mutagenic oxidative DNA lesion, and DNA strand breaks in nasal respiratory epithelium of children exposed to urban pollution. *Environmental Health Perspectives*, Jun, 107(6):469-74. (1999)

[153] Ritz B; Yu F. The effect of ambient carbon monoxide on low birth weight among children born in southern California between 1989 and 1993. *Environmental Health Perspectives*, Jan, 107(1):17-25. (1999)

[154] Dejmek, J. SG Selevan, I. Benes, I Solanski and RJ Sram. Fetal growth and maternal exposure to particulate matter during pregnancy. *Environmental Health Perspectives*, June 107(6): 475-480. (1999)

Exposure Levels

Outdoor pollution levels are at their worst on summer days when high temperature, sunlight and low air movement create the necessary environmental conditions for production of smog. This is a particular concern for children as they are most active outdoors during summer months. The pollutants forming the smog that covers Southern Ontario come primarily from local sources, however there is also significant contribution to ozone, acid aerosol and particulate levels here from pollutants that originate in the U.S. midwest.[155]

The current national objective for air quality is assessed as 82 ppb ozone. The highest levels of ozone in Canada are found along the Windsor - Quebec corridor, the country's industrial interior.[156] Therefore, the population in Southern Ontario is particularly exposed to bad air quality. When ozone levels are predicted to exceed 82 ppb Environment Canada issues air quality advisories that warn the public about the elevated health risks from exposure to smog. Environment Canada's data show that air quality advisories have occurred more frequently in these same regions (Southern Ontario and Quebec) compared to most other areas of the country.

Health Canada researchers speculate that there may be no threshold for ozone concentration below which no adverse health effects are observed.[157]

2.5.6 Summary

While this is not intended to be an exhaustive outline of the many chemicals present in our environment, it highlights a few key contaminants that are raising concerns regarding children's environmental health.

Persistent organic pollutants, like PCBs, dioxins and furans, are chemicals that are widespread in the environment because they decay gradually and bioaccumulate in the tissues of living organisms, including humans. They are implicated in many health effects in humans, including endocrine disruption, neurodevelopmental and immune system effects and cancer. There are many gaps and uncertainties in the science as yet, while a huge research effort is in progress. Levels of POPs continue to be detected in human tissue samples, including breast milk, in the Great Lakes region.

Pesticides are toxic to living organisms by design. They include several classes of compounds that are used for different purposes, many of which mean people are exposed to them involuntarily either through air, dust, food or objects in the environment. Individual pesticides differ in their toxicity to humans but there appears to be a wide range of potential or actual adverse health effects that include reproductive and neurodevelopmental effects, cancer, immune system effects.

Lead is a persistent metal whose dispersion through the environment is due mainly to human activities. Exposure to lead is via air, soil, dust, food drinking water, old paint, consumer products and across the placenta. Since the elimination of lead from gasoline, there has been a significant decrease in exposure to lead, however, there continue to be sources that may bring people, especially children, into involuntary contact with lead. Lead's effects on health are most notable in children because they absorb lead more readily, their developing systems are more sensitive and effects occur at much lower levels of exposure

[155] Ontario Medical Association, 1998. *OMA Position Paper on The Health Effects of Ground-Level Ozone, Acid Aerosols and Particulate Matter*. www.oma.org/phealth/ground.htm; and Spengler J.D. *et.al.*, 1996, *op.cit.*

[156] Ontario Medical Association. 1998, *op.cit.*

[157] Health Canada. Great Lakes Health Effects Program - GHLEP. *Outdoor air and your health: A summary of research related to the health effects of outdoor air pollution in the Great Lakes Basin.* Air Quality Health Effects Research Section, Environmental Health Directorate, Health Canada. (March 1996).

than in adults. Estimates from population studies of blood lead levels and from concentrations of lead in Ontario's food, air and water suggest that there should be continued vigilance concerning the potential for lead exposure.

Major pollutants of outdoor air include smog and ground-level ozone, fine particulate matter, nitrogen oxides, sulphur dioxide and carbon monoxide. These same pollutants may be present in indoor air. Other important indoor air contaminants include both biological (moulds, fungi and allergens from pets) and chemical (environmental tobacco smoke, volatile organic chemicals and carbon monoxide among others). Significant numbers of Ontario children with asthma suffer exacerbations which keep them from school, or send them to their doctors or emergency rooms, related to elevated levels of smog. Long-term exposure to high levels of pollutants is associated with effects on lung function. Exposure to contaminants in air is associated with a spectrum of effects on health. Populations living in areas of Ontario that are heavily industrialized or are in the region of transport of air contaminants are exposed to significant levels of pollutants.

2.6 HEALTH PROBLEMS RELATED TO ENVIRONMENTAL EXPOSURES

2.6.1 Introduction

Despite significant improvements in children's health around the world, fewer children dying of infectious diseases and malnutrition, a new face to childhood illness is emerging. In both developed and developing countries, the childhood burden of illnesses influenced by the environment is gaining greater attention. Landrigan and colleagues recently characterized this shift as "the new pediatric morbidity" referring to the rising incidence of conditions that are "known or suspected to be of toxic environmental origin."[158] Many different environmental contaminants have been implicated. These have differing effects upon pediatric physiology and hence, lead to different health effects. This section will review research into these known (or possible) health effects that may be associated with exposure to various environmental contaminants. These effects are also important as indicators relevant to monitoring the effects of toxic exposures on children's health.

2.6.2 Spontaneous Abortion, Stillbirth Rates

It is difficult to link spontaneous abortion and stillbirths directly to a specific environmental exposure since often women may not realize[159] they are pregnant at critical developmental periods when they might be exposed.

Much of the knowledge of environmental effects on reproductive outcome stem from studies of acute toxic exposures, frequently in the occupational setting. For example, high rates of spontaneous abortion occurred in mothers accidentally exposed to PCB contaminated oil in Taiwan and Japan; hairdressers and dry cleaners may have higher rates of early miscarriage due to exposure to solvents; and, maternal agricultural occupation and exposure to pesticides may be associated with higher risk of spontaneous abortion and stillbirth. Some of the environmental factors associated with adverse reproductive outcome

[158] Landrigan, P.J. *et.al.* Children's health and the Environment: A new agenda for prevention research. *Environmental Health Perspectives*. 106 (Suppl 3) (1998), 788.

[159] A large percentage of all embryos are lost within the first trimester of pregnancy (30-50% of all conceptions going undetected.) Up to 50% of early spontaneous abortions occur due to abnormal chromosome complement which occurs with increasing age of mother although the exact factors causing this are not well understood. The vast majority of early spontaneous abortions are of unknown cause.

include metals, such as lead, solvents, such as toluene and ethylene glycol ethers, pesticides and PCBs, and ionizing radiation (X-rays).

A recent study of over 5,000 women showed a greater risk of spontaneous abortion occurring in women who drank 5 or more glasses of tap water per day where they were consequently exposed to levels of disinfection by-products as indicated by trihalomethanes in excess of 75 micrograms per liter total.[160]

2.6.3 Congenital Malformations

Generally the fetus (and preconceptionally, a woman's eggs) is most vulnerable to chemical exposures. The first trimester when organogenesis and formation of major body structures occur represents the most vulnerable and sensitive period as described above, and can most often result in congenital abnormalities from environmental exposures.

For example, first trimester exposure of mothers to the pesticide benomyl has been implicated in several dozen Canadian, American and British cases[161] where babies had congenital anopthalmia or microphthalmia[162] indicating the very specific effects of that particular chemical on eye development *in utero*.

Exposure to pesticides both environmentally or due to occupation has also been associated with other congenital anomalies in offspring. For example, studies suggest elevated risk of limb anomalies, orofacial clefts[163] and hypospadias[164] and cryptorchidism[165] (in male infants).[166]

Based on a prospective study of 125 pregnant women, researchers from Toronto's Hospital for Sick Children conclude that those who were occupationally exposed to organic solvents during pregnancy had a 13-fold risk of fetuses developing major malformations[167] compared to controls.[168]

Researchers in the U.S. are currently looking at the possible role of environmental chemical exposures to explain the recent dramatic increase in neural tube defects (including anencephaly and spina bifida) in the Rio Grande Valley region of south Texas.[169]

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[160] Waller, K. *et.al.* Trihalomethanes in drinking water and spontaneous abortion. *Epidemiology*. 9 (1998), 134-40.

[161] See Dyer, Clare. U.S. court case starts over eyeless babies. *BMJ.* 312 (1996), 1247.

[162] Anophthalmia is absence of eyes. Microphthalmia is abnormally small eyes.

[163] Nurimen, T. 1995*, op.cit.*

[164] Hypospadias refers to malformations of the male genital tract.

[165] Cryptorchidism refers to the phenomenon of undescended testes.

[166] Weidner I.S., H. Moller, T.K. Jensen and N.E. Skakkebæk. Cryptorchidism and hypospadias in sons of gardeners and farmers. *Environmental Health Perspectives*. 106 (1998), 793-6.

[167] Khattak and colleagues define major malformations as, "any anomaly that has an adverse effect on either the function or the social acceptability of the child" (Khattak, S., G.K. Moghtader, K. McMartin, M. Barrera, D. Kennedy and G. Koren. Pregnancy outcome following gestational exposure to organic solvents: a prospective controlled study. *JAMA*. 281(12): 1106-9 (1999: 1107).

[168] *Ibid.*

[169] Haynes, R.C. A tradition of focusing on children's health. NIEHS News. *Environmental Health Perspectives* 106 (1998), A14-16.

2.6.4 Neurodevelopmental, Behavioural Effects

Developmental neurotoxicity and behavioural effects are of particular concern as more is learned about the nature of more subtle degrees of impairment that can occur with even low level exposures. The window of susceptibility to neurotoxic effects is broad because of the extensive age-related development that the brain and nervous system undergoes from fetal stage, through childhood and into adolescence.

Lead provides the best-documented example of a contaminant that causes neurotoxic developmental effects, especially at low levels of exposure. Acute lead poisoning (associated with blood lead levels in children above 80µg/dL) damages the peripheral nervous system, causing severe health effects such as muscle and abdominal pain, mental symptoms, paralysis, coma and death. There is a continuum of neurotoxic symptoms in children that are linked with blood lead levels below 80µg/dL.

The picture at lower levels of exposure is different. For blood lead levels between10 and 40µg/dL, the associated neurotoxic effects are "clinically invisible", however, we recognize their significance nonetheless. They are characterized by overall dysfunction of the central nervous system including developmental deficits that can be observed as lowered IQ, behavioural problems and poor performance in school. It has been determined that in children under age four who are exposed to lead, there is about a two point decrease in IQ with an increase in blood lead from 10 to 20µg/dL.[170] U.S. researchers are looking at the possible role of low dose lead exposure in contributing to attention deficit hyperactivity disorder (ADHD) in school age children.[171]

While the blood lead level of concern, set by the Center for Disease Control in the U.S., has dropped from 25 down to 10µg/dL,[172] it has recently come to light that there is no real "threshold" for neurotoxic effects, as they can be demonstrated down to blood lead levels of 1µg/dL.[173]

While the neurotoxic effects from heavy metals such as lead and methylmercury have been well characterized, there are fewer data on the potential for neurotoxic effects from other environmental contaminants. There is significant concern based on epidemiological and laboratory data, that early exposure to PCBs impairs neurological development and can lead to developmental deficiencies and learning disabilities in the young.[174]

Endocrine disruptors such as PCBs are being shown to affect thyroid function[175] and thyroid hormones are vital to proper development of various brain functions involved in learning and memory.[176]

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[170] Needleman, H.L. and C. Gatzonis. Low level lead exposure and the IQ of children. *JAMA*. 263 (1990), 673-78.

[171] Haynes, R.C. 1998, *op.cit.*

[172] Above this level, there are detectable decreases in IQ.

[173] Rice, D. 1998, *op.cit.*

[174] Jacobson & Jacobson. 1996, *op.cit.*; and Lonky, E., J. Reihman, T. Darvill, J. Mather and H. Daly. Neonatal behavioural assessment scale performance in humans influenced by maternal consumption of environmentally contaminated Lake Ontario fish. *J. Grt. Lakes Res*. 22 (1996), 198-212.

[175] Sher E.S., X.M. Xu, P.M. Adams, C.M. Craft and S.A. Stein, The effects of thyroid hormone level and action in developing brain: are these targets for the actions of polychlorinated biphenyls and dioxins? *Toxicol Ind Health* 14 (1998), pp. 121-58; and, Brouwer A *et.al.* Characterization of potential endocrine-related health effects at low-dose levels of exposure to PCBs. *Environmental Health Perspectives* 107 (1999) Suppl 4, pp. 639-49.

[176] Porterfield, S.P. Vulnerability of the developing brain to thyroid abnormalities: Environmental insults to the

There has recently been an important shift in understanding the impact of neurotoxic effects from subtle exposures. There is increased recognition that so-called "small" changes in function may still have "far-reaching consequences" and thus, "a small effect is not necessarily an unimportant effect."[177]

2.6.5 Growth

Physical growth comprises the overall structural and compositional changes that occur in the body. It is a complex, regulated phenomenon effected by growth processes that occur from the cellular up to the system levels. These component parts undergo growth at characteristic rates and times. The times of most rapid growth of the individual occur prenatally and during infancy and adolescence. While much of human growth is determined by genetics, a substantial influence on growth patterns comes from environmental effects and it is during these periods of rapid growth that there is greatest effect from environmental toxicants.[178] In particular, because growth in the womb and during infancy involves considerable cell proliferation (as opposed to later growth which mainly involves increase in the *size* of cells) effects on growth prior to age 2 are more likely to result in permanent reduction in body size that is seen in adulthood.

A review of population studies has demonstrated that exposure to various compounds (e.g. lead, PCBs, noise) can alter and delay normal growth both prenatally and postnatally.[179] Importantly, as with neurotoxic effects, growth impairment also appears to occur from exposure to levels that are otherwise too low to produce acute toxicity.[180]

Longitudinal and cross-sectional studies of lead exposure in U.S. children indicate that reduced birth weight and linear growth retardation are common effects of lead exposure. Prenatal lead exposure may also be the source of growth inhibition at later stages of childhood. Exposure to high levels of PCBs during pregnancy is also associated with significantly reduced birth weight and gestational weight for age. Mixtures of chemicals are also implicated in growth alteration. A new Jersey study demonstrated that infants whose parents resided near a hazardous waste landfill site had significantly lower birth weight.[181]

While the evidence is less clear, numerous population studies of the relationship between air pollution and growth effects appear to support the conclusion that even when socioeconomic factors are controlled for, juvenile growth is generally poorer in urban areas with greater air pollution.[182]

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thyroid system. *Environmental Health Perspectives*. 102 (Suppl 2) (1994), 125-130.

[177] Rice. 1998, *op.cit.*

[178] Karlberg, J. On the construction of the infancy-childhood-puberty growth standard. *Acta. Pediatr. Scand. Suppl.* 356 (1989), 26-37.

[179] Schell, L.M. Effects of pollutants on human prenatal and postnatal growth: Noise, lead and polychlorobiphenyl compounds and toxic wastes. *Ybk Phys Anth*. 34 (1991), 157-188; and Schell, L.M. Pollution and human growth: lead, noise, polychlorobiphenyl compounds and toxic wastes. In: *Applications of Biological Anthropology to Human Affairs.* CGN. Mascie-Taylor & G. W. Lasker (Eds.) (Cambridge: Cambridge University Press, 1992), pp. 83-116.

[180] Schell. 1991, *op.cit.*

[181] Berry, M. and F. Bove. Birth weight reduction associated with residence near a hazardous waste landfill. *Environmental Health Perspectives*. 105(8) (1997), 856-861.

[182] Schell. 1991, *op.cit.*

2.6.6 Immunological Effects

The immune system is the body's defense against infection from foreign agents, but it may also play a role in containing malignant cells and thereby resisting tumour formation and cancer. There is much uncertainty and less known about the immune system effects from exposure to environmental contaminants in humans. Two main immune system effects may be associated with exposure to toxins: 1) immune sensitization or heightened function, may allow for development of allergic reactions to antigens; and 2) immune suppression may render the individual more susceptible to infections and cancer. There is also some speculation (despite little scientific evidence) that certain autoimmune disorders, where the immune system fails to distinguish between self-cells and foreign cells, may be associated with environmental exposures.[183]

Regarding the immuno-suppressive effect, several studies have indicated an increase in incidence of infectious illnesses in the children of women who were exposed to high doses of PCBs either through accidental or occupational exposure.[184] Incidence of respiratory and sinus infections, gastrointestinal and dermatological symptoms, was pronounced from both prenatal (transplacental) and postnatal (via breast milk) exposure to high levels of PCBs. These symptoms appear to have been confined to the earliest stages of life. Altered T cell function has also been associated with exposure to PCBs pre- and post-natally[185] in both acute and low-level exposures.

While the exact immunological effects from pesticides are not well known, it is believed that they may potentially be immunotoxic to humans. For example, *in vitro* laboratory experiments have demonstrated that carbaryl, a carbamate pesticide, suppressed natural killer cells that are critical in combating cancers such as leukemia and lymphomas.[186] Other compounds from which there are presumed immunosuppressive effects, despite incomplete knowledge of the precise causal mechanisms, include: air pollutants such as, ozone, nitrous oxides, environmental tobacco smoke; and, metals such as, cadmium, lead and mercury. For example, animal studies have shown an association between cadmium exposure and malignancies.

Heightened immune response due to contaminant exposure can lead to hypersensitivity and allergic reactions. Asthma and allergies are immediate hypersensitivity reactions that can be provoked by exposure to certain organic compounds such as, isocyanates, freons, amines, anhydrides and some metals such as platinum. Medical case reports also suggest that pesticides may be immune sensitizers for some individuals. Skin irritation or dermatitis is a delayed-type hypersensitivity response and it can occur after exposure to certain pesticides, metals, rubber compounds and chemicals such as formaldehyde. The immaturity of the young immune system appears to be somewhat protective of sensitization in very young children, however, as children achieve greater immune competence with increasing age, the hypersensitivity response can be more readily induced.

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[183] Szentivanyi, A. *et.al.* Environmental immunotoxicology. In: *Environmental Medicine*. Brooks, Stuart M. *et.al.* (eds). (St. Louis: Mosby, 1995), pp. 139-155.

[184] Hara, I. Health status and PCBs in blood of workers exposed to PCBs and their children. *Environmental Health Perspectives.* 59 (1985), 85-90.

[185] Weisglas-Kuperus, N. *et.al.* Immunologic effects of background prenatal and postnatal exposure to dioxins and polychlorinated biphenyls in Dutch infants. *Pediatric Research*. 38 (1995), 404-410.

[186] City of Toronto, 1998, *op.cit.*

2.6.7 Asthma & Respiratory Diseases

A variety of contaminants, in both indoor and outdoor air, is associated with respiratory problems in children. The observed adverse respiratory health effects range from subtle, non-specific symptoms such as sore throat and redness to increased cough and wheeze, increased use of asthma medication, increased rates of asthma attacks, increased physician and hospital respiratory emergency visits and hospital admissions, permanent reduction in lung capacity and an increased risk for Sudden Infant Death Syndrome (SIDS). The "Health Effects Pyramid" (see Figure 2.3 below) illustrates how with increasing severity of symptom, there is an increasingly smaller proportion of the population that is affected. In other words, fewer people die from the effects of air pollutants, however, many more people do suffer some degree of respiratory impairment or illness.

Asthma is of greatest concern. In urban America, children are more likely to be hospitalized for respiratory problems, especially asthma, than due to any other cause.[187] There have been substantial increases in asthma prevalence, morbidity and mortality, in children of industrialized and industrializing nations, beginning in the 1970s. In Canada, figures for asthma prevalence among the young indicate that there has been a more than fourfold increase in the numbers of children under age 15 afflicted with asthma over the last 15 years.[188] The reasons for this increased asthma prevalence are not fully understood but it may reflect both environmental determinants and an increase in susceptible individuals with a westernized lifestyle. Outdoor air pollution appears to be more important as a risk factor that *worsens* existing disease and/or triggers symptoms, rather than as an explanation of *new* asthma cases.[189] As such, asthmatic children represent a particularly sensitive subgroup of children with respect to exposure to air pollutants.

Air quality, both indoors and outdoors, seems to be contributing to a higher burden of illness from asthma. New cases of asthma in children are likely *initiated* by exposure to factors such as indoor air allergens, including house dust mites, cats, cockroaches and molds. Exposure of children has increased as houses have become more air tight. Environmental factors that exacerbate asthma are ground-level ozone, particulates and acid aerosols.

Epidemiological studies do show clear associations between episodes of high air pollution and subsequent hospital visits for respiratory problems.[190]

[187] Gottlieb, D.J., A.S. Beiser and G.T. O'Connor. Poverty, race and medication use are correlates of asthma hospitalization rates: a small area analysis of Boston. *Chest*. 108 (1995), 28-35.

[188] Miller, Wayne and Garry B. Hill. Childhood asthma. *Health Reports*. Winter 10(3) (1998), 9-21. Statistics Canada, Catologue No. 82-003.

[189] Dockery *et.al.* 1996, *op.cit.*; and Becklake, M.R. and P. Ernst. Environmental factors. *Lancet*. 350 (Suppl ii) (1997), 10-13.

[190] Burnett *et.al.* 1994, *op.cit.*

There is evidence that lung growth, development and function may be compromised from longterm

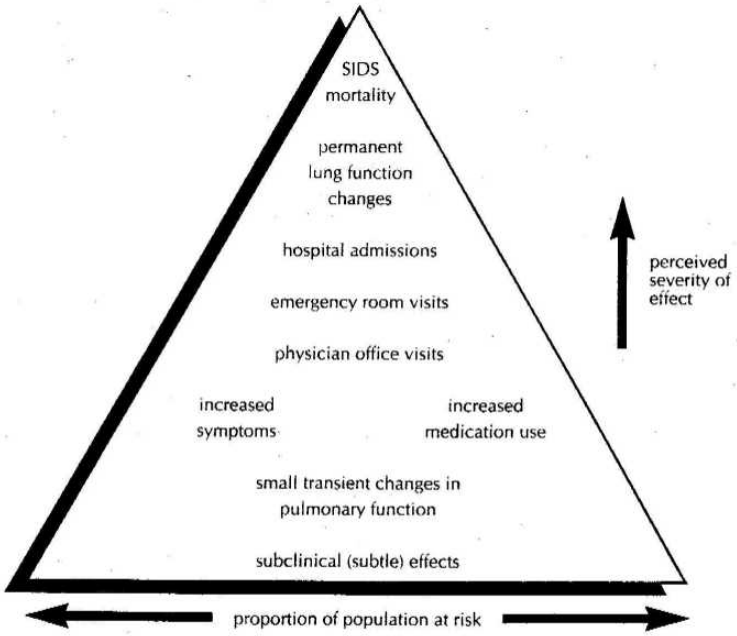

Figure 2.3. The Health Effects Pyramid.
(Source: Raizenne, M. *et.al.* Air pollution exposure and children's health. *CJPH*. 89 (Suppl 1) (1998), S44.)

exposure to acid aerosols, even at moderate levels.[191] Children exposed to higher levels of air pollutants are more likely to report episodes of bronchitis.[192] There is a suggestion that the above effects from air pollution may predispose children to developing other chronic respiratory illness or put them at higher risk of ill effects from other environmental exposures later in life.[193]

There is a need for further research that examines the links between children's respiratory illness and indoor and outdoor air quality. It is unlikely that outdoor air pollutants actually cause asthma, but the research is clear that air pollutants, especially smog, exacerbate asthma, leading to a significant burden of illness among Canadian and Ontario children and adolescents.

2.6.8 Reproductive & Endocrine Effects

The body's endocrine system produces hormones that are chemical messengers involved in regulation of a variety of body functions including, reproduction, the immune system and growth. Normally hormones fit into specific protein receptors on the surface of cell membranes in a "lock and key" fashion. The

[191] Raizenne, M. *et.al.* Air pollution exposures and children's health. *CJPH*. 89 (Suppl 1) (1998), S43-S48; and Stern, B.R., M.E. Raizenne, R.T. Burnett, L. Jones, J. Kearney and C.A. Franklin, Air pollution and childhood respiratory health: Exposure to sulfate and ozone in 10 Canadian rural communities. *Environ. Res*. 66 (1994), 125-42.

[192] Dockery *et.al.* 1996*, op.cit.*

[193] *Ibid.*

binding of a hormone to its specific receptor then prompts a cascade of biochemical responses that characterize normal endocrine functioning.

Endocrine disrupters are chemicals that, by virtue of some structural similarity to normal hormones, can also bind to the receptor sites. However, once bound, these chemicals do not elicit the normal biochemical response; they alter the activity of the endocrine system, either by mimicking (enhancing) or by blocking (inhibiting) normal hormonal functions. Figure 2.4 below illustrates the usual ways in which endocrine disrupters may interfere with hormone function.

Many different candidate contaminants have been judged to act as endocrine mimics or inhibitors.[194] The area of research that concerns hormone disrupters is still relatively limited in terms of what we can say are the effects on reproductive and endocrine development in humans.

Evidence of effects comes from three main sources:
1) observations of wildlife; 2) laboratory studies of animals and cell cultures; and 3) epidemiological observations in humans.

[194] Among these are many man-made compounds such as; 1, 1-dichloro-2,2-bis (p- chlorophenyl) ethane (p,p '-DDE), DDT, TCDD, Vinclozolin, PCBs, PCDFs, PCDDs, toxaphene, chlordane, kepone, hexachlorobenzene (HCB), methoxychlor, Bisphenol-A, phthalate esters; and some naturally occurring compounds like lead, mercury, phytoestrogens (as cited in Foster, Warren. Endocrine Disruptors & Development of the Reproductive System in the Fetus and Children: Is there Cause for Concern? *CJPH.* 89 (*Suppl* 1) (1998), S37-41, S52).

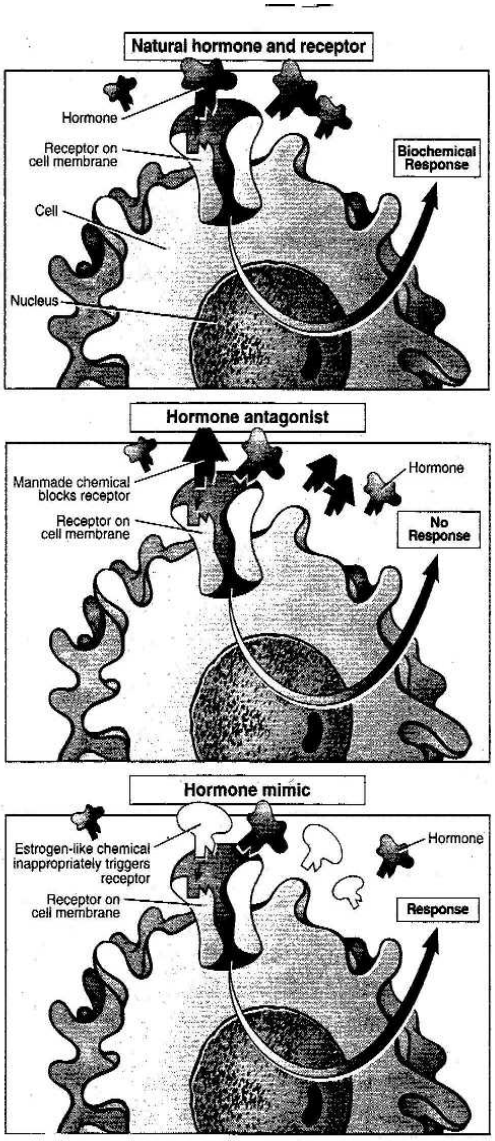

Figure 2.4. The mechanisms of endocrine disruption.
(Source: CALPIRG and PSR, 1998, *op.cit.*)

1) The effects of some of these contaminants have been well characterized in studies of wildlife populations (mainly birds, reptiles and fish) in heavily polluted areas.[195] They include ambiguous and/or congenitally malformed genitalia (producing masculinized females and feminized males), impaired fertility, abnormal reproductive development and behaviour. Observed effects also include, abnormal thyroid and immune system function and increased incidence of reproductive cancers.

2) Experimental data, exposing rats to various estrogenic and antiestrogenic compounds *in utero* and during lactation, have also demonstrated similar types of effects. *In vitro* laboratory data have demonstrated abnormal growth in human cells exposed to certain of these endocrine disrupters.[196]

   Vom Saal and colleagues have demonstrated the sensitivity of the developing rodent reproductive tract to even subtle alterations in hormone homeostasis. In the normal rodent fetus, intrauterine position, which determines hormone exposure, has an influence on subsequent behaviour, physiology and anatomy of the individual.[197] For example, female mouse fetuses who developed between two male fetuses thereby experiencing greater exposure to testosterone, were more aggressive, less able to attract mates, reached puberty later and had fewer cycles of heat.

   Animal models are also indicating that low level effects of PCBs may include disruption of thyroid hormone functioning that can directly impair brain development and functioning.[198]

[195] Colborn, T., D. Dumanoski and J.P. Myers. *Our Stolen Future*. (New York: Penguin, 1996)

[196] As found in: Soto A.M., K.L. Chung and C. Sonnenschein. The pesticides endosulfan, toxaphene, and dieldrin have estrogenic effects on human estrogen-sensitive cells. *Environ. Health Perspec*. 102 (1994), 380-83.

[197] vom Saal, F. The intrauterine position phenomenon: Effects on physiology, aggresive behavior and population dynamics in house mice. In: *Biological Perspectives on Aggression*. K. Flannelly, R.J. Blanchard and D.C. Blanchard (eds.) *Progress in Clinical and Biological Research*. v. 169. (New York: A.R. Liss, 1984), pp. 135-79.

[198] Sher *et.al.* 1998, *op.cit.*; and Brouwer *et.al.* 1999, *op.cit.*

3) The epidemiological evidence that shows clear causal relationships between exposure to hormone disrupters and reproductive effects is limited at best. The evidence of effects from the synthetic estrogen, Diethylstilbestrol (DES), given to pregnant women in the 1940s to 1970s to prevent spontaneous abortion, has served as a model for the potential reproductive effects from man-made estrogenic chemicals. Prenatal exposure to DES has been clearly linked to impaired reproductive function (including malformations and tumours) in adulthood among the exposed offspring. While DES is indicative of the effects of endocrine disruption *in utero*, it is not clear how well this predicts effects from exposure to environmental contaminants, as the potency of many of these ambient chemicals is assumed to be much less than either DES or endogenous estrogen.

   There have been marked trends observed in increased incidence of hypospadias and cryptorchidism, decreasing age at menarche[199] and increased infertility, occurring over the last several decades in industrialized western nations.[200] Although still being debated, there is concern that sperm counts may be declining as well. [201] These trends coincide with and are believed to be linked to the increased presence of culprit endocrine disrupters in the environment and as measured in human tissue samples from these populations.

   Ecological[202] epidemiology studies have shown an association between possible excess exposure to pesticides by place of residence[203] or by virtue of parental occupation (sons of gardeners and farmers)[204] and higher incidence of cryptorchidism and/or hypospadias.

All of these data taken together have led to a biologically plausible model of potential effects on human reproductive development even at low exposure levels. However, this hypothesis has not been confirmed and the "biological significance of the findings (above) for humans has yet to be established."[205] As such, there has been a call for increased epidemiological and experimental research to substantiate the hypothesized endocrine disruptive effects of environmental chemicals in humans. This is currently recognized as an important issue of concern in the protection of pediatric environmental health.

2.6.9 Cancer

Cancer is not a single disease but represents different diseases of varying etiology and causal mechanisms. Recognized mechanisms by which environmental contaminants can cause cancer include:

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[199] First menstrual period.

[200] Foster. 1998, *op.cit.*; and Klotz L.H., Why is the rate of testicular cancer increasing? *CMAJ*. 160 (1999), 213-4.

[201] For example, Canadian researchers have found regional differences and trends of both decline and increase in sperm concentration. See Younglai E.V., J.A. Collins & W.G. Foster, Canadian semen quality: an analysis of sperm density among eleven academic fertility centers. *Fertil. Steril*. 70 (1998), 76-80.

[202] Epidemiological studies that examine and compare disease rates in different groups and look for associations between environment or other group factors that might explain variation in rates from one group to another. Ecological studies might also compare time trends and look for changes in exposure among various groups that may correlate with observed changes in disease rates.

[203] Garcia-Rodriguez J., M. Garcia-Martin, M. Nogueras-Ocana, *et.al.* Exposure to pesticides and cryptorchidism: Geographical evidence of a possible association. *Environmental Health Perspectives*. 104 (1996), 1090-95.

[204] Weidner I.S., H. Moller, T.K. Jensen and N.E. Skakkebæk. Cryptorchidism and hypospadias in sons of gardeners and farmers. *Environmental Health Perspectives*. 106 (1998), 793-6.

[205] Foster. 1998*, op.cit.,* p. S39.

1) Genotoxicity - DNA mutations are produced that alter cell properties (i.e. the contaminants act as cancer initiators).

2) Cancer promotion - tumour production is accelerated.

3) Immunotoxicity - immunosuppression occurs disrupting the body's ability to eliminate cancer cells.

4) Peroxisome proliferation - production of this process encourages development of cancer.

The etiology of childhood cancer is not well understood, but greater exposure to environmental contaminants (particularly at tissue-specific windows of vulnerability) is a potential factor associated with its appearance.

Childhood cancers are relatively rare (in epidemiological terms) and therefore, difficult to study in samples of adequate size. Although the number of children affected is small, there is evidence of increased incidence[206] of childhood cancers. In Canada, there has been a 25% increase in the last 25 years in cancer incidence among children under 15 years of age.[207]

Certain types of childhood cancers have shown considerable increases, namely, acute lymphoid leukemia, tumours of the CNS and bone tumours.[208] Exposures to pesticides pre-conceptionally, prenatally and during childhood, both in the environmental and occupational settings have been associated with moderate increases in childhood brain tumours and leukemias.[209] There are considerable difficulties in establishing clear exposure-outcome relationships between pesticides and pediatric cancer. However, there is definite cause for concern and greater methodological precision is necessary to further understand cancer etiology in children.

Because of the long latency of most carcinogens, childhood exposures do have implications for most adult onset malignancies. For instance, it is now well established that childhood exposure to ultraviolet radiation that leads to severe sunburn is a strong risk factor for adult development of melanoma. Although most other types of cancers are more difficult to link to the causal exposures (and do not directly affect children), there is still good reason to prevent exposure to carcinogenic substances at the youngest ages possible.[210]

2.6.10 Environmental Chemical Sensitivity

Environmental or Multiple Chemical Sensitivity is a phenomenon that describes individuals who exhibit a cluster of symptoms, such as, headache, breathing difficulties, fatigue, muscle aches and inability to think and function, for which there appears to be no demonstrable clinical basis. Individuals frequently report symptoms after exposure to what would normally be low levels of chemicals (triggers) and they sometimes recall that their illness began after a distinct episode of over-exposure to some chemical (an

[206] This may in part be due to earlier and improved diagnosis.

[207] Canadian Institute of Child Health. What on Earth? Proceedings from National Symposium on Environmental Contaminants and the Implications for Child Health - Canadian Institute for Child Health. (May 1997); and National Cancer Institute of Canada. Canadian Cancer Statistics. (Toronto, Canada. 1995)

[208] Daniels, *et.al.* 1997, *op.cit.*

[209] *Ibid.*

[210] Canadian Institute of Child Health. 1997*, op.cit.*

initiating event).[211] In most cases, the causal associations between exposure and hypersensitivity symptoms are unproven. There has been no consensus on how such syndromes are defined, what the cause of sensitivity is, nor the mechanism by which symptoms might be triggered.[212]

Despite problems with the diagnosis, etiology and treatment of environmental hypersensitivity disorders,[213] and recognition of the fact that not all individuals are equally likely to exhibit such responses to environmental stimuli, the fact remains that sufferers of MCS do experience considerable ill health effects.

There is much less known about environmental hypersensitivity in children. The diagnosis and prevalence among children are not well characterized. Hypersensitive children are known to develop strong reactions to a variety of allergens, such as moulds, house dust mites, tobacco smoke and to have reduced tolerance for synthetic chemicals found in food, air and water. Case study reports indicate that learning disabilities and behavioural and attention problems may be associated with sensitization to environmental irritants in some children.[214]

2.6.11 Summary

Environmental health researchers increasingly recognize that a variety of health problems may be attributed in part to exposure to environmental toxins. Some studies have noted increases in spontaneous abortion and stillbirth rates among women exposed to various contaminants during pregnancy. In many cases, these reflect acute doses, either from accidents or occupational exposure to substances such as solvents, pesticides, PCBs, metals and ionizing radiation. However, there is some evidence that exposure to environmental levels of certain substances, e.g. chlorine disinfection by-products in drinking water, or pesticides, may elevate the risk of adverse pregnancy outcomes. Many congenital birth defects (e.g. malformed genitals, eyes, limbs, and other conditions such as cleft lip and palate) may also reflect environmental exposures during pregnancy.

Impairment of behavioural, cognitive and neurological development may result from exposure early in life to neurotoxins. Lead is the best characterized example of a neurotoxin that has measurable effects on child cognition and behaviour (e.g. lowered IQ, reduced attention span, behavioural problems) even at very low levels of exposure.

There have been substantial increases in childhood asthma, allergies and respiratory problems in recent decades. Environmental factors such as ozone, sulphates and particulate matter exacerbate asthma in asthmatic children sending more children to emergency rooms and hospitals. Immune system functioning may be compromised by exposure to contaminants, either by heightening the immune response (as may explain some of the increase in prevalence of respiratory problems) or via suppression of the immune response, thereby leaving the body more vulnerable to infections and cancer. An increase in certain

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[211] Kipen, H.M., N. Fiedler and P. Lehrer. Multiple Chemical Sensitivity: A primer for pulmonologists. *Clin. Pulm Med*. 4 (1997), 76-84.

[212] Kipen, H.M. and N.L. Fiedler. MCS, Unexplained Symptoms and the Environment. *Risk Policy Report*. 6(1) (1999), 30-33.

[213] Of note, there are a host of other medically unexplained syndromes that appear to have a striking similarity of symptoms and may represent related health problems (Kipen & Fiedler, 1999, *op.cit.*). These include, Chronic Fatigue Syndrome, Fibromyalgia, Irritable Bowel Syndrome, Sick Building Syndrome, Gulf War Syndrome, chronic hypoglycemia, among other conditions that appear to be increasing in prevalence.

[214] Canadian Institute of Child Health. 1997, *op.cit.*

childhood cancers, such as leukemia, bone and brain tumours has also been documented and may be related to immune suppressive effects, or, to the direct genotoxic effects of environmental toxins.

Finally, the potential for environmental chemicals to act as endocrine disrupters (either hormonal mimics or blocks) has elicited considerable concern. The effects of hormone disruption on reproductive development and behaviour and thyroid or immune system functioning have been widely documented in wildlife and from laboratory animal studies. The effects in humans are not fully confirmed, although various trends observed in industrialized countries (e.g. declining sperm counts, increasing prevalence of prostate, testicular, and breast cancers, reproductive organ abnormalities and fertility problems) may be linked with the presence of endocrine disruptors in the environment.

2.7 TRENDS IN CHILDREN'S ENVIRONMENTAL HEALTH PROBLEMS

Evidence of a "new pediatric morbidity" is most convincingly provided by figures that indicate a rising incidence of many of the above health conditions in children. The following presents some of the available data for the trends in certain of these environmentally-related diseases for Canadian children.

Health Canada compiles national statistics on birth defects through the Canadian Congenital Anomalies Surveillance System (CCASS). The most recent published information (for 1989-1991) on the rates of selected birth defects shows that several provinces have high rates for certain anomalies compared to the national averages. For example, Ontario has high rates for abdominal wall defects, while Saskatchewan has high rates of cleft lip/palate and Alberta has a high rate of limb reduction anomalies.[215] A Birth Defects Atlas of Ontario: 1978-1988 was developed as part of the Great Lakes Health Effects Program. During that time period there was a rate of 509 defects per 10,000 births and distinct geographic variability in birth defect risk. Rates were about 40% higher than the provincial average in Kingston, Manitoulin Island and Thunder Bay. Southwestern Ontario had birth defect rates that were 10 to 30% lower than the overall average. This study concluded that there is little evidence of excess risk at the local level and that these data are limited by the fact that they do not identify specific causal factors that may explain variability in rates of birth defects.[216]

The National Cancer Institute of Canada provides Canadian cancer statistics, however, because of the rarity of cancer in children, the trends are difficult to detect. For the period 1990-1994, there was an annual average of 879 children (aged 0 to 14) diagnosed with cancer.[217] Leukemia accounts for the greatest proportion (31%) of new cancer cases, followed by brain and spinal cord cancers (19%) and lymphomas at 11%.[218] Recent analysis of trends indicate that testicular cancer incidence has increased by 59.4% between 1964 and 1996 in Ontario among men aged 15 to 59 years.[219] The greatest relative increase in testicular germ cell cancer has occurred among younger men, ages 15 to 29 years and more

[215] Rouleau, J. T.E. Arbuckle, K.C. Johnson & G.J. Sherman. Status Report: Description and limitations of the Canadian Congenital Anomalies Surveillance System (CCASS). *Chronic Diseases in Canada.* Winter 16(1) (1995). Http://www.hc-sc.gc.ca/hpb/lcdc/publicat/cdic/cdic161/cd161e_e.htm.

[216] Health Canada. 1998a*, op.cit.*

[217] The National Cancer Institute of Canada: Canadian Cancer Statistics 1999, Toronto, Canada, (1999). Http://www.cancer.ca/stats/childe.htm

[218] *Ibid.*

[219] Weir, H.K., L.D. Marrett and V. Moravan. Trends in incidence of testicular germ cell cancer in Ontario by histologic subgroup, 1964-1996. *CMAJ.* 160 (1999), 201-201.

recent cohorts of men appear to be at increased risk of developing this cancer.[220] These trends agree with reports of increased incidence in testicular cancer worldwide.[221] Some researchers suggest that this trend reflects an increase in exposure during the prenatal period to environmental toxins that are estrogen mimics.[222] Non-Hodgkin's Lymphoma, a cancer that can affect children, is one among six types of cancer that has shown an average annual increase in incidence among Canadians from1987 to 1994.[223]

New Statistics Canada research estimates that in 1994/95, asthma prevalence among those aged 0 to 14 years, was at 11.2% (affecting about 672,000 children). In 1978/79 there was only a 2.5% asthma prevalence rate which indicates that there has been more than a fourfold increase in numbers of children afflicted with asthma in under two decades.[224] A Health Canada study found even higher prevalence of asthma in schoolchildren aged 5 to 19 in nine health units across the country.[225]

2.8 THE FUTURE OF CHILDREN'S ENVIRONMENTAL HEALTH

2.8.1 Introduction

Researchers of children's environmental health issues are in agreement that we are still missing substantial pieces of the puzzle. There have been several important strides taken to improve the information base and to set a comprehensive agenda for enhancing knowledge of children's environmental health risks. In Canada, the Canadian Institute of Child Health (CICH) has spearheaded the initiative to address the question, "do children require special protection from environmental contaminants?" Their work includes an extensive literature review summarizing current information on the impacts of environmental contaminants on children's health, as well as a national symposium[226] that brought together presenters from a variety of backgrounds, all with the common aim of pushing "children's environmental health to the forefront of the scientific, government and public agendas"[227] in Canada. In the United States, the Children's Environmental Health Network, which unites delegates from the arenas of research, policy, medicine and advocacy groups, has been a driving force behind bringing a "child-centred", prevention focus to public health, policy and research. It has also sponsored several national research conferences that have helped establish the agenda for pediatric environmental health research and policy.

The work by these two organizations (and others [228]) has served to crystallize key issues regarding

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[220] *Ibid.*

[221] Klotz *et.al.* 1999*, op.cit.*

222 Sharpe R.M. and N.E. Skakkebæk. Are oestrogens involved in falling sperm counts and disorders of the male reproductive tract? *Lancet*. 341 (1993), 1392-5.

[223] The other five cancers reflect adult increases and include prostate and kidney cancer in males and thyroid, lung and breast cancer in females. The National Cancer Institute of Canada: Canadian Cancer Statistics 1999, Toronto, Canada, (1999).

[224] Miller & Hill. 1998*, op.cit.*

[225] Childhood Asthma in Sentinel Health Units: *Report of the Student Lung Health Survey Results 1995-96*. Health Canada (September 1998).

[226] Canadian Institute of Child Health. 1997*, op.cit.*

[227] Canadian Institute of Child Health. A message from the Canadian Institute of Child Health. *CJPH*, 89 (*Suppl* 1) (1998), S3.

[228] The work of many organizations can be cited as having influenced thinking in children's environmental health.

children's environmental health in North America. Here we identify some of these important observations and issues that must be acknowledged and addressed in order to further the goal of protecting and improving children's health in this province.

2.8.2 Specific Exposures/Priority Contaminants

- There is a significant burden of illness and spectrum of effects from indoor and outdoor air pollutants. Indoor air quality is an underestimated source of exposure, requiring further study and monitoring.

- Lead is still a major contaminant of concern for children. In Canada, exposure is mainly through old paint and consumer products (rather than gasoline). There is recognition of more subtle, yet important health and developmental effects from even low-level exposure, warranting further efforts to prevent exposure.

- Reduction of pesticide residues in the North American diet, as recommended by the seminal work of the National Academy of Sciences,[229] is driving new child-centred standards in the U.S.

- The potential for numerous chemicals that are present in the environment to disrupt normal endocrine and reproductive function from exposures during development represents a considerable public health concern.

2.8.3 Gaps in Knowledge

- Of all chemicals in use and being produced, only a relatively small fraction have been tested to assess for any or all types of health effects. Aside from lead, mercury and PCBs, there is relatively little information on the specific health impact of many environmental contaminants in *children*. We cannot necessarily extrapolate from knowledge of the effects in adults. In other words, children are not just "little adults."

- Equally, we cannot definitively extrapolate from the health effects observed through animal studies alone. Animal models of development and physiology are not directly comparable to those for humans, although, they may in some instances, be the only option for predicting health effects in people.

- Frequently there is imperfect knowledge of the mechanisms by which environmental contaminants may lead to particular health effects. For example, although there is modest evidence for cancer stemming from childhood exposure to pesticides, the process remains speculative. In part, this stems

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For example, *Our strength for tomorrow: valuing our children*. Part 3: Child health and the environment Abridged version, Report by Task Force on Child Health, College of Family Physicians of Canada? (CFPC Task Force on Child Health, 1997); What on Earth? A National Symposium on Environmental Contaminants and the Implications for Child Health, 25-27 May 1997, Ottawa, Canada, Canadian Institute of Child Health; selected papers published in *Canadian Journal of Public Health*, Vol 89 *Suppl* 1, May/June1998; *The Air Children Breathe: The Effects on Their Health*, January 19, 20, 1998, Educational Forum jointly sponsored by Pollution Probe and CICH; and Miami declaration of G8 leaders. See also multiple cites for U.S. EPA and others in Sections 4.2 and 4.4 of Chapter 4, below.

[229] National Research Council. 1993, *op.cit.*

from our incomplete knowledge of many aspects of normal developmental processes and physiological parameters.

- Much less is understood of low-level exposures (to organic chemicals and toxic metals) but they may: predispose to chronic illness; decrease higher brain function, especially learning; impair fetal and childhood development.

- In many cases, measuring subtle health effects in children is extremely difficult to do objectively.

- There are also significant information gaps regarding how long after exposure at an early age might health effects appear (i.e. the latency period), the effects of life-long low-dose exposure, as well as effects that may occur in one generation as a result of the previous generation's exposures (transgenerational effects).

- It is imperative to focus on determining the effects of cumulative and multiple (mixed), synergistic (combined or interactive) exposures to environmental chemicals (i.e. beyond the single pollutant approach) as there are gaps in our understanding of these mechanisms. Real-world exposures to environmental contaminants rarely mirror that seen in the controlled laboratory experimental situation.

2.8.4 General Concerns

- Children's health problems that are or may be related to environmental contaminants are on the rise in Canada and other industrialized countries. These include health conditions such as asthma, childhood cancers, learning disabilities, among others.

- There is growing evidence of health problems from exposure to low levels of pollutants, that is, at levels that are close to or **below** current alert thresholds.

- Differences in vulnerability to contaminants need to be better understood. For instance, the individual (genetic) variability that influences the health effects a person may experience when they are exposed to contaminants.

- We must also recognize that there are groups, who as a result of geographic, social, economic, political or cultural circumstances, are much more likely to be exposed to environmental contaminants. In particular, the fact that many low income level and aboriginal children experience greater environmental health risks is an issue of significant concern.

- A prevention approach is necessary to protect children's health, rather than merely implementing the tenets of reduction and regulation.

- There is a significant need for greater education of the public, health professionals and policymakers as to the avoidable, preventable nature of environmental diseases in children.

2.9 CHAPTER SUMMARY

The body of this chapter describes many contaminants, health effects, environmental media and routes of exposure and the particular susceptibility of children because of both increased exposure and increased sensitivity.

What are the issues of main concern for children, their parents, communities, researchers and policy? We prioritize these according to two important criteria: 1) numbers of children affected and, 2) severity of outcome.

2.9.1 Number of Children Affected

The issues of main concern here are very different. Historically, there was widespread exposure of children to lead because of its presence in gasoline. As a result, the health effects of lead exposure in the young are extremely well characterized. We also know a lot about asthma. The research questions about what we don't know, and how to proceed, can be well defined. We are dealing with a very common illness, that physicians diagnose and treat frequently. But the scale of the problem, in terms of numbers affected, is enormous. Endocrine disruption, on the other hand, is an area of research much less well defined than the effects of air pollution or exposure to lead. The notion of endocrine disruption is unsettling, the animal evidence is troubling, and the major uncertainties of critical concern to society, creating new challenges for policy.

Lead

Children are particularly vulnerable to health effects from lead, even at very low levels of exposure. Children are also predisposed to higher exposure to sources of lead contamination. We now believe that lead may permanently alter a child's physical, mental, intellectual and behavioural development and that there may be no "safe" threshold for these developmental delays. Many of the previous sources of lead exposure have been eliminated or significantly reduced. However, this was not before generations of children suffered the consequences from exposure to lead in gasoline and consumer products. There is still reason to remain vigilant about the possibility for many children to be exposed through new or unexpected sources of lead, because of their unique behaviour and because of the persistence of lead in the environment.

Asthma

There has been a dramatic increase in the prevalence of asthma in the last 20 years, affecting 672,000, or over 11% of Canadian children in 1994/95. Asthma is the most common chronic condition of childhood; a major public health issue.

We do not know what is driving the increased prevalence of childhood asthma, but areas of research must include familial, allergenic and environmental factors. We do know that exposure to both outdoor and indoor pollutants, and Environmental Tobacco Smoke, makes asthma worse in asthmatics. The role of pollution in the causation of asthma must be studied more.

Endocrine Disruption

Endocrine disruption is a relatively new concept in environmental health. There has been an explosion of research and understanding, but it remains an area of enormous scientific uncertainty. The story of lead suggests that uncertainty should not supersede a precautionary approach when the stakes are so high (i.e. widespread exposure combined with profound health effects).

We are all exposed to endocrine disruptors. They are measurable at very low levels in the body fluids and tissues of all our children. Some of them (persistent organochlorines) are stored in the body for long periods of time. The significance of this is unclear. Wildlife, exposed to higher levels of persistent organochlorines, have been found to suffer reproductive dysfunction and sexual abnormalities. At present we live with uncertainty as to whether endocrine disruptors might be affecting cancer rates, reproductive function and development, neuro-development, the immune systems and thyroid function of children. Of immediate concern is the levels of two persistent organochlorines with endocrine disruption action, dioxin and PCBs, in the breast milk of mothers in the Great Lakes Region. Although breast milk is clearly considered best for the baby, this is an area that warrants continued scientific and immediate

policy attention. We need to focus on reducing the chances that these substances continue to end up in the environment and ultimately, in human tissues, being passed on from generation to generation.

2.9.2 Severity of Outcome

Childhood Cancer

Cancer is the most feared of childhood diseases. It is the second leading cause of death in 0-14 year olds. Of great concern is the increased incidence of certain childhood cancers, especially leukemia and brain tumors. What role do environmental agents play in the etiology of cancer? What is the relationship between genetic, developmental and environmental factors? Great strides have been made in the treatment of childhood cancer, offering hope to affected children and families. But the ultimate victory over cancer will be prevention of the disease. Hopefully the search for, and elimination of, environmental causative or promoting agents, will be a huge step in this direction.

Neurodevelopmental Effects

Lead levels in the blood of Ontario's children dropped steadily as lead was removed from gasoline. How many children were affected prior to this we do not know. We do know that lead, even at levels previously common in Ontario, and certainly common in other parts of the world that still use leaded gasoline, has been consistently associated with lower scores on tests of intellectual function, and with reading disabilities and failure in school. The loss of human potential has probably been substantial.

There are other chemical exposures that affect the child's brain in this period of rapid and critical development. Less is known of them than lead. There are concerns with regard to mercury, organochlorines, pesticides, and manganese, which has recently been readmitted into Canadian gasoline. The gaps in knowledge should not restrict us in our efforts to protect the potential that is our children.

2.10 RECOMMENDATIONS

In light of the foregoing discussion on the future of children's environmental health we can make a number of key recommendations that will further the incipient trend to protect the health of Canadian children.

1. Children's exposure to environmental contaminants needs to be more accurately characterized, estimated and assessed including baseline data on exposure, emissions, biomarkers and health effects. For children's exposure to pesticide residues in food, the 1993 United States National Research Council report clearly demonstrated that the data were incomplete, and that children differ from adults in terms of food consumption, both quantitatively and qualitatively. These differences and information gaps also occur for a variety of other contaminants and routes of exposure. A key part of the solution to this problem should be to mirror in Canada the data collection model used in the United States: the National Health and Nutrition Examination Survey (NHANES). In particular, efforts should include data collection similar to the proposed National Longitudinal Cohort of Environmental Impacts on Children and Families currently being designed by the Centers for Disease Control in Atlanta. Further, efforts to marry databases and expand this data collection system to include all of North America should be encouraged.

2. There is a need to enhance knowledge of the critical periods and vulnerable systems during development of the fetus, infant and child such that we can better prevent compromise to children's health throughout their lives. We know that lead exposure prior to age 2 has marked effects on nervous system development and behaviour. Better understanding is required as to the influence (if any) of endocrine disruptors and air pollutants at early stages in development, and whether they predispose children to health effects later in life.

3. There is a need for greater understanding of specific pediatric health problems that have an environmental basis and that are increasingly prevalent, including asthma, cancer, and perhaps, learning disabilities. For asthma in particular, which affects nearly 13% of Canadian children, a concerted research effort should be funded and promoted to investigate the links between asthma and both indoor and outdoor environments.

4. Attention must also be focused on identifying those children whose risk of exposure and/or susceptibility to environmental contaminants is compounded by other factors. Children from lower income families and aboriginal children, children whose parents work in occupations that expose them to contaminants that might be brought into the home, children residing in agricultural regions and the children of families that eat sport fish and wild game are all at heightened risk for exposure to environmental contaminants and subsequent environmental health problems. Additional research is necessary to determine links between environmental contamination, poverty and other broad determinants of health.

5. Although the federal government does provide some funding for research on children's environmental health (see Chapter 1), given the significant gaps in information identified in this study and through the preceding recommendations, the government should further support Canadian research that fills those data gaps. To that end, (and similar to the circumstances in the U.S.) we recommend that government-funded centres of excellence for the study of environmental health be established which would include children's health as an important focus. Such centres should encourage collaboration and coordination of research efforts between government and universities.

6. In the clinical setting, pediatric environmental health clinics should be established, within academic teaching hospitals, to provide a clinical service, to promote teaching of health professionals and to conduct appropriate health research. Such clinics should incorporate the information and methods recently promoted by the American Academy of Pediatrics in its Handbook of Pediatric Environmental Health.

7. Strategies to prevent environmental exposures should also become part of the clinical protocol for expectant and nursing mothers and parents with young children. Physicians, nurses, midwives and social workers need to be educated and patient materials need to be developed.

8. For pesticides in particular, and as also noted in the Pesticides Case Study, the difficulty must be recognized of identifying cases of exposure to pesticides in a clinical setting because of the non-specific nature of symptoms. Hence, university and college curricula must educate health professionals (including family physicians, pediatricians, obstetricians, midwives, and nurse practitioners) about the adverse health effects of pesticides (both acute and chronic). Further to the preceding two recommendations, an important part of such clinical education would be to learn environmental history taking similar to the methods recently promoted by the American Academy of Pediatrics in its Handbook of Pediatric Environmental Health. These strategies should also become part of the clinical protocol for expectant and nursing mothers and parents with young children.

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