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Environmental Health Committee

Environmental Standard Setting and Children's Health

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Environmental Standard Setting and Children's Health

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Executive Summary

Human behaviour has resulted in the introduction of many tens of thousands of different chemicals to the environment over the last half-century. The rate of production of new chemicals has overtaken the capacity to fully characterize their potential to cause harm to people. The state of the environment plays a critical role in causing or augmenting ill effects to human health and well being. Today's children are growing up in an environment that is radically different from that of their parents and grandparents, one that has an incomparable potential to impact on health throughout their lives.

The surface has only been scratched in our understanding of just how detrimental toxic environmental exposures during childhood can be to lifelong health. However, there are clear indications that children's health is being measurably compromised by environmental factors.

The challenge to protect children's health is enormous; society is contending with an environment in which there is both ample opportunity for exposure and limited information on the risks from those exposures.

The primary and immediate goals in response to these concerns are to prevent, or at the very least, reduce exposure to environmental contaminants in children and to better identify the risks to children from toxic environmental exposures. These, then, are the ultimate challenges of the standard setting processes. The question remains, however, how effective have the standard setting regimes been in responding to these challenges?

The following document is the initial product of the *Children's Health Project* that represents a 20-month collaboration between the Canadian Environmental Law Association and the Ontario College of Family Physicians' Environmental Health Committee. This report summarizes the findings of a lengthy investigation into the adequacy of the standard setting process for protecting the health of children in Canada and specifically, in the province of Ontario. The report also provides a detailed review of research into the greater susceptibility and exposure of children to environmental contaminants.

CHAPTER SUMMARIES AND CONCLUSIONS

Introduction

Chapter One lays out the overview, structure, rationale, methods and bounds for the study. The study defines "childhood" according to current practice in environmental health research as all stages prior to maturity, from *in utero* up to and including adolescence. The project team determined that the contaminants chosen for consideration would be primarily chemical and metal pollutants. This admittedly leaves out several significant types of environmental toxins that may or do affect children,[1] however, it was felt that focus on a smaller number of equally important environmental contaminants would realize greater gains without duplicating the efforts of others. In addition, the study focuses on reviewing specific areas of standard setting, again for chemical and metal pollutants, and addresses air, pesticides, consumer products and toxic substances. These areas reflect standards that have greatest relevance to the chemical exposures of children, in terms of known or suspected avenues of increased risk and, as well, they represent areas of standard setting most directly determined by evaluations of human health effects. We recognize that a key component of analyzing the adequacy of standard setting regimes

[1] For example, environmental tobacco smoke and other indoor air pollutants, physical contaminants like radiation and EMFs, and biological agents such as moulds, fungi and bacteria.

was to provide a thorough review of the scientific risk assessment process that is embedded in most regulatory frameworks.

The review of standard setting includes coverage of some areas in depth (air, pesticides, lead) and overviews of others. Since Phase I of the *Children's Health Project* provides a foundation for further work, opportunities for additional legal and policy analysis are identified. Two case studies, one concerned with lead, and the other with pesticides, allow for more extensive review and analysis of the information on exposure and health effects and the standard setting regimes in two areas that continue to be significant to children's environmental health in this country. Finally, **the central questions for the study were to decipher whether the regulatory framework as it exists in Canada is *intentionally* protective of children and where this is so, whether children's health is indeed protected.**

Exposure and Health Effects

In its summary of the extensive scientific literature characterizing children's greater exposure and susceptibility to environmental contaminants, Chapter 2 highlights some of the key trends in recent scientific understanding of children's environmental health. The conclusion is that this information must inform both scientific assessment of risks and regulatory decision-making.

There is increasing evidence of health effects from various pollutants occurring at very low levels of exposure. In some cases, it is speculated that there is no threshold below which children are safe from the effects of these contaminants. Examples include lead, ozone and particulate matter, all three of which have clear effects on children's health in particular. With most environmental contaminants we can characterize the outcome of exposure in terms of a pyramid of effects. At the apex of the pyramid, thankfully, few children suffer from fatal effects, but, toward the base there are increasing numbers exhibiting subclinical, yet often very important compromises to their health and well-being.

There is great concern among scientists because of the universality of some of these exposures. Air pollution and persistent organic pollutants (POPs) that appear in the food chain can potentially reach virtually all children. The latter is of exceptional concern because of the biologically plausible hypotheses surrounding their role as endocrine disruptors.

It is critical for the protection of all children that the variability inherent in human exposure or response to environmental contaminants is brought to the fore. Both exposure and susceptibility to health effects are mediated by genetic, social, economic and cultural factors. In particular, poor children and aboriginal children are generally more often at greater risk of environmentally related health problems. In Ontario for example, while the most recent data on blood lead levels indicate an *average* that is below the intervention level, the *distribution* of those values demonstrates that some portion of those children is close to or above the level for health effects from lead. Children living in poverty are at greater risk of reaching or surpassing that intervention level of exposure.

When determining the potential for exposure to a given contaminant, exposure assessments must account for the complexity and great variety of exposure pathways and media through which children may become exposed. In particular, we underscore the fact that the regulatory framework has not routinely considered exposures during the prenatal (when the child was in the womb) and early postnatal (via breast milk, foods or consumer products) periods. These represent significant exposure routes for children and ones that can have important impact because of the characteristic developmental windows of vulnerability during these times.

Similarly, it is increasingly recognized that exposures to contaminants that occur early in life may have long lasting or delayed consequences that may translate to more serious health problems later in life. For example, exposure to carcinogens may not result in cancer until later years, childhood exposure to air pollution may predispose to respiratory disease in adults and, exposure to lead prior to age two is associated with permanent effects on growth and neurocognition and behaviour.

Newer data that are gaining wider acknowledgement suggest we must be ever vigilant in expanding knowledge of the health effects from children's exposure to environmental contaminants. Delayed neurotoxic effects and acceleration of aging from early lead exposure, damage to DNA of immune cells after exposure to air pollution and the effects on the thyroid and immune systems from persistent organic pollutants are but a few examples of recent, notable research results.

Lastly, in Canada, it is clear that for most types of environmental exposures there is relatively much greater exposure in the Great Lakes basin compared to elsewhere. The Great Lakes Health Effects Program (GLHEP) found that measures of contaminants in human tissues were often consistently greater for populations living in this region. Of note, because contaminants such as PCBs and dioxin are present in breast milk in concentrations that approach or even far exceed the current guidelines of Health Canada, breastfed infants are being exposed and we are not certain what the effects of that exposure will be. Breast feeding continues to be the recommended method of infant feeding, however, greater attention must be paid to preventing further breast milk contamination and therefore exposure of children to toxins at a vulnerable age.

The Standard Setting Framework

Chapter 3 aims to make sense of how the legal and policy system operates. It is solely descriptive and lays out the basic regulatory framework and division of responsibilities between the federal and provincial governments. Two overarching policies of direct relevance to children's health are introduced: the *Toxic Substances Management Policy* and the *1997 Declaration of the Environment Leaders of the Eight on Children's Environmental Health.* The effectiveness and implementation of both of these policies are central to this study.

The federal and provincial government departments responsible for standard setting affecting children are Health Canada, the Pest Management Regulatory Agency (PMRA), Environment Canada and in Ontario, the Ministry of the Environment. Federal, provincial and territorial cooperation and partnership is sought through the Canadian Council of Ministers of the Environment (CCME). Under the CCME, a multi-lateral agreement, the Canada-Wide Accord on Environmental Harmonization, has been established that has far-reaching implications for standard setting across Canada.

Chapter 3 provides, for each of these agencies, summaries of their self-described authority, responsibilities and coordination with other departments. A brief description of recent trends in funding for each agency is also provided.

Risk Assessment and the Precautionary Principle

The focus of Chapter 4 is a lengthy, critical review of the theoretical foundations of standard setting, namely the processes of risk assessment and risk management. The focus is on the use of risk assessment for deriving health-referenced standards. By the late 1970s, risk assessment became the regulatory tool of choice that increasingly replaced early decision-making that, in some cases, banned very hazardous substances (such as DDT and PCBs) due to their inherent toxicity. Instead, risk assessment enabled continued use of toxic chemicals at scientifically sanctioned "acceptable" levels. During the process of bringing risk assessment in greater synchrony with the increased knowledge of environmental health issues, attention has focused on continually refining rather than replacing risk assessment. Criticisms and some fundamental limitations of the system have been identified for well over 10 years and have yet to be adequately addressed. One of the first problems identified was the disproportionate focus in risk assessment towards managing cancer risks. It was not until the mid-1980s that the U.S. Environmental Protection Agency (EPA) began to add some consideration of developmental risks into its risk assessment protocol. More fundamental limitations concern the numerous stages wherein inference and judgement are applied to compensate for large gaps in data and methodologies rendering risk assessment anything but a wholly objective scientific exercise.

Critical problems with risk assessment surround the characterization of exposure and dose-response. For the vast majority of chemicals, we do not know exactly how much of a particular substance, or combination of substances, to which people will be exposed in the course of its/their use, emission and path through the environment. It is also exceedingly difficult to determine what the relationship is between the amount that reaches the tissues and the response of the body to that dose.

More fundamentally, risk assessment enables risk calculations that allow for "acceptable" levels of chemical exposure that may cause one-in-a-million or one-in-ten-thousand risks (of cancer, birth defects, etc.) across a population. However, this game of odds becomes useless if further research confirms the suspicion that chemicals such as endocrine disruptors are capable of exerting population-wide effects at current levels of exposure. Nor is it appropriate to make such calculations for chemicals that are persistent and bioaccumulative. Risks will continue to increase for chemicals that do not break down and which accumulate in animal fat, breast milk, etc. Such risks will affect some people more seriously than others depending on the flow of persistent chemicals through the environment. The predicted avenues of exposure and the health endpoints that are used to assess risk clearly have implications for the ability of ensuing policy decisions to protect children's health.

The ever-increasing complexity of risk assessment methodologies has been matched and consistently overcome by the greater complexity of the problems they attempt to address including accounting for the special exposure circumstances and vulnerabilities of children. For those risk assessment advocates or practitioners who recognize the significance of the data gap, and many do not, the problem is considered inevitable and insignificant and a key solution is seen as the need to improve techniques of risk characterization and communication.

Issues of ethics and equity are also highlighted. Risk assessment is a complex, opaque system and that complexity and lack of transparency afford the opportunity for obscuring the value judgements it includes as well as the manipulation of policy-makers. Chemicals are assessed in isolation and each is treated in essence as "innocent until proven guilty." Risk assessment demands that decisions as to the harmfulness of toxic chemicals be determined according to the very high standard of proof demanded of scientific inquiry. This standard is nearly impossible to achieve given the arguably problematic scientific foundation of risk assessment. Yet, when action is taken only in the face of rigorous proof of harm, the chemicals ostensibly have greater rights than the human population. Each chemical, assessed in isolation is allotted an "acceptable" risk level whereas the human population does not have the same right to avoid the cumulative risk of real-world exposure circumstances to many different chemicals in the environment.

The critique of risk assessment, its assumptions and practice, is supported by a summary of the science behind the assessment, namely the relevant principles of epidemiology and determination of causation. The summary highlights the fact that scientific inquiry is extremely cautious and demands a rigorous degree of certainty in order to make definitive statements about causation. **We conclude that the scientific standard of proof is an inordinate, unbalanced and unfair burden to demand prior to the establishment of protective standards**. This standard of proof is particularly inappropriate when at issue is the prevention of harm to children's health. We conclude that the demand for such a standard of proof will very likely contribute to undue exposure and possibly irreversible health effects before protective action is taken. Such exposure occurred for millions of children exposed to lead from gasoline. As the Lead Case Study illustrates, the lessons of that cautionary tale need to be applied to the regulation of pesticides and other toxic substances.

The objective of child-protective standards would be better served by standard setting that weighed more in favour of the legal concepts of "duty of care", "the balance of probabilities" and the medical dictum of, "do no harm." Our review of the "science behind the assessment" highlights the observation that science and policy are, and should be, separate entities because of the incorporation of a broader range of values and considerations in the latter. However, given the often shaky scientific foundations that exist when

standards must be set, a "weight-of-evidence" approach needs to be applied throughout both the scientific and policy stages of the standard setting exercise.

We conclude that a new paradigm is necessary to supplement and in some instances to replace the current risk assessment framework in science and policy. The paradigm, borrowing concepts from the legal context, centres on shifting the burden of proof that is required in regulatory decision-making on to the parties wishing to create environmental contamination, a reverse onus approach. The paradigm must also incorporate the notions of making decisions that reflect prudent, protective judgement and precautionary inference, including consideration of the weight of evidence. While weight of evidence is increasingly used in standard setting, it is applied in too limited a manner revealing that only minor first steps are being taken towards a more precautionary paradigm.

The review of risk assessment includes a close look at recent steps taken by the Environmental Protection Agency to implement the *Food Quality Protection Act* in the United States since 1996. The review focuses on the practical challenges of implementing this regulatory device that was intended to enhance protection for children from exposure to pesticides through their diet, but has yet to achieve those original goals to any significant degree. At every step of the risk assessment process there has been ongoing fine-tuning and generation of ever more complex, voluminous and numerous guidelines. These efforts have served to effectively place increasing constraints on application of the key progressive elements of the FQPA, in an endless quest for definitive scientific evidence. In particular, with respect to the extra 10-fold safety factor, decisions as to when it is applied (rarely, as it turns out) appear to fall into the black box known as "scientific judgement." The renewed practice of human testing by pesticide companies has been a perverse, unintended result of attempts by the pesticide industry to avoid the 10-fold safety factor. This phenomenon is currently under scrutiny and further highlights the ethical and equity issues enmeshed in the current risk paradigm.

It is important however to recognize that valuable and progressive elements exist in the FQPA including at least the *concept* of, the 10-fold child-protective safety factor as well as requirements to aggregate chemical exposures and to assess groups of chemicals with common mechanisms of toxicity. The latter two are extremely important attempts to assess real-world exposure circumstances and to move beyond the ponderously slow chemical-by-chemical assessment approach. Unfortunately, the long-standing, central limitations in the science within risk assessment are brought into stark relief in trying to implement these progressive ideas. Ideally, however, Canadian regulatory agencies can and should learn from the FQPA experience, avoid the pitfalls and adopt its strengths.

Chapter 4 also reviews the precautionary principle, a contrasting as well as complementary paradigm whose acceptance has gained international approval, at least on paper. It mandates that in the policy arena, where potential for harm exists together with a great degree of scientific uncertainty, measures to avoid such harm should be adopted without delay. Considerable debate exists as to the appropriate definition and ways of implementing a precautionary approach. In Canada, it is acknowledged as having informed the *Oceans Act*, the revised *Canadian Environmental Protection Act*, the Canada-Wide Accord on Environmental Harmonization and legislation in two maritime provinces, although in none of these instances is it explicitly stated how the precautionary principle is to be applied and implemented, nor is implementation occurring. Canadian regulators also appear to be adopting the Rio Declaration definition which qualifies that "cost-effective" measures be taken to avoid harm. A preferable alternative is the *Wingspread Statement on the Precautionary Principle* which states:

When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause-and-effect relationships are not fully established scientifically.

Air

Chapter 5 examines whether the regulation of contaminants in air, which involves both federal and provincial governments, has been intentionally protective of children. The approaches differ depending

on the jurisdiction, however there are some commonalties. In terms of recent policy on air pollutants, both jurisdictions have focused largely on reviewing standards for so-called "priority" substances as defined by the "risk" paradigm, while there has been continued reliance on out-dated standards for other substances. For each of these reviews the documentation states that there is regard for balancing cost-effectiveness with considerations of human health. **The attention to cost-effective measures has led to examples where industry's concerns have taken precedence over those of protecting the most sensitive receptor.**

The Ontario Ministry of Environment's recently revised Standard Setting Plan holds promise of improvements as it states that it will consider the most sensitive receptor in hazard analysis. This may be children, or presumably children will also be protected if a more sensitive receptor, such as an ecosystem effect, is chosen. Another positive feature of this plan is that it applies a multi-media, pathways approach when determining the most sensitive receptor. It remains to be seen, however, whether the risk management phase of the standards reviews will in fact carry forth the progressive aims of this plan that might protect children.

The federal process for setting air standards has moved to the Canada-Wide Standards approach which is a stakeholder, rather than a health-based approach. Unanimous consensus is required for adoption of new standards and this has resulted in some standards being driven to levels that are less protective of health and the environment under the influence of those jurisdictions with the greatest problems for a particular contaminant. Accordingly, **the Canada-Wide Standards stakeholder approach to setting standards is neither intentionally, nor actually protective of children, but is heavily shaped by the risk management phase of the process.**

Toxic Substances

Chapter 6 provides primarily an overview of the regulation of toxic substances. The chapter considers a broad variety of vehicles such as the: Great Lakes Water Quality Agreement, *Canadian Environmental Protection Act*, Toxic Substances Management Policy, *Pest Control Products Act*, Canada-Wide Standards, current negotiations towards an international treaty on Persistent Organic Pollutants (POPs) and Ontario's Municipal Industrial Strategy for Abatement (MISA). Similar themes recur in this chapter as in the previous one (i.e. that a risk framework is applied, that a small number of priority substances are targeted for review and that in most cases, there is a stated commitment to cost-effectiveness and reasonably achievable solutions). Another recurring theme is that the legislative framework does not incorporate in any meaningful way the precautionary principle or pollution prevention as outlined in Chapter 4.

However, chapter 6 also highlights the few instances in the Canadian regulatory scene where there is at least an attempt to regulate substances because they are deemed inherently toxic. (These attempts too, are not without their flaws.) **Under the Toxic Substances Management Policy, persistent bioaccumulative substances are slated for virtual elimination which is defined as a lack of measurable release, rather than the more protective action which would eliminate *use* of the substance.** In Ontario, the MISA regulations were not risk-based but intended to reduce pollution as much as possible while calling for the use of the best available technology economically achievable. The focus of the MISA regulations, passed in 1995 and 1996, is on persistent and bioaccumulative substances. This focus brought about a significant reduction in toxic pulp mill emissions. The Hazardous Contaminants and Water Resources Branches of the Ministry of the Environment also have focused, at least in past, on inherently hazardous substances that they state should ideally not be allowed to enter the environment. They cite that the focus on inherent toxicity is a direct result of the lack of exposure data that would be necessary in the formal risk assessment approach. Despite these progressive approaches in the past, regulatory action by the Province on toxic substances has been minimal in recent years.

Consumer Products

Stemming from the review in Chapter 7, it is apparent that both the *Hazardous Products Act*, the vehicle by which Health Canada controls the sale, importation and advertisement of consumer and industrial products, and Health Canada's role in its enforcement, are of limited value in protecting children's health. The Act does not define general product requirements and it is a product-centred approach that is reactive rather than preventive. In other words, **no mechanism is in place for formal pre-market assessment of consumer products such as toys or equipment and furniture that is intended for children's use.** Product inspection can only take place, for products regulated under the Act, when Health Canada receives a complaint, or if a Health Canada inspector believes there to be a potential risk from a product. In both instances this represents post-market assessment, once the goods have already been made available to unwitting consumers. If Health Canada's risk assessment determines that there is indeed a risk, it has no power to mandate product recalls. Instead, it relies upon voluntary industry action and may issue warnings and advisories to the public. Stronger action, such as the adoption of a regulation under the Act, will ensue only when Health Canada deems that the above strategies are insufficient to protect the public from risk.

The two case-studies provide greater depth for critical analysis of the standard setting regime in two specific contexts that have vast implications for children's health in Canada, namely regulation of lead and of pesticides.

Lead: The Cautionary Tale

Case Study #1 deals with standard setting to protect children from lead. It represents the "cautionary tale" and illustrates central problems with risk assessment and risk management. For example, industry-controlled information and research limited and biased the understanding of low-level lead exposure in children for many years. Assumptions of safety were made in the absence of proof of harm despite early warnings of neurological effects in children and evidence from animal studies that raised concerns about the potential dangers to children from low-level lead exposure. The science was extremely complex. Validated testing protocols for measuring multiple sources and exposure pathways and then body burdens were expensive and, for blood-lead, intrusive. Complex and age-sensitive measurement tools were necessary to discern subtle effects in childhood neurodevelopment. Such tools have only recently been developed to discern and measure effects of lead on behaviour. Research results were hotly contested and findings of health effects were elusive and subtle requiring the application of complex meta-analyses to discern significant effects.

Regulatory agencies, particularly Health Canada in its reaction to lead in gasoline, waited while the debates continued and did not act in a precautionary manner until exposure was high enough for investigators to reveal incontrovertible evidence of harm. Lead is now one of the most extensively studied pollutants in the world. Clear causal associations are apparent between lead and adverse neurobehavioural effects in children. The data are extensive enough that two powerful meta-analyses have verified the realization that an increase in blood-lead levels from 10 µg/dL (micrograms per deciliter) to 20 µg/dL results in an IQ deficit of approximately 2 points. The Lead Case Study summarizes the variety of adverse neurological and neurobehavioural effects that have been revealed as associated with exposure to lower and lower levels of lead.

The regulation of lead did not keep step with the scientific evidence of risks to health. The use of lead in gasoline represented an effective way to expose literally millions of children around the globe to unsafe levels of lead. **Phase-out of lead in gas was a significant step in reducing the exposure of children to lead.**[2] However, significant problems remain in Canadian regulations for lead in paint and the process for

[2] It should be noted that leaded fuel is still commonly used in many developing nations.

dealing with lead in other unexpected sources, including products such as mini-blinds and toys. Proposals to regulate lead in consumer products promise to make Canada a world leader in the control of lead in children's products. However, this regulation is not likely to be in place until at least 2001 and may not survive opposition from the toy industry or the possibility of international trade obligations trumping attempts at domestic regulation.

Average blood lead levels in Canadian children have been on a steady decline since the move away from leaded gasoline, however, **1994 estimates suggest that over 66,000 children still had blood-lead levels in the range known to have health effects**. Since scientific research has determined that there likely is no threshold for health effects in children from lead exposure, it should remain an issue of concern and greater awareness.

The Lead Case Study makes five key conclusions that highlight aspects of the cautionary tale. First, regulatory action on dangerous, persistent substances must include a precautionary and preventative approach. Second, it is essential that research on pollutants be independent of the industries responsible for the contaminant emissions. Third, lack of proof of harm must not be considered proof of safety. Fourth, when the insistence on "sound science" serves to delay regulatory standards a dangerous "Catch-22" situation is created wherein once the scientific data are conclusive enough to prove harm there has already been extensive contamination and harm done. Fifth, Health Canada's regulation of current uses of lead is largely ineffectual, although their stated commitments hold considerable promise. In the meantime, consumers cannot assume that products for sale have undergone independent or legislatively required testing to ensure children's safety.

Pesticides

The regulation of pesticides to protect children's health is the focus of Case Study #2. The information on pesticides contrasts with that of lead, in that there is relatively more limited information on what represents a varied group of environmental contaminants. The comprehensive review of literature concerning exposure to and health effects from pesticides in children concludes that the potential for the health of children in Ontario to be affected by pesticides is undeniable. Studies point to a wide variety of possible health effects in children from pesticides, many of which are serious and in some cases, life-threatening. Although the body of evidence for pesticides is not as weighty as that for lead, the data do tend towards implicating pesticides as inducing damage to children's immune, endocrine, nervous and reproductive systems, as well as congenital anomalies and cancer. Both exposure and susceptibility to the effects from pesticides are documented as being greater in children as compared to adults based on current scientific research. It is likely that many Canadian children are enduring the negative effects of pesticides, including those that are: from poor homes that may be treated with pesticides; children living in agricultural areas and the children of agricultural workers; aboriginal children exposed through their traditional diet and mother's milk; and children with chemical sensitivities and immune deficiencies. **The cumulative effects of being exposed to many different pesticides over a lifetime represent an unquantified and unacceptable risk to all Canadian children.**

Moreover, this investigation revealed that children's health is at risk because of inherent weaknesses in the Canadian regulatory system that governs pesticides. The analysis focuses on the many unfulfilled promises that are part of federal government commitments, some dating as far back as 1994, to improve the regulation of pesticides. The most serious shortcomings highlighted in the critique centre on aspects of the work by the Pest Management Regulatory Agency (PMRA), in particular, the inaccessibility, lack of clarity and contradictory nature of its risk assessment and risk management process. **The review concludes that rather than Canadians having a regulator for pesticides, the pesticide industry has a "customer service department" in the Pest Management Regulatory Agency.** In order to honour existing commitments as well as prevent harm to children from pesticides, the Case Study also concludes

that the PMRA is in need of both significant expansion and, more important, re-orientation towards a mind-set that gives first priority to health promotion and prevention of harm.[3]

OVERALL STUDY CONCLUSIONS

Several recurring observations have been made throughout this study that bear highlighting separately.

In terms of the state of knowledge regarding pediatric environmental health, much has been achieved in terms of an enhanced awareness of children's environmental health issues among health scientists. However, researchers acknowledge that the issues are complex, there remain significant gaps in information and measurement tools to discern exposure and effects, and that generally research has progressed slowly.

Regarding the regulatory realm, a fundamental conclusion is that the disconnect between the twin processes of risk assessment and risk management as they are applied in standard setting regimes perpetuates a questionable notion that risk assessment is the "objective," science-based part of this dual exercise. This report concludes that the gaps in information and methodologies are too profound for risk assessment to be considered an entirely objective activity. Rather, risk assessment is a combination of science and conjecture and like the risk management phase, it is replete with opportunities for value judgements and bias to influence decisions.

The many judgement calls throughout risk assessment and risk management are recognized as a necessary part of exercises that seek to set standards in the face of large areas of uncertainty. However, the insistence on "risk assessment as science" sets up regulatory agencies for failure in terms of setting protective standards. This failure results from the insistence on scientific standards of proof *before* protective action is taken. When science is unequal to this task, i.e., in the majority of cases, regulatory agencies wait and see or bow to industry pressure for an unreasonably high standard of proof of harm. This situation becomes even more complicated when industry pays for or controls the generation of scientific information about its own pollutants.

The cautionary tale in the Lead Case Study illustrates both a public health success story and the failure of risk assessment. The Pesticides Case Study and Chapter 4 reveal that the lessons have not been learned. The decision to follow the U.S. Environmental Protection Agency "science-based" risk assessment approach to revising standards for questionably safe pesticides perpetuates existing problems. The regulation of air pollution, toxic substances, and consumer products applies the same approach. Under the risk assessment paradigm, children are being exposed and will continue to be exposed unduly to environmental and consumer product contaminants as governments wait for definitive proof of harm and delay decision-making that would better protect them.

Another overall conclusion is that even where risk assessment exercises may provide for protective standards, the risk management phase or policy-making step is not transparent, is highly malleable and often results in weakening of the initially proposed standards such that they are no longer particularly protective of human health.

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[3] It is important to note that, since the Pesticides Case Study was published earlier than the main study it is current to December 1, 1999. More recent announcements by the PMRA state that it will follow the lead of the United States Environmental Protection Agency with respect to pesticide re-evaluation. Hence the conclusions and recommendations of the Case Study regarding the conduct and transparency of the PMRA's risk assessment process for both new and currently registered pesticides need to be considered in light of when they were made. The reader is also reminded that the U.S. EPA's re-evaluation strategy is the subject of a detailed up-to-date review in Chapter 4 of this study.

A further (and related) conclusion is that preventive action in standard setting has rarely occurred. In the increasingly rare instances where toxic substances have been banned or severely restricted, action has only been triggered by clear evidence of harm in the environment and in human populations (or sub-populations) often from levels that were initially assumed to be safe. For many more substances, weak or non-existent regulation has continued, often for long periods of time, in the face of scientific complexity and uncertainty but with indications of serious risk. Even with increased awareness of the need to protect sensitive populations, including children, regulatory action has been minimal. The regulation of lead is a clear example of the lack of prevention within the risk assessment approach to standard setting. Scientists fear that regulation of pesticides and endocrine disruptors may not heed the advice of that cautionary tale. The complexity of scientific investigation into the single pollutant lead, pales in comparison to the effort necessary to understand the multiple exposure pathways and health effects of hundreds of pesticides (dozens of which are known or suspected to cause serious health effects), and tens of thousands of endocrine disruptors.

Many declarations and policy statements have been made at the international, national and provincial level espousing the need to act in a more precautionary manner to prevent harm from environmental contaminants for children or the environment in general. However, very limited progress has occurred to ensure their effective implementation.

Finally, the choice to focus the study on chemical and metal pollutants as areas of greatest concern for children's environmental health and those for which standards tend to be health-referenced, sent an Ontario-focused effort into large areas of federal jurisdiction. **A consistent finding was an extreme reluctance on the part of the federal government to effectively regulate chemical or metal pollutants in a timely manner or at all.**

MAIN STUDY RECOMMENDATIONS

Multiple recommendations stem from this report. They reflect the specific analyses conducted as outlined above and are contained in each of Chapters 2, 4, 5, 6, 7 and the two case studies.

The fundamental theme of the recommendations is that in order to significantly improve the current standard setting framework, there is need for a shift in paradigm to one that incorporates a precautionary approach at every stage in the process. In specific terms, a weight of evidence approach and the reverse onus burden of proof will ensure that prudent, timely decisions are made to provide standards that protect children's health.

A fundamental recommendation is the need to expand research, in both clinical and academic settings, on children's environmental health. In particular, research that improves our understanding of the how, why, when and what of children's environmental health is necessary. We need to better understand how contaminants travel through the environment, why children's unique behaviours expose them to contaminants, how much exposure and to which contaminants, when there are critical windows of developmental susceptibility, and what are the health effects from exposure. The focus of research should be on the health effects that potentially affect many children (e.g. asthma, neurodevelopmental and neurobehavioural effects) and as well, on characterizing the children at highest risk for environmental health problems.

We need to improve monitoring of the environment and population health. This includes monitoring of air, food and water quality, the potential health effects in wildlife, as well as population health surveys (along the lines of the National Health and Nutrition Examination Survey – NHANES - in the U.S.). This monitoring will allow for establishing a baseline of exposure and the ability to track trends in environmental health.

Health professionals of all sorts can play a role (alongside academic researchers) in expanding such information and understanding. Therefore, there is need to incorporate pediatric and general environmental health education modules into curricula, particularly in the training for family physicians, obstetricians, pediatricians, and midwives, nurse practitioners and social workers.

Recommendations for regulatory action are often but not exclusively focused on the federal government. However, changes to, and where necessary replacement of, risk assessment and risk management practices are relevant for all regulatory agencies involved in standard-setting. In addition to the problems associated with standards generated by risk assessment and risk management, the reluctance, inertia and slowness of the federal government to regulate toxic substances, consumer products or pesticides is a serious problem and unnecessarily puts children's health at risk. The situation is made worse by the removal of some areas of standard-setting to the Canada-Wide Standards process established under the Environmental Harmonization Accord; a process which this review finds is neither intentionally nor actually setting standards to protect children's health.

Political will and government funding is a final ingredient that is critical to achieving success in the realm of research and education in children's environmental health issues and in setting child-protective standards.

Consolidated List of Recommendations

In addition to the preceding summary of recommendations, the following list consolidates the recommendations made in Chapters 2, 4, 5, 6, 7, and in the two Case Studies. Only a selected list is included from the 45 recommendations in the Pesticides Case Study. As well, some recommendations are repeated since they are relevant in more than one chapter.

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

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.

Chapter 4: Risk Assessment and the Precautionary Principle

Risk Assessment

9. The use of "comparative risk assessment" and "cost-benefit analysis" in environmental standard-setting should be monitored and evaluated for effectiveness in environmental and health protection versus their narrower ability and purpose of cutting costs.

10. All regulatory agencies in the federal and provincial government need to explicitly acknowledge the scientific uncertainties and limitations of risk assessment for deriving environmental standards.

11. The harmonization (either NAFTA-imposed or as cost-saving measures) of Canadian pesticide standards with those being developed in the U.S. should be undertaken as a preliminary step towards, or at least should not undermine Canada's ability to move towards, more precautionary standards. Such standards should include more rigorous and stepwise application of child-protective safety factors during both exposure assessment and dose-response assessment, as well as assessments of aggregate exposure, cumulative and synergistic effects, and the ability to implement a full ban on persistent organic pollutants. Child-protective safety factors and a weight-of-evidence approach should continue through to the risk management stage of setting new or revised standards for pesticides and all environmental pollutants.

12. Further research is necessary regarding whether and how commitments made under international trade agreements constrain Canada's ability to set protective standards. In addition, given the influence on Canada of standard setting in the United States, further research is required to determine the degree to which final standards established in the United States are set at numbers influenced by the possibility of legal challenge, including on a constitutional basis, so as to be able to recognize when a resulting standard is weaker than it should be in the Canadian legal context.

13. The Canadian Pest Management Regulatory Agency, as part of a government-wide approach, should immediately implement a policy of refusing to accept from pesticide companies new or existing toxicity test data derived from experiments on human "volunteers."

Precautionary Principle

14. Although the federal government has committed to the precautionary principle in the *Canadian Environmental Protection Act*, the *Oceans Act*, and in other policy pronouncements, there is little evidence that the principle has been operationalized. It is therefore recommended that the federal government develop a national implementation strategy to further the precautionary principle that includes:

(a) Change in the burden of proof: a process that ensures that those parties creating a threat of harm, such as those that produce a new substance that is being assessed or that introduce new products, have the onus to establish that such substances or products are safe, rather than having government establish that they pose a risk of harm;

(b) Weight of evidence: a protocol that allows decisions at each step in a risk based decision-making process (i.e., during all stages of both risk assessment and risk management) to be based on the weight of evidence approach rather than waiting for an extremely high standard of proof;

(c) Pollution Prevention: a commitment to operationalize pollution prevention through the development of a regime for bans and phase-outs of inherently toxic substances as well as pollution prevention standards for industrial sectors;

(d) Just Transition: a commitment to ensure the application of the principles of Just Transition for workers affected by toxic substance phase-down and phase-out;

(e) Public Participation: in recognition of the political and ethical implications of environmental and risk-based decisions, a commitment to make these decision-making regimes more transparent and open to the involvement of the public.

15. At this time, there is little evidence in provincial law or policy that Ontario is committed to the precautionary principle. It is recommended that the province of Ontario development a regulatory commitment to the precautionary principle together with a strategy to operationalize the principle similar to that described in Recommendation 5 above.

16. It is recommended that both Ontario and Canada adopt a definition of the precautionary principle that is more expansive than the definition found in the Rio Declaration, and preferably one similar to that found in the *Wingspread Statement on the Precautionary Principle*, which states:

    When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause-and-effect relationships are not fully established scientifically.

Chapter 5: Air

Air: Recommendations for Ontario

17. The Ontario standard setting plan is proceeding and should be encouraged to reach timely results with respect to the priority substances identified for review. However, the ultimate standards adopted in the group of eighteen contaminants currently under review and the fifteen yet to be proposed are highly dependant upon the approaches taken by Ontario in the next "risk management" stage of the process. The Ministry of the Environment should follow through with the development of a transparent, detailed and specific plan for finalization of these standards, as soon as possible. For carcinogens, the standards should in all cases be established at the risk level of no greater than ten to the minus six; with specific time frames for compliance being specified in the standard, if not immediately. No time frames should exceed five years for any substance, regardless of "implementation issues."

18. Research with respect to the evidence and data gaps for non-carcinogenic risks (for example endocrine disruptors and other health end-points) is a high priority for incorporation into standard-setting exercises and should be supported by the Ontario government.

19. Ontario should proceed with its own review of all of the priority air contaminants, originally identified, regardless of whether any of these are also in a Canada-Wide Standard or other federal provincial process. Ontario should ensure that all of the air contaminants in the province are regulated in the same manner and to the same risk levels.

20. Ontario should place special emphasis on standards for nitrous oxides and particulate matter in its own standard setting process because of the impact of these contaminants on children's health and because of the levels in which they are found in the Ontario environment.

21. Ontario should drastically improve its ozone commitment and should actively work to support a stringent ozone Annex between Canada and the United States.

22. Ontario should immediately repeal its plan with respect to emissions trading in the electricity sector and replace it with a plan that will ensure improved air quality from this sector within five years.

Air: Recommendations for Canada

23. The Canada-Wide Standards process under the Environmental Harmonization Accord Standard Setting Sub-Agreement is ineffective for protecting children's health and should be repealed with respect to air contaminants.

24. The Federal Minister of the Environment should establish standards on a health protective basis rather than pursuant to a Canada-wide consensus approach, and without risk management considerations.

25. Health protective standards should be published regardless of implementation issues.

26. Where implementation barriers are identified that require industry sector adjustments, sectoral time frames for compliance should be immediately established and subject to third party review.

27. All opportunities to improve current commitments (for example, shorter time frames, or more stringent standards) should be vigorously pursued.

28. A stringent ozone Annex should be reached with the United States as soon as possible.

Chapter 6: Toxic Substances

29. Environment Canada should clarify what it means by "cost effective measures" when applying the precautionary principle and ensure that "cost effective" comprehensively accounts for human health costs, particularly for children, affected by exposure to toxic substances.

30. Environment Canada should commit to take regulatory action on all substances found to be toxic under the *Canadian Environmental Protection Act* and employ processes such as the Strategic Options Process as a means to consult stakeholders on those regulatory initiatives.

31. Environment Canada should exercise its discretion under the *Canadian Environmental Protection Act* to require pollution prevention planning for all "CEPA-toxic"substances up to and including establishing timetables for phase-down and phase-out of inherently toxic substances.

32. Resources and efforts should be applied to in-depth focussed research on the effects of toxic substances on vulnerable populations, particularly children. This focussed research should directly inform the assessment processes within *CEPA* as well as in Ontario processes.

33. Criteria should be established to identify as "*CEPA* toxic" or "*CEPA* –equivalent" those substances not currently subject to *CEPA* to ensure they are made subject to the Toxic Substances Management Policy.

34. The ARET (Accelerated Reduction/Elimination of Toxics) program should not be renewed until an in-depth, impartial assessment is undertaken. Unless that assessment reveals unequivocal evidence of sustainable and

actual progress, toxic substances should not be dealt with through voluntary measures but through regulatory measures.

35. The Canada-Wide Standards process under the Environmental Harmonization Accord Standard-Setting Sub-Agreement should be repealed with respect to toxic substances.

36. The federal government should take a leadership role in the negotiation of the proposed Legally Binding Treaty on Persistent Organic Pollutants. In particular, Canada should support language in the treaty that calls for the elimination of both products (such as pesticides) and by-products (such as dioxins) in the proposed treaty as opposed to a mere reduction regime supported by some countries.

37. The province of Ontario should re-vitalize its list of candidate substances to be phased out or restricted, and this list should be developed using the precautionary principle.

38. The province of Ontario should enhance its policy and legal framework for pollution prevention.

Chapter 7: Consumer Products

39. The *Hazardous Products Act* should be amended to provide Health Canada with the power to issue mandatory consumer product recalls.

40. Health Canada should develop a proactive Materials Use Policy that incorporates a precautionary and preventative approach to avoiding the use of toxic substances in consumer products.

41. An area for further research beyond the scope of the present study should include a review of the child-specific *Hazardous Products Act* regulations reviewed herein to determine whether they were developed in a precautionary manner or in reaction to identified hazardous or lethal situations.

42. Further research is necessary to investigate the impact of international trade agreements on both the ability and inclination of Canadian regulatory agencies to set child-protective domestic regulations.

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

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

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

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

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

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

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

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

Case Study #2: Regulating Pesticides to Protect Children's Health

50. The *Pest Control Product Act*'s core test for judging the acceptability of a pesticide (unacceptable risk of harm) should be specifically defined so that it can be applied in a transparent and consistent manner throughout the risk assessment-risk management process. An essential amendment to the Act, to complement Recommendation 5 below, is to designate persistent and bioaccumulative substances as presenting an unacceptable risk of harm.

51. The *Pest Control Products Act* should be amended to include a requirement to act in a precautionary manner, for example, when the weight of evidence points to the potential for "unacceptable risk of harm." In keeping with this approach, Canada should follow Sweden's lead with legislative amendments to specify inherent characteristics of pesticides that justify de- registration including criteria such as very high acute toxicity, endocrine disruption, probable human carcinogenicity, and neurotoxicity all of which should be considered synonymous with "unacceptable risk of harm."

52. The Pest Management Regulatory Agency (PMRA) should fulfill its commitment to incorporate the Toxic Substances Management Policy (TSMP) in pesticide regulation. This activity should include immediate bans (or de-registrations) on pesticides which are persistent and bioaccumulative (Track 1 substances) without wasting time and resources on re- evaluation. In keeping with this approach, the PMRA should immediately revise its TSMP Implementation Policy to eliminate the ability to register Track 1 pesticides and to cancel registration of pesticides contaminated with persistent organic pollutants pursuant to the TSMP.

53. Several toxicity tests that are currently conditionally-required should become standard requirements. This includes developmental neurotoxicity testing on young animals, which is particularly important for gauging risks to children's health. Similarly, tests for endocrine disruption that are protective of children should be made a standard PMRA test requirement.

54. The PMRA should consider the potential effects on human health of occupational/bystander and food/drinking water exposures on an aggregated basis.

55. The PMRA should consider the potential effects on human health of cumulative exposures to pesticides that act via common mechanisms of toxicity.

56. The PMRA should adopt a requirement similar to that found in the U.S. *Food Quality Protection Act*, mandating the application of an uncertainty factor with a minimum value of 10 in order to account for potential pre- and post-natal developmental toxicity and the incompleteness of toxicity and exposure data for children. The uncertainty factor could have a higher value in situations of relatively high uncertainty regarding toxicity and children's exposure.

57. The PMRA should expeditiously complete on-going re-evaluations including several that were initiated close to 20 years ago, such as for pentachlorophenol.

58. The PMRA should expeditiously fulfill its commitment and complete development of its policy on formulants. The PMRA should release its policy to the public for comment and revision. Once completed, the PMRA should effectively implement and enforce its policy. The policy should set out how the PMRA will use the EPA formulant classification system and toxicological database. The policy should also include an explicit enumeration of rigorous testing requirements for new and non-EPA-listed formulants. These requirements should be effectively enforced.

59. The PMRA should develop a pesticide reduction policy and should apply its policy to all PMRA decisions and activities including as a first priority the reduction of pesticides important in children's diets and in use categories of most relevance to children's exposure circumstances including parks and institutional facilities geared primarily to children.

60. The PMRA should ensure that the public has access to basic information that is essential to an understanding of the risks posed by pesticide exposure. Information availability requires that:

    a) The PMRA disclose all pest control product ingredients and provide access to all information upon which registration and other regulatory decisions are based;
    b) If necessary, the public health and environmental protection provisions in the *Access to Information Act* be invoke; and
    c) Public notification mechanisms regarding the initiation and status of new regulatory decisions be developed.

61. The federal government should fulfill its commitment and legislate an adverse effects reporting requirement that explicitly includes information regarding the adverse effects of pesticide exposure on children. To be effective this reporting system requires first that:

    a) effort is placed on ensuring the education of primary care health-care practitioners (i.e., family physicians, pediatricians, emergency room physicians, obstetricians and midwives, nurse practitioners and social workers about the health effects, both acute and chronic, of pesticides on children in order that they can better clinically detect these cases; and
    b) a central registry be established, federal or provincial, of adverse clinical responses to pesticides, in an attempt to gather appropriate data.

62. In recognition of the greater exposure and sensitivity in children to the toxic effects of pesticides, the federal government's National Children's Forum must allocate the necessary resources to honour longstanding domestic and international commitments to improving legal and policy tools, including application of the precautionary principle, to protect children from toxic substances, including pesticides.

Chapter 1 - Introduction

Chapter 1 - Introduction

1.1 Overview and Study Boundaries

This study represents Phase I of the *Children's Health Project,* a collaboration of the Canadian Environmental Law Association and the Ontario College of Family Physicians Environmental Health Committee. It addresses the risks to children's health from environmental contaminants and the adequacy of regulatory responses. It explores whether the rules or standards in place and the manner in which those standards are set ensure that children's health is protected.

Several very large areas of inquiry are at issue here: the amount and availability of toxins in a child's environment; the effects of toxins on a child's health; and the control of those toxins by regulatory agencies. In each of these areas there are thousands of different types and sources of toxic substances, all with varying degrees of usage and ability to create environmental or consumer product contamination. Equally as diverse are the potential health effects from toxic substances. There are also numerous regulatory agencies at the federal and provincial levels of government often with overlapping, conflicting or confusing mandates. The passage of time is also an important consideration in this investigation. Regulatory responses in recent years have increasingly recognized children as a sensitive sub-population. A review of regulatory action must therefore make a distinction between current and past approaches or techniques to the evaluation of environmental contaminants.

Over all of these areas of inquiry hangs a cloud of uncertainty and controversy due to the enormous complexities involved, incomplete information, constraints on both resources and research capabilities, differences in scientific opinion, and vested or competing interests of different stakeholders.

The study focus is children in Ontario. A wide net is cast across the internationally reported and peer-reviewed literature concerning the exposure to and effects of environmental contaminants in children. However, exposure data from Ontario, where these are known, are provided to be able to talk about children in Ontario.

Similarly, the review of standard-setting addresses the Ontario and federal governments. Hence, although more information is provided with respect to Ontario, for those standards set by the federal government, the study provides a review and analysis that is applicable to standards affecting children across Canada. Central to the analysis is a review of the history, ongoing evolution and appropriate role for risk assessment in standard-setting.

The focus is also primarily on chemical and metal pollutants. Time and budget constraints did not permit a more comprehensive review. Not included are physical contaminants (including electromagnetic frequency, radiation, radon or radionuclides), biological agents (such as mould, fungi, bacteria) or environmental tobacco smoke and other indoor air pollutants. Although these are important areas for children's health, the focus on chemicals and metals provided some bounds on a very large field of inquiry.

The study is further bounded by the exclusion of some areas of standard-setting. Included are detailed reviews of standard-setting for air, pesticides, consumer products and toxic substances. These areas were chosen since they represent the most significant areas of chemical exposure for children and are also the areas where standard-setting is primarily focused on evaluations of human health effects. Categories that are not included in detail are water, food (other than pesticides, i.e., there is no review of standards for genetically-modified foods, food additives or food irradiation), soil, or *new substances* as these are defined

under federal environmental law. The reviews of standard-setting are mainly descriptive and provide either preliminary analysis and in some cases recommendations for further detailed analysis and/or action. A very detailed review and analysis of standard-setting is provided in two case studies: the first on the "cautionary tale" of lead and the second on the regulation of pesticides by the federal government. The review of risk assessment in Chapter 4 is relevant to all categories where human health effects are assessed and standards established.

A central question for this study is whether the rules in place concerning environmental contamination are *intentionally protective* of children's health. Further, where standards are or are increasingly established to be *intentionally protective* of children, the question remains whether children's health is indeed protected. This study provides a foundation for answering these questions and a detailed set of recommendations for further research and action steps, including educational activities. Phase II of the *Children's Health Project* is now underway with the translation and summary of this large study into a variety of academic and popular educational media and the preparation of further educational materials on standard-setting.

1.2 Toxic Substances and Children's Health: A Growing Concern

There is growing world-wide concern that toxic substances in the environment and consumer products are, or may be, particularly harmful to the health of children. We know that children's health has been harmed and continues to be harmed by certain environmental contaminants. Historically, lead is perhaps the most well known example. By the 1980s, environmental lead contamination, primarily from the use of lead in gasoline, was high enough to affect the health of literally millions of children in most industrialized countries, including Canada. Average blood-lead levels were at or above the range where lead can lower IQ, reduce attention span, and create behavioural problems. This situation has improved dramatically with the removal of the primary exposure source via the decision to ban gasoline lead additives.

Recent decades have seen substantial increases in childhood asthma, allergies and respiratory problems. Environmental contaminants such as ozone, sulphates and particulate matter aggravate symptoms associated with these conditions and may be causes. According to the Ontario Medical Association:

> ... the available evidence on the impact of air pollution on children shows that, when all the data are taken together, there is no doubt that relatively low levels of pollution are responsible for increased morbidity in children.[1]

The concern extends far beyond cases for which clear, causal connections have been established. Increasing concern exists about a wide range of chemicals that, both animal studies and human clinical evidence suggests, are neurotoxic, suspected or known carcinogens, or that can impair immune system functioning. Childhood cancers, such as leukemia, bone and brain tumours are rising and may be related to immune suppressive effects, or, to the direct genotoxic effects of environmental toxins. More question marks surround the relationship between exposure to environmental contaminants and adverse pregnancy outcomes including spontaneous abortions, increased rates of stillbirth, and anatomical abnormalities in the newborn.

Endocrine disrupting chemicals are also of concern. Studies in wildlife and laboratory animals are showing effects of hormone disruption on reproductive development, behaviour and thyroid or immune system

[1] Ontario Medical Association, 1998. *OMA Position Paper on The Health Effects of Ground-Level Ozone, Acid Aerosols and Particulate Matter*, p. 11. www.oma.org/phealth/ground.htm

functioning. Disturbing trends in human populations in industrialized countries include declining sperm counts, increasing prevalence of prostate, testicular, and breast cancers, reproductive organ abnormalities and fertility problems, all of which may be linked with the presence of endocrine disrupting chemicals in the environment. Further, there is increasing understanding as to the notion of "windows of vulnerability." These windows are highly sensitive stages during fetal, infant, child and adolescent development when chemical exposures that may be otherwise safe for adults or even during other periods of childhood, might cause or contribute to serious or fatal health effects in later life.

Chapter 2 provides a detailed review of the relationship between children's health and environmental contaminants by addressing the factors influencing children's exposure, uptake and susceptibility to contaminants and the issues of concern at each of the developmental stages of children. Note that this study defines "children" as including each of the developmental stages discussed in Chapter 2, from *in utero* to adolescence. Chapter 2 explores the diverse environmental media and exposure routes for contaminants and the many factors influencing contaminant exposure and susceptibility that are unique to children and that frequently place children at greater risk. The chapter goes on to describe a range of contaminants and their known effects focusing on classes of chemicals and metals for which the existing knowledge base reveals cause for significant concern. A detailed review is provided of the known, suspected and controversial nature of health problems in children related to environmental exposures and concludes with an assessment of trends in children's environmental health problems and the future of children's environmental health. Building upon Chapter 2, the two case studies provide greater detail in each of the areas noted above. The first addresses a single pollutant, lead, and all of its many exposure sources and well-known health effects. The second addresses pesticides as a class of pollutants and focuses on those for which the greatest amount of concern exists about possible health effects.

1.3 Toxic Substances: Underestimated Volumes and Information Gaps

Every year, more substances are introduced into the Canadian marketplace. These are added to the more than 23,000 substances in commercial use in Canada. In addition, there are over 500 active ingredients in pesticides registered for use in Canada, over 300 of which were approved for use before 1981 (and over 150 approved for use before 1960), under far less rigorous testing requirements than exist today.

Of the over 23,000 substances in commercial use in Canada, probably less than 15% of these has a full or comprehensive data set evaluating toxicological properties.[2] A preliminary first step towards addressing this problem has been taken by Health Canada with the creation of a detailed inventory of 72 activities within that agency on children's health and the environment.[3] Further "gaps analysis" continues across the federal government.

Additional gaps in information occur with respect to pollutant releases. Estimates are incomplete

[2] The figures for south of the border are comparable. The U.S. Office of Technology Assessment (OTA) estimates that there are over 62,000 chemicals in commerce in the United States with 1500 new chemicals introduced each year. Concerning the approximately 15,000 chemicals that are deemed to be produced in high volume, the EPA recently reported that complete health and environmental effects data are available for only 7% of these. Source: Congressional Research Service, Issue Brief to Congress. No. 94036: *The Role of Risk Analysis and Risk Management in Environmental Protection*. November 5, 1999. By L. Schierow, pp. 8-9. Available at: www.cnie.org.nle.rsk-1.html

[3] Inventory on Children's Health and the Environment – Activities at Health Canada. Fax memo to Loren Vanderlinden from Monica McAuley, Program Development Bureau, Health Protection Branch, Health Canada, April 9, 1999.

concerning the total volumes of toxic substances that are released to the environment. The Canadian pollution inventory, the *National Pollutant Release Inventory* (NPRI), reports that for 1997, over 160,000 tonnes of chemicals, (over 15,000 tonnes of which are toxic and carcinogenic), were either released into the Canadian environment or transferred from source facilities.[4] Despite the huge volumes of chemicals these numbers suggest, the NPRI focuses only on large facilities, includes only a limited number of substances, does not include mobile sources and as a result reflects only a small fraction of total pollutant emissions to the environment. The total volume reported in the NPRI is derived from fewer than 2000 reporting facilities. Hence, similar to the Toxic Releases Inventory (TRI) in the United States, inventories like the NPRI and the TRI may only estimate some 5% of actual emissions to the environment.[5]

According to annual reports released for three years in a row by the Commission for Environmental Cooperation, Ontario was, in 1994 and 1995, the third worst polluter in North America, behind Louisiana and Texas, and as of 1996 was ranked the second worst. Over 68 million kilograms of pollutants were released into the environment or transferred off-site in 1996 from reporting facilities in Ontario.[6] The most recent reporting of NPRI data for 1997 reveals a continuation of a trend towards lower overall emissions in Ontario by reporting facilities during the 1994-1997 period. Whether or not this drop in 1997 data will affect Ontario's ranking as among the top polluters in North America will depend upon the annual comparisons of all North American data conducted by the CEC. That comparison will likely be reported, as in years past, in August of this year (2000).

1.4 ARE REGULATIONS KEEPING UP?

Regulatory responses have not, in past or currently, always adequately considered children's health. Children can be more vulnerable to environmental contaminants than adults for many reasons mostly related to immaturity and sensitivities associated with growth and development. Exposure can also be much greater, again due to numerous circumstances that are unique to children. Both exposure and uptake can be greater under conditions of poverty, an increasing reality for large numbers of children in Ontario.

The reality that children's exposure patterns and special vulnerabilities are not adequately accounted for in regulatory controls on toxic substances was recognized in 1997 when Canada signed the *1997 Declaration of the Environment Leaders of the Eight on Children's Environmental Health*, which states:

> We pledge to establish national policies that take into account the specific exposure pathways and dose-response characteristics of children when conducting environmental risk assessments and setting protective standards.[7]

This report is, in part, an assessment of whether this commitment is being achieved to protect children in

[4] Environment Canada, 1999. *National Pollutant Release Inventory - 1997.*

[5] United States General Accounting Office, *Toxic Chemicals: EPA's Toxic Release Inventory is Useful but Can be Improved.* GAO/RCED-91-121, June, 1991, p.3; see also: United States General Accounting Office, *Pollution Prevention: EPA Should Re-examine the Objectives and Sustainability of State Programs.* GAO/PEMD-94-8, January, 1994, p.54 and multiple additional cites therein.

[6] See the three annual reports of the Commission for Environmental Cooperation, 1997, 1998 and 1999. All entitled *Taking Stock - North American Pollutant Releases and Transfers – 1995 (-1996)(-1997).* Legal Deposit-Bibliotheque national du Quebec. Pages 21, 31 and 47, respectively.

[7] *1997 Declaration of the Environment Leaders of the Eight on Children's Environmental Health*, Environment Leaders' Summit of the G7 countries plus Russia (the "Eight"), Miami, Florida. May 6-7, 1997.

Ontario. With a focus on Ontario, the scope of this report is both provincial and federal insofar as laws, regulations and policies exist at both jurisdictional levels that will affect the health of children in Ontario. Chapter 3 provides an introduction and overview to this constitutional context whereby roles and responsibilities are shared and/or divided between the two levels of government. For each of the federal and provincial departments, or joint federal-provincial organizations, relevant to this inquiry, Chapter 3 describes the relevant mandates, specific regulatory or policy tools that are applied to standard-setting as well as a sense of how these players relate to each other to accomplish their particular standard-setting functions.

The central scientific, legal and policy analysis of this report is the review in Chapter 4 of *risk analysis* or *risk assessment.* Chapter 4 looks at how standard-setting to assess human health impacts has been a roughly 30 year process of refining *risk analysis* or *risk assessment* methods. A detailed look at trends and recent activity in the United States is included due to the significant impact on Canada of approaches taken in the United States. The "science behind the assessment" takes a detailed look at how inferences of causation can be drawn from epidemiological evidence. The Chapter also contrasts the standard or burden of proof in scientific versus legal settings and looks at how "weight-of-evidence" and "precautionary inference" approaches offer useful input to alternative models for setting environmental standards. To conclude, the Chapter addresses the role of the *precautionary principle* or a *precautionary approach* to standard setting.

Chapters 5 through 7 then provide detailed reviews and/or overviews of actual standard-setting processes and techniques with a focus on health-based standards and the predominance of risk assessment in their derivation. Again, the choice was made to focus on those human-health based standard-setting areas which are also of increasing concern for children's health, i.e., air, consumer products, and toxic substances. In addition, more detailed regulatory analyses and recommendations contained in the two case studies provide illustrations and lessons for the study as a whole.

1.5 Research Methods

This review of children's health issues and environmental standard setting was assembled by a team of environmental, legal and medical researchers.

The summary of the relationship between children's health and environmental contaminants (in Chapter 2 and in the two case studies) was founded on an extensive review of the most current literature available. Researchers accessed a variety of sources including research publications (both primary and secondary sources), unpublished documents (conference proceedings, reports and data from research agencies, government ministries and public interest groups, among others) and electronic sources (web pages for a variety of agencies). Data gathering was both systematic and exploratory. Medline was the primary tool used to search published sources, and Internet search engines such as Lycos, were used to search the web. Data were gathered for a number of different subject areas, reflecting the fact that Chapter 2 examines children's environmental health issues from several perspectives. Among the topics researched were: growth and development patterns in the young, developmental toxicology, physiological and behavioural features of children affecting exposure, environmental contaminants of concern (as defined in Chapter 2, for the purposes of this study) and their health effects, children's health problems related to environmental exposures, and environmental exposure monitoring data relevant to children in Ontario.

The review of standard setting in this report included an extensive review of the available literature using research methods and information sources similar to those noted above. Extensive use was made of the websites of regulatory agencies and non-governmental organizations in Canada and the United States. Particularly useful for the US research was the availability of Congressional Research Service Reports on

the website of the Committee for the National Institute for the Environment, based in Washington.

The involvement throughout the project of the librarian with the *Resource Library for the Environment and the Law* (located in the offices of the Canadian Environmental Law Association) provided valuable research assistance and cataloguing expertise. Since Phase I of the *Children's Health Project* is intended to provide a foundation for further work, the extensive collection of research materials for both the health and legal sides of this project have been exhaustively referenced. This detailed referencing has been done to facilitate print and internet access to source materials during Phase II as well as for use by other researchers working in this field.

The literature reviews for both the health and legal areas of research were supplemented by interviews of key informants in medicine, pediatrics, public health, research agencies, government departments, non-governmental organizations, public interest groups and community associations. Key informants were an invaluable resource providing important theoretical perspective and contextual information that allowed the research team to more adequately characterize environmental health and standard-setting issues for Ontario's children. They were also helpful in directing team members to additional key informants and current literature on children's environmental health and standard-setting.

1.6 REFERENCES CITED

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

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

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

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[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		• The **most** vulnerable periods according to different body systems are:[9]
→ Embryonic	= implantation to 8 weeks	CNS, brain - weeks 3 through 5+
→ Fetal	= 9 weeks to birth	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	= birth to 1st month	• Brain is 10% of adult volume at birth, ∴ undergoes considerable growth postnatally (until about age ten)
→ neonatal	= 1 to 12 months	• Myelination of nerves (until adolescence)
→ postneonatal	postnatal	• 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

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[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)

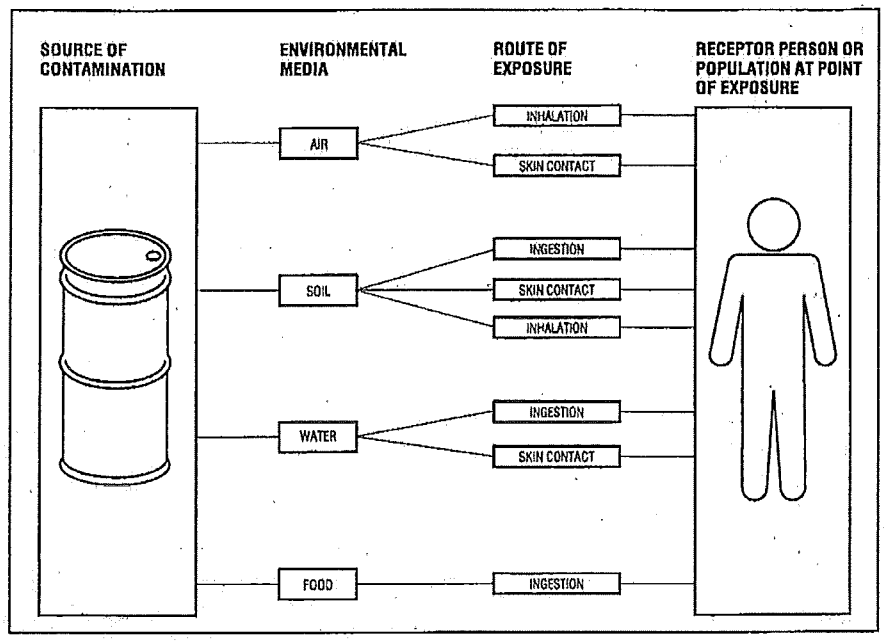

Figure 2.1. The pathways of human exposure to environmental contaminants.
(Source: Health Canada, 1995a, *op.cit.*)

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.

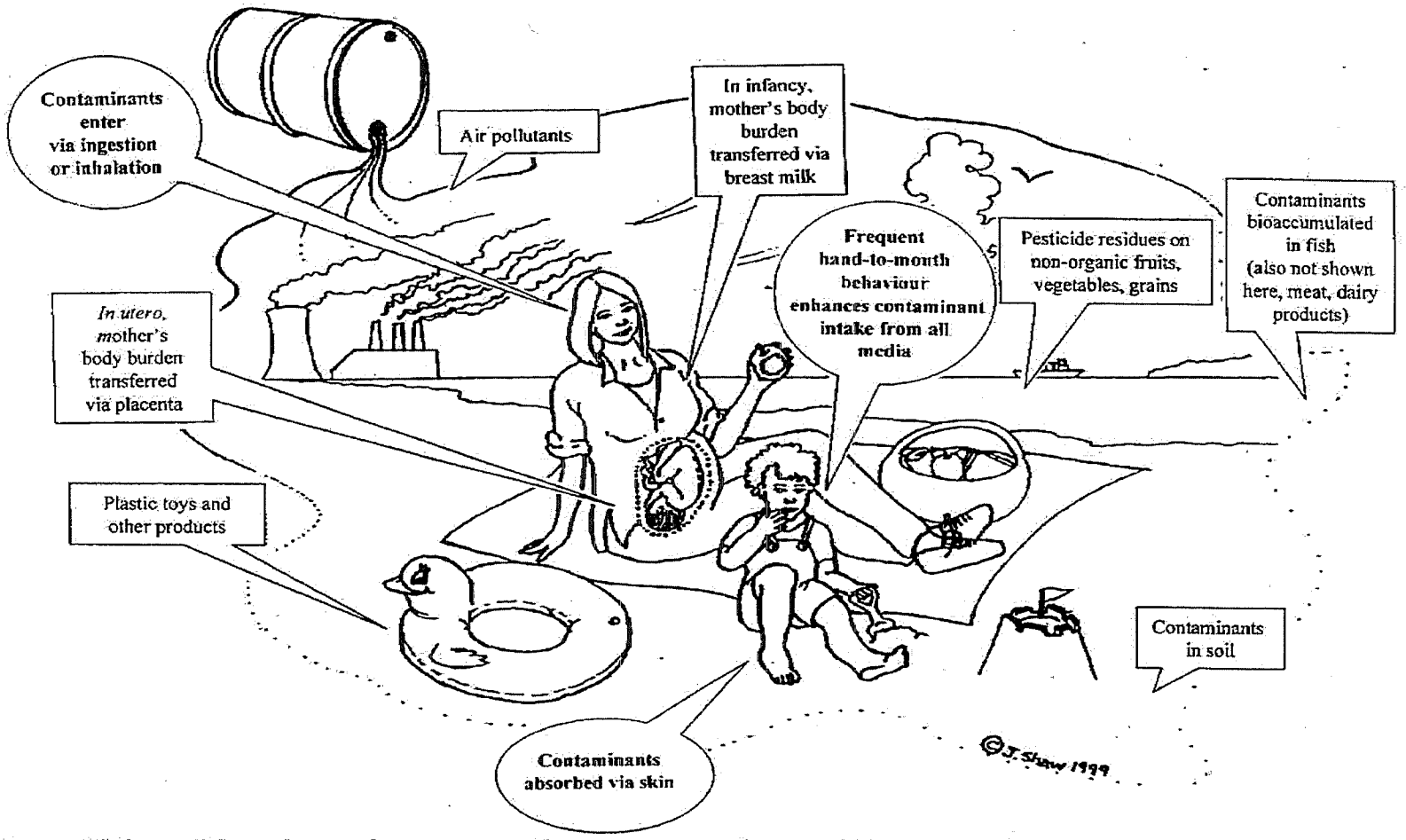

Figure 2.2. Some of the pathways of exposure to environmental contaminants for children.
(Illustration by Jacqueline Shaw.)

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

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[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

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[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

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[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

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[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

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[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]

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[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

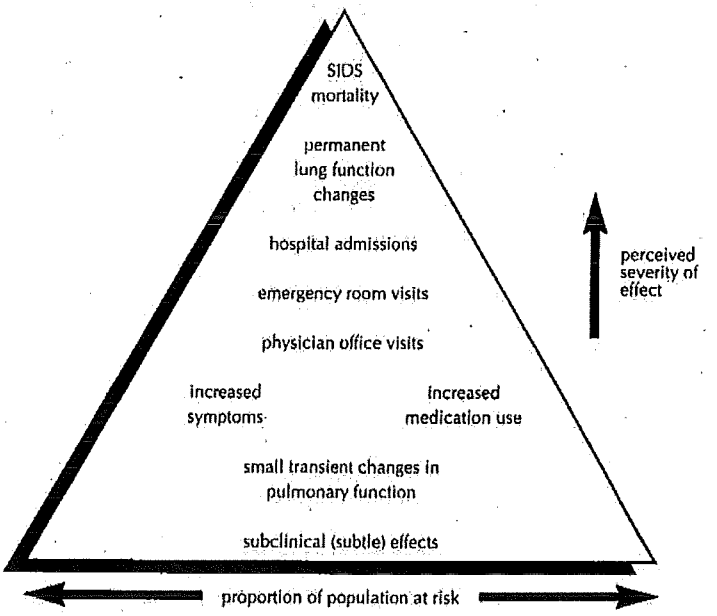

Figure 2.3. The Health Effects Pyramid.
(Source: Raizenne, M. *et.al.* Air pollution exposure and children's health. *CJPH.* 89 (Suppl 1) (1998), S44.)

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).

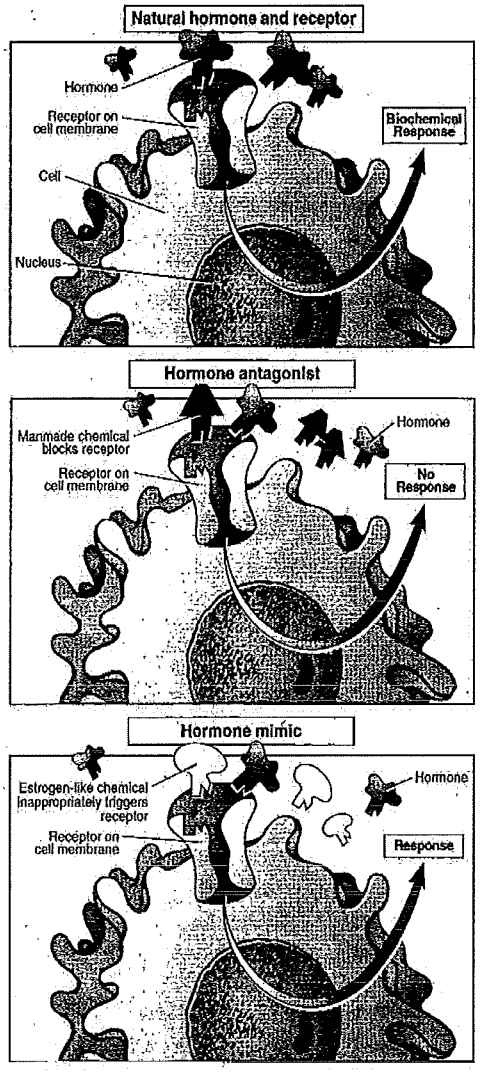

Figure 2.4. The mechanisms of endocrine disruption. (Source: CALPIRG and PSR, 1998, *op.cit.*)

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

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[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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Chapter 3: The Standard Setting Framework

Chapter 3: The Standard Setting Framework

3.1 INTRODUCTION

It is clear from the preceding review that environmental contamination can be a significant factor in the health of children. As the summary for Chapter 2 notes, very large numbers of children have been and continue to be affected by contaminants such as lead as well as by air pollutants that can contribute to serious respiratory problems, including asthma. For the less understood pollutants, i.e., the vast majority of them, the potential exists for equally huge numbers of children to be affected. For example, although there is a high degree of uncertainty as to the nature of the causal relationship (if any) between chemicals capable of endocrine-disrupting effects and negative health effects in children, the potential scale of the problem is immense in terms of numbers of children that could be affected. The severity of health effects is also deeply troubling. Increases are occurring in the rates of childhood cancer and learning disabilities. Again, although there is a high degree of uncertainty, environmental pollutants could well be contributing factors. Although still relatively rare, such effects can be either fatal or have very serious, often permanent, negative health effects.

The public policy response to these issues is a complex story. One objective of this report is to simply make sense of how the system operates in order to then assess whether it is responding adequately to the known problems and potential risks to children.

To begin, this chapter describes the basic regulatory framework of the federal and provincial governments for the standard-setting areas covered in this study. It summarizes, using information provided by these government agencies, the governmental players involved, their goals/mandates and legal and policy tools and their interactions with each other. It in no way attempts to assess the validity of the self-descriptions provided. An assessment of whether the goals as stated have been accomplished have been accomplished with respect to children's health is the subject of chapters 4 -7 and particularly the two case studies.

Additionally, the standard setting areas covered in this review are rapidly changing, and thus the regulatory framework is stated as at the date of writing. Before describing the individual departments of relevance to the standard-setting reviewed in this study, the constitutional context is described whereby powers are divided between the federal and provincial governments.

3.2 THE CONSTITUTIONAL CONTEXT

As a Constitutional Monarchy, Canada's legislative powers are split between the Federal, Provincial, and increasingly, the First Nations levels of government. Britain provided Canada with its rights and responsibilities under the *Constitution Act, 1867*[1]. The *Constitution Act, 1982*[2] in effect "patriated" this legislation, and provided that henceforth all constitutional changes would be made in Canada.[3]

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[1] *Constitution Act, 1867*, (U.K.), 30 & 31 Vict., c.3 (formerly the *British North America Act*).

[2] *Constitution Act, 1982*, being Schedule B to the *Canada Act 1982* (U.K.), 1982, c. 11 [hereinafter *Constitution Act*].

[3] M. Fagan & D. Lloyd, *Dynamic Canada: The Environment and the Economy* (Toronto: McGraw-Hill Ryerson, 1991) at 210.

The *Constitution Act* provides for the federal division of legislative powers. It confers specific exclusive powers on both the Parliament of Canada and the Provincial legislatures and leaves to the Parliament a residual power to make laws for the peace, order and good government of Canada.

The *Constitution Act* does not specifically mention jurisdiction in respect of the environment. As a result, the courts have had the task of determining the authority for environmental law-making on the basis of the other powers the Act assigned to the federal and provincial governments. The courts have given legal authority for some environmental matters to the federal government, some to the provinces, and some to both levels ("concurrent powers").

Because there are areas of separate responsibility and areas of overlap, environmental standard setting entails considerable interplay between both levels of government. The regulation of pesticides provides a good example of the overlap that occurs between federal and provincial jurisdictions. The federal government determines which pesticides are permitted for use in Canada and specifies labelling and some use restrictions, while the provincial government decides on the allocation of the permitted uses through a system of permits and licenses.

In addition, "health" is not mentioned specifically in the *Constitution Act.* Thus it is not in the exclusive domain of the federal Parliament or the provincial legislatures. The courts have recognized that federal authority in matters of health protection derive largely from its constitutionally conferred power over criminal law. Criminal law allows Parliament to prohibit a public evil. The federal government can formulate and enforce regulations that establish the parameters of such prohibition. Under the *Constitution Act* the federal government has power to regulate "trade and commerce," but only when it crosses provincial/territorial or national borders.[4] The *Constitution Act* gives the provinces responsibilities over hospitals, property, civil rights and matters of a local nature.

3.3 THE FEDERAL GOVERNMENT

3.3.1 Introduction

Responsibility within the federal government for matters of health and environmental protection resides within the Departments of Health Canada, Environment Canada and Agriculture and Agri-Food Canada. For the purposes of the standard-setting areas covered in this study, only the first two departments are investigated here. Also relevant is the Pest Management Regulatory Agency, which reports to the Minister of Health.

The authority, responsibilities, structure and activities of each of these agencies is described below. Of note first however are two overarching policies that apply, in the first instance, to the federal government as a whole, and in the second, to Health Canada's activities related to children's health.

Toxic Substances Management Policy

In 1995, the Government of Canada introduced a Toxic Substances Management Policy. The policy calls for virtual elimination from the environment of toxic substances that result from human activity and that are persistent and bioaccumulative.[5] It also calls for "cradle-to-grave" (i.e., throughout their entire

[4] Health Canada, *Shared Responsibilities, Shared Vision: Renewing the Federal Health Protection Legislation, A Discussion Paper* (Ottawa: Minister of Public Works and Government Services Canada, 1998) at 6-7; http://www.hc-sc.gc.ca/hpb/transitn/index.html .

[5] http://www.ec.gc.ca/toxics/toxic1_e.html.

lifecycle) management of all other substances of concern that are released to the environment. The policy puts forward a preventive and precautionary approach to deal with all substances that enter the environment and could harm the environment or human health. The policy is intended to guide federal regulatory and non-regulatory programs by defining the ultimate management objective for a substance.

The policy is to be applied in all areas of federal responsibility and is to serve as the centrepiece of the Government of Canada's position in seeking to deal forcefully with toxic substances from domestic and foreign sources. It is intended to provide decision makers with direction, and sets out a framework to ensure that federal programs are consistent with the objectives of the policy.

The policy has two key management objectives. The first is the virtual elimination from the environment of toxic substances that result predominantly from human activity and that are persistent and bioaccumulative (Track 1 substances). The second is the management of other toxic substances and substances of concern, throughout their entire life cycles, to prevent or minimize their release into the environment (Track 2 substances).

1997 Declaration of the Environment Leaders of the Eight on Children's Environmental Health

The second overarching policy of note for federal government activities is Health Canada's 1997 Sustainable Development Strategy. As noted in Chapter 1, Canada was a signatory to the *1997 Declaration of the Environment Leaders of the Eight on Children's Environmental Health* and pledged to establish national policies that more appropriately take children into account when setting protective standards. Within Health Canada, this commitment has been further refined in that department's 1997 Sustainable Development Strategy[6], referring to the G8 declaration as follows:

> in accordance with the 1997 Declaration of the Environmental Leaders of the Eight on Children's Environmental Health [the Department plans to] implement actions on protecting children's health with respect to environmental risk assessments and standard setting, children's exposure to lead, microbiologically safe drinking water, air quality, emerging threats to children's health from endocrine disrupting chemicals and environmental tobacco smoke ....[7]

The implementation and effectiveness of both of these policies is the subject of this study. The TSMP is discussed specifically in Chapter 6: Toxic Substances.

3.3.2 Health Canada

3.3.2.1 Authority, Responsibilities and Co-ordination with Other Departments

The stated mission of Health Canada, a federal department, is "to help the people of Canada maintain and improve their health."[8] The mandate of Health Canada is to provide national leadership, collaboration and co-ordination in health policy, regulations, disease and injury prevention, health promotion, health information and knowledge, and First Nations and Inuit health, in addition to the delivery of services. It works with other federal departments and agencies to reduce health and safety risks to Canadians. It also

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[6] *Sustaining Our Health: Health Canada's Sustainable Development Strategy*, November 1997. (www.hc-sc.gc.ca/susdevdur).

[7] *Ibid.* at 26.

[8] *Health Canada: Performance Report for the Period Ending March 31, 1999* (Ottawa: Minister of Public Works and Government Services Canada, 1999) at 10-11; (http://www.tbs-sct.gc.ca/tb/key.html). See also Health Canada Web Site: http://www.hc-sc.gc.ca/english/about.htm#org.

administers a number of Acts which are pertinent to the present study: (1) the *Food and Drugs Act;*[9] (2) the *Hazardous Products Act;*[10] (3) the *Canadian Environmental Protection Act,*[11] which it co-administers with Environment Canada; and (4) the *Pest Control Products Act,*[12] administered by the Pest Management Regulatory Agency (PMRA) which reports directly to the Minister of Health. The PMRA is described further below and its operations are discussed in detail in the Pesticides Case Study.

Health Canada provides assistance to other departments and agencies whose responsibilities touch on health. For example, Health Canada's responsibilities under the *Canadian Environmental Protection Act* (CEPA), are discharged in close co-operation with Environment Canada and the provinces. Section 6 of CEPA establishes a Federal-Provincial Advisory Committee composed of representatives from Environment Canada, Health and Welfare Canada and the provinces. The Committee is responsible for co-ordinating action on the control of toxic substances including facilitating the establishment of nationally-consistent objectives.

3.3.2.2 Structure and Relevant Activities

Table 3.1: Health Canada - Internal Organization

Branch	Directorate / Bureau
Health Protection Branch	***Environmental Health Directorate / Environmental Health Program (EHP)***: • Bureau of Chemical Hazards • Product Safety Bureau • Radiation Protection Bureau • Office of Tobacco Control • Commercial Products Bureau • Toxic Substances Research Initiative Secretariat
Medical Services Branch	
Health Promotion and Programs Branch	
Policy and Consultation Branch	
Information, Analysis and Connectivity Branch	
Corporate Services Branch	

Source: http://www.hc-sc.ca/english/about.htm#org.

The above table sets out the 6 branches of Health Canada. Within each branch there are various Directorates which are further sub-divided into Bureaux or Offices. The Directorate or Program within the Health Protection Branch of relevance to this study is the Environmental Health Directorate/Environmental

[9] *Food and Drugs Act,* R.S.C 1985, c. F-27.

[10] *Hazardous Products Act,* R.S.C. 1985, c. H-3.

[11] *Canadian Environmental Protection Act,* R.S.C. 1988, c. C-15.3.

[12] *Pest Control Products Act,* R.S.C. 1985, c. P-9.

Health Program (EHP). Within the Environmental Health Directorate, there are several bureaux, all of which are listed. The following sub-sections provide the self-described mandate as well as the legislative or policy tools administered by the departments of relevance to this study.

Health Protection Branch (HPB)

The mandate of the HPB is "to protect Canadians against health risks in two broad areas, products and disease."[13] The HPB is set up to assess the safety, effectiveness and quality of drugs and medical devices. Its focus is to protect Canadians from potential health hazards associated with tobacco, cosmetics, food and radiation-emitting devices, pesticides, certain consumer products and working and living environments.[14] Further, it is to develop and enforce regulations under consumer and environmental protection laws, including the *Food and Drugs Act* and *CEPA*.[15]

Environmental Health Directorate

The Environmental Health Directorate, along with Regional Offices located throughout Canada comprises the Environmental Health Program (EHP). The role of the EHP is "to protect and improve the well-being of the people of Canada by assessing and managing the risks to health associated with the natural and technological environments."[16] The EHP administers legislation and agreements undertaken by the Canadian Government in the areas of safe living and working environments. Further, its mandate is to identify, assess and manage health risks and provide sound and timely advice on environmental factors that can influence health and safety. The two bureaux relevant to this study are the Bureau of Chemical Hazards and the Product Safety Bureau. Also relevant is the Toxic Substances Research Initiative Secretariat. Each is described in turn below.

Bureau of Chemical Hazards

The Bureau of Chemical Hazards concentrates on the effects on human health of chemicals and biological agents in the environment.[17] Its role is to identify chemicals to which people are exposed, assess their toxicity and quantify risk. It studies the health risks posed by chemicals and micro-organisms in air, soil, drinking water, and water for recreational uses. It manages health risks through the introduction of regulations, standards, and guidelines and by providing advice to other government departments, the provinces and the public.

The Bureau consists of three Divisions. The first is the Environment and Occupational Toxicology Division which conducts laboratory research and field studies to determine the toxic effects of chemical pollutants found in the natural environment, indoor environment and in the work place. The second is the Environmental Substances Division which conducts a mandatory assessment and management of health risks of new and existing chemicals and also assesses the products of biotechnology as prescribed under *CEPA* (see Environmental Contaminants project area, below). The third is the Environmental Health Effects Division which, among other responsibilities, monitors, researches and performs mathematical modelling to assess human exposure to chemical and microbial contaminants in natural and built environments and health effects associated with such contaminants, and develops health-based guidelines for air, drinking-water, and recreational-water quality.[18]

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[13] Health Canada, *Information: Health Protection Branch - Facts*, October 1998a.

[14] *Ibid.*

[15] http://www.hc-sc.ca/english/about.htm#org

[16] http://www.hc-sc.gc.ca/ehp/ehd/who/index.htm

[17] http://www.hc-sc.gc.ca/ehp/ehd/bch/index.htm

[18] http://www.hc-sc.gc.ca/ehp/ehd/who/bch.htm

The Bureau's New Chemical Substances and Biotechnology Products project area has responsibilities under the *CEPA*. Its role is the risk assessment of new chemicals and products of biotechnology for potential health risks.[19]

The Bureau's Environmental Contaminants project area deals with risk assessment and management activities for "Priority" chemicals and toxicology research in support of *CEPA*.[20] The project encompasses work associated with the Priority Substance List (PSL) established under *CEPA*. Substances on this list are of the highest priority for assessment to determine whether environmental exposure to them risks the health of Canadians or the environment.[21] The responsibility for assessing Priority Substances is shared by Health Canada and Environment Canada, and the PSL is established by both the Ministers of Health and the Environment.

Product Safety Bureau

The Product Safety Bureau's mandate is "to regulate and monitor compliance for the advertisement, sale and importation of hazardous or potentially hazardous products that are not covered by other legislation and to provide clients with information."[22] It administers the *Hazardous Products Act,*[23] which provides the authority to control, restrict or prohibit certain materials as well as the sale, importation and advertisement of other dangerous or potentially dangerous consumer and industrial products. Regulations are enforced through product inspections and market sampling.[24]

Toxic Substances Research Initiative Secretariat

The Toxic Substances Research Initiative Secretariat is a program within the Environmental Health Directorate, which provides funding for applied toxic substances research.[25] The Initiative supports research projects from both within and outside the federal government, as well as promoting partnerships between researchers in both sectors.

The guiding principle of the Initiative is to support scientific excellence and federal public policy objectives by, among other things, "[p]lacing emphasis on biological and chemical research which would benefit ecosystem health and priority population groups at risk, i.e., children, Aboriginal people and the elderly."[26]

3.3.2.3 Resources

Like many federal government departments, Health Canada has recently experienced significant budgetary cuts. For 1997, Canada's spending on health was estimated to be nine per cent of Gross Domestic Product, down from 10.3 per cent in 1992, placing Canada in the middle of the Group of Seven countries in relative

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[19] *Ibid.*

[20] *Ibid.*

[21] http://www.hc-sc.gc.ca/ehp/ehd/bch/env_contaminants/psap/psap.htm

[22] http://www.hc-sc.gc.ca/ehp/ehd/psb/mandate.htm

[23] *Hazardous Products Act*, R.S.C. 1985, c. H-3.

[24] *Ibid.*

[25] http://www.hc-sc.gc.ca/ehp/ehd/tsri/index.htm

[26] *Ibid.*

expenditure for public health.[27]

In 1998, the Health Protection Branch budget was approximately $230 million.[28] This budget had been reduced by about $25 million from its 1993/94 level. Additional funding was provided to new and priority areas such as AIDS and cancer research.[29]

In the 1999 budget, however, the Government of Canada allocated $65 million over the next three years to enhance its food safety programs in Health Canada and to develop new food safety and nutrition policies.[30]

3.3.3 Pest Management Regulatory Agency

3.3.3.1 Authority, Responsibilities and Co-ordination with Other Departments

The Pest Management Regulatory Agency (PMRA) is a governmental agency which reports to the Minister of Health. For a much more detailed account of the PMRA, see the Pesticides Case Study.

Established in 1995, following the recommendation of the Pesticide Registration Review Team, the PMRA administers the *Pest Control Products Act*[31] for Health Canada. The PMRA's stated goal is "to protect human health and the environment while supporting the competitiveness of agriculture, forestry, other resource sectors and manufacturing."[32] It is "responsible for providing safe access to pest management tools while minimizing the risks to human and environmental health."[33]

One of the PMRA's stated roles is to "consult and liaise with other federal departments, provincial and territorial governments, other national governments and international co-ordinating bodies."[34] The Alternative Strategies and Regulatory Affairs Division of the PMRA liaises with other federal government departments through the Policy Council and individual Memoranda of Understanding, and with provincial governments through the Federal-Provincial-Territorial Committee on Pesticide Management and Pesticides.

The PMRA also collaborates with pesticide regulators in the United States and is involved in several international pesticide fora. Policies and methodologies developed in these fora can significantly influence PMRA pesticide regulation.

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[27] *Health Canada: Performance Report for the Period Ending March 31, 1998b* (Ottawa: Minister of Public Works and Government Services Canada, 1998); (http://www.tbs-sct.gc.ca/tb/key.html).

[28] Health Canada, 1998a, *op.cit.* at 3.

[29] *Ibid.*

[30] *The Canada Food Safety and Inspection Bill and Health Protection Legislative Renewal, April 1999:* http://www.hc-sc.gc.ca/hpb/transitn/food_e.html

[31] *Pest Control Products Act*, R.S.C. 1985, c. P-9 [hereinafter *PCPA*]. Products regulated under the *PCPA* include herbicides, fungicides, insecticides, biological agents, antimicrobial agents, and growth regulators. See *Pest Control Products: Registration Handbook* (November 30, 1998) at 1 (Part 2); (http://www.hc-sc.gc.ca/pmra-arla/hndbk-e.html).

[32] *Ibid.*

[33] *Ibid.*

[34] *Ibid.*

Under the North American Free Trade Agreement (NAFTA), a Technical Working Group on Pesticides was established in June, 1997 by Canada, Mexico and the United States. The Working Group seeks to establish work sharing as a routine practice among members by the year 2002. A further goal is the establishment of a North American market for pesticides, while maintaining high levels of environmental and health protection. To achieve these goals, the Working Group proposes to develop a common data submission format, a coordinated review process, and to take steps to minimize trade problems resulting from different maximum residue limits for commodities that are traded among the three countries. The realization of these objectives will require the harmonization of data requirements, relevant test protocols, data submission and study report formats, data review and risk assessment practices, regulatory decision making, and administrative processes and procedures.[35]

In 1996, the PMRA and the U.S. Environmental Protection Agency established joint-review processes for reduced-risk chemical pesticides. The basis for joint-review is the desire to avoid the introduction of trade barriers if Canada does not co-ordinate its re-evaluation program with other countries, especially the United States. The implications of this situation are discussed further in Chapter 4. Very few Joint Reviews have been conducted. The PMRA has also stated that it plans to co-ordinate its re-evaluation (of registered pesticides) program with the EPA.[36]

In addition to working with the United States, the PMRA also collaborates with the Pesticide Programme established by the Organization for Economic Co-operation and Development (OECD).

The OECD Pesticide Program was established in 1992 in response to the huge workload faced by individual countries with their re-evaluation/re-registration programs. These programs require the completion of new risk assessments for hundreds of pesticides that have been on the market for many years. The goal of the Pesticide Program is to assist OECD countries to share the work of undertaking pesticide risk assessments and finding new approaches to risk reduction. In furtherance of this goal, the Program has developed a database to facilitate contacts between countries that would like to exchange reports or collaborate on assessing a pesticide, and has developed common guidelines for data submissions and for government evaluation reports. The Pesticide Program also contributes to the OECD Environmental Health and Safety Program efforts to develop and harmonize test guidelines and assessment methods. The Pesticide Program works to harmonize core data requirements such as the basic studies required for pesticide registration.[37]

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[35] NAFTA Technical Working Group on Pesticides. *A North American Initiative for Pesticides: Operation of the NAFTA Technical Working Group on Pesticides*. November, 1998. http://www.hc-sc.gc.ca/pmra-arla/qinter2-e.html .

[36] Pest Management Regulatory Agency, Regulatory Proposal, PR099-01. A New Approach to Re-evaluation. December 3, 1999. http://www.hc-sc.gc.ca/pmra-arla/qcont-e.html to obtain via download: pro9901e.pdf

[37] For a description of current projects, see PMRA. *PMRA Table of Current OECD Pesticide Projects*. February 1999. Document No. OECD99-01. http://www.hc-sc.gc.ca/pmra-arla/qinter-e.html .

3.3.3.2 Structure and Relevant Activities

The Agency is organized into six divisions. Five of these divisions, and their corresponding responsibilities, are laid out in the following table (not included is the Laboratory and Services Division - LSD).

Table 3.2: Pest Management Regulatory Agency - Internal Organization

Division	Responsibility
Submission Management and Information Division (SMSD)	• Manages and tracks submissions • Conducts scientific screening of potential registrants
Product Sustainability and Co-ordination Division (PSCD)	• Undertakes efficacy assessments, sustainability and value assessments
Health Evaluation Division (HED)	• Provides expertise on human health hazards, risk assessment and risk mitigation • Conducts toxicology evaluation and exposure assessment.
Environmental Assessment Division (EAD)	• Provides expertise on environmental hazards, risk assessment and risk mitigation • Conducts assessments of the • environmental fate and effects of pest control products
Alternative Strategies and Regulatory Affairs Division (ASRAD)	• Directs the development, review and assessment of policies, regulations, programs and legislative amendments, including those related to sustainable pest management

Source: Health Canada, *The Pest Management Regulatory Agency: Overview Document*; (http://www.hc-sc.gc.ca/pmra-arla/over-e.pdf).

Several groups advise the PMRA, as described in the following table:

Table 3.3: Pest Management Regulatory Agency - Advisory Bodies

Advisory Body	Membership	Responsibility
Economic Management Advisory Committee	Representatives of the manufacturers and users of pest control products that are economically impacted by PMRA decisions.	Provides advice on mechanisms to improve efficiency and cost effectiveness.
Pest Management Advisory Council	Representatives of pesticide manufacturers, users and environmental and health groups, as well as individuals with appropriate expertise.	Makes recommendations regarding PMRA management, priorities and strategies. Acts as a forum for the exchange of ideas and advice.
Policy Council	PMRA Executive Director, Assistant Deputy Ministers of Agriculture and Agri-Food, Environment, Fisheries and Oceans, Health, Industry and Natural Resources.	Provides a forum for the exchange of information and advice between federal government departments and the PMRA.
Federal - Provincial - Territorial Committee on Pest Management and Pesticides	PMRA; provincial and territorial government representatives.	Promotes information exchange, the provision of advice and the harmonization of appropriate programs and policies.

Source: *Pest Control Products: Registration Handbook* (November 30, 1998); (http://www.hc-sc.gc.ca/pmra-arla/hndbk-e.html).

3.3.3.3 Resources

The PMRA budget is approximately $28 million.[38] When first created, it had a budget of approximately $34 million, with a mandate to reduce this amount through Cost Recovery fees received from industry.[39] However, not all of these fees were recovered, leaving PMRA short of its target revenue.[40] This short-fall has, at least in the past, been covered by Agriculture and Agri-Food Canada, and Health Canada.[41]

In 1997-98, the PMRA's actual net expenditures were $15.7 million.[42] In 1998-99, the PMRA's actual net

[38] Julia Langer, Director, Wildlife Toxicology Program, Worldwide Fund for Nature (WWF), Toronto, January 4, 1999 (pers. comm.).

[39] The revenues comprise one-time fees charged for review of applications for registration and annual maintenance fees for the right and privilege to sell registered products in Canada. Health Canada, 1998b, *op.cit.* at 90.

[40] In 1997-98, PMRA experienced a revenue shortfall of $4 million in maintenance fees. This resulted in delaying the re-evaluation of registered products as well as delaying the elimination of the backlog. See *ibid.*

[41] *Supra* note 38.

[42] Health Canada, 1998b, *op.cit.* at 88.

expenditures increased to $18.5 million.[43]

3.3.4 Environment Canada

3.3.4.1 Authority, Responsibilities and Co-ordination with Other Departments

Environment Canada, established in 1971, is the primary federal agency responsible for environmental protection, with a mandate flowing from the *Department of the Environment Act.*[44] The Act provides the following mandate: "to preserve and enhance the quality of the natural environment (including migratory birds and other non-domestic flora and fauna), conserve and protect water resources, carry out meteorology, enforce the rules of the Canada-U.S. International Joint Commission, and co-ordinate federal environmental policies and programs."[45] Environment Canada's stated Mission is to "make sustainable development a reality in Canada by helping Canadians live in an environment that is protected, respected and conserved."[46] Environment Canada also states that "science and technology are the foundation for all of the department's policies, programs, technological solutions, services and operations."[47]

Environment Canada administers, or has some role in the administration of, a number federal statutes including, of importance to this study, the *Canadian Environmental Protection Act* (CEPA). First proclaimed in 1988 and revised in 1999, the "renewed" CEPA has as its focus pollution prevention rather than pollution controls, expands several legislative authorities and creates some new ones. For instance, it requires that all 23,000 substances in use in Canada be examined, introduces new deadlines for taking action on toxic substances, and requires virtual elimination of the most dangerous toxic substances. It addresses concerns about enforcement by giving "peace officer" status and expanded powers to enforcement officers.[48]

Environment Canada states that its long-term goal is the understanding, and prevention or reduction of the environmental and human health threats posed by toxic substances and other substances of concern.[49] To this end, Environment Canada is implementing the new CEPA in conjunction with Health Canada (including the Pest Management Regulatory Agency), Industry Canada, Natural Resources Canada and Agriculture and Agri-Food Canada.[50] The ability of the federal government to set standards on toxic chemicals is significantly influenced by the Canada-Wide Accord on Environmental Harmonization (see Section 3.5 below concerning Federal-Provincial-Territorial Co-operation and Partnerships).

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[43] http://www.ec.gc.ca/toxics/toxic1_e.html at 49.

[44] *Department of the Environment Act*, R.S.C. 1985, c. E-10.

[45] *Environment Canada, 1999-2000 Estimates: A Report on Plans and Priorities*, at 6; http://www.ec.gc.ca/rpp/index.htm

[46] *Ibid.* at 5.

[47] *Environment Canada: Performance Report For the Period Ending March 31, 1999* (Ottawa: Minister of Public Works and Government Services Canada, 1999); (http://www.tbs-sct.gc.ca/rma/dpr/98-99/EC98dpre.pdf)

[48] *Ibid.* at 18.

[49] Health Canada, 1998a, *op.cit.* at 7.

[50] *Ibid.*

3.3.4.2 Structure and Relevant Activities

Environment Canada is organized into seven headquarter organizations, and five integrated regions:

Table 3.4: Environment Canada: Headquarter Organizations And Regions

Headquarter
Office of the Minister and Deputy Minister
Atmospheric Environment Service
Environmental Conservation Service
Environmental Protection Service
Corporate Services
Policy and Communications
Human Resources Directorate

Regions
Atlantic
Quebec
Ontario
Prairie and Northern
Pacific and Yukon

Source: *Environment Canada, 1999-2000 Estimates: A Report on Plans and Priorities*; http://www.ec.gc.ca/rpp/index.htm

Environment Canada is also organized along four business lines. Business lines are the forums for setting national direction, resource allocation and accountability:

Table 3.5: Environment Canada: Business Lines And Structure

Business Line	Structure
A Clean Environment	Atmospheric Change; Toxics; Compliance and Enforcement; Technologies, Jobs and Capacity Building; Partnerships for Sustainable Development; and Emergency Prevention and Preparedness
Nature	Biodiversity/Wildlife; Conserving Canada□s Ecosystems; Compliance and Enforcement; Information Products and Services; and Partnerships for Sustainable Development
Weather & Environmental Predictions	Weather & Environmental Predictions; Atmospheric Change; and Information Products and Services
Management, Administration & Policy	Management and Administration; Information Products and Services, & Partnerships for Sustainable Development

Source: *Environment Canada, 1999-2000 Estimates: A Report on Plans and Priorities*; http://www.ec.gc.ca/rpp/index.htm

3.3.4.3 Resources

Environment Canada has undergone significant changes in the past decade. Since the mid-1990s, Environment Canada has had its budget cut by some 40%.[51] Environment Canada's total net budget for the fiscal year 1996-1997 was $621.3 million, however this had decreased to $551.0 million in 1998-1999. Of that amount, only $358.2 million went toward environmental protection and emissions reductions.[52] $55.8 million of this amount was allocated for the region of Ontario.[53]

In May of 1998, the Standing Committee on Environment and Sustainable Development released a scathing report on Environment Canada's enforcement record.[54] Although there are many dimensions to the problem, the lack of funding was a key issue in the discussion.

On the other hand, to meet the government's obligations under the revised *CEPA*, an additional $42 million over three years was announced in the 1999 Federal budget.[55]

3.4 THE PROVINCIAL LEVEL

3.4.1 Ministry of the Environment

3.4.1.1 Authority and Responsibilities

The Ontario Ministry of the Environment bears primary provincial responsibility for environmental protection. It has the ability to control provincial sources of pollution through its power to refuse approval or to impose conditions on an approval for any facility that may cause pollution. The MOE administers the *Environmental Protection Act*,[56] the *Environmental Assessment Act*,[57] the *Ontario Water Resources Act*.[58] the *Pesticides Act*[59] and the *Environmental Bill of Rights*.[60]

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[51] Standing Committee on Environment and Sustainable Development, *Third Report: Enforcing Canada's Pollution Laws: The Public Interest Must Come First!*, May 1998, at 6; http://www.parl.gc.ca/InfoComDoc/36/1/ENSU/Studies/Reports/ensurp03-e.htm

[52] Canadian Institute for Business and the Environment, Special Report on Environment Canada's Budget of $551.0 Million in 1998-99 (1998) 2:14 *The Gallon Environment Letter* 1. The federal government combines the budgets of weather reporting and environmental protection. Canada's weather reporting function in 1998-1999 was allocated $192.8 million, or 35% of Environment Canada's budget.

[53] *Ibid.* at 4.

[54] Standing Committee on Environment and Sustainable Development, 1998, *op.cit.*

[55] *Environment Canada, 1999-2000 Estimates: A Report on Plans and Priorities*, at 12; http://www.ec.gc.ca/rpp/index.htm.

[56] *Environmental Protection Act*, R.S.O. 1990, c.E.19, as amended.

[57] *Environmental Assessment Act*, R.S.O 1990, c.E.19, as amended.

[58] *Ontario Water Resources Act*, R.S.O. 1990, c. O.40.

[59] *Pesticides Act*, R.S.O. 1990, c. P.11 (hereinafter *Pesticides Act*).

The MOE participates in a number of collaborative initiatives including: (1) the development of Canada-Wide Standards (see section 3.5 below); (2) the work of the Federal-Provincial Advisory Committee (FPAC), set up under CEPA to deal specifically with air issues of national concern; and (3) work under the Canada-Ontario Agreement Respecting the Great Lakes Basin Ecosystem.[61]

3.4.1.2 Structure and Relevant Activities

The Ministry is organized by division, as outlined in the table below. Each division is headed by an Assistant Deputy Minister.

Table 3.6: Ontario Ministry Of The Environment - Internal Structure

Division	Branches
Operations	• Approvals • Investigations and Enforcement • Environmental Assessment
Environmental Sciences and Standards	• Environmental Monitoring and Reporting - Standards Development Branch • Environmental Partnerships
Integrated Environmental Planning	• Waste Management Policy • Land Use Policy • Air Policy and Climate Change • Water Policy Branch
Corporate Management	
Intergovernmental Relations Office	
Communications	

Source: http://www.ene.gov.on.ca/envision/org/org-moee.htm

Table 3.6, above, sets out the six Divisions of the Ontario Ministry of the Environment. Only three of the Divisions will be examined in greater detail, below.[62] Only those Branches having relevance to the present study are listed in the table.

Operations

The Operations Division is responsible for delivering programs to protect air quality, to protect surface and ground water quality and quantity, to manage the disposal of wastes, to ensure an adequate quality of drinking water and to control the use of pesticides. Additionally, this division is responsible for administering the ministry's approvals and licensing programs as well as an investigative and enforcement

[60] *Environmental Bill of Rights,* 1993, S.O. 1993, c.28, as amended.

[61] See http://www.ene.gov.on.ca/envision/news/coamb99.htm , Media Backgrounder (September 21, 1999).

[62] Descriptions of Ministry Divisions and Branches obtained from: http://www.ene.gov.on.ca/envision/org/op.htm; http://www.ene.gov.on.ca/envision/org/op.htm ; and http://www.ene.gov.on.ca/envision/org/essd.htm

program to ensure compliance with environmental laws. The division has a province-wide network of regional, district and area offices. As well, it includes the Approvals Branch, the Investigations and Enforcement Branch and the Spills Action Centre.

Approvals Branch
Under the *Ontario Water Resources Act* and the *Environmental Protection Act*, the Approvals Branch reviews and approves applications for new or modified waste, water and sewage facilities or facilities which may emit a contaminant, including noise, to the air or water. It administers licence-issuing functions relating to pesticides and septic system haulers and installers. It provides technical advice and guidance to agencies delivering the subsurface sewage disposal program in Ontario.

Investigations and Enforcement Branch
The Investigations and Enforcement Branch is responsible for all aspects of environmental enforcement within the ministry including for the *Environmental Protection Act*, *Ontario Water Resources Act*, *Environmental Assessment Act*, and *Pesticides Act*.

Environmental Sciences and Standards Division
The stated intent of the Environmental Sciences and Standards Division is to provide the best available science and technology to support all decisions about the natural environment, and to implement those decisions by developing ecosystem-based programs and partnerships, setting scientifically credible standards, monitoring the environment and providing valuable analytical and scientific expertise. This division provides these services through its four branches: Environmental Monitoring and Reporting, Standards Development, Laboratory Services, Environmental Partnerships and through the Drive Clean Office, some of which are discussed below.

Laboratory Services Branch
The role of the Laboratory Services Branch is to provide analytical support for the ministry's environmental monitoring and regulatory programs. Further, its role is to provide analytical method development and support standard setting. Its role is also to ensure the data quality of ministry compliance, enforcement and emergency analytical testing.

Standards Development Branch
The Standards Development Branch is responsible for: (1) developing and promulgating environmental standards to protect both human and ecosystem health and the quality of the natural environment; (2) providing toxicological advice and diagnostic services on environmental contaminants and pesticides; (3) assessing the performance of new and emerging environmental technologies and promoting technology transfer; (4) administering the *Pesticides Act* and providing direction on the responsible use of pesticides in Ontario.

Environmental Partnerships Branch
The task of the Environmental Partnerships Branch is to develop, deliver, measure and provide advice on innovative, non-regulatory approaches that promote environmental protection. The branch works in areas such as pollution prevention, resource conservation, environmental management systems, green industry development and improving the efficiency and effectiveness of water and sewage infrastructure.

Integrated Environmental Planning Division
The responsibility of the Integrated Environmental Planning Division is to integrate the overall policy development and planning functions of the Ministry. This involves integrating and synthesizing all information, data and perspectives on the many aspects of the Ministry's mandate. The division consults extensively on developing policies, strategies and programs that support the Ministry's core business of

conservation and environmental protection. It is organized into the following branches: Waste Management Policy, Land Use Policy, Water Policy, and the Air Policy and Climate Change Branch, one of which is discussed below.

Air Policy and Climate Change Branch
The role of the Air Policy and Climate Change Branch is to develop policies and programs for the improvement and protection of Ontario's air quality. The branch develops policy regarding smog issues, acid rain, inhalable and respirable particulates and ozone-depleting substances. Further, it is responsible for the development of Ontario's position on climate change and other activities to reduce greenhouse gas emissions.

3.4.1.3 The Pesticides Act and the Pesticides Advisory Committee

In addition to the Divisions and Branches described above, the Ministry has established, under the *Pesticides Act*, a Pesticides Advisory Committee to provide advice on a range of matters relating to pesticides and pest control. Since the Pesticides Case Study focuses on the federal regulatory system for pesticides, the provincial powers and responsibilities are described here.

While the federal *Pest Control Products Act* determines which pesticides may be utilized in Canada, the Ontario *Pesticides Act* further refines this system. The provincial law prohibits the sale and use of a pesticide product unless it is registered under the federal *Pest Control Products Act* and classified in one of six schedules, or is exempt from classification. Pesticide products "are classified into six schedules on the basis of their toxicity, environmental or health hazard, persistence of the active ingredient or its metabolites, concentration, usage, federal class and registration status." The schedules also determine who may buy, sell and use the pesticide, as well as the applicability of obligations such as the need for safety equipment, warning signs and safety testing. Hence, it is this classification scheme that provides the basis for the regulation, distribution, availability and use of pesticide products in Ontario. The *Pesticides Act* "allows Ontario to be more restrictive than the federal government, but not less restrictive."[63]

The Pesticides Advisory Committee, established under authority of the *Pesticides Act*, is firstly responsible for reviewing the *Pesticides Act* and Regulation 914.[64] As of April 1, 1999, Regulation 914, amended to O. Reg. 129/98, gives the Pesticides Advisory Committee the authority to approve the classification of a new product. Prior to this regulation, the Committee only had authority to review and recommend the classification of a new product, but had no approval authority. Additionally, Regulation 914 "now allows the sale and use of classified products as soon as the approved product is posted on the Internet, instead of being published in the Ontario Gazette."[65] The Pesticides Advisory Committee is also responsible for recommending changes and amendments to the Act to the Minister of the Environment. Next, it is responsible for advising the Minister of the Environment on matters relating to pesticides and pest control. Lastly, it is responsible for classifying all federally registered pesticides for sale, storage and use in Ontario.[66]

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[63] Ontario, Pesticides Advisory Committee, *Ontario Guidelines for Classification of Pesticides Products*, (Toronto: Queen's Printer for Ontario, April 1999) at 1.

[64] R.R.O. 1990, Reg. 914; O. Reg. 129/98.

[65] Ontario Ministry of the Environment website, *Pesticide Classification (April 1999)*; http://www.ene.gov.on.ca/envision/news/licensing.htm.

[66] Pesticides Advisory Committee website (http://www.opac.gov.on.ca/frmwelc.htm)

3.4.1.4 Resources

The operating budget for the Ministry of Environment has been dramatically cut in recent years. The Ministry's budget was cut from $365.4 million in 1994-1995 to $211.0 million in 1997-1998, a 44 per cent reduction.[67] Most recently, in 1999, the Ontario government announced it was further cutting the Ministry's budget to $165 million,[68] representing less than half the budget of five years ago.

Funding for the development of environmental programs and standards was cut 99 per cent from $51 million in 1994-1995 to $500,000 in 1997-1998.[69] Further, Ministry staff positions were cut 36 per cent from 2430 in 1995 to 1550 in 1997.[70]

3.5 FEDERAL - PROVINCIAL - TERRITORIAL CO-OPERATION AND PARTNERSHIPS

3.5.1 Canadian Council of Ministers of the Environment

3.5.1.1 Authority and Responsibilities

The Canadian Council of Ministers of the Environment (CCME) was formed in 1989. It is comprised of environment ministers from the federal, provincial and territorial governments. These 14 ministers usually meet twice a year "to discuss national environmental priorities and determine work to be carried out under the auspices of CCME."[71] The CCME mandate is to work to promote co-operation on and co-ordination of interjurisdictional issues such as waste management, air pollution and toxic chemicals. CCME members propose nationally-consistent environmental standards and objectives. CCME does not have the authority to implement or enforce legislation, and thus each jurisdiction decides whether to adopt CCME proposals.

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[67] Canadian Institute for Business and the Environment, Ontario Environment Budget Cut 44% (1997), July 23, *The Gallon Environment Letter* 1.

[68] J. McCarten, Ontario Detailing First Wave of New Spending Cuts, *Canadian Press Newstex* (18 November 1999).

[69] Canadian Institute for Business and the Environment, 1997, *op.cit.*

[70] *Ibid.*

[71] CCME Web Site; http://www.ccme.ca/1e_about/1e.html

3.5.1.2 Structure and Relevant Activities

In January 1998, the CCME endorsed and announced several initiatives, including the Canada-wide Accord on Environmental Harmonization (discussed further below). Table 3.7 shows the CCME internal organization.

Table 3.7: Canadian Council Of Ministers Of The Environment - Internal Organization

Initiatives	Structure
Environmental Planning and Protection Committee	**Steering Committee:** • comprised of senior staff of each jurisdiction • provide on-going advice to the Council of Ministers • co-ordinate specific CCME projects
	Canada-Wide Standards:[72] • Particulate Matter • Mercury • Dioxins & Furans • Ground-Level Ozone • Benzene • Petroleum Hydrocarbons
	Hazardous Waste
	Water Quality Guidelines
	Economic Integration
	Soil Quality Guidelines
	Packaging
	National Air Issues Co-ordinating Committees - Other Air Issues
New Sub-agreements under Harmonization Accord	**Monitoring and Reporting**
	Enforcement
	Environmental Emergencies
	Research and Development

Source: http://www.ccme.ca/1e_about/1ea.html.

[72] This is not a permanent list, and new substances can be added in the future. See discussions below and in Chapters 5 and 6 regarding Canada-Wide Standards.

3.5.1.3 Canada-Wide Accord on Environmental Harmonization

In 1993, the CCME identified harmonization of environmental management in Canada as a top priority[73] and developed the Canada-Wide Accord on Environmental Harmonization (discussed further in Chapter 4). On January 29, 1998, all jurisdictions except for Quebec[74] signed the Accord. While not a statute, the Accord holds the potential to have a dramatic impact on the way that standards are developed at both the federal and provincial levels.

The Accord is a multilateral umbrella agreement, whose intent is to provide a framework for achieving harmonization.[75] It provides a framework for the development of ancillary Sub-agreements on specific areas of environmental management. Three Sub-agreements have been developed to date: (1) Canada-wide Environmental Standards Sub-agreement; (2) Canada-wide Environmental Inspections Sub-agreement; and (3) Sub-agreement on Environmental Assessment. Further sub-agreements are currently in the process of negotiation, including ones on enforcement, monitoring and reporting, environmental emergencies, and research and development.[76]

The Canada-wide Environmental Standards Sub-agreement is described below and its implications are discussed further in Chapters 5 and 6.

Canada-Wide Environmental Standards Sub-agreement: Canada-Wide Standards
The Canada-Wide Environmental Standards Sub-agreement "sets out the principles underpinning the development of Canada-wide Standards (CWS) for environmental quality and human health, and commits the governments to participate in their development. Such standards could include guidelines and objectives, as well as legally enforceable standards."[77] The focus of the Sub-agreement is on "ambient standards, so that all Canadians can expect a common high degree of environmental quality."[78] Ambient standards are described as levels of environmental quality for specific media (for example, air, water, soil, or sediment).[79]

One of the stated underpinnings of the development and attainment of Canada-wide Environmental Standards is the Precautionary Principle. The agreement states that, "where there are threats of serious or irreversible environmental damage, lack of full scientific certainty shall not be used as a reason for postponing the development and implementation of standards."[80]

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[73] House of Commons Canada, Standing Committee on Environment and Sustainable Development, *Report: Harmonization of Environmental Protection: An Analysis of the Harmonization Initiative of the Canadian Council of Ministers of the Environment,* December 1997; http://www.parl.gc.ca/InfoComDoc/36/1/ENSU/Studies/Reports/ENSURP01-E.htm

[74] Quebec indicated it still required certain conditions to be met before it would sign the Accord and Sub-agreements. For instance, Quebec would like Parliament to adopt amendments to federal legislation that recognize the need to reduce overlap and duplication between jurisdictions. See *ibid.*.

[75] Standing Committee on Environment and Sustainable Development, 1997, *op.cit.*

[76] CCME Website, "Guide to the Canada-Wide Accord on Environmental Harmonization"; http://www.ccme.ca/3ea_harmonization/3ea1_accord/3ea1a.html

[77] Standing Committee on Environment and Sustainable Development, 1997, *op.cit.* at 6.

[78] CCME, 1998, *op.cit.*

[79] http://www.ccme.ca/3e_priorities/3ea_harmonization/3ea2_cws/3ea2b.html

[80] http://www.ccme.ca/3e_priorities/3ea_harmonization/3ea2_cws/3ea2a.html

The Sub-Agreement "calls for governments to establish priorities for the development of CWSs and to allow for public involvement."[81] The priority setting phase consists of three stages; nomination, screening, and selection.[82]

In November of 1999, the ministers agreed on draft Canada-Wide Standards for four priority pollutants: particulate matter; ground-level ozone; benzene; and mercury.[83] Ministers agreed to take the standards back to their Cabinet colleagues, who have six months to consult on these before they are finalized and formally adopted at the CCME meeting in the spring of 2000.[84]

Provincially, Ontario has posted three Notices of Proposal for Policy on its *Environmental Bill of Rights* Registry Web Site.[85] Particulate Matter and Ozone are combined in one proposal, while Benzene and Mercury each have their own proposal. Consultations are occurring in early 2000 (see Chapter 5 for more discussion of Ontario's air standards).

Currently, additional CWSs are under development for dioxins and furans; petroleum hydrocarbons; mercury from other sources; and benzene in air - Phase 2.[86]

3.5.1.4 Funding

The CCME is organized, funded and operated consistent with the following principles: the core business is funded according to the CCME funding formula - Canada 1/3 and the remaining 2/3 divided among the other jurisdictions pro-rated to population.[87]

If Ministers decide to take on issues in addition to those identified through the annual priority setting process or issues outside the core business, support is funded outside the formula, and partnerships are sought.[88]

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[81] http://www.ccme.ca/3e_priorities/3ea_harmonization/3ea2_cws/3ea2d_public/3ea2d.html

[82] *Ibid.*

[83] CCME, Environment Ministers Meet at Kananaskis, Kananaskis, Alberta, November 30, 1999; http://www.ccme.ca/1e_about/1eg_communiques/1eg7.html See also Ontario Ministry of the Environment, *In Brief: Canada-wide Environmental Standards: Ontario's Role* (Toronto: Queen's Printer for Ontario, December 1999).

[84] *Ibid.*

[85] http://www.ene.gov.on.ca/envregistry/012643ep.htm. See also: Ontario Ministry of the Environment, *In Brief: Ontario and the Canada-wide Standards for Particulate Matter and Ground-level Ozone* (Toronto: Queen's Printer for Ontario, December 1999) at 1; for the Benzene (Phase 1) proposal, see Ontario EBR Registry Number "PA9E0014"; http://204.40.253.254/envregistry/012642ep.htm; and for Mercury, see Ontario EBR Registry Number PA9E0013; http://204.40.253.254/envregistry/012641ep.htm

[86] CCME, *Canada-wide Standards - Overview*; http://www.ccme.ca/pdfs/cws_bkgoverview_e.pdf

[87] http://www.ccme.ca/le_about/led.html

[88] *Ibid.*

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Chapter 4 - Risk Assessment and the Precautionary Principle

Chapter 4 - Risk Assessment and the Precautionary Principle

4.1 INTRODUCTION

The various governmental agencies – provincial, federal and inter-governmental – described in Chapter 3 are responsible for setting standards, guidelines and policy on matters regarding environmental contamination. At issue in this study is whether or not these standards and guidelines, and the policies used to establish them, adequately account for (or in the past, accounted for) the health of children in Ontario. It is clear from the review in Chapter 2 that some environmental pollutants are affecting children's health and many other pollutants place children at risk. The level of uncertainty as to the extent and severity of these risks is significant. Yet the potential exists for both very large numbers of children to be affected and for the increased incidence of typically rare but very serious or even fatal health effects.

To continue this review, the present chapter begins with an historical look at standard setting approaches. Although there are different approaches to standard setting, the underlying framework for most of them is risk assessment.[1] This chapter focuses on a description and evaluation of risk assessment in order to determine how and to what extent the health concerns outlined in Chapter 2 are addressed. This focus on risk assessment is also necessary to appreciate and evaluate the detailed standard setting explored in subsequent chapters, including the two case studies. The setting of standards for air quality (Chapter 5), toxic substances (Chapter 6), consumer products (Chapter 7), lead (Case Study #1), and pesticides (Case Study #2), have all relied upon and increasingly rely upon the application of risk assessment. As noted in Chapter 1, the choice of focusing on standards for air, toxic substances, consumer products and pesticides was made to scope a large inquiry and to focus on areas most relevant to children's health in terms of known or suspected avenues of increased risk.

An evaluation of the health effects of environmental contaminants and of standard setting approaches also requires an appreciation of key issues surrounding interpretation of scientific evidence. Section 4.3 of this Chapter, the Science Behind the Assessment, explores these issues in depth. The chapter then focuses on recent examples of standard setting that recognize the increased risks to children of environmental contamination.

The review of risk assessment in this chapter takes a close look at the situation in the United States. This review is necessary for two reasons. Much of what is applied in Canada, both in absolute terms in the adoption of specific standards and in methodological terms in the development of risk assessment and risk management disciplines, comes from the United States. This situation is increasingly true due to efforts to achieve harmonization of standards under the terms of international trade agreements. To understand the application of risk assessment in Canada requires an understanding of the U.S. approach. Moreover, the Canadian Pest Management Regulatory Agency (PMRA) has stated that its pesticide re-evaluation process will borrow heavily from the ongoing pesticide re-evaluation process occurring in the United States. Since pesticides and children are of significant concern in both countries and the subject of a detailed case study herein (Case Study #2), the choice was made to focus this review of risk assessment in the United States on the implementation of the *Food Quality Protection Act* of 1996.

Finally, the shortcomings and challenges posed by risk assessment are contrasted to a review of the Precautionary Principle or a precautionary approach to standard setting.

[1] For brevity the term risk assessment is used in this introduction to include risk assessment and the related procedure of risk management. The two terms and their variations are defined further below.

4.1.1 Standards and Standard Setting

For the purposes of this discussion, the term "standard" includes any regulatory limit, including a full ban, on chemical or metal substances emitted to the environment. Since children's exposure to toxic substances occurs via numerous environmental pathways but also via manufactured goods, (such as paint, children's toys, plastic mini-blinds, etc.), this review of standards includes consumer products. Since pesticides are not environmental contaminants in the same sense as most but are toxic substances that are intentionally released into the environment, this review also includes the approval process for the use of pesticides.

The term "standard setting" refers to the various processes by which these regulatory limits or standards are set or the use of pesticides is approved.

The focus is on what can be called "health-referenced" standards, or those standards that are derived through an evaluation or estimation of the human health effects of the contaminant in question. Other types of standards can include "environment-referenced" standards where the evaluation process concerns environmental or ecological effects. Often, of course, a consideration of both health and environmental factors is included in a standard setting exercise. Another category is a "technology-referenced" standard, such as a limit on air or water emissions based on the "best available technology economically achievable," or BATEA. While this latter approach may set standards that are based solely on what is technically and economically achievable, it is generally the case that such standard setting occurs due to a prior understanding or concern about environmental or health imperatives or usually both.

As discussed more fully throughout this Chapter, the derivation of "health-referenced" standards during risk assessment includes both an estimation of exposure and an evaluation of health effects. An alternative approach to health-referenced standards is to eliminate the exposure assessment component and establish standards based on "inherent hazard" or "inherent toxicity." Advocates of this approach see it as a means of fast-tracking inherently toxic contaminants towards their ultimate elimination via regulatory phase-down if necessary but ultimately towards a total phase-out. This approach is discussed further in Section 4.5 below with respect to implementation of a precautionary approach to standard setting.

4.1.2 "Safe" Levels, Safety Factors, Threshold and Non-Threshold Effects

The history of standard setting approaches is one of increasing complexity of techniques mostly preoccupied with the establishment of "safe" or "acceptable" levels of contaminants. In some early cases, evidence of environmental persistence and/or harm in humans or wildlife was used in many industrialized countries as justification for banning outright some chemicals (e.g., the pesticides DDT and mirex and the entire class of chemicals known as PCBs). These early decisions to ban substances were examples of standards that recognized the "inherent toxicity" of the substances in question. More often however, evidence of harm was only suspected, difficult or impossible to prove, and hotly contested by the industries responsible for the contamination. As Section 4.3 below explores in detail, drawing inferences of causality in environmental health matters is extremely difficult. The "cautionary tale" of lead, described in Case Study #2, illustrates all of these aspects of standard setting. Over more than thirty years, lead standards have been constantly revised (up to and including a ban on lead additives in gasoline), as evidence of harm increased and within an adversarial climate of stiff industry opposition to regulation.

Early approaches to standard setting intended to protect human health from environmental exposure to

chemicals took a variety of forms. In many cases, health effects from toxic substances would be more or less understood due to their use and control in occupational settings. These occupational standards would have been derived from animal testing as well as knowledge of health effects among occupationally exposed workers. Somewhat arbitrarily, standards for environmental exposure might have been set at 10 times or 100 times the level considered safe in an occupational setting. This notion of using multipliers or "safety factors" in order to set standards for chemical exposures at levels 10 times, 100 times, etc., lower than the level where health effects are known or detected continues to be a key aspect of ever-more refined standard setting approaches to this day.

The application of safety factors, implying that safe levels of exposure are achievable, has been a key foundation from which risk assessment has grown. Indeed, the practice of setting standards based on a scientific determination of an "acceptable" level of risk developed since the 1970s largely as a substitute for bans or phase-outs of chemicals. However, with greater understanding of the mechanisms of toxicity of certain classes of chemicals, the notion of "inherent toxicity" has arisen, or has perhaps been revived, whereby substances are identified as toxic without the need for scientific determinations of harm. Substances that are considered inherently toxic are those that, by virtue of their molecular structure, are persistent and bioaccumulative and for which risk-based standards cannot establish "safe" levels of exposure. Other inherent characteristics may also justify classification of chemicals as inherently toxic such as very high acute toxicity, ability to cause endocrine disruption, probable human carcinogen, and neurotoxic or developmental neurotoxic effects. These distinctions are central to key aspects of the precautionary approach to standard setting discussed in Section 4.5 below, in Chapter 6 and the Pesticides Case Study.

Standard setting in both occupational and environmental settings also has often included making a distinction between chemicals for which a threshold is or is not apparent. In other words, in the case of chemicals with a threshold, the evaluation (using animal studies or the results of occupational exposure, accidents, etc.) determines the lowest point, or threshold, at which a health effect is detected. These threshold levels are called the Lowest Observed Adverse Effect Level (LOAEL). Lower limits are also calculated where no health effects are discernable. Also called the No Observed Adverse Effect Level (NOAEL), regulatory limits for human exposure to chemicals with threshold effects are often set by applying safety factors (typically between 10 and 1000) to NOAELs derived from animal studies.

Of course, considerable debate has occurred over whether or not health effects in fact do occur below these thresholds. Again, the example of lead is one where the threshold for adverse effects has been progressively lowered (see e.g., Figure 8.2 in the Lead Case Study) from occupationally derived standards steadily downward to a point where there is increasing agreement that, for some health effects, there is probably no safe level of lead in young children.

In the case of non-threshold chemicals, investigations are not able to discern any level or threshold below which certain effects (often called the most sensitive effect or the critical effect) do not occur. Such health effects are often various forms of cancer. The long history of the study of asbestos provides one of the best examples of a chemical for which no threshold is apparent. Regardless of a historical progression towards lower and lower levels of asbestos exposure, occupationally exposed individuals consistently experience excess rates of cancer.[2] For non-threshold effect chemicals, the safety factor applied has often been higher such as 1000 times the lowest dose where cancer has been seen to occur. Or, more typically for carcinogens, the safety factor approach is replaced by the use of mathematical models that assume a linear dose-response relationship. Using these models, a standard is set with the intention of ensuring that there is only a one-in-a-million chance for the cancer to occur across an exposed population often assuming a 70-year or "lifetime" exposure period.

[2] See review in Epstein, S. *The Politics of Cancer, Revisited.* East Ridge Press, 1998, pp. 54-68.

4.2 RISK ASSESSMENT AND RISK MANAGEMENT

4.2.1 Definitions

Since at least the mid-1970s, the still-evolving system by which these standards for threshold and non-threshold chemicals have been set has included *risk assessment* and *risk management.* Terminology and definitions have varied over time and in different countries and experts disagree on how "risk" and related terms should be defined. The Canadian Standards Association (CSA) has developed a series of definitions for a range of terms and these are reproduced in Figure 4.1.

Figure 4.1: Risk Management Definitions According to the Canadian Standards Association[3]

Hazard: a source of potential harm, or a situation with a potential for causing harm, in terms of human injury, damage to health, property, the environment, and other things of value, or some combination of these.

Hazard Identification: the process of recognizing that a hazard exists and defining its characteristics.

Risk: the chance of injury or loss as defined as a measure of the probability and severity of an adverse effect to health, property, the environment, or other things of value.

Risk Analysis: the systematic use of information to identify hazards and to estimate the chance for, and severity of, injury or loss to individuals or populations, property, the environment, or other things of value.

Risk Assessment: the overall process of risk analysis and risk evaluation.

Risk Communication: any two-way communication between stakeholders about the existence, nature, form, severity, or acceptability of risks.

Risk Control Option: an action intended to reduce the frequency and/or severity of injury or loss including a decision not to pursue the activity.

Risk Estimation: the activity of estimating the frequency or probability and consequence of risk scenarios, including a consideration of the uncertainty of the estimates.

Risk Evaluation: the process by which risks are examined in terms of costs and benefits, and evaluated in terms of acceptability or risk considering the needs, issues, and concerns of stakeholders.

Risk Management: the systematic application of management policies, procedures, and practices to the tasks of analyzing, evaluating, controlling, and communicating about risk issues.

Risk Perception: the significance assigned to risks by stakeholders. This perception is derived from the stakeholders' expressed needs, issues, and concerns.

In the United States,[4] a slightly different set of definitions exists. Several of these are provided for

[3] Canadian Standards Association, *Risk Management: Guidelines for Decision-Makers* (CAN/CSA-Q850-97), July, 1997.

[4] Source for the three definitions and four-step framework for EPA risk analysis described in this section: Congressional Research Service (CRS) Report 98-618, *Environmental Risk Analysis: A Review of Public*

comparison to those developed by the CSA and because they underlie the United States Environmental Protection Agency (EPA) approach to be discussed in detail below. They are also the most relevant and useful for a discussion of health-referenced standards.[5]

In the context of the EPA approaches to standard setting, *environmental risk assessment*, can be defined as:

> Any formal or informal scientific procedure used to produce a *quantitative* estimate of environmental risk. For example, risk assessment is often used to estimate the expected rate of illness or death in a human population exposed to a hazardous chemical based on the number of experimental animals affected by various doses of the chemical as measured in laboratory experiments.

Environmental risk analysis is defined more broadly to include:

> Any *quantitative* or *qualitative* scientific descriptions of an environmental hazard, the potential adverse effects of exposure, the risks of these effects, events and conditions that may lead to or modify adverse effects, populations or environments that influence or experience adverse effects, and uncertainties with regard to any of these factors.

There is an underlying four-step process within risk assessment and risk analysis that originated in the United States during the late 1970s. Much has happened since this basic framework was established, as this Chapter discusses in detail, but the four steps remain relevant today. They include:

Hazard identification: determining whether a particular chemical causes a particular health effect.
Dose-response assessment: determining the relationship between magnitude of exposure and probability the health effect will occur.
Exposure assessment: determining the extent of exposure before or after application of regulatory controls.
Risk Characterization: describing the nature and often the magnitude of risk, including attendant uncertainty.

Finally, *risk management* is the policy making step. To complete the set of definitions from the U.S. approach, *risk management* can be defined as:

> The process of deciding what should be done about a hazard, the population exposed, or adverse effects, implementing the decision, and evaluating the results. Decision makers may consider social, political, economic, legal, ethical, and engineering information as well as scientific risk estimates in choosing among available risk management options. Risk management decisions often require value judgements on such questions as "What level of risk is acceptable?" and "What level of expenditure is reasonable?"

Policy Issues. 40 p., Appendix. July 15, 1998. (Hereinafter: CRS Report 98-618.) Part VIII, Appendix, pp.3-4. Available at: www.cnie.org/nle/rsk-11g.html.

[5] Note that there are many areas where risk assessment and risk management are applied including the setting of standards, environmental assessment and planning decisions, remediation of contaminated lands or hazardous waste sites, and many non-environmental settings as well. Approaches and frameworks differ in each of these areas and this study is only concerned with risk assessment and management with respect to the setting of standards. For an overview of a variety of risk assessment and management frameworks, see: Dyck, W, *et.al.*, *Current Directions in Environmental Risk Assessment and Management*, Network for Environmental Risk Assessment and Management (NERAM), February, 1999. Available at: www.neram.ca.

4.2.2 Towards a Consistent Approach

Techniques for evaluating hazards and measuring risks pre-date the environmental and health concerns that became the subject of policy and legislation in the late 60s and early 70s. Early techniques were developed often for engineering and/or insurance purposes (risk of death, chance of floods, etc.) and were subsequently borrowed and adapted to assess environmental risks. Several different agencies within the U.S. government, often regulating the same industries for different purposes, developed their own techniques for assessing risk and devising regulatory standards. Predictably, different conclusions were reached by agencies with different mandates. Such differences were understandable given the level of complexity and inherent uncertainty involved in making risk calculations. Much criticism resulted.[6]

A first step towards coordinating risk assessment procedures was to establish an Interagency Regulatory Liaison Group (IRLG). Five different agencies were ultimately part of the ILRG including the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), the Consumer Product Safety Commission (CPSC), the Food and Drug Administration (FDA), and the Food Safety and Quality Service of the Department of Agriculture. The ILRG focused on cancer risks and in 1979 proposed a "cancer policy" to coordinate risk analysis and risk management across their members respective agencies and within the constraints of the statutes each administered. The agreement reached among these agencies included a consistent approach for cancer risk assessment procedures. In particular, the proposed policy included a consistent approach to the choice of "inference options" or "default assumptions" that need to be applied throughout risk assessment in order to compensate for gaps in data and scientific theory and methodology. Although a first step towards addressing the problem of inter-agency differences in risk assessment procedures, as well as getting a grip on the many assumptions inherent in the process, the proposed policy was condemned by many as allowing policy prescriptions to influence scientific judgements.

The ILRG was disbanded in 1981 and the U.S. Congress turned to the National Academy of Sciences to address both the substance of risk assessment procedures and the issue of interagency coordination. The result was a pivotal study that had a far-reaching influence on risk assessment practices. *Risk Assessment in the Federal Government: Managing the Process*[7] reviewed the various agencies practices and found the institutional arrangements to be basically sound. It recommended a framework for cancer risk assessment which has continued to be refined to the present day. Additional key recommendations included the need to separate risk assessment from risk management (along the lines of the definitions noted in Section 4.2.1 above) and to develop risk assessment guidelines for the federal government as a whole.

The report also identified the many gaps in both data and theory that exist in risk assessment. It identified at least 50 "inference choices" that are necessary during cancer risk assessment that cannot be made on a scientific basis. Herein lies the central criticism of risk assessment that has been part of an extensive and vocal critique, mostly championed by environmental organizations at least since the NAS report was published. The list in Figure 4.2 provides some examples of the inference choices or subjective judgements that are necessary during risk assessment. Despite the many inherent and fundamental limitations of risk assessment identified, the NAS report nevertheless concluded that risk assessment

[6] Historical account summarized from: CRS Report 98-618, Part II (available at www.cnie.org/nle/rsk11a.html) and Part VII (available at www.cnie.org/nle/rsk11f.html).

[7] National Academy of Sciences, *Risk Assessment in the Federal Government: Managing the Process*. Washington, D.C., National Academy Press. 1983.

required refinement, (through the development of detailed guidance documents), not replacement.

Figure 4.2 : Some Subjective Judgements in Risk Assessment[8]

What kinds of evidence are needed to demonstrate carcinogenicity?
How important are toxicity studies that show an effect relative to studies that show no effect?
How are benign and malignant tumours in animals counted?
What are the appropriate dose levels for experiments?
How should animal doses be compared to human doses?
How should animal effects be compared to human effects?
Are the effects observed at high doses expected to occur at low doses?
Should different chemical carcinogens be treated differently?
How should carcinogenicity be compared to mutagenicity? To birth defects?

The NAS report contributed to increasing consistency and coordination in risk assessment approaches across the various agencies.[9] It also led to amendments in 1990 to the *Clean Air Act* which established a Risk Assessment and Management Commission, also more recently called the Presidential/Congressional Commission on Risk Assessment and Risk Management. This group ultimately concluded that detailed interagency guidelines were not possible given different departmental mandates. It recommended a general framework instead which was published in 1997.[10]

Among all the agencies, EPA has consistently taken the lead in developing and revising risk assessment guidelines. EPA was the first to propose an interim guideline for its cancer risk assessments in 1977. Using the framework proposed in the 1983 NAS report, EPA finalized its guideline for cancer risk assessment in 1986.[11] This guideline also included early consideration of developmental risks (from chemicals that can cause mutations or damage to human development) and guidance on assessing exposure (to both individual chemicals and chemical mixtures). Subsequently, a revised guideline for developmental risks was published in 1991[12] and for exposure in 1992.[13] During the late 1990s these guidelines have continued to be revised and additional guidance documents have been developed in response to new requirements flowing from the *Food Quality Protection Act* of 1996 (discussed further in Section 4.4 below).

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[8] Source: Adapted from Rushefsky, M. *Making Cancer Policy*. Albany, N.Y. State University of New York Press, 1986, p.40, as cited in CRS Report 98-618, Part VII (available at www.cnie.org/nle/rsk11f.html).

[9] For example, the NAS risk assessment framework was adopted by the White House Office of Science and Technology Policy in 1985. Subsequent documents contained similar government-wide guidance including the "Regulatory Impact Analysis Guidance" contained in the 1991-1992 *Regulatory Program of the United States*, the 1994 *Draft Principles for Risk Assessment; Management, and Communication* and the 1996 Office of Management and Budget report, *Economic Analyses of Federal Regulations Under Executive Order 12866*. See more detailed review in CRS Report 98-618, Part II.

[10] The Presidential/Congressional Commission on Risk Assessment and Risk Management. *Framework for Environmental Health Risk Management.* Final Report, Volume 1, 1997, and *Risk Assessment and Risk Management in Regulatory Decision-Making*, Final Report, Volume 2, 1997.

[11] *51 Federal Register* 33992-34054, Sept.24, 1986.

[12] *56 Federal Register* 63798-63826, Dec.5, 1991.

[13] *57 Federal Register* 22888-22938, May 29, 1992.

4.2.3 The "Delaney Paradox"

The continuing acceptance, and indeed frequent insistence by industry, of risk assessment for the setting of environmental standards, faced a fundamental problem in the United States with the so-called "Delaney clause" in the U.S. federal *Food, Drug and Cosmetic Act.*[14] This clause, named after the Congressman who had been its author in 1958, prohibited FDA approval of any food additive found to cause cancer in animals or humans. This clause set up a conflict between the FDA in its approval process for food additives and the EPA, responsible for approving pesticides. A federal appeals court ruling in 1992[15] stated that an EPA finding of cancer-causing pesticide residues in processed foods would be a violation of the Delaney clause. This case forced the U.S. government on the road to choosing between weakening the Delaney clause or banning cancer-causing pesticides. They ultimately chose the former.

The Delaney paradox was resolved in favour of risk assessment. The "zero cancer risk" policy demanded by the Delaney clause was clearly at odds with the commercial interests of pesticide manufacturers and such a policy, if applied more broadly, also was not supported by other corporations wanting to continue releasing carcinogenic chemicals to the environment. It was also dismissed as the product of an earlier time when scientific techniques were considered crude.[16] Advocates of risk assessment pointed to the need to use "good science" that could determine levels of "safe" or so-called *de minimus* or "negligible" risk; levels so low that there would be, according to the risk assessors, no cause for concern. These low levels of risk are the one-in-a-hundred-thousand, or one-in-a-million or one-in-ten-million risk levels that are established using risk assessment techniques for incorporation into environmental standards.

4.2.4 Science or Pseudo-Science?

Although risk assessment is routinely described by its proponents as an objective, fact-based scientific activity, it is not, and probably never will be.[17] While it can provide a generally reliable means of predicting acute effects from high dose exposures, it falls far short in the most important area of environmental concern: chronic effects from long-term, low dose exposure. As for assessing the real-world situation of exposure to and the interactive effects of multiple chemicals in the environment, it fails miserably. There are simply too many uncertainties inherent in the process in terms of 1) basic insufficiency of data; 2) lack of methodologies for key steps in the process; and 3) the difficulty of reproducing or ensuring consistency and equal levels of professionalism and expertise across highly

[14] *Federal Food, Drug, and Cosmetic Act (FFDCA),* 21 U.S.C. 301 (1996).

[15] Les v. Reilly, 968 F.2d 985 (9th Cir. 07/08/1992).

[16] There was an extensive, multi-year debate over the Delaney Clause with environmental advocates often insisting on its retention and industry equally opposed to its rigidity. In *Our Children's Toxic Legacy*, (Yale University Press, 1996, p. 282) John Wargo argues that the Delaney Clause was unreasonable because: 1) it applies only to a small portion of pesticides, those that are carcinogenic and concentrate during food processing, and neglects other health effects; 2) scientific evidence supports the claim that some compounds pose cancer risks that approach zero, but are not definitively zero and forcing these compounds off the market may cause more risky substitutes to be used; 3) uncertainty in cancer studies often is substantial due to reliance on animal studies; 4) the costs of achieving a zero risk pesticide environment are enormous and difficult to justify given the possibilities of reducing other types of more certain health risks at lower costs; and 5) the clause has caused scarce regulatory resources to be disproportionately devoted to manage cancer risks rather than other types of effects such as neurological and reproductive damage.

[17] CRS Report 98-618, Part II. See also: Congressional Research Service Issue Brief for Congress No. 94036: *The Role of Risk Analysis and Risk Management in Environmental Protection.* November 5, 1999. Available at: www.cnie.org/nle/rsk-1/html. (Hereinafter: CRS Issue Brief No. 94036.)

complex analyses.[18] Moreover, the presentation of results and their incorporation into policy decisions, the risk management extension of the exercise, is equally subject to the value judgements and guesswork that are central to the "science" of risk assessment.

Just looking at cancer risk assessment, which is, arguably the most well developed and reliable of any form of risk assessment addressing chronic health effects, several important points can be made that challenge the notion that risk assessment is objective, fact-based and "good science." Recall that the NAS 1983 report identified at least 50 "inference choices" that are necessary during a cancer risk assessment that cannot be made on a scientific basis, many of which directly influence the policy choices made about the chemical under investigation.

The conclusion of the NAS study was, as previously noted, the need to develop better risk assessment guidance documents and to continually improve the database upon which risk assessment depends. The result over time has been a steady increase in the sophistication of risk assessment procedures, particularly with respect to cancer. However, the amount and significance of inference choices has not changed very much. Rather, because of a long-term focus on cancer as the most serious of a variety of possible chronic health effects, a great deal of research has been conducted on whether and at what dose, chemicals contaminants can cause cancer. There also have been a lot of cancer risk assessments conducted and revised in light of new and emerging information as well as increasing agreement about key areas where judgement calls are made (including the many areas noted in Figure 4.2).

The result of all of this work in cancer risk assessment has been a reduction in the range and variability of risk estimates but not necessarily a reduction in cancer risk. On the contrary, cancer risks have very likely increased as this chemical by chemical approach has proceeded and devised one-in-a-million cancer risk estimates for hundreds of different chemicals. The result in terms of cancer risk is 100s-in-a-million and perhaps even thousands-in-a-million. The actual risk level is not one-in-a-million since each chemical is assessed separately and considered in isolation from any other cancer risks that may exist from either similar or dissimilar cancer-causing or potentially cancer-causing chemicals in the environment. Nor has cancer risk assessment been conducted on more than a mere handful of chemicals by comparison to the many tens of thousands of chemicals in commercial use for which almost no toxicological information exists at all (as described and referenced in Chapter 1).

This numbers game is particularly abhorrent to those who criticize the ethics of risk assessment (discussed further below). Another way of describing the risk result of this chemical by chemical assessment and generation of "negligible" risk estimates, is to think in terms of even just ten substances with a one-in-a-million excess cancer risk (two very conservative assumptions). This situation would work out to a risk level of ten in a million or one-in-one-hundred-thousand (10/1000000 = 1/100000). For risk assessments conducted at the one-in-one hundred thousand risk level (and there are many; even at a one-in-ten thousand level), the number gets even worse, i.e., 10/100000 = 1/10000 and 10/10000=1/1000. Risk assessment proponents would rightly point out that these simple calculations incorrectly assume that all exposures are additive. Although each person could be exposed to each and every one of the ten chemicals at the exposure levels assumed, such additive exposure may not be the case. Nevertheless, risk assessment proponents would never advocate that 1/1000 or one-in-a-thousand is an "acceptable" risk of cancer from environmental exposure to contaminants. Taking these calculations further, if the number of carcinogens being released is more than 10, (an entirely reasonable assumption), the risk level continues to increase. If excess deaths due to other mechanisms (non-cancer) from these chemicals are added, the risk number is worse yet. And such calculations still have not accounted for synergistic effects or inter-

[18] See for example, the error in dust exposure calculations in Health Canada's risk assessment for plastic mini-blinds, as discussed in Chapter 8, Section 4.6.4.

generational effects. In other words, no matter how much the individual risk assessment process is refined, it is still counting trees and missing the forest in terms of real risks to people.

The long period of time during which cancer research and risk assessment has occurred also contributed to a situation where carcinogenicity was, and to a considerable extent still is, heavily relied upon as a surrogate measure for any chronic health effects. This situation resulted in the near total exclusion in risk assessments of other less understood and less studied effects such as reproductive, neurological or neurodevelopmental effects, or immunological and endocrine effects. Notably, these other potential effects are particularly relevant to children's health.[19]

Other central criticisms of the scientific shortcomings of risk assessment include the fact that uncertainties and errors can result from:

- *small population generalizations* – i.e., when extrapolations are made from high concentrations of chemical exposures in small populations to predict health effects in large populations exposed to lower concentrations of the same chemical.
- *generalizations from animal studies to human health* – i.e., when extrapolations are derived from animal studies (both high dose, short term exposure and low dose, long term exposure) to predict human health effects.
- *ignoring background sources* – i.e., the tendency to ignore or be unaware of background sources of exposure to chemicals affecting people or ecosystems leading to exceedances of threshold values established through risk assessment.
- *ignoring multiple chemical exposure* – i.e., the inability of risk assessment to accommodate real world situations of multiple chemical exposures of varying dose and duration or to assess the possible cumulative or synergistic effects of such multiple exposures.
- *the "healthy white male" as the norm* – i.e., the tendency to exclude the most sensitive segments of the population from calculations of risk by not including a wide enough margin of safety (and even assuming safe levels are known or knowable).
- *major limitations in animal testing* – i.e., the fact that animal bioassays do not always extend over entire lifetimes, dosing generally begins after weaning, thereby skipping *in utero* and neonatal periods comparable to the first 3-6 years of human life, the complication of the "wasted dose" which is the difference between the lifetime dose and the dose that actually causes disease, and the inappropriate assumption that negative results in animal bioassays indicate safety for humans.

The above list is drawn from analyses published mostly during the early 1990s.[20] Within the above list, it

[19] Note however that considerable work has continued in the development of additional risk assessment guidance documents for these other health effects. However, despite the existance or evolution of guidance for the evaluation of these other effects, they may not necessarily inform the risk assessment process if they are not part of "core testing" requirements. This matter is discussed further in Section 4.3.1 below with respect to concerns about endocrine disrupting effects at vulnerable periods of very low dose exposure and with respect to the use of developmental neurotoxicity tests.

[20] See for example: Benbrook, C.M., *et.al.*, Consumers Union, *Pest Management at the Crossroads.* (Consumers Union of the United States, New York, 1996) Chapters 3 and 4; Chess, C. and D. Wartenberg, The Risk Wars: Assessing Risk Assessment, *New Solutions* 3(2) (1993), pp.16-25; Chociolko,C., The Experts Disagree: A Simple Matter of Facts Versus Values?, *Alternatives* 21(3) (1995); Costanza, R. and L. Cornwell, The 4P Approach to Dealing with Scientific Uncertainty, *Environment* 34(9) (1992); Ginsberg, R., Quantitative Risk Assessment and the Illusion of Safety, *New Solutions* 3(2) (1993), pp. 8-15; Gregory, M., Pesticide Reform in Arizona: Moving Beyond Risk Assessment and Clean-up to Exposure Prevention, *Arizona Toxics Information*, (1991); Gregory, M., Some Unacceptable Risks of Risk Assessment, *Pesticides and You*, Spring (1995), p.14-16; Gutin, J., At Our Peril: The False Promise of Risk Assessment,

is important to note the difference between the first two points and the final four. For the first two, there is no way around the need to make such generalizations and extrapolations. Problems of uncertainty, variability, error, and gaps in data will exist but inferences have to be drawn from the information that such studies can provide (as described further in Section 4.3 below). The final four points however are shortcomings of a different kind. They represent problems of fundamental gaps in information and methodology to assess both real-world exposure and actual risks to sensitive populations or life stages. While refinements in risk assessment continue and have begun to address some of these shortcomings, many fundamental limitations remain. The work within the United States EPA to implement the *Food Quality Protection Act* is particularly illustrative of this ongoing debate (as discussed in Section 4.4 below).

Uncertainty, variability, error and large gaps in basic data and methodology occur in two of the four risk assessment steps described in Section 4.2.1 above. Of the four steps: hazard identification, *dose-response assessment, exposure assessment*, and risk characterization, the second two (in italic) are especially difficult due to a basic lack of both critically important scientific and/or empirical data and assessment methodologies. Even when risk assessors are considering a single chemical at a time, basic scientific and/or empirical data and methodologies are lacking to be able to calculate exposure and a dose-response relationship. Of course, this problem is greatly magnified when considering multiple exposures and the chance of cumulative or synergistic effects.

To illustrate,[21] risk assessors simply do not know exactly (or in some cases even remotely) how much of a pesticide (or a group of pesticides) makes up a child's exposure. They do not know whether the adverse effect levels detected in laboratory experiments on rats or dogs are comparable, or even approach the range of possible adverse effects in a human fetus, infant, child or adolescent. To be able to carry through to the risk characterization step and assign exposure and dose-response numbers for incorporation into a risk management strategy such as setting a standard for exposure or permitting the use of a pesticide, gaps are filled by the "inference choices" noted above. Also called "science policy choices" or "default assumptions," these gaps in critically important scientific and empirical data and methodologies are filled by what is essentially guesswork. It may be the product of "best guesses" or "informed guesses" or "the informed judgement of experts" but it is still guesswork, not science.

Nor is it simply a matter of doing more research or spending more money to fill in these gaps. It is certainly true that more research can and does eliminate data gaps and uncertainty. Improvements in methodology can also reduce the broad range in risk estimates that risk assessments can generate. However, the enormity of the data collection task is formidable. According to one risk assessment expert and advocate, "toxicologists know a great deal about a few chemicals, a little about many, and next to nothing about most."[22] Further, the key methodological gaps are even less easy to fill and proposals for addressing them are controversial as Section 4.4.3 below explores in more detail.

Greenpeace Magazine, 16(2) (1991); Highland, J., *Risk-Benefit Analysis in Regulatory Decision-Making*, Toxic Chemicals Program, Environmental Defense Fund, undated; O'Brien, M., Alternatives to Risk Assessment, New Solutions 3(2) (1993), pp.39-42; Smith, C., K. Kelsey, and D. Christiani, Risk Assessment and Occupational Health: Overview and Recommendations, *New Solutions* 3(2) (1993), pp.26-38; Thornton, J., Getting Burned: Risk Assessment is the Real Threat to the People Who Live Near Toxic Waste Incinerators, and Risking Democracy, *Greenpeace Magazine* 16(2) (1991), p.15 and p.17.

[21] Adapted from example in: Risk Assessment –Part 2, Judge Breyer's Prescription for Risk, *Rachel's Hazardous Waste News*, #394, June 16, 1994. See also: *Rachel's Hazardous Waste News* Part 1, The Emperor's Scientific New Clothes, #393, June 9, 1994; Part 3, Which Problems Shall We Ignore?, #395, June 23, 1994; and The Ethical Hazards of Risk Assessment, #519, November 7, 1996.

[22] Rodricks, J., *Calculated Risks*. Cambridge University Press, New York (1992), p. 192.

Finally, and perhaps most fundamentally, the assigning of individual risk levels for each chemical is essentially a game of odds that cannot address two of the most serious issues of toxic chemical pollution: inherent toxicity and population-wide effects such as may be occurring with endocrine disrupting chemicals. Risk assessment enables risk calculations that allow for "acceptable" levels of one-in-a-million or one-in-ten-thousand risks (of cancer, birth defects, etc.) across a population. However, the odds game becomes useless if further research confirms the suspicion that chemicals such as endocrine disruptors are capable of exerting population-wide effects at current levels of exposure.[23] Nor is it appropriate to make such calculations for chemicals that are persistent and bioaccumulative. Risks will continue to increase for chemicals that do not break down and which accumulate in animal fat, breast milk, etc. Such risks will no doubt affect some people more seriously than others depending on the flow of persistent chemicals through the environment.

4.2.5 Politics, Ethics and Equity

The political and ethical hazards of risk assessment stem directly from the combination of guesswork and science described above. Despite its gaps in basic information and methodologies to implement key steps, risk assessment is enormously complex and the domain of specialized experts. This complexity makes several things possible. Value judgements and questionable assumptions can be concealed. Policy-makers can be manipulated or misled during the political decision-making or risk management phase. An intellectual elite and those wealthy enough to hire them can dominate discussions, the political process and the outcome.

It is not surprising that a methodology that requires the making of frequent "inferences choices," or "science policy choices," or what many consider to be significant and influential value judgements, will raise important issues of ethics and social equity. Commentators frequently note that risk assessment tends to impose risk on those that are often most susceptible to harm, and the least able to confront or resolve the source of harm, including the poor, the elderly, children (including fetuses) and minority groups. Moreover, risks can be imposed on these groups without their consent and under circumstances where those being placed under the highest risk receive little to none of the benefits that result from whatever activity the risk assessment sanctions. As noted above, the political malleability of the process provides the opportunity for those with money and power to influence the outcome.[24]

Two additional ethical issues arise directly from the shaky scientific foundation of risk assessment. First, the vast ignorance about the toxic effects of chemicals leads to each chemical being treated as "innocent until proven guilt." Risk assessment calculations (where these have been done at all) guarantee the granting of a risk level (e.g., the one-in-a-million, also called the "negligible" cancer risk level) or a risk range (between one-in-10,000 and one-in-10-million) for each chemical and often in each medium. Hence risk calculations have customarily been done to establish the one-in-a-million risk level for a chemical in fish, meat, air, drinking water, etc. Although a multi-media approach may be conducted so that a risk assessment accounts for aggregate exposure via different pathways, this may be very difficult, may apply controversial methods or may not occur at all. It is almost certain that aggregating exposure (to ensure lower risk calculations for each individual pathway) did not occur in the past for evaluations of chemicals,

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[23] Colborn, T, D. Dumanoski, and J. Peterson Myers, *Our Stolen Future* (Dutton, New York, 1996), see in particular, Chapter 11 (Beyond Cancer) and Chapter 11 (Flying Blind).

[24] See multiple sources *supra* notes 19 and 20, in particular, O'Brien, M. and J. Thornton, *Rachel's Hazardous Waste News*, #519; and se also: Silbergeld, E., The Risks of Risk Assessment, *New Solutions* 3(2) (1993), pp.43-44.

particularly pesticides, that have been in use for a very long time.

This approach of providing a guaranteed risk level for each chemical is exactly that; each chemical is entitled to its one-in-a-million risk level. The human population on the other hand is faced with hundreds if not thousands of these one-in-a-million risk levels (and of course many additional risks remembering that only a relative few chemicals have been assessed). In effect, the chemicals have greater rights than the human population. While each chemical is allotted a one-in-a-million risk level, (or sometimes even higher risk levels), the human population does not have the right to avoid the cumulative risk of real-world exposure circumstances. In addition, some people are more exposed than others. For example, a one-in-a-million risk level may be established for chemicals emitted for particular air emissions or water effluents or leachate from landfills. The risks however can be borne disproportionately by the population living nearby, not the hypothetical population that informed the risk assessment calculation. Even if, as some do, the risk calculations account for the localized circumstances of the exposed population, these are still groups of people disproportionately exposed to toxic chemicals, and this is often the case because they are poor or otherwise disenfranchised from the political decisions flowing from risk calculations.

Similarly, as Chapter 2 and the Pesticides Case Study describe, children are known to be more highly exposed and susceptible to the harmful effects of pesticides than are adults. They are clearly at risk from current exposure levels, and the lion's share of the risk in Canada is probably due to the over 73% of pesticide active ingredients that were not evaluated with children's circumstances in mind. Nevertheless, the fact that the pesticides are approved for use is given as justification by government officials at all levels (federal, provincial and municipal) that the pesticides are safe. Or, if statements by such officials are more accurate and specific, it is stated that risk levels have been calculated and found to be acceptable. Although there is strong evidence to show that children are at risk from current pesticide exposure, risk assessment calculations for approving additional pesticides can proceed regardless and the long overdue re-evaluation of currently approved pesticides continues very slowly within a political and funding backwater.

The second ethical problem with this approach of guaranteeing a risk level to each chemical is that risk assessment has only recently begun to consider health end-points other than cancer. There may in fact be other end-points such as endocrine disruption and neurodevelopmental effects, which may occur at even lower exposure levels or under different circumstances than the cancer risk assessment considered. These other unknown or poorly understood effects have to be assumed to be non-existant. Alternatively, they require the application of default assumptions and there is great uncertainty as to whether these assumptions adequately inform the risk assessment calculations. Further, those chemicals which are unidentified, untested, or otherwise not part of the analysis, (including the real-world situation of complex mixtures of small amounts of chemicals) must also be assumed to be safe as they are simply not part of the risk assessment exercise. Again, the chemicals are given the right to an inherently incomplete risk level calculation; the exposed human population does not have the same right to be exposed to no more than a specific level of risk. Under this ethically slippery construct, it is not surprising that the human population is experiencing a rise in the chronic health effects that toxic environmental contaminants are known or suspected of causing when "one-in-a-million" risk levels are doled out for every chemical that comes along.

The situation is not improved by the fact that the critique of risk assessment, as summarized here, is frequently not accepted by risk assessment practitioners or advocates as valid or worthy of consideration. Instead, it is seen as an unjustified attack on their scientific credentials. This reaction is ironic since what is at issue is the very lack of scientific integrity at key steps within an exercise that otherwise should be, and must be, highly dependent on "good science." A further irony is contained in the position advanced

by industry and the right-wing press,[25] generally in reaction to public concerns about toxic chemicals and insistence on better regulation. These concerns are dismissed as emotional and unscientific. The solution offered is the "objective science" of risk assessment. Like this industry approach, risk assessment practitioners often react to the critique of risk assessment by ignoring it. Instead, their non-self-critical approach is one of essentially pretending that the gaps in data and methodology are insignificant in terms of presenting barriers to continued application of what is, again, seen and described as an objective, fact-based scientific exercise. Such an approach is evident in the document prepared by the Canadian-based Network for Environmental Risk Assessment and Management (NERAM) entitled "*Current Directions in Environmental Risk Assessment and Management.*"[26]

Alternatively, for those risk assessment practitioners and advocates that recognize the scientific limitations of the process, the approach is to accept the level and degree of default assumptions as inevitable and a valid part of the exercise and something that ever more effort at refining techniques will ultimately overcome. In the meantime, they consider the solution to the problem to be a matter of improvements in risk characterization and communication.[27]

Finally, there is a very important distinction to be made between the United States and Canada in terms of the political forces that are brought to bear on risk management decisions due to underlying differences in the legal context in the two countries. This distinction stems from a fundamental difference in the United States whereby property rights are afforded to persons under the U.S. Constitution. Such rights provide persons (and by extension, corporations) in the U.S. with the ability to challenge and constrain environmental regulation (via litigation) in a way that does not exist in the Canadian legal context.[28] Therefore, decisions to harmonize Canadian standards or standard setting approaches with the U.S. may unnecessarily constrain the ability of Canadian regulatory agencies to set protective standards. It should therefore be of serious concern to Canadians if standard setting derived under, and constrained by, a different constitutional context, is borrowed by Canadian regulators who are not similarly constrained. More legal research is needed in this area.

4.2.6 Risk Assessment and Cost-Benefit Analysis

The increasing prevalence of risk assessment and risk management in environmental decision making has been the subject of much debate and controversy. A related trend is the corresponding increase in demands and/or requirements for cost-benefit analysis of environmental decisions. Part of the story of the increasing use of risk assessment and management practices is the fact that risk assessment is often a pre-

[25] See for example: Corcoran, T. The mad voyage beyond zero risk, *Financial Post*, May 8, 1999; and Junk Science, Junk Policy? Managing Risk and Regulation, Fraser Institute Conference, April 29, 1999, Ottawa.

[26] Dyck,W. *et.al.*, NERAM, 1999, *op.cit.*

[27] See for example, Stern, P. and H. Fineberg, (eds) *Understanding Risk: Informing Decisions in a Democratic Society*, Committee on Risk Characterization, Commission on Behavioral and Social Sciences and Education, National Research Council, (1996) 264 p.

[28] See e.g., *Industrial Union Department v. American Petroleum Institute, et.al.*, [1980] U.S.S.Ct. #78-911, 78-1036; 48 LW 5022. In this U.S. Supreme Court case dealing with an occupational benzene standard, brought by the American Petroleum Institute, Mr. Justice Powell, concurring with the majority said, "I conclude that the statute [the *Occupational Health and Safety Act*], requires the agency [the Occupational Health and Safety Agency] to determine that the economic effects of its standard bear a reasonable relationship to the expected benefits . . . It is simply unreasonable to believe that Congress intended to pursue the desirable goal of risk-free work places to the extent that the economic viability of particular industries is threatened . . . [the] regulations would impair the ability of American industries to compete . . . [and] would result in a serious misallocation of resources," p.5038.

requisite when a cost-benefit analysis is done for an environmental matter. Similarly, there is increasing insistence among politicians and industry representatives, particularly in the United States, for the application of *comparative risk analysis,* an approach that evaluates environmental hazards as a group to be able to assign priorities and budget allocations based on relative magnitude of risk. Application of such an approach pre-supposes that a quantitative risk assessment has been conducted for each hazard in the comparison.[29]

A form of comparative risk analysis is apparent in Health Canada's proposed decision-making framework being developed as part of its review of health protection operations. The three-part framework includes: issue identification, risk assessment and risk management. Intended to be comprehensive and consistent across all aspects of Health Canada's mandate, the draft framework focuses on general principles more than the details of risk assessment. The draft framework moves Health Canada towards a "population health approach" to risk assessment and management including a prioritization of risks so that "the most important risks are addressed." The draft speaks of putting risks "in a broad context," and though it is not explicit, there is the danger that, in a time of deregulation and cuts to departments, this will become a way of avoiding assessments and interventions that are thought to be less important. Such prioritization is described in the document as being more accountable for the "wise use of limited resources." Risk prioritization though, often falls into the trap of comparing very different types of risks, natural and human-made, voluntary and involuntary, as if they have the same importance in society. This approach is echoed in the statement that "every choice brings with it some degree of risk and that certain risks are shared by society as a whole."[30] In reality, as discussed above, risks are often unevenly shared in society, and those shouldering much of the burden often have little input into decisions.

Insistence on the evaluation of (readily quantifiable) economic impacts has often served to limit or reduce safety margins for standards derived from evaluation of uncertain (and difficult to quantify) risks. Canadian decisions in the early 1980s on lead in gasoline are a case in point (see Section 8.4.1.2 in the Lead Case Study). As the harmonization of standard setting continues between the U.S. and Canada,[31] the treatment of economic impacts in standard setting becomes increasingly important. Alongside this increased harmonization has been large cuts in staff and resources in both the federal and Ontario governments undermining their ability to create, implement and enforce toxic substance regulation. As discussed for separate departments in Chapter 3 above, between 1994 and 1998, the departments of Fisheries and Oceans, Health, Environment and Natural Resources reduced their scientific personnel by 17%. The departments admitted that these cuts included a reduction in the resources available for the assessment of toxic substances.[32] Deeper cuts have occurred at the provincial level in Ontario. Meanwhile, governments are increasingly contracting out the work of conducting human health risk assessments to private firms that generally see the world through the eyes of their corporate clients. For example, as noted in the Canadian-based Network for Environmental Risk Assessment and Management

[29] For more on this topic see CRS Report 98-618, Parts I, II, III and V, *op.cit.*, and Congressional Research Service, Issue Brief for Congress, *Environmental Risk and Cost-Benefit Analysis: A Review of Proposed Legislative Mandates, 1993-1998*, January 22, 1999. RL30031. Available at: www.cnie.org/nle/rsk-24.html. See also description in Chapter 3 of arrangements reached under NAFTA by the Pest Management Regulatory Agency and the U.S. Environmental Protection Agency.

[30] Health Protection Branch, Health Canada. *Health Canada Decision-Making Framework for Identifying, Assessing and Managing Health Risks, Draft.* (Oct 1, 1999), pp.1,6,18

[31] This increased harmonization is occurring internationally as well and flows from the implementation of international trade agreements, particularly the North American Free Trade Agreement (NAFTA). See information regarding "Trade Negotiation and Agreements" and "Regional and Bilateral Agreements" at www.dfait-maeci.gc.ca/tna-nac/reg-e.asp

[32] Federal Commissioner for the Environment and Sustainable Development. *1999 Annual Report.* s.3.60, 3.61

report addressing the state of risk assessment in Canada: "industry is facing increasing pressure to be competitive in the global marketplace and would prefer a move towards a "risk-based" approach," which would "enable industry and government to set priorities and better focus their resources on high risks that are of greatest concern."[33]

Debate over the use of risk assessment in the context of cost-benefit analysis or in assigning environmental priorities within overall governmental priorities is beyond the scope of this report. Nevertheless this context is important because risk assessment of environmental hazards is considered by its proponents as providing a scientifically sound basis for assisting with broader economic decision making. Such confidence is undermined however, by the fact that central and well-founded criticisms of risk assessment and risk management include evidence of frequently shaky scientific foundations combined with multiple opportunities for value judgements and bias.

4.2.7 Summary

The two disciplines generally known as risk assessment and risk management have developed and been applied for roughly thirty years to the task of regulating toxic substances in the environment. Risk assessment is routinely characterized as the "scientific" stage of the exercise while risk management is considered the policy-making step.

The ever-increasing complexity of risk assessment methodologies has been matched and consistently overcome by the greater complexity of the problems they attempt to address. An underlying four-step process at the heart of risk assessment includes: hazard identification; dose-response assessment; exposure assessment; and risk characterization. The second two of these four steps have consistently suffered from large gaps in data and methodology providing many opportunities for uncertainty, variability and error. Gaps have been filled with "inference choices," or "science policy options," or what critics have accurately labelled as guesswork, not science. For those risk assessment advocates or practitioners who accept this criticism, and many do not, the problem is considered inevitable and insignificant and a key solution is seen as the need to improve techniques of risk characterization and communication.

Important issues of ethics and equity arise since the complexity of risk assessment makes it the domain of specialized experts and those wealthy enough to hire them. The combination of science and guesswork provides numerous opportunities for value judgements and bias to enter risk calculations. Critics charge that risks can be disproportionately assigned to those unable to avoid them (the poor, children, etc.) and who do not share equally in the benefits. Each chemical is treated as "innocent until proven guilty" and chemicals arguably have greater rights than the human population.

When chemicals are assessed one at a time, in isolation from other chemicals, risk levels are assigned regardless of risk levels that already exist or that are yet to be calculated for new chemicals. When risk levels are assigned without accounting for all relevant health effects, or for the cumulative or synergistic effects of chemicals acting in combination, these additional risks have to be ignored and they do not inform the risk calculations. As more and more chemicals continue to have the right to be assigned a risk level (alongside the many thousands of chemicals that have never been adequately assessed), the human population does not have the same right to be exposed to no more than a specified level of risk.

The assigning of individual risk levels for each chemical is also a game of odds that cannot address two of

[33] Dyck, *et.al.*, NERAM, 1999, *op.cit.* p.1.

the most serious issues of toxic chemical pollution: inherent toxicity and population-wide effects such as may be occurring with endocrine disrupting chemicals. Risk assessment enables risk calculations that allow for "acceptable" levels of one-in-a-million or one-in-ten-thousand risks (of cancer, birth defects, etc.) across a population. However, the odds game becomes useless if further research confirms the suspicion that chemicals such as endocrine disruptors are capable of exerting population-wide effects. Nor is it appropriate to make such calculations for chemicals that are persistent and bioaccumulative. Risks will continue to increase for chemicals that do not break down and which accumulate in animal fat, breast milk, etc. These risks will of course be highest for children and other vulnerable populations than for the adult population at large.

The increasing insistence on both cost-benefit analysis and comparative risk analysis as a means of assigning government priorities and budget allocations may perpetuate the inequities that can arise from risk calculations.

4.3 THE SCIENCE BEHIND THE ASSESSMENT – EPIDEMIOLOGY AND CAUSATION

4.3.1 Introduction

Before addressing some specific examples of the application of risk assessment to the special circumstances of children, it is important for this review of risk assessment and the review of children's health effects in Chapter 2 and the two case studies, to address key scientific and methodological issues. Central questions arise as to whether and how causal connections can be shown to exist between exposure to environmental contaminants and health effects in humans.

4.3.2 Sources of Data

Epidemiological studies examine the patterns of occurrence of health problems in different groups of people with the aim of determining causes for such patterns. Epidemiological studies are generally considered an important source of information for evaluating the health risks to humans from exposure to environmental contaminants, particularly for formal risk assessments. The strength of evidence from epidemiologic studies depends upon the quality of information identifying: a) populations at risk;[34] b) an estimate of the level of exposure;[35] and c) a measure of the dose[36] received, and the proportion of the population that exhibits the particular response (i.e., health problem). The degree to which this

[34] Population at risk refers to the group of individuals among whom the particular health problems might be observed, or all people who are susceptible to or could have the disease or health problem (or a representative sample of them (see: Fletcher *et.al.*, *Clinical Epidemiology: The Essentials*. (Williams and Wilkins, Baltimore, 1988). In environmental health studies these would be people who by virtue of their occupation, residence, activities or physiology are exposed to a given environmental chemical.

[35] Exposure refers to "the extent of contact between the toxicant and the surfaces of the human body." See: Roberts, J.R., P.B. Curry, R.F. Willes, M.F. Mitchell, S.Narod and L.C. Neri, Epidemiological evidence of the effects of pesticides on human health in Canada. Monograph II. In: *Strengths and Limitations of the Benefit-Cost Analyses Applied to the Assessment of Industrial Organic Chemicals Including Pesticides*. Associate Committee on Scientific Criteria for Environmental Quality. National Research Council of Canada. NRCC No. 22852 (1985: 1).

[36] Dose refers to "the amount of toxicant in the critical organ or tissue" (Roberts *et.al.*, *op.cit.* 1985: 1).

information is known or characterized depends in large part on the strength of the research design.[37]

4.3.3 Study Design in Epidemiology

Epidemiological studies examining environmental health effects are typically observational. In other words, they observe the effect of various influences that occur by chance (i.e., the so-called "natural experiments"). This observational approach is in contrast to randomized controlled trials (frequently used in drug trials or clinical epidemiology) where, with adequate sample size, blinding of subjects, therapists and researchers, standardized measurement techniques and analysis, the unique effects of a single factor can be studied. Such experimental studies provide the strongest evidence for establishing cause-and-effect relationships, however, when investigating potentially harmful agents epidemiologists do not have this option. For ethical reasons, humans are not subjected to clinical trials of non-therapeutic agents such as pesticides. [However, some pesticide companies have crossed this line in the past and have recently increased the practice of using human "volunteers" to determine NOAELs for their products. This renewed and highly controversial practice is discussed further in Section 4.4.3.2 below.]

Observational studies are the only alternative to experiments but they also vary in their strength and the degree to which they are subject to biases. There are three basic observational study designs including: 1) cohort studies; 2) case-control studies; and 3) ecological studies, in decreasing order of statistical power.[38]

Cohort Studies

Cohort studies examine groups of people who share some attribute, such as, birth year, a particular occupation, or living in a neighbourhood at a certain time. They are also called prospective studies since the direction of research is forward in time. The investigator starts with the defined group or cohort and follows them through time to assess the development of disease or health problems. Researchers compare those who develop disease with those who don't to see which factors vary between the two groups. The cohort study represents the most powerful observational study design having fewer biases and typically with better estimation of exposure. One weakness of cohort studies is that they may need very large sample sizes to adequately detect weak risk associations especially between rare health outcomes, such as cancer, and environmental contaminant exposures. An example of a cohort study is that examining the effects of perinatal exposure to PCBs on neurodevelopment in infants whose mothers ate contaminated fish from Lake Michigan during their pregnancy. The study has followed the developmental characteristics of these children who were exposed to PCBs *in utero* and via breast milk for more than a decade now.[39] Several other examples of cohort or prospective studies evaluating the health effects of lead in children are discussed in Case Study #1.

Case-Control Studies

In case-control (also called retrospective) studies, subjects with (i.e., cases) and without (i.e., controls) a particular health problem are matched to be similar for certain variables such as, age, sex, etc., and are compared for differences in factors such as exposure. In contrast to the cohort study design, this type of study is best suited to, and statistically stronger for, studying rare health effects with a long latency period

[37] Note that b) and c) would correspond to the second two steps of risk assessment noted above, .i.e., exposure assessment and dose-response assessment. The ability of risk assessment to accurately or even adequately deal with these two steps is central to the review of risk assessment in this chapter.

[38] Power refers to the ability of a study to detect an association or a significant difference when one actually exists.

[39] 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.

such as cancer. Because these studies are retrospective, they often rely on the recollection of the subjects for details of past exposure. Accurate estimation of exposure is therefore plagued by the recall bias of subjects which may over- or under-emphasize certain details of their exposure history. They are also vulnerable to selection biases since the comparability of cases and controls rests on the assumption that they have had an equal chance of being exposed to the particular agent. (See Figure 4.3 for clarification of case control studies.) Case-control studies examining environmental causes of breast cancer have compared DDT and DDE levels in breast tissue and exposure histories to assess differences between women with breast cancer and those without.[40]

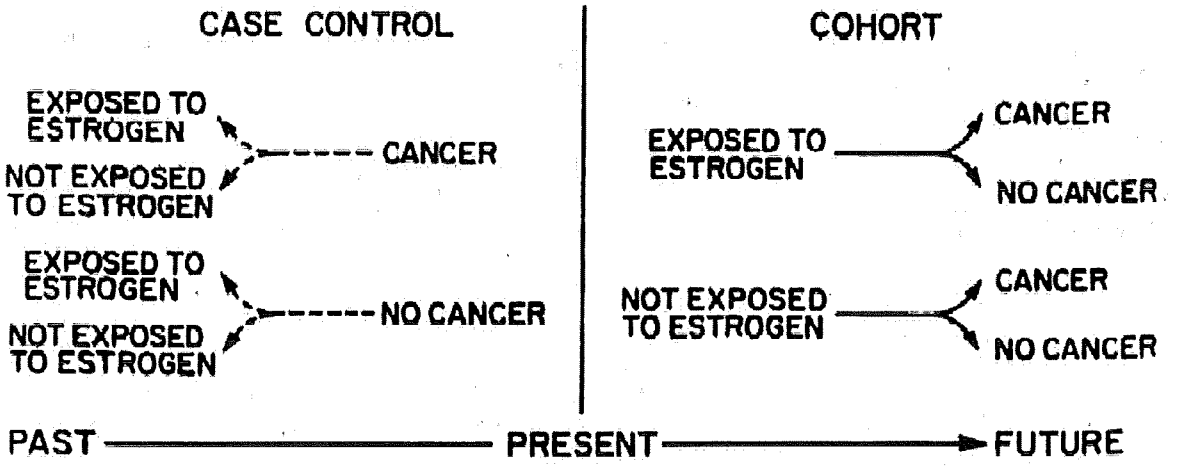

Figure 4.3 A comparison of cohort and case control research: studies of exogenous estrogens as a risk factor for endometrial cancer. (Source: Fletcher, 1988, *op.cit.* p.194).

Ecological Studies

The third type of observational study important in environmental health is the ecological design. Here, the unit of study is the population or community (vs. individuals) and the patterns of health problems and exposures are often tabulated from data (usually cross-sectional) that are already available such as mortality records, cancer registry data, etc. Data are assessed for correlations between disease and various risk factors such as, geographical location, time trends, occupation, social class, etc. (Ecological studies are often characterized as being descriptive as opposed to case-control and cohort studies which are considered analytical.) There have been many epidemiological studies that have shown associations between, for example, difference in breast cancer incidence and differences in diet, or incidence of Alzheimer's disease and levels of aluminum in local drinking water. One ecological epidemiology study has shown an association between possible excess prenatal exposure to pesticides by place of residence and an increase in rates of undescended testes in male infants.[41] Ecological studies represent statistically the weakest epidemiological design. Researchers can calculate those with particular risk factors and the proportions with a particular health outcome, but they cannot prove that the associations are necessarily causal. There is often no control over other factors that may confound or modify or be causing the effects

[40] van't Veer, P., *et.al.*, DDT (dicophane) and postmenopausal breast cancer in Europe: case-control study. *BMJ*, Jul.12, 315(1997)(7100):81-5.

[41] 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.

observed. Ecological studies are important however, for initial investigation and generating hypotheses as to possible important associations between environmental factors and health risks. Epidemiologists then rely on inferences from the analytical studies (i.e. cohort and case-control studies) to test hypotheses and to ascertain the statistical confidence in association between exposure and disease incidence.

4.3.4 Inferences of Causality in Environmental Health Studies

A fundamental step in interpreting epidemiological data is evaluating causality of the association between agent and hazard, a process that applies directly to the hazard identification element of risk assessment.[42] It is notably difficult to prove causal relationships in epidemiology. It is often only possible to increase the confidence in a cause and effect relationship observed between two factors. "When experiments are not possible and only observational studies are available, deciding whether something is a cause requires *judgement*, based on all the evidence."[43] Epidemiologists refer to criteria developed by Bradford-Hill in 1965.[44] These criteria for causation include a series of conditions that if met, can increase the confidence that an association is "real" and hence help guide the judgement as to whether a given environmental factor is a cause of disease or adverse health effect. Bradford-Hill's scheme suggests that the important criteria to consider in inferring causality include:

- *Strength of the association* - there should be a significantly higher relative risk of observing the health effect in the exposed versus the unexposed population. In this case, we might assume that such effects are not occurring by chance alone and that the evidence is stronger for a causal relationship between the environmental factor and the health effect. Note that Bradford-Hill stated that one should not too readily dismiss a cause and effect relationship just because the observed association is slight.
- *Consistency* - the association is observed by different people in different circumstances.
- *Specificity of the association* - it may be established that one cause leads to one effect. This criterion is not to be over-emphasized because diseases may have more than one cause.
- *Temporality* - which observed event came first of the associated events. This may be difficult to determine if one is examining cross-sectional data since they do not directly indicate the sequence of events.
- *Biological gradient* - whether there is a dose-response curve if it can be revealed.
- *Plausibility* - consistency with known biology, but Bradford-Hill cautioned that this requirement cannot be demanded because it is dependant on the knowledge of the day and the study may disclose something new. For example, the attention paid to researching the effects of endocrine disruptors on human health has been strengthened by "suggestive evidence of a possible role of man-made chemicals in developmental abnormalities of the reproductive tract."[45]
- *Coherence* - that the cause and effect relationship observed does not seriously conflict with the generally known facts.
- *Reversibility of an experimental/intervention effect* – if possible, it may be shown that

[42] Samet, J.M., R. Schnatter and H. Gibb, Invited commentary: Epidemiology and Risk Assessment. *Am. J. Epid.* 148(1998):929-936.

[43] Fletcher, *et.al.* 1988, *op.cit.*, p.216, emphasis added.

[44] Bradford-Hill, A., The environment and disease: Association or causation? *Proc. Roy. Soc. Med.* 58(1965): 295-300.

[45] Foster, W. Endocrine Disruptors and Development of the Reproductive System in the Fetus and Children: Is there Cause for Concern? *CJPH* 89 *Suppl* 1) (1998): S37-41, S52. S37.

removal of the proposed causal agent is associated with a decreased risk of disease.

- *Analogy* - comparison with another similar drug or disease. For example, the fact that measles causes other de-myelination disorders (subacute sclerosing panencephalitis) has been suggested as evidence for a role of the measles virus in the etiology of multiple sclerosis.[46] Analogy is relatively the weakest argument for causality.

Alongside the above scheme, several additional points regarding inference of causality were raised. First, it is important to remember that hard and fast rules cannot be laid down, and none of the factors individually can answer the question of cause and effect.[47]

Second, with respect to tests for significance, their role is to remind us that the "play of chance" can create certain effects; but that significance tests provide nothing else to the "proof" of the hypothesis. Far too often a conclusion of "no difference" is drawn from a finding of "no significant difference." Decisions in real life must be governed by the seriousness of the consequences - for instance restricting a drug administered to pregnant women on slight evidence, but requiring stronger evidence before forcing lifestyle choices on people.[48]

More recently, it has been suggested that an additional criterion should be added whereby the significance factor is combined with a reporting of the "power" of the statistical test that is used. Reporting the power would assist in preventing "type II" errors: that is to reduce the probability of concluding that there is no effect when that conclusion is in error.[49] To clarify, there are two opposing opportunities for error in making causal inferences from epidemiological studies. Type I errors involve accepting spurious associations as causal. Type II errors involve missing true causal associations. Scientific rigour is more often focused on avoiding Type I errors. This focus ensures that scientific literature is not overly burdened with false claims and that time is not wasted on correcting them. However, this preoccupation with avoiding Type I errors can result in true associations being overlooked.[50]

Further work in understanding the ideas of causation has included examination of the need to replace a paradigm in which the search is for "simple schemes of single causes" with a "scheme of multiple causes", especially in examining ecological causes.[51] While causation analysis needs to be kept arbitrarily simple in order to conduct useful work, a causation analysis is needed that allows for recognition of multiple causes. Two aspects of multiple causation include that one type of exposure or experience can give rise to a myriad of effects or manifestations among those exposed; while a single type of manifestation in different individuals may arise as a result of many diverse exposures or experiences. Such an approach would allow an investigator to begin with either causes or effects. Beginning with a cause might reduce the likelihood of missing the possibility that one cause may give rise to more than one effect. An investigator could begin with one independent variable and search out its effects. This

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[46] Fletcher, *et.al.* 1988, *op.cit.*

[47] Bradford-Hill, A. 1965, *op.cit.*

[48] *Ibid.*

[49] Bertell, R., Weight of Evidence versus Proof of Causation, In: *Applying Weight of Evidence: Issues and Practice*, A Report on a Workshop held October 24, 1993. International Joint Commission, June 1994, pp. 27-32.

[50] See for example, with respect to the literature on lead and IQ, a discussion of six flaws in study design or interpretation that have systematically reduced the risk of Type I errors but at the cost of increased risk of Type II errors. In: Needleman, H. and D. Bellinger, The Health Effects of Low Level Lead Exposure, *Annu. Rev. Publ. Health*, 12 (1991): 111-40.

[51] Susser, M., The Logic of Multiple Causes, Chapter 4 in *Causal Thinking in the Health Sciences: Concepts and Strategies in Epidemiology*. (Oxford University Press. 1973), pp. 42-47.

approach is more useful for studying the health of populations, and has application in environmental health studies.[52]

Further refinements have included a pragmatic definition of cause: "a cause is something that makes a difference"[53] and simplification of the Bradford-Hill criteria. Those criteria that are "most useful and least tautologic" in assisting with causal inference are: Strength; Specificity; Consistency; and Predictive Performance.[54] Methodological criteria for judging epidemiologic studies have also been suggested since it may be possible in advance to determine how conclusive a study is likely to be.[55] The "markers" for this purpose include: how certain is it that there has been exposure to a specific toxicant; how accurate is the knowledge of the biologic effects of an exposure on human populations; how specific is the health outcome that is to be measured; and, is there a large exposed population or relatively common adverse health effect to be measured?

From the foregoing discussion, it is important to keep in mind the elements of judgement that make up the "science" of determining causation, regardless of which paradigm is followed. As well, the elements of those judgements are not fixed but evolving as contributing factors become better understood. The implications from these points are further discussed in Section 4.3.7 below with respect to the "Implications for Decision Making and Policy Setting". It is worth remembering that:

> Epidemiologists have come to understand that the data and the assumptions used in sound causal inference and those used in sound decision making are not the same.[56]

4.3.5 Limitations of Epidemiological Studies for Risk Assessment

Direct human evidence is often not available or may be of limited use to risk assessments for a variety of reasons. It is beyond the scope of this report to adequately analyse all the limitations presented when using epidemiological data in risk assessment.[57] However, we can briefly address some of the key issues that limit use of human data for the purposes of assessing environmental health risks. Some methodological or analytical weaknesses of epidemiological studies surround identification of health effects, exposure assessment, and sample size and representativeness.

Accurate identification of health effects is important. For example, different types of cancer represent distinct disease processes and so must be specifically defined. Many health effects of concern (including cancer, respiratory problems and neurological effects) may not appear for long periods following the causal exposure or they may occur as a result of progressive accumulation of damage that doesn't produce

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[52] *Ibid.*

[53] See: Susser, M., *Epidemiology, Health & Society: Selected Papers.* (New York: Oxford University Press, 1987); and Susser, M., What is a cause and how do we know it? A grammar for a pragmatic epidemiology. *Am. J. Epidemiol.* 133(1991): 635-648.

[54] Susser, M., 1991, *op.cit.*

[55] Frank, J.W., B. Gibson, and M. Macpherson, Information Needs in Epidemiology: detecting the health effects of environmental chemical exposures. In: *Information Needs for Risk Management Environment Monograph No. 8,* D.D. Fowle, A.P. Grima and R.E. Munn (eds.) (Toronto: Institute of Environmental Studies, University of Toronto, 1988), pp. 129-44.

[56] Susser, M., 1991, *op.cit.*

[57] For recent critical discussions of the role of epidemiology in risk assessments see sources such as, Samet J.M., *et.al.. op.cit.*; and Herz-Picciotto I., Epidemiology and quantitative risk assessment: A bridge from science to policy. *Am.J.Pub.Health* 85(1995): 484-491.

identifiable effects. These types of effects are difficult to associate with a specific exposure with any degree of certainty (because of the time lag) and they are also not easily detected. With "rare" health effects such as cancer, it is also difficult to collect data on a large enough sample. Larger sample size increases the ability (statistical power) to detect real associations between exposure and outcome.

Where those conducting a risk assessment are interested in quantifying dose-response relationships, epidemiological studies are often only able to address the exposure-response relationship, i.e., there is no way of accurately determining what proportion of the amount to which people are exposed actually reaches the body tissues. Even so, risk assessors are also frequently unable to accurately determine the degree of exposure to a contaminant of interest. They can often only infer exposure from job description in occupational studies, or by place of residence or subject recall in exposures of the population at large. In many instances, exposure can only be characterized as a dichotomous variable with subjects designated as either "exposed" or "not exposed." Biological exposure data (for example, measures of contaminants from urine or blood samples) improves accuracy in assigning the dose-response relationship. However, such measurement adds extra expense and logistical problems, especially in large epidemiological studies and is often not an option in retrospective studies.

Another problem of exposure assessment is that there may frequently be multiple exposures from multiple sources. For example, children can be exposed to many different pesticides via contamination of food, drinking water, and home, school and playground surroundings. Children experience exposure to other contaminants as well and via various pathways. Multiple exposures are especially the case for those who are exposed to contaminants in both occupational and environmental settings. For instance, pesticide workers are routinely exposed to several pesticides, and other toxicants such as solvents, emulsifiers as well as "inert" ingredients.[58] This multiple exposure makes it very difficult to attribute observed health effects to exposure to a specific pesticide. A similarly complex picture exists for the children of these workers since they may be additionally exposed to pesticides on their parents' clothing, shoes, etc., or due to living very near to where their parents work. This exposure is in addition to the range of pesticide residues and contamination to which all children are exposed.

Factors other than the exposure of interest may also confound the observations. For example, poor nutrition will enhance the uptake of lead and hence, the lead-based health effects in children. Lastly, the choice of human population samples for epidemiological studies is often opportunistic. As a result there may be inadequate representation of the effects in all population subgroups (the healthy worker effect[59]) especially particularly sensitive ones, such as children or the elderly.

As a consequence of these weaknesses in epidemiological data, risk assessors rely on other types of evidence (such as animal experiments and wildlife studies) which may provide only a prediction of the nature and magnitude of the health effects in humans. However, reliance on wildlife and animal studies alone would also have limitations.[60] It is insufficient for public policy and public protection to focus solely on cancer testing or bio-accumulation. Effects may be produced at extremely low levels, but at extremely sensitive times in the development of embryos. Rather than "the dose is the poison;" the

[58] Roberts, J.R., *et.al.*, 1985, *op.cit.*

[59] In occupational epidemiology if non-exposed workers are the control sample, they are less representative of the general population, since employed people are on the whole, in better health compared to the general population which includes people with a broader range of states of health from poor to good (Roberts, J.R., et al, *op.cit.*, 1985).

[60] Colborn, T.E., A.Davidson, S.N.Green, R.A. Hodge, C.I.Jackson, and R.A.Liroff, Human Health, Chapter 7 in *Great Lakes Great Legacy?* (Washington, Ottawa: The Conservation Foundation and the Institute for Research on Public Policy, 1990).

timing may be the poison. Extremely small amounts of dioxins exposed to the mother at day 15 in a rat's gestation or at day 56 in a human's gestation may irreversibly affect sexual differentiation in the offspring.[61]

4.3.6 Weight of Evidence

The term "weight of evidence" refers to an approach to estimating human health risks from toxic environmental exposures that makes use of all the best available science and data collected by accepted scientific methods.[62] In particular, it considers data from a variety of sources including toxicological (animal) studies, wildlife studies, and epidemiological studies of both acute and chronic exposures in humans. The more extensive the research, and the more consistent the results across different studies and in different species, the stronger is the weighting given to a judgement that a given contaminant may pose a risk to human health. Weight of evidence comes into play when identifying potential human health hazards, especially when appropriate human data are lacking and inferences have to be made about the degree of proof that is provided by existing toxicological data. A "weight of evidence" approach was taken in the 1990 work, *Great Lakes Great Legacy?*[63] It reviewed the available evidence from:

wildlife (replication in laboratories, observation of health effects such as wasting, birth defects, immune suppression and target organ damage in offspring, cancerous tumours);
public health data (cancer incidence, cancer mortality, and reproductive outcomes);
human exposure from tissue analysis (pesticides and pesticide break down products found in human tissues); and
studies of individuals, (such as from eating Great Lakes fish).

Exposure pathways addressed included: ingestion of food, drinking water, inhalation, body contact with water and from other less well understood pathways.

The approach to assessing "weight of evidence," appropriately requires a differentiation between "science" and "policy;" the latter being informed by many disciplines, including science, but also ethics, values, opinion, conflicting interests and perspectives.[64] The next section, Implications for Decision Making and Policy Setting, explores this difference further.

Related to the "weight of evidence" approach is the need to consider[65] newly developing methods for using epidemiologic evidence in decision making and standard setting, such as geographically-based health information and meta-analysis. Meta-analysis is a means to review or re-review a wide range of previously conducted studies and can assist in gathering the most relevant evidence and in analysing the collective implications of a range of studies. It has been particularly useful in the understanding of the health effects of low-level lead exposure in children (see Section 8.3.4.1 in Case Study #1). Geographical information systems are becoming more important as databases are created and the ability to map the

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[61] Colborn, T, Listening to the Lakes, *Pesticides and You*, June, 1992: 4-8.

[62] Sixth Biennial Report Under the Great Lakes Water Quality Agreement, International Joint Commission, 1992. Available at: www.ijc.org/comm/6bre.html

[63] Colborn, T.E., *et.al.* 1990, *op.cit.*

[64] Weinberg, J. and J. Thornton, Scientific Inference and the Precautionary Principle. In: *Weight of Evidence: Issues and Practice, A Report on a Workshop held October 24, 1993.* (International Joint Commission, June 1994), pp 20-6; see also Fox, G. Scientific Principles. In: *Weight of Evidence: Issues and Practice, A Report on a Workshop held October 24, 1993.* (International Joint Commission, June 1994), pp 2-5.

[65] Pershegen, G. Environmental epidemiology in public health. *Lancet* 352(1998): 417.

information becomes more widespread.[66]

4.3.7 Implications for Decision Making and Policy Setting

The fact that efforts to determine causation and interpretation of epidemiological and other scientific studies involves considerable judgement has important implications for decision making and policy setting based on those studies. As noted above,[67] "science" is different from "policy." Policy is informed by many disciplines, including science, but also ethics, values, opinion, conflicting interests and perspectives. The foregoing review of the way in which the "science" is conducted illustrates that it is an impossible demand of science to provide the policy answers. Furthermore, the judgements and conclusions based on "science" may be far from certain even in terms of the limited questions that science attempts to answer. Accordingly, decisions must be made, based on all of the best available information. While the results of "science" (epidemiological studies; assessments as to contributors to the questions of "causation", etc.) are important contributors to the decisions, science is incapable of playing the role of the sole determinant of these questions.

Standard setting is primarily a policy-making exercise. Decisions on policy entail a review of the science, together with many other judgements. A "weight of the evidence" approach is appropriate for policy making as to standards, i.e., in the risk management process itself, not solely at the hazard identification stage. An important question in that context is what "burden of proof" to demand; where to place the "burden of proof"; and what elements of "proof" to consider in making standard setting decisions.

There is a history of considering differences in required burdens of proof in legal decision making. Two commonly applied standards are the usual civil "balance of probabilities" (which means "is the contested fact more likely than not?") and the criminal law standard of proof of the contested facts as being "beyond a reasonable doubt." The reasons for the differences vary with the reasons behind the court proceedings that apply these different burdens. In criminal proceedings, the legal system has institutionalized an approach that, ideally, makes it extremely unlikely that an innocent person would be wrongly convicted. It is understood and accepted in that approach that sometimes, "guilty persons" will not be convicted. This is because the value of freedom for innocent persons is strongly protected by our legal system. On the other hand, for civil disputes – that is, disputes between two parties over contracts, tortious claims and other such matters – the value is on expeditious resolution of disputes based on defendable and reasonable evidence. The burden is slightly higher on the party claiming a legal wrong has been committed, but they need not satisfy the decision maker "beyond a reasonable doubt" – it is only necessary to show that their claim is more likely than not "true" based on the evidence.

Because standard setting is intended to protect human health and welfare, ecosystems and other very high values, the "burden of proof" that is required in standard setting should be one that is more likely to be protective of those desired values. Too often, however, a protective approach has not been the case due in part, to a mis-application of the ideas of causation and the statistical significance testing that is applied to epidemiological studies.

In epidemiological studies, statistical tests that estimate the likelihood that the study has produced the correct answer (e.g. a causal link is present) have been set, usually at 95% or 99% "confidence" levels. It is important to remember that these confidence levels are arbitrary cut-off points chosen for convenience

[66] Another new measurement tool for assessing complex data includes the Child Behavior Check List, discussed in Section 8.3.5 of the Lead Case Study.

[67] Weinberg and Thornton, 1993, *op.cit.*

and consistency; they have "no sound logical basis and [remain] unjustified."[68] They indicate the statistical likelihood that an association shown in a study is purely due to chance. Said another way, scientific convention takes the level of less than 5% or less than 1% as a limit in judging whether an effect is to be considered significant or not. The value that the scientific method is protecting in this approach is a value to base hypotheses and further work on studies that meet this extremely rigorous test.[69] These tests do *not* mean that when the confidence level is less than 95% or less than 99% that the association is not present. They just mean that as the confidence level decreases, it becomes more and more possible that the association that was found is an artifact of chance.

However, to base standard setting decisions on the same approach *before* establishing protective measures or refusing to allow additional exposures raises the likelihood that too much exposure is allowed. One noted legal text on evidence discussed the possibility in some circumstances of a third standard of proof. It was described as that of "clear, strong and cogent" evidence.[70] There are also legal evidentiary tools that assist decision makers, such as the establishment of common inferences and presumptions. The "presumption of innocence" is an example. In deciding who should bear the risk from environmental contaminants, the burden should be shifted once there is epidemiological evidence showing an increase in incidence of the harm under study.[71] Normally the legal concepts of duty of care, the failure of which may lead to legal liability, are based on a "balance of probabilities" or "50% plus one" likelihood standard. Standard setting policy decisions should follow a paradigm in which it is *at least* "more likely than not" that the appropriate protective decision has been made – that is, that a standard is set that is protective of children's health. An approach that requires human epidemiological evidence demonstrated at a 95% or 99% confidence interval *before* taking protective action would not meet this requirement.[72] On the other hand, an approach that truly weighs all of the available evidence and arrives at a prudent protective judgement based on all of that "weight of the evidence" would be more likely to meet this standard.

In considering this issue, "precautionary inference" is proposed as a method to make scientific judgements when data are incomplete or inconclusive, and where significant harm may follow from a false negative judgement.[73] This approach would be a reversal of the current scientific and policy framework. For instance, since data are lacking for most chemicals, if a given chemical belongs to a class for which it is plausible to presume that members of that chemical class may be persistent toxic substances, the onus, under this approach, should be reversed. Hence, using a reverse onus approach, specific exceptions could be made upon proof that a particular chemical is not a persistent toxic substance. Similarly, for those engaging in processes that mix chemicals and release the products of those mixtures, the onus should be on them to demonstrate that the processes do not result in the release of persistent toxic substances. These recommendations are specifically directed to the area of environmental

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[68] Cornfield, J., Recent methodological contributions to clinical trials, *Am.J.Epidemiol.* 104(1974):553-58, as discussed in Needleman and Bellinger, 1990, *op.cit.*

[69] The intention, as noted above with respect to drawing causal infererences from epidemiological studies, is to avoid Type I errors (i.e., accepting spurious associations as causal). Notably, one of the six flaws found by Needleman and Bellinger, 1990, *op.cit.*, in the literature on lead and children's IQ, was a tendency to overvalue the status of the P value (or confidence level) as a criterion for inferring causality. The result was an increased tendency to overlook causal connections, i.e., an increase in Type II errors.

[70] Cross, Sir Rupert and C. Tapper, *Cross on Evidence*, 6th edition, (London (UK) Butterworths, 1985).

[71] Harris, O.F., Toxic Tort Litigation and the Causation Element: Is there any hope of reconciliation? *Southwestern Law Journal* I 40(Sept.1986): 909-965.

[72] Jenicek, M., Rules of Evidence: Criminality and Causality. In: *Epidemiology: The Logic of Modern Medicine.* (Montreal: Epimed, 1995), pp 192-4.

[73] Wineberg and Thornton, 1993, *op.cit.*

contamination and health damage. Rather than the traditional epidemiological approach, in which all confounding variables cannot be controlled and the "webs of cause and effect ... are too complex to be fully illuminated by the tools and models currently available...", a precautionary inference approach would rely on "an integrated body of evidence from laboratory experiments, wildlife studies and epidemiological investigations... to consider [not] whether causal relationships have been definitively proven, but whether the body of evidence suggests a plausible hypothesis that harm has occurred."[74]

Herein lies the central difference between standard setting approaches that apply risk assessment versus a precautionary approach. As the next two sections and indeed the rest of this report explores, in both the U.S. and Canada, the application of risk assessment has predominated and contaminants largely have been considered "innocent until proven guilty." Section 4.5 below however, addresses these issues (weight of evidence, burden of proof, and precautionary inference) again with respect to the implications of a precautionary approach to standard setting that is protective of children's health.

In light of the foregoing discussion of science and policy, and for the purposes of introducing more detailed discussion of the application of risk assessment, it is worthwhile asking a central question.

For a given standard or proposed standard, is the best hypothesis, based on all of the evidence, that harm is *not* likely to occur to children? If not, the standard should be improved (made more strict) until the best hypothesis on all of the evidence is that at that standard, harm to children is *not* likely to occur. For areas of uncertainty that make it difficult to assess this question, the approach should be modified by a precautionary approach. In that case, the standard should be made appropriately more rigorous unless and until the uncertainty is resolved to demonstrate on "clear, strong and cogent evidence" that at the permitted exposure level, no harm to children will result. As the discussions in Section 4.4 and subsequent chapters reveal, a precautionary approach has not been followed for the majority of standards affecting children's health.

4.3.8 Summary

The progress of scientific inquiry is an extremely cautious effort of avoiding errors and drawing conclusions only when a high level of certainty can be gleaned from the evidence. As sources of information for assessing human health risks of environmental contaminants, the three types of epidemiological studies have important strengths and key weaknesses. The strength of scientific evidence from epidemiological studies depends on the quality of the underlying information. Unfortunately, for epidemiological studies addressing environmental contaminants, basic information and methodologies are either lacking or fundamentally constrained. These constraints apply to the scientific work necessary to undertake the two risk assessment steps at issue in this chapter, dose-response assessment and exposure assessment.

The weaknesses inherent in individual studies and epidemiological data in general make it very difficult to draw causal inferences. In drawing inferences of causality from epidemiological evidence, investigators must look at a body of evidence as to environmental factors and human health effects. A detailed set of criteria is applied to make inferences as to causality. Even with these criteria, a great deal of judgement must be applied within the "science" of determining causation.

Risk assessors must rely on additional evidence from animal experiments and wildlife studies which can only provide a prediction of the nature and magnitude of health effects in humans. Such predictions are

[74] *Ibid.*

also limited by the many differences between animal and human species and the shortcomings in methodologies to assess effects at sensitive and/or developmental lifestages.

A "weight of evidence" approach to estimating human health effects considers the best available science from the full range of sources. It differentiates between "science" and "policy," the latter including science but also ethics, values, opinions, and conflicting interests and perspectives. The complexity of these inquiries is also assisted by new and developing methods of data evaluation such as meta-analysis and spatial analysis such as via geographical information systems.

Many implications arise when applying judgement and non-scientific values to the process of weighing a body of evidence and setting policy or standards for exposure to contaminants. Key among them is the choice made as to the "burden of proof" demanded. Because standard setting is intended to protect human health and welfare, ecosystems and other very high values, the "burden of proof" that is required in standard setting should be one that is more likely to be protective of those desired values. However, standard setting rarely applies such a protective approach. Instead, protective standards generally are not set until rigorous scientific inquiry has been applied to the available (and always incomplete) information in order to verify proof of harm. The result is delay in setting protective standards and the greater likelihood of too much exposure before protective action is taken.

A more appropriate standard of proof would incorporate the legal concepts of duty of care, based on a "balance of probabilities" or "50% plus one" likelihood standard. Standard setting policy decisions should follow a paradigm in which it is *at least* "more likely than not" that standards have been set that will be protective of children's health. Where data are incomplete or inconclusive, the approach of "precautionary inference" is a more prudent and appropriate means of making scientific judgements particularly since significant harm may flow from incorrectly assuming that no harm is possible from the environmental contamination being regulated. This approach reverses the current scientific and policy framework, recognizes the inherent shortcomings of information and methodologies, and would set protective standards first. Such standards would be made less stringent only when the uncertainty as to the toxicity of the chemical hazard is resolved via "clear, strong and cogent evidence" that, at the permitted exposure level, no harm to children or other sensitive populations will result. Such a "reverse onus" approach would place the scientific burden of proof on those wishing to create environmental contamination while regulatory agencies could apply precautionary inference to the setting of protective standards including a "weight-of-evidence" approach.

4.4 ASSESSMENT OF CHILDREN AT RISK

4.4.1 Introduction

The 1990s saw an explosion of publishing, mostly in the U.S., about the health effects in children of environmental contaminants, particularly pesticides.[75] The highly influential 1993 report of the U.S.

[75] See for example: CALPIRG – California Public Interest Research Group Charitable Trust and PSR – Physicians for Social Responsibility (Greater SF Bay Area and LA Chapters). *Generations at Risk: How Environmental Toxicants May Affect Reproductive Health in California* (November, 1998); Canadian Institute for Child Health. *Proceedings from National Symposium on Environmental Contaminants and the Implications for Child Health*, (May, 1997); Consumers Union. *Do You Know What You're Eating? An Analysis of U.S. Government Data on Pesticide Residues in Foods*. Consumers Union of the United States, Inc. Public Service Projects Department, Technical Division, Groth, E., C.M. Benbrook and K. Lutz, (February, 1999); Consumers Union. Worst First: High Risk Insecticide Uses, Children's Foods and Safer

National Research Council, *Pesticides in the Diets of Infants and Children*, set the stage for the ensuing debate over whether and how pesticides and other contaminants could be regulated to protect children's health. The NRC review of children's health issues with respect to pesticides is summarized in Chapter 2 and Case Study #2 of this report.

The policy conclusions drawn by the NRC significantly influenced subsequent changes to U.S. law and policy concerning pesticides and other environmental contaminants. Legal reforms included the passage in 1996 of the *Food Quality Protection Act* and amendments to the *Safe Drinking Water Quality Act.* Also in 1996, the EPA confirmed policy commitments to consider risks to infants and children during risk assessments throughout its environmental policy-making.[76] This commitment to protecting children's health was made government-wide under President Clinton's April, 1997 Executive Order entitled "Protection of Children from Environmental Health Risks and Safety Risks."[77] In addition to broadening the consideration of children's health issues from pesticides to all environmental contaminants and all government departments, the Executive Order focused primarily on research. It established an inter-departmental Task Force (the Task Force on Environmental Health Risks and Safety Risks to Children) and led to subsequent decisions to establish Centers of Excellence across the country to address the many research needs within the field of children's environmental health.

These and many other legal and policy initiatives are all part of the story of how children's environmental health risks have been addressed in the United States in recent years.[78] Risk assessment has been central to all of these new developments. However, for the purposes of addressing the role of risk assessment and informing an understanding of its application by Canadian regulatory agencies, this review focuses on the recommendations of the NRC report and their subsequent implementation in the *Food Quality Protection Act* of 1996.

4.4.2 The NRC Benchmark

The NRC reviewed in detail the shortcomings in exposure assessment and toxicity testing for pesticides as these relate to the special circumstances of children (in all life stages including prenatal, neonatal, and adolescence). Key gaps were identified in terms of both data and methodologies for assessing exposure to, and metabolism and toxicity of, pesticides during children's developmental stages. For the two key areas of risk assessment uncertainty – exposure assessment, and dose-response assessment – the NRC made several important recommendations.

Alternatives. (Washington: Consumers Union of the United States, September, 1998).; Davies, Katherine. Pesticides and Your Child. AN Overview of Exposures and Risks. Prepared for the Campaign for Pesticide Reduction (CPR!), Ottawa, Ontario. (1998); National Research Council. *Pesticides in the Diets of Infants and Children.* (Washington: National Academy Press, 1993); Repetto, R. and S.S. Baglia. *Pesticides and the Immune System: The Public Health Risks.* (Washington: World Resources Institute, 1996); Schettler, T., G. Solomon, M. Valenti and A. Huddle, Generations at Risk: Reproductive Health and the Environment. (MIT Press: Cambridge, 1999); Wargo, J. 1999, *op.cit.*; etc.

[76] United States Environmental Protection Agency, Office of the Administrator. *Environmental Health Threats to Children*, EPA 175-F-96-001, September, 1996. Available at: www.epa.gov/epapages/epahome/epadocs/child.htm

[77] Executive Order No. 13045, Protection of Children from Environmental Health Risks and Safety Risks, April 27, 1997. Available at: www.epa.gov/children/document/executive.htm

[78] For a detailed overview of numerous EPA programs see: The EPA Children's Environmental Health Yearbook, June, 1998. Available at: www.epa.gov/ocepa111/NNEMS/oeecat/docs/1075.html ; see also the activities of the Tolerance Reassessment Advisory Committee at: www.epa.gov/oppfead1/trac

For dietary exposure calculations, the ability to quantify pesticide exposure from data about pesticide residues on food was found lacking. Inconsistent and weak data collection and analysis of pesticide residues have made interpretation difficult. These shortcomings have been particularly problematic for determining the potential for pesticide residue exposure in the major foods consumed by children. Pesticide residues in water had been largely overlooked.

The NRC recommended standardization and coordination of data collection and analysis, the need to pay special attention to collecting sufficiently large samples of data by children's age sub-categories, and also paying attention to child-specific consumption patterns including water consumption. For non-food sources of pesticides, the NRC noted the many additional routes of exposure (as discussed in Chapter 2 and Case Study #2) and found that these exposures were not considered in setting pesticide tolerances. Hence, the report recommended that risk estimates for individual pesticides consider exposure from all sources as well as the need to estimate intake of multiple pesticides with a common toxic effect. Exposure distributions, in contrast to point estimates, were considered a more relevant means of characterizing exposure and for contributing to evaluations of both acute and chronic toxicity. The report recognized however that the identified shortcomings in data collection as well as methodological problems undermine the ability to conduct these three kinds of exposure estimation (i.e., calculations of: exposure from all sources; exposure to multiple pesticides with common toxic effects; and exposure distributions). Hence additional recommendations were made concerning methods to estimate exposure.

To address shortcomings in the area of dose-response assessment, numerous recommendations were made for new toxicological tests and new pharmacokinetic[79] models to include specific consideration for children's unique biochemical and physiological characteristics. Refinements in animal testing were also recommended to evaluate toxicity to developing organ systems, and to evaluate developmental impacts on the central nervous system, the endocrine and immune systems and the reproductive system. For example, recommendations were made for modified testing for chronic toxicity/carcinogenicity in rats to address *in utero* exposure during the last trimester, exposure during lactation and following weaning, and oral exposure through diet. Similarly, recommendations were made for improved or new testing requirements to assess developmental and functional neurotoxicity and to expand acute and subchronic neurotoxicity testing for all food use pesticides (i.e., to expand beyond an earlier focus on only the organophosphate and carbamate pesticides).

Perhaps most significantly, the NRC report made two recommendations that attempted to address central criticisms of risk assessment – i.e., the large gaps in data and methodology, and for the latter, the problem of not assessing real-world combinations of chemical exposures. To address the "data gap," particularly as it relates to children, the NRC recommended the use of an additional 10-fold margin of safety. While many recommendations, noted above, were made to fill the data gap, this additional margin of safety was intended for situations where information is incomplete. To address real-world combinations of chemicals, the NRC found that exposure estimates and dose-response assessments should not be restricted to the impact of a single pesticide but should be required to assess the cumulative effect of pesticides with a common toxic effect.

[79] Pharmacokinetics is the process of uptake, biotransformation, distribution, metabolism and elimination of drugs and their breakdown products by the body. Toxicokinetics is a variant on this term used to refer to the body's reaction to toxic chemicals.

4.4.3 The Food Quality Protection Act

The NRC report recommendations noted above were adopted almost entirely in the *Food Quality Protection Act* (FQPA).[80] Adopted by Congress with only a single dissenting vote in August of 1996, the new law fully embraced risk assessment as the means to evaluate existing and new pesticides for their impacts on children.[81] It was described as being guided by the principles of "sound science" and "health-based approaches to food safety."[82] The FQPA amended the *Federal Insecticide, Fungicide, and Rodenticide Act* (FIFRA),[83] governing registration, sale and use of pesticide products, and the *Federal Food, Drug, and Cosmetic Act* (FFDCA),[84] under which EPA sets allowable pesticide residue levels for food, also called food tolerances.

Large public interest and environmental groups supported the new law because it required, (or they thought it would require), an additional ten-fold safety factor in risk calculations to provide added protection for children. The pesticides industry supported the new law in exchange for removal of the disliked Delaney Clause and the establishment of a regulatory regime founded on science-based, risk assessment. Those who have consistently criticized risk assessment however opposed the new law as legalizing statistically predictable deaths from toxic residues on foods. They also mourned the loss of the precautionary ethic contained in the Delaney Clause.[85]

To resolve the "Delaney Paradox," the new law replaced the conflict between FIFRA and FFDCA by establishing a new standard of food safety. The new approach would establish legally allowable limits of pesticide residues on food (tolerances) that would ensure a "reasonable certainty of no harm" while recognizing the benefits of using pesticides on food crops. Hence pesticide tolerance levels established under FFDCA were required to ensure that only "safe" residues were left on food. "Safe" levels were to be calculated (as recommended in the NRC report) by taking into account aggregate exposure from all dietary and non-dietary sources and all routes of exposure (including drinking water, household sources but excluding occupational sources). For all tolerance levels established prior to the passage of the FQPA, re-evaluation against this new standard was required.[86] Note that this new set of rules governs the "exposure assessment" step of risk assessment.

For the "dose-response assessment" step of risk assessment, the new law set out a number of requirements for assessing the risk of pesticide residues allowed by a tolerance. The FQPA requires EPA (again, as recommended in the NRC report) to consider:

- the susceptibility of children to exposure and/or to adverse health effects;
- potential effects of *in utero* exposure;

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[80] *Food Quality Protection Act,* Pub. L. No. 104-170, 110 Stat. 1489 (1996).

[81] In the same year, Congress also passed another risk-based law with amendments to the *Safe Drinking Water Quality Act* to explicitly require the assessment of all impacts on children and other sensitive populations for setting drinking water standards.

[82] United States Environmental Protection Agency, *1996 Food Quality Protection Act: Implementation Plan.* Prevention, Pesticides and Toxic Substances, March, 1997(hereinafter: USEPA, FQPA Implementation Plan).

[83] *Federal Insecticide, Fungicide and Rodenticide Act (FIFRA),* 7 U.S.C. s/s 135 (1972).

[84] *Federal Food, Drug, and Cosmetic Act (FFDCA),* 21 U.S.C. 301 (1996).

[85] Stroshane, T., U.S. Food Quality Protection Act: Will the Risk Cup Runneth Over? *Global Pesticide Campaigner*, 9(1) (1999), pp.1,4-8.

[86] USEPA, FQPA Implementation Plan, *op.cit.*

- potential disruptive effects on endocrine systems; and
- cumulative risks due to exposure to the pesticide and to other pesticides that may have a similar toxic effect (i.e., common mechanism of toxicity").[87]

To specifically address the particular susceptibility of children to pesticide exposure, FQPA requires EPA to:

> Use an extra 10-fold safety factor to take into account potential pre- and post-natal developmental toxicity and completeness of the data with respect to exposure and toxicity to infants and children. A different safety factor may be used only if, on the basis of reliable data, such a factor will be safe for infants and children.[88]

In exploring how the risk assessment basis of the FQPA has been implemented, it is worth recalling the 1983 report by the National Research Council. Recall that *Risk Assessment in the Federal Government: Managing the Process* noted that it was necessary to make at least 50 "default assumptions" during cancer risk assessment that are not based on science. Recommendations were made to separate risk assessment from risk management and to develop detailed guidelines for the risk assessment part of the process. Work progressed on a number of guidelines, particularly on cancer risk assessment.

A second influential report by the National Research Council, published in 1994, revisited these issues and found that very little had changed in terms of the amount and necessity of applying "default assumptions" in risk assessments. However, that report, *Science and Judgement in Risk Assessment*,[89] supported the continued use of "default assumptions" thus validating the manner in which risk assessment practice and guidance documents had continued to be developed.[90] The result of both of these influential NRC reports, recommending the development of detailed guidance on risk assessment, has been the generation of an enormous amount of documents. Over the course of many years of iterative drafts, guidance documents have been proposed, consulted upon, revised, and in some cases finalized, for exposure assessment,[91] developmental toxicity,[92] carcinogenicity,[93] reproductive toxicity,[94] neurotoxicity,[95] among others.

Despite the existence of all of these guidelines (in either draft or final form) prior to the passage of the FQPA, they all fall short of providing the additional guidance necessary to implement key aspects of this

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[87] *Ibid.* See also: Congressional Research Service, CRS Issue Brief for Congress, *Pesticide Residue Regulation: Analysis of Food Quality Protection Act Implementation.* RS20043, August 3, 1999. Available at: www.cnie.org/nle/pest-10.html (hereinafter: CRS Issue Brief RS20043, 1999).

[88] USEPA, FQPA Implementation Plan, *op.cit.*

[89] National Research Council. *Science and Judgement in Risk Assessment.* (Washington, D.C., National Academy Press, 1994).

[90] It also recommended the development of systematic and transparent guidelines that would ensure clear communication to the public of the "default assumptions" applied during the risk assessment process. The NRC published further on this matter in 1996 in: Stern, P.C. and H.V. Fineberg (eds.) *Understanding Risk: Informing Decisions in a Democratic Society*; Committee on Risk Characterization, National Research Council, 264 p.

[91] *57 Federal Register* 22888-22938, May 29, 1992.

[92] *56 Federal Register* 63798-63826, Dec. 5, 1991.

[93] *61 Federal Register* 17959-18011, April 23, 1996.

[94] *61 Federal Register* 56273-56322, October 31, 1996.

[95] *63 Federal Register* 26925-26954, May 14, 1998.

new law. For example, it has been necessary to develop two additional guidelines for improving exposure assessment, one for data requirements[96] and another for performing aggregate exposure and risk assessments.[97] Still more guidelines are proposed for first identifying and then assessing chemicals with a common mechanism of toxicity (two different guidelines)[98], another is proposed for screening chemicals (87,000 of them) for their potential as endocrine disruptors.[99] Thereafter, presumably additional guidance will be necessary for both exposure assessment and toxicity testing (dose-response assessment) for endocrine disruptors.

As of February 2000, none of these guidelines is yet finalized and all are hotly debated. Particularly contentious is the treatment of the application of the 10-fold safety factor. Notably, the approach has not been to simply apply the 10-fold safety factor. Rather, it has been either omitted or, in many cases, been only partially applied. Two draft policies[100] on its application are also the subject of debate. Despite the promise of a simpler system, FQPA implementation has been mired in controversy and delay and environmental group critics contend that overall risks from pesticides to children have likely only increased since its passage.[101]

Once again, the two areas where data and methodology are most lacking – exposure assessment and dose-response assessment – call into question the ability of risk assessment to deliver on the FQPA's promise of protecting children using "sound science." This fundamental shortcoming of risk assessment serves the pesticides industry very well since it can insist on more scientific evidence any time a risk assessment results in a conclusion that would require reduction or elimination of a pesticide product. The industry influence is equally pervasive at early stages since the insistence on science-based approaches also occurs at the level of the drafting of guidelines that steer the entire process. The result of this influence is a tendency across all of the proposed guidelines to increasingly limit and constrain the application of the key progressive elements of the FQPA. This situation is discussed further below with respect to the application of the 10-fold safety factor and the requirements for assessing aggregate exposure and the cumulative effects of chemicals with a common mechanism of toxicity.

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[96] United States Environmental Protection Agency, Scientific Advisory Panel (SAP), *Draft – Exposure Data Requirements for Assessing Risks from Pesticide Exposure of Children*, March 8, 1999. Available for review but not for citation or quotation at: www.epa.gov/oscpmont/sap/1999/may/10xdoca3.pdf

[97] United States Environmental Protection Agency, Scientific Advisory Panel (SAP), *Draft – Guidance for Performing Aggregate Exposure and Risk Assessments*, February 1, 1999. Available at: www.epa.gov/oscpmont/sap/1999/february/guidance.pdf

[98] United States Environmental Protection Agency, Office of Pesticide Programs, *Guidance for Identifying Pesticide Chemicals and Other Substances That Have a Common Mechanism of Toxicity*, January 29,1999. Available at www.epa.gov.fedrgster/EPA-PEST/1999/February/Day-05/6055.pdf ; and United States Environmental Protection Agency, Scientific Advisory Panel (SAP), *Preliminary Draft - Proposed Guidance on Cumulative Risk Assessment of Pesticide Chemicals That Have a Common Mechanism of Toxicity*, August 29, 1999. Available at www.epa.gov/oscpmont/sap.1999/september/cumdoc.pdf and not for citation or quotation.

[99] *63 Federal Register* 71541-71568, December 28, 1998.

[100] United States Environmental Protection Agency, *Draft – Toxicology Data Requirements for Assessing Risks of Pesticide Exposure to Children's Health*, Report of the Toxicology Working Group of the 10X Task Force, April 28, 1999; and United States Environmental Protection Agency, Office of Pesticide Programs, *Draft – The Office of Pesticide Program's Policy on Determination of the Appropriate FQPA Safety Factor(s) for Use in the Tolerance-Setting Process*, May, 1999. Both available at: http://www.epa.gov/oppfead1/trac/science/index.htm#additional

[101] Letter from Kenneth Cook, president, Environmental Working Group to Vice-President Albert Gore, October 28, 1998. Available at: www.ewg.org as cited in Stroshane, T. 1999, *op.cit.*

As the weighty and bewildering pile of guidance and policy documents continues to be developed, negotiated, and disputed, the EPA's 1997 Implementation Plan for the FQPA, has been relied upon for interim strategies to begin the massive task at hand.

Overall, the new law required EPA to reevaluate 33% of existing residue limits for food-use pesticides by August 3, 1999, 66% by August 3, 2002, and 100% by August 3, 2006. Over 9,000 pesticide tolerances were in line for re-evaluation when the FQPA was passed and tolerances for the riskiest pesticides were to be evaluated first. This requirement proved very difficult since the pesticide industry and the farming community mounted stiff opposition over the prospect of losing some of the riskiest pesticides, particularly the carbamates and organophosphates. Nor did data collection or methodology exist to adequately assess these riskier products according to the new FQPA requirements; hence the need for the additional detailed guidance noted above.

Instead of addressing the riskiest first, EPA met its numeric deadline by re-evaluating over 3,000 tolerances. However, it concluded that the majority of these tolerances posed no significant risks as many were for outdated practices, i.e., the residue limits were on crops no longer treated with the pesticide in question. Hence of the nearly 1000 pesticide uses that were revoked due to this reevaluation, many were for inactive uses. In addition, during 1998, EPA reported registering 27 new pesticides and over 100 new food uses for previously registered pesticides.[102]

4.4.3.1 The 10-Fold Safety Factor

The FQPA was passed in response to the alarm bell sounded by a renowned group of scientists that children faced unacceptable risks from existing pesticide exposure. The surface impression of an additional 10-fold margin of safety to specifically protect children was understandably interpreted as a step forward; protection for children would be increased 10-fold. However, this increased margin of safety has not occurred.

The qualifying started with the FQPA Implementation Plan and the results were soon apparent. In the first year after the law was passed, of 90 reassessed pesticides, the EPA applied the 10-fold safety factor only 9 times. In other cases, a factor of 3 or less was applied.[103] The Implementation Plan justified this approach and it has been a matter of ongoing controversy.

For the purpose of background information to this discussion it is worth noting first that the approach of applying "safety factors" in risk assessment is one of the key areas of "default assumptions." More typically called "uncertainty factors," it is standard practice to apply two 10-fold safety margins (i.e., an overall 100-fold margin) to the No Observed Adverse Affect Level (NOAEL), determined using animal toxicity studies. The animal NOAEL is divided by 10 to account for unknown differences between animals and humans. Hence the NOAEL for humans is assumed to be 10 times lower than for the laboratory test animals. An additional 10-fold safety margin is added to account for unknown differences in sensitivity among people. Hence, the two 10-fold uncertainty factors result in a NOAEL for humans that is 100 times lower than the NOAEL observed in the animal tests. It has also been standard practice in risk assessments to decide, using scientific judgement, whether and if to apply an additional uncertainty factor (typically in the range between 3 and 10) to account for incomplete test data such as incomplete information about risks to children.

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[102] United States Environmental Protection Agency, *Pesticide Program Highlights from Fiscal Year 1998*, November , 1998 as cited in Stroshane, T., 1999, *op.cit.*

[103] Stroshane, T.,1999, *op.cit.*

It is this final and additional 10-fold safety factor, above and beyond the routine application of the 100-fold safety factor, that has been the subject of debate under the FQPA. The direction given in the statute is that EPA shall add the additional safety factor to protect infants and children against threshold effects unless EPA determines based on reliable data, that a different margin will be safe. EPA chose right from the start to interpret the FQPA mandate to mean that the application of the additional child-protective 10-fold safety factor would remain a matter of scientific judgement.[104]

Instead of adding the 10-fold safety margin where data are incomplete, EPA interprets this direction to mean that where data are incomplete it will use scientific judgement to decide on adding a safety factor of between 3 and 10 based on *how much* information is incomplete. Hence, incomplete information does not mean the application of a 10-fold safety margin but instead the application of "default assumptions," more guesswork, as to whether data are incomplete *enough*. As well, the EPA states that "where reproductive and developmental data have been found acceptable by EPA, and the data do not indicate potential pre- or post-natal effects of concern, the additional tenfold margin of safety will not be applied."[105]

This approach is apparently consistent with recommendations made in 1996 by the FIFRA Scientific Advisory Panel. However, it could well ignore the potential for adverse effects of endocrine disrupting chemicals that may be hazardous to fetuses, infants, children and adolescents at particularly low levels of exposure during periods described as "windows of vulnerability" (see discussion in Chapter 2, Section 2.3). Assessing the latter using animal testing is rarely required during standard risk assessment procedures.

The result of this exercise of deciding, in the face of incomplete information, how much of a safety factor to apply, depends on how much information the risk assessors are addressing in the first place. An important criticism came from a group of environmental, children's health and public interest organizations writing to the EPA in 1999 concerning the EPA's evaluation of organophosphates, pesticides that are known to adversely affect brain development in young animals through multiple pathways.[106] They noted the recommendations of the 1993 NRC report and the EPA's own Toxicology Work Group that developmental neurotoxicity testing (DNT) should be a "core" or required test for all risk assessments on organophsophates. The EPA only requires neurotoxicity tests on adult animals despite having had a validated Developmental Neurotoxicity Test Guideline (OPPTS 870-6300) since 1991. By not requiring DNT, the result in terms of the application, qualified application, or decision not to apply the 10-fold safety margin for children is clear. If the risk assessors do not look for effects, they will not find them and will therefore conclude that the safety factor is unnecessary.

A recent example of EPA choosing to apply only a 3-fold safety factor to comply with the FQPA child-safety requirement is the preliminary risk assessment for the pesticide chlorpyrifos, one of the organophosphates and widely used in over 800 pesticide products. This risk assessment is discussed further with respect to "Implications for Canada" in section 4.4.3.4 below.

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[104] USEPA, FQPA Implementation Plan, *op.cit.*

[105] *Ibid.* p.13.

[106] Letter to Carol Browner, Administrator, United States Environmental Protection Agency from: Learning Disabilities Association of America, Consumers Union; Natural Resources Defense Council; Science and Environmental Health Network; Physicians for Social Responsibility; and U.S. Public Interest Research Group, Re: Developmental Neurotoxicity Testing Data Gaps and the Children's 10X Safety Factor, May 12, 1999.

4.4.3.2 Human Testing of Pesticides

Perhaps the most unexpected and certainly the most perverse result of the new 10-fold safety margin intended to protect children, is the recently renewed[107] and increasing practice, by pesticide companies, to seek and often pay human "volunteers" to test for pesticide NOAEL's. The intention of this renewed testing is to eliminate one of the core 10-fold uncertainty factors by providing data on a NOAEL derived directly from experiments on humans. The result, if these studies are accepted, could well be to *increase* pesticide tolerances directly as a result of the passage of the supposedly child-safety-focused FQPA. Said another way, the FQPA requirement for an additional safety factor has unintentionally created an incentive to test pesticides in humans.[108]

At least twelve unpublished studies conducted by pesticide companies have been submitted to EPA and more are expected. They are systemic toxicity studies to establish a human NOAEL. Most were conducted in England and Scotland, often seeking volunteers from among company employees or offering to pay "volunteers" from the public at large.

The pesticides most commonly being studied in the human experiments are organophosphates and carbamates, the two categories of pesticides that have been the subject of the most heated debate in the U.S. during their reevaluation. The FQPA requirement to address the riskiest first as well as to first aggregate exposure from, and then assess chemicals with, common mechanisms of toxicity led to an initial focus on these two groups of pesticides. Since the 10-fold safety margin would be very likely applied to chemicals known or strongly suspected to negatively affect developing nervous systems, human testing offers a potential way out of lower tolerance levels.[109]

The human testing of pesticides has arisen within a policy vacuum at the EPA. It has been greeted with disgust and outrage and is opposed on moral, ethical and scientific grounds by the public interest, farmworker, religious, environmental, consumer, health and medical communities in the United States.[110] A 1998 report[111] and subsequent evaluations by the above-noted groups have charged that the practice is scientifically dubious and ethically indefensible. When the story hit the headlines in 1998, the EPA responded with concern and referred the matter to its independent Science Advisory Board for advice.

The referral to the Science Advisory Board (SAB) has resulted in a deeply controversial investigation. A Joint Science Advisory Board-Science Advisory Panel (SAB/SAP) Subcommittee on Data from Human

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[107] *Ibid.*; the 1972 study cited therein is: Coulston, F., L. Golberg, and T. Griffin, 1972. Safety Evaluation of DOWCO 179 in Human Volunteers, Institute of Experimental Pathology and Toxicology, Albany Medical College, Albany, New York. MRID No. 95175. HED Doc No. 000179, 03822, 04363.

[108] Staff Background Paper for November 30, 1999 Meeting of SAB/SAP Joint Subcommittee on Data from Human Subjects. Available at: www.epa.gov/oscpmont/sap/1999/november/background-1130.pdf

[109] Indeed, the process of revising down tolerances for these chemicals according to an additional 10-fold safety factor might well conclude that many ought to be banned to ensure avoidance of children's health effects.

[110] See multiple letters to Carol Browner, Administrator, United States Environmental Protection Agency, from the farmworker community (www.cehn.org/cehn/farmltr.html), the religious community (www.cehn.org/cehn/reliltr.html), the consumer and environmental community (www.cehn.org/cehn/consltr.html) and the health and medical community (www.cehn.org/cehn/htletter.html) Re: Human Testing of Pesticides, November 18/19, 1999.

[111] Environmental Working Group, *The English Patients: Human Experiments and Pesticide Policy*, July, 1998. Available at: www.ewg.org

Subjects is advising on policy to ensure that EPA can rely on data meeting the highest ethical and scientific standards.

The standard approach to toxicity testing at EPA has been the use of its authority to specify what tests are required and how they should be performed (via the guidelines discussed above). EPA has never developed guidelines for testing pesticide effects or establishing NOAELs in humans nor have such tests been considered necessary, or to be encouraged.[112] Pesticide companies and their farming supporters argue that human tests are more appropriate and reliable in making accurate estimates of human health risk during a risk assessment exercise.[113] In seeking the SAB/SAP committee's advice, EPA wants a policy that applies the protection of the Common Rule (see below) to this new area of inquiry but that also recognizes the wide range of human research that already exists in less controversial circumstances. EPA notes that general standards of conduct will apply to all research but specific standards of conduct and acceptability are necessary in this new area of research.

The SAB/SAP Committee has not been able to agree on this contentious issue. The rift in the Committee has delayed the setting of a policy by EPA. At issue has been debate over whether this testing, as science, is dubious or ethical. John McCarthy of the American Crop Protection Association states that testing pesticides on humans is no different from testing the toxicity of new drugs. Bioethicists disagree pointing out that pesticides are not therapeutic agents.[114]

The comparison to clinical drug trials is important because it takes this issue directly and appropriately into the field of medical ethics. The history of abuse within medical experimentation is a horrific tale. It runs the gamut from the appalling practices of systematic torture and total control over "patients" by the German Nazi doctors through to the Tuskegee syphilis study[115] and the seminal work of Henry Beecher.[116] The history of abuse has provided detailed understanding of the conditions necessary for ethically justifiable research. The Nuremburg Code was the first attempt to enshrine ethical conduct in medical research; the most recent expression of policy for the protection of human subjects in research is The Common Rule.[117]

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[112] Staff Background Paper for November 30, 1999 Meeting of SAB/SAP Joint Subcommittee on Data from Human Subjects. Available at: Available at: www.epa.gov/oscpmont/sap/1999/november/background-1130.pdf

[113] Stroshane, T. 1999, *op.cit.*

[114] Ibid.; Staff Background Paper, *op.cit.*; and Joint SAB and SAP Open Meeting, November 30, 1999. Data from Testing on Human Subjects Subcommittee, Baskerville Transcription, Vienna, VA. Available at: www.epa.gov/oscpmont/sap/1999/november/jointsab.sap.pdf

[115] The Tuskegee syphilis study was one of the most condemned experiments in US medical history. Although a cure for syphilis was found during the course of this multi-year investigation, the hundreds of poor southern black men infected with the disease and involved in the study were not given treatment so that researchers could learn more about the disease by seeing the study through to its fatal conclusion.

[116] Henry Beecher wrote the seminal work in medical ethics in the 1960s after conducting an exhaustive review of unethical conduct in medical research. He drew distinctions between the most heinous examples of the Nazi doctors and the Tuskegee syphilis study but also found extensive unethical conduct within medical studies reported in the peer-reviewed medical literature.

[117] The Federal Policy for Human Subjects Protections (The Common Rule): From the *Final Report, National Committee on Human Radiation Experiments, 1995*, (as reproduced in Environmental Working Group, 1998, *op.cit.*), sets out the responsibilities and obligations of those conducting research on human subjects to ensure protection of their subjects rights and well-being and to ensure the application of informed consent requirements.

Two mandatory components of such rules have included the notion of informed consent and a responsible investigator. While it is beyond the scope of this report to explore the details of these two components, several important points are relevant. First, there is a crucially important set of conditions to be met for the ethically justified medical research on humans. There are issues of scientific adequacy, therapeutic value, protection of subjects and informed, comprehending and voluntary consent. As a two-way transaction, informed consent is a matter of shared decision-making. Hence, informed consent is considered possible only between adults, not between an adult and a child. The irony here is brutal since the experiments conducted by pesticides companies on humans are being done for the purpose of avoiding safety factors intended to protect children.

A key aspect of the notion of a responsible investigator revolves around the central ethical problem of medical experimentation. Beecher's work addressing the continuum of abuse noted above found that while the Nazis had a systemic and racist contempt for their subjects, the less horrendous forms of abuse in medical research stem from a conflict of goals of the physician-researchers. Beecher found that the central ethical problem of medical experimentation concerned balancing the interests of individual subjects with the goals of both helping future patients and advancing careers. Clearly, a central issue to consider in the applicability of The Common Rule to toxicity testing of pesticides on human subjects is the vested interests of pesticide company investigators. Research results that would enable sustained or increased pesticide sales are comparable to the conditions upon which ethical conduct rules have had to be established within the sphere of medical research.[118] A related issue in these studies is whether the alleviation of poverty motivated the human subjects to participate.

TheSAB/SAP Subcommittee on Data from Human Subjects met in December of 1999 to re-consider these issues after failing to agree on policy proposals at an earlier meeting in August. No policy has yet been proposed by the committee or EPA. Meanwhile, EPA states that it expects to receive more results from pesticides companies conducting human testing for pesticide toxicity. As unpublished reports, the nature of informed consent within these studies is not the subject of independent peer review. Nor is there any independent review on the extent to which The Common Rule is being applied.

4.4.3.3 Aggregate Exposure and Common Mechanisms of Toxicity

In the absence of data and exposure models (and guidelines governing their collection/application) to measure aggregate exposure to pesticides, the EPA came up with the notion of "the risk cup" as an interim approach to assessing aggregate exposure. The logic is based on the concept that the total level of acceptable risk from all sources is contained or aggregated in the Reference Dose (RfD). For a chronic health hazard like cancer, the RfD is the daily exposure level over a seventy year period (an average lifetime) that does not, according to the risk assessment calculations, create appreciable risk. For aggregate exposures, the risk cup is filled up with an assessment of the combined exposure estimates from multiple sources including all dietary and non-dietary sources and drinking water, but excluding occupational exposures. Calculations and default assumptions are applied for areas of exposure where data are incomplete. So long as the RfD has not reached 100%, EPA can consider registering additional uses of the pesticide in question or setting new tolerances.

As with exposure assessment for individual pesticides, the degree of uncertainty and gaps in data are enormous for these "risk cup" calculations. This interim approach continues to be applied as debate

[118] For two reviews of these issues see: Roy, D.J., J.R. Williams and B.M. Dickens, *Bioethics in Canada,* Chapter 13: When Treatments are Uncertain: The Ethics of Research with Human Beings, Prentice-Hall, 1994; and Pence, G.E., *Classic Cases in Medical Ethics*, Chapter 9: The Tuskegee Syphilis Study, McGraw-Hill Inc. 1990.

surrounds two proposed guidelines concerning the assessment of aggregate exposure.[119] In both proposed guidelines, critics charged that the EPA's risk assessment methodology refinements focus almost exclusively on ways to reduce exposure estimates despite repeated criticisms pointing to evidence that such exposure estimates should be raised.[120]

In particular, the well documented problem of organophosphates posing an unacceptable risk to children at current levels of exposure is cited by environmental group critics of these efforts at refining risk assessment techniques. Action is justified now, they say, without waiting for further complex risk assessment refinements. EPA has been repeatedly criticized as delaying such action to reduce organophosphate exposure. Instead, environmental groups and their hired experts have carefully documented the apparent one-way effort to refine exposure assessments by focusing almost exclusively on measures that will tend to reduce the final risk estimates. This effort within EPA appears to be in response to industry criticism that actual or "real" use of these pesticides is not informing risk calculations. The critics counter that the exposure averaging techniques being proposed also ignore real world circumstances. The actual circumstances of exposure, they charge, serve to increase exposure in a manner that EPA is factoring out. Further, critics charge that EPA is:

> "...removing more and more of the default assumptions that were built into previous risk assessments, and which presumably were intended to ensure that risk assessments erred on the side of overstating rather than understating true pesticide toxicity and exposures. In many cases, these health-protective defaults are being replaced by new assumptions with no validation using real world data that can assure the risk estimate will not understate the true risk."[121]

The groups are concerned that, due to pressure from registrants, these older, default assumptions are being replaced with new assumptions that claim to better approximate true exposure or toxicity. However, EPA readily admits that the data gaps remain enormous. Environmental groups charge that these proposed "refinements" in calculating and assessing exposure are a systematic attempt to eliminate the more "conservative" default assumptions with less conservative assumptions. They also note that the historically more conservative assumptions were claimed by EPA as being more than adequate to protect all Americans from pesticide risks. This claim is clearly not the case as evidenced by the NRC's 1993 report, numerous other authoritative reports, and the decision by Congress to pass the FQPA.

The reevaluation of organophosphates is equally controversial in the area of assessing common mechanisms of toxicity. The proposed guideline[122] is extremely rough and posted to the internet for

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[119] United States Environmental Protection Agency, Office of Pesticide Programs, *Draft - Guidance forPerforming Aggregate Exposure and Risk Assessments.* February 1, 1999. Available at: http://www.epa.gov/fedrgstr/EPA-PEST/1999/November/Day-10/6043.pdf (note that this version is dated November, 1999 and is revised from the February 1, 1999 draft reviewed for this report and by the environmental organizations noted below); and United States Environmental Protection Agency, Office of Pesticide Programs, *Draft – Choosing a Percentile of Acute Dietary Exposure as a Threshold of Regulatory Concern.* Final document posted to 65 *Federal Register,* 15330 –15333, March 22, 2000. Likewise, the research for this report reviewed the April 7, 1999 draft of this document.

[120] Environmental Working Group, Comments on the Office of Pesticide Programs Proposed Science Policy for "Choosing a Percentile of Acute Dietary Exposure as a Threshold of Regulatory Concern," June 7, 1999; and Natural Resources Defence Council, Comments in response to Public Docket #OPP-00591, Data for Refining Anticipated Residue Estimates Used in Dietary Risk Assessments for Organophosphate Pesticides, June 9, 1999. Both (and several related documents) available at www.ecologic-ipm.com/whatsnew.html

[121] Natural Resources Defense Council, June 9, 1999, *op.cit.* Section III.

[122] United States Environmental Protection Agency, Scientific Advisory Panel (SAP), *Preliminary Draft - Proposed Guidance on Cumulative Risk Assessment of Pesticide Chemicals That Have a Common Mechanism of*

discussion purposes and not for citation or quoting. Although it is slightly unfair to comment on it in this context, it is worth noting that it is clearly on the same continuum. Since it has been the subject of consultation, analyses and responses to it have been prepared by the same environmental groups that have been part of thorough-going critiques of this entire process of refining risk assessment techniques. The most notable impression of this document is, like its companions discussed above, an obvious pre-occupation throughout with exclusionary criteria and/or techniques for narrowing the scope of what is cumulatively assessed.

4.4.3.4 Implications for Canada

The Canadian Pest Management Regulatory Agency (PMRA) has stated that its re-evaluation of pesticides[123] used in Canada will make use of the U.S. EPA pesticide reviews "to the extent possible" and it will "implement approaches (increased safety factors for sensitive populations, aggregate exposure and cumulative risk assessment) taken by the EPA for tolerance reassessment under the FQPA *where necessary and appropriate* (emphasis added).[124] There is no legislative requirement in Canada to include the new approaches contained in the FQPA. However, additional arrangements to harmonize approaches and standards have been established (as described in Chapter 3) and these flow from commitments made under international trade agreements to move towards harmonization of pesticide standards between the two countries. The PMRA also made clear its commitment to risk assessment in the January, 2000 "draft guide to risk assessment and risk management in the PMRA." The draft states: "there is a broad international consensus among regulatory agencies that the acceptability of a chemical should be predicated on the degree of risk rather than simply the hazard (or inherent toxicity) of a chemical." [125] While this rather self-serving view may serve to maintain the *status quo,* it is not particularly accurate, at least not in terms of trends discussed in Section 4.5 below, or when the views of commentators outside of "regulatory agencies" are taken into account. Unfortunately such a view serves to pre-empt the progress embodied in various international agreements to which Canada is a signatory (also discussed in Section 4.5 below).

There are benefits and pitfalls from hitching the Canadian wagon to the FQPA star. On the one hand or perhaps on the surface, the FQPA includes some important and progressive advances over previous U.S. approaches and the existing Canadian regulation of pesticides. However, the reality of FQPA implementation is another matter. The decision to put total faith in the "sound science" of risk assessment underlies much of the problems that have arisen and the concerns that remain. Within the bewildering array of documents that have been drafted, and as yet rarely finalized, to guide the risk assessment process in the application of new requirements flowing from the FQPA, the central limitations of risk assessment are not overcome. Rather, the problems of gaps in data and methodologies for both exposure assessment and dose-response assessment are magnified most particularly in the area of aggregating exposure and assessing cumulative effects. Nor has the additional, supposedly child-focused and much heralded, 10-fold safety factor been applied at key steps along the risk assessment path. Rather, it has

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Toxicity, August 29, 1999. Available at www.epa.gov/oscpmont/sap.1999/september/cumdoc.pdf and not for citation or quotation.

[123] The long overdue task of pesticide re-evaluation by the PMRA needs to address 73% of pesticide active ingredients registered for use in Canada including potentially hundreds if not thousands of pesticide products and formulants (see Chapter 9, the Pesticides Case Study).

[124] Pest Management Regulatory Agency, *Re-evaluation Document: Re-evaluation of Organophosphate Pesticides.* REV99-01, June 29, 1999, pp. 1, 4.

[125] Pest Management Regulatory Agency. *Risk Assessment and Risk Management in the Pest Management Regulatory Agency (Draft).* (Jan 17, 2000), p.6.

been treated as an add-on at the end of the traditional process where often it has been either circumscribed or omitted altogether. Finally, the unexpected renewal and increasing practice by pesticide companies testing their products on human "volunteers" to avoid and perhaps weaken pesticide regulation is a perverse and sad result of a law intended to increase regulation for the sake of protecting children from known risks.

A recent example illustrates many of these points. Dursban or chlorpyrifos is one of the most widely used of the organophosphate pesticides and has been the subject of a re-registration application in the U.S. during 1999. The Reregistration Eligibility Decision (RED) Document[126] for chlorpyrifos clearly reveals an exposure problem for U.S. children. However, without a final decision as to methodology, an aggregate exposure assessment was not conducted and the risk assessment is not informed by aggregate exposure calculations despite the fact that this chemical is the active ingredient in over 800 commonly used products. Nor has the risk assessment been informed by as-yet unknown or uncalculated cumulative risks of this pesticide with others having a common mechanism of toxicity. Despite the above gaps in information the risk assessors chose to apply only a 3-fold additional safety factor within the FQPA requirement. The situation could be worse since chlorpyrifos was the subject of one of the first of the human dosing experiments to determine a human NOAEL.[127] However, as part of this re-registration review process and likely due to a great deal of public pressure, and the child-specific health risks of this chemical, the EPA decided to eliminate the results of the human testing results of chlorpyrifos from its re-registration decision on this chemical.[128]

The Canadian PMRA apparently continues to rely on this 1972 human study[129] as the basis for its Tolerable Daily Intake (TDI) for chlorpyrifos. Notably, in light of the EPA re-registration review, twelve prominent scientists have recently called for tight restrictions on agricultural use of chlorpyrifos and for a full ban on all applications in the residential setting, schools or childcare facilities because of concerns about neurological effects in children.[130]

The expensive and time-consuming nature of a chemical-by-chemical approach to regulation embodied in risk assessment is only slightly improved in the FQPA requirement to assess the "worst first" and to assess groups of chemicals with common toxic effects. The outcome so far has been much the same as in past however with only a handful of chemicals having been assessed and only the most egregious

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[126] United States Environmental Protection Agency Office of Prevention, Pesticides and Toxic Substances, internal memorandum and attached reports re: Chlorpyrifos: Health Effects Division Preliminary Risk Assessment for the Reregistration Eligibility Decision (RED) Document. October 18, 1999. 66 p. Available at: www.epa.gov/oppsrrd1/op/chlorpyrifos/hedassessment.pdf

[127] Coulston, F. *et.al.*, 1972, *op.cit.*

[128] United States Environmental Protection Agency, Office of Pesticide Programs, Memorandum regarding *Chlorpyrifos – Replacement of Human Study Used in Risk Assessments*, Report of the Hazard Identification Assessment Review Committee, June 2, 1999. Available at: http://www.epa.gov/oppsrrd1/op/chlorpyrifos.htm

[129] D.L. Grant and Associates, Ltd., *Chlorpyrifos: Review of the basis of the human health assessments conducted by PMRA and identification of uncertainties in the assessments.* Report prepared for Pollution Probe, Toronto, Ontario. December 13, 1999.

[130] Of these twelve scientists, two are former EPA officials. Dr. Philip Landrigan, director of the Center for Children's Health and the Environment at Mount Sinai School of Medicine in New York, was a senior advisor on children's health to EPA in 1997 and 1998. Dr. Lynn Goldman, adjunct professor at Johns Hopkins University School of Hygiene and Public Health, was assistant administrator for the EPA's Office of Prevention, Pesticides and Toxic Substances from 1993 to January of 1999. See: Environment News Service, Americscan: April 13, 2000 at www.ens.lycos.com/ens/apr2000/2000L-04-13-09.html

examples having been the subject of use restrictions. For example, in August of 1999, EPA took regulatory action to reduce residue limits for two organophosphates, methyl parathion and azinphos methyl.[131] However, now-illegal residues remain on the bulk of the apple crop sold throughout the winter of 1999-2000 according to recent test results by the Environmental Working Group. They found unsafe residues of methyl parathion in 2 of 25 bags tested and residues of aziphos-methyl in 14 of 25 bags tested. The tests also revealed residues of endosulfan, recommended for a ban by the Washington State Department of Ecology.[132] Despite having stated that Canada will follow the FQPA lead, no regulatory action on azinphos methyl use in Canada has been announced (methyl parathion is not registered for use in Canada) although a report is apparently in preparation.[133] Nor has any Canadian action been taken on restricting the use of chlorpyrifos, or Dusrban. In contrast, recent media reports indicate that the U.S. EPA will be announcing use restrictions for this pesticide to reduce exposure to children.

Actual reductions in pesticide use and exposure under the FQPA have been limited by the insistence on the "sound science" of risk assessment. Implicit in this approach is the standard of proof demanded by scientific inquiry. The lack of data and methodologies to provide this standard of proof undermine the notion that risk assessment is "sound science" or that it can deliver child-protective standards quickly or perhaps at all. Rather, the science necessary to deliver protective standards tends to demand that exposure to pesticides reaches measurable (according to rigorous scientific standards) and often excessive levels and that health impacts of such exposures have been both detected and verified by defensible scientific inquiry. The situation is reminiscent of the early days of the cautionary tale of lead, documented in Case Study #1, when scientifically defensible proof of both exposure and harm was uncertain but deeply troubling; comparable to the situation with many pesticides in common use today. The regulatory reforms provided by the FQPA offer very limited and mostly inadequate solutions to this problem.

4.5 THE PRECAUTIONARY PRINCIPLE

4.5.1 Introduction

Sections 4.2, 4.3 and 4.4 above describe the nature, scope and the limits of risk assessment. These sections provide considerable commentary on the scientific limits, (such as attempting to generalize animal studies to human health ignoring background sources, ignoring multiple chemical exposure, among many others), the gaps and deficiencies in data and methodologies and those limits pertaining to epidemiological and causation issues.

Two responses have emerged to respond to these criticisms of risk assessment. Predominantly, as discussed in Sections 4.2 and 4.4 above, the response has been to find risk assessment basically sound and in need of ever more complex refinement. To a certain extent, this refinement has included incorporation of the weight-of-evidence approach discussed in Section 4.3.6.

The other response is to provide a new "overlay" on risk assessment that instills in effect a new approach. This new approach incorporates the precautionary principle.

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[131] EPA Press Release, *EPA Acts to Reduce Children's Exposure to Two Older, Widely Used Pesticides*, August, 2, 1999; available at: www.ecologic-ipm/epapr080299.html).

[132] See: Environmental Working Group, *A Few Bad Apples... Pesticides in Your Produce; Why Supermarkets should "Test and Tell,"* March, 2000. Available at: www.ewg.org/pub/home/reports/fewbadapples/foreword.html

[133] Personal communication with Adrian Carter, Pest Management Regulatory Agency, May, 2000.

Simply put, the precautionary principle provides a policy framework to make decisions to protect human health and the environment in the face of scientific uncertainty. As summarized by one commentator, the principle has a "dual trigger," namely, "If there is a potential for harm from an activity and if there is uncertainty about the magnitude of impacts or causality, then anticipatory action should be taken to avoid harm."[134]

Although simply put, the definition of the principle, the legal basis, the scope of its application, its core elements and how to implement it, have been but a few issues raised by the concept and which are now being debated both within the context of international law and domestic legislation.

One of the key issues raised by the precautionary principle is how the principle relates to risk assessment. While the precautionary approach is not usually viewed as an alternative to risk assessment, it is at times regarded as a threat to the "sound science" and the rigour that is supposedly inherent within risk assessment. This section provides some context on the precautionary principle in terms of its origins, definition and application. It also builds and relies upon the discussions of "weight of evidence", "burden of proof" and "precautionary inference" contained in the review of "The Science Behind the Assessment" in Section 4.2 above. The components of the precautionary approach are reviewed again here in terms of their relevance to children's health. The present status of the precautionary principle in Canada is then discussed.

4.5.2 Evolution of Principle

Although the articulation of the precautionary principle as a distinct, recognizable principle is fairly recent in origin, the key motivation and thinking behind the term is anything but new. Its basis is rooted in public health thinking of attempting to anticipate harm in the face of uncertainty,[135] or colloquially put, "an ounce of prevention is worth a pound of cure." The legal and policy footing of the term can be traced through its recognition in a number of international treaties and conventions commencing in the early 1980s, followed by some modest efforts to incorporate the concept domestically.

4.5.2.1 Precautionary Principle and International Law

One of the on-going debates is whether the precautionary principle has emerged into a customary rule or norm of international law.[136] A customary norm or rule creates an obligation upon all states to follow the rule. While most seem to agree that the principle is an international custom, it is unclear whether the principle has evolved into a binding rule. Evidence or indicators that the principle has emerged as an international custom is derived from the fact that a large number of treaties and conventions incorporate the principle, it is found in state practice to some extent, and it exists within commentary from the international court of justice, among other indicators. The list of international treaties, conventions, agreements and statements where the concept is mentioned is impressive, including the *Protocol on*

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[134] Raffensperger, C. and J. Tickner (eds.), *Protecting Public Health and the Environment: Implementing the Precautionary Principle* (Washington, D.C.: Island Press, 1999), Introduction, p. 1.

[135] *Ibid.*, pp. 4-7.

[136] For a more detailed discussion on this topic, see: Castrilli, J.F., *The Precautionary Principle and Canadian Environmental Law: From Principle to Practice*. A Report Prepared for Pollution Probe, 1999, p. 6; and McIntyre O. and T. Mosedale, The Precautionary Principle as a Norm of Customary International Law. *Journal of International Law*, 9(1997) p.221.

Substances that Deplete the Ozone Layer (1987); *Bamako Convention on Hazardous Wastes within Africa* (1991); the *Rio Declaration on Environment and Development* (1992); and the *Framework Convention on Climate Change*, (1992), to name but a few.[137] Moreover, many earlier international agreements have been interpreted as applying or implicitly recognizing the precautionary principle. The clearest example perhaps is the discussion by the International Joint Commission (and one of its advisory bodies, the Science Advisory Board) in its interpretation of the *Great Lakes Water Quality Agreement.*[138]

Certainly most treaties and conventions in the past decade have incorporated the precautionary principle in some fashion.[139] This trend is continuing. In January of 2000, the principle was incorporated into the *Cartagena Protocol on Biosafety to the Convention on Biological Diversity.*[140] At the 4th International Negotiating Conference for the proposed Legally Binding Treaty on Persistent Organic Pollutants held in Bonn, Germany in March of 2000, the use of the precautionary principle received, and is anticipated to continue to receive, extensive debate as to how to implement the principle within that regime.

4.5.2.2 Approaches in Other Countries

The Maastricht Treaty, which formed the European Union, commits the environmental policy of the community to the precautionary principle. A number of countries, including the Netherlands, U.K, and Sweden have been studying how to implement the principle while Hungary and Brazil have already adopted it.[141] In February of 2000, the European Commission issued a Communiqué formally recognizing the principle and providing guidance as to how to further it.[142]

According to one commentator, the United States has yet to adopt the precautionary principle as an "explicit basis" for environmental decision-making. However, there is an emerging debate on the topic, especially since the U.S. did sign the *Rio Declaration on Environment and Development* (which obliges countries to adopt the principle) and there is some articulation of the concept in a few national environmental laws. As well, the principle is recognized in the 1996 statement of guiding principles for sustainable development by the U.S. President's Council on Sustainable Development.[143] However, the concept of pollution prevention, a component of the precautionary principle (described in the next

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[137] For a review of treaties and conventions that have incorporated the precautionary principle see: Raffensperger and Tickner, 1999, *op.cit.*, Appendix B, Uses of the Precautionary Principle in International Treaties and Agreements in U.S. Legislation. This is a summary review with more extensive treatment in: Hickey, J. and V. Walter, Refining the Precautionary Principle in International Environmental Law, *Virginia Environmental Law Journal*, 14(1995) pp. 423 –436, as cited in Appendix B of Raffensperger and Tickner, 1999, *op.cit.*

[138] See: International Joint Commission, *Sixth Biennial Report on Great Lakes Water Quality* (Ottawa/Washington, 1992); International Joint Commission, *Seventh Biennial Report on Great Lakes Water Quality* (Ottawa/Washington, 1994); International Joint Commission, *1993-95 Priorities and Progress Under the Great Lakes Water Quality Agreement* (IJC, 1995).

[139] *Ibid.*

[140] The Protocol can be found at: www.biodiv.org/biosafe/BIOSAFETY-PROTOCOL.htm. For commentary on this issue, see: Swenarchuk, M., The Cartagena Biosafety Protocol: Opportunities and Limitations. Canadian Environmental Law Association, February, 2000. Available at: www.web.net/cela/Trad&Env/biosafe.htm

[141] Tickner, J., *Precautionary Principle: Current Status and Implementation.* Lowell Center for Sustainable Protection, March, 2000, p. 1.

[142] *Ibid.*

[143] *Ibid.*, pp. 1 and 2.

section), is a part of the policy mainstream in the U.S. Approximately 25 states now have toxic use or pollution prevention laws now in place, although they vary greatly in scope and effect.

The Canadian position with respect to the precautionary principle is discussed below.

4.5.3 What is the Precautionary Principle?

While a strong argument can be made that the precautionary principle is becoming enshrined in both international law, and perhaps less so, national law, there are enormous debates that remain as to both the definition and the implication of the principle.

4.5.3.1 Definitions

Clearly, there is no consensus on how to define the "precautionary principle." The definition is important is since it either expands or constrains the scope of the concept. A comparison of two definitions illustrates the point. The *Rio Declaration on Environment and Development*[144] states the definition as follows:

> In order to protect the environment, the precautionary approach shall be widely applied by states according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation.

While this definition speaks to "serious or irreversible damage" and "cost-effective" measures, other definitions do not have such qualifications. For example, the *Wingspread Statement on the Precautionary Principle*[145] states:

> When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause-and-effect relationships are not fully established scientifically.

As noted , even though Canada seems to be tending to accept the Rio Definition, certainly the concept will continue to evolve as the policy debate matures. Indeed, one can expect a continuous rigorous debate on the definition for a number of reasons. Foremost, the definition will determine the scope of application of the concept. Second, the definitions that have evolved also implicitly or explicitly refer to a number of elements or components that seek to implement the principle. These components are discussed in the next section.

4.5.3.2 Components of the Precautionary Approach and their Relevance to Children's Health

As the debate as to the definition of the principle continues, the literature on the topic is rich in outlining the key components or policy implication emanating from the principle. While no attempt will be made to outline the full range of possible components or policy implications, those most germane to the present discussion pertain to: onus of proof; the weight of evidence approach; prevention-based tools and

[144] June 14, *International Legal Material* 31(1992) p.849.

[145] The Wingspread Statement is reproduced in: Raffensperger and Tickner, *op.cit.*, Appendix A.

standards; and public participation.[146] These components are discussed generally, but some effort is also made to relate these components to implications for risk assessment referred to in section 4.2 and 4.3, as well as some commentary on the implications for children's health.

Burden/ Onus of Proof

One of the common noted elements of the precautionary principle is that, where there is a threat of harm, the onus should be on those threatening such harm to establish the activity will not cause harm to the environment or human health. As discussed in detail in Section 4.3.7, standard setting is primarily a policy-making exercise and decisions on policy entail a review of the science, together with many other judgements. While there is increasing agreement and application by regulatory agencies of the need to apply a "weight of evidence" approach in standard setting, disagreement remains concerning the "burden of proof." In particular, the questions (discussed in more detail in Section 4.3.7) include: what "burden of proof" should be demanded; where should the "burden of proof" be placed; and what elements of "proof" should be considered in making standard setting decisions.

If environmental standards are to protect human health and welfare, ecosystems and other very high values, the "burden of proof" that will be protective of those desired values cannot rely strictly upon the statistical significance testing that is applied to epidemiological studies. To base standard setting decisions on scientifically derived inferences of causation *before* establishing protective measures or refusing to allow additional exposures will result in potentially hazardous exposure to contaminants. The Lead Case Study provides the cautionary tale of the perils of this rigid approach.

The legal concepts of duty of care based on a "balance of probabilities" or "50% plus one" likelihood standard are valid here and can reasonably be applied. Standard setting approaches that would truly weigh all of the available evidence and arrives at a prudent protective judgement based on all of that "weight of the evidence" would be the most likely to create standards that are protective of children's health.

Again, as is explored in more detail in Section 4.3.7 above, "precautionary inference" is a preferred method for making scientific judgements when data are incomplete or inconclusive, and where significant harm may follow from a false negative judgement, i.e., in matters typical of environmental contamination and health damage. The risk assessment approach of contaminants largely being considered "innocent until proven guilty" is reversed and the burden of proof is on demonstrating lack of harm. Standards would be set at rigorous levels of safety and not lowered unless and until the ever-present uncertainty is resolved to demonstrate on "clear, strong and cogent evidence" that at the permitted exposure level, no harm to children will result.

Weight of Evidence Approach

Although, as noted above, regulatory agencies are increasingly applying a weight-of-evidence approach, another question implicit within the precautionary principle is the determination of how much evidence is required that harm may occur before precautionary action will be taken. Is it necessary that there will be absolute proof of harm or only a mere suspicion?

One commentator summarized the preferred approach this way:

> Decision –making about associations or likelihood of harm under the Precautionary Principle

[146] The criteria identified are derived from a review of the literature, and in particular, see: Castrilli, J.,F., 1999, *op.cit.*, pp.11-13; and Raffensperger and Tickner, 1999, *op.cit.*, A Map Toward Precautionary Decision Making, pp. 166-177. Other components identified include use of the "polluter pays" principle, evaluating alternative activities, technologies and chemicals, ongoing monitoring, investigation and information dissemination, strong enforcement, among others.

> should be based on a "weight-of-evidence" approach, rather than on some quantitative probability of harm (as is the case with risk assessment approaches). The weight-of-evidence approach to decision-making takes into account the cumulative weight of information from numerous sources that address the question of injury or the likelihood of injury to living organisms."[footnotes omitted][147]

As discussed in detail in Section 4.3.6 above, a wide array of evidence is at issue when identifying potential human health hazards, especially when appropriate human data are lacking and inferences have to be made about the degree of proof that is provided by existing toxicological data.

Prevention-Based Tools and Standards

Another element of the precautionary principle calls for the use of prevention-based tools and standards aimed at avoiding or preventing harm from some activity. In other words, rather than focusing on the proof of harm, a focus would be on designing products and activities such that the threat of harm would be avoided. Examples of such measures in this context would include recognition of inherent toxicity as the basis for phasing out of dangerous substances, the establishing of pollution prevention standards, the development and encouragement of clean technologies; methodologies to promote alternatives, to name but a few.[148] It is important that principles of Just Transition be applied so that workers affected by the phase-down and phase-out of toxic chemicals are able to at the very least obtain alternative training and employment. Ideally, the expertise of these affected workers can assist with the process of workplace transition and transformation.

Public Participation

One of the implementing mechanism for the precautionary principle relates to greater public participation in environmental decision-making. This mechanism is important since the implementation of the principle requires "the need to balance value judgements before decision-makers when health and environmental risks of activities are being evaluated."[149]

4.5.4 Precautionary Approach in Canada

Canada's initiatives to embrace the precautionary approach has been described as "hesitant hugs." Although Canada has perhaps accepted the approach in principle in various legislative enactments, it has yet to provide any specific roadmap with respect to its practical implementation.[150]

Federally, both the *Oceans Act* and the new *Canadian Environmental Protection Act* formally commit to the precautionary approach. In the *Oceans Act*, the precautionary principle is recognized in the preamble and is required to be considered when the Minister of Fisheries and Oceans develops a national oceans management strategy.[151]

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[147] Tickner, J., A Map Toward Precautionary Decision Making, Raffensperger and Tickner, *op.cit.*, p. 169.

[148] Castrilli, J.,F., 1999, *op.cit.*, pp. 11; and Raffensperger and Tickner, 1999, *op.cit.* p.171.

[149] Castrilli, J.,F., 1999, *op.cit.*, pp. 13; and Raffensperger and Tickner, 1999, *op.cit.* p.175-6.

[150] VanderZwaag, D., The Precautionary Principle in Environmental Law and Policy: Elusive Rhetoric and Embraces , *Journal of Environmental Law and Practice* 8(355)(1999), p. 369. See also: Moffet, J., Legislative Options for Implementing the Precautionary Principle *Journal of Environmental Law and Policy* 7(1997), p. 157.

[151] *Oceans Act*, S.C. 1996, c. 31, Preamble and section 30.

In the *Canadian Environmental Protection Act* (CEPA), which was recently revamped and passed into law in 1999,[152] as with the *Oceans Act*, the precautionary principle is mentioned in the preamble. It also outlined those administered duties committed to by government, namely:

> In the administration of this Act, the Government of Canada shall .. exercise its powers in a manner that protects the environment and human health, applies the precautionary principle that, where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation, and promotes and reinforces pollution prevention approaches..."[153]

Hence, while the Act embraces in a general way the precautionary principle, using the language from the Rio Declaration, the general view is that the Act falls short of implementing the principle in a meaningful way.[154] Some of the highlights of the law in this regard (as well as some of the limits), include:

- *Virtual Elimination*: Part 5 of CEPA contains a number of provision that recognize the inherent toxicity of substances. For those substances designated to be persistent, bioaccumulative and toxic, the goal is "virtual elimination." Despite this goal, the law is drafted is such a way that there is no actual requirement to achieve virtual elimination, but instead requires the setting of interim targets taking into account social, economic and technical matters.

- *Weight of Evidence*: When the government is conducting and interpreting the results of toxicity assessments pursuant to the Act, the Ministers of the Environment and Health "shall apply the weight of evidence approach and the precautionary principle."[155]

- *Pollution prevention plans*: Under Part 4 of CEPA, the government is empowered to require pollution prevention plans for those substances found to be toxic under the Act and placed on the Toxic Substances List. These provisions, however, are completely discretionary on the part of the Minister of the Environment and these powers are only for substances found to be toxic under the Act..

Because the Act is only to be proclaimed in the spring of 2000, the interpretation and application of these provisions remains to be seen.

The federal and provincial governments have also recognized the precautionary principle, at least in principle, in the *Canada-Wide Accord on Environmental Harmonization* which was concluded on January 29, 1998. The intent of the Accord is to avoid overlap and duplication between federal and provincial governments in areas of shared jurisdiction. However, the Accord has been seriously criticized by non-governmental groups as a mechanism to devolve federal environmental roles and responsibilties to the provinces.[156] It is too early to tell whether this devolution of responsibility will mean that Canada's

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[152] *Canadian Environmental Protection Act*, 1st Sess. 36th Parl. 1997-98-99.

[153] *Ibid.*, section 2(1)(a).

[154] VanderZwaag, D., 1999, *op.cit.* p. 371.

[155] *Canadian Environmental Protection Act*, 1st Sess. 36th Parl. 1997-98-99, section 76.1.

[156] For example, see: Canadian Environmental Law Association and the Canadian Institute for Environmental Law and Policy, *Brief to House of Commons Standing Committee on Environment and Sustainable Development Regarding the Canadian Council of Ministers of the Environment (CCME) Environmental "Harmonization" Initiative*, CELA Brief No. 332; CIELAP Brief No. 97/4 (October 1997).

international commitments to the Precautionary Principle will be implemented. However, if the progress so far on Canada-Wide Standards is any indication, the prospects are bleak (see Chapter 5 for further discussion).

In addition to acknowledging the need for pollution prevention, the Accord adopts the Rio Declaration of the precautionary principle as it states under a section entitled "Principles":

> Where there are threats of serious or irreversible environmental damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation (precautionary principle);

There is no mention in the Accord of implementation measures for this principle ensuring that, so far at least, the commitments remain primarily rhetorical.

According to one commentator,[157] only two provinces, namely Nova Scotia and New Brunswick, have legislation that acknowledges the precautionary principle.[158] Neither of these statutes though provide implementing mechanisms.

4.5.5 Summary

The precautionary principle is now recognized as a legitimate public policy principle, although it has yet to be fully implemented. Its acceptance as an international law norm suggests that it is just a matter of time before domestic law will more routinely and effectively reflect this principle.

While the precautionary principle will probably not replace risk assessment, it will certainly promote risk assessment in a different light. Rather than being the core of standard setting exercises, risk assessment would be just another tool along side other important tools. The components of the precautionary principle, if implemented, would profoundly recast how environmental standard setting takes place. These components are essentially direct responses to the limits of risk assessment identified in sections 4.2 and 4.3 above. The onus of proof, weight of evidence and pollution prevention are a number of the key elements that ensure that real progress can be made towards more protective standards. It should be noted however, that these elements are anything but radical. By and large, some of these components are slowly drifting into the lexicon of decision-makers while other are already firmly entrenched (such as pollution prevention) although there is an enormously long way to go yet.

While Canada has made some modest gains in furthering the precautionary approach through recognizing it in such statutes as the *Canadian Environmental Protection Act* and the *Oceans Act*, its implementation is anything but certain.

4.6 CONCLUSIONS

This Chapter provides a foundation for answering the question as to whether environmental standard setting, via the predominant approaches of risk assessment and risk management, is or can be, *intentionally* protective of children. It is clear from this historical review that risk assessment approaches

[157] VanderZwaag, D. 1999, *op.cit.*, p. 372.

[158] See: *Environment Act*. Statutes of Nova Scotia 1994-95, c. 1, s. 2(b)(ii); *Clean Air Act*, Statutes of New Brunswick 1997, C-5.2, 2(b).

to standard setting have evolved over time and continue to do so. These changes have attempted to resolve gaps in data and methodology including better accounting for children's health effects. However, the ever-increasing complexity of risk assessment methodologies has been matched and consistently overcome by the greater complexity of the problems they attempt to address, including accounting for the special exposure circumstances and vulnerabilities of children.

Advances in risk assessment guidance and methodologies continue to be undermined by central problems that have been with risk assessment from the start. Even when risk assessment approaches have been modified to specifically account for children's health, as is being attempted under the *Food Quality Protection Act* in the United States, the outcome in terms of actual reduction in risk has so far been minimal as well as highly acrimonious, controversial and slow. Moreover, the application of additional child-specific safety factors has been mainly an exercise in minimal afterthought rather than application of precautionary measures at each step where uncertainty exists throughout the process. Methodologies to overcome key barriers (aggregate exposure, cumulative effects) are barely developed, controversial, and have yet to be employed to any significant extent.

These problems frequently stem from the incorrect assertion that risk assessment is an objective science-based activity. Although risk assessment is routinely characterized as the "scientific" stage of the exercise (while risk management is considered the policy-making step), two of the four key steps in risk assessment suffer from large gaps in data and methodology providing many opportunities for uncertainty, variability and error. When gaps have to be filled with "science policy options," or informed guesswork, the risk assessment exercise can no longer claim to be objective and scientific. This Chapter's review of the "science behind the assessment" explores the many reasons for the high degree of difficulty and scientific uncertainty in drawing inferences of causation in environmental health matters. A key issue for standard setting is a mis-application of the standard of proof demanded by scientific inquiry. While there are important reasons for maintaining this standard, not the least of which is ensuring the integrity of scientific inquiry, problems arise when the scientific standard of proof is applied to the only-partially scientific process of setting standards to limit exposure to contaminants.

The insistence on risk assessment to provide objective science-based standards has resulted in a demanding, time and resource intensive chemical-by-chemical approach. With so many chemicals to assess, so many gaps and uncertainties in data and a lack of methodologies to both assess exposure and health effects, it is distinctly unfair and illogical to insist on scientific standards of proof (of exposure and harm) before taking preventative action. Such an approach is doomed to failure in terms of being truly protective of children. Given these fundamental constraints, it is debatable whether individual techniques can be added to make risk assessment *intentionally* protective of children. Although standard setting agencies can and do increasingly apply a weight-of-evidence approach, the political forces brought to bear on the risk management side of the exercise can be formidable and can serve to remove any safety margins or precautionary influence on the final choices as to standards.

The assigning of individual risk levels for each chemical is also a game of odds that cannot address two of the most serious issues of toxic chemical pollution: inherent toxicity and population-wide effects such as may be occurring with endocrine disrupting chemicals. Risk assessment enables risk calculations that allow for "acceptable" levels of one-in-a-million or one-in-ten-thousand risks (of cancer, birth defects, etc.) across a population. However, the odds game becomes useless if further research confirms the suspicion that chemicals such as endocrine disruptors are capable of exerting population-wide effects. Nor is it appropriate to make such calculations for chemicals that are persistent and bioaccumulative. Risks will continue to increase for chemicals that do not break down and which accumulate in animal fat, breast milk, etc. These risks will of course be highest for children and other vulnerable populations than for the adult population at large.

Important issues of ethics and equity arise during risk assessment and risk management. Within the domain of specialized experts and those wealthy enough to hire them, the combination of science and guesswork provides numerous opportunities for value judgements and bias to enter risk calculations. Again, the chemical by chemical supposedly scientific process is a central part of the problem. Each chemical is treated as "innocent until proven guilty." By applying the high standard of proof demanded within scientific inquiry, chemicals essentially have greater rights than the human population. Assessed one at a time, in isolation from other chemicals, risk levels are assigned to new chemicals regardless of risk levels that already exist or that are yet to be calculated for new chemicals. Such assessments also underestimate risk since they rarely account for all relevant health effects or for the cumulative or synergistic effects of chemicals acting in combination. As more and more chemicals continue to have the right to be assigned a risk level (alongside the many thousands of chemicals that have never been adequately assessed), the human population does not have the same right to be exposed to no more than a specified level of risk.

Many implications arise when applying judgement and non-scientific values to the process of weighing a body of evidence and setting policy or standards for exposure to contaminants. Key among them is the choice made as to the "burden of proof" demanded. Because standard setting is intended to protect human health and welfare, ecosystems and other very high values, the "burden of proof" that is required in standard setting should be one that is more likely to be protective of those desired values. However, standard setting rarely applies such a protective approach. Instead, protective standards generally are not set until rigorous scientific inquiry has been applied to the available (and always incomplete) information in order to verify proof of harm. The result is delay in setting protective standards and the greater likelihood of too much exposure before protective action is taken.

A more appropriate standard of proof would incorporate the legal concepts of duty of care, based on a "balance of probabilities" or "50% plus one" likelihood standard. Standard setting policy decisions should follow a paradigm in which it is *at least* "more likely than not" that standards have been set that will be protective of children's health. Where data are incomplete or inconclusive, the approach of "precautionary inference" is a more prudent and appropriate means of making scientific judgements particularly since significant harm may flow from incorrectly assuming that no harm is possible from the environmental contamination being regulated. This approach reverses the current scientific and policy framework, recognizes the inherent shortcomings of information and methodologies, and would set protective standards first. Such standards would be made less stringent only when the uncertainty as to the toxicity of the chemical hazard is resolved via "clear, strong and cogent evidence" that, at the permitted exposure level, no harm to children will result. Such a "reverse onus" approach would place the scientific burden of proof on those wishing to create environmental contamination while regulatory agencies could apply precautionary inference to the setting of protective standards.

In contrast to risk assessment, the precautionary principle provides a policy framework to make decisions to protect human health and the environment in the face of scientific uncertainty. While the precautionary approach is not usually viewed as an alternative to risk assessment, it is at times regarded as a threat to the "sound science" and the rigour that is supposedly inherent within risk assessment. The components of the precautionary principle, if implemented, would profoundly recast how environmental standard setting takes place. These components are essentially direct responses to the limits of risk assessment. The onus of proof, weight of evidence and pollution prevention are a number of the key elements that ensure that real progress can be made towards more protective standards.

4.7 RECOMMENDATIONS

4.7.1 Risk Assessment

1. The use of "comparative risk assessment" and "cost-benefit analysis" in environmental standard setting should be monitored and evaluated for effectiveness in environmental and health protection versus their narrower ability and purpose of cutting costs.

2. All regulatory agencies in the federal and provincial government need to explicitly acknowledge the scientific uncertainties and limitations of risk assessment for deriving environmental standards.

3. The harmonization (either NAFTA-imposed or as cost-saving measures) of Canadian pesticide standards with those being developed in the U.S. should be undertaken as a preliminary step towards, or at least should not undermine Canada's ability to move towards, more precautionary standards. Such standards should include more rigorous and stepwise application of child-protective safety factors during both exposure assessment and dose-response assessment, as well as assessments of aggregate exposure, cumulative and synergistic effects, and the ability to implement a full ban on persistent organic pollutants. Child-protective safety factors and a weight-of-evidence approach should continue through to the risk management stage of setting new or revised standards for pesticides and all environmental pollutants.

4. Further research is necessary regarding whether and how commitments made under international trade agreements constrain Canada's ability to set protective standards. In addition, given the influence on Canada of standard setting in the United States, further research is required to determine the degree to which final standards established in the United States are set at numbers influenced by the possibility of legal challenge, including on a constitutional basis, so as to be able to recognize when a resulting standard is weaker than it should be within the Canadian legal context.

5. The Canadian Pest Management Regulatory Agency, as part of a government-wide approach, should immediately implement a policy of refusing to accept from pesticide companies new or existing toxicity test data derived from experiments on human "volunteers."

4.7.2 Precautionary Principle

6. Although the federal government has committed to the precautionary principle in the *Canadian Environmental Protection Act*, the *Oceans Act*, and in other policy pronouncements, there is little evidence that the principle has been operationalized. It is therefore recommended that the federal government develop a national implementation strategy to further the precautionary principle that includes:

   (a) Change in the burden of proof: a process that ensures that those parties creating a threat of harm, such as those that produce a new substance that is being assessed or that introduce new products, have the onus to establish that such substances or products are safe, rather than having government establish that they pose a risk of harm;

   (b) Weight of evidence: a protocol that allows decisions at each step in a risk based decision-making process (i.e., during all stages of both risk assessment and risk management) to be based on the weight of evidence approach rather than waiting for an extremely high standard of proof;

(c) Pollution Prevention: a commitment to operationalize pollution prevention through the development of a regime for bans and phase-outs of inherently toxic substances as well as pollution prevention standards for industrial sectors;

(d) Just Transition: a commitment to ensure the application of the principles of Just Transition for workers affected by toxic substance phase-down and phase-out;

(e) Public Participation: in recognition of the political and ethical implications of environmental and risk-based decisions, a commitment to make these decision-making regimes more transparent and open to the involvement of the public.

7. At this time, there is little evidence in provincial law or policy that Ontario is committed to the precautionary principle. It is recommended that the province of Ontario development a regulatory commitment to the precautionary principle together with a strategy to operationalize the principle similar to that described in Recommendation 5 above.

8. Both Ontario and Canada should adopt a definition of the precautionary principle that is more expansive than the definition found in the *Rio Declaration,* and preferably one similar to that found in the *Wingspread Statement on the Precautionary Principle*, which states:

> When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause-and-effect relationships are not fully established scientifically.

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Chapter 5: Air

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5.1 INTRODUCTION

This section considers the regulation of *outdoor* air contaminants,[1] an area that includes the involvement of both the federal and provincial governments. The provinces establish standards to regulate air contaminants at levels to prevent harm to human and ecosystem health, to prevent discomfort and loss of enjoyment of property, and to prevent damage to the physical environment. The federal government's role in the control of air pollution is to undertake research, enter into treaties, control transboundary air pollution and set standards to protect public health and safety.[2] This chapter provides a relatively detailed up date as to the status of Ontario provincial and federal air standard setting as of April, 2000. The legislative and regulatory framework is outlined, along with a brief description of the provincial approvals process for air approvals. The provincial standard setting plan is described in some detail; other aspects of provincial air regulation are briefly mentioned. On the federal side, the current Canada Wide Standards setting process is described in terms of current proposals; other aspects of federal and federal-provincial air standard setting are also mentioned more briefly. Attempts have been made to provide up to date references as of the date of this study so that those interested in further review regarding air standards can begin from the status as of April, 2000. However, it should also be mentioned that both federally and provincially, for particular air contaminants, there are many specific in depth consultation processes, with an array of participants or stakeholders, and so for a review of a particular contaminant, as opposed to an overall view which this chapter provides, the documentation available for the particular stakeholder process should be consulted.

5.2 PROVINCIAL REGULATION

Ontario's *Environmental Protection Act (EPA)*[3] is the principal statute governing air quality in the province. It establishes a general prohibition against discharging contaminants into the natural environment in excess of the amounts permitted by the regulations. "Contaminant" is defined to include a substance that causes an "adverse effect." "Adverse effect" is defined to include, among other things, "harm or material discomfort to any person;" "an adverse effect on the health of any person" and "impairment of the safety of any person." Because of the regulations, emissions may be permitted in accordance with a Certificate of Approval issued by the Ministry of the Environment (MOE). Specific provisions are provided for Certificates of Approval for all stationary sources that emit, or have the potential to emit outdoor air contamination.[4] Certificates of Approval are legally-binding licences that set out the conditions under which a facility can operate, including maximum permissible contaminant emission levels. The Ministry of the Environment has established standards and guidelines that inform the setting of these emission limits, as described below.

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[1] For information regarding indoor air pollutants and their effects on children, see Pollution Probe and The Canadian Institution of Child Health, *The Air Children Breathe: The Effects on Their Health.* Conference Proceedings, (January 19/20, 1998).

[2] Estrin, D. and Swaigan J. *Environment on Trial: A Guide to Ontario Environmental Law and Policy.* Third Edition, (1993).

[3] *Environmental Protection Act,* R.S.O. (1990), E. 19.

[4] *Ibid.*, EPA, s. 9. The Act and Regulations exempt a number of sources from this requirement. See also R.R.O. (1990), Reg. 346, s. 3.

5.2.1 *Ambient Air Quality Criteria*

Ambient Air Quality Criteria (AAQC)[5] are established under the *EPA* and limit total atmospheric contaminant levels. The Criteria are established for different time periods and set the maximum average contaminant concentration that is permissible during a particular time period. Hence, a one hour AAQ Criterion for a contaminant would limit the average atmospheric quantity of the contaminant that is present during a one-hour period at a particular point or receptor. AAQC are based on either human health or environmental effects, whichever is the most sensitive, and are normally set at a level that is not expected to cause adverse effects to a sensitive receptor, based on continuous exposure. Consequently, socio-economic factors including costs and technological feasibility are not considered in the setting of an AAQC. If odour or irritant effects are experienced at levels below health effects, then the AAQC are established based on that more sensitive impact. The Criteria are not themselves standards, but they may become indirectly enforceable by way of being included in a particular Certificate of Approval issued to a particular applicant for a specific facility or mobile source. Where relevant, they are used to guide the setting of individual Certificate of Approval limits.[6] Where National Ambient Air Quality Objectives (see below) exist, they inform the setting of AAQC.[7] The Canada Wide Standards process under CCME has largely usurped development of additional National Ambient Air Quality Objectives (NAAQO), in the sense that the federal government is devoting its resources to the CWS process although the authority to enact NAAQO is still in place. The Ontario Ministry of the Environment expects that in the future as new standards are developed (see description of this process later in this chapter), all of the criteria will be adopted as standards and there will no longer be air "guidelines" in use. This approach will be more consistent with current practice and more consistent with enforceability requirements.[8]

5.2.2 *Point of Impingement Standards*

Regulation 346[9] under the *EPA* sets out Point of Impingement (POI) standards for non-vehicular contaminant sources. These legally-binding standards limit the contaminant content of the emissions that are produced by individual facilities. A point of impingement is the location at which a contaminant first makes contact with a sensitive receptor following emission. The receptor may be human, animal or plant. For any given emission source, there exist multiple points of impingement, as the contaminant reaches different receptors (people, plants, wildlife) that are situated at different distances and in different directions from the emission source.[10] Schedule 1 to Regulation 346 establishes Point of Impingement limits for a number of contaminants. These standards are maximum average contaminant concentrations that are permitted over a half hour period at the Point of Impingement. They may not be exceeded unless

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[5] *Ambint Air Quality Criteria Regulation,* R.R.O. (1990), Reg. 337.

[6] Ontario Ministry of the Environment. *Backgrounder on the Development and Implementation of Air Quality Standards*, (not dated) [hereinafter *MOE Backgrounder*].

[7] Personal Communication, J. Smith, D. Harper, A. Socha, Standards Development Branch, Ontario Ministry of the Environment, (March 30, 1999).

[8] Personal Communication, S. Fleming, A. Socha, Standards Development Branch, Ontario Ministry of the Environment, (March 16, 2000).

[9] *General Air Pollution Regulation*, R.R.O. (1990), Reg. 346.

[10] Personal Communication, Doug Harper, Manager of Human Toxicology and Air Standards Section, Ontario Ministry of the Environment, (July 29, 1999).

an emission source is explicitly exempted by regulation.[11]

Regulation 346 sets out, in its Appendix, formulae to calculate the concentration of a contaminant at the different possible points of impingement, depending on variables including source concentration and a range of relevant environmental conditions, such as weather. In order to determine if it is in compliance with POI standards, an industry calculates its POI concentrations using these formulae, for the range of points of impingement that are relevant to its situation. The industry then compares the *highest* POI contaminant concentration calculated with the Regulation 346 standard.[12]

Once an Ambient Air Quality Criterion is developed for a particular contaminant, it is used by the MOE to set a Point of Impingement standard, via a series of established mathematical relationships. For example, a 24 hour AAQ Criterion is multiplied by 3 in order to derive a 24 hour POI limit. Similarly, an annual AAQ Criterion is multiplied by a factor of 15 to determine the annual POI limit. According to the MOE, these relationships between AAQC and POI standards are well developed. The limitation of this method, however, is that it fails to consider background contaminant levels. In other words, it works well for an individual facility, but does not take into account the emissions produced by other facilities. It does not guarantee, therefore, that if the POI limits derived in this manner were met by all contaminant sources, that the AAQ Criterion for total atmospheric contaminant levels would also be satisfied. According to the MOE, this is only a concern for a few contaminants such as nitrous oxides and particulate matter, where background levels are significant. For other contaminants, background levels are apparently minimal.[13] However, it should be noted that among the contaminants of greatest concern for respiratory impacts on children are nitrous oxide and particulates.

Because POI standards apply to existing sources, some socio-economic issues are sometimes considered by the MOE in their development. The MOE considers whether the POI standards are "technically feasible" and whether the "costs" of implementation are balanced by their "benefits."[14] However, new sources may be required to be built to the more recent POI standards where applicable.

5.2.3 Point of Impingement Guidelines

The provincial Ministry also makes use of Impingement Guidelines. Like POI standards, they are used to review Certificate of Approval applications and to approve new and modified emission sources. However, in contrast to POI standards, they do not automatically apply to emission sources and are only legally-binding when incorporated into a Certificate of Approval. While POI standards are developed for substances that are identified as being of relatively greater risk to human health and the environment, based on release quantities, the number of sources, and the potential for exposure at levels that may cause adverse effects, guidelines apply to substances that are released from relatively few sources and which the Ministry has determined are best managed on a case-by-case basis. POI guidelines are generally set to avoid adverse human health and environmental effects and accordingly, are not informed by socio-economic factors.[15] As indicated above, the MOE expects to move toward use of standards only as the

[11] *General Air Pollution Regulation*, R.R.O. (1990), Reg. 346, s. 5.

[12] Personal Communication, Doug Harper, Manager of Human Toxicology and Air Standards Section, Ontario Ministry of the Environment, (July 29, 1999).

[13] Personal Communication, Doug Harper, Manager of Human Toxicology and Air Standards Section, Ontario Ministry of the Environment, (July 29, 1999).

[14] *MOE Backgrounder,* undated, *op.cit.*

[15] *MOE Backgrounder*, undated, *op.cit.*

newer standards are developed.[16]

5.2.4 Ministry of the Environment Three Year Plan for Standard Setting

During the 1980s, air standard setting was undertaken by a committee comprised of representatives from the Ministries of Labour and Environment. When this group disbanded in the late 1980s, standard setting and revision was left without a formalized process or overseeing committee. Consequently, these activities were undertaken as they were perceived to be needed. In 1994, for example, a revised lead standard was developed.[17] In the mid-1990s, the Provincial Auditor criticized the MOE for its lack of a formal standard setting process and in response, the Ministry developed its Three Year Plan for Standard Setting.[18] The 1996 Plan[19] recognized that many of Ontario's air standards, some of which were established two decades previously, may not be adequately protective. The Ministry also acknowledged that the science of risk and exposure assessment had changed significantly. Accordingly, the Ministry undertook an assessment of its air standards in order to establish priorities for review. Following comparison to the standards in other jurisdictions, Ontario standards for 75 substances were deemed to be sufficiently stringent for the time being. These substances were set aside while higher priority substances are reviewed.[20] A decision by the MOE to accept these 75 standards was posted to the EBR on February 21, 2000.[21]

In its identification of priorities for review, the MOE's Standards Development Branch considered toxicity, whether the substance is present in the environment and to what extent, quantities of release, the number of sources and the potential for exposure to a contaminant.[22] Branch documentation states that it also considers new information related to environmental/human health effects, the need to meet MOE commitments to federal/provincial standard-setting working groups, information from tools such as the National Pollutant Release Inventory, and needs that have been identified in support of Ministry programs.[23]

Based on these criteria, over 70 substances were identified as priorities for evaluation and were included

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[16] Personal Communication March 16, 2000, *supra* note 8.

[17] See Section 8.4.3 of Case Study #1.

[18] Personal Communication, Doug Harper, Manager of Human Toxicology and Air Standards Section, Ontario Ministry of the Environment, (April 16, 1999).

[19] Previously available at http://www.ene.gov.on.ca/envision/standards/index.html

[20] Ministry of the Environment, Standards Development Branch, October, 1999, *Reviewing Ontario's Air Standards.* The jurisdictions to which each of the 75 standards was found comparable and a brief rationale statement for retaining the standard can be found in this document at Table 1 and Appendix A. (hereinafter, Reviewing, 1999).

[21] EBR posting EBR Registry Number PA9E0004, titled *Setting Environmental Quality Standards in Ontario: the Ministry of Environment's Standards Plan;* pdf file located at: http://www.ene.gov.on.ca/envision/env_reg/er/documents/2000/pa9e0004.htm. Written submissions permitted between November 5, 1999 and January 4, 2000; Decision posted Feb. 21, 2000. Based on the document, *Reviewing Ontario's Air Standards*, October 1999, Standards Development Branch, Ministry of the Environment (hereinafter, Standards Plan, 2000).

[22] Standards Plan, 2000, *supra* note 21, section 3.2

[23] Fax received from D. Harper, Manager of Human Toxicology and Air Standards Section, Ontario Ministry of the Environment, (June 1, 1999).

in the Ministry's Plan. In January, 1997, stakeholder consultation began on proposed air quality standards for an initial set of 14 of these substances.[24] These consultations were undertaken to solicit science and technology related information regarding the proposed standards and to seek feedback regarding timely and equitable implementation. In March, 1998, standards for 9 of these substances were posted to the *Environmental Bill of Rights*[25] Registry and the decisions for those 9 were posted in December 1998. The remaining 5 substances[26] were to undergo more comprehensive assessment. Initial standards were proposed for these substances in 1998; as of April , 2000, they are still undergoing revision and preparation of information drafts, and establishment of a risk management process.[27]

In January, 1999, information regarding an additional 18 substances[28] for review was posted on the Registry.[29] This group of 18 has been posted on the Registry with proposed Ambient Air Quality Criteria (AAQC) and Point of Impingement (POI) Standards, along with detailed rationale documents for each of the eighteen chemicals.[30] For fifteen of these eighteen standards, the AAQC is proposed to be more stringent; for two of these standards, (methyl isobutyl ketone and toluene), the AAQC is proposed to remain the same; while for one of these eighteen standards, (isopropyl benzene), the proposed AAQC is to be made less stringent. For those proposed to be more stringent, the factors range from 1.5 times more stringent to 500 times more stringent, with the upper end of this range being for the air carcinogens (acrylonitrile, chloroform and propylene oxide). However, this type of comparison is crude at best and the proposals should be reviewed specifically for further information.[31] For these eighteen proposed air standards, the POI half-hour standard is proposed to be more stringent for 13 of them and to remain the same for five of them. However, all of them are proposed to be standards rather than guidelines. An interim standard is proposed as a range, from the proposed standard, up to a specified amount, for nine of them; for the other nine, the ultimate proposed standard does not require an interim range.[32]

There will be a further group of 15 substances for which the review process will begin shortly, but as of April, 2000, these have not yet been posted to the Registry.[33] They are expected to be posted to the EBR later in the year 2000.[34]

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[24] 1,4 dichlorobenzene, acetaldehyde, arsenic, cadmium, carbon tetrachloride, chromium VI, cyclohexane, dichloroethane, formaldehyde, methylene chloride, nickel, styrene, tetrachloroethylene, trichloroethylene. Supra, footnote 21, Table 1(a).

[25] *Environmental Bill of Rights*, R.S.O. (1993), c. 28.

[26] cyclohexane, cadmium, chromium VI, nickel and arsenic.

[27] Standards Plan, 2000, *supra* note 21, table 1(a).

[28] acetonitrile, acrylonitrile, ammonia, chlorine, chloroform, ethyl ether, ethylbenzene, hydrogen chloride, isopropylbenzene, vinylidene chloride, methanol, methyl ethyl ketone, methyl isobutyl ketone, mineral spirits, n-hexane, propylene oxide, toluene, xylenes.

[29] *MOE Backgrounder*, undated, *op.cit.*

[30] Ministry of Environment, Consultation on 18 Ontario Air Standards: Information Summary, http://www.ene.gov.on.ca/envision/env_reg/er/registry.html (hereinafter, Consultation on 18)

[31] Consultation on 18, *supra* note 30, Table I.

[32] Consultation on 18, *supra* note 30, Table I.

[33] Personal Communication, J. Smith, D. Harper, A. Socha, Standards Development Branch, Ontario Ministry of the Environment, (March 30, 1999); See also Standards Plan, 2000 *supra*, note 21.

[34] This next group of fifteen (actually to be sixteen with the addition of uranium), include: acetone, acetonitrile, acrolein, acrylamide, cyclohexane, Di(2-ethylhexyl)phthalate, Di-n-octylphthlalate, Hexamethylene diisocyanate monomer and buiret, Hydrogen cyanide, Hydrogen fluoride, Methane di-phenyl diisocyanate,

In addition to the 70 substances identified as priorities for standards revision, there also remains the possibility of additional standards development or revision in response to new information. For example, the uranium standard is to be released shortly because of concerns arising in Port Hope, Ontario. Other reasons for additional standards to be developed or revised include new information from other sources such as IARC (International Agency for Research on Cancer) carcinogens, EPA IRIS (Environmental Protection Agency (U.S.) Integrated Risk Information System), World Health Organization, and the National Toxics Program.

Following the above groups of proposals, thirty-three of the 70 air priorities identified in the Standards Setting Plan will remain for air standards development. Of these, eight are being developed under either the Canada Wide Standards process or under a Federal-Provincial Working Group under CEPA (benzene, mercury, NOX, ozone, particulate matter, total reduced sulphur, sulphur dioxide, chlorinated di-benzo-p-dioxins (CDD's and furans). Further comment as to some of these latter is found below in the review of the federal role.[35]

Those POI standards that are revised through this process automatically apply to all emission sources, regardless of whether a facility holds a Certificate of Approval authorizing the application of the former emission standard. In contrast, POI guidelines do not automatically apply to emission sources, but rather, are applied when a source seeks to renew its Certificate of Approval.[36] Accordingly, as POI guidelines are replaced by standards, they will apply to all emission sources.

5.2.5 The Standard Setting Process

The MOE's stated policy is to set its standards to be protective of the most sensitive receptor. When available information indicates that children are the most sensitive receptor, as was the case with lead and is the case with a forthcoming uranium standard, the Ministry reports that it will base its standard on the protection of children.[37]

To assess the risk posed by a particular contaminant, the Ministry relies on risk assessments developed by other regulatory agencies such as Environment Canada, Health Canada, the U.S. Environmental Protection Agency (EPA), individual state EPAs and the World Health Organization (WHO).[38] The MOE acknowledges that "there is considerable variability among regulatory agencies on the types and number of ambient air quality standards, guidelines and exposure limits." Reasons can include different endpoints or types of effects, different averaging times and different methodologies. Differences also include considerable differences in the way standards are developed; some jurisdictions basing results almost exclusively on results of detailed risk assessments; others extrapolating from workplace exposure limits.[39] Ministry staff advise that their review of these other jurisdictions' risk assessments is thoroughly

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Methyl isocyanate, phenol, toluene diisocyanate, uranium and vinyl chloride. *supra* note 30, page 6.

[35] Consultation on 18, *supra* note 30, Table II.

[36] Personal Communication, Doug Harper, Manager of Human Toxicology and Air Standards Section, Ontario Ministry of the Environment, (July 29, 1999).

[37] Personal Communication, J. Smith, D. Harper, A. Socha, Standards Development Branch, Ontario Ministry of the Environment, (March 30, 1999).

[38] *MOE Backgrounder,* undated, *op.cit.*

[39] Reviewing, (1999), *supra* note 20.

documented, including their assessments as to whether the judgments made by that jurisdiction at various steps in the hazard assessment process are consistent with the judgments that would be made in Ontario's risk assessment process.[40] Ministry staff also advise that their preference, where possible, is the work of U.S. EPA or California, at least as to the risk assessment aspect of the process (i.e. not necessarily the risk management approach).[41]

The Ministry inventories contaminant sources and ambient contaminant levels in order to estimate exposure levels in the province.[42] The MOE states that it considers multi-media exposure.[43] While exposure is generally assessed based on adult body weight and breathing rate, the MOE asserts that its standards are protective of children based on the use of very large uncertainty factors.[44]

To set a standard, the MOE turns once again to the standards developed in the jurisdictions listed above. It may also develop a standard independently, which is usually based on existing scientific information. The Ministry develops a proposed *Rationale Document for the Development of Ontario Air Standards* which sets out the range of standards that the Ministry is considering for adoption.[45] The Ministry solicits information and commentary from stakeholders on this draft by organizing information sessions and posting the draft to the *Environmental Bill of Rights* Registry.[46]

According to the MOE, it then considers information received from the public and undertakes a preliminary risk analysis for the proposed standards. Where no substantive implementation issues have been identified by stakeholders, or where implementation can be achieved via ongoing or planned initiatives, the Ministry proposes an Ambient Air Quality Criterion, a Point of Impingement Guideline or Standard that is based on the Ambient Air Quality Criterion, and sets out an effective implementation date. The new standards are posted on the *Environmental Bill of Rights* Registry.

When Ministry assessments and stakeholder input indicate that a number of sources will be out of compliance with the range of standards being considered and will incur undue financial hardship or other economic consequences to be in compliance, or where more scientific information needs to be considered in the risk assessment, the Ministry states that it undertakes a more detailed assessment in the form of a *Detailed Risk Management Analysis*. For example, for the 18 standards currently under review, this Analysis has not yet been prepared; it will be undertaken after receipt of the comments from affected parties and the public as solicited on the *Environmental Bill of Rights* registry. The basis upon which risk management decisions will be made has not yet been determined, and a stakeholders advisory

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[40] Personal Communication, (March 16, 2000), *supra* note 8.

[41] For the MOE's description of the risk assessment process followed in some of the leading jurisdictions (U.S. EPA, California, Massachusetts, Michigan, and New York, see Reviewing, (1999), *supra* note 20.

[42] Personal Communication, *supra* note 8.

[43] Ontario Ministry of the Environment. *Workshop on Incorporating Risk Management Considerations in the Development of Ontario Air Standards: Report of the Standards Development Branch, Ontario MOE.* (September 25, 1998) [hereinafter, *MOE Workshop*]. In the October, 1999 Report , *supra* note 20, the description is that the Ontario MoE takes a multi-media protection approach; that is the multi-media aspect of the approach is that the most sensitive receptors may be non-human, i.e. vegetation, wildlife, accumulation in soil, or entering food-chain (at page 2).

[44] Personal Communication, J. Smith, D. Harper, A. Socha, Standards Development Branch, Ontario Ministry of the Environment, (March 30, 1999).

[45] *MOE Backgrounder,* undated, *op.cit.*

[46] *Ibid.*.

consultation to consider the parameters for the risk management stage of the standard setting for this group will be established. Ministry staff advise that they are concerned as to whether affected industry has demonstrated a "compelling" rationale to show that there are "major implementation issues" with the proposed protective standards. However, pending that process, interim standards will be set, which for carcinogens will be within a range corresponding to risk levels of one excess cancer in a population of 1,000,000 (10^{-6}) over a lifetime, up to, but no higher than 1 excess cancer in a population of 10,000 (10^{-4}) over a lifetime.[47] Ten to the minus six life time risk is the target standard; the risk management consultation is primarily to put the onus on the affected industry to show why they cannot meet that standard immediately; and if it is satisfactorily proven that there are some barriers to doing so, then to establish the time frame and conditions to move from the interim standard to the long term standard.[48] This includes an examination of the technical options available to reduce emissions and their socio-economic effects.[49]

In order to assess whether there are such "compelling implementation issues," the consultation document asked industry to provide the following information:

- the operations or processes which give rise to emissions of the substances under review;
- the reduction in emissions that would be required to bring the facility into compliance with the proposed standard;
- changes in equipment, potential additional systems or operations (including pollution prevention measures) necessary to achieve and maintain compliance with the proposed standard, including projected capital and annual operating costs of such changes, timing and any gains in productivity, recovered materials or reduced energy or raw material usage;
- if it is claimed that a standard is not technically achievable, to provide documentation of the conditions or circumstances that confirms this position;
- provide documentation as to the degree of reduction in emission and ground-level concentration that could be achieved in the operation, facility and/or firm;
- the earliest possible time frame for compliance with the standard; and
- whether the firm is involved in developing emission reduction strategies under the federal Strategic Options Process for this substance.[50]

The document also refers industry to other documents for assistance in preparing their submissions, including the *Framework for the Application of Socio-economic Analyses in Setting Environmental Standards*.[51]

The standards plan advises that "In the absence of specific, significant implementation issues the Ministry will proceed to finalize the proposed standards" and that "If stakeholders make no comments about the proposed standards, it will be presumed that they have no concerns or will have no difficulty, technically or financially, in complying with the proposed standard."

The Ministry states that one of the purposes of this analysis is to ensure that the costs and benefits associated with the standard are "balanced," and to verify industry claims such as cost burdens. The

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[47] Consultation on 18, *supra* note 30 at page 3.

[48] Personal Communication, (March 16, 2000), *supra* note 8.

[49] *Ibid.*

[50] For example, see the Toluene rationale document, one of the group of 18, note 57 below (detailed rationale).

[51] Economic Integration Task Group, CCME, (1998). http://www.mbnet.mb.ca/ccme/pfds/SEFrameENG.pdf

analysis is conducted by way of a multi stakeholder working group that includes representation from the major sources of a particular substance. The outcome of this process is to be a proposed Ambient Air Quality Criterion, a Point of Impingement Standard or Guideline and an effective implementation date. The standards are posted to the *Environmental Bill of Rights* Registry for final comment.

The MOE states that when a large number of sources are affected by a revised standard, other options include implementing "technology-based solutions," adopting a philosophy of continuous improvement with short-, medium-, and long-term plans for reducing emissions, setting interim Point of Impingement limits based on considerations of cost and technical feasibility (which are incorporated into Regulation 346 and are therefore binding), or setting a Point of Impingement limit at a level that is deemed "reasonable" in light of the costs imposed and benefits realized.[52] However, the MOE does not have a policy in place to guide the development of alternatives when proposed standards are deemed to be too onerous to industry.[53]

When considering cost-benefit arguments for air standards, the U.S. Environmental Protection Agency report, *The Benefits and Costs of the Clean Air Act Amendments of 1990* should be borne in mind.[54] That study reported that "The economic value of the public health and environmental benefits that Americans obtain from the Clean Air Act Amendments of 1990 exceed their costs by a margin or four to one. Included in the benefits is the prevention of thousands of premature deaths and millions of asthma attacks related to air pollution each year. By the year 2010, the benefits will total about $110 billion (U.S.) compared to the costs of achieving those health and ecological benefits at only about $27 billion.[55] The study was extensively peer reviewed at all stages of its design, research, analysis and report. These figures did not even include many additional benefits not yet quantified, such as the control of cancer-causing air toxics and benefits to crops and ecosystems. Some of the recommendations from this study are endorsed and repeated at the end of this chapter.

Toluene

One of the current proposals in the group of 18 substances on which stakeholder comment is being solicited is toluene. The EBR posting for toluene[56] provides a brief description of the rational for the development of the Ontario Air Quality Standards for Toluene and the detailed supporting document is also available.[57] Similarly, each of the other of the eighteen substances in the current review has a separate posting and rationale document.

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[52] *MOE Workshop, supra* note 43.

[53] Personal Communication, J. Smith, D. Harper, A. Socha, Standards Development Branch, Ontario Ministry of the Environment. (March 30, 1999).

[54] U.S. Environmental Protection Agency, "*The Benefits and Costs of the Clean Air Act Amendments of 1990*," www.epa.gov.oar/sect812

[55] Among the benefits counted by the study, to be achieved by 2010, are the prevention of 23,000 annual premature deaths, aversion of over 1,700,000 annual asthma attacks, prevention of 67,000 incidences of chronic and acute bronchitis, 91,000 occurrences of shortness of breath, 4,100,000 lost work days and 31,000,000 days of restricted activity, 22,0000 respiratory related hospital admissions, 42,000 cardiovascular hospital admissions and 4,800 emergency room visits for asthma -- in the U.S. alone.

[56] EBR Registry Number PA00#0018, Proposal for Policy, comment period from February 21, 2000 to May 21, 2000, *Rationale for the Development of Ontario Air Quality Standards for Toluene.* www.ene.gov.on.ca/envregistry/013213ep.html

[57] www.ene.gov.on.ca/envision/env_reg/er/documents/2000/pa9e0004.html

For the toluene standard, the current proposal is to leave the existing 24 hour Ambient Air Quality Criterion at 2,000 ug/m^3 and the existing half hour Point of Impingement standard at 2,000 ug/m^3. In other words, no change to the existing standard which was originally published in 1970 is proposed. The rationale includes that the health-based risk assessment from Health Canada was considered the most appropriate for developing the standard. The Health Canada Tolerable Concentration is 3,750 ug/m^3, but deriving a 24 hour AAQC from this concentration would result in an increase to the standard. As a result, the MOE is recommending no change to the existing standard.

Propylene Oxide[58]

Another of the current proposal by the MOE for 18 air standards is Propylene Oxide. The AAQC (24 hour) for this contaminant is proposed to be reduced from 4500 micrograms per cubic metre of air (ug/m^3) to 1.5 ug/m^3; the annual average AAQC to be .3 ug/m^3. The current half hour Point of Impingement guideline is 13,500 ug/m^3. The proposal is to change that to a standard, and to reduce the standard to 4.5 ug/m^3. An interim standard is proposed in the range of 4.5 to 450 ug/m^3. It is identified in the consultation document as a carcinogen. Propylene oxide is described as used mostly as an intermediate in the chemical production of urethane foams, other manufactures, sterilization and fumigation. The Rationale document identifies that the United States EPA identifies propylene oxide as a probable human carcinogen. The proposed standard is based on a risk level of one excess cancer risk per 1,000,000 people. The interim standard is proposed to allow for a phase-in period to achieve the new standard, and the range identified is that between a lifetime one in a million excess risk level and one in 10,000 excess cancer risk level. As was described in the toluene example, industry is asked to provide detailed information as to "the technical and economic considerations associated with achieving an interim POI standard" in the proposed range. They are also asked for information regarding the feasibility of achieving the final proposed POI standard.

Ethyl Ether[59]

The proposal for ethyl ether is to reduce the AAQC (24 hour) from 30,000 to 8,000 micrograms per cubic metre of air, and to reduce the POI guideline from 30,000 ug/m^3 to 700 mg/m^3, and as a standard, not a guideline. An interim range from 700 to 7,000 mg/m^3 is proposed. Ethyl ether is described as a solvent for waxes, fats, oils, perfumes, alkaloids and gums, as a reagent in certain chemical reactions, and as used in manufacture of gun powder and as a primer for gasoline engines. It is also used in medical therapeutic use. For this proposed standard, MOE based their AAQC proposal on the health-based Threshold Limit Value from the American Conference of Governmental Industrial Hygienists. The half-hour POI standard is based on odour effect. Again, industry and stakeholder comment as to implementation issues is being sought with respect to finalizing the interim standard. For this posting, the MOE included an appendix as to the agency-specific reviews of air quality guidelines for ethyl ether, including the standard in that jurisdiction, the documentation available, key references, a discussion of the peer review process, a brief discussion of the key risk assessment considerations, in this case, a discussion of threshold effects and non-threshold effects, a discussion of the key risk management considerations for the jurisdiction and a statement as to the utilization of a multimedia approach in the jurisdiction. This type of information is extremely useful and the MOE should be encouraged to continue making its reviews of other

[58] EBR Registry Number PA00E0017; Policy Posting, Written submissions due between February 21, 2000 and May 21, 2000. The detailed rationale document, *Rationale for the Development of Ontario Air Standards for Propylene Oxide*, February 2000, is available at: www.ene.gov.on.ca/envision/env_reg/er/documents/2000/pa9e0004.htm .

[59] EBR Registry Number PA00E0008, Policy Posting, Written Submissions permitted between February 21, 2000 and May 21, 2000. Detailed rationale document, *Rationale for the Development of Ontario Air Standards for Ethyl Ether*, February 2000 is available at: www.ene.gov.on.ca/envision/env_reg/er/documents/2000/pa9e0004.htm

jurisdictions' standards available in this manner.

5.2.5.1 Styrene: An Example

Styrene is one of the original 14 air contaminants identified for evaluation in 1996 under the MOE's Three Year Plan. The existing standard for styrene dates back to 1975 and is odour-based. In its evaluation, the Ministry reviewed the risk assessments and standards of other regulatory agencies, styrene's toxicity and styrene levels in Ontario. Two possible regulatory approaches existed, based on different health end-points. The first concerned neurological effects including fetal neurotoxicity (widely viewed to be among the most sensitive end-points), and injury to the central nervous system and the liver. The second was based on carcinogenicity. The latter was rejected on the basis that, in the opinion of the MOE, there was inadequate evidence in epidemiological and animal studies of a link between styrene exposure and cancer. In contrast, the State of Massachusetts characterizes styrene as a probable human carcinogen and estimates the additional risk of cancer from styrene at one in one hundred thousand with a lifetime exposure of 20 micrograms/cubic metre of air.

The MOE focused its neurotoxicity-based analysis on two sources: Health Canada and the World Health Organization. In 1993, Health Canada developed a Tolerable Daily Intake (TDI) for styrene under the *Canadian Environmental Protection Act*.[60] It recognized fetal neurotoxicity as being among the most sensitive of end-points for styrene. With this in mind, it developed a TDI value of 125 micrograms per cubic metre, based on the LOEL for neurotoxic effects observed in animal studies. The standard was specifically intended to be protective against fetal neurotoxicity. Health Canada then derived a Tolerable Concentration based on this TDI, and the breathing rates and body weights of 5 to 11 year olds. The result was a Tolerable Concentration of 92 micrograms per cubic metre.

The WHO also explicitly recognized, in the development of its 1997 standard for styrene, that neurotoxicity in the form of developmental impairment is among the most sensitive end-points for styrene. However, the WHO based its standard on data regarding the subtle neuro-psychological effects (such as reductions in visuomotor accuracy) observed in human occupational exposure studies. These data were adjusted by a factor of 4.2 to facilitate a conversion from occupational to continuous exposure. An additional safety factor of 10 was employed for inter-individual variation and finally, a further factor of 10 was applied because a LOAEL was adopted in place of a NOAEL. The WHO notes that the resultant level of 450 micrograms per cubic metre *should* be protective of neurological effects as observed in animal species (emphasis added).

In January, 1997, the MOE proposed a new styrene Ambient Air Quality Criterion of 125 micrograms per cubic metre, the value developed by Health Canada. Following stakeholder consultation, it switched to the considerably less stringent WHO standard, stating that this standard provides "a more balanced treatment of both the observations of fetal neurotoxicity in animal species and human exposure data."[61] Among industry stakeholder complaints was the claim that users of styrene in the manufacture of plastic resins and in the fibreglass industry would not be able to meet the Health Canada standard, based on existing knowledge and technology. Consequently, the existing MOE standard of 400 micrograms per cubic metre, which is 50 micrograms more stringent than the WHO standard, was deemed protective of health and remains unchanged.[62]

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[60] *Canadian Environmental Protection Act*, R.S.C., (1985), c. 16 [hereinafter *CEPA*].

[61] Ontario Ministry of the Environment. *Rationale for the Development of Ontario Air Standards for Styrene: Consultation Draft*, (1998) [hereinafter Styrene Rationale].

[62] Styrene Rationale, *supra* note 59.

Since this standard was proposed, the Ministry has issued a revised Standards Plan, as described above, and has revised their process to ask industry specific questions in justification of any claims that they cannot meet the proposed standard. For example, the EBR posting for comment on the current group of 18 standards asks Industry questions as to whether there are "compelling implementation issues" as described above. As well, the risk management approach intended to follow this round of consultation is yet to be developed.

5.2.6 Ozone-Depleting Substances General Regulation

The *EPA Ozone-Depleting Substances General Regulation*[63] was developed to reduce or eliminate the use of ozone-depleting substances in the manufacture of pressurized containers, flexible plastic foams and rigid insulation foams.

5.2.7 Acid Rain Regulations

The acid rain regulations were adopted under a 1985 program called Countdown Acid Rain, in order to impose total annual sulphur dioxide emission limits on the major sources of these contaminants in Ontario.[64] These include Algoma Steel,[65] Inco,[66] Falconbridge[67] and Ontario Hydro.[68]

5.2.8 The Environmental Protection Act Part III: Motors and Motor Vehicles

The provincial government is also involved in the regulation of mobile emission sources. Part III of the *EPA* prohibits the removal from vehicles of systems and devices designed to reduce contaminant discharges.[69] The *Motor Vehicles Regulation*[70] sets out maximum permissible emission levels for operating vehicles, including maximum levels of hydrocarbons, carbon monoxide and visible emissions.

5.2.9 Smog Plan

The Ontario Ministry of the Environment's 1998 Smog Plan is aimed at reducing, by 75%, the number of exceedances of its ozone Ambient Air Quality Criterion. It seeks to accomplish this by reducing total nitrous oxides (NOX) and volatile organic compound (VOC) levels by 45% (1990 background standard).

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[63] *Ozone-Depleting Substances General Regulation,* R.R.O. (1990), Reg. 356.

[64] Estrin and Swaigen. (1993), *op.cit.*

[65] Reg. 663/85.

[66] Reg. 660/85.

[67] Reg. 661/85.

[68] Reg. 662/85. Note that the regulation controlling sulphur dioxide and nitric oxide emissions from the fossil-fueled electric generating stations of Ontario Hydro is now *Ontario Hydro* R.R.O. (1990), Reg. 355.

[69] *Environmental Protection Act*, R.S.O. (1990), E. 19, s. 22.

[70] *Motor Vehicles Regulation,* O.Reg. 361/98.

This reduction is to be accomplished, by the year 2015, through a variety of voluntary initiatives.[71] Additional information regarding ozone standards development is described below, in review of the proposed Canada-Wide Standard for ground level ozone. In addition, Canada is currently involved in negotiation with the United States of an Ozone Annex to the 1991 Canada-U.S. Air Quality Agreement. One round of negotiations was held in February, 2000; the next round is scheduled for June 14-15, 2000 in Washington. The purpose of the negotiation is to address trans-boundary ground level ozone air pollution between Canada and the United States. This is a major issue for Ontario since much of the ozone pollution in Ontario is from U.S. sources. At the same time, Ontario's actions with respect to its own contribution to ozone pollution (which impacts U.S. receptors to the east as well as Ontario, Quebec and eastern Canada) are critical to the negotiations and have been highly contentious. Furthermore, in development of the Canada-Wide Standard for ozone, the current levels in Ontario are standing in the way of development of a standard that is based on the most health protective levels.

5.3 ADEQUACY OF PROVINCIAL AIR QUALITY STANDARDS: INITIAL INFORMATION

In 1998, the Ontario Medical Association (OMA) released *The Health Effects of Ground-Level Ozone, Acid Aerosols and Particulate Matter*,[72] a report examining the health effects and regulation of air pollution in Ontario. The OMA reported that air pollution is a significant contributor to health conditions in Ontario and that as many as 1800 Ontarians die prematurely every year as a result of exposure to air contaminants.[73] The study emphasized that children are at particular risk.

A particular concern identified by the OMA was the restructuring of the electricity generation sector. The OMA fears that following privatization, increased reliance on cheap coal-generated electricity will result in high levels of air contaminants including volatile organic compounds, particulate matter, sulphur dioxide, mercury and carbon dioxide. This concern was echoed in a report[74] produced for the Toronto Board of Health which recommended stricter emission limits for a number of contaminants[75] originating from the electricity sector.

The OMA concluded that Ontario air standards are insufficiently stringent and called for a number of reforms including:

- stiffer sulphur dioxide and nitrous oxides emission limits for the electricity sector;
- a province-wide sulphur dioxide emission reduction of 75%;
- legislatively-mandated nitrous oxides emission reductions at Ontario Hydro; and
- stricter vehicular emission standards.

The OMA also highlighted the fact that current standards are not respected, noting that annual average

[71] Ontario Ministry of the Environment. *Ontario's Smog Plan: A Partnership for Collective Action.* Steering Committee Report. (January 1998). www.ene.gov.on.ca/envision/programs/3573e.pdf

[72] Available at: http://www.oma.org/phealth/ground.html

[73] http://www.oma.org/pcomm/pressrel/1998/may12.html

[74] Perrotta, K. and de Leon, F. *Ontario's Changing Electrical Sector: Implications for Air Quality and Human Health.* (March 1999). Prepared for Toronto Department of Public Health.

[75] sulphur dioxide, nitrous oxides, carbon dioxide, mercury, arsenic, beryllium, cadmium, chromium, lead and nickel.

ground-level ozone levels in the Great Lakes Basin have consistently surpassed the National Ambient Air Quality Objective of 15 ppb for the past several years. The OMA drew attention to the associated issue of severe cuts to Ontario's Ministry of the Environment, noting for example, that there were 40% fewer staff in 1997 than in 1990.

Since this report, the Minister of Environment has announced that emissions trading in the electricity sector would be permitted. It will allow for trading with companies from other (non-electricity) sectors which have no emission caps; it will allow trading with non-Ontario firms and sectors which have no emission caps; and accordingly will allow increased use of coal fired power plants to supply Ontario electricity users.[76]

Accordingly, the concerns expressed by the OMA in its 1998 report remain outstanding and furthermore, the impacts on childrens' health are expected to get worse rather than better from the electricity sector.

5.4 FEDERAL REGULATION

5.4.1 National Ambient Air Quality Objectives

National Ambient Air Quality Objectives (NAAQO) serve as a benchmark for air pollution regulatory regimes across Canada. They are non-binding and are designed to guide regulators in the issuance of operating permits to pollution-generating facilities. As discussed above, the Ontario MOE reports that it utilizes NAAQOs when setting its own air standards.[77] To date, objectives have been established for five pollutants: sulphur dioxide, nitrogen oxide, carbon monoxide, ozone and particulates.

The objectives specify the maximum permissible concentration of an air contaminant resulting from the combined emissions of all sources in an area. They are set for different time periods including, for example, one hour, eight hour and twenty-four hour objectives. The objectives include three regulatory levels:

i) tolerable: intended to protect against adverse effects to human health. Concentrations above this level require prompt action in order to protect public health;

ii) acceptable: provide adequate protection against deleterious effects to the environment, personal comfort and well-being; and

iii) desirable: long-term goal for the improvement of existing air quality.

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[76] For a good review of these and other important criticisms of the Ontario proposals with respect to emissions trading as well as implications from Ontario Energy Board decision making, see Kim Perotta, Toronto Public Health, Speaking Notes, *Stationary Sources - Implementation Approaches and Issues Municipal Perspective*, reproduced in Appendix C to the Final Report, *Ground-level Ozone Management: Approaches, Mechanisms, and Implementation*, Stakeholder Consultation, Negotiation of an Ozone Annex to the Canada-U.S. Air Quality Agreement, (February 10, 2000) [compiled by Environmental Canada, International Smog Programs].

[77] Personal Communication, J. Smith, D. Harper, A. Socha, Standards Development Branch, Ontario Ministry of the Environment. (March 30, 1999).

5.4.1.1 Derivation of NAAQOs

The Working Group on Air Quality Objectives and Guidelines (WGAQOG), composed of federal, provincial and territorial representatives of environment and health, is responsible for the development of National Ambient Air Quality Objectives. The Working Group reports to the *CEPA* Federal-Provincial Advisory Committee.

There are several steps in the process of developing a NAAQO. First, substances must be proposed and accepted for NAAQO development. A scientific review of the substance is then undertaken, using available information, in order to identify dose-response relationships for a variety of receptor end-points. Based on the scientific data, a Reference Level is determined. This is the level above which there are demonstrated health and/or environmental effects. A risk assessment is undertaken and a Rationale Document is prepared. The latter summarizes the scientific information, risk assessment and exposure estimate. It is used to derive a recommended NAAQO, which is presented to the *CEPA* Federal-Provincial Advisory Committee, the National Air Issues Coordinating Committee, Environment Canada and Health Canada, for adoption.[78] These steps are described in detail below.

The Working Group, the *CEPA* Federal-Provincial Advisory Committee and the National Air Issues Coordinating Committee may all nominate substances as candidates for the development of a NAAQO. The Working Group determines whether it is appropriate to proceed with the development of an air quality objective for a nominated substance by considering a number of factors including the abundance of the substance in the Canadian environment, environmental persistence of the substance, the capability and likelihood of the substance to cause adverse effects to human health or the environment, the existence of sub-populations that are sensitive to the substance, environmental transformation of the substance, and the appropriateness of managing the substance via an air quality objective.

When a substance is accepted for the development of a NAAQO, the Working Group undertakes a scientific assessment. The review includes an evaluation of the substance's physical and chemical properties, sources, and environmental fate, behaviour and levels. Also considered are monitoring technologies for detecting the substance. The substance's toxicity is assessed and exposure estimates are undertaken. Finally, a risk characterization for the substance is prepared. Toxicity assessment, exposure estimates and risk characterization are described, in turn, below.

Toxicity assessment first involves a qualitative assessment. This step critically assesses the scientific data concerning the substance. It provides conclusions regarding the likelihood that a substance poses a hazard to human health or the environment, the nature and severity of its potential effects, and the conditions of exposure under which the effects occur. Quantitative assessment describes the dose-response curves for various end-points and receptors.

According to the Working Group, exposure assessment considers all routes and media. The Working Group reviews existing data and undertakes exposure studies. It assesses the spectrum of potential

[78] Federal-Provincial Working Group on Air Quality Objectives and Guidelines. *A Protocol for the Development of National Ambient Air Quality Objectives. Part 1: Science Assessment Document and Derivation of the Reference Level(s)*. (1996) [hereinafter, *WGAQOG Protocol*].

receptors and identifies any subsets that are likely to exhibit heightened sensitivity. Finally, it provides estimates of human and environmental exposure to the substance. The purpose of risk characterization is to compare data on probable exposure levels with those levels that cause adverse effects. Sensitive or susceptible populations are identified.

The science assessment stage concludes with the determination of one or more Reference Levels for the pollutant. The Reference Level is the level above which there are demonstrated effects on human health or the environment. Reference Levels are intended to serve as benchmarks against which proposed National Ambient Air Quality Objective levels can be compared. Science assessment documents are subject to both internal and external review.[79]

In the risk management stage, control technologies, economic factors and other management issues are considered. A Rationale Document is prepared and includes the recommended objective as well as the rationale, based on the above-described analysis, for this choice.

5.4.1.2 The Relationship Between National Ambient Air Quality Objectives and Canada-Wide Standards

Recently, the processes for the development of National Ambient Air Quality Objectives and Canada-Wide Standards[80] were integrated. Air pollutants identified by government as management priorities will be targeted for the development of *either* a Canada-Wide Standard or a National Ambient Air Quality Objective.

The Working Group on Air Quality Objectives and Guidelines first prepares a risk assessment report, as described above. This is followed by the development of either a Canada-Wide Standard or a National Ambient Air Quality Objective. The former is undertaken by a committee of federal, provincial and territorial environment and health representatives when a commitment has been made by the Environment Ministers. In the absence of such a commitment, a NAAQO is developed by the WGAQOG. Both processes take place in consultation with stakeholders.[81]

5.4.2 Canada-Wide Standards

Particulate matter (PM) and ground level ozone are of particular concern to children. In January, 1998, the Environment Ministers identified PM and ozone as priorities for the development of Canada-Wide Standards. A Canada-Wide Standards Development Committee of federal, provincial and territorial environment and health officials was established. The group's mandate is to make recommendations on the form, target date and level of Canada-Wide Standards for ozone and PM, to organize and participate in national stakeholder consultations for the proposed standards and to prepare an overview of the jurisdictional implementation plans. Canada-Wide Standards for PM, ground-level ozone, Benzene and Mercury were proposed [82] with notice in the Canada Gazette on February 5, 2000. [83] These Standards

[79] *WGAQOG Protocol, op.cit.*

[80] See discussion in Chapter 6: Toxic Substances.

[81] Letter from Vic Shantora, Federal Co-chair, CEPA Federal/Provincial Advisory Committee to P. Muldoon, (Aug 25, 1998).

[82] http://www.mbnet.mb.ca/ccme/3e_priorities/3ea_harmonization/3ea2_cws/3ea2i_overviews/3ea2i4.html

are being developed under the framework of the Canada-wide Accord on Environmental Harmonization and the Canada-wide Environmental Standards Sub-Agreement (discussed further in Chapter 6). The Strategic Priorities Directorate of the Canadian Council of Ministers of the Environment (CCME) advised that the federal and provincial Ministers are also to consider other options for PM and ozone, including setting a standard for PM_{10} (coarse particulate matter), shortening the time frame for meeting the ozone target which is now set at 2015 and undertaking to review the standard in three years.[84] Since the federal-provincial agreements as to the proposed standards may be signed after the coming into force of the *Canadian Environmental Protection Act, 1999*, the Minister of the Environment published the proposed agreements in the Gazette as that legislation requires. Therefore the legislative framework for the federal agreement would be *CEPA 1999*. The federal and provincial Ministers intend to sign agreements as to the proposed standards in spring of 2000.

Benzene

Benzene was selected as a Canada-wide environmental priority in recognition that it is carcinogenic with no threshold and with the aim of reducing Canadians' exposure to "this known human carcinogen."

The agreement with respect to benzene (proposed under the Canada-wide Environmental Standards Sub-Agreement) proposes that the CWS is a national target of 30% reduction in total benzene emissions from 1995 emission inventory levels to be achieved by the end of the year 2000 in phase 1 and phase 2 to be developed for discussion in the spring of 2001.

The rationale states that the CWS "represents a balance between the desire to achieve the best health and environmental protection possible and the feasibility and costs of reducing the emissions that contribute to elevated levels of benzene in ambient air. The primary long-term air quality management goal for non-threshold toxicants like benzene is to reduce exposure to the extent possible and practicable, thereby reducing the risk of the adverse effects of this pollutant on human health." The rationale argues that lack of scientific certainty is not to be used as a reason to postpone "cost-effective measures to prevent environmental degradation." However, the rationale also acknowledges that most of the measures needed to reach the CWS are already underway.

It is apparent that the risk management approach has been relied upon in developing the Benzene CWS such that the proposal will not reach the level that would be necessary to best protect against health and environmental impacts. It is also apparent that the driver for the standard selected is not the CWS process; it is merely a repetition of measures already underway.

Particulate Matter and Ozone

Again, the Canada-Wide Standards for particulate matter and ozone proposed in the February 5th *Gazette* notice have been developed under the Harmonization Agreement and Sub-Agreement. The rationale states that "Significant adverse effects have been demonstrated for the air pollutants PM and ozone on human health and the environment." The context statement states that the long-term air quality management goal for these two pollutants is to "minimize the risks of these pollutants to human health

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[83] Canada Gazette Part I, Vol. 134, No. 6, (February 5, 2000), p.320, Government Notices: Department of the Environment, Canadian Environmental Protection Act, 1999: *Agreements Respecting Canada-Wide Standards for Benzene - Phase 1, for Particulate Matter (PM) and Ozone, and for Mercury*. See also http://canada.gc.ca/gazett/hompar1_e.html for the Gazette website and http://www.ccme.ca for the CCME website with additional information on the Particulate Matter and Ozone options.

[84] Cynthia Wright, Director General, Strategic Priorities Directorate, (February 15, 2000), Letter to Stakeholders re Canada-wide Standards for PM and Ozone, Benzene and Mercury.

and the environment. However, recent scientific evidence indicates that there is no apparent lower threshold for the effects of these two pollutants on human health."

Again, the document states that the CWS's proposed at this time "represent a balance between the desire to achieve the best health and environmental protection possible in the relative near-term and the feasibility and costs of reducing the pollutant emissions that contribute to elevated levels of PM and ozone in ambient air." It continues, "As such, while they will significantly reduce the effect of PM and ozone on human health and the environment, they may not be fully protective and may need to be re-visited at some future date."

The CWS proposed at this time for PM is to deal with the "fine fraction", $PM_{2.5}$ for the interim period until 2005 when a planned review of the standard will be completed. There is to be consideration in the meantime as to whether the Ministers can agree on a PM_{10} standard. Similarly, there is to be consideration given to shortening the time frame from meeting the ozone target; namely reducing it from being reached by the year 2015 to 2012 or 2010. The need for reduction of transboundary flows of PM and ozone in certain regions in order to reach the targets is also acknowledged. Because most areas of Canada have ambient levels of PM and ozone better than the standards, but still above the levels associated with observable health effects, there is also included in the agreements, an Annex consisting of a guidance document for "Continuous Improvement and Keeping-Clean Areas-Clean".

This guidance document explicitly states that "There is a need to ensure that the public recognizes that CWS levels are only a first step to subsequent reduction towards the lowest observable effects levels. It would be wrong to convey the impression that no action is required in these areas or that it would be acceptable to allow pollutant levels to rise to the CWS levels." However, the measures called for in the Annex are very indeterminate: under the topic of Continuous Improvement areas, it states that "Jurisdictions should take remedial and preventative actions to reduce emissions from anthropogenic sources in these areas to the extent practicable." In Keeping Cleans Areas Clean, the document states that "Jurisdictions should work with their stakeholders and the public to establish programs that apply pollution prevention and best management practices." Examples cited include "strategies consistent with the CCME commitment to pollution prevention"; ensuring that new facilities and activities incorporate "the best available economically feasible technologies to reduce PM and ozone levels;" requiring capital upgrades to do the same, and "reviewing new activities that could contribute to an increase in PM and ozone levels with stakeholders and the public in terms of their social, economic and environmental merits." No process requirement or methodology for the latter is specified.

In the reporting methodologies section of the document is contained a statement that, "For the province of Ontario, a 45% reduction in NOX and VOC emissions from 1990 levels by 2015 will be considered the province's appropriate level of effort towards achieving the ozone CWS. Any remaining ambient ozone levels above the CWS in Ontario will be considered attributable to the transboundary flow from the U.S. of ozone and its precursor pollutants."

It is apparent from the document, that the proposed CWS's for ozone and particulate matter are not the optimum health based standards that one would hope for in protection of human health. Since these two substances are of particular concern to children and their respiratory health, we would conclude that the CWS process has not resulted in standards that are intentionally protective of children's health. Rather, the CWS's are heavily influenced by "risk management" considerations, including inter-jurisdictional and political considerations, not the least of which is the high ambient levels for these substances in Ontario from existing local and cross-border sources.

Mercury

The preamble to the Canada-wide Environmental Standards Sub-Agreement with respect to a Mercury CWS recites the high levels of mercury in fish and wildlife warranting "additional efforts to reduce atmospheric emissions derived from both deliberate use of mercury and from incidental releases of mercury." In addition to the health impacts, including those on sensitive populations, which the document describes as infants, children, and women of childbearing age, and those following traditional lifestyles, the document also describes that there is "additional, largely unquantified risk to fish-eating wildlife." The document, in describing how difficult it is to ascribe proportions of impact attributable to anthropogenic releases and how much to natural sources, states that "Because it is a natural and persistent bioaccumulative element which can be transported many miles in the atmosphere, mercury can have impacts many years and many miles removed from its original source.

A common thread through all mercury impacts is that deposition to water bodies from anthropogenic emissions poses a threat to human and ecosystem health, and that reduced deposition will contribute, in time, to reduced impacts." After reciting a list of jurisdictions in which mercury has been "consistently targeted for emission reductions", the agreement states that "Ministers of the Environment have thus agreed to undertake and promote the cost-effective actions to achieve further precautionary reductions in anthropogenic emissions (releases to the air) of mercury."

The document identified three major sectors as responsible for the bulk of mercury emissions: base metal smelting (the largest), waste incineration and coal-fired electricity. Standards to improve base metal smelting and waste incineration that are cost-effective have been identified. However, it states that "Efforts to develop a standard for the electricity generation sector have been complicated and progress has been delayed such that a work plan to develop standards for this sector will not be completed until early in 2000." For base metal smelting, existing facilities are expected to apply "best available pollution prevention and control techniques economically achievable" to achieve 2 g/Hg tonne of mercury per tonne of finished metals. For new and expanding facilities, the requirement is to apply "best available pollution prevention and control techniques to minimize mercury production throughout the life-cycle to achieve 0.2 g Hg/tonne for zinc, nickel and lead and 1 g Hg/tonne for copper. Existing facilities are expected to "make a determined effort" to meet the existing facilities standard by 2008; new and expanding facilities will be required to design for and achieve compliance immediately upon full scale operation. New or expanding incineration facilities, of any size, are to apply "best available pollution prevention and control techniques" to achieve the emissions limits which are specified according to whether they are municipal waste incinerators, medical waste incinerators, hazardous waste incinerators or sewage sludge incinerators. The highest emission levels are specified for the latter; the next highest for hazardous waste. Existing facilities are expected to apply "best available pollution prevention and control techniques" to achieve concentrations at the same levels as for new facilities for the specified waste stream, except that large medical waste incineration has a higher limit. New and expanding facilities must meet the targets immediately; existing facilities must meet the targets between 2003 and 2006 depending upon which waste stream they are incinerating.

For mercury from electricity generation, no CWS has yet been proposed. Clearly the jurisdictions are unable to arrive at a standard that is intentionally protective of children's health from this sector when no standard is even agreed. For the other two major sectors responsible for mercury emissions, that is, waste incineration and base metal smelting, it is apparent that a risk management approach has been taken in which the standards proposed are contingent upon availability of control equipment technologies and techniques. For existing facilities, there is the added consideration as to whether they are "cost effective" and "reasonably available".

The premise in setting standards for mercury is that the sectors must be allowed to continue in full

operation unimpacted by concerns for impacts on human health. Since children are identified as one of the key sensitive groups impacted by mercury pollution, this approach to the standard will mean that the impacts from existing mining and incineration facilities will continue, with reductions and improvements only subject to all of the caveats built into the CWS. In addition, these impacts are even more problematic for communities in Northern Ontario and elsewhere who live traditional lifestyles and who live off of the land. Members of First Nations communities who lead a traditional harvesting lifestyle are precluded from taking their wives and children with them on the land during the summer season, as they traditionally would do, because of the restrictions on fish consumption caused by the high mercury levels in some of the local lakes, resulting from mining operations. Mammals are also affected and alternative non-traditional food sources are not practical in these areas. Alternatively, if families feel compelled or desire to continue the practice of taking the whole family on the land, women of child-bearing age and children are exposed to troublesome levels of mercury in their foods.

5.4.3 The Canadian Environmental Protection Act [85]

5.4.3.1 National Emission Standards and Guidelines

National Emission Guidelines are suggested maximum pollutant emission levels for individual facilities and are based on the best practically-achievable technology. They are non-binding but are intended to be adopted as binding regulations by the provinces. National Emission Guidelines exist for emissions generated in arctic mining, coke ovens, asphalt paving and cement plants, among other sectors.

National Emission Standards are binding under the *Canadian Environmental Protection Act*[86] and set maximum emission levels for particular facilities. Standards have been established for lead from secondary lead smelters,[87] mercury from mercury-cell chlor-alkali plants,[88] vinyl chloride released in the manufacture of vinyl- and polyvinyl chloride,[89] and asbestos mines and mills.[90] Both National Emission Standards and Guidelines are developed by ad-hoc federal-provincial government and industry task forces that are established when there is a perceived problem with emissions from a particular industry.[91] This process has been overtaken by CCME processes pursuant to the Harmonization Agreement & the Standards Sub-Agreement thereunder.

5.4.3.2 Gasoline

The *CEPA Gasoline Regulations*,[92] which were implemented in response to health and environmental concerns, limit lead and phosphorous levels in gasoline.

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[85] *CEPA, op.cit.*

[86] *Ibid.*

[87] *Secondary Lead Smelter Release Regulations*, SOR/91-155.

[88] *Chlor-Alkali Mercury Release Regulations*, SOR/90-130.

[89] *Vinyl Chloride Release Regulations*, SOR/92-631.

[90] *Asbestos Mines and Mills Release Regulations*, SOR/90-341.

[91] Mellon *et al.*, *The Regulation of Toxic and Oxidant Air Pollution in North America.* (CCH, Toronto, 1986).

[92] SOR/90-247.

The *CEPA Sulphur in Gasoline Regulations*[93] limit sulphur content in gasoline that is produced or imported for use or sale in Canada and for gasoline that is sold or offered for sale in Canada. Sulphur oxides are a precursor to smog and acidic precipitation, and these regulations were adopted to address environmental and health concerns related to these pollutants.[94]

5.4.4 Automobile Emissions: The Motor Vehicle Safety Act

The federal government is authorized under the *Motor Vehicle Safety Act*[95] to set emission limits for vehicles that are imported into or manufactured in Canada. Regulations[96] under the act set out permissible exhaust emission levels for hydrocarbons, carbon monoxide, nitrous oxides, among other contaminants. Generally, these emission limits are based on the capacity of available emissions reduction technologies.[97] The act also permits the government to require the installation of specific pollution control equipment. The sale of cars in Canada that fail to meet these standards is prohibited.

5.4.5 The Canadian Council of Ministers of the Environment

5.4.5.1 Comprehensive Air Quality Management Framework Agreement

The National Air Issues Coordinating Committee (NAICC), formed of staff from the federal and provincial environment and energy departments, is mandated under this agreement to develop coordinated air issue management plans and strategies, and to track progress in achieving targets to reduce air pollution. The NAICC is organized into two groups, one dealing with climate change and the other addressing remaining air quality issues. The Committee is developing action plans to reduce smog and acidic emissions and is also working on international programs such as the Canada-U.S. Accord on Acid Rain.[98]

5.4.5.2 National Action Plan for the Environmental Control of Ozone-Depleting Substances (ODS) and their Halocarbon Alternatives

This plan updates the 1992 National Action Plan for the Recovery, Recycling and Reclamation of Chlorofluorocarbons (CFCs). Objectives of the new, 1998 Plan include the improved environmental management of all ozone-depleting substances and their alternatives and to decrease their emissions from all industrial sectors. This is to be accomplished through a number or initiatives including:

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[93] SOR/99-236.

[94] The regulations specify that: beginning July 1, 2002, gasoline sulphur concentrations will be limited to an average of 150 ppm; beginning January 1, 2005, average levels should not exceed 30 ppm. A never-to-be-exceeded limit of 300 ppm is mandated for 2004, which will drop to 80 ppm thereafter.

[95] *Motor Vehicle Safety Act*, R.S.C. (1985), c. M-10.

[96] *Motor Vehicle Safety Regulations*, C.R.C., c. 1038.

[97] Estrin, D. and Swaigen, J., 1993, *op.cit.*

[98] http://www.mbnet.mb.ca/ccme/3e_priorities/3eb1.html

- minimizing emissions during the installation, operation, maintenance, repair, disposal and decommissioning of systems and equipment;
- requiring recovery and recycling of these compounds in all industrial use sectors;
- identifying appropriate dates for the phase-out of specific uses of CFCs and halons or mandating total containment;
- developing a strategy for the disposal of surplus CFCs and halons;
- implementing environmental awareness training; and
- supporting use of non-ODS alternatives.[99]

5.5 CONCLUSION

The original question for the study was whether air standard setting is intentionally protective of children. The answer differs between jurisdictions and is mixed.

In Ontario, for those standards that remain unchanged and not yet reviewed, there is no evidence that the standards were intended to protect children in particular when originally set, and no evidence that they are in fact protective of children. However, the Ministry of the Environment's Standard Setting Plan, announced in 1996 and revised in 1999 holds out promise that matters will improve. For example, the MoE chooses the most sensitive receptor for its hazard analysis, and that may be children. The MoE takes into account a multi-media, pathways approach in considering who or what is the most sensitive receptor. Where there is a receptor more sensitive than children (for example, an ecosystem effect), then children should also be protected. What remains to be seen is whether after the risk assessment stage, when hazard is identified, whether the risk management stage results in standards that are in fact protective of children. For example, for the current group of 18 standards presently under review, the risk management stage has yet to be undertaken and the criteria for evaluation and application of any alleged obstacles to implementation of the new standards has yet to be developed. The methodology and results of this stage will be critical.

Federally, the various standard setting processes have moved primarily to the Canada-Wide Standards process under the federal-provincial Environmental Accord. As some of the contaminants selected as priorities have now had proposed standards published in the Canada Gazette, along with the applicable rationale, it appears that the resulting standards are heavily influenced by the "risk management" part of the process; in particular by the approach that requires a unanimous consensus based approach to the adoption of new standards. The jurisdictions with the biggest problems for the particular contaminant seem to be driving the standard to a lower level than that which would be most protective of health and the environment. Accordingly, this approach is not intentionally protective of children, nor actually protective of children. This is an unfortunate result given that previously, the National Ambient Air Quality Objectives were one of the main federal contributions to air standard setting and were health based objectives. Although not binding, they have been, and remain, an important consideration in provincial air standard setting, at least in Ontario. The Canada-Wide Standard approach is not a health-based approach; it is a stakeholders' approach. Progress for the contaminants under this approach will be slow, if at all, according to the rationale documents for the proposed standards.

For both Ontario and Canada, federally, of course, as discussed in chapter 4, there also remains the issue of exposure to multiple chemicals and additive or synergistic effects. There also remains the issue of

[99] http://www.ec.gc.ca/ozone/nap-pan/nap_e.html

whether the underlying studies that the MoE and other standard setters are relying upon have assessed any health endpoints other than cancer. Additional work is needed in Ontario, as well as elsewhere, to begin to address these inherent deficiencies in the science underlying the standard setting process. The weight of evidence approach described in chapter 4 needs to be applied at every stage in the process, from the original design and analysis of the underlying studies, through to the risk management or final standard setting decisions.

5.6 RECOMMENDATIONS

5.6.1 Recommendations for Ontario

1. The Ontario standard setting plan is proceeding and should be encouraged to reach timely results with respect to the priority substances identified for review. However, the ultimate standards adopted in the group of eighteen contaminants currently under review and the fifteen yet to be proposed are highly dependant upon the approaches taken by Ontario in the next "risk management" stage of the process. The Ministry of the Environment should follow through with the development of a transparent, detailed and specific plan for finalization of these standards, as soon as possible. For carcinogens, the standards should in all cases be established at the risk level of no greater than ten to the minus six; with specific time frames for compliance being specified in the standard, if not immediately. No time frames should exceed five years for any substance, regardless of "implementation issues."

2. Research with respect to the evidence and data gaps for non-carcinogenic risks (for example endocrine disruptors and other health end-points) is a high priority for incorporation into standard-setting exercises and should be supported by the Ontario government.

3. Ontario should proceed with its own review of all of the priority air contaminants, originally identified, regardless of whether any of these are also in a Canada-Wide Standard or other federal provincial process. Ontario should ensure that all of the air contaminants in the province are regulated in the same manner and to the same risk levels.

4. Ontario should place special emphasis on standards for nitrous oxides and particulate matter in its own standard setting process because of the impact of these contaminants on children's health and because of the levels in which they are found in the Ontario environment.

5. Ontario should drastically improve its ozone commitment and should actively work to support a stringent ozone Annex between Canada and the United States.

6. Ontario should immediately repeal its plan with respect to emissions trading in the electricity sector and replace it with a plan that will ensure improved air quality from this sector within five years.

5.6.2 Recommendations for Canada

7. The Canada-Wide Standards process under the Environmental Harmonization Accord Standard Setting Sub-Agreement is ineffective for protecting children's health and should be repealed with respect to air contaminants.

8. The Federal Minister of the Environment should establish standards on a health protective basis rather than pursuant to a Canada-wide consensus approach, and without risk management considerations.

9. Health protective standards should be published regardless of implementation issues.

10. Where implementation barriers are identified that require industry sector adjustments, sectoral time frames for compliance should be immediately established and subject to third party review.

11. All opportunities to improve current commitments (for example, shorter time frames, or more stringent standards) should be vigorously pursued.

12. A stringent ozone Annex should be reached with the United States as soon as possible.

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Chapter 6: Toxic Substances

Chapter 6: Toxic Substances

6.1 Introduction

This chapter provides an overview of several areas of international, federal and provincial efforts to control toxic substances in the environment. Instead of looking at individual standard-setting examples or regulations, general or overall approaches to risk assessment and risk management are addressed. As well, each area has been reviewed in terms of whether and how precautionary approaches such as weight of evidence and reverse onus are applied.

6.2 The Great Lakes Water Quality Agreement

The Great Lakes Water Quality Agreement was the culmination of multiple studies by the International Joint Commission (IJC), and an important landmark in the recognition of toxic substances in the environment. Originally signed in 1972, the updated 1978 agreement called for a prohibition of "the discharge of toxic substances in toxic amounts" and "the virtual elimination of the discharge of any or all persistent toxic substances."[1]

The agreement named over 350 *hazardous polluting substances*, defined as "any element or compound identified ... which, if discharged in any quantity into or upon receiving waters or adjoining shorelines, would present an imminent and substantial danger to public health or welfare."[2] These substances were divided between those known to have toxic effects and a potential for discharge, and those that may have such toxic effects and discharge potential. Toxicity was determined based on animal studies using specific criteria, with some risk of discharge being necessary for its inclusion. In some cases, the agreement outlined reduction targets for specific substances that were intended to protect the most sensitive aquatic species.[3] It also stated that levels should be based on combined exposures from various media, and on the interactive effects of toxic substances, calling for more research into these interactions.[4]

Further International Joint Commission reports continued to refine their assessment of toxicity, recommending a "weight of evidence" approach and supporting the concept of "reverse onus" where the responsibility for proving reasonable safety falls on the producer.[5] This combination of an assessment based on inherent toxicity and a reverse onus approach created a precautionary framework that emphasized human and environmental health.

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[1] *Revised Great Lakes Water Quality Agreement of 1978, Article II(a).*

[2] *Ibid., Article I(j).*

[3] *Ibid., Annex 1.*

[4] *Ibid., Annex 12 (6).*

[5] International Joint Commission. *5th, 6th and 8th Biennial Reports on the Great Lakes Water Quality Agreement.*

6.3 CANADIAN ENVIRONMENTAL PROTECTION ACT

6.3.1 CEPA, 1988

The 1988 *Canadian Environmental Protection Act* (*CEPA*)[6] takes a chemical-by-chemical approach to managing toxics in the environment. Several lists of chemicals are established for regulatory purposes. Overall, the Domestic Substances List (DSL) covers all substances in commercial use in Canada. It currently contains over 23,000 substances.

CEPA, 1988, defines a substance as toxic if it may be entering the environment at a level that may harm human health or the environment. Under the Act, a substance cannot be regulated merely for having the inherent potential to cause harm; it must also be shown to be entering or likely to enter the environment at levels sufficient to cause harm. Action can only then be taken after a substance is placed on the Toxic Substances List (TSL) which is a permanent list contained in Schedule 1 to the Act. The legislation outlines two ways in which substances reach the TSL. The first is through the creation of a Priority Substances List (PSL 1), with each of the chosen priority substances being assessed by the Ministries of Health and Environment.[7] Health Canada takes responsibility for the human health risk assessments and Environment Canada for the risk to ecosystems. Designation of a substance to a PSL is temporary until a decision is made to assign the substance to the TSL or not.

The risk assessments of PSL substances are externally peer reviewed and must also be approved by the Rulings Committee of the Bureau of Chemical Hazards in Health Canada and the Environment Canada-Health Canada *CEPA* Management Committee. If a substance is determined to be toxic according to the *CEPA* definition ("*CEPA*-toxic"), it is then recommended for the Toxic Substances List (Schedule 1). A substance is "*CEPA*-toxic" if it is persistent, bioaccumulative and primarily the result of human activity.

Listing as a toxic substance does not, however, require that action be taken. Appropriate controls are to be decided through a risk management phase that includes social and economic factors.[8] The Ministers must, nevertheless, on receiving notice of a Priority Substance's assessment as toxic, publicly outline a plan of action or inaction. The assessments of the 44 substances on the PSL1 were completed by 1994 with 25 substances declared toxic, 6 not toxic, and 13 undecided due to a lack of sufficient information.

A large number of those PSL1 substances declared *CEPA*-toxic have gone to a 'Strategic Options Process' (SOP), multi-stakeholder consultation. Also called Issue Tables, these multistakeholder groups look at technical, social and economic factors in providing advice to decision-makers. The stated objectives of the SOP are those of the federal Toxic Substances Management Policy (TSMP) (see below): "virtual elimination" of persistent and bio-accumulative substances and lifecycle management of the rest.[9] Following an assessment of the options available for controlling a substance, the Ministers of

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[6] *Canadian Environmental Protection Act*, R.S.C. 1988, s.11 (Hereinafter, *CEPA, 1988*).

[7] According to Section 33, the second way a substance can be placed on the TSL, without a Priority Substances evaluation, is if the Ministers are already "satisfied" the substance is toxic.

[8] Health Canada. *Human Health Risk Assessment for Priority Substances*. (1994), p.2.

[9] *Strategic Options for the Management of Toxic Substances from the Steel Manufacturing Sector*: Report of the Stakeholder Consultations, Draft #3. (Nov 1996), p.55.

Environment and Health may then propose a strategy of regulatory and/or non-regulatory instruments for eliminating the risk posed by a toxic substance.

Twenty-five substances have since also been named to the second Priority Substances List and are currently being reviewed.

The risk assessments conducted by both ministries, are based on methods developed by the U.S. Environmental Protection Agency and include the requirements of entry into the environment, potential exposure and evidence of effects (called the 3E approach).[10] In addition to quantitative analysis, Environment Canada states they use a "weight of evidence approach" in dealing with the "biases and uncertainties" associated with risk assessment.[11] Using a three-tiered method, Environment Canada starts with more conservative assessments and then progressively factors in distributions of exposure and effects to derive a more "realistic" picture.[12] Community and population models of ecosystems are then used to aid weight-of-evidence determinations, and the ecological significance of an organism is factored in to the decision as to whether the potential risks are important.[13]

Both Health Canada and Environment Canada admit that, because of scientific uncertainties, the use of risk assessment involves a fair amount of "professional" or "sound scientific judgement on a case-by-case basis."[14] In addition, uncertainty factors are routinely used in human health risk assessments to account for the inherent weaknesses of epidemiological and toxicological studies and for gaps in the data. For instance, following the models developed in the US, Health Canada uses 10-fold uncertainty factors to account for 'inter-species' differences (such as between rats and humans), and 'intra-species' differences (such as the different susceptibilities of children or the elderly) among others.

Health Canada calculates exposures for six different age groups, taking into account differences in food, water and air intake, as well as differences in other behaviour such as children's play habits. The department estimates "mean and reasonable worst case exposure" for the general population, but also for populations near point sources. Where priority substances are known to exist in consumer products, they also estimate the direct exposure to those substances when data are available.[15]

Health Canada calculates the maximum acceptable Tolerable Daily Intake (TDI) for a substance, the acceptable daily intake over a lifetime. This limit is based on the most sensitive effect (also called the critical effect) found, meaning the effect that occurs at the lowest dose. Effects on child development due to early exposure for example, only form the basis of the TDI if there are data showing that they occur at the lowest doses. If such evidence is unavailable, extra uncertainty factors are not used, as the fetus and the child may not necessarily be the most susceptible population for the specific substance.[16]

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[10] Environment Canada. *Environmental Assessments of Priority Substances Under the Canadian Environmental Protection Act, Guidance Manual Version 1.0.* (March 1997), pp.1-5.

[11] *Ibid.,* pp.1-4.

[12] *Ibid.*, pp.7-4.

[13] *Ibid.*

[14] *Ibid.*, p.1-3; and Health Canada. *Human Health Risk Assessment for Priority Substances.* (1994), p.2.

[15] Written communication, Ron Newhook, Bureau of Chemical Hazards, Health Canada, June 14, 1999.

[16] *Ibid.*

6.3.2 CEPA, 1999

The revised *Canadian Environmental Protection Act* was proclaimed in 1999 and includes a commitment to the precautionary principle, but only when such measures are considered "cost-effective."[17] *CEPA, 1999* also sets out new ways for substances to reach the Toxic Substances List, and incorporates the TSMP criteria and definition of "virtual elimination." Of the 23,000 substances on the Domestic Substances List, the list of substances currently used in Canada, these will now be assessed through one of three tracks. A flow chart describing this process is reproduced in Figure 6.1.

[17] *Canadian Environmental Protection Act*, R.S.C. 1999, s.2(a) (Hereinafter, *CEPA, 1999*). Environment Canada has not clarified what "cost effective" means in this context and could benefit from application of the kind of analysis conducted in the United States as to the health costs of air pollution (discussed in Section 5.2.5 of Chapter 5).

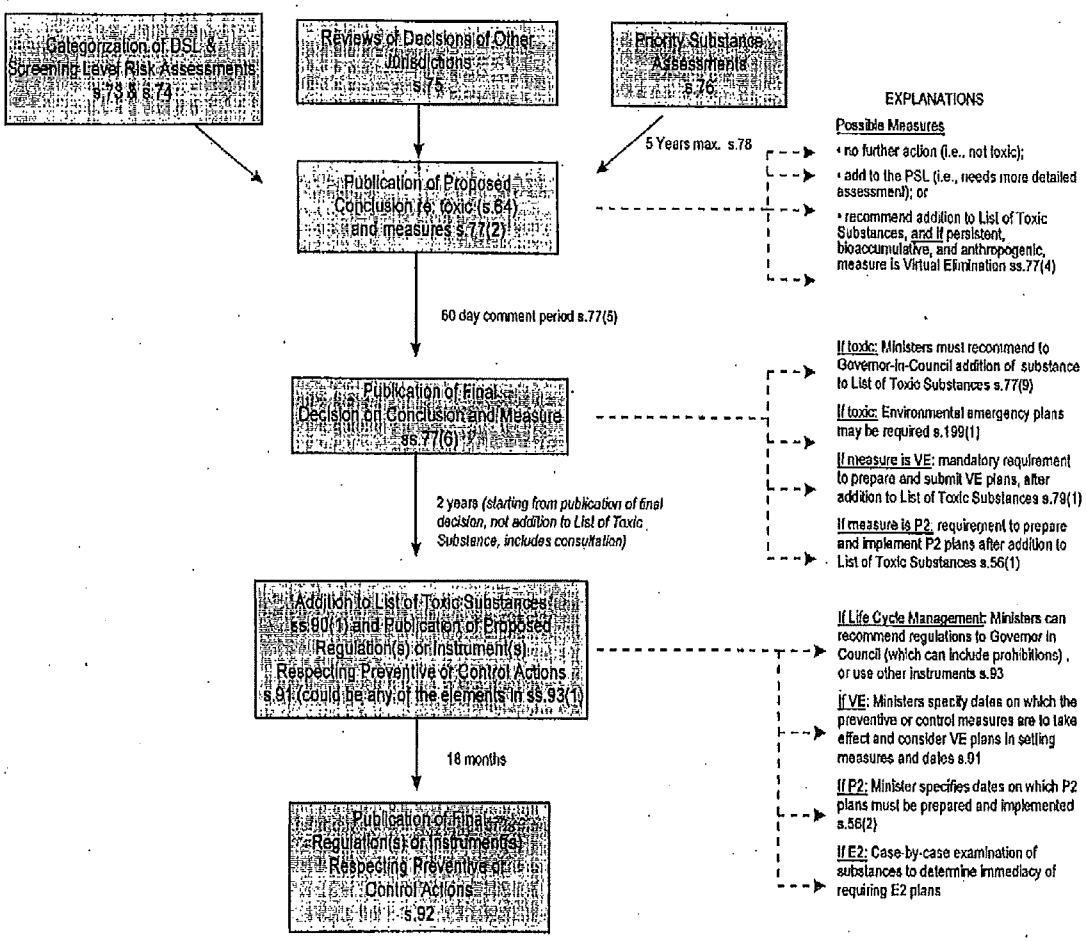

Figure 6.1. CEPA '99 Part 5 & 6.
(Source: Environment Canada, *A Guide to the New Canadian Environmental Protection Act*. March 2000, p.8.)

Those substances that have been nominated and chosen for a Priority Substance List are assessed for toxicity within five years, though this may be extended if there are insufficient data. Substances that have been banned in another country or jurisdiction, will have those decisions reviewed and will be added to either the PSL, if more assessment is considered necessary, or placed directly on the Toxic Substances List. The rest of the Domestic Substances List will be reviewed over the next seven years, first by categorizing substances based on their inherent toxicity, their persistence in the environment and their potential for bioaccumulation. All the substances will then undergo further assessments to determine if they are 'toxic' by the *CEPA* definition and will be added to the PSL or potentially to the TSL directly.[18] Where a substance is determined to be *CEPA*-toxic, i.e., persistent, bio-accumulative and primarily the

[18] Environment Canada. *A Guide to the New Canadian Environmental Protection Act*. (March 2000), p.7.

result of human activity, it will also be targeted for virtual elimination.[19]

Sections 80 to 89 of *CEPA* 1999 prescribe assessments for new substances to be used or imported into Canada that are neither on the Domestic Substances List, nor covered by other federal acts. Manufacturers or importers of these new substances must pay an assessment fee and provide the relevant information needed. In addition, the same procedure may be required for substances in use for which there are "significant new activities," but in both these cases, the ministers may waive the need for assessment information if they are satisfied that the substance will be contained so that it is not harmful to humans or the environment, or "if it is not practical or feasible to obtain the test data."[20]

6.4 TOXIC SUBSTANCES MANAGEMENT POLICY

In June of 1995, Environment Canada pre-empted the Standing Committee on the Environment and Sustainable Development's proposed changes to *CEPA* by releasing the Toxic Substances Management Policy (TSMP) two weeks before the Standing Committee issued its report.[21] The TSMP proposed a division of substances named toxic into a Track 1 for those that are persistent and bio-accumulative and a Track 2 for the rest. Track 1 substances would then be targeted for virtual elimination, and Track 2 substances would have 'lifecycle management' with attempts to reduce exposure at all stages.[22] The TSMP definition of virtual elimination is a lack of measurable release, an end-of-pipe approach, where the release of a substance must be below the 'Level of Quantification,' the lowest concentration that can be accurately measured using routine devices. This definition contrasts with the Standing Committee's proposal that virtual elimination include the elimination of use of a substance. By the TSMP definition, new persistent and bio-accumulative toxic substances can be manufactured or imported into Canada as long as there is no measurable release detected.[23]

The TSMP definition of virtual elimination was incorporated into *CEPA* 1999 referring only to the elimination of release, not of use. The Ministers of Environment and Health set the Level of Quantification for each substance named to a Virtual Elimination List. This level however, does not necessarily become the regulatory limit; the specific regulation is later determined by looking at other technical, social, political and economic matters.[24]

In addition to substances declared toxic through the *CEPA* processes, the TSMP, in theory, also applies to toxic substances that are '*CEPA*-equivalent,' for instance for pesticides, which are not covered by *CEPA*. According to the Federal Commissioner for the Environment and Sustainable Development's 1999 report however, the "departments cannot agree on other substances that could be considered *CEPA*-toxic

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[19] *CEPA, 1999*, s.77(3).

[20] Environment Canada. March 2000, *op.cit.*, pp.8-9.

[21] Standing Committee on Environment and Sustainable Development, House of Commons Canada, Report: It's About Our Health! Towards Pollution Prevention. CEPA Revisited. June, 1995.

[22] Environment Canada. *Toxic Substances Management Policy* (June 1995).

[23] Mausberg, B. et al. *A Response to the Proposed Toxic Substances Management Policy for Canada*, Canadian Environmental Law Association and Canadian Institute for Environmental Law and Policy (November, 1994).

[24] *CEPA, 1999,* s.65 (3).

equivalents or substances of concern. Criteria to identify them have not been established."[25]

6.5 PESTICIDES

The federal pesticide regime is distinct from that of other chemicals, pesticides being managed under the *Pest Control Products Act* (see Case Study #2 on Pesticides). While potentially harmful to humans, they are, by definition, toxic to parts of the ecosystem. Pesticides also differ fundamentally from other chemicals in that the release of a pesticide into the environment is essential to its use, and the elimination of release of pesticides is the same as the elimination of use.

Pesticides, unlike other chemicals in Canada, have historically required pre-market assessment and approval. The *Pest Control Products Act* requires that, to be approved, a pesticide's use must not lead to an "unacceptable risk of harm" and it must show efficacy for the purposes proposed.[26] This determination of efficacy is referred to as an assessment of "value," where it must be shown that a product does what it intends to do before it can be approved. Unacceptable risk of harm is not further defined however, and its interpretation is left to the discretion of the Pest Management Regulatory Agency, which has overseen pesticide regulation since 1995. The situation is further complicated because pesticides have not only active ingredients but also 'formulants,' vehicles that aid in their application, and these formulants may have safety profiles that differ considerably from the active ingredients. As discussed further in Case Study #2, pesticide formulants are not adequately regulated in Canada.

It is difficult to say how pesticides have been assessed for safety since they were first introduced in Canada. This difficulty arises since the majority of pesticide active ingredients were approved before 1981 when approval standards were less strict, 150 of these in use since before 1960.[27] No guidelines had been published by the PMRA describing their process for assessing new pesticides until the January 2000 draft report that describes the risk assessment and risk management framework the agency now uses. The draft also voices the PMRA's commitment to the precautionary principle and implementation of the TSMP and international agreements on substances such as the proposed Persistent Organic Pollutants treaty (see below).[28] A consistent approach to toxic substances has still not been reached across various departments however. The Federal Environment Commissioner notes for instance, that there are significant disagreements between the assessments of Priority Substances conducted under the *Canadian Environmental Protection Act* and the PMRA assessments of the same substances under the *Pest Control Products Act*.[29]

6.6 ACCELERATED REDUCTION/ELIMINATION OF TOXICS

The Accelerated Reduction/Elimination of Toxics (ARET) program is a voluntary pollution reduction

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[25] Federal Commissioner for the Environment and Sustainable Development. *Annual Report.* (1999), p.4.50.

[26] *Pest Control Products Act*, R.S.C. 1985, c.P-9.

[27] Federal Commissioner for the Environment and Sustainable Development. *Annual Report.* (1999), 3.75.

[28] Pest Management Regulatory Agency. *Risk Assessment and Risk Management in the Pest Management Regulatory Agency* (Draft). (Jan 17, 2000).

[29] Federal Commissioner for the Environment and Sustainable Development. *Annual Report.* (1999), 3.133.

framework involving 162 companies and organizations. Launched by the federal Minister of the Environment in 1993, the program's stakeholder committee chose 117 substances to be targeted for reduction from 2000 substances in the Chemical Evaluation Search and Retrieval System database (pesticides being excluded). In deciding upon the substances, "no consideration was given to quantities released, the medium of release or quantities in the environment," and the candidate lists were "not meant to imply that actual harm is currently being caused by these substances."[30] Instead toxicity was determined solely based on toxicological criteria originally developed by the Ontario Ministry of the Environment. According to ARET's third progress report though, only 500 of the 2000 substances had enough data to be considered for the list.[31]

The program is committed to a co-operative, voluntary approach they consider "faster and more effective than relying on regulations alone."[32] Although the original stakeholders committee included environmental non-governmental organizations, these groups questioned the viability of the voluntary program and eventually left over ARET's decision to focus on eliminating the release and not the use of substances. ARET follows the Toxic Substances Management Policy framework of two major tracks, with the first 30 substances being targeted for virtual elimination based on the TSMP definition of release below measurable levels. The remaining 87 substances, listed as toxic but not necessarily both persistent and bio-accumulative, are targeted for reduction below levels of harm. Eight of the substances on the present federal Toxic Substances List however, are not included in the ARET program, as well as 16 of those listed in the second Priority Substances List. ARET is currently under review and will either be changed or renewed in the near future.

6.7 THE CANADIAN COUNCIL OF MINISTERS OF THE ENVIRONMENT

The Canadian Council of Ministers of the Environment (CCME), though it does not have the authority to implement or enforce legislation, has also taken on a role in determining the safety or potential risks of substances. On January 29, 1998, all the Ministers of the Environment except Quebec's signed the Canada-Wide Accord on Environmental Harmonization and its sub-agreements, including the sub-agreement on Canada-Wide Standards. The accord created a multilateral process for screening and recommending controls for potentially toxic substances, and embraced the *CEPA* definition of the precautionary principle.[33] Though the Canada-Wide Standards sub-agreement does not alter the federal authority for managing toxic substances, it stipulates that when "a [provincial] government has accepted obligations and is discharging a role" under the agreement, the federal government will not also act in that role.[34]

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[30] Accelerated Reduction/Elimination of Toxics. *Environmental Leaders 2: Progress Report*, http://www.ec.gc.ca/aret/el2/el2covr.html.

[31] Accelerated Reduction/Elimination of Toxics. *Environment Leaders 3: Voluntary Action on Toxic Substances*, p.42 http://www.ec.gc.ca/aret/reports/aret_el3_e.pdf,

[32] Accelerated Reduction/Elimination of Toxics. *Environmental Leaders 2: Progress Report*, http://www.ec.gc.ca/aret/el2/el2covr.html.

[33] A Canada-Wide Accord on Environmental Harmonization. http://www.ccme.ca/3e_priorities/3ea_harmonization/3ea1_accord/3ea1.html

[34] Canadian Council of Ministers of the Environment. http://www.ccme.ca/3e_priorities/3ea_harmonization/3ea2_cws/3ea2a.html

The creation of Canada-Wide Standards for substances intentionally "incorporates socio-economic and technical factors" and they are intended to be "achievable targets."[35] The standards are meant to balance "the best health and environmental protection possible" with the "feasibility and costs of reducing emissions."[36] Though the CCME endorses risk assessment and management as the preferred methods for managing toxic substances, the standards are, in practice, numbers negotiated by 'stakeholders' in the process. The CCME too has incorporated the TSMP guidelines for toxic substances management. In addition, the multi-lateral approach to nominating substances proposed for the Canada-Wide Standards process includes an initial screening out of those substances that are not of "national significance" or sufficiently present in the environment.[37]

To date, Canada-Wide Standards agreements for Benzene, Mercury, Ozone and Particulate Matter have been proposed (these are discussed further in the previous chapter). They were accepted in principle by the Ministers in November of 1999 and have been taken back for ratification by their respective cabinets before the agreements will be signed. According to a senior official at Environment Canada however, the federal Environment Minister is committed to pushing for stricter standards than those now on the table.[38]

6.8 PERSISTENT ORGANIC POLLUTANTS (POPs)

Canadian plans for the control of toxic substances must also account for international agreements such as the Montreal Protocol on Ozone Depleting Substances. Likewise, in its draft risk assessment guidelines, the PMRA commits itself to making pesticide registrations compatible with international frameworks.[39]

In negotiation right now is an international treaty on Persistent Organic Pollutants (POP's). Based on 1992 Earth Summit commitments to the elimination of persistent, synthetic toxics, the Intergovernmental Forum on Chemical Safety (IFCS), agreed in 1996 on a list of 12 POP's for reduction. Endorsed by the United Nations Environment Program, meetings have taken place since 1998 to negotiate an international treaty.

The proposed document, commonly known as the proposed "Legally Binding Convention on Persistent Organic Pollutants," is expected to be completed by 2001. A fifth, and probably final, negotiating session is to be completed in South Africa in December of 2000. The proposed convention is intended to initially address 10 products and 2 by-products (namely dioxins and furans). A process is also included in the proposed convention for adding additional substances. The proposed convention focuses on persistent, bioaccumulative and toxic substances.

Article D of the proposed convention outlines the respective obligations for products and by-products and

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[35] Canadian Council of Ministers of the Environment. http://www.ccme.ca/3e_priorities/3ea_harmonization/3ea2_cws/3ea2b.html

[36] Canada-Wide Standard for Benzene. *Canada Gazette*, Part. II (Feb 5, 2000), p.321.

[37] Canadian Council for the Ministers of the Environment Policy for the Management of Toxic Substances. http://www.ccme.ca/3e_priorities/3ec_toxic/3ec1_toxic/3ec1a.html

[38] Personal communication, (April 5, 2000).

[39] Pest Management Regulatory Agency. *Risk Assessment and Risk Management in the Pest Management Regulatory Agency* (Draft). (Jan 17, 2000), p.4.

includes schedules for what substances will be eliminated or severely restricted. A number of contentious issues remain including the timeframes for eliminating some of the products, what exemptions are appropriate and whether the overall goal for by-products will be elimination or reduction.

6.9 ONTARIO

The 1986 Municipal Industrial Strategy for Abatement, or MISA, had an approach different from most toxics regulation in Canada. Based on the inherent toxicity of a substance, its persistence and its potential for bio-accumulation, MISA regulations called for reducing toxic discharges via use of the best available technology economically achievable (BATEA). Toxicity was determined using toxicological criteria by the Standards Branch of the Ministry of the Environment, and the technology standards were based on international surveys of the technology available and its costs. MISA regulations were therefore not risk-based, but intended to reduce pollution as much as possible while attempting to balance the feasibility of controls and their impact on industry. Notably, these regulations contributed to significant reduction in toxic emissions from pulp mills in Ontario.

In 1991, the Hazardous Contaminants and Water Resources Branches of the Ontario Ministry of the Environment established a list of candidate substances to be phased out or to have restrictions on use or release.[40] As with MISA, the ministry focused on persistent and bio-accumulative substances, and attempted to determine which were "the most inherently hazardous," those that "should ideally not be permitted to enter the environment." Using their toxicological scoring system, they looked at over 800 substances in the Chemical Evaluation Search and Retrieval System database, and used a cutoff score that would capture the 10-15% most hazardous. They then created a Primary List of 21 toxics and a Secondary List of 46, the Secondary List being those that were either not persistent or not bio-accumulative, or were both, but were less toxic. The inherent toxicity approach was chosen, according to the ministry's report, because of "the limited exposure information available," the extent of exposure being necessary for reasonable risk assessment determinations.[41] No activity has occurred on this list since 1994 or 1995.

In addition to the MISA regulations, the Ontario Drinking Water Objectives (ODWO's) and the Provincial Water Quality Objectives guide water quality in Ontario. The Drinking Water Objectives are guidelines for over 100 contaminants that must be met by water works to obtain Certificates of Approval. Ontario usually adopts the Canadian Drinking Water Guidelines developed by the Federal-Provincial-Territorial Sub-Committee on Drinking Water, and when it does develop independent objectives, the province generally follows the sub-committee's methods. Health Canada is responsible for the risk assessments that inform the objectives, while the provinces and territories incorporate the technical and socio-economic issues into the standard-setting process.[42]

Provincial Water Quality Objectives are driven primarily, but not exclusively, by the health of aquatic life. They are developed in co-operation with the CCME's water quality group with the intention of protecting "the most sensitive aquatic life-stage for an indefinite period of time, with an added margin of

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[40] Socha, A.C. et al. *Candidate Substances List for Bans or Phase-Outs*. Ontario Ministry of the Environment. (April 1992).

[41] *Ibid.* p.1.

[42] Ontario Ministry of the Environment , *Setting Environmental Quality Standards in Ontario: The Ministry of the Environment's Standards Plans*, undated, p.13-14.

safety."[43] The more than 240 objectives do not consider socio-economic or technical factors, and are not directly enforceable, but come in to play as the ministry decides on Certificates of Approval (for industrial facility emissions) based on the objectives.

Air pollution regulation in Ontario is built on the Ontario Ambient Air Quality Criteria (AAQC), which include standards for over 300 air pollutants. These are then used to calculate "Point of Impingement" standards under Regulation 346 of Ontario's *Environmental Protection Act* (see Chapter 5: Air). In addition, Ontario guidelines also include those for tissue residues of bio-accumulative substances, for vegetation contaminants, for lake fill quality and for sediment quality. Finally, based on Health Canada safety guidelines, Ontario publishes fish consumption guidelines in the *Guide to Eating Ontario Sport Fish*.

6.10 Conclusions

As far back as 1978, Canada agreed to the precautionary principle with respect to toxic substances when it concluded the *Great Lakes Water Quality Agreement*. The agreement, and its interpretation through the International Joint Commission, has provided the impetus for progressive policy development not only in North America, but globally as well. However, despite this early commitment, the Canadian federal government has been slow to implement an agressive toxic mangement regime.

Certainly both the Toxic Substances Management Policy and the recently enacted *Canadian Environmental Protection Act* provide some general support in principle for the goal of virtual elmination and the precautionary principle. However, at this point in time, it is unclear whether the general support will be operationalized into firm action. The historical support for voluntary initiatives (such as the ARET process) and the commitment to the *Canada-Wide Accord on Environmental Harmonization* may well undermine any legislative authority for clear action on toxic substances. The Accord in particular provides little direction for strong action, especially with the need to get support from the provinces and the federal government as a precondition to action.

The province of Ontario is not seen as a leader in issues pertaining to toxics management. Some of the initiatives, such as the Candidate List for Bans and Phase-Out, does not seem to have any currency. Moreover, its water program, MISA, was really a technology based approach to provide some regulatory foundation, although it was not seen as the exclusive program. Ontario's approach to air is discussed in Chapter 5. Its commitment to the precautionary principle is unclear at best. It also has decreased significantly its emphasis on pollution prevention and other such policies. In fact, it seems to now to rely more on the *Canada-Wide Accord* for its priority-setting.

6.11 Recommendations

1. Environment Canada should clarify what it means by "cost effective measures" when applying the precautionary principle and ensure that "cost effective" comprehensively accounts for human health costs, particularly for children, affected by exposure to toxic substances.

2. Environment Canada should commit to take regulatory action on all substances found to be toxic under the *Canadian Environmental Protection Act* and employ processes such as the Strategic

[43] *Ibid.*, p.14.

Options Process as a means to consult stakeholders on those regulatory initiatives.

3. Environment Canada should exercise its discretion under the *Canadian Environmental Protection Act* to require pollution prevention planning for all *CEPA* toxic substances up to and including establishing timetables for phase-down and phase-out of inherently toxic substances.

4. Resources and efforts should be applied to in-depth focussed research on the effects of toxic substances on vulnerable populations, particularly children. This focussed research should directly inform the assessment processes within *CEPA* as well as in Ontario processes.

5. Criteria should be established to identify as "*CEPA* toxic" or "*CEPA* –equivalent" those substances not currently subject to *CEPA* to ensure they are made subject to the Toxic Substances Management Policy.

6. The ARET (Accelerated Reduction/Elimination of Toxics) program should not be renewed until an in-depth, impartial assessment is undertaken. Unless that assessment reveals unequivocal evidence of sustainable and actual progress, toxic substances should not be dealt with through voluntary measures but through regulatory measures.

7. The Canada-Wide Standards process under the Environmental Harmonization Accord Standard-Setting Sub-Agreement should be repealed with respect to toxic substances.

8. The federal government should take a leadership role in the negotiation of the proposed Legally Binding Treaty on Persistent Organic Pollutants. In particular, Canada should support language in the treaty that calls for the elimination of both products (such as pesticides) and by-products (such as dioxins) in the proposed treaty as opposed to a mere reduction regime supported by some countries.

9. The province of Ontario should re-vitalize its list of candidate substances to be phased out or restricted, and this list should be developed using the precautionary principle.

10. The province of Ontario should enhance its policy and legal framework for pollution prevention.

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Chapter 7: Consumer Products

Chapter 7: Consumer Products

7.1 INTRODUCTION

This chapter addresses an area of exclusive federal jurisdiction: the standard-setting process governing consumer products. It describes the regulatory framework followed by Health Canada and focuses on those products of most relevance to children.. [For a more detailed review of lead in consumer products, see Section 8.4.6 of Case Study #1.] A Materials Use Policy is proposed as a means of proactively augmenting the current approach of reacting product-by-product to problems associated with hazardous substances in consumer products.

7.2 THE REGULATORY FRAMEWORK

Consumer products are regulated by Health Canada under authority of the *Hazardous Products Act.*[1] This Act controls the sale, importation and advertisement of dangerous or potentially dangerous consumer and industrial products. Such products are organized into two schedules under the Act, depending on the degree of hazard that they pose. *Prohibited* products (Schedule I, Part I) may not be advertised, sold or imported in Canada.[2] The advertisement, sale and importation of *restricted* products (Schedule I, Part II) is limited by conditions that are set out in product-specific regulations promulgated under authority of the Act.[3] Finally, *controlled* products (Schedule II) are relevant to the work place. Sections 7.3 and 7.4 discuss prohibited and restricted products respectively.

The Act does not include any general product requirements. Consequently, only those products that are included in one of the Act's two schedules are regulated, and in the case of restricted products, governance is confined to the terms of the relevant regulation. This approach greatly limits the applicability of the Act. It ensures the safety of only those individual products that have been centred out as requiring control measures via the drafting of a regulation. Because it is a product-centred approach, it is time and labour intensive. Moreover, in the few cases where more general provisions exist,[4] the applicability of the Act is not always clear, allowing for uncertainty regarding which products are required to satisfy safety stipulations, and which are not. The issue of children's products containing plastic is a case in point, as discussed in Section 7.4.1 below.

To determine the threat posed by a particular product, Health Canada undertakes a risk assessment.[5] If the assessment indicates that a consumer product poses an unacceptable risk to the public, the ministry has few available options for minimizing that risk. Specifically, Health Canada has no power to mandate product

[1] *Hazardous Products Act*, R.S.C. 1985, c. F-27.

[2] *Hazardous Products Act*, s. 4.

[3] *Ibid.*

[4] See, for example, Schedule I, Part II, paragraph 13 (p), regarding toys and other children's products that contain toxic substances.

[5] For a discussion regarding the Health Canada risk assessment for plastic mini-blinds, see section 8.4.6.4 in Case Study #1.

recalls.[6] On those occasions where a dangerous product is removed from retail shelves, it is the result of voluntary industry action. Health Canada's limited authority regarding product availability is the power, under the *Hazardous Products Act*, to seize products. This power is restricted to products that are regulated under the Act, and is primarily utilized at the point of product storage. A product seizure takes place when a provision of the *Hazardous Products Act* has been contravened. In contrast to a product recall, which would involve (if the federal government had the authority to require it) the removal of a product from store shelves, a seizure takes place at the point of product storage and is therefore of a much smaller scale than a full product recall and does not involve the removal of products from store shelves.[7]

The department's primary tool in the control of hazardous products, including both regulated and unregulated products, is the release of public advisories and warnings.[8] Advisories are issued in relation to a class of products, while warnings are specific to a particular product, of a specific brand. In those cases where an advisory or warning is deemed to be insufficient to protect the public, and industry chooses not to voluntarily recall its product, Health Canada's only option is to adopt a regulation under the *Hazardous Products Act.*

7.2.1 Product Inspection

The recent discoveries of so many unexpected and very hazardous sources of lead in consumer products (as discussed in the Lead Case Study) belies any assumptions that the public may have concerning product safety. Many people reasonably assume that if a product is on the shelf, especially if it is intended for children, that it has been tested in some way or is otherwise considered safe. However, for both regulated and non-regulated products, there is no mechanism formally in place regarding pre-market assessment. Health Canada simply doesn't have the resources to check products prior to them being made available to the public for consumption.[9]

> In the case of products that are regulated under the *Hazardous Products Act*, post-market inspection can take place when Health Canada receives complaints regarding a product. Health Canada maintains a database of such complaints which it assesses yearly for trends. An inspection can also be triggered when a Health Canada inspector believes there to be a potential risk from a product. In addition, a cyclical enforcement program is currently being developed by Health Canada. Under this program, all regulated products would be inspected at least once, and some several times, in the space of a six year period. The frequency of inspection for a given product would be dependent on a number of factors such as the risk it poses, exposure, and Health Canada's capacity to conduct investigations, among others.[10]

Products that are not regulated under the *Hazardous Products Act* are inspected on a case-by-case basis in

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[6] Unlike the *Hazardous Products Act*, both the *Food and Drug Act* and the *Radiation Emitting Devices Act* provide for product recalls however even under those statutes, the process is largely voluntary and industry-driven. For information on Product Recall Procedures under these two laws, see: www.hc-sc.gc.ca/hpb-dgps/therapeut/zfiles/english/crisis/recall_e.html

[7] Personal Communication, Andy Teliszewsky, Project Officer, Product Safety Bureau, Health Canada, May 19, 1999.

[8] Under authority of the *Department of Health Act*, R.S.C. 1996, c. H-3.2.

[9] Personal communication, Jonathan Williams, Product Safety Officer, Health Canada. (May 6, 1999).

[10] Personal communication, Greg Whalen, Product Safety Officer, Health Canada. (May 5, 1999)

response to complaints, or irregularities or potential dangers that are perceived by inspectors. The inspection process may involve a risk assessment (if Health Canada is not already aware of the risk involved), and a consideration of potential risk management actions. Options available to Health Canada include the issuance of a warning to the public via an advisory, pressuring industry to respond to the safety problem, and ultimately, the adoption of a regulation.[11]

7.3 PROHIBITED PRODUCTS

Products that are prohibited under the *Hazardous Products Act* and that are directly relevant to children include:

- furniture and other articles, intended for children, painted with a liquid coating material containing lead compounds of which the lead content is in excess of 0.50 per cent of the total weight of the contained solids, including pigments, film solids and driers;[12]
- toys, equipment and other products for use by a child in learning or play that:
  - have applied to them a decorative or protective coating that contains lead, antimony, arsenic, cadmium, selenium, barium or mercury;[13]
  - in whole or in part are made of, or impregnated with celluloid or cellulose nitrate;[14] or
  - contain a number of substances[15] that, under reasonably foreseeable circumstances, could become accessible to a child;[16]
- pencils and artists' brushes that have applied to them a decorative or protective coating that, when dry, contains more than 0.5 per cent weight to weight of lead;[17]
- among others.[18]

Note that a level of 0.5 per cent to weight of lead is equivalent to 5000 parts per million. As discussed in more detail in Case Study #1, this level was established in the early 1970s, based on health effect information that is now woefully out of date. A child exposed to dust or paint chips containing 5000 parts per million of lead would be in danger of serious lead poisoning. Further, the "prohibitions" in this section of the Act are mostly just a restatement of the restrictions, for example on lead content in paints and other coatings, noted in the regulations on restricted products discussed below.

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[11] Personal communication, Greg Whalen, Product Safety Officer, Health Canada. (May 5, 1999)

[12] *Hazardous Products Act*, *supra* note 1, Schedule I, Part I, s. 2.

[13] *Ibid.*, s. 9.

[14] *Ibid.*, s. 7.

[15] carbon tetrachloride, methyl alcohol, petroleum distillates, benzene, turpentine, boric salts, salts of boric acid or ethyl ether.

[16] *Hazardous Products Act,* Schedule I, Part I, s. 8.

[17] *Hazardous Products Act,* s. 18.

[18] See Schedule I, Part I, ss. 5, 10, 11, 13, 14, 15, 20, 21, 27, 28, 35.

7.4 RESTRICTED PRODUCTS

The *Hazardous Products Act* allows for the establishment of restricted products and the setting of regulations governing these products. Several of these regulations are relevant to children.

Toys, equipment and other products for use by a child in learning or play that contain a toxic substance are restricted products under paragraph 13 (p) of Schedule I, Part II of the *Hazardous Products Act*. This section includes all toxic substances that are not mentioned elsewhere in the Act, including heavy metals such as lead.[19] Under the *Hazardous Products (Toys) Regulations*,[20] (discussed further below), every product described in paragraph 13 (p) must meet at least one of three requirements:

> a) the product, by reason of its nature, physical form, size or any other characteristic, shall be such that the toxic substance or the substance or part containing the toxic substance cannot be ingested, inhaled or absorbed through the skin;
>
> b) the total quantity of the available toxic substance shall not exceed one-hundredth of the acute oral or dermal median lethal dose, whichever is the lesser, calculated for a child having a body weight of 10 kg; or
>
> (c) the toxicity of the toxic substance does not exceed the limits prescribed by Schedule I:
> A substance shall be considered excessively toxic for humans if:
>
> (a) the acute oral LD50 value for rat is 5 grms or less per kilogram body weight;
> (b) the acute dermal LD50 value for rabbit is 2 grams or less per kilogram body weight; and
> (c) where gas, vapour, mist or dust is likely to be encountered when the substance is used in any reasonably foreseeable manner, the LC50 value for a one-hour exposure determined using rats, is 20,000 parts per million by volume of gas or vapour or less, or 200 milligrams per litre by volume of mist or dust or less.

These Toy Regulation requirements also apply to playpens, carriages and strollers by authority of the *Playpens Regulations*[21] and the *Carriages and Strollers Regulations*.[22] Each of these regulations is further discussed in turn below in Sections 7.4.2 through 7.4.10.

First however, it should be noted that the general provisions suffer from a number of important shortcomings including the fact that it is not at all clear which products are included within paragraph 13 (p). Moreover, the toxicity tests that form the basis for regulatory action are difficult to decipher and are based on complicated laboratory data that are not readily available. It is difficult to determine whether a particular children's product is regulated under this section of the *Hazardous Products Act and Regulations*, and if so, whether it is in compliance with the regulatory stipulations.

The situation with respect to children's products made of or containing plastics is even more obscure. While this matter is also discussed in Case Study #1, it bears repeating in this context since plastic is a component of so many children's products, not just toys, and it appears to be very nearly unregulated by

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[19] Personal Communication, Jonathan Williams, Product Safety Officer, Health Canada, May 11, 1999.

[20] *Hazardous Products (Toys) Regulations*, C.R.C., c. 931.

[21] *Playpens Regulations*, C.R.C., c. 932.

[22] SOR/85-379.

Health Canada.

7.4.1 Children's Products Containing Plastics

Toys, equipment and other products for use by a child in learning or play that are or are likely to be used by a child of less than three years of age and which are made of or contain any plastic material are regulated under Paragraph 13 (r) of Schedule I, Part II. The *Hazardous Products (Toys) Regulations*, mandate that the use of resins, plasticizers, antioxidants, dyes, pigment and other substances in the manufacture of any plastic material found in these products be limited by those regulations that govern the manufacture of food packaging material and food containers. Toxic substances such as lead are routinely added to plastic toys to serve the above listed functions. *The Food and Drug Regulations*, under the *Food and Drugs Act*, stipulate that food may not be sold in a package that may yield to its contents any substance that may be injurious to the health of a consumer of the food. It is far from clear whether or how this standard for food packaging is applied to the plastics found in children's toys.

When the mini-blinds discovery was made Greenpeace and others had long been warning about the other hazards of polyvinyl chloride (PVC) plastics, most notably the fact that they create deadly dioxin when they are burned, for example in a garbage incinerator. The discovery of lead in mini-blinds made Greenpeace suspect similar contamination in children's products made with PVC plastics. In 1997, Greenpeace investigated the lead and cadmium content of a range of plastic children's products. During the course of this report's investigation, after several conversations with officials from Health Canada and Justice Canada, it was finally apparent that plastic children's products that are, or are likely to be used, by children of three years of age or older, are not regulated at all under (either) the *Hazardous Products Act*.[23] (or the *Food and Drugs Act*.)

Greenpeace revealed alarmingly high levels of both lead and cadmium in a variety of plastic children's products that were readily available and commonly used across Canada and the US.[24] Products tested include plastic backpacks, rain clothes and toy cables. While Health Canada has proposed, as a guideline, a maximum total lead content in children's products of 15 ppm,[25] lead levels as high as 18,750 ppm were found in the products tested. Further tests into the level of extractable lead in these products, as well as the release of lead-containing dust from the products revealed exceedances of the daily ingestion limits set by the European Union (0.7 µgrams) and the U.S. Consumer Product Safety Commission (15 µgrams). High levels of lead and cadmium in children's products were confirmed by Greenpeace in a further, 1998 study.[26]

Greenpeace has also revealed similarly dangerous levels of toxins called phthalates in these products. These plastic additives leach out of products when sucked or chewed by children. The specific chemical, diethylhexyl phthalate (DEHP – the plasticizer in pacifiers, nipples, teethers and flexible toys) was

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[23] Personal communication with Louise McGuier-Wellington, Justice Canada, May 18, 1999.

[24] Di Gangi, J., 1997. *Lead and Cadmium in Vinyl Children's Products: A Greenpeace Exposé*; Greenpeace Canada Briefing, Oct. 9, 1997, Vinyl Children's Products Pose Lead and Cadmium Hazard.

[25] Health Canada, *Strategy for Reducing Lead in Children's and Other Consumer Products*, Discussion Paper, Draft II, August, 1997.

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

assessed in the early 1990s under the *Canadian Environmental Protection Act* Priority Substances List process[27] and was found to be "*CEPA*-toxic" (see discussion of *CEPA* in Chapter 6). The only action taken since then by the federal government has been to suggest further research. There is no regulatory limit for phthalate levels in children's plastic products in Canada.

7.4.2 The Toys Regulations

Under the *Hazardous Products (Toys) Regulations*,[28] toys that contain a toxic substance[29] must meet at least one of the following requirements:

> a) it cannot be possible for the toxic substance or the part of the product containing the toxic substance to be ingested, inhaled or absorbed through the skin;
>
> b) the total quantity of available toxic substance cannot exceed one one-hundredth the acute oral or dermal median lethal dose (whichever is less), as calculated for a child with a body weight of 10 kg; or
>
> c) the toxicity of the toxic substance cannot be greater than the permissible toxicity limits set in Schedule 1 of the Regulation.[30]

Toys that contain a corrosive substance, irritant or sensitizer must meet one of the following requirements:

> a) the corrosive substance, irritant or sensitizer cannot come into contact with the skin, or
>
> b) the corrosive substance, irritant or sensitizer cannot be excessively corrosive or irritant or an excessively strong sensitizer as determined by tests set out in Schedule II of the Regulation.[31]

The Regulation also requires that the grade, quality, quantity and proportions of resins, plasticisers, antioxidants, dyes, pigments and other substances that are used in the manufacture of any plastic material that is in turn used in products that are, or are likely to be used by a child of less than three years of age satisfy the following requirements:

> a) the grade, quality, quantity and proportions of resins, plasticisers, antioxidants, dyes, pigments and other substances that will be permitted shall be those considered acceptable for use in the manufacture of food packaging materials and food containers; and
>
> b) substances other than heavy metals, heavy metal compounds, carbon tetrachloride, methyl alcohol, petroleum distillates, benzene, turpentine, boric acid, ethyl ether, and decorative or protective coatings containing lead, antimony, arsenic, cadmium, selenium, mercury or barium (in

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[27] Government of Canada, Environment Canada, Health Canada, *Canadian Environmental Protection Act: Priority Substances List Assessment Report, bis(2-ethylhexyl) phthalate*, 44 p. undated.

[28] *Hazardous Products (Toys) Regulations*, C.R.C., c. 931.

[29] other than a toxic substance named in item 8 of Part I of Schedule I of the Hazardous Products Act: carbon tetrachloride, methyl alcohol, petroleum distillates, benzene, turpentine, boric acid and ethyl ether.

[30] *Hazardous Products (Toys) Regulations*, C.R.C., c. 931, s. 10.

[31] *Ibid.*, s. 11.

accordance with section 9, Part 1, Schedule 1 stipulations) may be present in plastic materials in the amount of one percent or less.[32]

7.4.3 The Pacifiers Regulations

The *Hazardous Products (Pacifiers) Regulations*[33] stipulate that no product, or part or component of a product shall contain more than 10 micrograms/kg total volatile N-nitrosamines. It further requires that pacifiers meet the requirements of section 10 of the *Hazardous Products (Toys) Regulations.*

7.4.4 The Infant Bottle Nipples Regulations

Under authority of the *Hazardous Products (Infant Feeding Bottle Nipples) Regulations,*[34] infant bottle nipples that may be advertised, sold and imported into Canada are limited to those products and parts that do not contain greater than 10 micrograms/kg total volatile N-nitrosamines.

7.4.5 The Glazed Ceramics and Glassware Regulations

The *Hazardous Products (Glazed Ceramics and Glassware) Regulations*[35] establish cadmium and lead leachability limits for products completely or partially covered with a coating/glaze decoration containing cadmium or lead. This regulation requires that products that are not intended for food use, but that release lead or cadmium in excess of the leachability limits be identified as unsuitable for food use either through the use of a design feature or through the display of a warning.

7.4.6 The Children's Sleepwear Regulations

The *Hazardous Products (Children's Sleepwear) Regulations*[36] stipulate that no product that is treated with a flame retardant, and no component extracted or broken down from the treated product, and no flame retardant used to treat the product shall cause:

a) death as a result of oral exposure to a dose of 500 mg/kg body weight or less;
b) death as a result of dermal exposure to a dose of 1000 mg/kg body weight or less;
c) redness or swelling greater than established limits;
d) genetic mutation or chromosomal aberration; or
e) tumors.

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[32] *Ibid.*, s. 12.

[33] *Hazardous Products (Pacifiers) Regulations*, C.R.C., c. 930.

[34] *Hazardous Products (Infant Feeding Bottle Nipples) Regulations*, SOR/84-271.

[35] *Hazardous Products (Glazed Ceramics and Glassware) Regulations*, SOR/98-176.

[36] *Hazardous Products (Children's Sleepwear) Regulations*, SOR/87-443.

7.4.7 The Cribs and Cradles Regulations

The *Cribs and Cradles Regulations*[37] prohibit the advertisement, sale or importation into Canada of cribs or cradles that have applied to them a decorative or protective coating that contains:

a) lead pigments;
b) more that 0.5 per cent weight to weight of lead in the total solids contained in such a coating;
c) any compound of antimony, arsenic, cadmium, selenium or barium if more than one-tenth of one percent of such compound dissolves in five per cent hydrochloric acid after stirring for ten minutes at twenty degrees Celsius; or
d) any compound of mercury introduced as such.

7.4.8 The Carriages and Strollers Regulations

Under authority of the *Carriages and Strollers Regulations*,[38] these products must meet the requirements of section 10 of the *Hazardous Products (Toys) Regulations.* In addition, they may not contain any of the substances referred to in items 8 or 9 of Part I of Schedule I of the Act.[39]

7.4.9 The Playpens Regulations

The *Playpens Regulations*[40] dictate that all playpens must comply with paragraphs 10 (a) to (c) of the *Hazardous Products (Toys) Regulations.*

7.4.10 The Liquid Coating Materials (Paint) Regulations

The *Hazardous Products (Liquid Coating Materials) Regulations*[41] stipulate that paints, enamels and other liquid coating materials that contain more than 0.5 per cent weight to weight lead may only be advertised, sold or imported into Canada when:

- they are for use on the exterior surface of a building and are labeled according to stipulations set out in the regulation;
- they are for use on an interior or exterior surface, or on furniture for use in any industrial or commercial premises or any other premises not ordinarily used or frequented or likely

[37] *Cribs and Cradles Regulations*, SOR/86-962.

[38] *Carriages and Strollers Regulations*, SOR/85-379.

[39] Several examples of such substances include carbon tetrachloride, methyl alcohol, petroleum distillates and benzene. See the statue for a complete listing.

[40] *Playpens Regulations*, C.R.C., c. 932.

[41] *Hazardous Products (Liquid Coating Materials) Regulations*, C.R.C., c. 928.

to be used or frequented by children, as long as they are labeled according to regulation stipulations.

7.5 CONCLUSIONS

The answer to the question as to whether standards for consumer products are intentionally protective of children is a very qualified yes and limited to only those products for which regulations have been established in reaction to identified problems. But, for children's products containing plastic the answer is unclear and probably no. For lead in consumer products the answer is decidedly no. The matter of Health Canada's response to lead in consumer products is discussed more fully in Case Study #1. As discussed therein, while the regulation of lead in consumer products in Canada is a sorry tale, there are hopeful signs that this situation may be about to change, if international trade agreements and industry opposition do not undermine the preventative efforts currently proposed.

Only a qualified yes is possible in response to the question since the regulatory framework is an entirely reactive one. Only once problems or poisonings have been identified have regulations been established, after the fact, to be intentionally protective of children.

Like the chemical-by-chemical approach to regulating toxic chemicals or pesticides, the *Hazardous Products Act* only regulates those individual products for which problems have indicated the need for control measures via the drafting of a regulation. As a product-centred approach, it is time and labour intensive. Where a few more general provisions exist the applicability of the Act is not always clear. It becomes uncertain which products are required to satisfy safety stipulations, and which are not. Children's products containing plastic are a case in point.

Although many people might assume that if a product is on the shelf, especially if it is intended for children, that it has been tested in some way or is otherwise considered safe. No such pre-market assessment occurs for either regulated or non-regulated products. Some case-by-case inspection is done in response to complaints or irregularities or potential dangers that are perceived by inspectors.

When risks are identified, Health Canada has few options. Specifically, Health Canada has no power to mandate product recalls, it has limited power to seize products and in both cases it must rely on voluntary action by industry to remove dangerous products from retail shelves. The department's primary tool in the control of hazardous products, including both regulated and unregulated products, is the release of public advisories and warnings. Thereafter, Health Canada's only option is to adopt a regulation under the *Hazardous Products Act.*

The Act's provisions regarding the ability to set regulations for restricted products suffer from a number of important shortcomings. It is unclear which products can be included. The toxicity tests that underlie regulatory action are unclear and based on complicated laboratory data that are not readily available. It is difficult to determine whether a particular children's product is regulated under this section of the Act, and if so, whether it is in compliance with the regulatory stipulations. The situation with respect to children's products made of or containing plastics is even more obscure but appears to be very nearly unregulated by Health Canada. Further, despite the fact that the phthalate plasticizer in children's plastic products was determined more than 6 years ago to be "*CEPA*-toxic" (as defined by the *Canadian Environmental Protection Act)*, this determination has not resulted in regulatory action to control or eliminate this chemical in children's products.

The reactive product-by-product nature of the *Hazardous Products Act* provides an important but very

limited tool for ensuring that consumer products are safe for children. Clearly, there is an enormous range of materials contained within consumer products and with which children come in contact on a daily basis. A Materials Use Policy would be a more proactive public policy measure that could work towards ensuring the safety of consumer products in a precautionary manner. Such a policy would require that consumer products be manufactured with materials that are inherently safe and would give manufacturers an incentive to search for safe materials and to begin and/or phase-in product substitution. Under such an approach, companion regulations are necessary as technology-forcing measures that serve to phase-down and phase-out the use of inherently toxic and hazardous materials in products with which children come in contact. Along the line of the precautionary principle and pollution prevention recommendations made in previous chapters, such regulations need to encourage as well as force the phase-down and phase-out in consumer products of persistent, bioaccumulative and inherently toxic substances such as heavy metals, asbestos, endocrine disruptors, phthalates and chlorinated hydrocarbons.

7.6 RECOMMENDATIONS

Note that additional recommendations with respect to consumer products are included in Case Study #1 with respect to the regulation of lead.

1. The *Hazardous Products Act* should be amended to provide Health Canada with the power to issue mandatory consumer product recalls.

2. Health Canada should develop a proactive Materials Use Policy that incorporates a precautionary and preventative approach to avoiding the use of toxic substances in consumer products.

3. An area for further research beyond the scope of the present study should include a review of the child-specific *Hazardous Products Act* regulations reviewed herein to determine whether they were developed in a precautionary manner or in reaction to identified hazardous or lethal situations.

4. Further research is necessary to investigate the impact of international trade agreements on both the ability and inclination of Canadian regulatory agencies to set child-protective domestic regulations.

7.7 REFERENCES CITED

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Greenpeace Media Release, Nov. 16, 1998. Leading child health and environmental organizations urge removal of hazardous vinyl children's products from sale.

Health Canada, *Strategy for Reducing Lead in Children's and Other Consumer Products*, Discussion Paper, Draft II, August, 1997.

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

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

8.1 INTRODUCTION

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

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

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

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

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

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

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

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

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

8.2 EXPOSURE

8.2.1 Uses, Sources, Media and Routes of Exposure

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

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

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

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

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

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

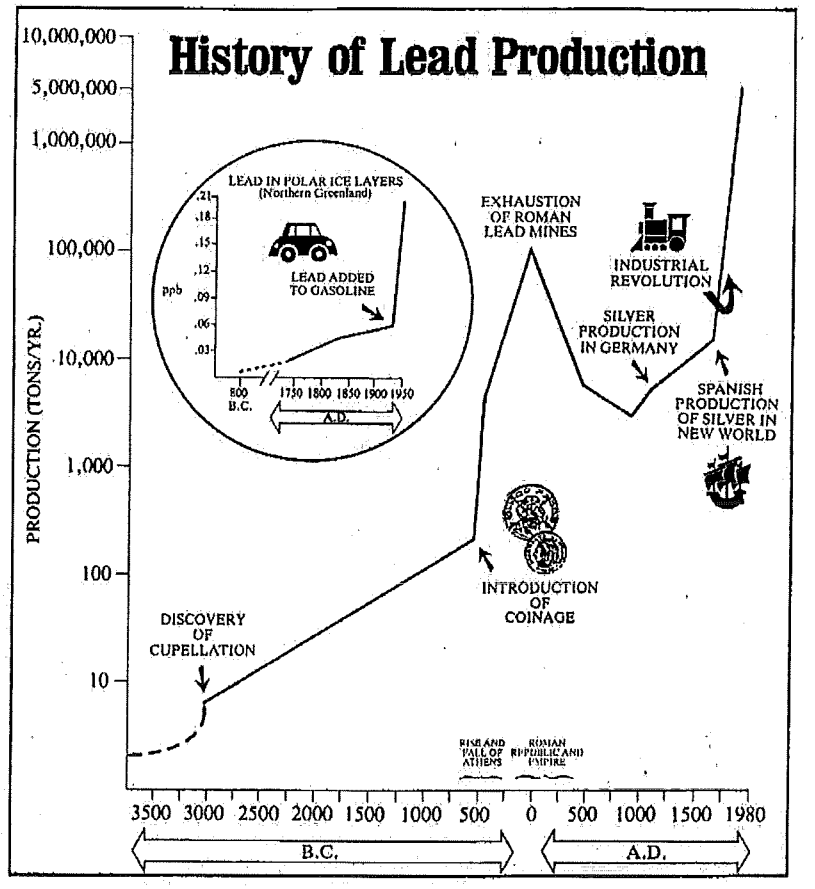

Figure 8.1. History of Lead Production.
Estimated tons of lead produced per year worldwide since about 3000 B.C. The present level is about 5 million tons per year. Note that the scale for tons per year is logarithmic (each unit of increase is ten times larger then the preceding unit of increase). The insert shows measurements of lead in parts per billion in polar ice layers since about 1750. (Source: Seattle, D.M. *et.al.*, 1980, *op.cit.*, and Murozumi, *et.al.*, 1969, *op.cit.* reproduced from Wallace and Cooper, 1986, *op.cit.*)

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

[4] See, for example, Centers for Disease Contro. *Preventing Lead Poisoning in Young Children.* United States Department of Health and Human Services. (1991)

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

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

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

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

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

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

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

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

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

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

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

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

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

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

8.2.2 Blood-Lead Surveys in Canadian Children

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

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

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

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

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

8.2.3 Risk Factors for Children

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

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

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

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

[15] Personal communication with Dr. Lesbia Smith, Ontario Ministry of Health. (May, 1999)

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

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

Nutritional status also significantly affects lead uptake and toxicity. Mushak and Crocetti[18] have reported on the large body of literature showing that deficiencies or alterations in essential nutrients like calcium, iron, phosphorus and zinc will enhance lead exposure and increase the degree of lead toxicity associated with such exposure. These investigators have further reported[19] on the socio-economic and demographic status of those most affected. In the United States, low income African-American children and African-American women of child-bearing age residing in densely populated urban areas have the highest elevations of blood-lead levels. Black male children are especially impacted. The study also reports that nutritional status in these population groups is sub-optimal particularly for calcium and iron. Both of these nutrients have a strong, inverse interactive effect on lead absorption and toxicity.
In Ontario, the first major study to determine blood lead levels and risk factors in Ontario children (conducted in 1984)[20] indicated that children who were younger, male, and from families of lower socio-economic status had higher blood lead levels. Higher levels were also strongly associated with several features of children's homes (apartment dwelling, fireplace, radiator heating systems, lack of an air filter, linoleum flooring and peeling paint in children's rooms or play areas) and neighbourhoods, (local traffic density, proximity to gasoline stations and industry) as well as local environmental levels (air and soil) of lead. The majority of these risk factors reflected the presence of lead in gasoline. The contribution of diet (and nutritional status) to childhood lead intake was not adequately addressed in this study, although it is a recognized risk factor.

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

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

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

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

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

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

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

8.3 HEALTH CONCERNS

8.3.1 Introduction

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

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

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

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

8.3.2 Lowering the "intervention" level

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

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

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

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

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

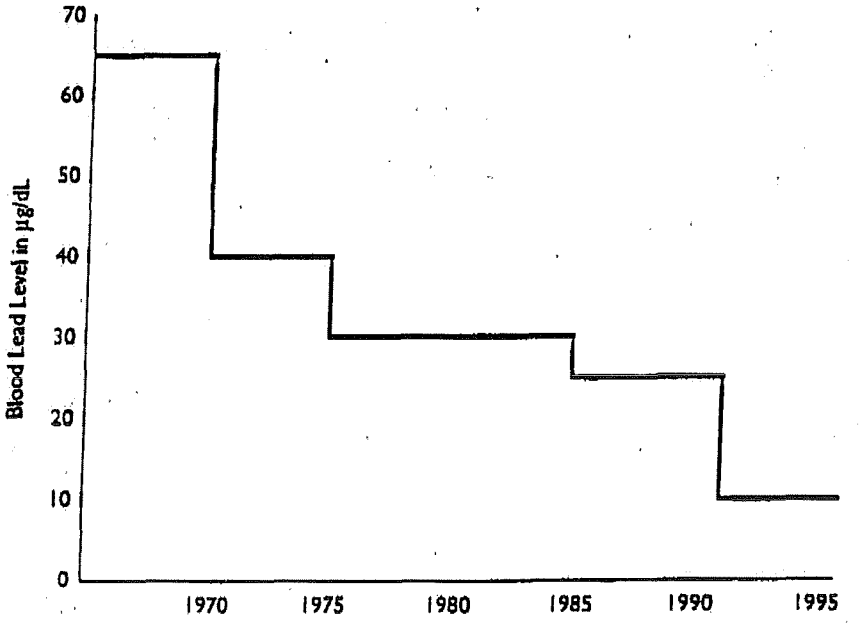

Figure 8.2. Lead and Public Health.
(Source: adapted from Centers for Disease Control, 1991, *op.cit.*, and Millstone, 1997, *op.cit.*)

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

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

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

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

[29] A substance in red blood cells that increases as blood lead increases.

of scientific literature. Lead is the most extensively studied pollutant. As Millstone[30] points out, debate during the last 30 years has not been about whether lead is poisonous but about:
* the levels at which adverse effects can be detected;
* whether or not there are thresholds below which those effects cease to occur; and
* whether or not the results of over-exposure persist or are merely transient.

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

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

8.3.3 A Systemic Poison

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

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

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

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

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[30] Millstone, Eric. *Lead and Public Health.* (Earthscan Publications Ltd, London, 1997), p.5.

[31] Calculated on the basis of an exposure level of 3.7 μg/kg/day for a 13 kg child over 14 days.

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

[33] *Ibid*, Sections 12 and 13, and Figure 13-4, (1986), pp. 13-31.

reduced gestational age, lower birth weight and smaller head circumference.[34] Lead has also been shown

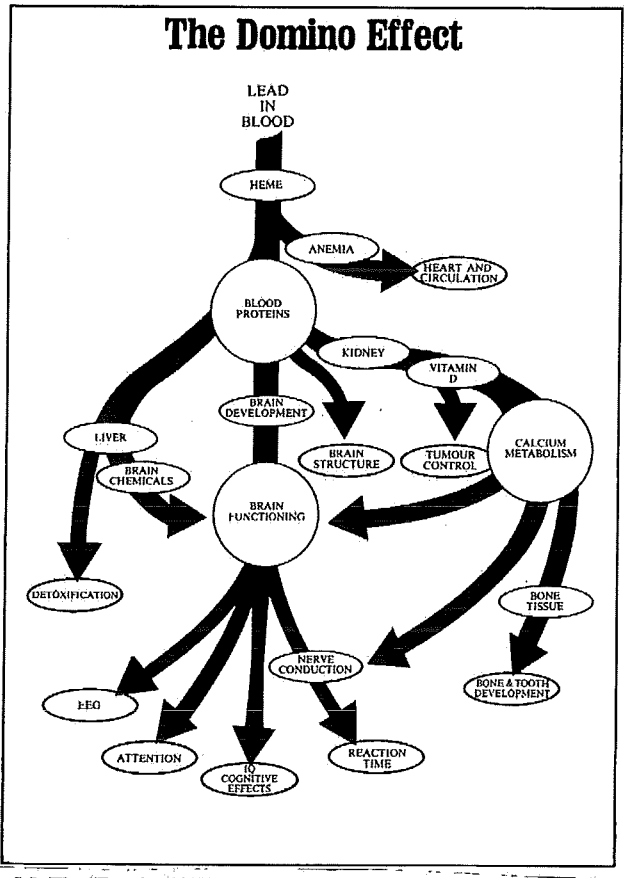

Figure 8.3. The "Domino" Effect.
The many impacts of lead-caused reductions in heme.
(Source: Adapted from U.S. Environmental Protection Agency, 1984, *op.cit.*, and reproduced from Wallace and Cooper, 1986, *op.cit.*)

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

to contribute to reduced stature in children[35] and to negatively affect children's hearing.[36]

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

8.3.4 Approaches to Studying the Neurotoxicology of Lead

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

Six different approaches to studying neurotoxic effects that have been used (roughly in temporal sequence) are:
1. clinical studies of children with high lead levels;
2. studies of "mentally retarded" or behaviourally deviant children;
3. chelation studies;
4. smelter studies;
5. general population cross-sectional studies; and
6. general population prospective/longitudinal studies.[39]

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

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

[37] Millstone, Eric. (1997) *op. cit.* Chapter 3.

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

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

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

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

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

8.3.4.1 Cross-sectional studies and meta-analysis [43]

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

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

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

[42] Millstone (*1997), op. cit.*, p. 37.

[43] As reviewed in Millstone (*1997), op. cit.*

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

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

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

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

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

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

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

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

[46] Fletcher, R.H., *et.al. Clinical Epidemiology: the essentials.* (Williams and Wilkins, Baltimore, 1988)

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

[48] World Health Organization's International Programme on Chemical Safety. *Inorganic Lead.* Environmental Health Criteria 165. (Geneva, 1995)

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

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

8.3.4.2 Prospective and Longitudinal Studies

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

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[49] Needleman and Gatsonis (1990), *op. cit.*

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

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

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

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

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

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

8.3.5 Lead and Behaviour

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

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

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

[55] World Health Organization's International Programme on Chemical Safety. *Inorganic Lead*, Environmental Health Criteria 165. (Geneva, 1995)

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

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

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

8.3.6 Summary

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

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

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

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

On a population basis, Needleman and Bellinger[62] postulate that there is probably an overall downward

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

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

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

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

[62] Needleman, H. and D. Bellinger. The Health Effects of Low Level Exposure to Lead. *Annu. Rev. Publ.*

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

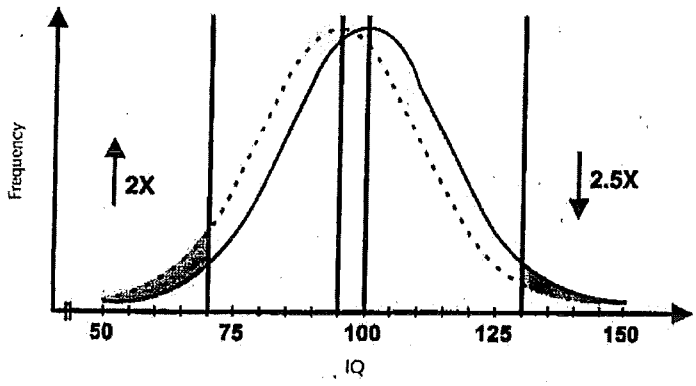

Figure 8.4. Normal Distribution of IQ.
Normal Distribution of IQ with a mean of 100 (solid curve) and decreased by five points (dashed curve). The filled area on the right tail of the distribution represents a decrease in the number of individuals with IQ's greater than 130 by a factor of 2.5, while the filled area on the left tail represents a doubling of the number of persons with IQ's less than 70 (defined clinically as mental retardation).
(Source: Rice, D.C. Issues in Development Neurotoxicology: Interpretation and Implications of the Data, *CJPH.* 89 (Suppl 1) (1998) 531-536, as adapted from Weiss, B. Risk Assessment: the Insidious Nature of Neurotoxicity and the Aging Brain. *Neurotoxicol.* (1990) 11-305.)

8.4 THE REGULATORY RESPONSE

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

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

[63] See detailed critique in Millstone, E. (1997), *Lead and Public Health, op.cit.*

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

8.4.1 Lead in Gasoline

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

[68] As quoted in Nriagu (1998), *op.cit.*

[69] As quoted in Rosner and Markowitz (1985), *op.cit.* p.350.

[70] By Nriagu (1998), *op.cit.*

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

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

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

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

Patterson's data were from his geochemical studies that revealed body burdens of lead in Americans to be at least 100 times above natural background values. By "showing the data" Patterson undermined the foundation of Kehoe's entire theoretical construct concerning the threshold of lead exposure and lead toxicity. Kehoe had determined from his occupational exposure studies that "safe" workplace levels for lead in air were 100 μg/m3. He then concluded that, since ambient air lead concentrations were about 1μg/m3, exposure to the public was 100 times lower than his scientifically derived "safe" level. However, when Patterson compared ambient levels (in the U.S.) to his biochemical data (of lead levels in remote locations indicative of actual background atmospheric lead levels), he showed that human activity was responsible for a 2000-fold increase in ambient levels. Patterson concluded that the average U.S. resident was being subjected to severe chronic lead insult.

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

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

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

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

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

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

8.4.1.1 Regulation of Lead in Gasoline in the United States

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

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

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

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

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

calculations and reached similarly categorical conclusions about the association between blood lead and gasoline lead.[75]

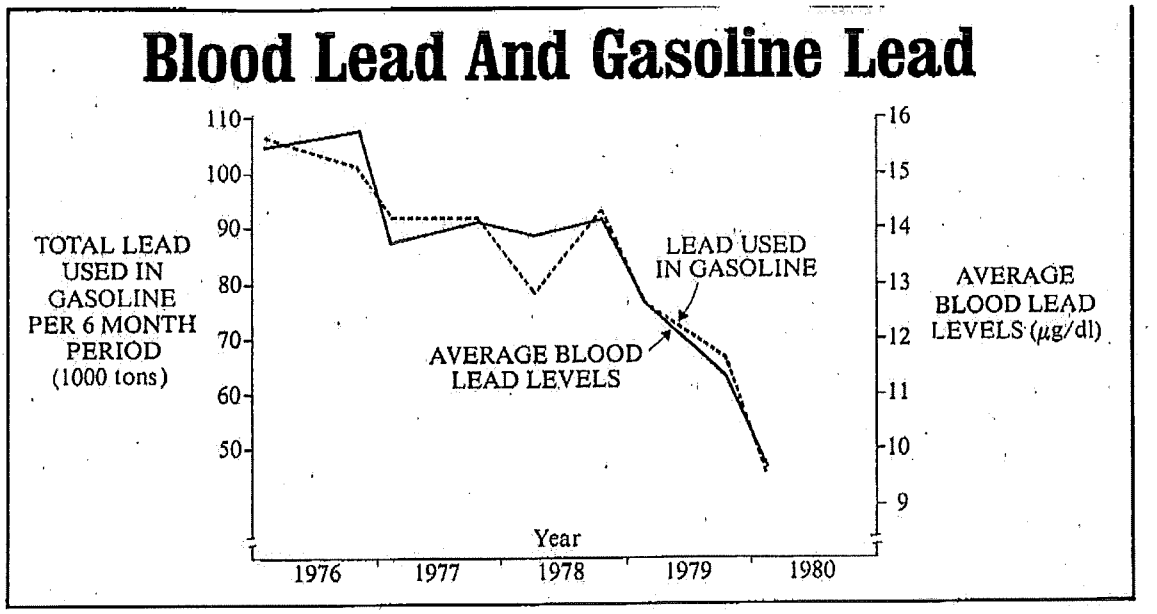

Figure 8.5. Blood Lead and Gasoline Lead.
Trends in the average blood lead levels of people living in the United States (February 1976 – February 1980) and lead used in gasoline production. Note: One ton equals about 0.98 metric tonnes.
(Source: Adapted from U.S. Environmental Protection Agency, 1985, *op.cit.* and reproduced from Wallace and Cooper, 1986, *op.cit.*)

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

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

[75] Ibid.

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

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

[78] U.S. Environmental Protection Agency. *Air Quality Criteria for Lead, Volumes I - IV*. Environmental Criteria

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

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

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

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

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

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

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

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

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

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

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

8.4.1.2 Regulation of Lead in Gasoline in Canada

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

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

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

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

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

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[84] Wallace, B and K.Cooper. *The Citizen's Guide to Lead: Uncovering a Hidden Health Hazard.* (NC Press, Toronto, 1986), p. 110.

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

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

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

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

> *We find that the report ignores many important scientific findings, fails to recognize the significance of the accumulated toxicological data, and adopts a policy of placing the burden of proof of adverse effects on Canadian citizens themselves. It shunts aside the most compelling priority, prevention. Instead of seeking to protect public health by recommending adequate safety margins, the report indicates that lead toxicity is not a significant problem until clinically overt disease becomes evident. To those of us who are familiar with the signs, symptoms and clinical course of lead toxicity in both children and adults, the Commission's archaic position yields a policy that irreversible consequences are the basis upon which exposure standards are erected. The Commission, however, should be guided by the knowledge that, once children develop overt symptoms, irreversible damage to the central nervous system is probably already present.*
>
> *Statements in the document, that adverse health effects of lead have not yet been established at blood lead values between 30-50 μg/dL and below 30 μg/dL, are applied to areas where substantial evidence of adverse health effects have been demonstrated in humans. This thesis, developed by the Commission, fails to reflect the current judgement of the scientific community; and, even where appropriate, the Commission suggests that evidence of a clearly recognized health hazard to children and the fetus should not be translated into policy, if the evidence is not beyond any "shadow of a doubt."*
>
> *The Commission's report seems to us a disservice to Canadian citizens and a retreat from informed regulation. Those of us who participated in the review process perceive insufficient evidence that our efforts were weighed seriously, thoughtfully, and impartially. It appears that we were used to provide a similacrum of objectivity.*

Similarly marked differences between the two countries are apparent in discussions of blood lead levels. An average blood-lead level in Ontario's child population of 10 μg/dL was described in 1984 by Ontario government agencies as "relatively low"[89] while in the U.S. such a level formed part of the basis for

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

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

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

justifying the need to "reduce the amount of lead in gasoline as quickly as possible."[90]

The Royal Society's Lead Commission interim report on lead in gasoline endorsed the Canadian government's go-slow position to reduce gasoline lead from 0.77 g/L to 0.29 g/L in 1987 concluding that:

> *The reductions in blood-lead levels achieved by these 1987 regulations will, in the opinion of the Commissioners, be sufficient to protect almost all segments of the Canadian population against the known harmful effects of lead exposure. There may be exceptions in certain urban hotspots, and among the industrially-exposed labour force.*"[91] (emphasis added)

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

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

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

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

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

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

[91] Royal Society of Canada's Commission on Lead in the Environment. *Lead in Gasoline: A Review of the Canadian Policy Issue*, (1985), p.xiv.

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

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

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

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

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

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

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

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

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

[96] *Ibid.* This estimate assumed a mean blood-lead level of 7 - 8 μg/dL, a geometric standard deviation of 1.42 - from the Ontario Blood-Lead Survey of 1984, in a log normal distribution.

[97] Personal communication with K.Cooper, Canadian Environmental Law Association. (October 1987)

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

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

8.4.2 Smelters and Soil

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

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

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

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

Independent testing by University of Toronto scientists confirmed excessive localized contamination

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

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

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

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

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

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

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

8.4.3 Ontario's Multi-Media Approach

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

In 1993 the Ministry published its rationale[101] for revising these three criteria and in 1994 published the

[101] Ontario Ministry of Environment and Energy. *Rationale for the Development of Soil, Drinking Water, and Air*

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

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

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

To determine soil, drinking water and air limits, the multi-media approach was structured to consider all potential sources of exposure simultaneously. The four media or exposure pathways considered were food, water, soil and air. Consumer products were not included in the analysis - a potentially significant shortcoming (see Section 8.4.6 below). The proposed limits for soil, drinking water and air (food limits are not within provincial jurisdiction) were health-based criteria derived using calculations that took into account the lead exposure that could be expected from each of the four media and the particular susceptibilities of children. For example, the proposals recommended revising the soil removal guideline (for decommissioning or clean-up of contaminated lands to a standard considered acceptable for residential redevelopment) from 500 ppm to 200 ppm. The lower level was derived by making several calculations:
1) an allocation factor of 64% (calculated as the amount of a child's lead intake that can be generally attributable to ingestion of soil or dust, based on typical soil lead concentrations of 150 ppm and house-dust lead concentrations of 200 ppm and considering child-specific lead absorption rates);
2) a soil/dust consumption rate of 80 mg/day;
3) assuming an average body weight of 13 kg (average weight of a child in the relevant age range); and
4) applying the above three calculations to the IOC_{pop} of 1.85 μg/kg/day (noted above).

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

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

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

[102] Ontario Ministry of Environment and Energy. Scientific Criteria Document for Multimedia Environmental Standards Development - Lead. (March 1994)

existing ODWO for lead was estimated to result in an approximate increase of lead exposure of 11% over the limit defined by the IOC_{pop}. It was also concluded that the practice of flushing standing water from pipes fairly consistently ensures that drinking water lead levels are below the 10 μg/dL limit. Similarly, the health-based criteria for the 30 day average ambient air quality criteria (AAQC) was 0.05 μg/m3. However, the recommended limit was 0.7 μg/m3 to take into account what was considered technically and economically achievable by a model secondary lead smelter. Again, a calculation was made finding that this increase above the health-based criteria could result in an approximate additional increase of 14% in total lead exposure over the limit defined by the IOC_{pop}.

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

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

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

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

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

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

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

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

8.4.4 Lead in Drinking Water

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

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

8.4.5 Lead in Food

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

[103] Ontario Ministry of Education, undated. Report on the Survey of Drinking Water in Ontario Schools for Lead.

[104] *Building Code Act*, R.S.O. 1992, c. 23.

[105] Ontario Ministry of the Environment. (1994), *op.cit.* Appendix D, Section D-2.2.2.

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

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

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

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

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

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[106] *Food and Drugs Act*, R.S.C. 1985, c. F-27.

[107] *Food and Drug Regulations*, C. R. C., c. 870, B. 15.001.

[108] SOR/79-249.

[109] SOR 86/258.

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

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

8.4.6 Lead in Consumer Products

8.4.6.1 Introduction

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

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

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

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

8.4.6.2 Lead in Ceramics, Glassware and Kettles

Regulations governing the amount of lead that can leach from glazed ceramics[114] and from kettles with

[111] *Hazardous Products Act*, R.S.C. 1985, c. H-3.

[112] Under authority of the *Department of Health Act*, R.S.C. 1996, c. H-3.2.

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

[114] Hazardous Products (Glazed Ceramics) Regulations,C.R.C., c. 925.

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

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

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

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

8.4.6.3 Lead in Paint

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

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[115] Hazardous Products (Kettles) Regulations, C.R.C., c. 927.

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

[117] SOR/98-176.

[118] Hazardous Products (Liquid Coating Materials) Regulations, C.R.C., c. 928.

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

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

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

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

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

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[119] Carriages and Strollers Regulations, SOR/85 - 379.

[120] Cribs and Cradles Regulations, SOR/86-962.

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

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

[123] Notice (Proposed Regulatory Text), *C.Gaz.* 1997.I.1759. Notice (Order), *C.Gaz.* 1997.I.1755.

[124] Personal communication with Barbara McElgunn, Learning Disabilities Association of Canada (June, 1998)

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

8.4.6.4 New and Unexpected Sources

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

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

Lead in Crayons

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

Lead in Plastic Mini-blinds

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

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

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

[127] Arizona Department of Health Services. *Miniblind Lead Warning Issued*, News Release. (December 7, 1995)

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

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

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

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

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

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

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

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

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

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

Lead in Children's Products

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

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

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

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

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

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

[134] Personal communication with Louise McGuier-Wellington, Justice Canada. (May 18, 1999)

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

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

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

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

And Still More Sources

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

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

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

8.4.6.5 Health Canada's Lead Reduction Strategy

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

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

[139] Bruening, K, *et.al.* Dietary Calcium Intakes of Urban Children at Risk of Lead Poisoning, *Environmental Health Persepectives* 107(6) 431-435 (1999).

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

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

[142] Personal communication, Jonathan Williams, Product Safety Bureau, Health Canada, March 27, 2000.

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

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

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

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

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

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

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

[143] Subramanian, M., Acting Director, Product Safety Bureau, Health Canada. Undated speech apparently delivered in February, 1999.

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

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

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

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

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

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

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

[147] Ibid.

[148] Ibid.

[149] "Tough limits on lead help our children: Ottawa to take 'strong stand' on kid's products." *Toronto Star*, May 14, 1999.

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

8.4.7 The OECD Declaration of Risk Reduction for Lead

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

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

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

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[150] Personal communication, Jonathan Williams, Product Safety Bureau, Health Canada, March 27, 2000.

[151] Ibid.

[152] Pearce, F. Lead trickles through European loophole...while industry blocks international ban, *New Scientist.* (July 15, 1995)

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

8.4.8 Blood-Lead Testing and Follow-Up

8.4.8.1 Approaches in the United States

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

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

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

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

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

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

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

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

[158] Nriagu, J.O. Clair Patterson and Robert Kehoe's Paradigm of "Show Me the Data" on Environmental Lead Poisoning, *Environmental Research* (Section A, 78)(1998), pp. 71-78.

8.4.8.2 Canadian Comparisons

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

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

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

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

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

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

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

8.4.8.3 Pediatric Management of Lead Toxicity in Canada

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

8.5 CONCLUSIONS AND LESSONS LEARNED

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

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

[160] Including recognition that there may be no threshold below which lead does not begin to harm a child's developing nervous system.

alongside increasing rates of child poverty.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

The lessons learned from lead include the following:

1. Regulatory action on dangerous substances must include a precautionary and preventative approach. The regulatory history and ongoing approach to the regulation of lead shows that this lesson has not been learned. The phase-out of highly dangerous substances has rarely been required. For lead in gasoline, phase-out occurred only after reaching health effect levels in huge numbers of children.

2. Independent research into pollutants is essential - both the agenda for research and the actual conduct of the work. When industry has exclusive control of the research into its own pollutants, the results cannot be considered independent.

3. Lack of proof of harm must not be considered proof of safety. Given the inherent complexity and inevitable uncertainty surrounding the investigation of the effect of toxic chemicals, the scientific standard of proof is far too high. For lead in gasoline, insistence on this standard of proof amounted to an approach of delaying regulatory action while conducting an enormous, uncontrolled experiment on children.

4. The insistence on "sound science" to set regulatory standards and in particular the insistence on solid exposure data and dose-response information sets up a "Catch-22" situation. The paradox is evident in the history of the scientific inquiry and regulation of lead. Ontario's multi-media standards for lead are an example. In the early 1990s, this multi-media risk assessment and risk management exercise was able to establish very credible, scientifically defensible regulatory standards given the extensive scientific information available about lead exposure and dose-response relationships. However, given the high baseline exposure created by historical circumstances of widespread environmental lead contamination, the standards were set at levels that provided for almost no safety margin between actual exposure levels and the levels permitted in the standards. For most pollutants however, levels of environmental contamination are much lower, as was the case with lead in the 1920s. For other pollutants, scientific

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

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

8.6 RECOMMENDATIONS

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

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

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

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

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

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

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

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Case Study #2: Regulating Pesticides to Protect Children's Health

Case Study #2: Regulating Pesticides to Protect Children's Health

9.1 INTRODUCTION

This case study addresses the health effects of pesticides and the regulatory response by the Canadian Pest Management Regulatory Agency (PMRA). To provide input to a Parliamentary Standing Committee review of pesticide regulation, it was published earlier than the main study. Information in this Chapter is therefore current to December 1, 1999. More recent information relevant to this chapter, particularly regulatory matters, is reviewed in Chapter 4. Most important, the PMRA has recently stated that it will follow the lead of the United States Environmental Protection Agency with respect to pesticide re-evaluation. Hence, the recommendations contained herein regarding the conduct and transparency of the PMRA's risk assessment process for both new and currently registered pesticides need to be viewed in that context.

In contrast to the first case study which summarizes the comparatively vast amount of health effect information on one specific contaminant, lead, this review examines the relatively more limited information on a varied group of environmental contaminants collectively called, pesticides.

On the regulatory side, this case study focuses on a review of the Pest Management Regulatory Agency (PMRA) while the Lead Case Study is a broader canvassing of regulatory controls on all aspects of lead use and environmental emissions. The Lead Case Study provides the regulatory "cautionary tale" since it documents how regulatory action on lead has been consistently denied or delayed in the face of troubling but inconclusive evidence of harm. The current situation with pesticides both in terms of the knowledge about human health effects, the troubling results from animal studies, and the inadequate regulatory response is very similar to the early chapters of the "cautionary tale" of lead.

9.1.2 Children: Greater Exposure and Potential for Serious Health Effects

In Canada, most pesticides are commonly applied in agriculture and by the forestry industry.[1] However, they are also frequently used in the household setting, both indoors and outdoors. Common household pesticide applications target garden weeds, insect infestations (indoors and outdoors), fleas on pets, and lice, scabies, bugs and bacteria on people. Pesticides are also used in wood preservation. Many pesticide uses can therefore bring people into contact with these chemicals through their living environment and via occupational exposure. Spraying (i.e., for crops, lawns, gardens or indoor pests) means wide, airborne dispersal of pesticide which allows for an effective route of exposure to humans via inhalation, ingestion or skin absorption. Some pesticides, or their breakdown products have been measured in trace or higher levels in soil, air, water and food.[2] Although there are assuredly health and other benefits to the

[1] Niedert, Eli, R.B. Trotman and P.W. Saschenbrecker. Levels and incidences of pesticide residues in selected agricultural food commodities available in Canada. *Journal of AOAC International*. 77 (1994), pp. 18-33.

[2] *Ibid*; U.S. Environmental Protection Agency, Atmospheric Research and Exposure Assessment Laboratory. *Nonoccupational Pesticide Exposure Study (NOPES)*. EPA Report Number EPA/600/3-90/003. Research Triangle Park, NC, (1990); National Research Council. *Pesticides in the Diets of Infants and Children*. (Washington: National Academy Press, 1993); Niedert Eli and P.W. Saschenbrecker. Occurrences of pesticide residues in selected agricultural food commodities available in Canada. *Journal of AOAC International*. 79 (1996), pp. 549-566; and Health Canada. *Persistent Environmental Contaminants and the Great Lakes Basin Population: An Exposure Assessment*. Minister of Public Works and Government

use of pesticides in terms of food production and eradication of household pests, such as cockroaches that may pose health problems,[3] the exposure to pesticides through a variety of media and pathways spells a potential health risk to people, especially children.

The 1993 report of the U.S. National Research Council, *Pesticides in the Diet of Infants and Children*[4] was a pivotal work that focused attention on the greater susceptibility and exposure of children to environmental contaminants in general, but particularly from pesticide residues in foods. Research since the NRC report lends further support to the conviction that there is potential for children to encounter widespread, low-level exposure to pesticides for two reasons: 1) pesticides are present in environmental media such as food, air, water, soil and dust, and, 2) children's small size and unique behaviours translate into relatively greater intake than adults of pesticides encountered in their environment.[5] Aside from these sources of pesticides that children (like adults) may ingest, inhale or absorb through their skin, children may also be exposed *in utero* via the placenta, as well as postnatally through their mother's milk.

The range of known or potential health effects from pesticides includes: abnormalities in physical development, cancer, immune system suppression, neurotoxicity, reproductive effects, and alterations in endocrine function. However, the NRC report also highlighted that the gaps in our knowledge regarding the effects from pesticide exposures at a young age and over the course of childhood development are such that we cannot be certain of the long term effects on children's health. It is clear that there is a growing consensus among researchers and medical practitioners around the globe that we should be concerned about the hazards of pesticides to children's health. There are still many unknowns and gaps in our knowledge. However, this situation does not exonerate pesticides, nor should it be used as a reason to delay regulatory action; particularly cautionary and preventative action.

Section 9.2 below reviews the circumstances by which children are generally more highly exposed to pesticides than are adults. Section 9.3 reviews the scientific literature concerning the health effects of pesticides including the results from both animal studies and those addressing human health.

9.1.3 The Public Policy Response

Given the range and severity of both the demonstrated and potential effects of pesticide exposure, adequate pesticide regulation is critical to human and environmental health, and in particular, children's health. Globally, governments have begun to recognize the dangers associated with pesticides and are reassessing the safety of those that are currently in use. These governments are also applying new knowledge and assessment techniques in their evaluation of pest control products.

Government efforts to minimize the risks associated with pesticide use have also resulted in a number of international initiatives, of which Canada is a participant. For example, Canada is a signatory to the 1997

Services, Canada. Catalogue No. H46-2/98-218E. (1998a).

[3] Toronto Public Health. *Cockroach Control in the Housing Sector: Evaluation of an Integrated Pest Management (IPM Demonstration Project for an Apartment Complex.* Prepared for the Ontario Ministry of the Environment (OMOE) and the Canada Mortgage and Housing Corporation (CMHC) (1998).

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

[5] Brenda Eskenazi, *et al.* Exposures of children to Organophosphate pesticides and their potential adverse health effects. *Environmental Health Perspectives* 107 (Suppl 3) (1999), pp. 409-419.

Declaration of the Environment Leaders of the Eight on Children's Environmental Health. In that declaration, Canada pledged to establish national policies regarding environmental hazards that, "take into account the specific exposure pathways and dose-response characteristics of children when conducting environmental risk assessments and setting protective standards."[6]

In Canada, a new federal government agency, the Pest Management Regulatory Agency (PMRA), was created in 1995 to regulate pesticide use across the country. Sections 9.4 through 9.12 below provide a detailed review of issues concerning pesticide regulation as undertaken by the federal Pest Management Regulatory Agency (PMRA). Section 9.13 addresses the political will and funding necessary to implement both existing unfulfilled commitments to improve federal pesticide regulation as well as additional recommendations arising from this review. Sections 9.14 and 9.15 provide, respectively, the case study conclusions and a consolidated list of recommendations.

9.1.4 Unfulfilled Commitments in Canada

Since 1994, the federal government has made a number of commitments to improve its regulation of pesticide use. However, this investigation reveals that the great majority of these commitments remain unfulfilled. The federal government's failure to improve its regulation of pesticide use seriously calls into question the capacity of the current pesticide regulatory system to protect children's health.

Despite its length, this case study is far from being a comprehensive investigation of the complex pesticide regulatory system. Instead, it focuses on a number of issues that are critical to pesticide regulation and the protection of children's health. Even this limited focus proved difficult, however, as no comprehensive documents have been produced by the PMRA regarding its risk assessment and management processes. Information concerning these processes is difficult to access and understand. At times, it appears contradictory. Lack of clarity on the application of risk assessment and risk management processes is of significant concern given the well-documented problems with the subjective nature of this evaluation and management tool. The additional and more fundamental shortcomings of risk assessment, including its inability to assess "real-world" combinations of chemicals in a child's environment or their cumulative or synergistic effects, have yet to be effectively addressed by any advocates or practitioners of risk assessment. The PMRA's failure to explicitly set out its risk assessment and risk management approach, in a format for public consumption, is a key criticism of the case study and a factor that limited its scope.

Nevertheless, detailed recommendations can be made with respect to improving the transparency and effectiveness of regulating pesticides to protect children's health. Indeed, many of the Case Study recommendations have to do with the detailed steps necessary to implement a wide range of unfulfilled government commitments with respect to pesticides management. These include the fact that the PMRA has so far failed to:

- adequately implement the Toxic Substances Management Policy;
- develop a regulatory policy on formulants;
- develop a national compliance policy;
- develop a re-evaluation policy and a comprehensive program of pesticide re-evaluation;

[6] *1997 Declaration of the Environment Leaders of the Eight on Children's Environmental Health*, as found in Pollution Probe and Canadian Institute of Child Health. *The Air Children Breathe: the Effects on Their Health*, Conference Proceedings, January 19 and 20, 1998.

- develop a pesticide risk reduction policy;
- produce Proposed Regulatory Decision Documents (PRDD) for proposed registration, re-evaluation and special review decisions;
- create a national database on pesticide use;
- require mandatory reporting of adverse effects by registrants; or
- support the integration of pest management with the broader goal of environmental sustainability including setting targets and establishing workplans for the reduction of pesticide use in all sectors.

9.1.4.1 The Environmental Commissioner's Report

This investigation confirms and expands upon the findings in the May, 1999 *Report of the Commissioner of the Environment and Sustainable Development to the House of Commons.*[7] That report included troubling criticisms of the federal government's ability to protect Canadians from the risks of toxic chemicals in general and the PMRA's regulatory management of pesticides in particular. The report was critical of the PMRA in many respects including identifying the existence of conflicts and lack of integration, cooperation or collaboration with other government departments, lack of public access to pesticides-related information and the long-identified problem of lack of effective or coordinated monitoring (of environmental fate, effects, etc.) to complement the federal research agenda for toxic substances, including pesticides.

The Commissioner also found that the PMRA procedures for applying risk assessment and risk management are inconsistent and sometimes in conflict with other government departments. Chapter 4 documents the shortcomings of risk assessment and the need to more effectively adopt a precautionary approach to the management of toxic substances, including pesticides. As the Commissioner's report notes, the Toxic Substances Management Policy is an over-arching tool which provides the federal government's most important basis for implementing a preventative and precautionary approach to harmful pesticides and industrial chemicals. Although limited progress has been made, neither the PMRA, nor any other federal government department, has adequately implemented this policy.

The lack of adequate resources in PMRA and other departments alongside increased demands and increased private sector influence over research agendas were also noted as problems for the federal government's management of toxic chemicals, including pesticides.

The Commissioner's review of the federal government's 13-year-old commitment to pesticide re-evaluation found the actions of the PMRA to be largely inadequate and concluded that no assurance exists that Canadians are not being exposed to unacceptable risks from pesticides needing to be re-evaluated. This case study reaches the same conclusion.

9.2 EXPOSURE

9.2.1 Contaminant Uses & Information

Pesticides are commonly used in several settings including agricultural, industrial, residential and

[7] *Report of the Commissioner of the Environment and Sustainable Development to the House of Commons.* (Minister of Public Works and Government Services, 1999) http://www.oag-bvg.ca

institutional, among others. In Canada, there are approximately 7500 pest control products registered for use and therein, approximately 600 pesticides are the active ingredients in the end-use products.[8]

Agriculture represents the vast proportion of annual pesticide use in the U.S. and Canada.[9] Nonagricultural uses of pesticides are varied and numerous. The most common nonagricultural use of pesticides is for structural pest control in commercial, institutional and residential buildings including school, day-cares, hospitals, stores, office buildings, sports facilities, homes, etc. Pesticide spraying is common on passenger aircraft that fly to or from specific international destinations and is also part of regular maintenance of airplanes by some airlines.[10] Weed management for home and commercial lawns, golf courses, parks, recreation areas, highways, railroad beds and power transmission lines represents another common nonagricultural use of pesticides. Industrial uses for pesticides are considerable. A substantial application of pesticides is as a preservative for wood that ends up in a variety of uses including railroad ties, utility poles, and lumber.[11]

Pesticides can also be found in products containing lanolin as a result of the practice of "dipping" sheep in pesticides. Since many pesticides tend to be lipophilic (they bind to oil/fat molecules), the oil-rich lanolin in the sheep wool can be contaminated with such pesticides. Metals, such as mercury, are used to inhibit the growth of fungus and moulds in paint. In response to evidence of hazardous levels of mercury vapours following paint application and the well known health hazards of mercury, new regulations to greatly reduce the allowable level of mercury in paint are being implemented in the United States and Canada is following suit.[12]

Pesticides are classified according to their chemical structure. Chemical structure is a key to the action of the pesticide and hence, its intended function. All pesticides are designed and intended to kill living organisms and this is achieved by different modes of action.

It is beyond the scope of the present document to adequately profile the full range of pesticides and their ingredients that may pose harm to human health. It is our intention to highlight those compounds that are most commonly used and hence, to which there is greatest possibility of human exposure. Table 9.1 in Appendix 2 to this Case Study summarizes the information on major classes of pesticides.

9.2.1.1 Insecticides

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[8] Pest Management Regulatory Agency. Product Group Counts, not including discontinued products: effective to September 30, 1998 (98-09D). Data provided by PMRA (As cited in: City of Toronto, Public Health, Environmental Protection Office. *Pesticides: A Public Health Perspective.* Unpublished report released October 30, 1998); and facsimile to the Canadian Environmental Law Association from Julie Chagnon, PMRA, March 19, 1999.

[9] Moses, Marion. Pesticides. In: *Occupational and Environmental Reproductive Hazards: A Guide for Clinicians.* Maureen Paul (Ed.) (Baltimore: Williams & Wilkins, 1993), pp. 296-309; and Agriculture Canada, Agri-Food Safety Division. *Annual Report.* (Ottawa, Ontario Canada. 1989)

[10] Northwest Coalition for Alternatives to Pesticides. "Flyers Beware: Pesticide Use on International and Domestic Aircraft and Flights." (1998) http://www.pesticides.org/AirlineSpray.html

[11] Moses, Marion. 1993, *op.cit.*

[12] See Section 8.4.6.3 of Case Study #1 for a discussion of Canada's revised regulations for the lead and mercury content of paint.

Organophosphates & Carbamates

The above two categories of pesticides are commonly used to control household pests such as ants, fleas, cockroaches, earwigs, wasps and silverfish. Organophosphates (OPs), the most widely used insecticide type, are designed to be neurotoxic to living organisms. OPs and Carbamates interfere with the activity of cholinesterase, an enzyme which breaks down acetylcholine, a neurotransmitter. When cholinesterase activity is inhibited, the neurotransmitter acetylcholine is not broken down and as a result there is overstimulation of nerve endings causing acute symptoms such as, serious sensory and behavioural disturbances, impaired coordination, muscle twitching, weakness, reduced heart rate, depressed cognition and coma. Because OPs are neurotoxic, can cross the placenta and have shown dose-related reproductive toxicity in animal studies, there is concern for potential developmental effects in humans.

Organochlorines

While organochlorine insecticides are no longer applied in agriculture in Europe and North America because of their environmental effects, they are still produced in some countries and exported for use in developing countries (on crops and in malaria control programs). Organochlorine insecticides were important pesticides for the precise reasons that they have been banned here since the 1970s. That is, because of their chemical stability and resistance to degradation they were highly efficient chemicals, but these same features, plus their long-range transport and cycling in the ecosystem, mean that they continue to contribute to the environmental load and human body burden measured globally. Until recently, the organochlorine Lindane was the active ingredient found in some medical treatments used against lice and scabies, such as Kwellada.[13] As will be discussed below, several banned organochlorine pesticides such as chlordane and toxaphene (among others) have been detected in the current food supply of Inuit in northern Canada.[14]

Pyrethrins/Pyrethroids

Pyrethrins are insecticides derived from chrysanthemums and pyrethroids are the synthetic versions of this type of compound. These chemicals attack the nervous system. They are generally deemed to be of low toxicity (both acute and chronic) to humans, hence their frequent use as a replacement for more toxic insecticides such as OPs and carbamates. They have also replaced Lindane in treatments for lice and scabies. However, recently, some pyrethroids have been associated with neurologic and respiratory reactivity as well as potential hormonal effects.[15]

Insect Repellants

The pesticide N,N-diethyltoluamil is the substance found in DEET, the most commonly used compound to repel mosquitoes and other insects. It enters the body by absorption through skin or with ingestion. It is commonly applied directly to the skin of children and adults by spray or cream.

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[13] The manufacturer of Kwellada recently released a new formulation that contains permethrin as the active ingredient.

[14] Chan, HM, *et al.* Evaluation of the population distribution of dietary contaminant exposure in an Arctic population using Monte Carlo statistics. *Environmental Health Perspectives* 105 (1997), pp. 316-21.

[15] Vijverberg, HP, van den Bercken, J. Neurotoxicological effects and the mode of action of pyrethroid insecticides. *Crit. Rev. Toxicol.* 21 (1990), pp. 105-126; Cantalamassa, F. Acute toxicity of two pyrethroids, permethrin, and cypermethrin in neonatal and adult rats. *Arch. Toxicol.* 67 (1993), pp. 510-513; Garey, J. Wolff, MS. Estrogenic and anti-progestagenic activities of pyrethroid insecticides. *Biochem. Biophys. Res. Comm.* 251 (1998), pp. 855-859; Go, V, Garey, J., Wolff, MS, Pogo, BGT. Estrogenic potential of certain pyrethoid compounds in the human breast carcinoma cell line MCF7. *Environ. Health Perspectives* 107 (1999), pp. 173-177, as cited in Landrigan et. al, Pesticides and inner-city children: Exposures, risks, and prevention. *Environmental Health Perspectives*, 107 Suppl 3 (1999), pp. 431-437.

Neurobehavioural effects have been observed with heavy exposure to DEET, in laboratory animals, workers and children.[16]

9.2.1.2 Herbicides

Chlorphenoxy Group
These herbicides (e.g. 2, 4-D, MCPA) are the most widely used in the removal of weeds such as dandelions. Therefore they are commonly applied in both agriculture and on lawns. They have low acute toxicity to humans, however, high level ingestion or absorption can cause symptoms such as nausea, vomiting, spasms, seizure and coma and there are reports of peripheral neuropathy[17] being a delayed effect of heavy chronic exposure to 2,4-D.[18]

9.2.1.3 Fungicides

Fungicides are active against many kinds of fungi and are used on seeds, crops and in gardens. Because fungicides are not easily absorbed by the human body they are considered to be of low toxicity, especially with short term exposure, however, they can produce a range of health effects from acute exposure in high doses as listed in Table 9.1. The fungicide Benomyl has been the subject of alleged prenatal exposures that led to babies born with eye defects.[19]

9.2.1.4 Other types of pesticides

This report is not intended to provide an exhaustive listing of all types of pesticides, but rather, to focus on those to which there is greatest opportunity of human exposure, those that are used in greatest volume and those that are particularly toxic to humans. For a thorough treatment of pesticide toxicology, the reader is referred to Hayes & Laws[20] which classifies all pesticides into *eleven* different categories, including several not covered here such as fumigants, rodenticides, biocides and metal-based pesticides.

9.2.1.5 Formulants

Formulants (also called "inert" substances in the United States) are added to pesticides and are distinct from the active ingredients in that they are not intended to affect the target pest. However, they are not necessarily inactive in their own right. Many formulants may independently cause health problems in humans, may increase exposure to the active ingredients in the product and, those that are volatile organic compounds, may increase concentrations of ground-level ozone.[21] These substances are

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[16] Rossenstock, Cullen (Eds.) *Textbook of Clinical and Occupational Medicine.* (New York: Saunders, 1994)

[17] Peripheral neuropathy denotes "damage to the nerves that stimulate the limbs" (City of Toronto, 1998: 17, *op.cit.*).

[18] Costa, L. Basic toxicology of pesticides. *Occupational Medicine: State of the Art Reviews.* 12(2) (1997).

[19] Dyer, C. U.S. court case starts over eyeless babies. *British Medical Journal.* 312 (1996), p. 1247.

[20] Hayes, W.L. and E.R. Laws. *Handbook of Pesticide Toxicology.* (New York: Academic Press, 1991.)

[21] For example, the formulant 4-nonylphenol (4-NP) is 75% by weight of the insecticide Matacil 1.8D, whose active ingredient is aminocarb. 4-NP is known to be a potent hormone disruptor that likely played a role in the

generally inadequately studied for health effects.[22] See section 9.10 below for a discussion of the regulation of pesticide formulants.

9.2.2 Exposure Sources, Routes, Media & Pathways

There are different routes by which humans are exposed to pesticides according to the mode of application and the pathway through which the chemical travels. All routes must be taken into account to gain a full estimate of total exposure. While exposure to pesticides via any single medium may be minute, when one accounts for the variety of sources of pesticides, the possibility exists for a considerably higher *overall* level of exposure. Table 9.2 summarizes many of the potential sources of exposure to pesticides that are most relevant to children.[23]

Exposure to pesticides is often indirect. That is, exposure can occur via media through which pesticides travel in the environment after they have been sprayed or applied. These media include water, air, soil, dust and sediments. Food is another important medium of indirect exposure. Because of our seasonal climate, Canadian food stuffs must come from foreign as well as domestic markets. Therefore, we have to be concerned about exposure to the pesticides that are used in other countries despite their being banned from use in this country. Since some pesticides become distributed throughout the global food chain, they may be ingested from imported agricultural foods that are treated directly with pesticides, or they may be ingested in the meat, milk and eggs of other animals that ingest them from foods or grasses. This is particularly the case with the persistent, bioaccumulating pesticides that become stored in animal tissues and therefore, become increasingly concentrated in organisms higher up in the food chain.

Pesticides enter the body by being ingested, inhaled or absorbed through direct skin contact. Pesticides may also cross the placenta to reach the developing fetus[24] and they may be transferred through mother's milk to the breastfed infant. These represent the main routes of pesticide exposure in humans and children, specifically.

There is little information that quantifies the activity patterns of Canadian children that render them vulnerable to exposure to pesticides. There is also, in general, relatively limited biological evidence of

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drastic decline in the Atlantic Salmon population after Matacil 1.8D application in eastern Canadian forests in the mid-1970s. See: Fairchild, W.L. *et.al.* Does an association between pesticide use and subsequent declines in the catch of Atlantic salmon (*Salmo salar*) represent a case of endocrine disruption? *Environmental Health Perspectives* 107(5) (1999), pp. 349-357.

[22] CALPIRG - California Public Interest Research Group (CALPIRG) Charitable Trust and PSR - Physicians for Social Responsibility (Greater SF Bay & LA Chapters). *Generations at Risk: How Environmental Toxicants May Affect Reproductive Health in California.* (Report released November, 1998.); and Davies, Katherine. *Pesticides and Your Child. An Overview of Exposures and Risks.* Prepared for The Campaign for Pesticide Reduction (CPR!). Ottawa, Ontario. (1998), 38 pp.; and City of Toronto, 1998, *op.cit.*

[23] See discussion in Chapter 2 and Figure 2.1 for clarification of the specific uses here for the terms: route, pathway and medium.

[24] Canadian and U.S. researchers recently presented startling results of a study which detected levels of p,p'-DDE, a breakdown product of the organochlorine pesticide, DDT, in 30% of a small sample of second trimester human amniotic fluid. (Foster, Warren et al, In utero exposure of the human fetus to xenobiotic endocrine disrupting chemicals: Detection of organochlorine compounds in samples of second trimester human amniotic fluid. Paper presented at 81st annual meeting of the Endocrine Society, San Diego, June 14, 1999.)

pesticide exposure in children.[25] Chapter 2 discusses in greater detail the reasons that children are more *likely* to be exposed to contaminants including pesticides. This has do to primarily with behavioural differences as well as the phenomenon wherein, because of their small size, children take in greater volume of substances from their environment, relative to their body size. Children's exposure to pesticides will differ according to the behaviours they exhibit that are characteristic for each developmental stage. For instance, young infants are more likely exposed to pesticides through breast milk, whereas older infants (over 6 months) will also be exposed via the foods they eat. When babies become more mobile they explore their environment more extensively and come into greater contact with the items that might harbour pesticide residues. Their hand-to-mouth and oral exploratory behaviour render them much more likely to ingest or absorb the pesticides that linger in their environment.

Table 9.2 Sources of Exposure Relevant to Children (Adapted from Davies, 1998:10, *op.cit.*)

1. The Home (in the child's home & homes of playmates)	*Applications of pesticides* • Indoor commercial application of pesticides to control rodents, cockroaches, ants, termites, earwigs, etc. • Homeowner/resident use of insecticide sprays, strips, baits • Application of insect repellents directly on skin or scalp (e.g. personal bug sprays, shampoos for lice, scabies) • Collars or powders to treat household pets for fleas, ticks, etc. • Commercial application of lawn and garden insecticides, herbicides and fungicides • Insecticides, herbicides and fungicides used in the garden or on the lawn by the homeowner or resident *Storage and handling of pesticides* • Storage of household pesticides in areas accessible to children • Disposal of pesticides in household garbage *Pesticide life cycle and pathways* • Pesticide residues in house dust and in soil tracked in from outdoors • Pesticide residues on furniture, drapes, toys, pet fur, absorbent items
2. Public Places (schools, daycare, etc.)	Commercial applications of pesticides for rodents, cockroaches, termites, weeds, mould and decay, etc. • Storage of pesticides in areas accessible to children • Disposal of pesticides and pesticide containers in regular school garbage • Maintaining playgrounds, playing fields • Wood preservatives on play structures • Pesticide application in other public places, e.g. airplanes, restaurants, malls, offices, etc.
3. Via Air & Water	Pesticides in indoor air (from uses above for household and public places) Pesticides in outdoor air • Pesticide drift from spraying (agricultural, municipal, household) • Long range transport of persistent pesticides (e.g. DDT) and incorporation into the food chain • Pesticides in drinking water - treated tap water or well water • Pesticides in swimming water - lake and river sediments, algicides in swimming pools

[25] Eskenazi et al. 1999, *op.cit.*

4. Via Food	•	Food crops that are routinely sprayed and form a significant part of juvenile diet. E.g. fruits, vegetable, grains
	•	Foods prepared from agricultural products. E.g. baby foods
	•	Bioaccumulation in other animals and their products. E.g. meat, fish, eggs, dairy products
	•	Mother's intake and transfer across placenta
	•	Mother's body burden transferred to breast milk.

9.2.2.1 Residential - Household & Garden

Children encounter considerable concentrations of pesticides in household dust as well as through air, soil and food, from residential application of pesticides.[26] Indoor application can lead to a much greater chance of exposure to pesticides. Whereas pesticides applied outdoors are subject to biodegradation from the effects of sunlight, wind and water, pesticides applied indoors may persist longer and linger on household surfaces such as furniture, floors and toys, as well as accumulate in house dust. Some particularly long-lasting pesticides have been measured in homes for *years* and even decades after a treatment.[27] Several studies suggest that children's exposure to pesticides may be higher than "other(s) living in the same contaminated environment, in part because young children spend more of their time indoors at home." [28]

Household uses also allow for greater risk of accidental exposures when pesticides are improperly applied or stored in the home or garden. Most pesticide poisonings result from home uses and children are at greatest risk of such accidental exposures.[29] According to data for Canada, accidental pesticide exposure accounts for about 4% of all reported childhood poisonings.[30]

Since young children are closer to the ground and frequently put objects and their hands in their mouths, they are much more likely to accidentally ingest the pesticide residues that persist and accumulate in the household setting. A recent experimental study showed that even two weeks after a spraying of

[26] Whitmore, R.W. et al. Non-occupational exposures to pesticides for residents of two U.S. cities. *Arch. Env. Contam. Toxicol.* 26 (1993), pp. 1-13.

[27] For example, chlorpyrifos was found to accumulate on surfaces that were not in the immediate area sprayed and to be detected in homes for several years after its use. Fenske, RA, et al. Potential exposure and health risks of infants following outdoor residential pesticide applications. *Am. J. Pub. Health* 80 (1990), pp. 689-693;, Wright, CG, et al. Chlorpyrifos in the air and soil of houses eight years after its application for termite control. *Bull. Environ. Contam. Toxicol.* 52 (1994), pp. 131-134. Homes where the organochlorine chlordane was used to kill termites have demonstrated detectable levels of the pesticide for over three decades after its use. See: Savage, EP. Termiticide use and indoor air quality in the United States. *Rev. Environ. Contam. Toxicol.* 110 (1989), pp. 117-130.

[28] Eskenazi et al. 1999: 410, *op.cit.*

[29] Roberts, J.R. et al. Epidemiologic evidence of the effects of pesticides on human health in Canada. Monograph II In: *Strengths and limitations of Benefit-Cost analyses Applied to the Assessment of Industrial Organic Chemicals Including Pesticides. Associate Committee on Scientific Criteria for Environmental Quality.* National Research Council of Canada. NRCC No. 22852. (1985)

[30] Canadian Hospitals Injury Reporting and Prevention Program (CHIRPP). Pesticide-related injuries and poisonings to children less than 20 years of age from the entire CHIRPP database as of December 1994. Laboratory Centre for Disease Control, Health Canada. (1995), 6 pp.

chlorpyrifos, a particularly long-acting organophosphate pesticide, there were sizeable levels of the pesticide measured on toys and other absorbent surfaces in the apartment.[31] The researchers had followed the manufacturer's directions as to what was deemed an appropriate period post-application.[32] They determined that even if parents' followed product instructions, children would still be exposed to significant amounts of chlorpyrifos after the "safe" period for re-entry. The findings of this and other studies suggest that indoor spraying exposes children to between 20 and 120 times the recommended reference dose of 3μg/kg/day of chlorpyrifos from all sources.[33]

Other indoor pesticide uses that allow for human, and especially, child exposures include: hanging no-pest strips, pet flea collars and shampoos, personal bug sprays, shampoo and lotion treatments for lice and scabies, antimicrobial soaps, etc. Use of herbicides, fungicides and insecticides in gardens and on lawns is another route of pesticide exposure. Children may be exposed while playing on grass in their home yard, schoolyards and daycares that have been treated with pesticides. These outdoor residues can also be brought indoors on clothing and shoes and by pets.

9.2.2.2 Agricultural & Industrial

There has been mounting concern over children's exposure to pesticides in food items from pesticide use on fruits, vegetables and grains during the growth season, and in some crops, to control pests during storage, transport and processing.

The main food crops that are eaten in highest proportions by children include apples, pears, peaches, grapes, oranges, green beans, peas, potatoes and tomatoes. Fruits, in particular, are very popular with children and infants, representing about 31% and 16% of the diets of nursing and non-nursing infants, respectively and over 11% of the diet of one to six year-old children.[34]

Consumers Union of the United States recently analyzed data on pesticide residues in over 27,000 samples of foods collected by the U.S. Department of Agriculture's Pesticide Data Program (PDP) between 1994 and 1997. Consumer's Union researchers calculated a Toxicity Index (TI)[35] for each food. The majority of foods had some degree of pesticide contamination, with TI values ranging from low to high (TI of 10 to 300[36]). Notably, there were unacceptably high[37] toxicity scores in seven of the fruits

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

[32] It is of note that Dow has recently withdrawn some products with chlorpyrifos from the market. (U.S. EPA. Agreement reached between EPA and chlorpyrifos pesticide registrants. EPA Press release. Washington, DC: U.S. Environmental Protection Agency, 6 June, 1997). It is not clear, however, if the ban on the part of Dow is comprehensive and is being honoured by other potential manufacturers (Personal communication. Julia Langer, Director, World Wildlife Fund. Phone interview, October 8, 1999).

[33] Davis, D.L., and A.K. Ahmed. Exposures from indoor spraying of chlorpyrifos pose greater health risks to children than currently estimated. *Environmental Health Perspectives* 106(6) (1998), pp. 299-301.

[34] Consumers Union. *Worst First: High-Risk Insecticide Uses, Children's Foods and Safer Alternatives.* (Washington: Consumers Union of U.S., Inc. Sept. 1998: 13)

[35] The TI value takes into account the amount of pesticide present in the food, the relative toxicity of the individual pesticide chemicals detected, and the frequency of pesticide detection. Foods with high TI values have relatively higher pesticide residue concentration or more toxic pesticide residues or both.

[36] TI values for all foods tested ranged from 0.01 to 5,376.

and vegetables popular among children such as, apples, grapes, pears, spinach, peaches, green beans, and winter squash.[38] Consumer's Union states that, with few exceptions,[39] "all of the residues ... are within U.S. legal limits for the pesticides on those foods."[40] There is concern, however, that the legal limits do not equate to a safe level for children when their particular dietary habits are taken into consideration. Sample calculations for several pesticides indicate that infants may be exposed to levels that exceed the ADIs by many times.[41]

Exposure levels in children are higher because of the low variety of food sources in their diets (for example, after milk or dairy products, apples constitute the next largest[42] component of the infant diet) and also because of their greater relative consumption of food. Children eat about three to four times the amount of food that adults do, when calculated on a per unit body weight basis.[43] For example, when a child eats one banana it is relatively the equivalent of an adult eating about five bananas.[44]

Organophosphate (OP) and carbamate insecticides are identified as high risk pesticides for three main reasons: 1) several individual chemicals of these two classes are relatively very toxic; 2) they often leave residues in food; and 3) their residues appear in foods that are consumed most by children.[45] Specific pesticides which contribute significantly to toxicity include methyl parathion (an OP) which, until recently (in the United States at least),[46] was heavily used on apples and green beans, and aldicarb (a carbamate) which has recently been detected in potatoes grown in the U.S.[47] Aldicarb was the subject of investigation in 1991 because of unusually high residues in bananas, as reported by the manufacturer. While the *average* aldicarb residue level was below the established TDI, the levels in some individual bananas, was enough that infants and toddlers would receive more than the allowable limit and potentially would become quite ill by eating only small portions of such "hot" bananas.[48]

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[37] High toxicity was defined as a TI value greater than 100. Foods with TI values less than 10 were considered "clean" and those with scores between 10 and 100 have low to moderate levels of pesticide residues (Consumer's Union, 1999: 1,*op.cit.*).

[38] Specifically, the seven foods with consistently high TI scores were: domestic and imported apples, grapes, pears, spinach; fresh peaches (both domestic and imported); U.S.-grown green beans; and U.S.-grown frozen and fresh winter squash (Consumer's Union, 1999, *op.cit.*).

[39] Violations (between 1 and 5% in different years sampled) were not typically due to excessive levels of legally registered pesticides, but low levels of pesticides not registered for use on a given food, including persistent types (Consumer's Union, 1999, *op.cit.*).

[40] Consumers Union. *Do You Know What You're Eating? An Analysis of U.S. Government Data on Pesticide Residues in Foods*. Consumers Union of United States, Inc. Public Service Projects Department, Technical Division, Groth, E., C. Benbrook, and K. Lutz. (February, 1999), p. 1.

[41] See for example, Schilter, B., A.G. Renwick and A.C. Huggett. Limits for pesticide residues in infant foods: A safety-based proposal. *Reg. Tox. Pharm.* 24 (1996), pp. 126-140.

[42] The proportion of the average infant dietary intake from apples, either fresh or from juice, is 15.5% for nursing, or 7.6% for non-nursing infants (NRC, 1993: 184, *op.cit.*).

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

[44] Goldman, L.R. Case studies of environmental risks to children. *The Future of Children.* 5 (1995), pp. 27-33.

[45] Consumers Union. 1998, *op.cit.*

[46] Note that in August of 1999, in response to children's health concerns, the United States Environmental Protection Agency moved to eliminate many uses of methyl parathion. See Section 4.3.4 of Chapter 4.

[47] Consumers Union. 1999, *op.cit.*

[48] As a result, the manufacturer voluntarily agreed to stop selling aldicarb for use on bananas (Goldman, 1995,

Not all food items carry equivalent risk of exposure to pesticides. Grain products have little evidence of insecticides used during the growing season, however they may have detectable residues from pesticides applied during storage, transport or processing of the grain. Processed foods are also generally found to have lower pesticide residues.[49]

Considerable environmental exposure to pesticides comes from the persistent organochlorine type, many of which have been banned or in restricted use since the 1970s. Organochlorine pesticides can still be measured in organic substances because of their tendency to bioaccumulate and appear in higher concentrations as one moves up the food chain. In most cases, they appear in the environment due to leakage from hazardous waste disposal sites, run-off from contaminated soils and via long-range atmospheric transport and deposition. They reach humans mainly through consumption of animal foods, especially fatty species, that are contaminated directly or due to the biomagnification phenomenon. Animal foods like freshwater fish, meat and dairy products are particularly likely to carry persistent pesticides in their fat stores. Lactation is one route of excretion of a mammal's stored burden of fat soluble contaminants. Therefore eating the fatty flesh and consuming the milk and milk products of animals means exposure to these persistent pesticides. Persistent pesticides may also be detected on some food crops grown in polluted soil.[50]

Those who work in the pesticide industry, whether in the manufacture, application, handling or transport of pesticides, will be occupationally exposed to higher levels of pesticides than the general population. The children of these workers may also be exposed to relatively higher levels as well since pesticide residues may be brought into the home environment on clothing and shoes. Women who work in the pesticide industry will also additionally transfer their intake of some pesticides to their children via the placenta or breast milk. Lack of basic information about the health effects of these occupational exposures is of concern (see discussion in Section 9.12.5 below).

9.2.3 Exposure Data for Ontario and Canada

Exposure estimates can be made based on a) the presence of contaminants in various media, including air, water, soil and food and, b) the most likely estimates of intake from inhalation, absorption and ingestion. The levels of pesticides measured in various human tissues can also provide an approximation of body burden which may indicate the degree of past exposure to pesticides.

9.2.3.1 Environmental Levels

The main source of information available to us concerning the presence of pesticides in the Canadian environment comes from published analyses of food levels.

The Canadian Food Inspection Agency was created in 1997 under authority of the *Canadian Food Inspection Agency Act*.[51] The agency, which reports to the Minister of Agriculture and Agri-Food, is

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op.cit.).

[49] Consumers Union. 1998, *op.cit.*; and Consumers Union. 1999, *op.cit.*

[50] Niedert, Eli, R.B., et. al. 1994, *op.cit.*

[51] *Canadian Food Inspection Agency Act*, R.S.C. 1997, c. C-16.5.

responsible for all federally-mandated food inspection, including that required by the *Food and Drugs Act*. Monitoring for pesticide residue levels for the period 1994-1998 revealed that 1.2% of the 6,879 domestic fresh produce samples had residue levels that were in *excess* of their Maximum Residue Levels (MRL).[52] The corresponding value for imported produce was 2% of 34,591 samples.[53] The bulk of the pesticides detected were those most commonly used, however, there were also some samples with measurable levels of pesticides that have been banned or are no longer used, including many of the organochlorine types. The banned, persistent pesticides were found more often in imported foods and in samples of food crops that mature on or below the soil, indicating that polluted soil is the likely medium by which they are transferred to foods. Noteworthy is the fact that of the Canadian food samples with higher pesticide residues, between 50 to 70% of these represented pesticides that were *not approved* for use by Agriculture Canada on the particular food crop.[54]

Market basket surveys are conducted by the Food Directorate at Health Canada checking about 100 different food items for a variety of environmental contaminants. Food that would represent approximately 80% of the Canadian diet is purchased, prepared and tested for pesticides and other toxins. Toxin concentration values are then multiplied by estimates of food intake (see below) to determine estimates of total intake of different pesticides. These data are compared with standards for intake set by the World Health Organization. [55]

9.2.3.2 Estimates of Intake

The Bureau of Chemical Safety at Health Canada evaluates potential exposures to pesticides through food by estimating consumption patterns of Canadians. The types of food and quantities eaten are then verified more directly via post-market surveillance. Post-market surveys are important for detecting differences between actual versus estimated exposures.[56]

The food consumption data specific for Canadian infants and children is sorely out of date, relying on data provided by the 1972 Nutrition Canada Survey.[57] The PMRA reports that it now uses the 1996 USDA *Continuing Survey of Food Intakes by Individuals.*[58] The Food Research Division of Health

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[52] Maximum residue limits are the regulatory criteria that establish limits for pesticide residues in foods. It is assumed that if residues do not exceed MRLs there is low likelihood of adverse health effects. See section 9.8.3 for further discussion of how MRLs are established.

[53] 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.

[54] Niedert, Eli, R.B. *et.al.* 1994, *op.cit.*; and Niedert and Saschenbrecker, 1996, *op.cit.*

[55] Personal Communication, Robert Dabeka, Food Directorate, Food Research Division, Health Canada. October 1999.

[56] For instance, using the example of the food additive aspartame, pre-market values predicted that adolescents would have greatest exposure to this substance, whereas, according to post-market surveys, it was revealed that infants and children under five had higher consumption relative to body weight, hence this group actually received the highest doses of aspartame.See: Conacher, H.B.S. Do current systems for control of potentially hazardous chemicals in food adequately protect the health of infants and children? In: Canadian Institute for Child Health, *Proceedings from National Symposium on Environmental Contaminants and the Implications for Child Health.* (May 1997), pp. 67-68.

[57] *Ibid.*

[58] Personal Communication, Danielle Prevost, Information Officer, PMRA, September 30, 1999.

Canada's Food Directorate, reports that it recognizes the need for more recent Canadian nutritional intake information, however, they have not been able to access such data for a Canadian sample.[59]

Doubtless eating patterns in North America have changed substantially over the last two or three decades making it vital to use the most recent nutritional intake data available. Comparison of two comprehensive U.S. national food consumption surveys, one from 1977-78 and a more recent one conducted in 1996 by the USDA, indicated that relative to two decades ago, American children now consume:[60]

- more beverages, especially packaged juice drinks and soft drinks;
- more grain-based snacks and combination foods like pizza;
- less milk and fat; and
- more foods away from home.

9.2.3.3 Body Burdens

The National Human Milk Survey has been responsible for collecting and analysing breast milk samples every five years for the last two decades and thereby monitors current exposures of breastfed infants and past exposures of mothers to various chemicals, including pesticides.[61]

The Great Lakes Health Effects Program (Health Canada) recently published extensive results including human exposure to chemical contaminants in the Great Lakes region. They provided information on the levels of the priority persistent contaminants, therefore this only reflects exposure to older organochlorine pesticides such as; aldrin/dieldrin, hexachlorobenzene, mirex,[62] toxaphene and DDT and its metabolite, DDE. They report that the DDT and DDE levels in blood, adipose tissue and breast milk are on a declining trend. While the levels are generally well below those that would cause any clinical symptoms for the average individual, the long-term persistence of these pesticides is an issue for children's health. There is concern, in particular, that breast-fed infants and adults and children who eat considerable amounts of freshwater fish and wildlife may be exposed to higher than the tolerable daily intakes for some of these pesticides.[63]

9.2.3.4 Communities at Risk

Farming families are at considerable risk for exposure to pesticides and particularly, the children are

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[59] Personal Communication, Robert Dabeka, Food Directorate, Food Research Division, Health Canada. October 1999.

[60] Consumers Union. 1998, *op.cit.*

[61] Conacher, H.B.S. 1997, *op.cit.*

[62] Mirex was used against fire ants in the southern United States and although incorporated as a fire retardant into plastic, rubber and paper products, was never registered as a pesticide in Canada.

[63] Riedel, D., N. Tremblay and 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); and Health Canada. 1998a. *Persistent Environmental Contaminants and the Great Lakes Basin Population: An Exposure Assessment.* Minister of Public Works and Government Services, Canada. Catalogue No. H46-2/98-218E.

unique by virtue of living where their parents work and potentially being exposed to occupational agents.[64] The Ontario Farm Family Health Study conducted by Health Canada has shown increased rates of fetal loss and decreased ability to conceive.[65] A large retrospective cohort study of Norwegian farm families has demonstrated that use of pesticides is associated with cancer at an early age in the offspring of farmers.[66] Health Canada's Pesticide Exposure Assessment Study which began in the spring of 1996, is assessing direct and indirect exposure to pesticides among applicators and their families, by data collected from diaries of handling practices, body fluid samples, well-water and swabs of household surfaces.[67] Results are still forthcoming from much of this study. However, results from well-water testing showed that 21% of wells tested had detectable (although non-violative) levels of a pesticide, usually atrazine,[68] indicating the persistence of this chemical and the general point that pesticides applied on crops can find their way into ground water which can then become a source of exposure for people. A recent pilot study in rural California suggested that children living in households with a farm worker were exposed to higher levels of pesticide residues in house dust. This study also predicts that these children could be exposed to levels of the pesticide diazinon that would exceed the U.S. EPA's reference dose simply through ingestion of household dust.[69]

Because of the persistence of the older, banned organochlorine pesticides and their tendency to bioaccumulate, those individuals who consume fish caught in the polluted waters of the Great Lakes are also more often exposed to these pesticides. Among the communities where children are most likely to be at risk for this type of exposure are aboriginal groups, who, because of their traditional culture, maintain use of wild fish and game foods, as well as families of anglers, and immigrant groups who may be less likely to know about fish advisories and guides to eating and preparing sport fish.[70]

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[64] Kristensen, P., A. Andersen, L.M. Irgens, A.S. Bye and L. Sundheim. Cancer in offspring of parents engaged in agricultural activities in Norway: Incidence and risk factors in the farm environment. *Int. J. Cancer.* 65 (1996), pp. 39-50.

[65] Health Canada. Health Protection Branch. Laboratory Centre for Disease Control. Looking at pesticides and pregnancy. *Farm Family Health.* 6 (1) (Spring 1998b) http://www.hc-sc.gc.ca/main/lcdc/web/publicat/farmfam/vol6-1/ff6-1j_e.htm ; and Curtis, K.M., D.A. Savitz,C.R. Weinberg, T.E. Arbuckle. The effect of pesticide exposure on time to pregnancy. *Epidemiology.*10 (1999), pp. 112-117.

[66] Kristensen, P., A. *et.al.*, 1996, *op.cit.*

[67] Health Canada. Health Protection Branch. Laboratory Centre for Disease Control. Researchers analyze pesticide exposure data. *Farm Family Health.* 5 (2) (Fall 1997a) http://www.hc-sc.gc.ca/main/lcdc/web/publicat/farmfam/vol5-2

[68] Atrazine has recently been found in rain water and drinking water samples in areas of Europe and the midwestern U.S. (*Rachel's Environment & Health Weekly*, Headlines: Pesticides in the News, #660. July 22, 1999); see also: Health Canada. Health Protection Branch. Laboratory Centre for Disease Control. Well-water contaminants: results from PEAS. *Farm Family Health.* 5 (2) (Fall 1997b) http://www.hc-sc.gc.ca/main/lcdc/web/publicat/farmfam/vol5-2

[69] Bradman MA, Harnly ME, Draper W, Seidel S, Teran S, Wakeham D, Neutra R. 1997. Pesticide exposures to children from California's Central Valley: results of a pilot study. *Journal of Exposure Analysis & Environmental Epidemiology.* 7(2):217-34.

[70] Health Canada. *Persistent Environmental Contaminants and the Great Lakes Basin Population: An Exposure Assessment.* Minister of Public Works and Government Services, Canada. Catalogue No. H46-2/98-218E. (1998a); and 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)

Researchers from McGill University and Universite de Laval have drawn attention to the fact that compared to all other groups in Canada, the Inuit in northern regions have an unparalleled exposure to persistent contaminants, including pesticides, as these have been detected in their traditional food supply (particularly fish and marine mammals) and breast milk.[71]

Urban children of low income families are an additional group at risk of exposure to pesticides. Living in poor neighbourhoods and poorer quality, older homes that may have insect or rodent problems, raises concerns that children of low income families are more likely to be exposed to pesticides applied in their home and surroundings.[72] There is evidence that those of low income or minority status suffer relatively greater health effects from exposure to environmental contaminants.[73] In the case of lead, we know that poor nutrition is a risk factor for greater health effects because it enhances lead uptake (see section 8.2.3 in Case Study #1for further details). It is worthy of investigating whether the same may be said for exposure to other contaminants such as pesticides.

9.2.4 Summary of Information on Pesticide Exposure

- Children are *relatively* more often exposed to pesticides compared to other age groups.
- There are numerous routes of exposure by which children may come into contact with pesticides, from everyday applications in their homes and yards, through to dietary exposure to residues from agricultural application.
- The main exposure routes of concern (and critical exposure periods) are: a) prenatal exposure from current maternal exposure or from mother's stored body burden, b) exposure from breast milk and/or cow's milk during infancy, c) skin absorption, ingestion and inhalation from indoor and lawn applications and d) dietary exposure from pesticide use on fruits and vegetables that are important food items for young children.
- While those who work with pesticides and people living in agricultural communities are at greatest risk for exposure to pesticides, monitoring data for Canada and Ontario indicate that there are detectable levels of currently registered and banned pesticides found in the environment and in certain foodstuffs, indicating the potential for general population exposure to these chemicals.
- Measures of the organochlorine pesticides (measured in blood, fat and breast milk of Great Lakes populations) indicate that most people carry a body burden of these persistent chemicals. There is concern regarding the effects from these same exposures which, in pregnant women and their unborn children, breastfed infants and children who eat freshwater fish or wildlife, may mean exposure to higher than acceptable doses of certain pesticides.
- Aboriginal children and especially, Inuit children are potentially exposed to the highest levels of persistent organochlorine pesticides because of the presence of these in traditional food items and their mother's milk.
- Poverty and minority status are additional factors that influence a child's exposure to pesticides.

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[71] See for example, Chan *et al.* 1997 *op.cit.*; Dewailly E *et al.* Inuit exposure to organochlorines through the aquatic food chain in Arctic Quebec. *Environ Health Perspect* 101 (1993), pp. 618-20. Berti, P.R., *et al.* Food Use and Nutrient Adequacy in Baffin Inuit Children and Adolescents. *Canadian Journal of Dietetic Practice and Research* 60 (1999, pp. 63-70.

[72] Landrigan *et.al.*, Pesticides and inner-city children: Exposures, risks, and prevention. *Environmental Health Perspectives*, 107 Suppl 3 (1999), pp. 431-437.

[73] Haynes, R.C. Environmental justice in action. *Environmental Health Perspectives.* 105 (1997), pp. 374-377.

Poor children are more likely to live in areas and in homes that have pest infestations and therefore they may be more often exposed to pesticides applied in their household and surroundings.

9.3 HEALTH CONCERNS

Ultimately, by their very nature, pesticides may be hazardous for human health since they are designed and intended to kill living organisms. They are also used in "situations that intimately affect human activity"[74] and therefore there is ample opportunity for humans to be exposed. There is thus good reason to be vigilant in continuing research to characterize the exact nature of their effects on human health.

The nature of the health effects from pesticides depends on the type of pesticide, the dose, timing and duration of exposure, as well as the particular susceptibility of the exposed individual.

9.3.1 Evidence

Evidence for the health effects of pesticides in humans comes from a number of sources including experimental toxicological studies on animals or on tissue or cell cultures in test tubes, and epidemiological data summarizing the effects in people mainly from occupational or accidental exposures. Few of these studies on humans have been able to specifically examine children's exposures.

As is discussed in greater detail below, Health Canada's Pest Management Regulatory Agency has a battery of toxicological testing requirements for *new* pesticides and hence there are more data available for more recently registered pesticides. Of concern, however, is the fact that many of the most commonly used pesticides on the market today were registered prior to the current testing requirements. Hence, the toxicological information on many widely used, older pesticides is incomplete and rarely accounts for certain subtle and delayed developmental effects, such as developmental neurotoxicity, behavioural effects and endocrine disruption, that have recently gained attention as health problems stemming from low dose exposures in animals and children.[75]

9.3.2 Animal & Experimental Studies

9.3.2.1 Reproductive/Endocrine Disruption

Several types of pesticides have been implicated as endocrine mimics or inhibitors, evidence being supported largely by the effects noted in exposed wildlife populations.[76] Exposure to organochlorine pesticides in animals has produced effects on estrogen, androgen, prolactin and thyroid hormone functioning, as well as fetal loss, reduced sperm counts, alterations in reproductive behaviour and defects

[74] Jackson, R.J. The hazards of pesticides to children. In: *Environmental Medicine*. Brooks, Stuart M. et al (Eds). (St. Louis: Mosby, 1995), p. 377.

[75] City of Toronto, 1998, *op.cit.*; and CALPIRG and PSR, 1998, *op.cit.*

[76] There has been much research published on the effects of endocrine disruptors as seen from wildlife studies. (Some of these are described in greater detail in Chapter 2.) An important series of papers came out of the symposium entitled "Estrogens in the Environment, III: Global Health implications" in 1994 (see *Environmental Health Perspectives* volume 103 Supplement 7, October 1995).

in the penis and testicles. Organophosphates and carbamates are also associated with hormonal changes and with abnormal sperm, ovarian follicles and eggs. Some herbicides are toxic to sperm and increase fetal loss. Certain fungicides are also associated with risk of fetal death and damage to testicles and sperm.[77]

9.3.2.2 Congenital Defects

A number of pesticides of several different types have produced birth defects in the offspring of exposed pregnant animals. As mentioned above, organochlorines and certain fungicides are associated with defects in male reproductive anatomy. Certain organophosphates and carbamates have shown dose-dependent effects that give rise to congenital anomalies of various types in rats, dogs and monkeys. These were associated with exposure to chlorpyrifos and carbaryl, respectively.[78]

Several fungicides have been associated with structural defects. Dithiocarbamate fungicides are associated with defects in the brain and limbs. The Registry of Toxic Effects of Chemical Substances (RTECS)[79] record for the fungicide Benomyl[80] lists reproductive effects from over a dozen different tests performed at the lowest published toxic dose on pregnant rodents. Among the observed developmental abnormalities listed are effects on the eye, ear, other craniofacial structures including the nose and tongue, the central nervous system, musculoskeletal system, urogenital system and the body wall. The observation of craniofacial defects in animals is disturbing in view of the recent concern surrounding babies with gross eye defects born to mothers who were presumed to have had exposure to Benomyl early in their pregnancy.[81]

9.3.2.3 Growth

In utero exposure to various types of pesticides, including organophosphates, carbamates, herbicides, and fungicides has affected fetal growth as indicated by experimental observations of smaller pup weight, stunted fetus and reduced postnatal weight gain.[82]

9.3.2.4 Neurodevelopmental Toxicity

Many pesticides function as poisons to the nervous system and therefore act as neurotoxins in animals. The criteria for assessing developmental toxicity from animal studies include premature death of the organism, structural abnormalities, alterations in growth and long-term functional deficits.[83]

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[77] CALPIRG and PSR, 1998, *op.cit.*

[78] *Ibid.*

[79] RTECS is compiled by the US National Institute for Occupational Safety and Health (NIOSH) and provides detailed toxicological profiles, reviews and citations on over 140,000 chemical substances. (See http://ccinfoweb.ccohs.ca/databases.)

[80] Benomyl is among selected pesticides listed as teratogenic by the U.S. Environmental Protection Agency See: Moses, 1993, *op.cit.*

[81] Dyer, C. 1996, *op.cit.*

[82] CALPIRG and PSR, 1998, *op.cit.*

[83] Moore, *et.al.* An evaluative process for assessing human reproductive and developmental toxicity of agents.

Organophosphates and carbamates are particularly neurotoxic and animal studies have demonstrated immediate and long-term delayed neurodevelopmental and behavioural effects associated with pre- and peri-natal exposure. Brenda Eskenazi and colleagues summarize the evidence from over 35 experimental studies investigating the effects of different OP pesticides on the developing nervous system.[84] Exposure to the organophosphate DFP in neonatal[85] mice was found to cause altered spontaneous motor behaviour as well as decreased brain neurotransmitter receptor levels in the adult at age 4 months.[86] Newborn rats exposed to levels of chlorpyrifos which did not produce overt toxicity of any kind did exhibit inhibition of protein and DNA synthesis in the brain when assessed as adults.[87] Pregnant mice exposed daily to diazinon at relatively low doses (0.18 mg/kg/day) had young with no visible defects at birth, however, these mice showed impairments of neuromuscular endurance and coordination when tested as adults.[88]

9.3.2.5 Carcinogenicity

There are limits in the ability of toxicological tests to identify substances that may be carcinogenic by any of the recognized mechanisms. (See Chapter 2, section 2.6.9 for an explanation of mechanisms of carcinogenicity). With this in mind, it is apparent that data on the carcinogenicity of pesticides and other environmental chemicals cannot help but be incomplete. Evaluation of pesticides for carcinogenicity in animals has suggested nonetheless that there are carcinogens represented from all major classes of pesticides including organochlorine and organophosphate insecticides, herbicides, fungicides and fumigants.[89] From over 45 pesticides identified by the International Agency for Research on Cancer (IARC) as being potential or known carcinogens in animals, almost half (n=20) are still registered and in common use in the U.S., including: the herbicide atrazine; insecticides dichlorvos, dicofol and lindane; and the fungicides captan, pentachlorophenol and creosote.[90]

An Italian experimental study which exposed rats to a mixture of 15 pesticides showed DNA damage at low doses, but paradoxically, not at high doses of the chemical mixes.[91] These investigators also determined that the toxicity of the mixture was significantly reduced if the fungicide Benomyl was

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Reprod. Toxicol. 9 (1995), pp. 61-95.

[84] Eskenazi *et al.* 1999, *op.cit.*

[85] Neurodevelopmental differences between rodent and human are such that the first 10 days postnatal are equivalent to the processes occurring the last trimester of human fetal gestation. See: Dobbing, J. and J. Sands. Comparative aspects of the brain growth spurt. *Early Hum. Devel.* 3 (1979), pp. 79-83.

[86] Ahlbom, J., A. Frederiksson and P. Eriksson. Exposure to an organophosphate (DFP) during a defined period in neonatal life induces permanent changes in muscarine receptors and behaviour in adult mice. *Brain Res.* 677 (1995), pp. 13-19.

[87] Whitney, K.D., F.J. Seidler and T.A. Slotkin. Developmental neurotoxicity of chlorpyrifos: cellular mechanisms. *Toxicol. Appl. Pharmacol.* 134 (1995), pp. 53-62.

[88] Spyker, J.M. and D.L. Avery. Neurobehavioural effects of prenatal exposure to the organophosphate Diazinon in mice. *J. Toxicol. Environ Health.* 3(5-6) (1977), pp. 989-1002.

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

[90] Hoar Zahm, S., M. Ward and A. Blair. Pesticides and cancer. *Occupational Medicine: State of the Art Reviews.* 12 (2) (1997)

[91] Lodovic, M. et al. Effects of a mixture of 15 commonly used pesticides on DNA levels of 8-hydroxy-2-deoxyguanosine and xenobiotic metabolizing enzymes in rat liver. *J. Env. Path. Tox & Onc.* 13 (1994), pp. 163-68.

excluded. Results such as these indicate that the mechanisms of toxicity and carcinogenicity for chemical mixtures are complex and unpredictable.

Several studies also indicate that when human tissue cultures are exposed to pesticides such as cypermethrin or diazinon, researchers have observed changes such as an increased number of micronuclei and increased sister chromatid exchange. These outcomes are DNA alterations that signal the potential for disruption of normal cell division to a cancerous pattern.[92]

9.3.2.6 Immune System Suppression

There appears to be substantial experimental evidence of immunotoxic effects from exposure to pesticides. In their report for the World Resources Institute (WRI), Repetto & Baliga[93] present the key points from this research which suggests that many types of pesticides, but especially organochlorine, organophosphate, carbamate and metal-based pesticides, alter immune function in animals and cell cultures. These are hypothesized to cause immunosuppressive effects manifest as diminished host resistance to infections, as well as promotion of tumour growth, providing an alternative mechanism for carcinogenicity from these substances. The array of studies conducted using many different pesticides, on different animal species and on cell cultures, and examining several different aspects of immune function alteration is too numerous to outline here, but the reader is referred to Repetto & Baliga and their cited sources for further detail. A critique by Acquavella and colleagues[94] suggests that the WRI review focussed too exclusively on acute, high dose toxicological studies, the doses for which would not directly reflect the (lower) exposures more typical for humans. It is their contention that 1) "in current practice, immunotoxicity is usually evaluated at doses lower than those producing overt toxicity"[95] and that, 2) pesticides are not routinely considered to be immunotoxicants.[96] Quite rightly, they also criticize the WRI report for failing to distinguish between findings that were statistically significant versus non-significant and for inaccurate citation of results from some studies.

These criticisms aside, observations of *wildlife* populations appear to demonstrate that environmental exposure to contaminants, many of which are the older, bioaccumulating organochlorine type pesticides, is associated with immunotoxicity in fish, birds and marine mammals.[97] An interesting prospective

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[92] Surralles, J., N. Xamena, A. Creus, J. Catalan, H. Norppa and R. Marcos. Induction of micronuclei by five Pyrethroid Insecticides in whole-blood and isolated human lymphocyte cultures. *Mutation Research.* 341 (1995), pp. 169-184.; Bianchi-Santamaria, A., M. Gobbi, M. Cembran and A. Arnaboldi. Human lymphocyte micronucleus genotoxicity test with mixtures of phytochemicals in environmental concentrations. *Mutation Research* 388 (1997), pp. 27-32; and City of Toronto, 1998, *op.cit.*

[93] Repetto, R. and S.S. Baliga. *Pesticides and the Immune System: The Public Health Risks.* (Washington: World Resources Institute, 1996), 103 p.

[94] Of note is the fact that the eight authors of this critique cite affiliation with five different chemical companies including Monsanto, Dow, du Pont de Nemours and BASF of the U.S. and Zeneca Co. of England. See: Acquavella, J. et al. A critique of the World Resources Institute's report on Pesticides and the Immune System: The Public Health Risks. *Environmental Health Perspectives* 106 (1998a), pp.51-54. This scientific critique was organized by the American Crop Protection Association.

[95] *Ibid*, Acquavella, J. et al. 1998a, p. 52.

[96] *Ibid*, p. 53.

[97] Repetto, R. and S.S. Baliga, 1996, *op.cit.*

experiment[98] with harbour seal pups captured from relatively unpolluted waters and housed for several years in controlled conditions determined that those fed herring from the polluted Baltic Sea exhibited substantially weaker immune responses and had a greater prevalence of infections compared to controls.[99] Interestingly, the Baltic Sea fish for the experiment was purchased from markets where it was intended for human consumption.

Despite contradictory opinions toward the WRI report, both the WRI researchers and critics from the chemical industry agree that the potential for immune system dysfunction from pesticides is an important issue that warrants: a) measures to restrict exposure to pesticides; b) improved and routine screening of pesticides for immunological toxicity; and c) further careful epidemiological studies that investigate the precise immune system effects in humans.[100]

9.3.2.7 Summary

Experimental (i.e. animal) studies indicate that several different types of pesticides have a variety of effects on reproduction, development, growth, neurological development, behaviour, cancer risk and the functioning of immune and endocrine systems.[101] The degree to which these observed effects are translatable to health risks in humans is not absolutely clear since some of these health effects have mainly been demonstrated only after exposure levels that are higher than those likely to be experienced by humans. Animal studies have also suggested, however, that there is increased sensitivity to pesticides in young, developing animals (both pre- and post-natally), that neurological and behavioural effects in the young may occur at low levels of exposure, manifesting at later stages in life, and that there is also the potential for transgenerational effects to occur.

9.3.3 Human Studies

Although there is extensive literature concerning the toxic effects of pesticides in humans from high dose, accidental exposures, there are limited data on low-level pesticide toxicity in humans, both in the young and in adults.[102] Researchers speculate that, while it is likely that the toxic effects of pesticides are similar between children and adults and across different species, there may be differences due to the unique windows of vulnerability at different times during development. For example, a number of studies looking at cancer outcomes have shown that the organ systems affected by pesticides vary according to species of mammal. The effects of pesticides during human adolescence cannot be replicated in weeks of animal maturation[103] and neurological, behavioural and possibly immunological

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[98] This work was conducted by researchers from the Netherlands National Institute of Public Health and Environmental Protection. See: De Swart , R.L., P.S. Ross, H.H. Timmerman, H.W. Vos, P.J.H. Reijnders, J.G. Vos and A.D.M.E. Osterhaus. Impaired cellular immune response in Harbour Seals (*Phoca vitulina*) feeding on environmentally contaminated herring. *Clin Exptl Immunol.* 101 (1995), pp. 480-86.

[99] *Ibid*, De Swart , R.L., *et.al.*, 1995.

[100] Robert Repetto & Sanjay Baliga. Response to the ACPA's critique. EHP 106(2) (1998), pp. A52-53; Acquavella *et.al.* , Response. Environmental Health Perspectives, 106(2) (1998b), p. A53.

[101] CALPIRG and PSR, 1998, *op.cit.*; and City of Toronto, 1998, *op.cit.*

[102] NRC. 1993, *op.cit.*; and Schilter, B, Renwick, G & Huggett AC. Limits for pesticide residues in infant foods: A safety-based proposal. *Reg. Toxicol. Pharm.* 24 (1996), pp. 126-140.

[103] This is a fundamental problem with animal studies in that the stages of development in animal species are not

outcomes cannot reliably be extrapolated from animal studies.[104] Therefore, although animal studies offer important experimental evidence, they are not always sufficient for estimating the risks to humans. The evidence from human accidental exposures does suggest that the young are more susceptible to ill effects from exposure to pesticides like OPs and carbamates.[105] This is because of the sensitivity of their developing systems, such as the brain and central nervous system and because they have insufficient activity of the necessary de-toxifying enzymes. [106]

Our knowledge of human health effects from pesticides comes largely from case reports of acutely exposed individuals (either accidental or occupational exposure) which indicates what the results of short-term, high exposure will be. We also gain information from examining the patterns of health problems at the population level, that is, from epidemiological studies. The latter is an important source of information for evaluating the health risks to humans from exposure to pesticides, because it indicates the health effects from real-world exposures, i.e. chronic or longterm exposures to lower levels of pesticides.
Epidemiological studies are observational, as opposed to being experimental, as we do not have the option of experimentally exposing groups of people to toxic chemicals.[107] Epidemiologists recognize that the strength of evidence from epidemiological studies depends upon the quality of information that identifies, among other factors, populations at risk[108], cases (i.e., individuals with a given health problem), exposure levels[109] and dose[110] received. (See Section 4.3 of Chapter 4 for a discussion of the fundamentals of epidemiological studies.) The challenges of epidemiology are such that it is very difficult to adequately determine all relevant variables so that cause-and-effect relationships are proven conclusively.[111] However, observational studies are the best possible evidence and the clear complement to animal studies for increasing information on human health effects from pesticides.

Epidemiological studies of the effects from pesticides have focused on two types of samples, 1) those exposed because of agricultural occupation and 2) those exposed by virtue of where they live (i.e. in areas where pesticides are routinely applied). Just about all human populations have had some exposure

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entirely comparable to human developmental periods.

[104] National Research Council, 1993 *op.cit.*; and Schilter *et al.*1996 *op.cit.*

[105] NRC, 1993, *op.cit.*; Schilter, *et al.* 1996, *op.cit.*; and Eskenazi et al. 1999, *op.cit.*

[106] *Ibid.*

[107] However, pesticide companies have crossed this line in the past and in recent years have increased the practice of testing pesticides on human "volunteers." See more detailed discussion in Section 4.4.3.2 in Chapter 4.

[108] Population at risk refers to the group of individuals among whom the particular health problems might be observed, or all people who are susceptible to or could have the disease or health problem (or a representative sample of them). (See: Fletcher RH. *et al. Clinical Epidemiology: The Essentials.* 1988. Baltimore: Williams & Wilkins.) In environmental health studies these would be people who by virtue of their occupation, residence, activities or physiology are exposed to a given environmental chemical.

[109] Exposure refers to "the extent of contact between the toxicant and the surfaces of the human body." See: Roberts, J.R. et al.,1985 *op.cit.*

[110] Dose refers to "the amount of toxicant in the critical organ or tissue." *Ibid*, Roberts et al, 1985: 1.

[111] This has been a criticism commonly invoked by spokespersons for the pesticide industry and it echoes the response of the tobacco industry to scientific studies that demonstrated an association between lung cancer and cigarette smoking. (Kelly Martin MD CCFP-EM, presentation to Standing Committee on Environment and Sustainable Development, December 1, 1999, Ottawa.)

to multiple pesticides.[112] Aside from case reports and anecdotal data of acute pediatric pesticide poisoning, there is also incomplete knowledge of the specific health effects in children since they have not been adequately studied.

9.3.3.1 Accidental Exposure

While those who work with pesticides undoubtedly receive the highest exposures, others, including children, may also receive high doses from the use of pesticides in buildings, by drift from aerial spraying or by accidental ingestion of improperly stored pesticides. Aside from the concern from low-level environmental exposure to pesticides, accidental poisoning is the most significant health hazard for children from pesticides.[113] Symptoms of acute exposure to pesticides are agent specific. That is, they reflect the intended action of each pesticide.[114] Symptoms may mimic those of more common conditions, such as diarrhea, influenza, or other uncommon conditions and therefore may not always be easily diagnosed, especially in children.

Large numbers of people were exposed to a metabolic product of the highly neurotoxic pesticide aldicarb in California in 1985.[115] Individuals reported symptoms of headache, nausea, vomiting, diarrhea, increased salivation, blurred vision and muscle twitching.[116] The common source of the poisonings was determined as contaminated watermelons. The accidental poisoning of seventy-nine people in Jamaica from flour contaminated with parathion demonstrated that proportionately more children than adults died from the exposure and that the reported fatal dose for children was twenty to thirty times *lower* than the estimated fatal dose for the adult.[117]

9.3.3.2 Occupational Exposure

Those with occupational exposure to pesticides, e.g., farm workers and pesticide mixers and applicators, have provided essential data on the range of potential effects in adults from acute exposure.

9.3.3.3 Reproduction, Fertility

Despite a dearth of published data on reproductive toxicity from pesticides currently in use, there is some evidence that acute exposures to pesticides may be associated with fertility problems in both men and women. Female farm workers in Ontario whose activities exposed them to pesticides had an increased time to pregnancy although the association was not significant.[118] There is evidence that a number of

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[112] CALPIRG & PSR, 1998, *op.cit.*

[113] Jackson, R.J. The hazards of pesticides to children. In: *Environmental Medicine*. Brooks, Stuart M. et al (Eds). (St. Louis: Mosby, 1995), pp. 377-382.

[114] *Ibid.*

[115] *Ibid.*

[116] Muscle twitching is evidence of denervation effects from exposure.

[117] Diggory HJ *et al.* Fatal parathion poisoning caused by contamination of flour in international commerce. *Am.J.Epidemiol.* 106 (1977), pp. 145-53.

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

different pesticides can cross the placenta and therefore can affect fetal development and survival.[119] Studies have found that women who work in agriculture or had environmental exposure to pesticides during pregnancy have higher risk of spontaneous abortion or stillborn babies, although there is inconsistency in these studies.[120] Research suggests that male fecundity may be affected by both occupational and environmental exposures to pesticides as determined by neuroendocrine status, testicular function and pesticides detected in seminal plasma.[121] Declining sperm count and increased abnormal sperm were found to be associated with exposure to 2,4-D in farm sprayers.[122] The Ontario Farm Family Health Study has shown that there was increased risk of miscarriage where fathers had participated in specific handling of herbicides and where they reported using certain pesticides such as thiocarbamates or carbaryl in the three months prior to conception.[123] Data are suggestive that reproductive risks are generally higher for maternal as opposed to paternal exposures.[124]

9.3.3.4 Developmental Malformations

There are few epidemiological studies with solid evidence of the effects of *in utero* exposure to pesticides. A Minnesota (ecological)[125] study of agricultural workers and the general population revealed higher rates of birth defects in infants conceived in spring and in areas where chlorphenoxy herbicides were used.[126] In Iowa, exposure to herbicides through contaminated municipal water was associated with an increased risk of fetal growth retardation in the general population.[127]

Based on experimental data in pregnant animals and several studies of women occupationally or environmentally exposed to pesticides during early pregnancy, it appears that pesticides may be associated with an elevated risk of specific birth defects (including cleft lip and palate, spina bifida and

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[119] Arbuckle,T.E. and L.E. Sever. Pesticide exposures and fetal death: A review of the epidemiologic literature. *Critical Reviews in Toxicology*. 28(3) (1998), pp. 229-270.

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

[121] Mattison, D.R. et al. Reproductive effects of pesticides. In: *The Effects of Pesticides on Human Health. Advances in Modern Environmental Toxicology.*Vol XVIII. Baker S.R. and C.F. Wilkinson, (Eds.) (Princeton: Princeton Scientific Publishers, 1990) pp. 297-389; and Whorton, D. et al. Infertility in male pesticide workers. *Lancet.* 2 (1977), p. 1259.

[122] Lerda, D. and R. Rizzi. Study of reproductive function in persons occupationally exposed to 2,4-dichlorophenoxyacetic acid (2,4-D). *Mutation Research.* 262 (1991), pp. 47-50.

[123] Health Canada. Health Protection Branch. Laboratory Centre for Disease Control. Looking at pesticides and pregnancy. *Farm Family Health.* 6 (1) (Spring 1998b) http://www.hc-sc.gc.ca/main/lcdc/web/publicat/farmfam/vol6-1

[124] Arbuckle,T.E. and L.E. Sever, 1998 *op.cit.*

[125] Ecological studies are 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.

[126] Garry, V.F. et al. Pesticide appliers, biocides, and birth defects in rural Minnesota. *Environmental Health Perspectives.* 104 (1996), pp. 394-399.

[127] Munger R. et al. Intrauterine growth retardation in Iowa communities with herbicide-contaminated drinking water supplies. *Environmental Health Perspectives.* 105 (1997), pp. 308-314.

limb reductions) depending on timing of the exposure.[128] The media has focused attention on reports of isolated cases in Canada, United States and Britain, where women with presumed first trimester exposure to the pesticide *Benomyl*, gave birth to babies with improper development of the eyes (i.e., either no eyes - anophthalmia, or small eyes - microphthalmia).[129]

A recent report by Weidner and colleagues[130] found an increased incidence of malformations of the penis (hypospadias) and of undescended testes (cryptorchidism) in the male children of a study sample of farmers and gardeners.

9.3.3.5 Neurotoxicity

Poisoning from organophosphate pesticides is associated with subsequent chronic or delayed-onset effects. There is evidence that peripheral neuropathy, a condition that affects nerves in the extremities, can manifest weeks after initial acute exposure and may persist or decrease over time.[131]

Cognitive and affective symptoms and long term neuropsychologic defects are also being recognized as chronic sequelae to an organophosphate pesticide poisoning. The largest study has shown that men who had experienced systemic poisoning (n=128), performed significantly worse on sustained visual attention[132] and mood scale[133] tests, out of 10 tests of neurobehavioural functioning performed.[134] Those who had suffered poisonings severe enough to be hospitalized performed relatively poorly on a third test, symbol digit,[135] suggesting a dose-response relationship between exposure and outcome in this case. This study also found some evidence of injury to the peripheral nerves demonstrated as reduced vibrotactile sensitivity[136] with more severe poisonings in general, and deficits in nerve conduction velocity specifically from chlorpyrifos poisoning.[137]

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[128] CALPIRG and PSR, 1998, *op.cit.*

[129] Dyer, 1996, *op.cit.*, p. 1247.

[130] 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), pp. 793-6.

[131] Cherniak, M. Toxicological screening for organophosphate-induced delayed neurotoxicity. *Neurotoxicol.* 9 (1988), pp. 249-272; and Kaloyanova, F. and M. E. Batawi. *Human Toxicology of Pesticides.* (Boca Raton, Fla: CRC Press, 1991)

[132] See: Steenland, K., B. Jenkins, R.G. Ames, M. O'Malley, D. Chrislip and J. Russo. Chronic neurological sequelae to organophophate pesticide poisoning. *Am. J. Epid.* 84(1994), p. 732): A sustained visual attention, or continuous performance test "requires pressing a key quickly when a certain letter appears amid a temporal sequence of various letters."

[133] *Ibid*: Tests of mood scale or affect, "measure the subjects' self-reported transient states of tension, depression, anxiety, fatigue and confusion." Higher scores mean the subject has a more heightened experience of these states and represents a worse performance.

[134] *Ibid*, pp. 731-736.

[135] *Ibid*: p. 732: The symbol digit, or coding speed test, "requires matching digits to symbols as fast as possible following an exhibited matched pattern."

[136] *Ibid*: Vibrotactile sensitivity is one test from a protocol of standard tests for neurological function. It assesses sensitivity to vibration "as a measure of the possible axonal degeneration in the sensory nerves of the index finger and big toe."

[137] *Ibid*, pp. 731-736.

While the focus of this case study is not adult exposures to high doses of pesticides, the above epidemiological findings related to occupational exposure are worth noting. These particularly worrisome health effects may signal the potential for serious long-term health problems and deficits in children because of the greater susceptibility of the developing brain and nervous system.

9.3.4 Human Studies - Chronic Effects

The more controversial and inconsistent data concern effects, especially delayed effects, from chronic, low-level exposure to pesticides.[138] Because this type of exposure has the potential to affect large numbers of children and because the observed health effects are severe, these types of exposures are of significant public health concern.

9.3.4.1 Cancer

There is evidence that rising incidence of certain cancers in children or young adults may be related to pesticide exposures either directly or via parental exposure.

Exposure to organochlorine insecticides and to herbicides such as the phenoxy acids 2,4-D and 2,4,5-T, is deemed to be a risk factor for development of non-Hodgkin's lymphoma (NHL), which, while a relatively rare cancer, has witnessed a rapid, steady increase in incidence worldwide.[139] Swedish researchers have demonstrated the association between NHL and exposure to phenoxy herbicides in a case-control epidemiological study.[140]

A recent report by Daniels and colleagues[141] analyzed the results of thirty-one previous studies examining the association between pesticide exposure and incidence of various childhood cancers. Such meta-analyses allow for greater confidence in the inferences made about health effects from pesticide exposure. This report highlighted some of the considerable difficulties in epidemiological studies where exposure to environmental contaminants is involved. Despite significant problems with lack of standardization in the methods used in these studies, Daniels and co-workers conclude that there is reason to suspect that pre-conceptional, prenatal and early childhood exposures to pesticides are associated with moderate increases in childhood brain tumours and leukemias. Home use of pesticides appeared to account for the greatest risk of these cancers.[142] For example, brain cancer was found to be in association with childhood use of Lindane for lice treatment.[143]

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

[139] Hoffman, W. Organochlorine compounds: Risk of Non-Hodgkins Lymphoma and Breast Cancer?. *Arch. Env. Health.* 51 (1996), pp.189-192.

[140] Hardell, L. and M. Eriksson, A case-control study of non-Hodgkin's lymphoma and exposure to pesticides. *Cancer* 85(6)(1999): 1353-60.

[141] Daniels, J.L. et al. Pesticides and childhood cancers. *Environmental Health Perspectives.* 105 (1997), pp. 1068-1077.

[142] *Ibid*; and Leiss, J.K. and D.A. Savitz. Home pesticide use and childhood cancer: A case-control study. *Am. J. Pub. Health.* 85 (1995), pp. 249-252.

[143] Davis, J.R. et al. Family pesticide use and childhood brain cancer. *Arch. Environ Contam Toxicol.* 24 (1993),

A large retrospective cohort study of Norwegian farm families has determined that there was increased risk of developing certain brain tumours, non-Hodgkins lymphoma, Wilms tumour and other cancers of infancy in farm children, associated with various proxy measures of parental pesticide exposure and use.[144]

There is no single explanation for how pesticide exposure might increase the risk of childhood cancers, however a number of possible causal mechanisms exist. Preconceptional exposure of the parents might increase inherited chromosomal mutations (i.e. those occurring in parents' eggs or sperm). Prenatal exposure from pesticides that cross the placenta may cause chromosomal mutations in the developing fetus. Finally, postnatal incidental exposure might result in alterations in the immune system, hormone functioning or DNA repair mechanisms in the young individual. All of these mechanisms would explain the appearance of cancer at a later stage in life.[145] For a more thorough discussion of the mechanisms of cancer causation, see Section 2.6.9 of Chapter 2.

9.3.4.2 Neurotoxicity

Despite the fact that the tolerable limits for pesticide exposure[146] are frequently based on neurotoxic effects[147] in exposed animals, there has been limited research concerning neurotoxicity of pesticide from real-world *human* exposure. Animal studies suggest that latent neurobehavioural disorders such as impaired endurance and coordination and behavioural alterations observed in adults were caused by exposure to organophosphates and carbamates in the neonate. Case-control and cross-sectional studies in humans have indicated that those with prior pesticide exposure have problems with memory loss and deficits in cognitive function later in life.

The mechanisms of action of certain pesticides such as organochlorines and organophosphates indicate that they do produce neurological symptoms after acute exposure. Subclinical neurological damage may also occur from lower level, long-term exposure to OP pesticides, although these studies are few in number and have inconsistent results, suggesting the need for well-designed, longer follow-up research.[148] Notably, few of these studies have specifically examined the potential for neurotoxic effects in children.

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pp. 87-92.

[144] Kristensen, P., A. Andersen, L.M. Irgens, A.S. Bye and L. Sundheim. Cancer in offspring of parents engaged in agricultural activities in Norway: Incidence and risk factors in the farm environment. *Int. J. Cancer.* 65 (1996), pp. 39-50.

[145] CALPIRG and PSR, 1998, *op.cit.*

[146] The term used to denote the exposure level for humans that will be without adverse health effects is the NOAEL or No Observed Adverse Effect Level. This is typically based on animal tests which determine the different health effects that occur at different levels or timing of pesticide exposure. For chemicals believed to cause cancer, however, there is thought to be no threshold dose, since there is believed to be a risk of developing cancer from even very low levels of exposure. For a thorough discussion of the rationale for calculating "safe" and "tolerable" levels of exposure to environmental contaminants, see discussion of risk assessment in Chapter 4.

[147] Pesticides are most often neurotoxic poisons by design.

[148] Steenland, K. 1996, *op.cit.*

A remarkable, recent study[149] compared two small samples of preschool children in northwestern Mexico for neurobehavioural impacts from pesticide exposure. Children from the Yaqui Valley Indian community are routinely exposed to aerial pesticide spraying as well as daily household bug spraying and there have been high levels of organochlorine pesticides measured in newborn cord blood and breast milk in this community. This group was compared to children from the foothills region, who are less exposed but are similar for several other features that might influence growth and development.[150] Children were assessed for cognition, memory and motor ability.[151] Although there were no differences in growth patterns between the two groups, children from the exposed valley community exhibited impaired stamina, gross and fine motor coordination, memory and drawing ability, as well as other differences in play behaviour. Figure 9.1 shows representative drawings of people done by the children in Guillette and colleagues' study.

[149] Guillette et al. An anthropological approach to the evaluation of preschool children exposed to pesticides in Mexico. *Environmental Health Perspectives.* 106 (1998), pp. 347-353.

[150] Groups were similar for genetic origin, living conditions, social and cultural behaviours, as well as diet and water mineral content.

[151] Children were asked to perform simple repetitive tasks such as jumping up and down on the spot, putting raisins into bottle caps, and catching balls, and they performed memory drills and were asked to draw a picture of a person.

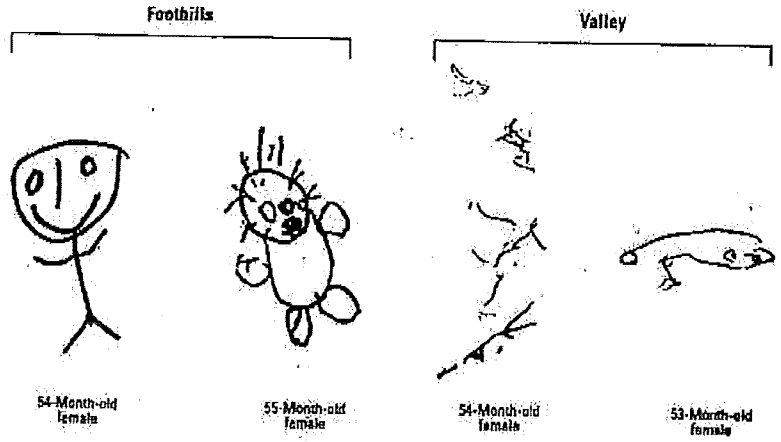

Figure 9.1(a). Representative drawings of a person by 4-year-old Yaqui children from the valley and foothills of Sonora, Mexico.

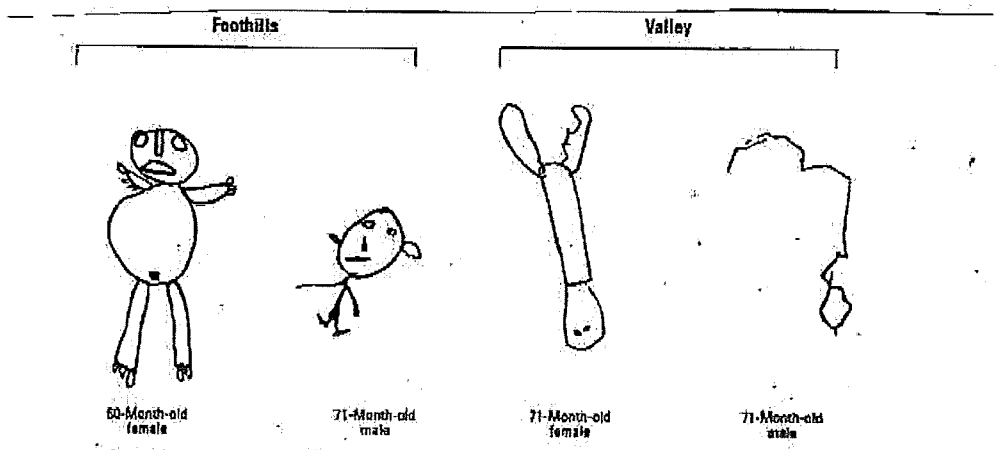

Figure 9.1(b). Representative drawings of a person by 5-year-old Yaqui children from the valley and foothills of Sonora, Mexico.
(Source: Guillette *et.al.*, 1998, *op.cit.)*

Recent reviews of the adverse health effects of exposure to OPs and carbamates have raised the question of their role in the etiology of respiratory problems. Eskenazi and colleagues present the hypothesis supported by some researchers, that there is a biologically plausible association between exposure to OP pesticides and respiratory disease in children. More of the experimental studies of OP exposure have focussed on their effects on the central nervous system. However, because OP pesticides inhibit acetylcholinesterase, they can cause disruption of both autonomic and parasympathetic control of airways and as such, may be important in the occurrence and severity of asthma in the young. This area is under-researched in terms of the risks to humans and there has been no research exploring the role of pesticide exposure in childhood asthma. A small number of epidemiological studies have shown however an association between increased occurrence of asthma among those exposed to select OP and carbamate pesticides.[152]

[152] See for example: Senthilselvan, A. et al. Association of asthma with use of pesticide. Results of a cross-sectional survey of farmers. *Am. Rev. Respir. Dis.* 146 (1992):884-887; Garry, V. et al. Survey of health and use

9.3.4.3 Immune System

There has been increasing attention paid to the possible immune suppressive effects from exposure to pesticides. The World Resources Institute report, *Pesticides and the Immune System*[153] presents evidence, mainly from epidemiological studies in the former Soviet Union, suggesting that pesticides may be linked to a weakening of the immune system which in turn increases susceptibility to infectious diseases and certain cancers. They also cite longitudinal evidence of increased rates of *otitis media* and altered T cell ratios from studies among Canadian Inuit children who are exposed to high levels of organochlorine chemicals (mainly PCBs but including DDT) via breastmilk and traditional food items. Despite the fact that human data to support immune system effects are incomplete, the majority of researchers conclude that this is an important area for further research and critical evaluation of scientific evidence nonetheless.[154]

9.3.4.4 Endocrine Disruption

Organochlorine pesticides have been linked to endocrine disruption involving estrogen, androgen, prolactin and thyroid hormone, as observed from animal studies. These pesticides interfere with hormone function by several mechanisms. Firstly, they may mimic or block at the receptor site for a given hormone. For example, the herbicide atrazine has been shown to have either estrogenic or anti-estrogenic effects depending on the experimental study design.[155] Endocrine disruptors may also act by altering the interaction between hormones and the carrier proteins that transport them through the circulation. For instance, some pyrethroid insecticides are known to displace testosterone from its carrier protein.[156] They are also implicated in causing reproductive developmental abnormalities in wildlife. There have been little data to confirm that these troubling effects occur in humans. Many of these organochlorine pesticides are no longer in use in the western world, however, they are still found in the environment because of their persistence, ability to bioaccumulate, use in many developing countries and long range transport. Levels of DDT and other organochlorine pesticides are detectable in human tissues including breast milk, therefore infants in the Great Lakes region are routinely exposed to these substances at a young age. Accordingly, these health effects are of concern for Ontario's children.

9.3.5 Summary of Human Health Effects from Pesticides

- The potential for the health of children to be affected by pesticides is undeniable.

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characterization of pesticide appliers in Minnesota. *Arch. Environ. Health.* 49 (1994):337-343; and Thrasher, JD, R. Madison & A. Broughton. Immunologic abnormalities in humans exposed to chlorpyrifos: preliminary observations. *Arch. Environ. Health.* 48 (1993): 89-93.)

[153] Repetto, R. and S.S. Baliga, 1996, *op.cit.*

[154] See for example, Repetto & Baliga, 1998, *op.cit.*; Acquavella *et.al.*, 1998a *op.cit.*, and 1998b, *op.cit.*; and Charles Marwick, Provocative report issued on use of pesticides. *JAMA* 275(12) (1996), pp. 899-900.

[155] Connor, K et al. Failure of chloro-s-triazine-derived compounds to induce estrogen receptor-mediated responses *in vivo* and *in vitro*. *Fundam. Appl Toxicol* 30 (1996), pp. 93-101.

[156] Eil, C & Nisula BS. The binding properties of pyrethroids to human skin fibroblast androgen receptros and to sex hormone binding globulin. *J Steroid Biochem.* 35 (1990), pp. 409-414.

- Toxicity studies suggest that young, developing animals are particularly susceptible to adverse effects from pesticides. Animal studies also suggest that the effects of pesticides may be transmitted from generation to generation.
- Children are likely particularly susceptible to the acute and chronic effects of pesticides because of their immature systems, long period of development and their unique behaviour and diet that brings them into greater contact with sources of pesticides.
- Most data on human health effects comes from studies of those who handle pesticides, or from farming communities. Except for clinical information from accidental exposures, there are few epidemiological studies that specifically examine exposures to pesticides in children.
- Depending on the specific pesticide and duration and timing of exposure, pesticides have been associated with a variety of health outcomes in people. There is an association between pesticide exposure and reproductive effects such as decreasing fertility in both males and females, as well as increased risk of spontaneous abortion. Chromosomal abnormalities have been observed after exposure to some pesticides and this also has implications for reproduction. Developmental problems, including appearance of certain birth defects, *in utero* growth retardation and low birth weight have also been observed. There is recent startling evidence of neurobehavioural deficits in Mexican children heavily exposed to pesticides, confirming data from animal studies. There appears to be a higher risk for some childhood cancers such as leukemia and brain tumours associated with prenatal and early exposure to certain pesticides.
- When we assess the effects of pesticides on animals in the lab and the wild, there are other outcomes that are of concern, such as the potential for endocrine disruption, neurobehavioural problems and immune system and respiratory effects.
- While there are certainly gaps in our knowledge, when the weight of evidence from animal models, acute human exposures and epidemiology is considered collectively, the potential for health problems from pesticides respresents an unacceptable risk to humans, and children in particular.

9.4 PESTICIDE REGULATION

As noted in Section 9.1 above, this case study focuses on a review of the activities of the federal government's Pest Management Regulatory Agency (PMRA). The discussions of Risk Assessment and the implementation of the *Food Quality Protection Act* in the United States are particularly relevant to this case study.

Also as noted in Section 9.1.4 above, the following review, despite its length, focuses only on the Pest Management Regulatory Agency (PMRA) and therein, on issues critical to pesticide regulation and the protection of children's health. This review addresses the PMRA risk assessment and risk management processes with respect to new pesticide registrations, the review of pesticides already registered and the evaluation of formulants. It also addresses the PMRA's consideration of pest management alternatives and the management of information. Since the case study was completed in December of 1999, reference to Chapter 4 is necessary for discussion of more recent developments with respect to the PMRA's policy on the use of risk assessment and its proposals for pesticide re-evaluation.

Detailed recommendations are made with respect to improving the transparency and effectiveness of regulating pesticides to protect children's health. Many of the recommendations have to do with the detailed steps necessary to implement a wide range of unfulfilled government commitments with respect to pesticides management. To reiterate, these unfulfilled commitments include the fact that the PMRA has so far failed to:

- adequately implement the Toxic Substances Management Policy;
- develop a regulatory policy on formulants;
- develop a national compliance policy;
- develop a re-evaluation policy and a comprehensive program of pesticide re-evaluation;
- develop a pesticide risk reduction policy;
- produce Proposed Regulatory Decision Documents (PRDD) for proposed registration, re-evaluation and special review decisions;
- create a national database on pesticide use;
- require mandatory reporting of adverse effects by registrants;
- support the integration of pest management with the broader goal of environmental sustainability including setting targets and establishing workplans for the reduction of pesticide use in all sectors.

The analysis that follows addresses these and additional issues from which 45 recommendations arise. The last five of these recommendations have to do with both political will and resources. The federal government's stated commitment to the well-being of Canadian children is hollow if it does not address the undeniable risks of current levels of pesticide exposure.

9.5 THE PEST MANAGEMENT REGULATORY AGENCY

Pesticide regulation in Canada is primarily a function of the federal government, which determines whether a pesticide may be used in this country. The provinces exercise control over the sale and use of federally-approved pesticides. In addition, municipal governments regulate pesticide use on lands and within buildings that they own or control. Increasingly, municipalities are responding to local concerns and are seeking to control pesticide use on privately-owned municipal land, as well.

In 1990, the Pesticide Registration Review Team, a multistakeholder group charged with studying and making recommendations to improve the federal pesticide regulatory system, issued its *Recommendations for a Revised Federal Pesticide Management Regulatory System (hereinafter, The Blue Book)*. The federal government response, a 1994 publication entitled, *Government Proposal for the Pesticide Management Regulatory System (hereinafter, The Purple Book),* outlined its plans for the implementation of Blue Book recommendations. One such recommendation was the creation of a government agency that would facilitate pesticide regulation by a single government department. This recommendation was satisfied in 1995 with the establishment of the Pest Management Regulatory Agency (PMRA), which reports to the Minister of Health.[157]

The Agency's goal is to protect human health and the environment while supporting the competitiveness of agriculture, forestry, other resource sectors and manufacturing.[158] It is responsible for providing safe access to pest management tools while minimizing the risks to human and environmental health. The PMRA administers the *Pest Control Products Act*[159] for Health Canada and sets food Maximum Residue Levels for pesticides on food under the *Food and Drugs Act*.[160] The Agency's specific roles include, but are not limited to the following:

- processing registration applications;
- conducting risk, efficacy and value assessments for potential registrants;
- determining Maximum Residue Limits for pest control products;
- developing effective information and communications;
- reviewing public comments;
- auditing compliance and enforcing the *Pest Control Products Act*;
- coordinating and monitoring the implementation of policies;
- developing risk-reduction policies for all use sectors; and
- identifying areas where research is needed.[161]

The Agency is organized into five divisions:

- The Submission Management and Information Division manages and tracks submissions and conducts scientific screening of potential registrants.
- The Product Sustainability and Co-ordination Division undertakes efficacy, sustainability and value assessments.
- The Health Evaluation Division provides expertise on human health hazards, risk assessment and risk mitigation. It conducts toxicology evaluation and exposure assessments.
- The Environmental Assessment Division provides expertise on environmental hazards, risk assessment and risk mitigation. It conducts assessments of the environmental fate and effects of pest control products.
- The Alternative Strategies and Regulatory Affairs Division directs the development, review and

[157] PMRA. *Strategic Plan 1998-2003* (undated) http://www.hc.sc.gc.ca/pmra-arla/stratp-e.html [hereinafter *PMRA Strategic Plan*].

[158] PMRA. *Overview Document* (undated) [hereinafter *PMRA Overview Document*].

[159] *Pest Control Products Act*, R.S.C. 1985, c. P-9.

[160] *Food and Drugs Act*, R.S.C. 1985 c. F-27.

[161] *PMRA Strategic Plan, op.cit.*

assessment of policies, regulations, programs and legislative amendments, including those related to sustainable pest management.[162]

Several groups advise the PMRA. The Economic Management Advisory Committee includes representatives from among the manufacturers and users of pest control products that are economically impacted by PMRA decisions. The Committee provides advice to the PMRA on mechanisms to improve efficiency and cost effectiveness. The Pest Management Advisory Council includes representatives of pesticide manufacturers, users, and environmental and health groups, as well as individuals with appropriate expertise. It makes recommendations regarding PMRA management, priorities and strategies. It also acts as a forum for the exchange of ideas and advice. The PMRA Policy Council includes the PMRA Executive Director, and the Assistant Deputy Ministers of Agriculture and Agri-Food, Environment, Fisheries and Oceans, Health, Industry and Natural Resources. It provides a forum for the exchange of information and advice between federal government departments and the PMRA. Finally, the Federal - Provincial - Territorial Committee on Pest Management and Pesticides includes provincial and territorial government representatives. It promotes information exchange, the provision of advice and the harmonization of appropriate programs and policies.[163]

The PMRA has committed to fully implementing the regulatory reforms that were identified by the Pesticide Registration Review Team, by the year 2003. Its first strategic objective in that process is to protect health, safety and the environment from the risks of pesticides through the use of sound, progressive science including innovative approaches to sustainable pest management. The PMRA will reportedly meet this objective by strengthening its risk management framework, incorporating the consideration of sustainability into that framework, ensuring that registered pest control products meet current safety standards through re-evaluation and special review, developing innovative approaches to sustainable pest management and ensuring that pest control products are used legally and according to label instructions, among other strategies.[164]

A further strategic objective is to establish an open, transparent and participatory regulatory process. The Agency aims to achieve this objective by establishing a clear regulatory framework, developing an open and transparent decision-making process, soliciting public input on major regulatory decisions and inviting stakeholder participation in regulatory development.

The final objective is to effectively manage the human and financial resources of the PMRA which is to be achieved through a number of initiatives including, but not restricted to work sharing with international partners, increased electronic dependence and the implementation of sound financial management practices.[165]

9.6 THE PEST CONTROL PRODUCTS ACT

The PMRA's authority for pesticide regulation is found in the *Pest Control Products Act*,[166] an act that

[162] *PMRA Overview Document, op.cit.*

[163] PMRA. *Registration Handbook*. 1998. http://www.hc-sc.gc.ca/pmra-arla/hndbk-e.html [hereinafter *PMRA Registration Handbook*].

[164] *PMRA Strategic Plan, op.cit.*

[165] *Ibid.*

[166] *Pest Control Products Act*, R.S.C. 1985, c. P-9.

has been the subject of a lengthy process of prospective amendment. Proposals for legislative amendments were announced by the government in 1995 and are outlined in the government's 1994 *Purple Book*. The primary objectives of the reforms are to enhance health and environmental protection and to increase the openness and transparency of pesticide regulation. The Pest Management Advisory Council is now in discussion with the PMRA regarding the proposed amendments. The *PCPA* amendment process is not covered in any detail in this report.[167]

The existing *Pest Control Products Act* establishes the pesticide registration process and dictates that only registered pesticides may be used in Canada. The regulations to the act specify that in order to approve a pesticide for registration, the registering authority must be of the opinion that:

1) the pesticide's use will not lead to *an unacceptable risk of harm* (emphasis added) to:
(i) things on or in relation to which the control product is intended to be used, or
(ii) public health, plants, animals or the environment; and
2) the product has merit or value for the purposes claimed, when the control product is used in accordance with label directions.[168]

The burden of proof in the registration process lies with the applicant, who must provide sufficient information to allow the PMRA Registrar to determine the safety, merit and value of the product for which registration is sought.[169] However, the act does not define its standard of *unacceptable risk of harm*. Moreover, a clear process, with explicit criteria for determining what constitutes unacceptable risk, has never been laid out,[170] thereby permitting considerable discretion in the assessment of the statutory requirements.

Applicants for whom registration is denied may appeal the decision to an administrative tribunal.[171] The public is denied a corresponding right of appeal when a registration is granted. Once registered, a product retains this status for five years at which point a registrant may apply for renewal. This renewal is generally a *pro forma* exercise which requires little more than confirmation that the product formulation is consistent with documents on register with the PMRA.[172]

Registration of a product may be suspended or canceled by the PMRA when the safety, merit or value of the product is no longer acceptable. The registrant may appeal a suspension or cancellation, in which case the minister must appoint a review board and hold a hearing. The board prepares a report, including recommendations, which it files with the minister. The minister has final power regarding registration of the product.[173] Review boards have been utilized three times. The most recent was in 1985 to hear *Monsanto's* appeal regarding the banning of its herbicide *Alachlor,* one of the pesticides tested by the infamous Industrial Bio-Test Laboratories, indicted in the United States for fraudulent health and safety

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[167] For more information see http://www.hc-sc.gc.ca/pmra-arla/future-e.pdf .

[168] *Pest Control Products Regulations*, C.R.C., c. 1253, ss. 18(c) and (d).

[169] *Ibid.*, s. 9(1).

[170] *Report of the Commissioner of the Environment and Sustainable Development to the House of Commons.* Minister of Public Works and Government Services Canada, 1999 http://www.oag-bvg.ca , *op.cit.* [hereinafter *Report of the Commissioner*].

[171] SOR/92-585, s. 2(F).

[172] *PMRA Registration Handbook, op.cit.*

[173] *Pest Control Products Regulations*, C.R.C., c. 1253.

testing on over 100 pesticides. The *Alachlor* review board recommended reinstatement of *Alachlor's* registration despite finding that the herbicide was a potential human carcinogen. The board also overstepped its authority by basing its findings on *Alachlor* in terms of comparisons and assessments of the commercial viability of its alternative, *Metolachlor*, a pesticide that it had not reviewed in any detail. The review board's report was heavily criticized. In 1988, the Minister of Agriculture issued a decision to maintain the ban on *Alachlor*.[174]

Recommendations

1. The *Pest Control Product Act's* core test for judging the acceptability of a pesticide (*unacceptable risk of harm*) should be specifically defined so that it can be applied in a transparent and consistent manner throughout the risk assessment-risk management process. An essential amendment to the Act, to complement Recommendation 5 below, is to designate persistent and bioaccumulative substances as presenting an unacceptable risk of harm.

2. The *Pest Control Products Act* should be amended to include a requirement to act in a precautionary manner, for example, when the weight of evidence points to the potential for "unacceptable risk of harm". In keeping with this approach, Canada should follow Sweden's lead with legislative amendments to specify inherent characteristics of pesticides that justify de-registration including criteria such as very high acute toxicity, endocrine disruption, probable human carcinogenicity, and neurotoxicity all of which should be considered synonymous with "unacceptable risk of harm."

3. To more effectively implement Recommendations 6 - 31 below, the PMRA should publish a guideline to make its risk assessment and risk management process more transparent. The guideline should include detailed descriptions of its decision-making process including the manner in which children's health interests are taken into account. It may be necessary that the guideline be legislated in the form of a regulation under the *Pest Control Products Act*, in order the ensure that it is implemented.

4. The public should be placed on an equal footing with industry regarding the appeal of a registration decision. To do so, the public must be granted the authority to challenge the approval for registration of pest control products.

Note that several additional recommendations noted in the sub-sections below will involve additional amendments to the *Pest Control Products Act*.

9.7 PMRA AND THE TOXIC SUBSTANCES MANAGEMENT POLICY

The 1995 Toxic Substances Management Policy (TSMP) is a federal government initiative to guide the management of toxic substances (also discussed in Chapter 6). It applies to all substances that are subject to federal regulation. Under the Policy, toxins are categorized as following either Track 1 or Track 2. Track 1 substances are "CEPA-toxic" or equivalent; i.e., they are persistent, bioaccumulate and are primarily the result of human activity. The long-term goal of the TSMP regarding Track 1 substances is their virtual elimination.

Track 2 substances are those that are of concern because of their potential to harm the environment or human health, but which fail to meet all of the Track 1 criteria of CEPA-toxicity: persistence;

[174] See more detailed accounted in: Estrin D. and Swaigen J. *Environment on Trial: A Guide to Ontario Environmental Law and Policy*. 1993, pp. 630-1.

bioaccumulation; and predominantly anthropogenic production. The TSMP aims to control Track 2 substances by preventing or minimizing their release through all stages of their life cycles.[175] Essentially all pesticides can be considered as Track 2 substances since they require some management framework in terms of use and exposure to the public.

The Track 1 list currently includes 12 chemicals, 9 of which are pesticides.[176] Most of the Track 1 pesticides have been used in Canada. All of them are no longer allowed for use although some may enter the country on imported food, flowers, etc., and of course via the movement and cycling of persistent pollutants in the global environment. Notably however, three Track 1 chemicals are or may be found in several commonly used Track 2 pesticides as contaminants generally due to problems with the manufacturing process. The worst example is pentachlorophenol, a commonly used wood preservative for which the manufacturing process unavoidably creates dioxins, furans and hexachlorobenzene (HCB).[177] In other examples, HCB contamination can occur in the pesticides Atrazine and Chlorothalonil, both commonly used across Canada. Dioxin contamination can occur in 2,4-D, Mecoprop and MCPA, also commonly used pesticides. Further, the pesticide Dicofol, registered as an ingredient in 13 pesticide products across Canada is contaminated with DDT, an ingredient used in its manufacture. And finally, the pesticides Endosulfan and Chlopyralid, also in common use in Canada, can be contaminated with HCB or PCBs. As part of a global campaign to eliminate persistent organic pollutants (POPs), some groups have called for the registration of these POPs-contaminated pesticides to be cancelled.[178]

In implementing the TSMP, the PMRA reports that it will assess whether new pest control products are candidates for Track 1 or Track 2 classification. According to the PMRA's 1999 *Strategy for Implementing the TSMP,* new products containing Track 1 active ingredients or formulants will be denied registration. Significantly, exceptions to this rule include emergency use, critical need situations and situations where significant risk reduction can be achieved. The PMRA has not set out which situations this wide range of exemptions embodies. Similar exemptions exist for allowing the approval of new products that contain Track 1 substances as microcontaminants.

Currently-registered products will reportedly also be screened to identify those that contain Track 1 substances. While the focus is on Track 1 substances in active ingredients and formulants, the PMRA implementation plan also states that it will review current levels of microcontaminants in pest control products for their continued acceptability. Virtual elimination is identified as a long term goal for Track 1 substances. More immediate actions that may be taken include strengthening partnerships with industry and users, including efforts to reduce use, and consideration of the possible replacement of chemicals of concern. The PMRA states that it will also use the results of its screening activities to assist in the setting of priorities for re-evaluation and special review.

According to the PMRA, its current management practices meet the TSMP requirements for Track 2

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[175] PMRA. *The Pest Management Regulatory Agency's Strategy for Implementing the Toxic Substances Management Policy.* 1999. Document No. Dir99-03, http://www.hc- sc.gc.ca/pmra-arla/qpubs2-e.html.

[176] The list includes dioxins, furans and PCBs and the pesticides: Aldrin, Chlordane, Dieldrin, DDT, Endrin, Heptachlor, Hexachlorobenzene, Mirex and Toxaphene.

[177] Like many other pesticides requiring reevaluation, Pentachlorophenol has languished on the PMRA re-evaluation shelf since 1992 - see Section 9.9 below).

[178] World Wildlife Fund, Inuit Circumpolar Conference, Inuit Tapirisat of Canada, *POPs in CANADA: Persistent Pollutants, Persistent Threats.* Map, March, 2000.

substances.[179] However, most of this chapter disputes this claim. Given the problems discussed in detail below with respect to existing (currently registered) pest control products, formulants, sustainable pest management and information issues, it is doubtful that current PMRA practices satisfy the TSMP requirement to prevent or minimize Track 2 substance release through life cycle management.

In his 1999 audit, the Commissioner of the Environment and Sustainable Development found that application of the TSMP to pesticide regulation has been limited. The PMRA lacks detailed plans for the removal of substances that are identified for virtual elimination, as well as plans to prevent or minimize releases of pesticides that are identified for life cycle management. The Commissioner recommended that the PMRA identify specific substances subject to life cycle management and that it develop and apply strategies for implementing its management approach.[180]

Recommendation

5. The PMRA should fulfill its commitment to incorporate the TSMP in pesticide regulation. This activity should include immediate bans (or de-registrations) on pesticides which are persistent and bioaccumulative (Track 1 substances) without wasting time and resources on re-evaluation. In keeping with this approach, the PMRA should immediately revise its TSMP Implementation Policy to eliminate the ability to register Track 1 pesticides and to cancel registration of pesticides contaminated with persistent organic pollutants pursuant to the TSMP.

9.8 THE REGISTRATION PROCESS: NEW PRODUCTS

9.8.1 Introduction

The process for determining whether a pest control product is safe for registration has come under review, globally. A number of international initiatives have been developed to coordinate national efforts in this area. The NAFTA Technical Working Group on Pesticides, of which Canada is a member, proposes to develop a common data submission format and a coordinated review process among its membership. The realization of these objectives will require the harmonization of data requirements, relevant test protocols, data submission and study report formats, data review and risk assessment practices, regulatory decision making, and administrative processes and procedures.[181] Similarly, the Organisation for Economic Co-operation and Development's Pesticide Programme works to develop and harmonize test guidelines and assessment methods.[182]

Currently, registration of new pest control products in Canada involves three steps:

i. a decision is made as to whether the pesticide is safe for registration;
ii. a maximum residue limit is established for the product that sets out the maximum quantity of pesticide residue permitted on food products; and finally,

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[179] *Ibid.*

[180] *Report of the Commissioner, op.cit.*

[181] NAFTA Technical Working Group on Pesticides. *A North American Initiative for Pesticides: Operation of the NAFTA Technical Working Group on Pesticides.* November, 1998. http://www.hc-sc.gc.ca/pmra-arla/qinter2-e.html .

[182] For a description of current projects, see PMRA. *PMRA Table of Current OECD Pesticide Projects.* February 1999. Document No. OECD99-01. http://www.hc-sc.gc.ca/pmra-arla/qinter-e.html .

iii. any use restrictions necessary to minimize the risks associated with the pesticide's use are established and must be printed on the product label.

These three processes are described, in turn, in sections 9.8.2, 9.8.3 and 9.8.4, below.

This approach to pesticide registration suffers from a number of significant shortcomings which are addressed in the case study recommendations. Some of the more glaring deficiencies include the PMRA's failure to aggregate pesticide exposure from all media, and its failure to consider the potential cumulative and synergistic effects of exposure to multiple pesticides (all pesticides are considered in isolation from each other and from other toxic substances). These flaws are set out in detail in the discussion below.

9.8.2 Registration

In order to determine whether a pest control product is safe for use and should therefore be registered in Canada, the PMRA carries out product risk assessments.[183] These assessments are carried out on a chemical-by-chemical basis, as new pest control products are submitted for registration. Two risk assessments are undertaken by the PMRA for each new product: one considers the hazards to human health caused by exposure to the pesticide; the other assesses the environmental hazards associated with the pesticide's use.

Each risk assessment involves two major steps. First, any hazards associated with use of the product are identified and assessed. This process is described below in section 9.8.2.1. Secondly, the potential exposure of humans and the environment to the pest control product is estimated, as described in section 9.8.2.2. The hazards and expected exposure levels associated with a pesticide are then used to make a determination about its safety. The PMRA states that its risk assessments for new products include a specific consideration of children and their unique characteristics.[184]

Following completion of the risk assessments, a value assessment of the pest control product is also conducted. This is described in section 9.8.2.3 below. The outcome of all of these assessments is a decision regarding whether to register the pest control product. What remains unclear in PMRA documentation however, is the relative importance that the PMRA assigns in the registration decision-making process to the three determining factors of risk to human health, environmental risk, and pest control product value.

Regulations under the *Pest Control Products Act*[185] specify the range of information required by the PMRA in order to complete the assessments identified above. Information requirements vary depending on the nature of the product, the manufacturing process and the submission (whether it is a new registration or an amendment). Generally, the applicant must submit scientific studies addressing:

- pest control product effectiveness;
- occupational safety;

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[183] Risk assessment is discussed more fully in Chapter 4.

[184] Letter from Christine Norman, Acting Head, Occupational Exposure Assessment Section, PMRA to Canadian Environmental Law Association, May 26, 1999 [hereinafter *PMRA Letter*].

[185] *Pest Control Products Act*, R.S.C. 1985, c. P-9.

- safety to host plant, animal, or the article in relation to which the product is to be used;
- effects to non-target organisms;
- pest control product and residue persistence, retention and movement;
- analysis methods for detecting the control product and its residues in food, feed and the environment;
- detoxification or neutralization methods;
- disposal methods for the control product and its empty packages;
- the stability of the product during storage and display;
- analysis methods for detecting the active ingredient; and
- the compatibility of the control product with other products.[186]

These statutory requirements are supplemented in practice with the requirement for studies of bystander exposure.[187] Where the control product is intended for use on foods for human consumption, the applicant must also provide the results of animal tests that consider the risks of the control product or its residues, to human health.[188] In practice, this requirement has been extended to all pest control products, regardless of whether they are used in connection with food commodities.[189]

9.8.2.1 The Risk Assessment Process: Hazards

Depending on the nature of the pesticide and the application, the PMRA states that its information requirements regarding human health risks could include studies of acute, short-term and long-term toxicity, carcinogenicity, reproductive toxicity, teratology, genetic toxicity, metabolism and toxicokinetics, neurotoxicity, immunotoxicity and endocrine disrupter potential. To provide guidance to registrants regarding which studies are required, the PMRA has characterized pesticides according to their use. Sample pesticide use-site categories (USCs) include Greenhouse Food Crops, Stored Food and Feed, and Indoor Hard Surfaces. Each USC has an associated list of required and conditionally-required data that are set out in data-code (DACO) tables.[190]

Testing for endocrine disruption potential, immunotoxicity and developmental neurotoxicity are not required for all pest control products.[191] According to the PMRA, it uses a two-tiered testing system. Only when certain indicators appear in the core toxicology tests are these more specialized tests required.[192] The PMRA has not made clear, however, how this testing system operates, including an identification of the triggers for additional testing requirements.

In addition, it is not clear whether the PMRA's data requirements test for all potential endpoints or whether the tests that are required are adequate to gauge the risk of causing these endpoints. This is

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[186] *Pest Control Products Regulations*, C.R.C., c. 1253, s. 9(2)(b)(i) to (xi).

[187] *PMRA Letter*, *op.cit.*

[188] *Pest Control Products Regulations*, C.R.C., c. 1253, s. 9(2)(b)(i).

[189] *PMRA Letter, op.cit.*

[190] PMRA. *Organizing and Formatting a Complete Submission for Pest Control Products*. 1998. Document No. Pro98-02. http://www.hc-sc.gc.ca/pmra-arla/qpubs2-e.html [hereinafter *PMRA Submission*].

[191] See *Ibid.*, and PMRA Data Code Tables.

[192] *PMRA Letter, op.cit.* and Personal Communication, Christine Norman, Acting Head, Occupational Exposure Assessment Section, PMRA. June 10, 1999.

particularly important with respect to tests that assess the risks to children's health. For example, the PMRA reports that its neurotoxicity tests are being refined to better investigate the effects of pesticides on neurodevelopment.[193] Even the Minister of Health recognizes the limitations of current PMRA testing requirements: "[a]lthough the current toxicology package allows for an assessment of the potential for endocrine disruption, it is recognized that these test methods can be enhanced to include additional endpoints and that more specific methods need to be developed."[194]

The PMRA evaluates the required studies and makes a judgement regarding the pesticide's safety. Two assessments are involved. The first gauges the risk to human health from occupational and bystander exposure to pesticides. The second considers the risks from exposure to pesticide residues on food and in drinking water. Significantly, the combined risks to human health from these two sources of exposure (occupational/bystander and food/water) are never considered. In other words, the combined or cumulative effects of different sources of exposure to a particular pesticide are never examined by the PMRA. In addition, the effects of cumulative exposure to multiple products with common mechanisms of toxicity are not considered in the PMRA's risk assessment process.[195] This means that all determinations regarding safe exposure levels consider each pesticide in isolation. For example, the herbicide Sulfosulfuron has been found to be carcinogenic to animals. However, the PMRA has concluded that the toxin should pose no carcinogenic risk to humans as long as human intake is below the threshold concentration needed to cause damage. Consequently, the pesticide has been approved for registration, in the absence of a consideration of whether exposure to other chemicals, which may have similar mechanisms of action, would put human intake over the cancer threshold.[196]

To gauge the risk to human health from occupational and bystander exposure, the PMRA identifies the most appropriate no observed adverse effect level (NOAEL) reported in the studies. Choosing which NOAEL is appropriate is based on considerations such as the route and duration of exposure, the species that were tested in toxicity studies and the endpoint of toxicological concern. The PMRA does not explain how this decision-making process takes place.

Once chosen, the NOAEL is divided by uncertainty factors to account for data limitations and response variability.[197] According to the PMRA, additional uncertainty factors are applied beyond the traditional 100-fold uncertainty factor (which accounts for inter- and intra-species difference) to account for severity of endpoint and lack of data.[198] The adjusted NOAEL is intended to quantify a safe level of exposure.

The PMRA next considers the risk posed by exposure to pesticide residue in food and drinking water. The PMRA reports that the most appropriate NOAEL is divided by a safety factor, typically of 100. The PMRA has not set out whether the same NOAEL is used in occupational/bystander exposure assessments and food residue assessments. Nor does the PMRA explain how it determines which safety factor to apply to the NOAEL. This calculation results in the derivation of the acceptable daily intake (ADI). The

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[193] *PMRA Letter, op.cit.*

[194] Letter from the Honourable Allan Rock, Minister of Health, to Julia Langer, World Wildlife Fund, June 29, 1998.

[195] PMRA. *Presentation Materials on Submission Review and Decision-Making and the FQPA.* February 1999 [hereinafter *PMRA Presentation Materials*].

[196] PMRA. *Proposed Regulatory Decision Document: Sulfosulfuron.* 1998. Document No. PRDD98-01 http://www.hc-sc.gc.ca/pmra-arla/qpubs2-e.html [hereinafter *PMRA Sulfosulfuron PRDD*].

[197] *PMRA Overview Document, op.cit.*

[198] *PMRA Presentation Materials, op.cit.*

ADI is the amount of pesticide that toxicologists consider safe for humans to consume every day, for a lifetime. The ADI is used to set maximum residue limits for the pesticide, described below in section 9.8.3.

According to the PMRA, risk assessments for carcinogens are based on the weight of the scientific evidence. The assessments involve an evaluation of the entire data package.[199] The PMRA does not describe how this process is undertaken.

The PMRA reports that the increased sensitivity of the young (especially infants and children), as well as pregnant women, are considered during the risk assessment process to provide additional protection where warranted.[200] However, the PMRA fails to set out under which conditions this additional protection would be warranted or what form it would take.

In addition to the human health assessment described above, the PMRA assesses the environmental risk of using the pesticide. The PMRA examines reports of scientific investigations regarding environmental fate and toxicity, including effects on non-target species. Based on this information, the no observed effect concentration (NOEC) for the pesticide is determined by the PMRA.[201] The NOEC is intended to quantify a safe level of exposure.

Recommendations

6. The PMRA should set out exactly how its two-tiered system of testing requirements functions. The trigger points for additional testing requirements should be made explicit.

7. Several toxicity tests that are currently conditionally-required should become standard requirements. This includes developmental neurotoxicity testing on young animals, which is particularly important for gauging risks to children's health. Similarly, tests for endocrine disruption that are protective of children should be made a standard PMRA test requirement.

8. There is a need for a detailed examination of the toxicity tests required by the PMRA in order to assess their adequacy. An investigation should be undertaken regarding whether the PMRA requires testing for all potential endpoints and whether the tests that are required are adequate to gauge the risk of causing these endpoints.

9. The PMRA should consider the potential effects on human health of occupational/bystander and food/drinking water exposures on an aggregated basis.

10. The PMRA should consider the potential effects on human health of cumulative exposures to pesticides that act via common mechanisms of toxicity.

11. The PMRA should describe how it chooses a NOAEL for occupational/bystander assessments and food residue assessments from the available alternatives.

12. The PMRA should set out how it determines which uncertainty factors to apply to the occupational/bystander and food residue NOAELs.

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[199] *PMRA Overview Document, op.cit.*

[200] PMRA. *PMRA Position on FQPA Science Policies.* 1998.

[201] *PMRA Overview Document, op.cit.*

13. The PMRA should adopt a requirement similar to that found in the U.S. *Food Quality Protection Act,*[202] mandating the application of an uncertainty factor with a minimum value of 10 in order to account for potential pre- and post-natal developmental toxicity and the incompleteness of toxicity and exposure data for children. The uncertainty factor could have a higher value in situations of relatively high uncertainty regarding toxicity and children's exposure.

14. The PMRA should explain precisely how it incorporates considerations regarding the increased sensitivity of the young and pregnant women into its risk assessments and should set out under which conditions it considers additional protection for these groups to be warranted.

15. The PMRA should set out precisely how its risk assessments are undertaken for potentially cancer-causing pesticides.

9.8.2.2 The Risk Assessment Process: Exposure

In addition to assessing the toxicological hazards posed by a particular pesticide, the PMRA estimates the exposure of Canadians to the pesticide. It then compares this expected exposure level to the safe exposure level determined through the hazards assessment, in order to gauge whether use of the pesticide will be safe.

Information regarding expected human exposure levels to a pest control product must be submitted to the PMRA by the registrant. This information includes values for occupational and bystander exposure. The adjusted occupational/bystander NOAEL that is described in section 9.8.2.1 is divided by this estimated exposure level to determine the pesticide's margin of safety (MOS). A MOS of 100 is typically considered acceptable to account for variability in response to pesticide exposure, both within species (differences between adults and children) and between species.[203]

Information regarding expected pesticide residue levels in food and drinking water is also considered and is compared to the ADI that is derived through the process described in section 9.8.2.1. This information is used to set maximum residue limits for the pesticide, as described in section 9.8.3 below.

In addition to data regarding human exposure, information in support of an environmental exposure assessment must be provided by the registrant. These data are used to generate an expected environmental concentration (EEC) for the substance. The most sensitive test species NOEL is then compared to the EEC in the form of a ratio. According to the PMRA, many factors determine how large this ratio must be in order for the risk to be judged acceptable.[204] However, the PMRA does not describe what these factors are, their relative weights and how they are applied. Such clarification is essential to know whether the PMRA adequately accounts for uncertainty and gaps in data and for the unique exposure circumstances of children.

As part of a movement to harmonize their regulatory approaches, the EPA and the PMRA are working together to develop Standard Operating Procedures (SOPs) for residential exposure assessments and a

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[202] *Food Quality Protection Act*, Pub. L. No. 104-170, 110 Stat. 1489 (1996).

[203] *PMRA Overview Document, op.cit.*

[204] *Ibid.*

harmonized post-application exposure guideline.[205]

Recommendation
16. The PMRA should set out which factors it considers when making determinations regarding how large the ratio between the NOAEL for the most sensitive test species and the EEC must be in order for the risks associated with a pesticide to be judged acceptable, as well as their relative weight, and the manner in which they are applied.

9.8.2.3 Value Assessment

Finally, the PMRA considers the value of registering the applicant's product by examining economics (the value of the product to the sector) and sustainability (contribution to integrated pest management; comparison to alternative products and practices). The value assessment also considers efficacy, that is, whether the use of the product contributes to pest management and whether the application rates are the lowest possible to effectively control the target pest.[206]

The PMRA fails to describe how its value assessments are used in pest control product registration decisions and in particular, the weight these assessments are given relative to the risk assessments undertaken by the PMRA.

Recommendation
17. The PMRA should set out how the results of its value assessment are used in the regulatory decision-making process.

9.8.3 Maximum Residue Limits (MRLs)

If a pesticide is intended for use on, or affecting, food commodities, the maximum residue limit (MRL) of the pesticide on food products must be determined as part of the registration process. MRLs are set by the Food Residue Exposure Assessment Section of the Health Evaluation Division of the PMRA, under authority of the *Food and Drugs Act*.[207] This work takes place at the same time as the hazard, exposure and value assessments described above.

Limits are established for parent pesticides as well as any degradation products, metabolites or impurities that are of toxicological concern. Together these compounds are termed the residue of concern (ROC). The applicant is required to develop analytical methods to identify all of the components of the residue of concern.[208]

Applicants are obligated to submit the scientific data necessary to assess whether any residues will result from proposed pest control product use and to determine a MRL. Required information includes:

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[205] PMRA. *The PMRA Position on FQPA Science Policies*. 1999.

[206] *PMRA Overview Document, op.cit.*

[207] *Food and Drugs Act*, R.S.C. 1985 c. F-27.

[208] PMRA. *Residue Chemistry Guidelines*. 1998. Document No. Dir98-02. http://www.hc-sc.gc.ca/pmra-arla/qpubs2-e.html. [hereinafter *PMRA Residue Chemistry Guidelines*].

- the results of plant and animal metabolism studies that determine the metabolic fate, translocation and deposition of the active ingredient after application to plants or administration to livestock;
- pesticide residue in water, fish and irrigated crops following direct application to water;
- pesticide residues in food and feed that result from the treatment of food and feed handling establishments;
- residue deposition in meat, milk, poultry and eggs;
- residues on and in plant material following pesticide exposure;
- data regarding whether residues in raw commodities degrade, reduce or concentrate during food processing; and
- data to assess whether pesticide residues are stable during the storage of analytical samples.[209]

The PMRA also relies on information regarding the relative consumption of various crops in the Canadian diet. This information is used to guide pesticide testing requirements. Crops that constitute a greater proportion of Canadians' diet require a higher number of crop residue trials. These trials measure the type and amount of residue left on a particular crop, growing in a variety of locations, when a pesticide is applied according to label directions.

To set an MRL, the registrant proposes a value which, based on field trial data, reflects the maximum residue level that could occur on food at the point of sale. The MRL must include all components of the ROC and should reflect residue values on the raw agricultural commodity. The MRL should be large enough to include any residue values that could reasonably be expected, and should not be an average value.[210]

The proposed MRL is then used to generate potential daily intake (PDI) estimates of pesticide residue, based on food and drinking water consumption patterns.[211] To estimate consumption patterns, the PMRA reports that it relies on the 1996 US Department of Agriculture Continuing Survey of Food Intakes by Individuals.[212] In addition, as part of a movement to harmonize its pesticide regulatory approach with that of the US Environmental Protection Agency (EPA), the PMRA reports that it has adopted the computer-based probabilistic models used by the EPA to assess dietary exposure. The PMRA has stated that it intends to harmonize as far as possible with the EPA on the use of models (including probabilistic models) to generate better estimates of drinking water exposure. Finally, the PMRA reports that it plans to begin aggregating dietary and non-dietary exposure data..[213]

The MRL is accepted on condition that the PDI will not exceed the pesticide's acceptable daily intake (ADI). According to the PMRA, residue consumption assessment includes consideration of different consumption patterns, including those of children.[214]

If the PDI exceeds the ADI, the PMRA applies use restrictions, such as approving the pesticide for fewer crops, imposing lower application rates, or increasing the time between spraying and harvest, in order to

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[209] *Ibid.*

[210] *Ibid.*

[211] *PMRA Overview Document, op.cit.*

[212] Personal Communication, Danielle Prevost, Information Officer, PMRA. September 30, 1999.

[213] PMRA. *The PMRA Position on FQPA Science Policies.* 1999.

[214] *PMRA Overview Document, op.cit.*

lower the PDI.[215]

A serious limitation of this process is the fact that the exposure estimate only considers pesticide intake from food and water sources. Other sources, such as soil and dust, are significant for children. In addition, no consideration is made of the cumulative intake of pesticides that act via similar mechanisms of toxicity.

The NAFTA Technical Working Group on Pesticides reports that it plans to take steps to minimize trade problems resulting from different maximum residue limits for commodities that are traded among the three member countries. Concern has been expressed that in harmonizing MRLs among trading partners, there will be pressure to weaken Canadian MRLs in order to place them in line with the least stringent standards of its trading partners. The outcome could be universally weak standards.[216]

Recommendations

18. Pesticide intake via soil and dust should be included in exposure estimates.

19. The PMRA should consider cumulative exposure to multiple pesticides that act via similar mechanisms of toxicity in its risk assessments.

20. The PMRA should ensure that the negotiation of MRLs between trading partners is a transparent process and that the strength of Canada's MRLs is not compromised.

9.8.4 Use Restrictions[1]

Use restrictions may be employed as an additional risk management tool for new pest control products that are approved for registration via the process describe above. These restrictions are used to manage pesticide exposure in order to ensure that it does not exceed the pesticide's acceptable daily intake. The restrictions can prohibit product use on certain crops, vary post application intervals including the interval between the last application and harvest, vary application rates and frequencies, prohibit the use of the product around environmentally sensitive areas and impose restrictions related to conditions such as wind speed at the time of use. Any measures determined to be necessary in order to bring the risks associated with a pesticide within acceptable limits must be set out, in detail, on the product label.[218]

Use restrictions can be employed as a primary method of risk management. For example, the herbicide Sulfosulfuron has been found to be very toxic to non-target terrestrial and aquatic plants, and it is reported that spray drift and run off following application have the potential to significantly affect terrestrial and aquatic plant habitat. The PMRA's management solution for this potentially significant source of contamination is to require the establishment of a buffer zone of 30 m between sprayed areas and sensitive terrestrial areas, and a zone of 6 m between sprayed areas and the edge of sensitive aquatic

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[215] Personal Communication, Christine Norman, Acting Head, Occupational Exposure Assessment Section, PMRA, October 6, 1999. See also next section, 9. 8.4, for discussion of pesticide use restrictions.

[216] Canadian Environmental Law Association, *The Environmental Implications of Trade Agreements*, CELA Brief No. 231, August, 1993.

[217] Use restrictions may be imposed by provincial pesticide regulators, as well. The division of responsibilities over pesticides between the federal and provincial governments is described in Chapter 3.

[218] PMRA. *Regulatory Decision Making*. January 1998.

areas, as well as label instructions not to spray prior to forecasted rain.[219]

Given the high toxicity of many pest control products to non-target organisms, and high level of reliance that can be placed on the applicator to minimize the associated risks, it is vital that the PMRA effectively monitor and enforce use restrictions. Yet the PMRA has failed to adequately fulfill this role, as described in Section 9.12.2 below.

In its 1994 Purple Book commitments, the government pledged to legislate strengthened *Pest Control Product (PCPA)* enforcement provisions and to develop a national compliance policy.[220] According to the PMRA, it currently employs a number of strategies for ensuring compliance with the *PCPA*. Inspections are used for educational purposes and to assess compliance. PMRA inspectors can also undertake an investigation when they suspect *PCPA* infractions. An investigation involves information gathering and evaluation, and appropriate enforcement measures.
The range of possible enforcement measures include:

- *registration cancellation or suspension*
- *education letters*: if there is a problem but no infraction, or when the infraction cannot be attributed to the person in question
- *notice of violation warnings*: if an infraction is detected for the first time and has not caused significant harm
- *imposition of violation penalties*: if a previous warning has been issued, or the infraction presents a risk, etc.
- *prosecution*: in more serious situations.[221]

The PMRA has not developed a national compliance policy.[222] In his 1999 report, the Commissioner of the Environment and Sustainable Development assessed the PMRA's enforcement record. Incredibly, the Commissioner reported that the PMRA has the equivalent of a mere 44 officers to inspect farms, food processing plants, commercial application facilities, retail outlets, pesticide registrants and formulators, lawn care companies, and others, *nationwide.* Not surprisingly, the Commissioner found that, "[t]he PMRA does not know the extent to which users comply with directions on pesticide labels."[223] The Commissioner found that inspections are primarily undertaken in response to known or suspected violations, and are not used to systematically monitor compliance.[224]

In addition, the Canadian Food Inspection Agency, the government agency responsible for all federally-mandated food inspection, reports that the rate of pesticide MRL exceedance on domestic crops tripled between 1991 and 1998. This increase is the result of several practices including: application of a pesticide to a crop for which it is not approved, application of more than recommended amounts of pesticide and the application of a pesticide too close to the harvest.[225]

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[219] *PMRA Sulfosulfuron PRDD, op.cit.*

[220] *PMRA Strategic Plan, op.cit.*

[221] PMRA. *Compliance and Enforcement Policy Guideline*. 1998. Document No. B98-01. http://www.hc-sc.gc.ca/pmra-arla/qpubs3-e.html .

[222] Personal Communication, Danielle Prevost, Information Officer, PMRA. September 30, 1999.

[223] *Report of the Commissioner, op.cit.*, at 4-30.

[224] *Ibid.*

[225] Eli Neidert and Glenn Havelock, Canadian Food Inspection Agency. *Report on Levels and Incidences of Pesticide Residues in Selected Agricultural Food Commodities Available in Canada During 1994-1998.*

Within the context of clearly inadequate inspection and enforcement capacity, heavy reliance upon pesticide label instructions and restrictions to avoid pesticide risks to human and environmental health, is a poor risk management strategy.

Recommendation

21. The PMRA should reduce the reliance on pesticide label instructions and restrictions for the management of pesticide risk to human and environmental health and in the interim, given the importance of label compliance, the PMRA should improve its inspection and enforcement operations to ensure appropriate pesticide use. The PMRA must not hesitate to apply the full range of enforcement penalties that are available to it, in order to guarantee compliance. Enhanced enforcement should be guided by a national compliance policy, which the PMRA committed itself to develop in its 1994 *Purple Book.*

9.9 Existing (Currently Registered) Pest Control Products

The discussion thus far has focused on products that are new to the regulatory system. Although the data requirements for these new pest control products have improved with time, over 300 of the 500 active ingredients found in currently-registered pest control products were approved prior to 1981, and more than 150 others were originally registered pre-1960.[226] Health and environmental standards were less stringent and scientific methods less reliable when these pesticides were registered for use. For example, until recently, estimates of pesticide exposure through food consumption relied on a 1975 Department of National Health and Welfare study, *Nutrition Canada Food Consumption Patterns Report.* Incredibly, according to this outdated study, no significant differences existed between the diets of infants and the general population, with the exception of oats.[227] This information is clearly inaccurate.[228]

Although registration of existing products expires every five years, registration renewal is a formality and is routinely given as a matter of course. The only inquiry that takes place at the time of re-registration considers whether the product label information and formulation ingredients are consistent with those that are held on file by the PMRA. No consideration is given as to whether these older products satisfy current assessment requirements or whether their MRLs are within current ADI levels.[229]

The PMRA committed itself, in its *Strategic Plan*, to remedy this problem by means of a comprehensive program of pesticide re-evaluation.[230] The Agency defines re-evaluation as the assessment and reconfirmation of the acceptability of older compounds in light of modern technology and scientific standards.[231] This language reveals a bias towards maintaining the *status quo* and upholding the registration status of older pesticide products.

November, 1998.

[226] *Report of the Commissioner, op.cit.*

[227] *PMRA Residue Chemistry Guidelines, op.cit.*

[228] See section 9.2.3.2 above.

[229] *PMRA Registration Handbook, op.cit.*

[230] Pesticides may be re-evaluated at any time to ensure their safety, under authority of the *Pest Control Products Regulations*, s. 19.

[231] *PMRA Strategic Plan, op.cit.*

The PMRA's re-evaluation commitment is to review registered products and their maximum residue limits to ensure that they meet current safety standards. Its goal is to re-evaluate all products registered as of December 31, 1994, by 2005/6.[232] It proposes to do this in coordination with the United States Environmental Protection Agency and the European Union, through shared pesticide reviews. The rationale is to save time and resources, and to eliminate the potential for trade difficulties resulting from differential pesticide standards among these trading partners. In particular, the PMRA plans to draw heavily from US data reviews.[233]

In certain circumstances, the PMRA can also undertake a "special review" of an existing pest control product.[234] These reviews are initiated when specific concerns about a product, based on new evidence or regulatory action taken in other countries, indicate that there may be a significant risk of harm to human health, safety or the environment, or that the product is no longer efficacious. In contrast to re-evaluation, which is meant to be undertaken on all older pesticides in order to assess compliance with current risk assessment requirements, special reviews are carried out in response to indications that there may be a problem with a particular pesticide. The PMRA concluded a special review of Carbofuran in 1998 after concerns were expressed regarding its effects on wildlife. To date this is the only special review that the PMRA has initiated,[235] although it has indicated that it plans to review pest control products containing the organochlorine insecticide *Lindane*.[236]

The Commissioner of the Environment and Sustainable Development examined pesticide re- evaluation and special review in his 1999 Report.[237] He noted that the federal government has formally recognized the need for pesticide re-evaluation for over 13 years but has failed to meet its long-standing commitment to implement a re-evaluation program. Prior to the creation of the PMRA, Agriculture Canada made commitments to re-evaluate priority pesticides that were not fulfilled. Similarly, the PMRA was charged in 1995 with the development and implementation of a pesticide re-evaluation program. Despite these commitments, the Commissioner found that:

> [t]here is no clearly delineated process that identifies steps to be followed in undertaking re-evaluations or special reviews, the roles of each of the various participants, the criteria to be used in making decisions, and the respective accountabilities.[238]

In contrast, the US legislated a pesticide re-evaluation requirement in 1988 that has resulted in a number of pesticide use de-registrations.[239]

The PMRA has committed no budgetary allowances to re-evaluation. Instead, funds for this endeavour are to come from efficiencies in other areas of PMRA's functions. Specifically, resources that result from

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[232] Pest Management Advisory Council. *Draft Meeting Report*. April 16, 1999.

[233] *PMRA Strategic Plan, op.cit.*

[234] Under authority of s. 19 of the *Pest Control Products Regulations*.

[235] *Report of the Commissioner, op.cit.*

[236] PMRA. *Special Review of Pest Control Products Containing Lindane*. 1999. Document No. SRA99-01. http://www.hc-sc.gc.ca/pmra-arla/qpubs2-e.html .

[237] *Report of the Commissioner, op.cit.*

[238] *Ibid.*, at 3-32.

[239] *Ibid.*

efficiencies and cost savings are to be shifted from the area of new product evaluation.[240] It is unclear exactly what this means. Moreover, revenues generated this way have been much lower than PMRA predictions, resulting in delays in re-evaluation activities. A study comparing government expenditures on the registration of new products versus the re-evaluation of old products found that Canada lags behind the US, UK and Australia in its financial commitment to re-evaluation.[241]

In his report, the Commissioner concluded that:

> Canada's track record for the re-evaluation of pesticides [is] one of inaction and unfulfilled commitments" and that, "[w]ithout an effective re-evaluation program, there is no assurance that Canadians are not being exposed to unacceptable risk.[242]

The Commissioner recommended that the PMRA develop and implement a program of re- evaluation that identifies priorities and a schedule for completion, and that the PMRA develop and document the processes to be followed for pesticide re-evaluations and special reviews. The processes should include a clear definition of responsibilities, timelines and reporting. In addition, the Commissioner recommended that priorities for re-evaluation should be determined in consultation with other government departments including Environment Canada, Health Canada, Natural Resources Canada, Fisheries and Oceans, as well as other stakeholders.[243]

Recommendations[244]

22. The PMRA should expeditiously complete on-going re-evaluations including several that were initiated close to 20 years ago, such as for pentachlorophenol.[245]

23. The PMRA should fulfill its commitment to establish a comprehensive pesticide re-evaluation and special review policy that includes responsibilities, methods for reporting and systems of accountability. The special review process should clearly set out the conditions necessary to trigger a special review. The PMRA should establish a re-evaluation program that sets out priorities and firm deadlines.

See also: Recommendation number 44, in section 9.13 below.

9.10 FORMULANTS

In addition to active ingredients, which are the substances that cause harm to a pest, many pesticides contain ingredients called formulants or inerts. These substances are added for a number of reasons such as to facilitate application or enhance effectiveness, and can often represent over 90% of a pesticide formulation. The concern with formulants is that many are toxic substances. In fact, some substances that are used as formulants in one pesticide product are the active ingredient in others. Some have been

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[240] *Strategic Plan, op.cit.*

[241] *Report of the Commissioner, op.cit.*

[242] *Ibid.*, at 3-31.

[243] *Ibid.*

[244] See also Recommendations for Sustainable Pest Management, Section 9.11 below.

[245] *Report of the Commissioner, op.cit.*The re-evaluation of pentachlorophenol was initiated in 1992.

classified as hazardous air and water pollutants (naphthalene)[246] and others are known carcinogens (crystalline silica, ethylbenzene).[247] Still others have been linked to birth defects (ethylbenzene),[248] central nervous system disorders (xylene),[249] and damage to internal organs (chlorobenzene, toluene).[250]

In its *Blue Book* of 1990, the Pesticide Registration Review Team recommended that the federal government develop a regulatory policy on formulants and in its 1994 response, the government committed the PMRA to develop such a policy.[251] Despite this commitment, the PMRA does not have a written policy regarding the regulation of pesticide formulants. Moreover, the PMRA's progress in addressing formulant safety is constrained by resource restrictions.[252] In practice, the PMRA closely follows the U.S. Environmental Protection Agency (EPA) approach to formulant regulation. The PMRA reports that it is currently developing a formulant policy that will also be based on EPA work.[253]

9.10.1 EPA Regulation

The EPA's policy on pesticide inerts took effect in 1987. On the basis of toxicity, it characterized the existing 1200 pesticide inerts as belonging to one of 4 lists:

- List 1: Inerts of toxicological concern based on carcinogenicity, adverse reproductive effects, neurotoxicity, other chronic effects, developmental toxicity, ecological effects and the potential for bioaccumulation. There were initially approximately 50 substances on this list; there are now 8.
- List 2: Potentially toxic inerts/high priority for testing. Many are structurally similar to those known to be toxic. There were initially approximately 60 substances on this list.
- List 3: Inerts of unknown toxicity. Inerts are placed on List 3 when there is no basis for listing them on any of the other three lists. Initially there were approximately 800; there are now 1500 listed substances.
- List 4: Inerts of minimal concern. There were initially approximately 300 listed substances.[254]

The composition of these lists has changed as substances were found to no longer be present in pesticide products, were reassigned to other lists, were reclassified as active ingredients or as their use was phased out. Inert classification can change at any time as new information becomes available.[255]

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[246] Northwest Coalition for Alternatives to Pesticides. *Worst Kept Secrets: Toxic Inert Ingredients in Pesticides.* January, 1998.

[247] Are "Inert" Ingredients in Pesticides Really Benign? In: *Journal of Pesticide Reform*, Summer 1999, Vol. 19, No. 2.

[248] *Ibid.*

[249] Northwest Coalition for Alternatives to Pesticides, 1998, *op.cit.*

[250] Hammond, M., Citizens for Alternatives to Pesticides. *Pesticide Bylaws: why we need them; how to get them.* 1995.

[251] *PMRA Strategic Plan, op.cit.*

[252] Facsimile document from Doreen Riedel, Evaluation Officer, PMRA, August 6, 1999.

[253] Personal Communication, Doreen Riedel, Evaluation Officer, PMRA, June 29, 1999.

[254] http://www.epa.gov/opprd001/inerts .

[255] Facsimile document from Doreen Riedel, Evaluation Officer, PMRA, August 6, 1999.

The EPA's regulatory efforts initially focused on List 1. It has taken a number of steps to better regulate these compounds including: encouraging registrants to substitute inerts on Lists 1 and 2 with other inerts; requiring that List 1 substances be included on product labels; and requiring that inerts of toxicological concern that remain in products are subject to data call-ins, that is, that registrants be required to provide the EPA with adequate toxicity and exposure data, as well as data on environmental fate, ecological effects and residue chemistry, to permit the EPA to assess product safety.[256]

The EPA has completed its assessment for the majority of current List 1 substances. It is now undertaking a similar assessment of List 2 substances, including a data call-in. In addition, the EPA has begun toxicological and ecological assessments to support reclassification determination for List 3 inerts. In the interim, use of these substances, including the 1500 List 3 formulants of unknown toxicity, continues.[257] In addition to these activities regarding existing formulants, the EPA requires that a minimal data set and scientific review be undertaken for all new inert ingredients.[258]

The Northwest Coalition for Alternatives to Pesticides (NCAP) is critical of the EPA's progress in reducing the dangers from toxic inert ingredients since the 1987 launching of its inert policy. This group found that a high number of inerts on the EPA's lists are what the NCAP calls "active inerts." These inerts have been, or continue to be registered for use in pest control products as active ingredients. Because these substances must be registered for use as active ingredients, the EPA necessarily has significant toxicity information on these substances. Yet 70% of the active inerts used at the time of the NCAP's inquiry were categorized as List 3 substances, that is, substances of *unknown* toxicity, with the associated lenient regulatory requirements.

In addition, using the US EPA inert lists of 1995, the NCAP found that a high number of inerts, including List 3 inerts, are considered hazardous air and/or water pollutants under the US *Clean Air* and *Clean Water Acts.* Several inerts, some of which are List 3 inerts, are classified as extremely hazardous substances under the US *Superfund Amendments and Reauthorization Act.* Another group of inerts, many of which are once again found on List 3, have been identified as occupationally-hazardous substances by the US Occupational Safety and Health Administration. Moreover, a number of listed inerts, the majority of which are found on List 3, include substances identified by the International Agency for Research on Cancer (IARC) and the US National Toxicology Program as known or suspected carcinogens. In 1987, when the EPA inerts policy was introduced, a number of chemicals such as bitumens, butylated hydroxyanisole and potassium bromate had been identified by the IARC as possible carcinogens. Coal tar was a known carcinogen at the time. Ten years later, these substances remained on List 3. Moreover, because inerts are not tested for carcinogenicity, it is impossible to know how many more List 3 inerts are possible carcinogens.

The NCAP has concluded that based on the identified hazards of List 3 substances described above, it is dubious whether the EPA's policy of encouraging pesticide manufacturers to replace List 1 and List 2 substances with List 3 substances will be any more protective of human and environmental health. NCAP calls for full ingredient disclosure on all pesticide products and extensive toxicity testing on the end use product, which would include any inert ingredients.[259]

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[256] http://www.epa.gov/opprd001/inerts/fr52.htm .

[257] http://www.epa.gov/opprd001/inerts/lists.html .

[258] http://www.epa.gov/opprd001/inerts/fr52.htm

[259] Northwest Coalition for Alternatives to Pesticides, 1998, *op.cit.*

9.10.2 PMRA Regulation

The PMRA largely follows the EPA approach to formulant regulation. However, the PMRA does not have comprehensive data regarding which formulants are in Canadian-registered products, and which of these are found on the EPA inerts lists. Failing adequate categorization of Canadian-registered formulants, it is doubtful that these substances are being effectively regulated. The PMRA reports that it is addressing this critical information gap.[260]

Under current PMRA practice, those formulants that are identified as EPA List 1 substances are only permitted for use in Canada when there are no substitutes for these substances, or when there is a negligible potential for exposure to these formulants. List 1 formulants may also be approved for use in certain use site categories. Registrants must request a waiver to use List 1 formulants and they must list these substances, as well as their concentrations, on their product labels.[261]

According to the PMRA, this has not always been the practice with List 1 substances, which were not as tightly screened in the past. Moreover, in practice, use of List 1 formulants may not always respect regulatory restrictions. For example, List 1 formulants may not consistently be listed on product labels. The PMRA reports that it is currently addressing this problem.[262]

In 1998, the Ottawa-based Campaign for Pesticide Reduction (CPR!) obtained a PMRA list of the over 4700 formulants present in pesticides registered in Canada. According to this group, only 2.4% of the substances present on the PMRA list are categorized by the EPA as known or possible toxins. The remainder are classified as being of unknown toxicity.[263] Given the known hazards associated with a large number of List 3 inerts, which are described above, these substances should be more tightly regulated. Continued use of those List 3 substances for which there is limited data regarding potential adverse effects should be prohibited. It is unclear what work the PMRA is undertaking to address the gaps in information that exist regarding the safety of these substances.

In addition to the individual formulants that are identified on the EPA lists, many pest control products contain formulant mixtures. These mixtures are often sold under a trade name and their constituents are not disclosed. Most formulant producers are American. Canadian producers of pesticide active ingredients purchase formulants from these companies or from their Canadian partners. In most cases, the pesticide registrant is not aware of the composition of the formulant mix; it is merely added to the active ingredient and offered for sale. The composition of these formulant mixtures is generally considered to be confidential business information.

Consequently, in effect, the EPA does not list these mixtures on its inerts lists and it does not disclose their composition to third parties, including the PMRA. The EPA has access to this information through its legislated right to demand such information from suppliers. There is no corresponding legal obligation in Canada.[264] Consequently, in effect, the PMRA has been forced to seek this information from U.S. formulant producers.

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[260] Facsimile document, Doreen Riedel, Evaluation Officer, PMRA, August 9, 1999.

[261] *Ibid.*

[262] *Ibid.*

[263] Campaign for Pesticide Reduction. *Pesticide Watch.* Vol 2, Issue 1. April 1998.

[264] Personal Communication, Doreen Riedel, Evaluation Officer, PMRA, June 29, 1999.

The PMRA also manages individual formulants that are not included on the EPA inerts lists. When a non-EPA-listed substance is identified in a Canadian-registered product, a reviewer searches on-line databases for toxicity information on the formulant. The PMRA reports that once its Formulants Policy is in place, it will be able to obtain this information directly from the manufacturer. It is proposed that the PMRA will require the same data as are currently required by the EPA for formulants. These include 90-day oral and dermal toxicity, genotoxicity and ecotoxicity studies. The results of these studies are used to determine whether long term studies are also required for risk assessments.[265]

In the meantime, PMRA documentation indicates that the required testing on non-EPA listed formulants is minimal.[266] According to PMRA documents, toxicological testing is undertaken on pesticide active ingredients and on "end-use products," which are the final formulation of the active ingredient combined with any formulants. PMRA toxicological testing requirements for end-use products, which capture the potential adverse effects of formulants, are far less stringent than those for active ingredients. In fact, in a recent Decision Document regarding the registration of the herbicide Sulfosulfuron, the PMRA stated that the environmental impact of a pesticide is assessed through studies on the product active ingredient. The PMRA may then request studies on the formulated product, on a case-by-case basis, if there are reasons for concern. In the case described, no environmental toxicity studies of the final mixture were required.[267] In contrast to testing requirements on the active ingredient, no long-term animal toxicology or special studies (includes multigeneration-reproductive, teratogenicity, genotoxicity and neurotoxicity tests) are required on the end-use product.[268] Moreover, examining the data requirements for specific Use Site Categories,[269] there are far fewer environmental toxicity data requirements for end-use products than for active ingredients. In addition, all of the end-use product data are conditionally, and not mandatorily required.

The PMRA states that it is concerned about the additional costs that will be imposed on pesticide manufacturers when they are forced to make changes to their pesticide registrations due to revisions in formulant requirements.[270]

With the exception of List 1 substances and petroleum distillates, the names of other formulants present in a pesticide need not be disclosed on the product label.[271]

Recommendations

24. The PMRA should expeditiously fulfill its commitment and complete development of its policy on formulants. The PMRA should release its policy to the public for comment and revision. Once completed, the PMRA should effectively implement and enforce its policy. The policy should set out how the PMRA will use the EPA formulant classification system and toxicological database. The policy should also include an explicit enumeration of rigorous testing requirements for new and non-EPA-listed

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[265] Facsimile document from Doreen Riedel, Evaluation Officer, PMRA, August 6, 1999.

[266] See, for example, Use Site Data-code Tables and *PMRA Submission, op.cit.*

[267] *PMRA Sulfosulfuron PRDD, op.cit.*

[268] *PMRA Submission, op.cit.*

[269] Livestock for Food and Terrestrial Food Crops.

[270] Personal Communication, Doreen Riedel, Evaluation Officer, PMRA, June 29, 1999.

[271] *Registration Handbook, op.cit.* and Facsimile transmission from Doreen Riedel, Evaluation Officer, PMRA, August 6, 1999.

formulants. These requirements should be effectively enforced.

25. The PMRA should immediately complete its assessment of formulants in Canadian-registered pesticides in order to determine which are on the EPA lists and which are not. This is a vital precursor to effective pesticide regulation.

26. The PMRA should more effectively regulate the use of List 3 formulants of known or suspected toxicity. The PMRA should aggressively investigate the safety of List 3 formulants that truly are of unknown toxicity. In accordance with the precautionary principle, use of these formulants should be prohibited until their potential effects are understood.

27. All formulants should be listed on pest control product labels. The requirement to include List 1 substances on product labels should be more aggressively enforced.

28. The PMRA should make active use of the re-evaluation process to assess the safety of formulants that until now, have not been rigorously considered.

29. The PMRA should be granted legislative authority to demand formulant composition information from registrants. It is unacceptable that acquisition of this information is contingent on the good will of U.S. formulant suppliers.

30. The PMRA should expedite its work on the identification and risk assessment of non-EPA-listed formulants that are present in products registered in Canada. Pesticide registrants should be required to provide the PMRA with adequate data to assess the toxicological hazard of such formulants.

31. In his next report, the Commissioner of the Environment and Sustainable Development should investigate the adequacy of PMRA measures to ensure the safety of pest control product formulants.

See also: Recommendation 45, in section 9.13 below.

9.11 SUSTAINABLE PEST MANAGEMENT

In its 1990 *Blue Book*, the Pesticide Registration Review (PRR) Team recommended that the government establish a Pest Management Promotion Office. The office was intended to harmonize pest management with the goal of environmental sustainability, including the development of targets and work plans for the reduction of pesticide use. In its 1994 response to the PRR Team's recommendations, the government committed itself to the establishment of an Alternatives Office within the PMRA in order to fulfill this objective including, in particular, the development of a pesticide risk reduction policy.[272]

An Alternative Strategies and Regulatory Affairs Division has since been created within the PMRA. It is charged with encouraging the development and adoption of sustainable pest management systems. Its functions include providing leadership and support in the development of policies, programs and projects related to sustainability.[273]

According to the PMRA, pest management systems that are compatible with sustainable development are

[272] *PMRA Strategic Plan, op.cit.*

[273] *Ibid.*

those that:

- meet society's needs for human health protection, food and fibre production and resource utilization;
- conserve or enhance natural resources and the quality of the environment for future generations; and
- are economically viable.[274]

An important component of sustainable pest control is integrated pest management (IPM). IPM aims to prevent pest problems from occurring and eliminate or reduce reliance on chemical pesticides. Some components of IPM can include cultural strategies such as good farming methods, the use of pest-resistant crop varieties, adapting planting times, maintaining soil fertility and nutrient balance, and preserving biodiversity. A further strategy is to ensure the presence of natural pest enemies.

Integrated Pest Management definitions and practices vary, and herein lies the problem. "Classical" IPM, applied in conventional agricultural systems, allows the use of pesticides in some instances. In contrast, IPM that is part of sustainable agricultural systems strictly adheres to the use of ecologically-sound management practices. Whether this includes chemical pesticide use, in tightly restricted conditions, is controversial. This form of IPM sees economic factors as being secondary to ecological integrity, even if the result is diminished profit margins. It also involves redesigning agricultural systems so that they are ecologically sound in terms of energy, water, mineral and biotic cycles, as well as being as pest-stable as possible.[275]

The danger with these divergent visions is that they are all advanced, by different groups, under the name of IPM. The result is that IPM has lost meaning. This is particularly dangerous when regulators, whose intentions are to make subtle, if any real change to the status quo, commit to implementing IPM. Reliance on the ambiguous IPM concept permits these agencies to put off instituting the type of real change that is necessary in order to move towards true sustainability in agricultural practices.

According to the PMRA, IPM aims to ensure that pesticide application takes place only when warranted, at the most appropriate time, and that the benefits of pesticide use are maximized by being undertaken as part of a larger, integrated strategy that includes non-pesticide tools. This approach is intended to reduce the adverse health and environmental effects of pesticides and to slow the development of pest resistance to pest control products.

The PMRA elements of IPM include:

- identifying potential pest organisms;
- monitoring pest and beneficial organism populations, pest damage, and environmental conditions;
- managing ecosystems to prevent organisms from becoming pests;
- using injury thresholds in making control decisions;
- reducing pest populations to acceptable levels using strategies that may combine biological, cultural, mechanical, behavioural and, when necessary, chemical controls; and
- evaluating the effects and efficacy of pest management strategies.[276]

According to the PMRA, it facilitates the use of sustainable pest management approaches in Canada

[274] http://www.hc-sc.gc.ca/pmra-arla/adifs-e.html .

[275] Integrated Pest Management: A Second Look. *Journal of Pesticide Reform*. Winter 1998, Vol. 8, No. 4.

[276] http://www.hc-sc.gc.ca/pmra-arla/adifs-e.html .

through a number of initiatives including:

- moving from regulation on a product-by-product basis, to a systems approach that incorporates risk reduction and integrated pest management;
- facilitating access to new technologies such as reduced-risk and biopesticide products;
- product labeling for resistance management;
- participation in international risk reduction projects through NAFTA and the OECD, including multilateral sustainable pest management projects and the OECD Pesticide Risk Reduction Activities in Canada survey; and
- the development of national strategies for sustainable pest management for particular commodities and sectors.[277]

The final initiative above is undertaken through voluntary IPM Partnership Projects. These projects include the participation of grower organizations, pesticide manufacturers, federal government departments, provinces, research establishments and non-governmental organizations. Working groups are established to develop, communicate and monitor the adoption of IPM strategies for particular crops and pests. IPM Partnership Projects aim to identify innovative approaches to pest management, facilitate access to new technology and technology transfer, incorporate current research, and highlight emerging research needs. Project areas include the Colorado potato beetle, canola, and urban landscapes, among others.[278]

The steps undertaken in the establishment of an IPM Partnership Project include:

- the identification of stakeholders;
- the establishment of a working group that includes all interested parties;
- the selection of a smaller steering group;
- the gathering of technical information;
- the development of an IPM program and documents;
- the publication and dissemination of the IPM program; and
- the undertaking of follow-up activities such as an assessment of the program's effectiveness.[279]

The PMRA's vision of IPM merely tinkers with the status quo. Its focus is on reducing the risks associated with chemical pesticides and fails to question the more fundamental issue of how to reduce our dependence on these toxic agents. In addition, this extremely limited version of Integrated Pest Management is used by the PMRA as a reactive tool, once a control product has been registered. According to the Environment Commissioner, PMRA IPM initiatives, "lack focus and clear goals and are largely reactive."[280] Moreover, IPM application takes place on a voluntary basis and on a small scale. The principles upon which IPM is based do not guide the pesticide registration process. Apart from ensuring that the least pesticide necessary is applied, which should be an automatic pesticide use restriction, it appears that considerations of integrated pest management are not included in the registration process.

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[277] http://www.hc-sc.gc.ca/pmra-arla/adpst1-e.html .

[278] *Ibid.*

[279] *Ibid.*

[280] *Report of the Commissioner, op.cit.*

Integrated pest management considerations should be included in the re-evaluation process. For example, should less toxic or non-chemical alternatives exist, a pesticide should be de- registered.

Not only has the PMRA failed to implement a genuine and effective IPM program, it has not fulfilled its commitment to develop a risk reduction policy. The Commissioner of the Environment and Sustainable Development recently reported that rather than implement an agency-wide strategy to guide the integration of risk reduction measures into its activities, the PMRA deals with each pesticide individually. Furthermore, the PMRA does not assess which pesticides pose the greatest risk, in order to set management priorities. In contrast, many other countries have established programs and policies that focus on reducing the use and risks of pesticides. The US Department of Agriculture, for example, has set the goal of establishing 70% of US agricultural land under IPM by the year 2000.[281] The Commissioner recommended that the PMRA develop, in consultation with other federal departments and in conjunction with the provinces, a national pesticide risk reduction strategy. The strategy should inform new pesticide registration, existing pesticide re-evaluation and special review, and Agency programs for promoting pesticide alternatives.[282]

Some commentators argue that a risk reduction policy, should the PMRA ever develop one, is an inadequate strategy for dealing with the dangers inherent in pesticide use. Instead of reducing the risks associated with pesticide use, they advocate a strategy to reduce reliance on pesticides. This approach is certain to reduce exposure to pesticides and their dangers, and avoids the lengthy pesticide-by-pesticide approach that would be necessary in a risk reduction approach.[283]

Recommendations

32. The PMRA should develop a pesticide reduction policy and should apply its policy to all PMRA decisions and activities including as a first priority the reduction of pesticides important in children's diets and in use categories of most relevance to children's exposure circumstances including parks and institutional facilities geared primarily to children.

33. The PMRA should reassess its IPM program and make the establishment of sustainable agricultural practices the goal of this program. The program should have, as its focus, the reduction of chemical pesticide use. IPM considerations should be integrated into all stages of pesticide decision-making including a consideration, in the registration process, of whether lower risk or non-chemical alternatives exist, in some cases preempting the need for new registrations. Once registered, pest control product use should be guided by the principles of integrated pest management.

34. The PMRA should do more to facilitate the widespread adoption of IPM. The PMRA should develop a national policy, with clear goals, and a sustainable funding program in order to fulfill this goal.

9.12 INFORMATION

9.12.1 Public Access to Information

[281] *Ibid.*

[282] *Ibid.*

[283] Facsimile document, Julia Langer, World Wildlife Fund, July 5, 1999.

Public access to information regarding pesticide safety and the pesticide regulatory process is limited. The public is not notified when a registration, re-evaluation or other regulatory process begins.[284] Access to the information upon which regulatory decisions are based is also restricted.

Virtually all of the information that the PMRA uses in its regulatory decision-making processes, including pesticide formulations (ingredients) and the results of health and environmental toxicity studies, originates with the manufacturer. The *Pest Control Products Act* is silent on the issue of public access to this information. Consequently, the *Access to Information Act*[285] applies. Under this Act, public access to corporate information can be denied if the information is classified as confidential business information which includes:

- financial, commercial, scientific or technical information that is confidential;
- information the disclosure of which could reasonably be expected to result in material financial loss or gain to a third party or that could prejudice the competitive position of the third party; and
- trade secrets.[286]

The Act does not define "trade secret." In order to preempt abuse of these provisions, section 20 provides that if disclosure would be in the public interest as it relates to public health, public safety or the protection of the environment, and if the public interest in disclosure out weighs the importance of non-disclosure in financial terms, the government may disclose confidential information, with the exception of trade secrets.[287]

Pesticide manufacturers claim that the information that they are required to supply to the PMRA is confidential business information and the PMRA treats it as such. Consequently, the public has no access to information regarding the composition of pest control products, including the presence and relative quantities of formulants, contaminants or by-products, or their potential hazards. Only the name of the product's active ingredient appears on the label.[288] Similarly, access to hazard information, which is derived from the toxicological studies undertaken by potential registrants, is limited. This restriction includes information regarding the product's toxicity, persistence and bioaccumulative potential, routes of exposure and environmental fate.[289] The public should have access to basic information that is essential to an understanding of the risks posed by pesticide exposure including disclosure of all pest control product ingredients.

The PMRA claims that its policy of restricting public access to pesticide information is mandated by the *Access to Information Act*. However, its policy of non-disclosure was established prior to the enactment of this statute.[290] Moreover, the Act authorizes the disclosure of information regarding public health and

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[284] Campaign for Pesticide Reduction. *Pesticides: The Right to Know*. Fact Sheet. (undated).

[285] *Access to Information Act*, R.S.C. 1985, c. A-1.

[286] *Ibid.*, s.20(1).

[287] *Ibid.*, s.20(6).

[288] See section 9.10 on formulants for a description of the limited exceptions.

[289] Davies, Katherine. *The Right to Know About Chemical Pesticides: A Discussion Paper,* prepared for the Canadian Labour Congress and the Campaign for Pesticide Reduction (undated), and Campaign for Pesticide Reduction. *Pesticides: the Right to Know Fact Sheet* (undated).

[290] Davies, Katherine. *The Right to Know About Chemical Pesticides: A Discussion Paper,* prepared for the Canadian Labour Congress and the Campaign for Pesticide Reduction (undated).

environmental concerns under section 20, described above. It is for precisely those reasons set out in section 20 that affected members of the public seek to have pesticide formulation and hazard information released. Given the explicit allowance in the Act for public availability of health and environment-related information, the continued treatment of pesticide formulation and hazard information as confidential business information is questionable.

In its 1990 *Blue Book*, the PRR Team recommended that Proposed Regulatory Decision Documents (PRDDs) be prepared for all proposed registrations of new active ingredients and for registrations that may result in substantially increased pesticide use or exposure. In response, the government committed to the production of PRDDs for proposed registration, re-evaluation and special review decisions. The government directed that PRDDs include the risk and value assessments upon which regulatory decisions are based. The public is then afforded the opportunity to comment on PRDDs.[291] The PMRA is to respond to public comments and release its decision in a Regulatory Decision Document. If the decision is favourable, the pesticide can then be registered or re-registered, as the case may be.

The PMRA does not produce the PRDD. Rather, the only PRDDs produced have been with the explicit agreement of the registrant. Content can therefore be controlled by the registrant.[292] PRDDs are not released without the authorization of the pesticide manufacturer[293] and the PMRA has not set out how it utilizes public comments to strengthen PRDDs and the regulatory decision-making process.

Upon registration, therefore, public access to information regarding a pesticide is limited to the product label and the PRDD, if one has been prepared.

Recommendations

35. The PMRA should ensure that the public has access to basic information that is essential to an understanding of the risks posed by pesticide exposure. Information availability requires that:

a) The PMRA disclose all pest control product ingredients and provide access to all information upon which registration and other regulatory decisions are based;
b) If necessary, the public health and environmental protection provisions in the Access to Information Act be invoke; and
c) Public notification mechanisms regarding the initiation and status of new regulatory decisions be developed.

36. The PMRA should fulfill its commitment regarding PRDD production, making the documents as comprehensive as possible. The PMRA should clearly set out its policy for the incorporation of public comments and concerns regarding PRDDs.

9.12.2 Research and Monitoring: The Fate and Effects of Pesticide Use

While the PMRA has decision-making power over pesticide use, responsibility for pesticide research and

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[291] *PMRA Strategic Plan, op.cit.*

[292] Personal Communication, Julia Langer, World Wildlife Fund. June 14, 1999. See also PMRA web site http://www.hc-sc.gc.ca/pmra-arla

[293] Personal Communication, Christine Norman, Acting Head, Occupational Exposure Assessment Section, PMRA, June 10, 1999.

monitoring rests with the research-oriented federal departments such as Environment Canada and the Department of Fisheries and Oceans. Environment Canada, for example, spent approximately $1.3 million on pesticide research and monitoring in 1997/8. This research, regarding the fate and effects of pesticides, is relevant to product re-evaluation, special review and risk reduction activities. Despite the importance of this work to PMRA operations, the PMRA does not cooperate effectively with the research-oriented departments. A system for the sharing of research findings and the setting of research priorities between departments has not been established.[294]

For example, in the past, Environment Canada has requested information from the PMRA that is necessary to guide its pesticide research. However, relying on the *Access to Information Act*, the PMRA claims that this information is confidential business information and refuses to share it with its regulatory counterparts. In an attempt to resolve this problem, Environment Canada and the PMRA signed a 1998 memorandum of understanding setting out their respective roles and responsibilities. Implementation of this memorandum has barely begun. A similar memorandum of understanding exists between the PMRA and the Department of Fisheries and Oceans. This document remains unsigned as a result of unresolved conflicts between the departments.[295]

In his recent audit, the Commissioner of Environment and Sustainable Development recommended that the PMRA, Environment Canada and the Department of Fisheries and Oceans implement their memoranda of understanding. This should include the development of a plan to guide research and monitoring, the exchange of results, and the consideration of results during regulatory decision-making. The Commissioner further recommended that the PMRA ensure that its registration decisions do not conflict with other federal legislation. When such conflicts do occur, the Agency should consult with other departments prior to making a registration decision.[296]

Noting that there is no pesticide monitoring in many parts of the country, the Commissioner further recommended that the PMRA, Environment Canada, the Department of Fisheries and Oceans, Health Canada, and Natural Resources Canada, together with other partners, identify monitoring needs for pesticides and develop and maintain an inventory of current monitoring programs, to be used to determine information gaps that need to be filled.[297]

Recommendation

37. The PMRA and its research and monitoring counterparts should establish and implement a plan for the collaborative gathering, sharing and use of vital pesticide information.

9.12.3 Adverse Effects Reporting

Reports regarding adverse effects resulting from pesticide use are an important source of information on previously-assessed pest control products. The Pesticide Registration Review Team recommended, in 1990, that registrants be required to report information indicating that a pest control product may cause unreasonable adverse effects on human health, safety or the environment.[298] Legislation in the U.S.

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[294] *Report of Commissioner, op.cit.*

[295] *Ibid.*

[296] *Ibid.*

[297] *Ibid.*

[298] *PMRA Strategic Plan, op.cit.*

requires manufacturers to provide the government with any reports it may receive regarding unexpected adverse effects resulting from pesticide use.[299] However, the *Pest Control Products Act* has no such requirement.

The federal government, in its 1994 *Purple Book*, committed to establishing an adverse effects reporting requirement.[300] The government has not yet fulfilled this commitment. Such a requirement should be established, as should an effective system of information exchange and a central database for this critical information. The reporting requirement should apply to doctors and should include information regarding affected individuals' occupation and place of residence. It must be recognized however that reporting of adverse effects by physicians is not a simple matter. In a busy practice it can be difficult to file such reports even for more routine situations such as adverse effects of medications. Some physicians may not even know reporting forms exist or where they should be submitted or may not consider the effort worthwhile. For pesticides, reports may arise as they do currently from accidental exposures but more subtle effects that are difficult to substantiate or for which associations may be hypothetical are unlikely to be reported. The British Medical Association made useful observations and recommendations on this matter in its report, *Pesticides, Chemicals and Health.*[301] The BMA states that physicians have an important role to play in providing guidance on the hazards of toxic chemicals but that they require sufficient training to recognize symptoms of exposure. The non-specific nature of symptoms underscores both the difficulty of the physician's task and the need for increased training in basic toxicology as well as the opportunity to update and refresh their existing toxicological training.[302]

Recommendation

38. The federal government should fulfill its commitment and legislate an adverse effects reporting requirement that explicitly includes information regarding the adverse effects of pesticide exposure on children. To be effective this reporting system requires first that:

> a) effort is placed on ensuring the education of primary care health-care practitioners (i.e., family physicians, pediatricians, emergency room physicians, obstetricians and midwives, nurse practitioners and social workers about the health effects, both acute and chronic, of pesticides on children in order that they can better clinically detect these cases; and
> b) a central registry be established, federal or provincial, of adverse clinical responses to pesticides, in an attempt to gather appropriate data.

9.12.4 Pesticide Use Database

The Pesticide Registration Review Team identified the need for a national database of information on pesticide sales. In response, the government committed the PMRA to implement such a database. However, the Commissioner of the Environment and Sustainable Development recently reported that of OECD member states, Canada is one of only two nations that do not collect data on pesticide sales. The Commissioner noted that,

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[299] *Report of the Commissioner, op.cit.*

[300] *PMRA Strategic Plan, op.cit.*

[301] British Medical Association, *The BMA Guide to Pesticides, Chemicals and Health* (Edward Arnold, London 1992).

[302] Recommendations on this matter are contained in Chapter 2.

[w]ithout such data, Canada has no ability to measure amounts of pesticides used and released into the environment. This information is needed to monitor the risks to health, safety, and the environment and to measure the extent to which lower-risk pesticides and non-pesticide alternatives are being adopted.[303]

The PMRA reports to be working on the establishment of such a database.[304]

Recommendation
39. The PMRA should promptly establish an enforced pesticide sales and use reporting requirement and a pesticide database. The database should be organized by active ingredient and should include detailed information regarding the quantities and locations of pesticide sales and use. Particular emphasis should be placed on reporting information relevant to assessing the effects of pesticide use on children. This information should inform pesticide regulatory decision-making and must be publicly accessible.

9.12.5 WHMIS

The Workplace Hazardous Material Information System (WHMIS) is designed to secure the right of workers to know about hazardous materials that are present in the workplace. WHMIS is legislated under the Federal *Hazardous Products Act*[305] and includes requirements for hazardous material labeling, the preparation and provision of material safety data sheets (MSDSs), which include a list of hazardous ingredients and their toxic properties, and worker education and training programs. Under WHMIS, hazard information cannot be claimed as confidential business information and there are ingredient disclosure requirements. WHMIS requirements also include a public right to know component. Users of controlled products can request the toxicological data upon which the MSDS is based.

Pesticides are exempted from WHMIS requirements and the *PCPA* does not require the preparation of MSDSs. Consequently, people that work with pesticides are in the anomalous position of being denied access to information regarding the formulation and hazards of the chemicals to which they are exposed. In 1990, the Sectoral Committee on Pesticides, which is composed of workers, industry and government representatives, recommended to the Parliamentary Standing Committee on Consumer and Corporate Affairs and Government Operations that WHMIS requirements be applied to pesticides, including the requirements that MSDSs be provided by pesticide suppliers for pesticides intended to be used in the workplace, that pesticide suppliers disclose the presence of formulants, and that pesticide labels should conform to WHMIS standards, among other recommendations.[306] These recommendations have not been realized. Because of the possible transgenerational effects on the children of occupationally-exposed parents, WHMIS requirements are important not only to workers, but also to their children.[307]

Recommendation
40. The anomalous situation of WHMIS requirements not being applied to pesticides requires correction. Because of the possible transgenerational effects on the children of occupationally-exposed parents,

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[303] *Report of the Commissioner, op.cit.* at 4-30.

[304] *PMRA Strategic Plan, op.cit.*

[305] *Hazardous Products Act*, R.S.C. 1985, c. H-3.

[306] Davies, Katherine. *The Right to Know About Chemical Pesticides: A Discussion Paper*, prepared for the Canadian Labour Congress and the Campaign for Pesticide Reduction, (not dated).

[307] See Section 9.3.3.2 above.

WHMIS requirements are important not only to workers, but also to their children. WHMIS requirements for Material Safety Data Sheets (MSDSs) must be applied to pesticide suppliers for pesticides intended for use in the workplace. Pesticide suppliers should also be required to disclose the presence of formulants, and pesticide labels should conform to WHMIS standards.

9.13 Political Will and Funding

The federal government's stated commitment to the well-being of Canadian children in the recently established National Children's Forum is laudable. This commitment is hollow however if it does not ensure that adequate resources exist to regulate toxic chemicals, including pesticides, in a manner protective of children's health. The following recommendations are made to ensure the implementation of the preceding recommendations those that precede

Recommendations

41. In recognition of the greater exposure and sensitivity in children to the toxic effects of pesticides, the federal government's National Children's Forum must allocate the necessary resources to honour longstanding domestic and international commitments to improving legal and policy tools, including application of the precautionary principle, to protect children from toxic substances, including pesticides.

42. The many recommendations noted above have significant resource implications in six major areas, including: 1) legislative amendments; 2) additional requirements in risk assessment and risk management procedures; 3) the re-evaluation of existing pesticides; 4) research and monitoring; 5) inspection and enforcement; and 6) development/refinement of guidelines and policy in key areas including risk assessment and risk management, the Toxic Substances Implementation Policy, formulants, integrated pest management, etc. Accordingly, for federal budget calculations, the PMRA should be required to prepare a detailed accounting of the resources necessary to implement these recommendations including an indication of short, medium and longer term priorities.

43. In setting priorities for the implementation of unfulfilled commitments and other necessary objectives for improving the pest management regulatory system, immediate attention and resources should be given to re-evaluation of existing pesticides, implementation of the precautionary principle, development of a formulants policy, and development and promotion of sustainable pest management alternatives.

44. In the establishment of an adequate and guaranteed resource base for the pesticide re-evaluation program, funding for re-evaluation must not be made contingent on the generation of funds from efficiencies created in other areas.

45. In the development of a policy on formulants, the PMRA should not be guided in the development of its formulant policy solely by the costs that would be borne by registrants for potential amendments to their registrations.

9.14 Conclusions

The potential for the health of children in Ontario[308] to be affected by pesticides is undeniable. Definitive

[308] While the focus of this study is Ontario, the results of this case study are applicable to all Canadian children.

proof of harm will never be available; not unless human children are made the subjects of controlled experiments investigating the effects of long-term, low dose exposure to the full range of pesticides encountered in their daily lives. Ethically, such experiments would not be condoned. However, in this context, it is exceptionally troubling to conclude that a multitude of uncontrolled experiments on the effects of long-term, low-dose exposure to pesticides on children's health is occurring *by default* as pesticide use continues in a wide variety of applications. It is equally disturbing that pesticide companies have renewed and expanded the practice of using human "volunteers" to determine human NOAELs in attempts to avoid more stringent regulation (see Section 4.4.3 of Chapter 4).

This report summarizes a broad array of observational studies from the peer-reviewed scientific literature[309] documenting the known or suspected health effects of pesticides. Possible health outcomes are extremely serious and in some cases life threatening. The literature points to the increased likelihood of damage to childrens' immune, endocrine, nervous and reproductive systems, as well as various cancers. Although more research needs to be done, this does not exonerate pesticides as human toxins, especially since children are far more vulnerable to pesticides than adults. For numerous reasons documented herein, both exposure and sensitivity to pesticides is greater in children than adults. Not only is there potential for harm, but in all likelihood some Canadian children are now enduring the negative effects of pesticides.

For instance, evidence suggests that the immune systems of Inuit children are being jeopardized by exposure to many persistent chemicals, including DDE (a by-product of the pesticide DDT) through their mothers' breast milk and through their traditional diet. Children in agricultural areas may also be at risk of cognitive deficits (nervous system damages) without obvious clinical symptoms of pesticide exposure. Pesticide use in the home puts children and pregnant women and their babies at risk of health problems, including cancer and reproductive problems in later life. Children from poorer families, living in older housing, and children with chemical sensitivities or immune system problems are also more likely to be affected by pesticides. Lastly, many commonly used pesticides can be detected in our food supply, frequently at levels that would not be safe for young children.The cumulative effects of being exposed to many different pesticides over a lifetime represents an unacceptable risk to all Canadian children.

Moreover, this investigation reveals that children's health is at risk because of the inherent weaknesses of the Canadian regulatory system governing pesticides and the lack of capacity to implement existing laws and policies. It is clear that children's health is at risk because of an inadequate regulatory system, a system the federal government promised to fix as far back as 1994. This study finds that the great majority of prior commitments remain unfulfilled. In addition to numerous unfulfilled commitments on matters highly significant to children's health protection, this review found serious shortcomings in the fundamental aspects of work conducted by the PMRA. In particular, the inaccessibility, lack of clarity and contradictory nature of the PMRA's risk assessment and risk management process is of significant concern. This problem is in addition to the more fundamental shortcomings of risk assessment in general as discussed in Chapter 4.

Put starkly, this review concludes that Canadians don't really have a regulator for pesticides. Rather, Health Canada has set up the Pest Management Regulatory Agency to be not much more than a "customer service department" for the pesticides industry. Children are being impacted by pesticides as

[309] The evidence gathered here is intentionally and overwhelmingly from the peer-reviewed scientific literature, government reports or from the proceedings of conferences presenting the results of scientific inquiry; all of which is, as much as is possible and as far as can be determined, indepedent of funding from companies involved in either the sale of pesticides or the evaluation of pesticides on behalf of the pesticide industry.

the federal government is knowingly refusing to act or delaying action to make legislative changes and spend the necessary resources.

The study provides 45 recommendations covering a broad range of regulatory issues, including:

- Changes to the *Pest Control Products Act.* For example: clarifying the core test for judging the acceptability of a pesticide; ensuring use of the precautionary approach when the weight of evidence suggests a potential unacceptable risk of harm; and enhanced citizen rights to appeal a registration decision.

- Implementation of the Federal *Toxic Substances Management Policy* including immediate bans (or de-registrations) on pesticides which are persistent (stay in the environment a long time) and bioaccumulative (accumulate in fat cells) without wasting resources on re-evaluation.

- Revisions to the registration process for new products to ensure a broader array of impacts on children is taken into account including developmental neurotoxicity and endocrine disruption and impacts from cumulative exposures to pesticides.

- Implementation of a detailed regulatory policy on pesticide formulants.

- Improved inspection and enforcement by the Pest Management Regulatory Agency (PMRA) to ensure appropriate pesticide use.

- Development and application by the PMRA of a Sustainable Pest Management Policy to reduce overall pesticide use.

- Improvements to public access to information that is essential to the understanding of the risks posed by pesticides exposure.

Finally, several recommendations are made to ensure the political will and resources necessary to do the job are in place and applied on a priority basis to urgent and overdue matters. It is also crucial that the resources necessary to honour both unfulfilled commitments and additionally necessary measures to regulate pesticides are not exclusively tied to "cost-recovery" mechanisms. It is perverse for the federal government to insist that the numerous shortcomings of its regulation of pesticides must be paid for by revenues obtained from the approval of new pesticides or the achievement of "efficiencies" in other areas of the PMRA. This department is in need instead of both significant expansion and, more important, re-orientation towards a mind-set that gives first priority to health promotion and prevention of harm.

9.15 Consolidated List of Recommendations

The Pest Control Products Act

1. The Pest Control Product Act's core test for judging the acceptability of a pesticide (unacceptable risk of harm) should be specifically defined so that it can be applied in a transparent and consistent manner throughout the risk assessment-risk management process. An essential amendment to the Act, to complement Recommendation 5 below, is to designate persistent and bioaccumulative substances as presenting an unacceptable risk of harm.

2. The Pest Control Products Act should be amended to include a requirement to act in a precautionary manner, for example, when the weight of evidence points to the potential for "unacceptable risk of harm." In keeping with this approach, Canada should follow Sweden's lead with legislative amendments to specify inherent characteristics of pesticides that justify de-registration including criteria such as very high acute toxicity, endocrine disruption, probable human carcinogenicity, and neurotoxicity all of which should be considered synonymous with "unacceptable risk of harm."

3. To more effectively implement Recommendations 6 - 31 below, the PMRA should publish a guideline to make its risk assessment and risk management process more transparent. The guideline should include detailed descriptions of its decision-making process including the manner in which children's health interests are taken into account. It may be necessary that the guideline be legislated in the form of a regulation under the Pest Control Products Act, in order the ensure that it is implemented.

4. The public should be placed on an equal footing with industry regarding the appeal of a registration decision. To do so, the public must be granted the authority to challenge the approval for registration of pest control products.

Implementation of the Toxic Substances Management Policy

5. The PMRA should fulfill its commitment to incorporate the TSMP in pesticide regulation. This activity should include immediate bans (or de-registrations) on pesticides which are persistent and bioaccumulative (Track 1 substances) without wasting time and resources on re-evaluation. In keeping with this approach, the PMRA should immediately revise its TSMP Implementation Policy to eliminate the ability to register Track 1 pesticides and to cancel registration of pesticides contaminated with persistent organic pollutants pursuant to the TSMP.

The Risk Assessment Process: Hazards

6. The PMRA should set out exactly how its two-tiered system of testing requirements functions. The trigger points for additional testing requirements should be made explicit.

7. Several toxicity tests that are currently conditionally-required should become standard requirements. This includes developmental neurotoxicity testing on young animals, which is particularly important for gauging risks to children's health. Similarly, tests for endocrine disruption that are protective of children should be made a standard PMRA test requirement.

8. There is a need for a detailed examination of the toxicity tests required by the PMRA in order to assess their adequacy. An investigation should be undertaken regarding whether the PMRA requires testing for all potential endpoints and whether the tests that are required are adequate to gauge the risk of causing these endpoints.

9. The PMRA should consider the potential effects on human health of occupational/bystander and food/drinking water exposures on an aggregated basis.

10. The PMRA should consider the potential effects on human health of cumulative exposures to pesticides that act via common mechanisms of toxicity.

11. The PMRA should describe how it chooses a NOAEL for occupational/bystander assessments and food residue assessments from the available alternatives.

12. The PMRA should set out how it determines which uncertainty factors to apply to the occupational/bystander and food residue NOAELs.

13. The PMRA should adopt a requirement similar to that found in the U.S. Food Quality Protection Act, mandating the application of an uncertainty factor with a minimum value of 10 in order to account for potential pre- and post-natal developmental toxicity and the incompleteness of toxicity and exposure data for children. The uncertainty factor could have a higher value in situations of relatively high uncertainty regarding toxicity and children's exposure.

14. The PMRA should explain precisely how it incorporates considerations regarding the increased sensitivity of the young and pregnant women into its risk assessments and should set out under which conditions it considers additional protection for these groups to be warranted.

15. The PMRA should set out precisely how its risk assessments are undertaken for potentially cancer-causing pesticides.

The Risk Assessment Process: Exposure

16. The PMRA should set out which factors it considers when making determinations regarding how large the ratio between the NOAEL for the most sensitive test species and the EEC must be in order for the risks associated with a pesticide to be judged acceptable, as well as their relative weight, and the manner in which they are applied.

Value Assessment

17. The PMRA should set out how the results of its value assessment are used in the regulatory decision-making process.

Maximum Residue Limits

18. Pesticide intake via soil and dust should be included in exposure estimates.

19. The PMRA should consider cumulative exposure to multiple pesticides that act via similar mechanisms of toxicity in its risk assessments.

20. The PMRA should ensure that the negotiation of MRLs between trading partners is a transparent process and that the strength of Canada's MRLs is not compromised.

Use Restrictions

21. The PMRA should reduce the reliance on pesticide label instructions and restrictions for the management of pesticide risk to human and environmental health and in the interim, given the importance of label compliance, the PMRA should improve its inspection and enforcement operations to ensure appropriate pesticide use. The PMRA must not hesitate to apply the full range of enforcement penalties that are available to it, in order to guarantee compliance. Enhanced enforcement should be guided by a national compliance policy, which the PMRA committed itself to develop in its 1994 Purple Book.

Existing (Currently-Registered) Pest Control Products

22. The PMRA should expeditiously complete on-going re-evaluations including several that were initiated close to 20 years ago, such as for pentachlorophenol.

23. The PMRA should fulfill its commitment to establish a comprehensive pesticide re-evaluation and special review policy that includes responsibilities, methods for reporting and systems of accountability. The special review process should clearly set out the conditions necessary to trigger a special review.

The PMRA should establish a re-evaluation program that sets out priorities and firm deadlines.

Formulants

24. The PMRA should expeditiously fulfill its commitment and complete development of its policy on formulants. The PMRA should release its policy to the public for comment and revision. Once completed, the PMRA should effectively implement and enforce its policy. The policy should set out how the PMRA will use the EPA formulant classification system and toxicological database. The policy should also include an explicit enumeration of rigorous testing requirements for new and non-EPA-listed formulants. These requirements should be effectively enforced.

25. The PMRA should immediately complete its assessment of formulants in Canadian-registered pesticides in order to determine which are on the EPA lists and which are not. This is a vital precursor to effective pesticide regulation.

26. The PMRA should more effectively regulate the use of List 3 formulants of known or suspected toxicity. The PMRA should aggressively investigate the safety of List 3 formulants that truly are of unknown toxicity. In accordance with the precautionary principle, use of these formulants should be prohibited until their potential effects are understood.

27. All formulants should be listed on pest control product labels. The requirement to include List 1 substances on product labels should be more aggressively enforced.

28. The PMRA should make active use of the re-evaluation process to assess the safety of formulants that until now, have not been rigorously considered.

29. The PMRA should be granted legislative authority to demand formulant composition information from registrants. It is unacceptable that acquisition of this information is contingent on the good will of U.S. formulant suppliers.

30. The PMRA should expedite its work on the identification and risk assessment of non-EPA-listed formulants that are present in products registered in Canada. Pesticide registrants should be required to provide the PMRA with adequate data to assess the toxicological hazard of such formulants.

31. In his next report, the Commissioner of the Environment and Sustainable Development should investigate the adequacy of PMRA measures to ensure the safety of pest control product formulants.

Sustainable Pest Management

32. The PMRA should develop a pesticide reduction policy and should apply its policy to all PMRA decisions and activities including as a first priority the reduction of pesticides important in children's diets and in use categories of most relevance to children's exposure circumstances including parks and institutional facilities geared primarily to children.

33. The PMRA should reassess its Integrated Pest Management program and make the establishment of sustainable agricultural practices the goal of this program. The program should have, as its focus, the reduction of chemical pesticide use. IPM considerations should be integrated into all stages of pesticide decision-making including a consideration, in the registration process, of whether lower risk or non-chemical alternatives exist, in some cases preempting the need for new registrations. Once registered, pest control product use should be guided by the principles of integrated pest management.

34. The PMRA should do more to facilitate the widespread adoption of Integrated Pest Management.

The PMRA should develop a national policy, with clear goals, and a sustainable funding program in order to fulfill this goal.

Public Access to Information

35. The PMRA should ensure that the public has access to basic information that is essential to an understanding of the risks posed by pesticide exposure. Information availability requires that:

a) The PMRA disclose all pest control product ingredients and provide access to all information upon which registration and other regulatory decisions are based;
b) If necessary, the public health and environmental protection provisions in the Access to Information Act be invoke; and
c) Public notification mechanisms regarding the initiation and status of new regulatory decisions be developed.

36. The PMRA should fulfill its commitment regarding PRDD production, making the documents as comprehensive as possible. The PMRA should clearly set out its policy for the incorporation of public comments and concerns regarding PRDDs.

Research and Monitoring

37. The PMRA and its research and monitoring counterparts should establish and implement a plan for the collaborative gathering, sharing and use of vital pesticide information.

Adverse Effects Monitoring

38. The federal government should fulfill its commitment and legislate an adverse effects reporting requirement that explicitly includes information regarding the adverse effects of pesticide exposure on children. To be effective this reporting system requires first that:

a) effort is placed on ensuring the education of primary care health-care practitioners (i.e., family physicians, pediatricians, emergency room physicians, obstetricians and midwives, nurse practitioners and social workers about the health effects, both acute and chronic, of pesticides on children in order that they can better clinically detect these cases; and
b) a central registry be established, federal or provincial, of adverse clinical responses to pesticides, in an attempt to gather appropriate data.

Pesticide Use Database

39. The PMRA should promptly establish an enforced pesticide sales and use reporting requirement and a pesticide database. The database should be organized by active ingredient and should include detailed information regarding the quantities and locations of pesticide sales and use. Particular emphasis should be placed on reporting information relevant to assessing the effects of pesticide use on children. This information should inform pesticide regulatory decision-making and must be publicly accessible.

Workplace Hazardous Materials Information System (WHMIS)

40. The anomalous situation of WHMIS requirements not being applied to pesticides requires correction. Because of the possible transgenerational effects on the children of occupationally-exposed parents, WHMIS requirements are important not only to workers, but also to their children. WHMIS requirements for Material Safety Data Sheets (MSDSs) must be applied to pesticide suppliers for pesticides intended for use in the workplace. Pesticide suppliers should also be required to disclose the presence of formulants, and pesticide labels should conform to WHMIS standards.

Political Will and Funding

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42. The many recommendations noted above have significant resource implications in six major areas, including: 1) legislative amendments; 2) additional requirements in risk assessment and risk management procedures; 3) the re-evaluation of existing pesticides; 4) research and monitoring; 5) inspection and enforcement; and 6) development/refinement of guidelines and policy in key areas including risk assessment and risk management, the Toxic Substances Implementation Policy, formulants, integrated pest management, etc. Accordingly, for federal budget calculations, the PMRA should be required to prepare a detailed accounting of the resources necessary to implement these recommendations including an indication of short, medium and longer term priorities.

43. In setting priorities for the implementation of unfulfilled commitments and other necessary objectives for improving the pest management regulatory system, immediate attention and resources should be given to re-evaluation of existing pesticides, implementation of the precautionary principle, development of a formulants policy, and development and promotion of sustainable pest management alternatives.

44. In the establishment of an adequate and guaranteed resource base for the pesticide re-evaluation program, funding for re-evaluation must not be made contingent on the generation of funds from efficiencies created in other areas.

45. In the development of a policy on formulants, the PMRA should not be guided in the development of its formulant policy solely by the costs that would be borne by registrants for potential amendments to their registrations.

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Appendix 1: DOCUMENT "KEY"

This document "key" is provided to assist the reader with the numerous abbreviations made, throughout the text, to several key reference materials.

The Blue Book
Recommendations for a Revised Federal Pesticide Management Regulatory System, Pesticide Registration Review Team, 1990.

The Purple Book
Government Proposal for the Pesticide Management Regulatory System. (Federal Government response to "the Blue Book"). 1994.

PMRA Letter
(Letter from Christine Norman, Acting Head, Occupational Exposure Assessment Section, PMRA to Canadian Environmental Law Association, May 26, 1999)

Report of the Commissioner
Minister of Public Works and Government Services Canada. *Report of the Commissioner of the Environment and Sustainable Development to the House of Commons.* (1999) http://www.oag-bvg.ca

PMRA Submission
PMRA. *Organizing and Formatting a Complete Submission for Pest Control Products.* (1998) Document No. Pro98-02. http://www.hc-sc.gc.ca/pmra-arla/qpubs2-e.html

PMRA Overview Document
PMRA. *Overview Document* (undated)

PMRA Sulfosulfuron PRDD
PMRA. *Proposed Regulatory Decision Document: Sulfosulfuron.* (1998) Document No. PRDD98-01 http://www.hc-sc.gc.ca/pmra-arla/qpubs2-e.html

PMRA Registration Handbook
PMRA. *Registration Handbook.* (1998) http://www.hc-sc.gc.ca/pmra-arla/hndbk-e.html

PMRA Strategic Plan
PMRA. *Strategic Plan 1998-2003* (undated) http://www.hc.sc.gc.ca/pmra-arla/stratp-e.html

Appendix 2. ***Table 9.1 Summary of Information on Selected Common Pesticides*** [i]

Pesticide Chemical Class	Some Common Examples	Uses	Biological Action	Long Term Effects in Mammals	Level of Concern	Level of Uncertainty	Status in Canada	Other Concerns
Organophosphates	*Insecticides*: e.g., Chlorpyrifos (also Parathion, Methyl parathion, Malathion, Diazinon, Dichlorvos) *Herbicides*: e.g., Glyphosate	▪ Insecticides primarily, some herbicides ▪ Both home-use and commercial pesticide applications	▪ Inhibit cholinesterase enzyme	e.g. Chlorpyrifos ▪ Neurodevelopmental effects in animals ▪ Dose-related reproductive toxicity in animals ▪ Teratogen ▪ Alters protein & DNA synthesis in brain ▪ Latent peripheral neuropathy (humans) ▪ Cognitive, affective & neuropsychologic symptoms (humans) ▪ Suspected assoc'n with birth defects (Chlorpyrifos in humans)	High ▪ Data from acute exposures suggest young more susceptible to ill effects than adults ▪ Substantial toxicologic evidence for neuro-developmental and growth effects in developing animals ▪ Able to cross placenta ▪ Chlorpyrifos measured in homes 8 yrs after use ▪ Residues appear frequently on food items commonly eaten by children	High to Moderate? ▪ Few studies of subtle long term effects from *in utero* exposure ▪ Uncertainty regarding human immune system suppression	▪ Most registered ▪ PMRA announced re-evaluation of 26 organophosphate pesticides, June 1999. ▪ Review ongoing and awaiting outcome of studies by U.S. EPA	▪ Tetrachlorvinphos contaminated with dioxins
Carbamates	e.g., Aldicarb, Basudin, Carbaryl, Propoxur	▪ Insecticides mainly ▪ Both home-use and commercial pesticide applications	▪ Inhibit cholinesterase enzyme ▪ Anti-cholinesterase effects are <u>reversible</u>	▪ Similar to above ▪ Prevalence of asthma sig. associated with use of carbamates in farmers (Sask.) ▪ Increased risk of miscarriage where male farmers exposed to certain carbamates (Can.)	High ▪ Data from acute exposures suggest young more susceptible to ill effects than adults ▪ Several individual carbamates relatively toxic to humans ▪ Residues appear frequently on food items commonly eaten by children	High to Moderate? ▪ Uncertainty regarding human immune system suppression	▪ Most registered ▪ Aldicarb banned in 1964 ▪ No reviews pending	
Organochlorines	e.g., DDT, aldrin, dieldrin, lindane, chlordecone, toxaphene, hexachlorobenzene (HCB), pentachlorophenol (PCP)	▪ Variety of uses ▪ Previously used as: ❖ Insecticides, (DDT vs. mosquitoes, Lindane for lice); ❖ Herbicides (dicamba); ❖ Rodenticides; ❖ Fungicides (HCB, PCP), ❖ Wood preservative (PCP), and ❖ Veterinary use	▪ Interfere with transmission of nerve impulses ▪ Primarily disrupt CNS	▪ Carcinogens (human data) e.g., brain cancer with use in childhood of Lindane anti-lice treatment ▪ Chromosomal abnormalities (human data) ▪ Suspect teratogens ▪ Fetotoxins ▪ Behaviour changes (human data) ▪ Reproductive effects (human data)	High ▪ Developing animals more sensitive than adults ▪ Exposures to pregnant rodents → effects seen in offspring ▪ Transfer across placenta ▪ Found in mother's milk ▪ Persistent, bioaccumulative and biomagnifying ▪ Potential for serious delayed effects (e.g., endocrine disruption, cancer) ▪ Transgenerational effects	Moderate ▪ Reasonably good data for effects from chronic exposure including human studies ▪ Uncertainty regarding effects as endocrine disruptors and immune system suppressants in humans	▪ Most banned as primary ingredients in 1970s, '80s ▪ Designated Track 1 substances, targeted for virtual elimination ▪ Lindane, methoxychlor & PCP still registered ▪ PCP under ongoing re-evaluation as a wood preservative (Feb 1994). ▪ Lindane under special review ▪ Tributyl tin (TBT) used on ocean-going vessels to prevent barnacle build up	▪ Some are contaminants of currently registered pesticides (e.g. DDT & dicofol-containing products; HCB & several registered pesticides – Atrazine, Endosulfan, etc.) ▪ May appear in imported produce and flowers and in domestic meat and dairy items.

Pesticide Chemical Class	Some Common Examples	Uses	Biological Action	Long Term Effects in Mammals	Level of Concern	Level of Uncertainty	Status in Canada	Other Concerns
Pyrethrins & Pyrethroids	e.g., Pyrethrin, Cyfluthrin, Permethrin, Cypermethrin	▪ Insecticides used for a variety of types such as lice, cockroaches ▪ Used in crops such as nut, fruit, various vegetables, mushroom, potato, cereals ▪ Used in greenhouses and home gardens for termites	▪ Inhibit sodium & potassium conduction in nerve cells ▪ Block transmission of nerve impulses ∴ paralyze the nervous system ▪ Permethrin is a stomach and direct contact poison	▪ Suspect mutagens? ▪ Suspect teratogens? ▪ Suspect carcinogens ▪ Immunotoxins ▪ Lower hormone release from brain ▪ Associated with neurologic and respiratory reactivity ▪ Permethrin causes liver enlargement	Medium to High ▪ Some may be cumulative ▪ Potential endocrine disruptors ▪ Toxic to wildlife	High ▪ Studies show variable results viz. chronic effects in animals	▪ Most registered in Canada	
Amides	e.g., DEET (N,N-diethyl-*m*-toluamide)	▪ Insect repellent effective against mosquitoes and biting flies ▪ Applied on human skin, clothing, pets, tents, screens, etc.	▪ Not fully understood	▪ Neurotoxin ▪ Evidence for dermal and neurobehaviroual effects in workers, children ▪ Clinical reports of various neurotoxic effects in children such as toxic encephalopathy, seizures	Medium to high ▪ Readily absorbed via skin or ingestion ▪ Distributed to all organs, including brain ▪ Crosses the placenta ▪ Excreted mainly in urine but also in milk ▪ Commonly used on children ▪ Concern that children at greater risk of adverse rxs	High ▪ Effects in children known mainly from clinical reports	▪ Ongoing review along with several other personal insect repellents, announced in June 1990 by Health Canada ▪ 160 DEET-containing products registered ▪ (U.S. has 53 registrants of DEET-containing insect repellents)	
(Chlor)phenoxy group	e.g., 2,4-D (2,4-dichloro-phenoxyacetic acid)	▪ Herbicides ▪ Used in agriculture, forest management against broad leaf weeds ▪ Found commonly in home garden products	▪ Acts as a synthetic growth hormone (plants) ▪ Action not fully understood for animals	**e.g., 2,4-D** ▪ Suspect mutagen ▪ Teratogen ▪ Delayed fetal development ▪ Suspect fetotoxin ▪ Immunotoxin ▪ Toxic injury to liver, kidney, CNS ▪ Reproductive effects in male farm sprayers ▪ Carcinogen (assoc'n with Non-Hodgkin's Lymphoma in exposed workers) ▪ Higher rates of birth defects in exposed populations	High ▪ Widely used ▪ Agriculture and Agri-food Canada discovered dioxin contamination in 2,4-D products	Low to Medium ▪ Vast amount of study on 2,4-D due to its association as a component of Agent Orange	▪ Usage in Ontario > 140,000 kg annually ▪ Ongoing re-evaluation in Canada (announced in October 1980; latest update, November 1994) ▪ Restricted use pesticide in the U.S.	

Pesticide Chemical Class	Some Common Examples	Uses	Biological Action	Long Term Effects in Mammals	Level of Concern	Level of Uncertainty	Status in Canada	Other Concerns
Bipyridil	e.g., Diquat, Paraquat	▪ Herbicide dessicant	▪ May interfere with cellular respiration & damage cell membranes ▪ Non-selective toxicity	▪ Suspect mutagen ▪ Suspect teratogen ▪ Suspect embryo & fetotoxin ▪ Liver damage ▪ Cataracts Human ▪ Damage to gastrointestinal tract, kidneys, liver, heart ▪ Reported fatalities from skin absorption ▪ Paraquat implicated in Parkinson's disease	High ▪ Variety of health effects in humans ▪ Potential for long-term, delayed effects	High	▪ Diquat & Paraquat registered in Canada ▪ Both slated for review by PMRA	Paraquat ▪ Banned in Sweden (1983) ▪ Banned in Netherlands (1989)
Triazine	e.g., Atrazine	▪ Herbicide ▪ Agricultural use – control of broad leaf and grassy weeds in various crops such as corn, Christmas trees	▪ May disturb vitamin metabolism	▪ Immunotoxin ▪ Adrenal, liver heart damage ▪ Effects on ovary ▪ Possible human carcinogen (EPA) ▪ Suspect endocrine disruptor	Medium to high? ▪ May contaminate groundwater ▪ Moderately persistent to persistent ▪ Health Canada study found atrazine in well water in PEAS ▪ Used in large volumes ▪ Contamination with HCB	Medium	▪ Registered in Canada ▪ Annual usage 2 million kg ▪ (Restricted use in U.S. since 1990) ▪ Slated for review by PMRA	▪ Atrazine contaminated with HCB
Fungicides	e.g. Benomyl (class - benzimidazole), [Also ziram (dithiocarbamate), captan (phthalimide), methyl mercury (organic metal), PCP (organochlorine) see above]	▪ Variety of classes used against various types of fungus ▪ e.g., Benomyl used against fungal diseases in field crops, fruits, nuts, ornamentals, mushrooms, turf	▪ Modes of action vary depending on class of each fungicide ▪ e.g., Benomyl interferes with cellular respiration	**e.g., Benomyl** ▪ Skin disorders ▪ Listed as teratogen by U.S. EPA ▪ Suspect carcinogen ▪ Suspect mutagen ▪ Liver & testes damage ▪ Reduced sperm ▪ Blood damage	Medium to high? ▪ Moderately persistent ▪ Anecdotal reports of association with gross eye defects in children of mother's allegedly exposed to Benomyl during pregnancy	Medium to High **e.g. Benomyl** ▪ Birth defects vary depending on route of exposure in test animals	**Benomyl** ▪Registered in Canada ▪Slated for review by PMRA ▪Restricted use in U.S. since 1982	

[i] Information compiled from several sources including: Briggs, Shirley, A. and staff of Rachel Carson Council. *Basic Guide to Pesticides: Their Characteristics and Hazards.* (Washington: Taylor & Francis, 1992); CALPIRG & PSR, 1998, *op cit.*, City of Toronto, 1998 *op cit.*; PMRA personal communication April 2000 and internal data file 405acti1.xls; Eskenazi et al. 1999, *op cit.*; EXTOXNET. Extension Toxicology Network. A pesticide information project of cooperative extension offices of Cornell University, Michigan State University, Oregon State University and University of California at Davis. Pesticide Information Profiles. Available at: http://pmep.cce.cornell.edu/profiles/extoxnet; and World Wildlife Fund, Inuit Circumpolar Conference, Inuit Tapirisat of Canada, 2000, *op cit.*

Appendix A: Consolidated List of References

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Appendix B: List of Acronyms

AAQC	Ambient Air Quality Criteria
ADI	Acceptable Daily Intake
CCME	Canadian Council of Ministers of the Environment
CDD	Chlorinated di-benzo-p-dioxins
CELA	Canadian Environmental Law Association
CEPA	*Canadian Environmental Protection Act*
CPR!	Campaign for Pesticide Reduction
CPSC	Consumer Product Safety Commission
CRS	Congressional Research Service
CSA	Canadian Standards Association
CWS	Canada Wide Standards
DACO	data-code
DNT	Developmental Neurotoxicity Testing
DSL	Domestic Substances List (under CEPA)
EEC	expected environmental concentration
EPA	Environmental Protection Agency (United States)
EPA	*Environmental Protection Act* (Ontario)
FDA	Food and Drug Administration (United States)
FFDCA	Federal Food, Drug and Cosmetics Act (United States)
FIFRA	Federal Insecticide, Fungicide and Rodenticide Act (United States)
FQPA	*Food Quality Protection Act* (United States)
FR	Federal Register (United States)
IARC	International Agency for Research on Cancer
IPM	integrated pest management
LOAEL	Lowest Observed Adverse Effect Level
MOE	Ministry of Environment (Ontario)
MOEE	Ministry of Environment and Energy (Ontario, prior to 1995)
MOS	margin of safety
MRL	Maximum Residue Limits
MSDS	material safety data sheets
NAAQO	National Ambient Air Quality Objectives (Canada)
NAFTA	North American Free Trade Agreement
NAICC	National Air Issues Coordinating Committee (Canada)
NAS	National Academy of Sciences (NAS)
NCAP	Northwest Coalition for Alternatives to Pesticides
NHL	non-Hodgkin's Lymphoma
NOAEL	No Observed Adverse Effect Level
NOEC	no observable effect concentration
NOX	Nitrogen Oxides
NRC	National Research Council (United States)
OCFP	Ontario College of Family Physicians
OECD	Organization for Economic Cooperation and Development
OPP	Office of Pesticide Programs (US EPA)
OPs	Organophosphates
PCPA	*Pest Control Products Act* (Canada)
PDI	potential daily intake
PDP	United States Department of Agriculture's Pesticide Data Program

PMRA	Pest Management Regulatory Agency (Health Canada)
POI	Point of Impingement (Ontario)
ppm	parts per million
ppt	parts per trillion
PRDD	Proposed Regulatory Decision Documents
PRR	Pesticide Registration Review
PSL	Priority Substances List (under *CEPA*)
ROC	residue of concern
RTECS	Registry of Toxic Effects of Chemical Substances
SOP	Strategic Options Process
SOPs	Standard Operating Procedures
TDI	Tolerable Daily Intake
TI	Toxicity Index
TRAC	Tolerance Reassessment Advisory Committee (United States)
TSL	Toxic Substances List (under CEPA)
TSMP	Toxic Substances Management Policy
USC	use-site categories
USDA	United States Department of Agriculture
VOC	Volatile Organic Compounds
WGAQOG	Working Group on Air Quality Objectives and Guidelines (Canada)
WHMIS	Workplace Hazardous Materials Information System
WHO	World Health Organization
WRI	World Resources Institute