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Children's Clean Air Act Backgrounder

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## BACKGROUND

### 1) Definition of the Problem:

#### a) Scientific Problem

Children's health problems that are or may be related to environmental contaminants are on the rise in Canada. These include health conditions such as asthma, childhood cancers, learning disabilities, among others.<sup>1</sup>

Children are more vulnerable than adults to environmental pollutants due to:

- their physiological makeup, their activities and pastimes and as a result of the fact that their bodies are still growing and developing.<sup>2</sup>
- Children spend more time outside than do adults, and when outdoors, they tend to be more active than adults, breathing faster during play activity, and therefore increasing exposure to outdoor air pollutants.<sup>3</sup>
- 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.<sup>4</sup>
- They are also more often involuntarily exposed to environmental chemicals because they are still dependant on adults for their supervision and care and are not sufficiently cognitively developed to avoid environmental exposures on their own.<sup>5</sup>

Differences between children and adults:

- Children are smaller than adults, they breathe more rapidly than do adults, and as a result of having smaller lungs, they have a much greater surface area to volume ratio, resulting in a greater dose of pollution delivered to their lungs.<sup>6</sup>
- As a result children's absorption of contaminants through inhalation is greater than that of adults.<sup>7</sup>
- Children have narrower airways than adults and irritation or inflammation caused by air pollution that would produce only a slight response in an adults can result in a potentially significant obstruction in the airway of a young child.<sup>8</sup>
- Because children's lungs and airways are still developing, they are especially sensitive to air pollution. (p44)
- Overall children's developing organs and tissues are more susceptible to damage from some environmental contaminants than are adult organs and tissues.<sup>9</sup>

The Case for including the effects of environmental pollutants on humans prior to birth:

- Developing organisms, especially during embryonic and fetal periods and early years of life, are often particularly susceptible, and may be more exposed than adults, to many environmental factors, such as polluted air, and chemicals.<sup>10</sup>

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<sup>1</sup> Canadian Environmental Law Association, *Environmental Standard Setting and Children's Health* (Canadian Environmental Law Association, 2000) online: <<http://www.cela.ca/coreprograms/detail.shtml?x=1326>> at 76.

<sup>2</sup> Ibid, at 30.

<sup>3</sup> Ibid, at 44.

<sup>4</sup> Ibid.

<sup>5</sup> Ibid, at 30.

<sup>6</sup> U.S. S.B. 25, *An act to amend ... the Health and Safety Code, relating to environmental health protection*, 1997-98, Reg. Sess., Cal., 1998 (enacted).

<sup>7</sup> CELA, *supra* note 1, at 30.

<sup>8</sup> S.B. 25, *supra* note 25.

<sup>9</sup> Ibid.

<sup>10</sup> Children's Environment and Health Action Plan 2004, passed at the Fourth Ministerial Conference on Environment and Health [Action Plan 2004].

- After the first two weeks in utero, the organ systems and body structures are rapidly developing and if these developmental processes are interfered with, major anatomical abnormalities can occur that would be irreversible. Thus, interference by environmental contaminants could be potentially damaging during this period, even at very subtle exposure levels.<sup>11</sup>
- During fetal development, important *physiological defects* and *minor anatomical abnormalities* may occur.<sup>12</sup>
- For example, exposure to higher levels of carbon monoxide in late pregnancy may be associated with significantly increased risk of low birth weight and there is some evidence to suggest that early maternal exposure to high particulate levels carries greater odds of intrauterine growth retardation. The health effects of air toxics include reproductive effects, as well as cancer.<sup>13</sup>

#### After Birth

- Children's metabolic pathways, especially in the first months after birth, are immature. Their ability to metabolize, detoxify, and excrete many toxins differs significantly from that of adults. Children are undergoing rapid growth and development, and their developmental processes are easily disrupted, thus creating special vulnerabilities.
- Children's organs undergo primary differentiation and very rapid growth prenatally, and the development of some organs continues months and even years after birth.<sup>14</sup>
- The lungs undergo extensive growth after birth and thus are among the most particularly sensitive organs (the brain and the nervous system are others).
- Children's developing lungs and extremely permeable skin present large surface areas through which chemicals and toxins can be easily absorbed, and the developing nature of children's organs renders them particularly ill-equipped to handle toxic contaminants.<sup>15</sup>

#### Types of Pollutants and their Sources

- There is a significant concern regarding the potential for long-term toxicity, including the carcinogenic, neurotoxic, immunotoxic, genotoxic, endocrine-disrupting and allergenic effects of many air pollutants, chemicals, heavy metals, and toxins.<sup>16</sup>
- Exposure to a host of gaseous agents – primarily from fossil-fuel power and heating plants (SO<sub>2</sub>) and from road traffic (NO<sub>x</sub> and VOCs) – and to suspended particulate matter from energy production, diesel engines and ore processing increases bronchial and lung morbidity and exacerbates pre-existing respiratory pathology.<sup>17</sup>
- Sulphur dioxide and acid aerosols, ozone, oxides of nitrogen, particulates 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.<sup>18</sup>
- The interactions between NO<sub>x</sub>, VOCs and oxygen under the effect of sunlight result in the production of ozone, hydroxy radicals and a number of organic irritants that induce lacrymation and mucosal irritation.<sup>19</sup>
- 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

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<sup>11</sup> CELA, *supra* note 1, at 34.

<sup>12</sup> *Ibid.*

<sup>13</sup> *Ibid.*, at 59.

<sup>14</sup> North American Commission for Environmental Cooperation, "North American Workshop on Risk Assessment and Children's Environmental Health: Background Document," (February 2003) online: <[http://www.cec.org/files/pdf/POLLUTANTS/RACEHW-feb03\\_en.pdf](http://www.cec.org/files/pdf/POLLUTANTS/RACEHW-feb03_en.pdf)> [NA Commission for Environmental Cooperation].

<sup>15</sup> CELA, *supra* note 1, at 31.

<sup>16</sup> Action Plan 2004, *supra* note 10.

<sup>17</sup> *Ibid.*

<sup>18</sup> CELA, *supra* note 1, at 43.

<sup>19</sup> Action Plan 2004, *supra* note 10.

as there is considerable long-range transport of air pollutants in atmosphere. Ontario reports that 50% of its smog originates in the U.S.<sup>20</sup>

#### Effects of Air Pollution:

- Childhood asthma and other pediatric respiratory ailments are increasing dramatically and are substantially exacerbated by environmental pollutants in the air, including emissions from fossil fuel combustion and other sources.<sup>21</sup>
- Chronic respiratory diseases and asthma are aggravated by some forms of air pollution, lung function may be reduced, and exposure may contribute to the incidence of lung cancer.<sup>22</sup>
- From 1985-2000, there was a fourfold increase in the number of children under 15 who were afflicted with asthma in Canada.<sup>23</sup> 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. As such, asthmatic children represent a particularly sensitive subgroup of children with respect to exposure to air pollutants.
- 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. It has also been associated with increased hospital emergency visits and admissions, and increased school absences.<sup>24</sup>
- Smog has also been shown to lead to significant increases in mortality in the general population. Data suggests that there are 5000 premature deaths a year from air pollution in 11 Canadian cities alone.
- 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.<sup>25</sup>
- Epidemiological studies do show clear associations between episodes of high air pollution and subsequent hospital visits for respiratory problems.<sup>26</sup>
- Another Canadian study found that moderate levels of ozone and sulfate resulted in statistically significant decreases in lung function.<sup>27</sup>
- Long-term exposure to acid aerosols may adversely affect lung growth, development and function. 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.<sup>28</sup>
- There is evidence that lung growth, development and function may be compromised from long-term exposure to acid aerosols, even at moderate levels. 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.<sup>29</sup>

#### Scientific Conclusions:

- Children have more future years of life than most adults, and thus have more time to develop chronic diseases triggered by early exposures.
- Many diseases that are caused by toxicants in the environment require decades to develop. Carcinogenic and toxic exposures sustained early in life, including prenatal exposures, in some cases appear more likely to lead to disease than similar exposures encountered later.
- Developing systems may not be able to repair damage caused by certain environmental toxicants. Therefore, there is high risk that the resulting dysfunction will be permanent and irreversible.

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<sup>20</sup> CELA, *supra* note 1, at 43.

<sup>21</sup> 1997 Declaration of the Environmental Leaders of the Eight on Children's Environmental Health.

<sup>22</sup> Action Plan 2004, *supra* note 10.

<sup>23</sup> CELA, *supra* note 1, at 66.

<sup>24</sup> *Ibid.*, at 58.

<sup>25</sup> *Ibid.*, at 59.

<sup>26</sup> *Ibid.*, at 66.

<sup>27</sup> *Ibid.*, at 59.

<sup>28</sup> *Ibid.*

<sup>29</sup> *Ibid.*, at 67.

## b) Problem with the Regulatory Framework

The Canada Public Health Association, passed a motion in 1999, where they recognized that the protection of children from environmental contaminants is an important but generally neglected issue that must be addressed with some urgency.

### Problems with the current framework:

- CEPA does not address children's special vulnerabilities.
  - Children are not mentioned in the standard setting process;
  - In the process of providing permits that allow the emission of certain substances, the impacts on children's health are not considered;
  - In fact, the potential effects on children's health from the emission of substances into the ambient air are not mentioned anywhere in CEPA.
- 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.<sup>30</sup>

In 2002, the Canadian federal government solicited feedback and comment from the public on a discussion document entitled, "A Canadian Perspective on the Precautionary Approach/ Principle." The document proposed to outline "broad, guiding principles to support consistent, credible and predictable policy and regulatory decision making when applying the precautionary approach/principle." However, the federal government discussion paper did not include any discussion regarding better protection of vulnerable populations such as children.<sup>31</sup>

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<sup>30</sup> Ibid, at 76.

<sup>31</sup> Ibid, at 23.

### c) Inadequate Science/Research

- 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.”<sup>32</sup>
- 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).<sup>33</sup>
- 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.<sup>34</sup>
- 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.<sup>35</sup>
- Most of the studies on the risk of harm from environmental pollutants have been completed on animals or healthy adults. However, it is not possible to definitively extrapolate from the health effects observed through animal studies, as animal models of development and physiology are not directly comparable to those for humans.<sup>36</sup>
- 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.<sup>37</sup>

### d) Inadequate Standards

The risk assessment process that is used in standard setting suffers from large gaps in data and methodology providing many opportunities for uncertainty, variability and error. The risk assessment exercise can no longer claim to be objective and scientific.<sup>38</sup>

- The risk assessment process utilized in the standard-setting approach is fairly capable of predicting acute effects from high dose exposures, but falls short in its ability to detect chronic effects from long-term, low dose exposure and in assessing the interactive effects of multiple chemicals in the environment, it fails miserably.<sup>39</sup>
- 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 especially particularly sensitive ones, such as children or the elderly. This has sometimes been referred to as the healthy worker effect.<sup>40</sup>

### The Evaluation Process:

- Key gaps have been identified in toxicity testing, risk assessment uncertainty, exposure assessment, and dose-response assessment, as they relate to the special circumstances of children (prenatal through adolescence).<sup>41</sup>
- Procedures and/or definitions vary but the four basic steps include: hazard identification, dose-response assessment, exposure assessment, and risk characterization. The second two are especially difficult due to a

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<sup>32</sup> Ibid, at 75.

<sup>33</sup> Ibid at 76.

<sup>34</sup> Ibid.

<sup>35</sup> Ibid, at 75.

<sup>36</sup> Ibid.

<sup>37</sup> Ibid, at 31.

<sup>38</sup> McClenaghan, Theresa et al., (2003) CELA Publication No. 466 “Environmental Standard Setting and Children's Health: Injecting Precaution into Risk Assessment” 26.

<sup>39</sup> Ibid, at 3.

<sup>40</sup> Ibid, at 14.

<sup>41</sup> Ibid, at 8.

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 calculate exposure and a dose-response relationship. This problem is greatly magnified when considering multiple exposures and the chance of cumulative or synergistic effects.

- In order to address this: exposure estimates and dose-response assessments should not be restricted to the impact of a single pesticide but should be required to assess aggregated exposures to pesticides with a common toxic effects.<sup>42</sup>
- 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 “inference choices.” These gaps in critically important scientific and empirical data and methodologies are filled by what is essentially guesswork. It may be the product of modeling including “informed guesses” or “the informed judgement of experts” but it is still largely guesswork, not science.<sup>43</sup>
- Standard setting in environmental settings has often included making a distinction between chemicals for which a threshold is or is not apparent. The evaluation 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.
- However, the stated risk level is not actually accurate, as in this process, each chemical is assessed separately and considered in isolation from any other risks that may exist.<sup>44</sup>
- Considerable debate has occurred over whether or not health effects in fact do occur below these thresholds. The example of lead is one where the threshold for adverse effects has been progressively lowered 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.<sup>45</sup>

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 threshold values established through risk assessment being exceeded.
- *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).

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<sup>42</sup> Ibid.

<sup>43</sup> Ibid, at 12.

<sup>44</sup> Ibid, at 10.

<sup>45</sup> Ibid, at 6.

- *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.<sup>46</sup>
- 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.<sup>47</sup>

#### The way forward:

- Decision –making about associations or likelihood of harm under the Precautionary Principle 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.”[Raffensperger, C. and J. Tickner (eds.), *Protecting Public Health and the Environment: Implementing the Precautionary Principle* (Washington, D.C.: Island Press, 1999), Introduction, p.169]<sup>48</sup>
- A “precautionary inference” has been proposed as a method to make scientific judgments when data are incomplete or inconclusive, and where significant harm may follow from a false negative judgment. With precautionary inference, 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 relevant uncertainty is resolved to demonstrate on “clear, strong and cogent evidence” that at the permitted exposure level, no harm to children will result.<sup>49</sup>
- 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. Standard setting approaches should weigh all of the available evidence and arrive at a prudent protective judgment based on all of that “weight of the evidence.” The question to be answered should be: based on all of the evidence, is harm *not* likely to occur to children? 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. To this point, a precautionary approach has not been followed for the majority of standards affecting children’s health.<sup>50</sup>

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<sup>46</sup> Ibid, at 11. The above list is drawn from analyses published mostly during the early 1990s. 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*, *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.

<sup>47</sup> Ibid, at 15.

<sup>48</sup> Ibid, at 22.

<sup>49</sup> Ibid, at 21.

<sup>50</sup> Ibid at 19,

- 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.<sup>51</sup>
- 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.

#### Conclusions:

- Government agencies should explore their options for causing the least possible environmental damage: “All potentially environmentally degrading activities, public or private, should be subject to public scrutiny of alternatives. The public deserves to know that those who pollute, extract, consume, emit, incinerate, or abandon are aware of their technological options for minimizing disturbance of the environment.”<sup>52</sup>
- Successfully applying a precautionary approach to these decisions will require rapid evolution of environmental decision-making tools beyond traditional risk assessment, with its tendency to concentrate on only those select few pollutants subject to a regulation at hand; in isolation from other sources of that pollutant, other dangerous substances, other media, and the natural ecosystem as a whole; and to ignore the totality of how people actually live in their homes and their communities.<sup>53</sup>
- Cumulative impacts should take into consideration exposures or public health and environmental effects from combined emissions and discharges, in a geographic area including environmental pollution from all sources, whether single or multi-media, routinely, accidentally, or otherwise released. Impacts take into account sensitive populations and socio-economic factors, when data is available.<sup>54</sup>
- The development of child-specific risk assessment methods, improved understanding of mechanisms underlying children's sensitivity to environmental toxicants, and consideration of child-specific toxicity and exposure information are all important considerations.<sup>55</sup>
- The scientific basis on how, when, and by how much children differ from adults in their susceptibility to environmental threats must be better understood. In addition, risk assessment methods should be fully adapted to utilize child-specific susceptibility information to improve public health evaluations when such information is available. The mechanisms that underlie the susceptibility of children to environmental exposures that are relevant for both toxicity and exposure assessment should be put into the context of the risk assessment framework. The health implications of children's sensitivities can only be understood through the utilization of currently available information regarding child specific hazard and exposure, and support of continued research.<sup>56</sup>

## **2) Governmental Action in other Jurisdictions**

### Legislation Passed in the United States:

Executive Order 13045 on Health Risks to Children, passed by the White House in 1997.

State of California's Senate Bill 25, December 7, 1998 – “An act to amend ... the Health and Safety Code, relating to environmental health protection.”

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<sup>51</sup> Ibid, at 22.

<sup>52</sup> Montague, Peter. “Risk Assessment: Modern Environmental Protection – Part 3,” *Rachel's Environment and Health News Biweekly* #706 (August 17, 2000).

<sup>53</sup> Raffensperger, Carolyn. “Cumulative Impacts and Precaution,” *The Environmental Forum* (March/April 2005).

<sup>54</sup> Ibid.

<sup>55</sup> NA Commission for Environmental Cooperation, *supra* note 14.

<sup>56</sup> Ibid.

European Union Conventions:

Third Ministerial Conference on Environment and Health (1999) World Health Organization and Commission of the European Communities.

Children's Environment and Health Action Plan 2004, passed at the Fourth Ministerial Conference on Environment and Health.

International Agreements:

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