Toxic Chemicals and Children's Health in North America

A Call for Efforts to Determine the Sources, Levels of Exposure, and Risks that Industrial Chemicals Pose to Children's Health

cec.org

Commission for Environmental Cooperation

Disclaimer

This publication was prepared by the Secretariat of the Commission for Environmental Cooperation (CEC). The views contained herein do not necessarily reflect the views of the CEC, or the governments of Canada, Mexico or the United States of America.

The National Pollutant Release Inventory (NPRI) and the Toxics Release Inventory (TRI) data sets used in this report are constantly evolving, as facilities revise previous submissions to correct reporting errors or make other changes. For this reason, Canada and the United States each "lock" their data sets on a specific date and use those locked data sets for annual summary reports. Each year, both countries issue revised databases that cover all reporting years. The CEC follows a similar process. For the purposes of this report, the TRI data set of June 2004 and the NPRI data set of July 2004 were used. The CEC is aware that changes have occurred since this time to both data sets for the reporting year 2002 that are not reflected in this report.

Publication details

Publication type: CEC Project Report
Publication date: May 2006
Original language: English
Review and quality assurance procedures:
Reviewed by the Parties: 1 June–13 July 2005 *and* 26 October–25 November 2005
Peer review (expert meeting): 18–19 November 2004
Peer review of draft: 1 June–1 August 2005
Public review: 8 April–15 June 2004

For more information: see Acknowledgements.

Published by the Communications Department of the CEC Secretariat.

For more information about this or other publications from the CEC:
Commission for Environmental Cooperation
393, rue St-Jacques ouest, bureau 200
Montréal (Québec) Canada H2Y 1N9
Tel : (514) 350-4300 Fax : (514) 350-4314
info@cec.org http://www.cec.org

Please cite this document as: Commission for Environmental Cooperation 2006. *Toxic Chemicals and Children's Health in North America: A Call for Efforts to Determine the Sources, Levels of Exposure, and Risks that Industrial Chemicals Pose to Children's Health.* Montreal: Commission for Environmental Cooperation.

ISBN: 2-923358-35-X

Legal Deposit: Bibliothèque nationale du Québec, 2006
Legal Deposit: National Library of Canada, 2006

Disponible en français – Disponible en español

Printed in Canada on paper containing 100% post-consumer waste fibers.

Toxic Chemicals and Children's Health in North America

A Call for Efforts to Determine the Sources, Levels of Exposure, and Risks that Industrial Chemicals Pose to Children's Health

Commission for Environmental Cooperation – May 2006

Table of Contents

Tables and Figures

Figure

Table

Preface

There are many factors that interact to influence children's health. We have come to recognize that children interact with their environment differently from adults and that physical, biological and behavioral characteristics of children often make them more vulnerable to environmental contaminants. Thus, a better understanding of the underlying environmental and health factors can lead to an improved quality of life and well-being for our future generations.

To better understand the interaction between health and the environment, the CEC's governing Council adopted the Cooperative Agenda for Children's Health in North America, in 2002. The primary purpose of this initiative has been to foster collaboration and the sharing of expertise across Canada, Mexico, and the United States and to provide policy-makers with the information needed to adequately address the environmental risks to children's health. The present report is the fruit of that collaboration.

Previous trinational cooperation in this area resulted in the publication of *Children's Health and the Environment in North America: A First Report on Available Indicators and Measures* in January 2006, which highlighted progress and identified information gaps concerning the link between health and the environment. Now, as we look at toxic chemicals and the data from the national pollutant release and transfer registers (PRTRs) of Canada and the United States, we again find that further efforts are required and additional tools are needed to better understand the risks to children.

Those familiar with the CEC's annual *Taking Stock* report on North American industrial pollution will notice a few differences in how we have analyzed the data for the present report. For instance, to make the pollution data more meaningful and easier to interpret, we have adopted the parameter known as *toxic equivalency potentials*, or TEPs, for both carcinogenic and non-carcinogenic risks. The report also includes specific recommendations for action to protect children's health from toxic chemicals in our environment.

In order to place chemical pollution into an appropriate context, the report frames children's health in terms of the major factors involved with disease, disability and death. Looking at PRTR information from 2002—our most recent year for matched data—the report analyzes groups of chemicals that are known or suspected to cause cancer, learning and behavioral changes, and neurological or developmental damage. It also examines individual chemicals associated with health effects in children.

It finds that almost half a million tonnes of chemicals *known or suspected to cause cancer* were released and transferred in Canada and the United States in 2002. It also finds that there was a similar amount of releases and transfers of chemicals *recognized to cause developmental and reproductive damage*. In addition, the report looks at *suspected developmental and reproductive toxicants* and *suspected neurological toxicants*. There were over two million tonnes of releases and transfers of chemicals in these categories.

Unfortunately, these amounts are likely underestimates of the actual chemical load because the data do not include all chemicals or all sources. Furthermore, these are annual estimates; each year we are adding to the cumulative load of chemicals released into the environment. Also, chemicals that persist a long time in the environment and travel far from their points of origin may not be covered by the national PRTR databases.

The good news, however, is that the amounts of carcinogens, developmental toxicants and reproductive toxicants and neurological toxicants released and transferred have decreased overall by 7 to 28 percent from 1998 to 2002. Clearly, the national programs and legislation guaranteeing the principles of "community right-to-know" have helped to drive reductions in pollutant releases and transfers, as have the continuing efforts of industry to improve efficiency and incorporate pollution prevention strategies.

I trust this report will be a starting point for government and nongovernmental organizations, industry, and citizens alike to identify steps that can be taken to further reduce releases and transfers of chemicals, especially those of concern to children's health.

William V. Kennedy
Executive Director

Acknowledgements

A number of people have contributed greatly to this report. The CEC Secretariat gratefully acknowledges the work of Dr. Lynn Goldman, who worked tirelessly to develop the report and bring it to fruition. In the early stages, Dr. Goldman worked with colleagues from all three countries in drafting the report: Canada—Dr. Alan Abelsohn, Kathleen Cooper, Rick Findlay, Dr. Kapil Khatter, Theresa McClenaghan, and Pollution Probe; Mexico—Horacio Riojas Rodríguez and Isabelle Romieu; United States—Samar Khoury. In the report's final stages, she was assisted by Ruth Quinn, Ellen Wells and Jennifer Nielsen. The CEC Secretariat would also like to thank Dr. Catherine Miller, who conducted most of the data analysis, and Sarah Rang, who contributed greatly to the writing, editing and revision of various drafts. The members of the former Expert Advisory Board on Children's Health and the Environment in North America also deserve recognition for their work in reviewing the draft report and for their unwavering support for the CEC's work in this area. They are: Canada: Dr. Irena Buka, Dr. Claire Infante-Rivard, Jane MacDonald; Mexico: Dr. Irma Aurora Rosas Pérez, Dr. Alvaro Roman Osornio Vargas, Dr. Mariano Enrique Cebrian García; United States: Ms. Beatriz Barraza-Roppe, Dr. Bruce Lanphear, and Dr. Alan Wolf.

The CEC Secretariat is greatly indebted to the experts who reviewed the draft report and participated in the expert review meeting held in Montreal in November 2004. They are: Canada: Warren G. Foster, Geoffrey C. Granville, Donald T. Wigle; Mexico: Enrique Cifuentes, Álvaro Román Osornio Vargas; United States: Daniel A. Goldstein, Melanie Marty, and Jennifer Beth Sass. Also, our gratitude goes to John Buccini for his skillful chairing of the meeting, and to the government representatives and invited speakers who all contributed to making the meeting a success. Nor would this report be possible without the data provided by covered industries and reviewed, analyzed and compiled by governments to support these analyses.

People from all over North America also contributed to the development of this report by providing their comments during the public review period in April–June 2004. Twenty-six submissions were received from industry groups, companies, public interest groups and government who took the time to thoroughly review the draft report and provide the many helpful suggestions and comments that greatly improved the quality of the report. The Consultative Group for the North American PRTR (Pollutant Release and Transfer Register) Project also contributed feedback and ideas that informed the report's development. Additionally, the Secretariat submitted drafts of the report for review and comment by the Parties in late 2005.

A number of CEC Secretariat staff were involved in the development of this report. Erica Phipps, former program manager for the CEC's PRTR and Children's Health and the Environment initiatives, was responsible for guiding the report's development in its early stages. Victor Shantora, former head of Pollutants and Health, and Keith Chanon, current program manager, were instrumental in bringing the report to completion. Marilou Nichols, program assistant, provided indispensable administrative support and Joanne O'Reilly coordinated work on the preparation of the draft text. The CEC's publications staff handled the editing, translation and production of the document in the three languages. Evan Lloyd, director of communications, and Spencer Ferron-Tripp, media and outreach officer, were responsible for the launch of the document.

Executive Summary

Across North America, in every school, playground and home are the eager faces of our children. We do our best to ensure they grow up healthy. Social, biological and environmental factors interact in complex ways to affect their health. In this report, we focus on one of these environmental factors—toxic chemicals—that can affect children's health adversely.

There are many factors that interact to determine the health of children. Biological factors (age, genetics and gender), social factors (income level, culture and behavior), and broad environmental factors (lifestyle factors and exposures to pollutants) have all been documented as playing major roles in determining children's health. While the focus of this report covers the releases of and potential for exposure to certain industrial chemicals, and pollutants in air, water and the ambient environment, it is recognized that any effort to improve the health of children needs to take a broad approach that would include attention to lifestyle factors like diet, exercise and prevention of harmful exposures like tobacco smoke.

Children are uniquely vulnerable to many environmental threats to good health. Compared to adults, children inhale more air, breathe more rapidly, eat more food, and drink more water per kilogram of body weight. They live closer to the floor where some pollutants tend to accumulate, are more likely to ingest contaminated soil and dust, and spend more time outdoors. In addition to these increased pathways of exposure, children's bodies are also more vulnerable. There are periods of vulnerability in fetal development and childhood, when the lungs, brain, and immune, reproductive, and other systems are maturing. Harmful exposures during these critical developmental windows can lead to lifelong alterations in behavior and functional status, disease occurrence and development. Childhood is a critical life phase, through which we all pass; children's health cannot be separated from health in later life stages.

Children in North America

There are several childhood health effects that are of particular concern in North America. These include: cancer, developmental and learning disabilities and behavioral problems, birth defects, preterm birth, intrauterine growth restriction, asthma and other respiratory diseases, infections (respiratory and gastrointestinal) and injuries. In the absence of common reporting methods for diseases across North America, information must be drawn from national surveys in each country. This lack of a common reporting system is one of the common barriers to understanding the links between childhood diseases and their underlying causes (Goldman *et al.* 1999).

Sources of Information

Information about the amounts of chemicals being released from industrial facilities into the environment in North America is available through national pollutant inventories, known as pollutant release and transfer registers (PRTRs). These inventories, which cover specific chemicals and specified industrial sectors, have been developed by a number of countries around the world. Canada's PRTR is called the National Pollutant Release Inventory (NPRI) and the US inventory is called the Toxics Release Inventory (TRI). Mexico is implementing mandatory reporting under its PRTR, the *Registro de Emisiones y Transferencia de Contaminantes* (RETC), which until 2005 has been voluntary.

Every year in Canada and the US, industries that meet certain criteria must report on the amount of chemicals released into the air, land, or water, or injected underground. The amount of chemicals transferred off-site for disposal, treatment and recycling is also reported. This information is collected by regulatory agencies in national governments each year and compiled into annual reports and electronic databases, which are accessible to the general public.

This report analyzes publicly available data from Canada's National Pollutant Release Inventory (NPRI) and the US Toxics Release Inventory (TRI) for the reporting year 2002. At that time, reporting to Mexico's RETC was voluntary. Because of the differences between mandatory and voluntary data, data from Mexico's RETC are not included in this PRTR analysis. This report also matches the chemicals and industrial sectors that are in common between the NPRI and the TRI, thus creating a matched data set that is amenable to analysis. This matched NPRI-TRI data set therefore does not consider data which are unique to one system, such as on-site recycling, reporting from the metal mining sector and some chemicals such as ammonia and hydrogen sulfide.

Analysis of the Releases of Carcinogens, Developmental Toxicants and Neurotoxicants in North America

This report analyzes the chemicals from industry sectors reported to both the US TRI and the Canadian NPRI. Many of these chemicals can fall into the following categories: known carcinogens, known or suspected developmental toxicants and suspected neurotoxicants. An individual chemical may fall into more than one of these categories. Each year, certain industrial facilities must report to these registries on the amounts of the PRTR-listed chemicals released into the air, land, or water or

Children are uniquely vulnerable to many environmental threats to good health. Compared to adults, children inhale more air, breathe more rapidly, eat more food, and drink more water per kilogram of body weight.

injected underground in North America. For this report, releases are reported in metric tonnes ("tonnes") or in kilograms ("kg").

Total releases and transfers of these chemicals reported in 2002 to the Canadian and US PRTRs and entered into the respective PRTR datasets, by category, included almost one-half million tonnes each of carcinogens and of recognized developmental and reproductive toxicants, two and one-quarter million tonnes of suspected developmental and reproductive toxicants, and over two and one-half million tonnes of suspected neurotoxicants.

Toxic chemicals arising from two sectors, primary metals and chemical manufacturing, are responsible for a large percentage of total releases. Other sectors, such as manufacturers of rubber and plastics products, are also large emitters of these substances. Other large releases resulted from manufacturers of paper products and of transportation equipment. Three jurisdictions in North America (Texas, Ohio and Indiana) released the largest amounts of carcinogens on the two PRTR lists in 2002. Tennessee, Ontario and Texas released the largest amounts of recognized developmental and reproductive toxicants.

It is very encouraging to observe that the released quantity of known carcinogens has decreased by 26 percent from 1998 to 2002. Similar downward trends were found for developmental/reproductive toxicants, with a decrease in the United States and Canada of 28 percent from 1998 to 2002.

Interpreting PRTR Data

PRTR data provide important insights into the large amounts of chemicals entering our environment each year from industrial releases but they tend to underestimate the actual loads of chemicals into the environment because inventories, by design, collect information on a limited list of chemicals released or transferred, and only from larger industrial facilities.

Significantly, the data do not include emissions from mobile sources, agricultural sources (i.e., pesticide use), small sources, consumer products or natural sources.

PRTR data do not directly provide information on human exposure. The levels of human exposure to most of the chemicals and the relationship between human exposure levels and PRTR pollutant emissions are unknown. **Since the health risk posed by these chemicals depends on the amount of exposure or dose, as well as toxicity, it is not possible to estimate risks from PRTR data alone or the levels of risks to the health of children, or adults, from these releases.** Moreover, toxicity is a complex process that is highly dependent on such factors as the nature of the toxic effect, the potency of a substance and the timing of exposure in regard to "windows of susceptibility."

Despite these limitations, PRTR data are useful tools for developing strategies for the protection of children from potentially harmful chemicals. The reporting of releases of chemicals with the potential for reproductive, developmental, neurological or cancer toxicity to children can lead to further investigations such as monitoring for such chemicals in the air, water, soil and food in such communities, and biomonitoring of people to directly assess exposures to such chemicals. It can focus efforts around prevention of exposures from activities such as spills during transport, manufacture, and use of such chemicals. It can empower communities with information that allows them to participate in decisions about industrial activities in their communities. Finally, such data permit evaluation of efforts to reduce pollutants and waste generation by various industrial sectors.

Many Actions Are Underway to Reduce Chemical Loadings to the Environment

At multiple levels of government, in many industrial sectors and in many communities, there have been concerted efforts to reduce releases of chemicals into the environment and also to reduce children's exposure to toxic chemicals. The development of "green" industrial technologies and other forms of pollution prevention, new emission standards, the voluntary reduction of releases from companies, the requirement to report releases and transfers and community improvement programs have all helped to reduce releases. PRTR data reflect the emission reductions seen over the years in many chemicals. Well-tested processes exist to allow a continued reduction of releases. PRTRs are also valuable tools to provide the public with information relevant to their community, and to leverage industry to track and reduce their releases of chemicals.

Future Actions Are Needed

Important progress has been made in the past decades to recognize, prevent and reduce children's exposure to toxic chemicals, but more action is needed on the following fronts:

- *Monitor and reduce releases of toxic chemicals to the environment:* Specifically, we need to consider

children's health in the interpretation of PRTR data and establishment of priorities for emission reductions. We can develop methodologies to put such release data into the broader context of children's exposures. PRTR reporting in North America can be expanded to give a fuller picture, and harmonized to increase the information available on a North American basis. Governments and the Commission for Environmental Cooperation (CEC) should consider adopting a method such as the toxicity exposure potency factors used in this report, to give a clearer picture of hazard potential from releases. In so doing, data gaps in regard to hazard and exposure need to be filled. An effort should also be initiated to develop a North American approach to reporting information about pesticides, including their sales, use, concentrations, poisonings, exposure, and releases.

- *Monitor and reduce exposures to toxic chemicals:* Specifically, trilateral biomonitoring and other exposure monitoring activities under the CEC's North American Regional Action Plan (NARAP) on environmental monitoring and assessment should continue, particularly for exposures relevant to children's health. The US government should continue and expand its human biomonitoring efforts. Where excessive exposures are found, action should be taken to protect health, especially the health of children.
- *Track childhood diseases that may be related to the environment:* Across North America, efforts should be made to expand and harmonize efforts to track diseases that are possibly related to the environment and to exchange information about linkages between the environment and children's health.
- *Improve scientific knowledge:* The major gaps in our knowledge about the risks associated with exposure to toxic chemicals need to be filled and further study is needed to quantify the extent to which early-life exposure to environmental contaminants contributes to the leading causes of illness, hospitalization and death during childhood and delayed health effects later in life. In addition, governments in North America need to increase research efforts as well as efforts to provide expert assessments of children's health hazards. Finally, trinational cooperation on a longitudinal study of children's health would provide a wealth of relevant information.
- *Increase awareness of the role of toxic chemicals in children's health:* As new knowledge is acquired, efforts to prevent releases and exposures to chemicals can focus on opportunities to protect the health of children.

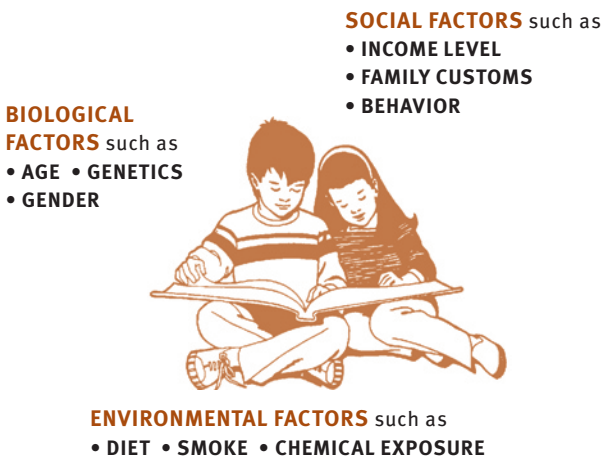
Introduction

Almost 120 million children live in North America. Many of them face economic, social and environmental challenges every day. More children than ever need daily medication to control asthma. Others struggle to control aggressive outbursts and understand difficult learning concepts. Too many, particularly in poorer areas, suffer from gastrointestinal disease. Children who live with parents or others who smoke at home are exposed whenever a smoker lights up another cigarette. Many factors are affecting the health of these children.

One of the goals of this report is to focus on one of these factors: chemical releases into the environment from industrial activities. Chemical industrial releases are one important part of the puzzle but do not give a full picture of risk since chemicals from industry are only one type of pollutant. Human exposure levels to these chemicals, and other sources of pollution, are beyond the scope of this report.

This report also aims to foster increased trilateral action to prevent and reduce children's exposure to harmful chemicals. Its focus is an analysis of available data on one category of pollutant, toxic chemicals from data obtained from the national pollutant release and transfer registers (PRTRs) in North America,[1] and emphasizes the reporting of chemical carcinogens, developmental toxicants and neurotoxicants. Although at this stage the data are available only for the United States and Canada, this report discusses in specific terms the potential impacts of these substances on the health of children in North America. It also describes the limits of what we know about these impacts based on present data. With its cross-border analysis of selected PRTR data, it provides a unique North American perspective as a basis for trilateral action.

Figure I-1. **Children's Health is the Net Result of Many Interacting Factors**



Children's Health Overview

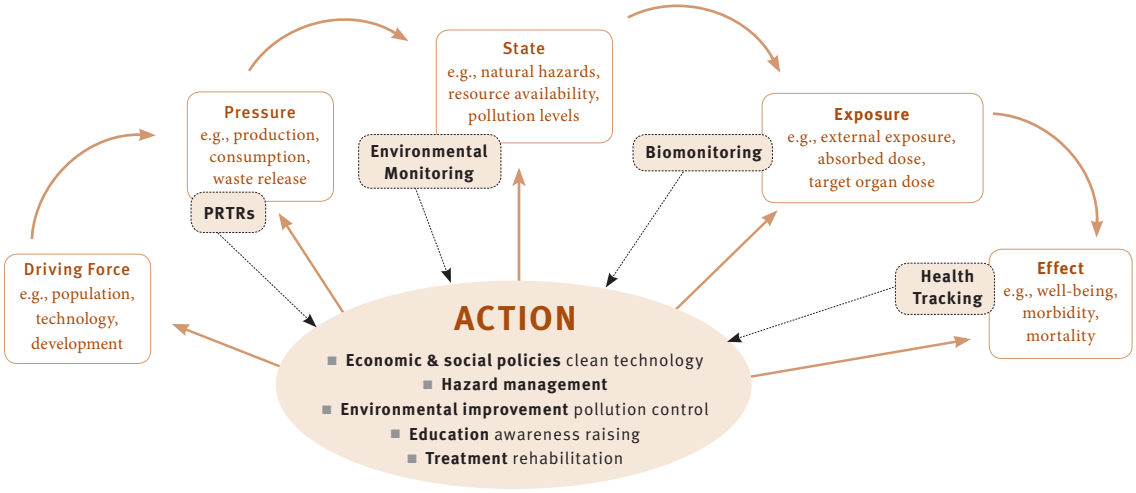
Health has been defined broadly as "a complete state of physical, mental and social well being" (WHO 1948), and more recently as "a positive concept emphasizing social and personal resources, as well as physical capacity" (WHO 1997).

Although the focus of this report is on chemicals released from industrial facilities and our state of knowledge about the potential impact of such substances on children's health, it is important to frame issues of environmental risk within the broader context of the health of children. Children's health is the net result of a complex interaction of social, biological and environmental factors (see **Figure I-1**). Social factors such as income level, educational attainment, family customs and behavior have been documented to play a major role in determining children's health. Biological factors such as age, genetics and gender all affect health. Environmental factors, such as diet, exposure to second-hand smoke, alcohol consumption, infectious agents, drugs and pharmaceuticals, injury hazards, and exposures to environmental pollutants such as radiation and chemicals contribute to disease and death in children (NRC and IOM 2004).

The WHO DPSEEA (Driving Force, Pressure, State, Exposure, Effect and Action) model (**Figure I-2**) is a useful framework for understanding the continuum, from drivers of environmental change (such as population and technology), to pressures (such as production, consumption and waste releases), to changes in environmental state (such as pollution levels), to exposure (external, internal and target organ doses), to effects on health. Government, the private sector and individuals can take action to positively effect environmental outcomes at all of these levels. Likewise, information can be used to provide feedback at all levels. Reports of chemical releases shed light on one of the initial links in this chain, namely, activities that

1. The Canadian National Pollutant Release Inventory (NPRI) and the US Toxics Release Inventory (TRI). Data from Mexico's *Registro de Emisiones y Transferencia de Contaminantes* (RETC) are not yet available.

Figure I-2: **Indicators of Environmental Health at Multiple Levels**



Source: WHO. DPSEEA model.

potentially create more pressure on the environment via the generation and release or transfer of wastes, specifically industrial activities within certain sectors such as manufacturing, mining, energy production, and waste disposal. However, such reports do not provide direct information about "downstream" effects. As shown in **Figure I-2**, *other* indicator systems are necessary to understand the state of the environment (e.g., environmental monitoring systems); exposures to human populations (e.g., human biomonitoring programs) and the state of health and well-being (e.g., tracking of mortality, diseases and measures of well being). Health effects also occur on a continuum and are related to dose and toxicity, as well as to timing of exposure; PRTR data do not inform us directly about these relationships. However, PRTR data are valuable for managing potential hazards at the facility and community levels.

Children Are Uniquely Vulnerable to Many Chemicals

Children are not small adults. Because of their unique physiology and developmental and behavioral characteristics, they are often more vulnerable to toxic chemicals. Such differences need to be taken into account when considering the potential impacts of environmental exposures (Daston *et al.* 2004). Compared to adults, children inhale more air, drink more fluids and eat more food per kilogram of body weight. Because of these differences, children often (but not always) have more intense exposure to chemical contaminants than adults (Miller *et al.* 2002).

Children also inhabit and interact with their environment differently. They live closer to the floor, where pollutants tend to accumulate, they are more likely to ingest or inhale particulates in contaminated soil and dust, and they spend more time outdoors. Because of these behavioral differences, children can have greater exposure to chemicals than adults (Goldman 1998).

In addition, because children's bodies are in dynamic states of growth and development, they can be more sensitive to chemicals than adults. A child's ability to break down and eliminate pollutants is poorly developed at birth, because the liver and kidneys are still developing. This means that at various stages of development, children may be more or less capable of breaking down, excreting, activating endogenous enzymes or inactivating toxic substances (Ginsberg *et al.* 2004, Hattis *et al.* 2003). Because children are at the beginning of their lives, effects with a long latency period may manifest themselves much later in life. These differences in children's size, behavior and development mean that they may be more susceptible to environmental contaminants like toxic chemicals, and that research is needed in order to identify and prevent such hazards (Landrigan 1998).

Children Have "Windows of Vulnerability"

Because children are rapidly growing and developing, there are time periods, or so called "windows of vulnerability," from gestation through adolescence where systems are particularly sensitive to damage. Any harmful exposure during these critical developmental windows can lead to lifelong alterations in behavior, disease, growth and development. The periods surrounding conception and during pregnancy, just after birth and during infancy, have long been recognized as critical windows for exposure to many contaminants but are receiving increased attention in recent years as we learn more about early human development. Currently, scientists are studying the sensitivity of the fetus to toxic chemicals and are increasingly recognizing the fetal stage as one of the most vulnerable developmental windows. For example, exposure to small amounts of chemicals during critical days of fetal development can change the architecture of the brain. This poses a new challenge: to identify when during a child's development the exposure to chemicals has taken place (Selevan *et al.* 2000).

Why a North American Report on Toxic Chemicals and Children's Health?

This report reflects the three governments' commitment to work together as partners through the Commission for Environmental Cooperation (CEC).

The preparation of this report by the Secretariat of the CEC was authorized under Council Resolution 02-06 "Cooperative Agenda for Children's Heath and the Environment in North America."

The report builds upon work of the CEC and the member states in:

- Analyzing chemicals reported to pollutant release and transfer registers in North America (*Taking Stock* reports);
- Coordinating trilateral efforts to monitor and reduce contaminants through the Sound Management of Chemicals (SMOC) initiative;
- Documenting the ability of some contaminants to travel long distances (*Continental Pollutant Pathways* [CEC 1997]);
- Presenting linkages between children's health and the environment (*Making the Environment Healthier for Our Kids: An Overview of Environmental Challenges to the Health of North America's Children* [CEC 2002]);
- Developing indicators of environmental effects on children's health in North America;
- Publishing the report *Health Impacts of Air Pollution on Morbidity and Mortality Among Children of Ciudad Juárez, Chihuahua, Mexico* (CEC 2003); and
- Publishing an inventory of North American power plant air emissions (CEC 2004).

Pollutant Release and Transfer Registers (PRTRs)—A Source of Information on Chemicals from Industrial Activities

PRTRs are important sources of information about the amount of chemicals being released into the environment from industrial facilities. Every year across North America, select industries report on the amount of chemicals released into the air, land, and water and injected underground. The amount of chemicals transferred off-site for disposal, treatment and recycling must be reported also. This information is collected by national governments each year and compiled into annual reports and electronic databases. This report analyses the matched data[2] reported to the Canadian National Pollutant Release Inventory (NPRI) and the US Toxics Release Inventory (TRI). The Mexican inventory, the *Registro de Emisiones y Transferencia de Contaminantes* (RETC), remains under development and data from Mexican facilities are not yet publicly available.

Environmental hazards come in a variety of forms, including: biologic agents (e.g., molds), radiation (e.g., ionizing, sunlight); air pollutants in smog, like nitrogen oxides, sulfur dioxides, particulate matter and ozone; greenhouse gases; and toxic chemicals and pesticides in water, land and food and other consumer products. PRTR data provide information on one category of pollutants: toxic chemicals released to the environment from industrial activities.

Methods and Scope of This Report

Building on methodologies developed for the CEC's annual *Taking Stock* report series, this report analyzes, from a children's health perspective, publicly available PRTR data.

It focuses generally on children up to the age of 18 years, depending on the data available. Exposure to chemicals prior to birth can also be important to a child's future development, and so is discussed in this report.

The report is arranged in the following way:

- **Chapter 1** describes the demographics and major causes of death, illness and disability for children in North America.
- **Chapter 2** describes the sources, pathways and health effects of chemicals.
- **Chapter 3** analyzes industrial pollutant release and transfer data for carcinogens, developmental toxicants and neurotoxicants, and other chemicals of concern to children's health.
- **Chapter 4** describes examples of current programs to prevent and reduce children's exposure to chemicals and provides an overview of recommendations for actions to reduce releases of and prevent exposure to toxic chemicals.
- A **Resources** section lists governmental agencies and other organizations which can provide further information.
- **References** for the entire report are to be found following the main body of the report. The reader is encouraged to explore these and other documents for understanding of particular issues in greater depth.
- **Appendixes** offer source data and supplementary information.
- **Tables** referred to in sections 1 and 3 follow the Appendixes.

This report focuses on selected chemicals and presents some forward-looking recommendations for action. It is not a report on risks, but rather information and analysis of the sources of some chemical industrial releases reported by the PRTRs of Canada and the United States.

2. A "matched" data set includes only those chemicals and those industrial sectors common to both systems. Thus, data on chemicals reported to one system but not the other are not included. Similarly, data from industrial sectors required to report to one system, but not the other, are not included.

1 Children in North America

1.1 Demographics

The nearly 123 million children in North America are our most precious resource. In 2003, the United States had the largest number of children in North America, with over 75 million children, followed by Mexico, with over 39 million and Canada, with nearly 7 million (**Figure 1-1**).

Comparing the three countries, in Mexico, children account for a larger share, over one-third (38 percent), of the total population. Children make up about one-fifth (22 percent) of the total population in Canada, and one-quarter of that in the United States (25 percent) (**Appendix A**). Mexico also has a larger percentage of children less than five years of age. Over 11 million children in Mexico, or nearly 11 percent of the population, are less than five years old. In Canada and the United States, about six percent of the population is less than five years old (**Figure 1-2**).

This difference in age distribution in North America is largely a result of differing birth rates. Mexico has the highest birth rate, with an average 2.5 births per woman. Next is the United States, with a birth rate of 2.1 and then Canada with 1.5 births per woman, over a reproductive lifetime (UNICEF 2005).

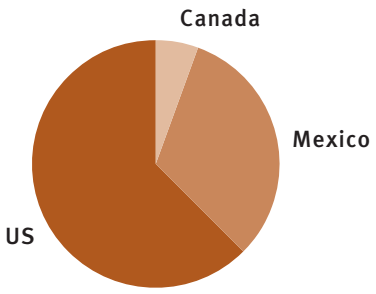
The numbers of children in North America will expand rapidly over the next decade. Mexico will have the largest percentage increase, with a projected population of 31.5 million children less than 15 years of age by 2015. The United States will have almost 66.8 million children less than 15 years of age by 2015. Canada is the exception to this, with the number of children under 15 years of age expected to decline in the future, from 6 million in 1998 to 5.05 million by 2015 (United Nations Population Division 2005).

1.1.1 Children Living in Poverty

Many of the children in North America—approximately 23 million, or 20 percent—live in poverty, which increases the likelihood of environmental health problems. Mexico and the United States now top the list of Organization for Economic Cooperation and Development (OECD) countries with the largest percentages of children living in "relative" poverty (living in a household where income is less than half the national median). About one in four children in Mexico (26 percent), one in five children in the United States (22 percent), and one out of six children in Canada (16 percent) are "relatively" poor (UNICEF 2000).

Poor children can have limited access to clean water, health care, food, and housing. Children in low-income homes or attending older schools in poor condition can be exposed to lead from deteriorated old paint and to pesticides from frequent applications used to reduce pest infestations. Parents or siblings may work in the dirtiest, most hazardous jobs, which increases the probability of "take home" exposures (Chaudhuri 1998). Also, poor children are more likely to live in polluted

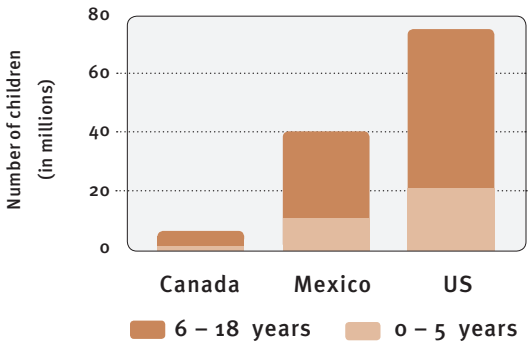
Figure 1-1 Distribution of Children, Ages 0 to 18 Years, in North America in 2003



Total – 122,635,000 children

Source: UNICEF 2005.

Figure 1-2 Age Distribution of Children in North America in 2003



Source: UNICEF 2005.

areas or close to polluting factories. They are also more likely to suffer from hunger and malnutrition, which can reduce the body's ability to withstand environmental pressures. For example, poor nutrition may result in more lead being absorbed in the body (see, for instance, Calderon *et al.* 2001, Bradman et al. 2001, and Mahaffey *et al.* 1986). Poor children can be therefore challenged by the combined threats of poverty, undernutrition and increased exposure to toxics. It should be noted that children don't necessarily have to be hungry to suffer poor nutrition. In North America, for example, where foods can be calorie-rich but nutrient-poor, even ample amounts of food can lead to malnourishment.

1.1.2 Children in Urban and Rural Environments

About three-quarters of the 122.6 million children in North America live in urban areas. The percentage of people living in urban areas is similar among the three nations (80 percent for Canada, 80 percent for the United States and 75 percent for Mexico) (UNICEF 2005). Children living in urban and rural areas may face different sources of environmental pollution. In Mexico, people in rural areas are less likely to have access to safe drinking water and sanitation services. It is estimated that in rural areas in Mexico, 28 percent of people lack access to improved drinking water and 61 percent to adequate sanitation services. For urban areas, three percent of Mexican people lack access to safe drinking water and ten percent to adequate sanitation services (UNICEF 2005).

1.1.3 Race and Ethnicity

The children in North America are from a variety of backgrounds. In Canada, children are predominately Caucasian. Approximately 1.3 percent of children under the age of 15 years old are of Asian background; over 0.5 percent of children have an indigenous background; and a smaller percentage come from black, Arab/ west Asian and Latin American backgrounds. In Mexico, almost 13 million people, or 13 percent of the total population, are indigenous (National Indigenist Institute 2001). About seven percent of the Mexican population speak an indigenous tribal language. According to the 2000 US Census, almost 30 percent of US children under the age of 15 are from minority groups. About 13 percent of US children have Latin American backgrounds, 12 percent are of African American descent, almost 4 percent of children have Asian heritage and about 1.3 percent are of indigenous heritage (FIRCFS 2001).

Ethnicity does correlate with differences in environmental exposure. Children from minority backgrounds are often at a greater risk of exposure to toxic chemicals. In the United States, several studies have noted a higher proportion of African American, Hispanic and Native American children who live within one mile of a US National Priorities List hazardous waste site. For example, African Americans are over-represented in many of the counties in the United States with the highest air emissions of developmental toxicants (Institute of Medicine 1999b).

1.2 Causes of Death in North American Children

The good news is that over the past 40 years in Canada, Mexico and the United States, infant and child (under five years old) mortality rates have decreased and life expectancies are rising (UNICEF 2005). Across North America, perinatal disorders, which include preterm birth, low birth weight and complications from pregnancy, labor and delivery are leading causes of infant mortality. Some of these perinatal disorders are the result of a number of factors, including poor nutrition, lack of medical care, cigarette and other smoke, infectious diseases and environmental and occupational exposures. In 1999, the leading cause of infant death in Canada was birth defects, accounting for 26.5 percent of all infant deaths, followed by preterm birth and sudden infant death syndrome (SIDS). Infant mortality due to major congenital anomalies has decreased significantly in Canada, from 3.1 per 1000 live births in 1981 to 1.9 per 1000 live births in 1995 (Health Canada 2003). Similar trends have been observed in the United States.

However, mothers, infants and children face different health challenges in each of the three countries of North America. In Mexico, 55 mothers die with every 100,000 live births (UNICEF 2003). In the United States and Canada the rate is much lower.

Mexican infants (less than one year old) are more likely to die than infants born in Canada or the United States. The rate of death from congenital malformations and from perinatal disorders in Mexican infants is more than twice that of Canada and the United States; the rate of death from infectious intestinal disease, 18 times; from influenza and pneumonia, 16 times; from unintentional injuries, three times; and from asthma, four times (see **Table 1-1**). The Mexican Ministry of Health has reported that asthma was the 11th-largest cause of mortality for children under five and ranked 16th among those ages 5 to 14 (SSA 2001). These increases in death due to infectious causes in Mexico continue through childhood. In contrast, in Canada and the United States, childhood cancer has become the most significant disease-related cause of death. From preschool age through adolescence, injuries take a prominent role in mortality in all three countries. School-age children in Mexico and in the United States are ten times more likely to die of asthma than those in Canada.

These disparities in mortality are known to result from a number of factors, most related to poverty, which are not the same from one country to another (Black *et al.* 2003). First, infants who live in conditions of poverty are more likely to live in circumstances that are associated with exposure to infectious agents. For example, pathogens contaminating food and drinking water and overcrowded living conditions are conducive to secondary spread of intestinal and respiratory pathogens from older children and adults to infants (WHO 2003). Second, children who live in conditions of poverty throughout North America are more likely to be less well nourished, which increases susceptibility to infectious diseases. In this regard it is heartening to see that rates of child mortality from infectious causes in the Americas have been decreasing over time; this decrease is attributed to better nutrition and safer water and food sup-

plies (*Epidemiological Bulletin* 1991). Likewise, the poorest children are most likely to live in the polluted environments. Severe air pollution is known to increase rates and severity of respiratory infections (Rosales-Castillo *et al.* 2001). Research continues to explore the potential negative effects of exposures to toxic substances in the environment on children's health as well as the interactions between environment and poverty. Poor infants and their families are less likely to benefit from preventive medical interventions such as vaccinations; in the United States, poor children are much more likely to have delayed immunizations (Wood 2003). Finally, infants in poverty, particularly in the United States and Mexico where there are more financial barriers to basic medical care, are more likely to have delayed access to medical care; even simple, yet sometimes live-saving, interventions, such as oral rehydration therapy for infants with severe intestinal disease, can be difficult to access (Gutierrez *et al.* 1996).

The different causes of death across the varied stages of childhood in North America suggest the need for multiple prevention strategies. In infancy, the priority may be on preventing preterm births; improving access to medical care for mothers during pregnancy, labor and delivery; and preventing congenital malformations. Across poor communities in North America, provision of sanitation and of safe drinking water is also a priority, as well as reduction of air pollution in severely polluted areas, which most certainly contributes to the morbidity and mortality from infectious respiratory diseases and asthma. For preschoolers, the priority may be prevention of injuries and, particularly in Mexico, the prevention of malnutrition, anemia and infectious diseases would contribute to marked improvements in children's health. For older children across North America, the prevention of injuries could be a priority and childhood cancer stands out as the most important disease-related cause of death.

1.3 Diseases Related to Environmental Pollution Affecting North American Children

As is noted above, infectious agents and injury play very significant roles in mortality (deaths) of children in North America; this is true for morbidity (disease) as well. The focus of this report is on releases of chemicals from industrial facilities that may affect children's health. In this regard, there are a number of health conditions in children that are significant and that may be associated with environmental pollution, as well as other factors. These include:

- Cancer;
- Learning, developmental and behavioral disabilities;
- Birth defects;
- Impaired endocrine function; and
- Respiratory problems, such as asthma.

The following is a brief overview of these health endpoints. Although important, other environmentally related diseases, such as gastrointestinal disorders, vector-borne diseases like malaria, and respiratory infections, are beyond the scope of this report.

1.3.1 Childhood Cancer

Although relatively rare, for children between the ages of 1 and 19, cancer ranked fourth as the cause of death, behind unintentional injuries, homicides, and suicide. A newborn has approximately a 0.3 percent probability of developing cancer by the age of 20 years (Ries *et al.* 1999).

For cancer in general, much of what we know about causal agents has to do with occupational exposures to adults (benzene, asbestos, ionizing radiation, arsenic) and lifestyle factors such as tobacco. Many possible factors can play a role in the development of childhood cancer, including genetic abnormalities, ionizing radiation, viral infections, certain medications, tobacco, alcohol, and industrial and agricultural chemicals (Zahm and Devesa 1995, Schmidt 1998, Birnbaum and Fenton 2003).

In Canada and the United States, leukemia is the most common childhood cancer, followed by brain cancers (NCIC 2002, Ries *et al.* 2001). In Mexico, mortality statistics may provide a better picture, due to under-reporting of cancer morbidity. In 1996, in Mexico, cancer was the eighteenth-leading cause of death in children aged five and under, and the eighth-leading cause in children 4 to 14 years old (SSA 1997).

Some types of childhood cancers are increasing. In the United States, overall childhood cancer incidence rates increased 13 percent from 1973 to 1997 (Ries *et al.* 2001). During that period in the United States, rates of increase for specific childhood cancers were: 30 percent for non-Hodgkin's lymphoma, 21 percent for brain cancer and 21 percent for acute lymphocytic leukemia (Ries *et al.* 2001). Some scientists feel that the increase in incidence is due to diagnostic improvements and reporting changes. Despite the above indications, more children are also surviving cancer (Ries *et al.* 2001). The decline in the death rate is due to improved treatment of common childhood cancers, especially leukemia (Ries *et al.* 1999). Unfortunately, the most common cancer treatment regimes involve chemicals and radiation, themselves cancer-causing agents. Because of this, childhood cancers often recur in adult life, making primary prevention of cancer an extremely important health goal.

Certain types of cancers are also increasing in young Canadian adults (ages 20 to 44), such as non-Hodgkin's lymphoma and thyroid cancer in both men and women, lung and brain cancer in women and testicular cancer in men (NCIC 2002). Data released in Canadian Cancer Statistics reported a long-term increase in testicular cancer in young males, with an average rate of 1.7 percent increase per year between 1987 and 1996 (NCIC 2002). Given that cancer in young adults reflects a relatively short latency, contributing factors could well have occurred during prenatal development and childhood. This increases our need to further understand risk factors and to eliminate or prevent these at as early an age as possible.

Epidemiological studies have reported that a range of environmental and medical exposures to chemicals are associated with childhood cancers, but clear scientific consensus exists only for diethylstilbesterol and radiation (Anderson *et al.* 2000).

There is limited, but not conclusive evidence that parental or childhood increased exposure to pesticides, such as home, lawn and garden pesticides, may confer an increased risk of a number of some childhood cancers such as leukemia, neuroblastoma, Wilms' tumor, soft-tissue sarcoma, Ewing's sarcoma, non-Hodgkin's lymphoma and cancers of the brain, and testes (Zahm and Ward 1998, Birnbaum and Fenton 2003). However, these studies rarely point to individual agents as being involved, are based on small numbers of exposed subjects, and have potential problems with recall bias among parents of children with cancer, compared with control parents. Another review of childhood brain cancer (Baldwin and Preston-Martin 2004) identifies parental occupational exposures and pesticides as among the exposures that may be involved with childhood brain cancer. Although they concluded that perinatal exposures were most likely linked to such cancer, they could make no firm conclusions about what causes childhood brain cancer. Most recently, evidence is accumulating that at least one type of childhood cancer, acute leukemia, begins prenatally with chromosomal breakages and translocations, but also requires environmental exposures later in conception or postnatally. Insufficient maternal levels of the B vitamin, folic acid, during conception may also play a role (McHale and Smith 2004).

Of particular concern more recently is the broader issue of perinatal and childhood carcinogenesis, which could be manifested as childhood cancer but also could result in increased risk of cancer over a lifetime. A scientific consensus is emerging that the *in utero* and early childhood period is a "critical window of exposure" for carcinogens; that is, that there is increased sensitivity of the fetus and young child to carcinogens (Anderson *et al.* 2000, Birnbaum and Fenton 2003, Hattis *et al.* 2004). For carcinogens that act via mutagenic mechanisms, the US EPA has just completed development of guidance to adjust the risk of cancer derived from (adult) animal models by 10-fold for the first two years of life and three-fold for years 3 to 15 (US EPA 2005a). Current regulatory standards do not reflect this consideration of increased carcinogenicity risk to the fetus and child (US EPA 2005a-b). In light of the human experience with diethylstilbestrol, there are concerns that carcinogens acting through some other mechanisms may also demonstrate increased carcinogenic risk to the fetus and child (Anderson *et al.* 2000).

1.3.2 Learning and Behavioral Disabilities

Another childhood health issue is learning and behavioral disabilities. Learning disabilities refer to a number of disorders which may affect the acquisition, organization, retention, understanding or use of verbal or nonverbal information. These disorders affect learning in individuals who otherwise demonstrate at least average abilities essential for thinking and/or reasoning. As such, learning disabilities are distinct from global intellectual deficiency. Learning disabilities range in severity and may interfere with the acquisition and use of one or more of the following: oral language (e.g., listening, speaking, understanding), reading (e.g., decoding, phonetic knowledge, word recognition, comprehension), written language (e.g., spelling and written expression) and mathematics (e.g., computation, problem solving). Learning disabilities may also involve difficulties with organizational skills, social perception, social interaction and perspective taking. Learning disabilities are lifelong (LDAC 2002).

Learning and behavioral disabilities result from many complex interactions of genetic, social and environmental factors, often during a critical time in a child's development. Toxic chemicals, one of the many interacting factors, are of special concern because they are a preventable cause of damage. Low-level exposures to some toxic chemicals have been found to cause changes in measures of ability such as intelligence, as assessed by IQ tests, of children. Relatively low-level prenatal and/or postnatal exposure to three substances in particular, lead, polychlorinated biphenyls (PCBs), and methylmercury, have been associated with small decreases in intellectual and neurological function. For example, high levels of exposure to lead, high enough to cause other symptoms of ill health, can also cause severe impacts such as mental retardation. However, lower-level exposures that do not cause noticeable symptoms are associated with an average decline in IQ scores. On a population basis, the impacts of widespread exposure to such a neurotoxicant can be profound; for example, a four-point shift downward in IQ for a population results in a quadrupling of the proportion of children with IQs of less than 80 (Bellinger 2004). Although the evidence for PCBs and methylmercury is less well established, expert scientific bodies have concluded that these also result in neurotoxicity to children exposed at levels found in the environment (NRC 1996, ATSDR 2000).

Major developmental disabilities exact a large toll on public health. Nearly 17 percent, or 12 million, of US children suffer from one or more learning, developmental or behavioral disabilities (CDC 2003b). Learning disabilities alone may affect 5 to 10 percent of US children (Goldman and Koduru 2000). In Canada, 28 percent of Canadian children (ages 0 to 11) have at least one identifiable learning or behavioral problem and 16 percent of Canadian children (ages four to five) show delayed vocabulary skills (Landy and Tam 1998). No comparable data are available for Mexico.

Attention deficit hyperactivity disorder (ADHD) is also a major problem for children in North America. For example, in the United States, methylphenidate (Ritalin), a central nervous system stimulant, has been prescribed to approximately 1.5 million US children to control ADHD. The number of US children taking this drug has doubled every four to seven years in the United States since 1971. ADHD is estimated to affect three to six percent of all school children, with some evidence to suggest rates as high as 17 percent in the United States (CDC 2003b). However, it is not clear whether the actual prevalence of the underlying disorder has increased, or whether this represents changes in diagnosis and treatment. ADHD seems to be strongly related to genetic inheritance but is also related to environmental factors. Exposures to some toxic chemicals such as lead, manganese, solvents, dioxins

and PCBs, and pesticides have to varying extents been linked to changes in behavioral areas such as activity levels and attention, but it is not yet known if these chemicals are related to ADHD (Goldman and Koduru 2000). For example, lead is known to cause reduced attention spans, distractibility and aggressive behavior in children at levels well below those that cause clinical symptoms (Lanphear *et al.* 2000). PCBs and methylmercury have also been reported to cause adverse impacts on IQ and behavior, with low-level exposure (Grandjean *et al.* 1997, Longnecker *et al.* 1997). Toxicology studies of primates indicate that exposure to lead and PCBs produces symptom manifestations that appear to be quite similar to ADHD (Rice 2000). Although these data are intriguing, at this time we do not have evidence from human studies to confirm or refute whether ADHD is related to exposure to environmental chemicals.

As many as 2 per 1000 US children may suffer from autism. For example, California's autism rates increased nearly 2.5-fold between 1987 and 1994. It is not yet known whether this increase is "real" or due to changes in diagnosis (Croen *et al.* 2002). Autism is believed to be caused by a combination of genetic and environmental factors interacting early in life. Recent investigations have not found associations between vaccinations (measles, mumps, rubella—MMR) or vaccine preservatives (thimerosal) and autism (Muhle *et al.* 2004). Recently researchers have reported that some autistic children have abnormal metabolic profiles that indicate an increased vulnerability to oxidative stress (James *et al.* 2004), perhaps a clue to the genetic and environmental origins of this devastating disease. However, the potential role of environmental factors in autism is unknown and largely unexplored.

1.3.3 Birth Defects

Birth defects are one of the leading causes of infant mortality in North America and are one of the top 10 causes of potential years of life lost. Nearly 1 out of every 28 US babies is born with a birth defect (March of Dimes 2002). For most birth defects, the cause or causes are unknown but are most likely to be due to gene-environment and gene-gene interactions. Improved monitoring of birth defects may help provide some answers.

Birth defects, congenital anomalies, and congenital malformations are terms used to describe an abnormality of structure, function or metabolism that is present at birth (even if not diagnosed until later in life). It has been estimated that around 20 percent of all birth defects are due to gene mutations, 5–10 percent to chromosomal abnormalities, and another 5–10 percent to exposure to a known teratogenic agent or maternal factor (Beckman and Brent 1984, Nelson and Holmes 1989). Together, these percentages account for approximately 30–40 percent, leaving the etiology of more than half of birth defects unexplained (Bishop *et al.* 1997). A teratogen is a factor that has an adverse effect on an embryo or a fetus between fertilization and birth (Health Canada 2002a). Examples of infectious agents that can be transmitted to the fetus and have an adverse effect include rubella, cytomegalovirus, varicella and toxoplasma. A number of drugs have clearly been shown to be teratogenic. The most commonly used teratogenic agent is alcohol. Fetal alcohol syndrome (FAS) has been recognized as one of the leading causes of preventable birth defects and developmental delay in children. Maternal age is a risk factor for congenital anomalies, specifically chromosome problems (Health Canada 2002a).

Major birth defects are detected in two to three percent of births every year. The total prevalence of birth defects has been stable over recent years. Today the most prevalent categories of major birth defects in Canada are musculoskeletal anomalies, congenital heart defects and central nervous system anomalies, such as neural tube defects (NTDs) (Health Canada 2003).

One type of birth defect that has been of particular concern in North America is neural tube defect, which includes anencephaly and spina bifida. The rates of anencephaly (where part or all of the brain is missing) vary among the three countries, with the highest rates in the United States, at 6 per 10,000 births, compared to Mexico, 5 per 10,000 and Canada, 2.4 per 10,000 (National Birth Defects Prevention Network 2000, INEGI 1999, Rouleau *et al.* 1995). These statistics are from national sources, and there are differences in collecting and reporting methods among the countries. Consequently, these must be interpreted with caution. However, such geographic variation may indicate a role for non-genetic factors such as diet (folic acid, in the case of anencephaly) and environmental exposures. In Canada and the United States, the prevalence of neural tube defects has declined over the past decade—due in part to increased intake of folic acid from fortified foods and use of vitamin supplements—but the number is still a concern.

A common birth defect in the United States is hypospadias (an abnormal formation of the penis in which the opening of the urethra does not emerge at the tip of the penis, but rather, lower down on the penis). Approximately 1 in every 125 US boys has hypospadias (Baskin *et al.* 2001). Reported rates of defects of the male reproductive system, such as undescended testicles and hypospadias, have doubled in the United States from 1970 to 1993 (Paulozzi *et al.* 1997). Some researchers have hypothesized that these birth defects are associated with exposure to persistent organic chemicals; however, there are other trends over time (such as improved case diagnosis and reporting and changes in diet) that could be involved as well (Skakkebæk *et al.* 2001).

1.3.4 Endocrine Toxicity

While the link between chemicals and cancer has been explored for many decades, only recently has more attention been focused on a wider range of subtle, non-cancer effects. Some chemicals are thought to alter and interfere with hormonal activity, causing significant health and developmental impacts. These chemicals are known as endocrine disrupters or hormonally active chemicals. Endocrine disruptors can interfere with the body's normal hormonal functioning by binding to receptors, blocking them, or

interfering with proteins which regulate the production, transport, metabolism and activity of hormones (Goldman and Koduru 2000). Endocrine disrupters can work at low doses, cause effects in the next generation, and might act only during critical windows of vulnerability (Melnick *et al.* 2002). Because of the various modes of action that these compounds work through, endocrine disruption has challenged traditional toxicity and health research.

Chemicals such as PCBs, pentachlorophenol, DDT, bisphenol A, and dioxins and furans have been found to have endocrine-disrupting properties in wildlife, laboratory animals and experiments on cells. In wildlife, increased mortality, altered sex ratios, thinning eggshells, and reduced immune and reproductive function have been linked to persistent organochlorine contaminants (Vos *et al.* 2000, Guilette and Gunderson 2001).

Based on animal toxicity studies, it has been hypothesized that endocrine disruptors may be associated with a variety of human health effects, including endometriosis, breast cancer, thyroid cancer, early onset of female puberty, infertility, testicular cancer, and abnormalities of the male reproductive organs such as hypospadias, undescended testicles, and reduced sperm counts (Foster 1998). However, it is difficult to extrapolate from studies in wildlife and the laboratory to human health endpoints, and this issue is quite controversial; currently there is much debate over human risks that might be associated with low-level exposures, for example with bisphenol A.

Four reports have suggested that the altered sex ratio (with fewer boys being born, compared to girls) observed in many countries could be a result of endocrine-disrupting chemicals acting at specific times of development (Figa-Talamanca *et al.* 2003, Mackenzie *et al.* 2005, Ryan *et al.* 2002, Schnorr *et al.* 2001). However, such a change was not observed in babies born after significant PCB exposures in Taiwan (Rogan *et al.* 1999, Yoshimura *et al.* 2001). At this time, it is uncertain whether or not sex ratio in humans can be affected by such exposures (Rogan and Ragan 2003).

An especially important—but largely missing—piece of the puzzle has to do with events prior to and around puberty. Timing of breast development (thelarche) in girls and onset of puberty in boys and girls is of particular concern, especially given the long-term trend of earlier puberty in girls that has been documented (Parent *et al.* 2003). There is evidence that pubertal developmental aspects are altered with exposure to lead (Selevan *et al.* 2003) and inconsistent evidence of effects of PCBs in humans (Denham *et al.* 2005, Gladen *et al.* 2000, Mol *et al.* 2002). However, there is also evidence that other trends, such as nutrition and obesity, may also play a role (Parent *et al.* 2003).

The potential for thyroid hormone disruption by a number of chemicals also has been noted and in toxicology studies dozens of chemicals have been identified that, at various doses, have the potential to affect thyroid hormone status (Howdeshell 2002). This is important because of the sensitivity of the developing brain to maternal thyroid hormone status (ACOG 2002). It has been hypothesized that some chemicals may have negative impacts on brain development via such a mode of action (Howdeshell 2002). In human populations there is some evidence that those who are more highly exposed to PCBs and dioxins have relatively lower thyroid hormone status (within the "normal" range), supporting this hypothesis (Kimbrough and Krouskas 2001, Porterfield 2000). At this time, much research is underway to explore this issue (Jahnke *et al.* 2004).

A recent global review of endocrine disruptors by the International Program on Chemical Safety, sponsored by the World Health Organization (WHO), the United Nations Environment Programme (UNEP) and the International Labor Organization (ILO), concluded that "the evidence that wildlife have been adversely affected by exposures to [endocrine disruptors] is extensive." The current evidence that human health has been adversely affected by exposure to endocrine disruptors was characterized as "generally weak." The report noted large gaps in knowledge, suggested that "concerns remain," and stated that there is an "urgent need" for studies in vulnerable populations such as infants and children (IPCS 2002).

1.3.5 Asthma and Other Respiratory Effects

The developing lung is a potential target for environmental contaminants. While children's bodies are growing, lungs are growing as well. Two recent studies in southern California found that children with higher exposures to air pollution (particles, nitrogen oxides and inorganic acids) have reduced lung growth (Gauderman *et al.* 2000, Gauderman *et al.* 2004). They also found that maternal smoking during pregnancy and environmental tobacco smoke in the home are associated with reduced lung growth (Gilliland *et al.* 2000).

Asthma is one of the diseases that have increased significantly in North America over the last 25 years. Reported prevalences of asthma are higher in the United States and Canada than in Mexico. This translates into millions of children in North America with asthma—approximately five million children in the United States alone.

Because the lungs of children are growing rapidly, there is also a concern for risk of exposure to carcinogens during childhood. That is because the process of cancer formation involves many steps, including mutations or other changes in DNA and cell division. Recent data on lung cancer patients would indicate this is more than a theoretical concern. Researchers showed that people who had started smoking before the age of 15 had twice the amount of DNA damage as those who started smoking after the age of 20 (given an equivalent lifetime exposure to tobacco smoke) (Wiencke *et al.* 1999). Also, the lifetime risk of lung cancer is very strongly increased by length of time since smoking initiation; given the overlap of carcinogens in mainstream tobacco smoke, environmental tobacco smoke and outdoor urban particulate air pollution, childhood exposure to the latter types of air pollution may substantially increase lifetime cancer risk independent of smoking.

Asthma is a disease of chronic airway inflammation and hyper-responsiveness to environmental triggers. Some of these triggers include mites, dander from pets, fungal spores, environmental tobacco smoke (i.e., second-hand smoke), viral infections and air pollution. Asthma is one of the diseases that have increased significantly in North America over the last 25 years. Reported prevalences of asthma are higher in the United States and Canada (up to 17 percent of the population suffers from it) than in Mexico (six percent) (ISAAC 1998, Public Health Agency of Canada 1999). This translates into millions of children in North America with asthma—approximately five million children in the United States alone (Mannino *et al.* 2002). Approximately 12 percent of Canadian children are asthmatic and 29,000 children are hospitalized each year due to asthma (Environment Canada 2002). United States asthma prevalence rates increased 74 percent from 1980 to 1995. The number of US children dying from asthma increased 2.5-fold from 1979 to 1996 (Wargo and Wargo 2002), supporting the notion that prevalence was increasing during the same time period (although in the United States asthma has not been a significant cause of childhood mortality during this time). In Mexico, asthma is reported to have been responsible for nearly 8 percent of childhood emergency room visits in one major pediatric hospital.

Pollutants such as ozone, particulates, sulfates and nitrogen oxides may aggravate asthma symptoms, resulting in a range of effects, from wheezing, to staying home from school, to visiting the doctor or an emergency room. Asthmatic children are more likely to visit emergency rooms as the levels of such air pollutants as ozone and particulates increase (Institute of Medicine 1999a). This disease is one of the leading causes of absenteeism; for instance, in Canada, asthma is responsible for 25 percent of all school absences (Environment Canada 2002). In Mexico, higher ozone levels (180–270 ppm) have been associated with absence from preschool due to respiratory illness (Romieu 1992).

Whether air pollution causes new cases of asthma is less certain. Some studies do provide support for the notion that air pollution doesn't just make asthma worse, but is actually associated with asthma causation. Children, in southern Californian communities with high ozone (smog) levels, who play three or more outdoor sports, are three times more likely to have asthma than children in such areas that do not play sports. Sports were not associated with asthma onset in low ozone areas (McConnell *et al.* 2002).

2 Types, Exposures and Potential Health Impacts of Chemicals

2.1 Types of Chemicals

Chemicals can be classified by their properties and uses.

2.1.1 Chemical Properties

Chemicals have specific physico-chemical properties such as molecular size, solubility and half-life that can determine their persistence in the environment, and their potential for accumulation in biological systems, including humans. Chemicals also exhibit potentially harmful characteristics, such as the ability to ignite, explode, corrode, etc. Toxicity, the potential to harm people, plants and animals, is another characteristic of a chemical. It is the chemical's inherent ability to cause a specific toxic effect. Any substance, even water, may cause toxic effects if ingested or inhaled in excessive quantities. Therefore, assessment of the risk posed by a substance involves consideration of dose as well as toxicity.

Persistence refers to the length of time a substance stays in the environment without breaking down to other chemicals. A substance may persist for less than a second, or indefinitely. So-called persistent chemicals remain in the environment for longer periods of time than non-persistent chemicals, usually weeks or years. Persistence, *per se*, is not a negative characteristic. It poses a problem only if it is coupled with toxicity.

Many chemicals are persistent in water, especially groundwater. Fewer chemicals persist in air. Sunlight, as well as oxygen and other constituents of outdoor air, can cause some chemicals to break down.

Metals, as well as certain inorganic and organic chemicals, are able to persist in air for long periods of time and thus can travel long distances from their source. This is why certain metals and organic chemicals are found in remote locations, such as the Arctic and Antarctic, far away from the chemical source areas where such substances have been manufactured and used.

Degradation in the environment is an important physico-chemical process that breaks down chemicals to other substances. Photo-degradation by sunlight, biodegradation by bacteria, and oxidation by oxygen can cause some less persistent chemicals to break down. The breakdown products can be more or less toxic, depending on the final product. Many organic products degrade to carbon dioxide and water.

Some chemicals have properties that make them bioaccumulative; that is, they accumulate in the tissues of living species. Chemicals that are bioaccumulative often show a pattern of higher and higher concentrations in tissues of organisms as one ascends the food chain from plants and plant-eating species to carnivores.

Chemicals with a combination of persistent, bioaccumulative, and toxic (PBT) properties are of particular concern because, once released to the environment, they can travel far from their source, remain in the environment for long periods of time, are toxic, and increase in concentration up the food chain. Some well-known PBT chemicals include dioxins and furans, lead, mercury, PCBs and hexachlorobenzene.

2.1.2 Chemical Uses

Generally, a distinction is made between chemicals on the basis of intended use and whether or not the production of the substance is deliberate. Types of chemical uses include the following:

- *Food additives* are substances in food that are added deliberately to change flavor, color, consistency or other attributes of food. In the US, these also include substances that are added inadvertently via migration of substances from packaging.
- *Pharmaceuticals* are chemicals that have medicinal properties and are marketed for health benefits. These include ingredients that are added for other useful properties such as for appropriate drug delivery and preservation.

- *Industrial chemicals* are chemicals developed or manufactured for use in industrial operations or research by industry, government or academia. They include metals, as well as polymers and organic chemicals. Most of the substances in the PRTRs are industrial chemicals. Many industrial chemicals also are used in consumer products, such as windshield washer fluids and household chemicals; such deliberate "releases" are not captured by pollutant registries.
- *Fuels* are used for the generation of energy, and include substances such as oil, natural gas, coal, but also include domestic and hazardous wastes used for energy recovery.
- *Manufacturing byproducts* and *breakdown products* are substances other than the principle product in a manufacturing process that are generated as a consequence of a manufacturing process. Byproducts of manufacture can be more toxic than the intended product of manufacture. For example, the highly toxic dioxin 2,3,7,8-TCDD was formed as a byproduct in the manufacture of the herbicide 2,4,5-T ("Agent Orange") and is considered the most toxic chemical in the dioxin family.
- *Combustion byproducts* are formed when chemicals are heated or burned. The most common combustion products of organic substances are carbon dioxide and water, but other more toxic substances may be formed, such as carbon monoxide. Minute quantities of dioxins and furans can be created during incineration, e.g., backyard burning, and even smaller amounts are produced in forest fires; these are not included in pollutant registries. The common pollutants that create smog and air pollution, such as ozone, nitrogen oxides, sulfur dioxides, and certain volatile organic compounds and particulates, are also formed in the burning of fossil fuels, known as combustion. Combustion can also contribute to the formation of greenhouse gases such as carbon dioxide and nitrous oxide.
- *Pesticides* are chemical substances or mixtures formulated for preventing, controlling, repelling, or mitigating any pest, including animals, plants and fungi. Categories of pesticides include insecticides for killing insects, herbicides for controlling weeds, fungicides for controlling fungi (e.g., on fresh produce), and rodenticides used to kill rodents such as rats and mice. Pesticides are frequently used in agriculture, industry, by municipalities, in institutions such as schools and hospitals, and in the home.

2.2 Chemical Sources

Chemicals are substances composed of one or more elements found in nature. All living and non-living systems are made up of chemicals from very simple to very complex structures. Chemicals are the building blocks of nature. Man-made chemicals can be found in nature, originating from numerous human activities such as agriculture, transportation, manufacturing, raw material extraction, waste disposal and treatment, and the use of many products, including pesticides, pharmaceuticals and consumer goods. Natural events, such as erosion and forest fires, can also release chemicals in the environment. On the most basic level, chemical uses are driven by societal forces such as the size (and wealth) of a population, the economy, technologies in use in the economy, and consumption patterns. These drivers encourage various kinds of industrial activities, which in turn become sources of chemicals in society. Sources of chemical emissions are many, including:

- Manufacturing plants;
- Electricity generating plants;
- Waste treatment, sewage and recycling plants;
- Small businesses, such as gas stations and dry cleaners;
- Mining, forestry, farming and fishing;
- Agricultural, home and institutional uses of pesticides;
- Vehicles, such as cars, trucks, buses and construction equipment; and
- Consumer products, such as toys, paints, solvents, household cleaners and building materials.

2.3 Chemical Exposures

The interrelationship between exposures to chemicals and health effects are rather complex and occur along a continuum, as shown in **Figure I-2.** Various monitoring tools are used to quantify the potential for exposure and for adverse outcomes at different points in this continuum. The following sections explore the possible routes of exposure through which a child can be exposed to various types of chemicals in his or her environment. First we discuss concepts of exposure assessment, including "routes of exposure"—how the child comes into contact with a chemical or mixture of chemicals—and then we look at "absorption and metabolism," i.e., the ways in which that substance moves within the body and affects its functioning.

2.3.1 Exposure Assessment

Exposure assessment is the determination or estimation of the magnitude, frequency, duration, and route of exposure to a chemical.[3] Routes of exposures are the ways in which chemicals can enter the body, which include inhalation, ingestion, skin absorption, or (rarely) injection. Examples of such media are:

- Air
- Water
- Food
- Land/soil
- Consumer products
- *In utero* (transplacental)
- Breast milk

Children eat more food and drink more water, per kilogram of body weight, than adults. Normal childhood behaviors result

3. **Hazard Assessment** is the process of determining whether exposure to a chemical or chemical mixture can cause an increase in the incidence of a particular adverse health effect and whether the adverse health effect is likely to occur in humans or environmental organisms. **Risk Assessment** is the determination of potential adverse health effects from exposure to chemicals, including both quantitative and qualitative expressions of risk.

Evolving Knowledge about How Pesticides Can Affect Children

Concern is growing over low-level, chronic exposures to pesticides which may interfere with immune, thyroid, respiratory and neurological processes in children (IPCS 1998) and may be linked to childhood cancers, endocrine disruption and developmental neurotoxicity in animals. Because they eat more fruits and vegetables per kilogram of body weight, and because their bodies are developing, children can be especially vulnerable to the health effects of pesticides (NRC 1993).

Insecticides have been of concern to children's health because they are often used in or around homes and on pets, and because they are often present as residues on the fruits and vegetables that children eat (NRC 1996). Three of the common groups of insecticides are **organophosphates**, such as chlorpyrifos (Dursban) and diazinon, **organochlorines**, such as DDT, and **pyrethroids**. Prenatal exposure to these chemicals may be of particular concern. For example, many years after DDT was banned in the United States, a study found that babies born in the early 1960s, whose mothers had higher levels of DDT during pregnancy, were more likely to have lower birthweight and to have been born preterm (Longnecker *et al.* 2001). Researchers in New York recently reported that babies born with higher levels of the insecticide chlorpyrifos in umbilical cord blood had smaller length and body weights. Babies born after 2001, when EPA phased down household and certain agricultural uses of chlorpyrifos, had lower blood levels of the pesticide and, at these lower levels, no association with fetal growth was noted (Whyatt *et al.* 2004).

PRTR data are a limited source of information about pesticides because:

- The US TRI requires reporting on a very limited number of pesticides, as does the Mexican RETC, but the Canadian NPRI requires none.
- Only manufacturers and blenders of pesticides are required to report to TRI. Farm, household or other institutional uses of pesticides are not reported (and probably could not feasibly be reported via the PRTR mechanism).
- Although PRTRs cannot provide a complete picture of pesticide impacts on communities, other types of reports could provide useful information. To date, no efforts are underway to establish such systems for North America but there are some efforts on national and subnational levels.

Pesticide Ingestions and Illnesses: Household pesticides are of concern because of their potential for accidental ingestion by children, especially curious toddlers. About 4 percent of all reported poisonings in Canadian children are the result of accidental pesticide exposure (Health Canada 1995). In the United States, the Toxic Exposure Surveillance System, a project of the American Poison Control Centers, reports that in the year 2003, about 50,938 children 0 to 6 years of age and 8,650 children 6 to 19 years old were cared for who had possibly been exposed to pesticides (there were many more additional telephone inquiries) (Watson *et al.* 2004). In the United States, pesticides account for 4 percent of all reported poison ingestions for younger children and 2 percent for older children. However, it is not clear how many of these ingestions caused toxic effects; in most cases, no medical treatment was required. There were two pesticide-related deaths among children six years of age or less (Watson et al. 2004). In Mexico, where pesticide poisonings are a reportable disease, children ages one to five have the highest rates of poisoning (1.5 cases per 10,000 people compared to 0.9 for infants of less than one year and 0.1 for older children 5 to 14 years old) (INEGI 1999). Pesticide poisoning is a reportable disease in certain states in the US (Calvert *et al.* 2004), but not nationally in the United States and not in Canada.

Pesticide Sales and Usage: The US EPA conducts a periodic survey of pesticide sales and use on a national basis. In 2001, there were nearly 5 billion pounds of pesticides used, of which about 1.2 billion pounds were agricultural and household pest control agents (Kiely *et al.* 2004). Usage patterns in the United States are significant on a North American basis because the US has such a large share of pesticide use globally—24 percent of agricultural and household pesticides—in relationship to population (Kiely *et al.* 2004) and because the US is a major exporter of pesticides to the rest of North America. Sales of pesticides increased by 50 percent from the mid-1960s through the mid-1990s and have leveled off since (Kiely *et al.* 2004). Increased sales have been seen particularly for house and garden uses, uses that are more likely to provide opportunities for direct exposure to children via improper storage of containers or pesticide spills in homes or lawns. Sales of pesticides also have increased in Mexico over time, from the 12,000 tonnes of pesticides sold within Mexico in 1960 to the 54,000 tonnes sold in 1986 (Ortega-Cesena *et al.* 1994). Pesticide imports into Mexico have also increased by 28 percent from 1999 to 2000 (*Subcomité de Comercio y Fomento Industrial* 2001). Unlike most OECD countries, Canada does not require reporting of pesticide sales data. This is changing now that Canada's recently-revised pesticide legislation is fully promulgated; however, for now, a reliable database on quantities of pesticides sold does not exist. Some of the states in the United States collect pesticide sales and usage reports (California, Massachusetts, New Hampshire, New York and Oregon). These state systems provide useful information about the types and quantities of pesticides used in particular areas, information that is important to communities (Kass *et al.* 2004), and also has been useful for research into pesticide health effects on children (Reynolds *et al.* 2005).

in greater intake of soil and dust (and any contaminants in those media). Toys or other products specifically manufactured for children are of particular concern; however, any product around the home may be ingested by children.

Improved scientific understanding and experience has brought a focus on children's health, and a growing awareness of the vulnerabilities of children *in utero*. Chemical exposures at this time can have significant, life-long and irreversible effects, depending on the timing of the exposure and developmental window. For example, pregnant women eating fish contaminated with methylmercury can pass along the chemical to the fetus through the placenta, and at certain levels this could result in decreased IQ. Although the strength of evidence for this has been confirmed by the US National Research Council (NRC 2000, Jacobson 2001), such effects have not been observed in all populations (Davidson *et al.* 2001).

Breastfeeding, which we know provides optimal nutrition to infants, can also be a significant pathway for children's exposure to some chemicals (Rogan 1996). Contaminants such as organochlorine pesticides, PCBs, dioxins, perchlorate, PBDEs and solvents may be present in breast milk. Some studies show that increased concentrations of contaminants in breast milk can increase the risk of infant infections (DeWailly *et al.* 2000, 2001). Infants, during breastfeeding, can be exposed to higher daily intake concentrations of some persistent organic pollutants per unit body weight than at any other time in their lives (Patandin *et al.* 1999a). The known neurotoxicity of PCBs and recent reports of rapidly increasing levels of PBDEs in human breast milk raise the possibility of preventable harm to current and future generations of breastfed children. However, breastfeeding confers numerous important nutritional and immunological advantages to the developing infant. One study showed that the benefits of breastfeeding outweighed the risks of the increased exposure to persistent toxic chemicals in the breast milk (Jacobson and Jacobson 2003). It must be emphasized strongly that breastfeeding is recommended as the optimum method of nourishing babies, as the benefits of breast milk have been judged to outweigh the risks from exposure to contaminants contained in it for most people (Brouwer *et al.* 1998)

2.3.2 Absorption and Metabolism

After exposures, chemicals are absorbed and metabolized in the human body. Levels of chemicals in the tissues can be measured by biomonitoring.[4] Chemicals are measured in various ways but most usually are monitored in blood or urine samples. Sometimes a breakdown product (or metabolite) is monitored when it is not possible to measure reliably the parent compound. At other times, a biochemical change is monitored as a surrogate for exposure.

For a health effect to occur, not only is an exposure required but the route of exposure is important because the dose must reach the target organ. Inhalation, ingestion, and dermal routes are important in this regard because: (1) the route may lead the substance directly to the target organ, such as the lungs (for example, the direct contact of air pollutants with lung tissue, the skin, and the gastrointestinal tract); and (2) the route may bypass the body's defense mechanisms (for example, chemicals not eaten in food are not passed through the liver and therefore may not be detoxified before being circulated through the rest of the body). Once there is exposure of the target organ to a chemical, in sufficient quantity, there can be a spectrum of effects ranging from biochemical alterations to disease, disability and death.

2.4 Potential Health Impacts

Tracking diseases in North America can be rather difficult and, within each country, there are numerous federal, state, and municipal regulatory agencies that oversee public health. Unfortunately, methods for reporting diseases are not uniform across North America. Although pieces of information can be drawn from national surveys in each country, this lack of a standardized reporting framework across the continent is one of the significant barriers to understanding the links between childhood diseases and their underlying causes (Goldman *et al.* 1999).

Individual differences in vulnerability also make assessment of health impacts difficult because the genetic make-up of some individuals can render them more sensitive to contaminants than others.

Furthermore, the type, nature and severity of a health effect may vary not only with the dose but also with the timing of a chemical exposure and the sex of the offspring. We know, for instance, that pregnant rats fed one meal containing dioxins on the critical fifteenth day of gestation produced male offspring with reproductive tract birth defects (Gray and Ostby 1995) and female offspring with persistently abnormal mammary gland development (Fenton *et al.* 2002).

Mixtures of chemicals can have different health and environmental effects from the effects of individual chemicals. Some mixtures can have effects that are greater than an individual chemical effect. In one study, a PCB compound (PCB153) given alone did not result in liver damage in rats, but when given with dioxin as a mixture produced 400 times the effect of the dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin [TCDD]) alone (van Birgelen *et al.* 1996). Alternatively, chemical mixtures can have competing effects, reducing the total chemical effects.

This observation of differing health effects of chemical mixtures poses real difficulties for toxicity testing and regulatory efforts, which often rely on chemical-by-chemical testing. This approach does not reflect the reality for children, who are exposed to a mixture of chemicals throughout their day. Our understanding of the effects of long-term, multiple, simultaneous, multi-generational exposures to low-level chemicals is just beginning. Creating a testing, standard-setting and regulatory framework that reflects "real life" exposures is one of our

4. Biomonitoring includes the assessment of human exposure to chemicals by measuring the chemicals or their metabolites (breakdown products) in human tissues such as blood or urine. Blood and urine levels reflect the amount of the chemicals in the environment that actually get into the body.

next great challenges (Bucher and Lucier 1998), recognizing of course, that it will not be possible to test all the permutations and combinations of all chemicals.

In the past, regulations have sought to identify a "threshold" below which a chemical does not cause health effects. For some chemicals and in fact for many effects, such thresholds seem to exist. However, for some health endpoints and chemicals, no such level does appear to exist. For example, on a theoretical basis, for genotoxic carcinogens, each decrement of exposure down to zero conveys some level of health risk. In these circumstances, most nations have adopted models that allow the identification of some very low level of risk such that there is a reasonable certainty that no one will be harmed by exposures to such chemicals since, in practice, "zero" exposure levels may be difficult if not impossible to achieve.

2.5 The Universe of Chemicals—What We Know and What We Don't Know

There are millions of chemicals that are known to exist in the world and some 100,000 chemicals that have been synthesized in large enough quantities to be registered in North America, Europe, or by other OECD countries (US EPA 1998). New chemicals are discovered every day, but few have commercial potential or are produced in significant enough quantities to warrant concern about exposures (outside the research laboratory) or to require notice to regulatory authorities.

In both Canada and the United States, there are procedures for assessing "new" chemicals (those that are not already listed on Canada's Domestic Substances List [23,000 chemicals] or the US Toxic Substances Control Act Chemical Substance Inventory [82,000 chemicals]). This amounts to 800 new chemical notifications per year in Canada and 1,500 in the United States. Guidelines describe the types of information to be submitted for assessment. (For more information on the US and Canada programs, see ‹www.ec.gc.ca/substances/nsb/eng/sub_e.htm› and ‹www.epa.gov/opptintr/newchems/›.[5]) Mexico does not have a consolidated list of "existing" chemicals but does have a catalog of pesticides that have been evaluated and allowed for import and distribution in the country (Cofepris 2005). The Ministry of Health (*Secretaría de Salud*) uses a number of lists to determine if a chemical is "new." An application must then be made to Mexican authorities before the new chemical can be manufactured or used. (See **Appendix E** for additional information regarding each country's regulatory programs.)

Screening and basic toxicity information is lacking on many existing chemicals. A 1998 EPA review found that no basic toxicity testing was publicly available for 43 percent of chemicals considered to be produced or imported in high volumes (one million pounds or 454,000 kilograms or greater annually) and that only seven percent of such chemicals had been evaluated for a full set of basic data for six end points (US EPA 1998), that is, for acute toxicity, repeated dose toxicity, developmental and reproductive toxicity, mutagenicity, ecotoxicity and environmental fate.

The OECD developed the Screening Information Data Set (SIDS) to provide an internationally agreed-upon set of test data for screening high production volume chemicals for human and environmental hazards. The SIDS data include: physicochemical properties (melting point, boiling point, vapor pressure, water solubility, and octanol/water partition coefficient), environmental fate (biodegradation, hydrolysis, and estimates of distribution/transport and photodegradation), ecotoxicity (acute toxicity to aquatic vertebrates, invertebrates, and plants), and studies in animals to assess human health effects (acute and repeat-dose toxicity, effects on the gene and chromosom, effects on reproduction and developmental effects).

Of the 830 companies making high production volume (HPV) chemicals, 148 had no test results available on their chemicals. The basic set of tests for one chemical costs about US$200,000 and can increase significantly when additional tests are required. Over the last five years, steps have been taken to fill these testing gaps through the voluntary High Production Volume Challenge Program in the US and the OECD HPV program (see text box), with the commitment to make data available for all HPV Challenge–sponsored chemicals in 2005. It is important to note in this context that the OECD HPV process includes only screening level toxicity analyses and not more comprehensive tests of developmental and reproductive toxicity, which are much more expensive.

In Canada, the Canadian Environmental Protection Act, 1999, requires the 23,000 existing chemicals on the Domestic Substances List to be categorized by 2006 and, if necessary, screened to determine whether they are toxic or capable of becoming toxic. The chemicals are categorized by persistence, ability to bioaccumulate, inherent toxicity to the environment and to humans and/or whether they have a high potential for exposure to Canadians. Screening assessments are being developed for these chemicals. Screening assessments recommend one of three outcomes:

- No further action is required on the chemical.
- The chemical should be placed on the Priority Substance List for further assessment.
- The chemical is toxic and should be placed on Schedule 1 for regulatory or other action. (For more information, see ‹www.ec.gc.ca/substances› and ‹http://www.hc-sc.gc.ca/hecs-sesc/exsd/›.)

2.6 Understanding Chemicals' Potential Risks to Children

Some of the HPV chemicals may be of particular concern to children's health. A set of 23 chemicals has been found in human tissue or the environment and identified by the US EPA for additional testing. Under the Voluntary Children's Chemical Evaluation Program, started in late 2000, 35 companies and 10 consortia have agreed to support additional testing for 20 chemicals. Companies

5. See also Chapter 4 below for more information on government regulatory programs in the North American countries and recommendations concerning them.

High Time to Focus on High Production Volume Chemicals (HPV)

Approximately 2,800 chemicals are known in the United States as high production volume (HPV) chemicals. These are substances that are produced in the United States and/or imported in volumes—at over 1 million pounds or greater (454,000 kg) per year. Pesticides, food additives, drugs, polymers and inorganic chemicals (such as lead, mercury, cadmium) are not included on the HPV lists produced by the US EPA or the list of over 4,000 HPV chemicals compiled by the Organization for Economic Cooperation and Development (OECD), using a somewhat different definition of "HPV."

Following the 1998 US EPA review indicating the lack of basic testing data for 93 percent of HPV chemicals, EPA issued the HPV Challenge Program. The goal of this program is to ensure that a baseline set of health and environmental data on the HPV chemicals is made available to the EPA and the public by 2005. Over 430 companies, some working through 155 consortia, have publicly committed themselves to sponsor HPV chemicals. Companies (or consortia of companies) volunteer to assess the current information on a particular chemical, conduct new testing as required and make the existing and new tests available to the public; this is called "sponsorship" of a chemical.

Companies have submitted plans for new testing of the HPV chemicals, and also summaries of existing information. These plans and summaries are available for public review at EPA's Chemical Right-to-Know web site at **‹www.epa.gov/chemrtk›**. According to Environmental Defense, an NGO partner with EPA and the chemical industry in the testing program that tracks these numbers, as of June 2004, 1,916 of the original 2,782 chemicals that needed additional testing had been sponsored by chemical companies, 532 (19 percent) were not sponsored, of which perhaps 50 percent are no longer in high production. Meanwhile, in the US EPA's 2002 chemical update, industry reported a total of 735 "new" HPVs in production. EPA and the chemical industry have not required that these be included in the voluntary program and only 112 of these have been sponsored (Denison 2004).

Two other similar HPV programs are also in progress: one testing approximately 4,000 chemicals identified through the OECD HPV Screening Information Data Sets program (SIDS) and the other developed by the International Council of Chemical Associations, testing approximately 1,000 high priority chemicals.

The end result? More publicly available baseline testing data on HPV chemicals. While still providing only the basic set of data (and not more detailed information about developmental effects), this will nonetheless significantly help our understanding of these HPV chemicals and their potential health and environmental effects.

will collect and develop, if need be, health effects and exposure information on their sponsored chemical and integrate this information into a risk assessment. Additional data needed to fully characterize the risks to children would also be identified.

The health effects information requested in the Voluntary Children's Chemical Evaluation Program is a subset of the test battery developed by the EPA to assess the impacts of pesticides on children's health, and is designed to assess some of the unique vulnerabilities and exposures that children may face (e.g., prenatal developmental toxicity, neurotoxicity, screening battery, and developmental neurotoxicity). Some of the chemicals included in this program are benzene, toluene, xylenes, and trichloroethylene. For more information, please see ‹http://www.epa.gov/chemrtk/vccep/index.htm›.

These initiatives in North America complement information developed globally under an international agency program for chemical testing organized by the OECD. Most of the data collected under the North American programs are available on the Internet, allowing for increased sharing of results among countries.

3 Releases of Chemicals: Data from Industrial Pollutant Release and Transfer Registers

3.1 Overview

Pollutant release and transfer registers (PRTRs) are innovative tools that can be used for a variety of purposes. They report on certain chemicals and thereby can help industry, government and citizens identify ways to prevent pollution, reduce waste generation, decrease releases and transfers and increase responsibility for chemical use.

As with any tool, however, an important consideration in making good use of PRTR data is to understand their limitations. For some toxics, such as benzene, mobile sources like automobiles may be the chief source of releases to the environment; PRTR data do not capture releases from mobile sources. For others, such as carbon tetrachloride, industrial sources are the main source, so PRTR data would provide a more complete picture of sources. For toxics such as mercury, for which the main route of exposure to people is through the food supply, PRTRs may capture sources and releases but will not provide information about exposures through consumption of contaminated fish and other foods. Likewise, since PRTRs were designed to report on industrial releases and transfers only, they do not contain data about "downstream" uses and exposure to a product. For example, a PRTR database would not contain information about exposure to benzene by workers (and consumers) who inhale vapors while pumping gas. Thus, while PRTR data are useful, they provide only a partial picture of chemicals in the environment and the potential for exposure.

It is also important to emphasize that the release of a substance from an industrial source does not automatically lead to human exposure. Moreover, the degree of human exposure is not necessarily proportionate to the number of tonnes released. There are many factors to consider in determining human exposure to *individual* environmental toxicants, including: the route of exposure; the duration and frequency of the exposure; the rate of uptake of the substance; individual age, gender, and ethnicity; and the disease, overall health, nutritional and pregnancy status of the individual. When it comes to examining human exposure to groups of environmental contaminants, the degree of human exposure cannot be aggregated in a corresponding manner to the aggregation in tonnage of industrial releases of a *group* of environmental toxicants (e.g., carcinogens). This is because, for example, a specific amount of one carcinogen does not necessarily have the same toxicity as the same amount of another carcinogen, meaning that the risks to human health could be considerably different.

In summary, PRTR data constitute only one part of the pollution "picture;" they do not necessarily include:

- all potentially harmful chemicals—just those on the mandatory lists of chemicals which must be reported;
- chemicals released from mobile sources such as cars and trucks;
- chemicals released from natural sources such as forest fires and erosion;
- chemicals released from small sources such as dry cleaners and gas stations;
- chemicals released from small manufacturing facilities with fewer than 10 employees;
- information on the toxicity or potential health effects of chemicals;
- information on risks from chemicals released or transferred; or
- information on exposures to humans or the environment from chemicals used, released or transferred.

PRTR data are just one source of information on toxic chemicals in the environment. Other sources include databases containing measurements of concentrations of chemicals in the air, land and water in the environment, inventories of chemicals such as specialized chemical and air pollutant inventories, hazardous waste databases, modeling estimates, actual levels (also

known as "body burden") in plants, fish and people, and industrial emission rates of chemicals.

3.1.1 North American PRTRs

Each country in North America collects information on chemical releases and transfers. Now coming up to its nineteenth year in operation, the Toxics Release Inventory (TRI) in the United States currently collects information on the releases and transfers of over 650 chemicals from over 24,000 facilities. For more information on the TRI program, please see ‹www.epa.gov/tri›.

In Canada, the National Pollutant Release Inventory (NPRI) collected its first information on pollutant releases and transfers for 1993. For the 2002 reporting year, the latest considered in this report, over 4000 facilities reported their releases and transfers of 273 substances. Fifty-eight of these chemicals have been declared toxic under the Canadian Environmental Protection Act of 1999. More information on the NPRI and a Citizen's Guide to NPRI can be viewed at Environment Canada's web site at ‹www.ec.gc.ca/pdb›.

The passage of legislation in 2001 provided Mexico with the enabling authority to put in place a system of mandatory reporting under its PRTR, the *Registro de Emisiones y Transferencia de Contaminantes* (RETC). Currently, approximately 300 industrial facilities under federal jurisdiction voluntarily report their annual releases and transfers of 104 chemicals. Work is underway to implement the mandatory reporting scheme. Information has been available by sector and by region only. For more information on Mexico's RETC program, see ‹http://www.semarnat.gob.mx/qroo/transparencia/retc.shtml›.

Each country has set up its PRTR to reflect local conditions, laws and objectives. Fortunately, a common set of elements allows much of the information collected in the Canadian NPRI and the US TRI to be matched. Comparable data are not yet available from the Mexican RETC.

The CEC, through its annual *Taking Stock* report, provides a North American perspective on the amounts of chemicals released to the air, land, water, and transferred off-site. The CEC takes the chemicals and elements common to both the NPRI and TRI data and produces a matched Canada/United States data set. Data from the mandatory RETC in Mexico will be included in future reports as they become available. This report complements the *Taking Stock* series by presenting the matched Canadian and US data sets from a children's health perspective.

3.2 PRTR Analysis

PRTR data are useful for identifying sectors and facilities that are releasing and transferring chemicals into the environment. Users can search these databases by chemical name, geographic coordinates or by industry sector to find out about the sources of particular chemicals of interest. Many of the PRTR chemicals are of particular concern for children's health because they have been linked to cancer, developmental and reproductive toxicity, or neurotoxicity. Some of these chemicals, such as lead, mercury and dioxins, have been identified in numerous reports as being of special concern to children's health. PRTR data also can reveal trends in releases and transfers of chemicals. This information can be used to help tailor programs and actions to reduce chemical releases and encourage pollution prevention, thereby helping to reduce children's exposures to chemicals.

This report presents findings from two approaches to analyzing PRTR data:

- the **health effects approach**: analyzing PRTR data using lists of chemicals with similar health effects; and
- the **chemical-specific approach**: analyzing PRTR data for specific chemicals of concern to children's health.

Within the health effects approach, we have analyzed the data based on total quantities as well as by using toxicity weighting factors to take into account the differing toxicities of the listed substances.

3.2.1 Description of the Matched PRTR Data

This report is based on publicly available data on chemicals and industrial sectors common to both the Canadian National Pollutant Release Inventory and also the US Toxics Release Inventory. The report is therefore based on a subset of the larger NPRI and TRI data sets. It is important to realize that some sectors with significant releases, such as metal mining, some chemicals with large releases, such as ammonia, and some chemicals with

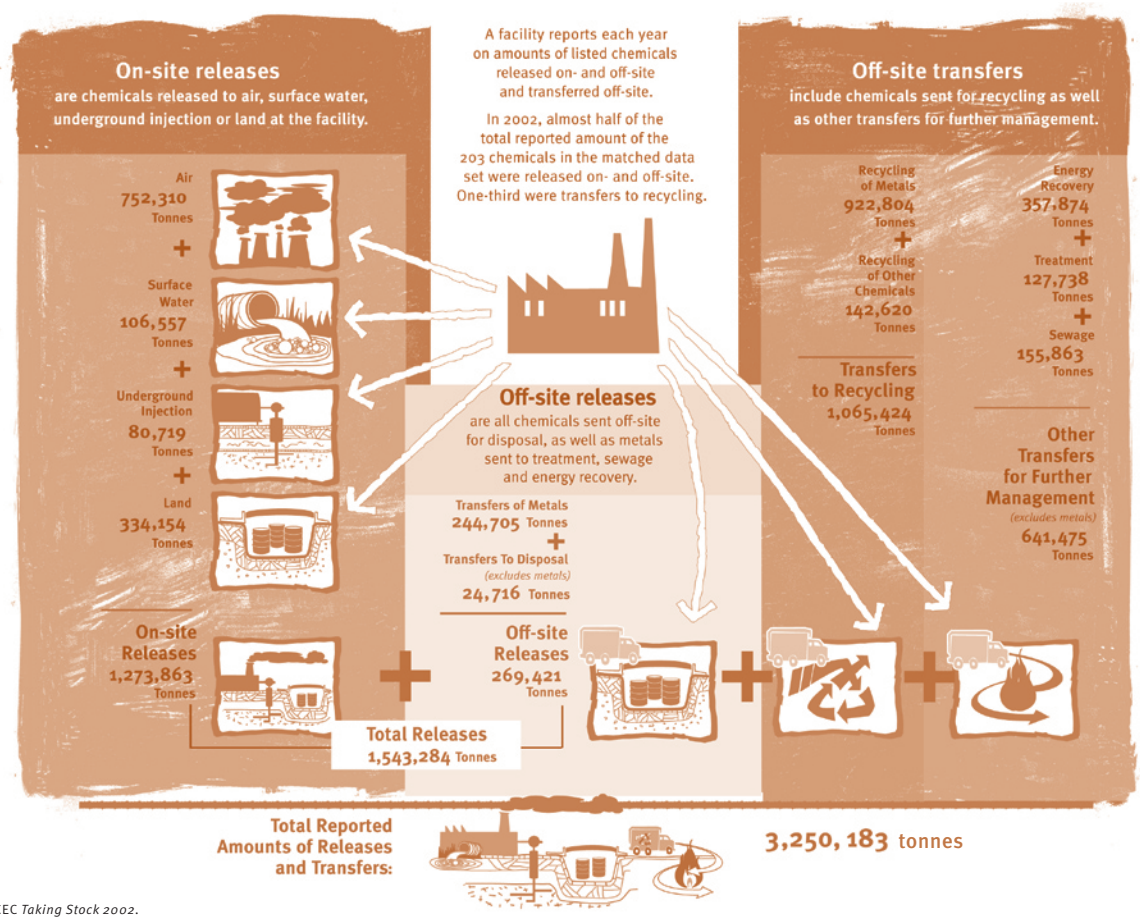
Taking Stock of Chemicals in North America

In Canada and the United States, factories, electric utilities, hazardous waste management/solvent recovery facilities and coal mines released and transferred over 3.25 million tonnes of chemicals in 2002. Over 179,000 tonnes of chemicals were released (on- and off-site) which are known to cause cancer, birth defects and other reproductive problems.

The five-year trend from 1998 to 2002 shows a decrease of 7 percent in the amount of chemicals released and transferred, as well as changes in how those pollutants are handled. The 18 percent reduction in chemicals released into the air was offset by a 4 percent increase in chemicals disposed in on-site landfills. Smaller reductions occurred in discharges to lakes, rivers and streams (a decrease of 8 percent) and in chemicals sent off-site for disposal in landfills (a decrease of 5 percent). There was a reduction in the release of chemicals which are known to cause cancer, birth defects and other reproductive problems. Total releases (on- and off-site) of these chemicals fell by 31 percent, compared to an 11 percent decrease for all chemicals.

The CEC's annual *Taking Stock* report and queries to the matched data set can be viewed at **‹www.cec.org/takingstock›**. *Taking Stock 2002* also presents data on many of the persistent, bioaccumulative, toxic chemicals (PBTs) such as dioxins/furans and hexachlorobenzene.

Figure 3-1 Releases and Transfers from Industrial Facilities in North America, 2002



Source: CEC *Taking Stock 2002*.

Note: Canada and US data only. Mexico data not available for 2002. Analyses are based on the matched set of chemicals and industry sectors for which comparable data are available for 2002. Total on-site releases are greater than the sum of the individual media because an NPRI facility can report only the total if it is less than one tonne.

environmentally significant releases, do not match between TRI and NPRI (because of differences in definitions or reporting requirements) and therefore are not part of this report.

In the future, data from Mexico may be available for inclusion in such an analysis. Currently, however, there are no comparable data from the Mexican RETC. The voluntary nature of the RETC program has resulted in relatively few reports being filed, and these reports are not publicly available by facility. The establishment in 2001 of a legal basis for mandatory PRTR reporting was a necessary prerequisite for the establishment of a system similar to NPRI and TRI.

The data used for the trend analysis are based on a set of chemicals and industries commonly reported in all years from 1998 to 2002. The year range of 1998–2002 was chosen so that several sectors that report large releases, such as utilities and hazardous waste/solvent recycling facilities, could be included in the trend analysis. These sectors started reporting to TRI in 1998. The primary chemicals of interest that are not included in the trend analyses include lead and mercury and their compounds. This is because the reporting thresholds for mercury and its compounds were lowered for the 2000 reporting year and the thresholds for lead and its compounds were lowered for the 2001 reporting year.

This report uses the following categories for presenting PRTR information. However, these summary classifications differ from those used by the separate countries' presentations of their data since each country collects data in somewhat different ways (see **Appendix C** for the details of how the reporting elements from each country are summarized). **Figure 3-1** presents these flows in a graphic manner:

- *Releases* are chemicals put into the air, water, land or injected underground.
- *On-site releases* are releases that occur at the site of the facility.
- *Off-site releases* are chemicals sent off-site to another location for disposal, as well as metals sent to treatment, sewage and energy recovery.
- *Total releases* are the sum of on-site releases and off-site releases.
- *Transfers* to recycling describe chemicals sent off-site for recycling.
- *Other transfers* for further management describes chemicals (other than metals) sent for treatment and energy recovery and to sewage plants.
- *Transfers for further management* represents the sum of chemicals sent for recycling and other transfers for further management.

- *Total reported amounts* is the sum of all above categories, i.e., total releases, recycling and other transfers for further management.

3.2.2 Methodology

Chemical lists

In this approach, four lists of chemicals with different health effects are used to analyze PRTR data:

1. Carcinogens
2. Recognized developmental and reproductive toxicants
3. Suspected developmental and reproductive toxicants
4. Suspected neurotoxicants

Chemical lists exist for other health effects such as respiratory toxicity, liver and kidney toxicity and endocrine toxicity. We chose these four lists based on the type of health effects of interest in children and the availability of matched data for PRTR chemicals.

For the purposes of this report, *carcinogens* refers to chemicals on the matched NPRI-TRI database that are recognized by the International Agency for Research on Cancer (IARC) to cause cancer in humans and/or animals and/or listed in the US National Toxicology Program (NTP 2004). There are various terms that are used for these. In the case of IARC these are called Groups 1, 2A and 2B carcinogens, depending on the degree of certainty for causing cancer (see ‹www.iarc.fr/›). In the case of the NTP, these are called chemicals that are "known" or "reasonably anticipated" to be human carcinogens (‹http://ntp-server.niehs.nih.gov/›).

For the sake of simplicity in this report, we refer to such chemicals as "carcinogens." Of the 203 chemicals in the matched TRI and NPRI 2002 data set, 55 have been determined by IARC and/or NTP to be known or suspected carcinogens on the basis of causing cancer to humans and/or animals and other scientific data. The chemical group chromium and its compounds is not included as a carcinogen in these analyses, despite the fact that one species—hexavalent chromium—is carcinogenic. Although hexavalent chromium is reported under NPRI separately from other chromium compounds, all chromium compounds are reported under TRI as a single amount.

Developmental and reproductive toxicants are those substances that can produce detrimental effects involving reduced fertility, and fetal and child developmental abnormalities. Some of these effects include structural abnormalities and other birth defects, low birth weight, growth retardation, fetal death, metabolic or biological dysfunction, as well as psychological and behavioral defects (Goldman and Koduru 2000).

The scientific determinations for whether chemicals are ***recognized*** **developmental and reproductive toxicants** were compiled by the State of California under Proposition 65. Of the more than 270 chemicals on the Proposition 65 list with such determinations, 21 chemicals with recognized developmental and reproductive toxicity matched the TRI and NPRI data and form the basis of the analyses in this report. Many of the chemicals listed on Proposition 65 are drugs, pesticides and different forms of PCBs or metals (e.g., arsenic trioxide). The PRTR database is restricted to chemicals manufactured or used in industrial operations and is, therefore, a shorter list. The full list of Proposition 65 chemicals is available at ‹http://www.oehha.ca.gov/prop65/prop_65_list/files/070904list.html›.

The scientific determinations for whether chemicals are ***suspected*** **developmental and reproductive toxicants** was compiled by a US nongovernmental group, Environmental Defense, using determinations that have been made by international agencies and the US government. This list, posted on their Scorecard web site as of July 2004, relies on various references, including determinations by the US EPA, by the State of California under Proposition 65, and by various other government and academic references. It identifies those chemicals with less weight of evidence that are considered suspected development and reproductive toxicants. Of the more than 300 chemicals with such determinations, 74 chemicals suspected to be associated with such developmental or reproductive effects matched the TRI and NPRI data and form the basis of the analyses in this report. The full Scorecard list of known and suspected developmental toxicants is available at ‹http://www.scorecard.org/health-effects/›, along with a full description of the methods that were used to compile this information.

It should be recognized that there are numerous limitations and uncertainties, which would be expected in the compilation of any such "list." Moreover, totaling chemicals by endpoint is probably more informative than lumping together all chemical releases. For example, it is recognized that all "carcinogens" do not cause cancer via the same mode of action and that it is therefore unlikely that the effects across all carcinogens would be additive. Most of these weaknesses derive from the limitations of the knowledge base that underlies these determinations, a knowledge base that needs to be strengthened with additional research.

Neurotoxicants are chemicals that alter the structure or functioning of the central and/or the peripheral nervous system. Symptoms of neurotoxicity include muscle weakness, loss of motor control, loss of sensation, tremors, and changes in cognition. Chemicals that are toxic to the central nervous system (the brain and spinal cord) such as mercury and lead can cause confusion, fatigue, irritability and behavioral changes. Chemicals that are toxic to the peripheral nervous system (all nerves except brain or spinal cord) can disrupt communication throughout the body (see ‹www.scorecard.org/health-effects/›).

Environmental Defense Scorecard also compiled a list of **suspected neurotoxicants**, as of July 2004, in consultation with government agencies. They found no recognized authoritative process for assessing neurotoxicants, so they were unable to compile a list of recognized neurotoxicants. In part this is because the term "neurotoxicant" covers a very wide range of

possible effects and possible dosages, ranging from substances which may only be able to cause very minor effects (e.g., nausea, dizziness), to major effects like lead-induced nervous system damage. Using government and academic sources, they were able to identify over 300 suspected neurotoxicants, of which 146 chemicals matched the TRI and NPRI data and so form the basis of the neurotoxicant analysis. The full Scorecard list of suspected neurotoxicants is available at ‹http://www.scorecard.org/health-effects/› along with a full description of the methods that were used to compile this information.

Because these types of toxicity are of particular concern for the health of children, this report addresses the following questions:

- *What quantities* of carcinogens/developmental and reproductive toxicants/neurotoxicants are released and transferred in Canada and the United States?
- *Which* carcinogens/developmental and reproductive toxicants/neurotoxicants are released and transferred in largest quantities?
- *Where* are the largest quantities of carcinogens/developmental and reproductive toxicants/neurotoxicants being released or transferred?
- Which industrial *sectors* are releasing the largest quantities of carcinogens/developmental and reproductive toxicants/neurotoxicants?
- Which *facilities* are releasing the largest quantities of carcinogens/developmental and reproductive toxicants/neurotoxicants?
- Have the quantities of carcinogens/developmental and reproductive toxicants/neurotoxicants released and transferred increased or decreased *over time*?

Appendix B provides a list of chemicals reported to both TRI and NPRI in 2002 that are considered carcinogens, recognized or suspected developmental and reproductive toxicants, or suspected neurotoxicants.

TRI facilities report separately for certain chemicals and their compounds, while in NPRI, a chemical and its compounds count as one category. For example, TRI lists both nickel and nickel compounds, counting them as two separate substances, while NPRI lists the single category, nickel and its compounds. Analyses of the PRTR data in this report add the TRI amount reported for the given chemical to the amount reported for its compounds, to correspond with NPRI practice.

3.2.3 Use of Toxicity Factors

One limitation of the PRTR approach is that quantities of chemical releases cannot tell us about risks to children unless we also have good information about exposure and toxicity. A toxic equivalency potential (TEP) approach has been developed by scientists at University of California, Berkeley, and reviewed by the EPA Science Advisory Board; this model takes into account relative toxicity as well as potential exposures through air and water (Hertwich *et al.* 1998). For known or suspected carcinogens, releases can be weighted by "benzene equivalents;" for noncancer effects, "toluene equivalents" are used. For most of the substances on the carcinogens list and the list of recognized developmental and reproductive toxicants, TEPs have been calculated and are shown in **Appendix B**. This method is further described in **Appendix D**. Even though the TEP approach is in an early stage, it does attempt to "weigh" the relative toxic potency of one substance against another by determining an equivalency relationship. It therefore may provide a better estimate of relative risk (and the relative importance of reductions) than an approach focused only on amount released.

This report provides an analysis of releases of these chemicals to air and water, applying the TEPs, in order to help provide an understanding of not only which chemicals have the highest releases but also how they compare in terms of toxicity. However, this analysis is limited, in the fact that a release does not directly correlate to actual exposures. As such, the findings of these analyses do not necessarily equate to levels of risk.

3.3 Findings from the PRTR Health Effects Approach

In this section, releases and transfers of carcinogens, recognized and suspected developmental and reproductive toxicants and suspected neurotoxicants are presented, based on the matched (TRI-NPRI) data set for 2002, with trends established from the 1998–2002 data set. These trends are based on the chemicals that were commonly reported over this time period. They do not include lead and its compounds because the reporting threshold was lowered during this time period.

More information on these releases and transfers from the matched data set can be found on the CEC *Taking Stock Online* web site at ‹www.cec.org/takingstock›. With its user-friendly "query builder," the web site enables users to generate their own reports on chemicals, sectors, facilities and trends of particular interest.

In addition to presenting the data on releases to air, water and land, we also present rankings based on releases to air and water that have been "weighted" for toxicity through the application of toxic equivalency potentials.

3.3.1 Releases and Transfers of Carcinogens

What quantities of carcinogens are released and transferred in Canada and the US?

In Canada and the United States, PRTR facilities released and transferred almost half a million tonnes (472,600 tonnes) of carcinogens of various types in 2002. Of these carcinogens, approximately 62,300 tonnes were released into the air; as much again were disposed of (mainly into landfills, including 39,000 tonnes on-site and 36,300 tonnes off-site); and 700 tonnes were released into water (about one hundred times less than air). More than half of the tonnage of carcinogens was reported transferred for the purpose of recycling. While prevention of pollution/waste at the source is the ideal, such recycling is preferable to releases to the environment and indicates that steps are being taken to

avoid such releases. However, the recycling facilities themselves also need to prevent environmental releases and occupational exposures that may result from the recycling activities (Landrigan *et al.* 1989) (**Table 3-1**).

Carcinogens make up approximately 15 percent of the total amount of chemicals released and transferred in Canada and the US (3.25 million tonnes). US (TRI) facilities were responsible for 87 percent of the total reported releases and transfers of carcinogens, while Canadian (NPRI) facilities accounted for 13 percent.

Which carcinogens are released and transferred in the largest quantities?

In 2002, the carcinogens released and transferred in the largest quantities were:

- Lead and its compounds (211,200 tonnes)
- Nickel and its compounds (82,900 tonnes)
- Styrene (33,100 tonnes)
- Dichloromethane (also known as methylene chloride) (27,900 tonnes)

The metals lead and nickel and their compounds were landfilled (on- and off-site) and recycled in large quantities; while not desirable, such modes of disposal are likely to minimize opportunities for exposure. In contrast, large amounts of styrene and dichloromethane were released into the air or sent off-site for further management, which includes use for energy recovery, treatment and transfers to sewage. Of note is that styrene was assessed in Canada under the first Priority Substances List and it was concluded that it is not CEPA–toxic to human health (see ‹http://www.hc-sc.gc.ca/hecs-sesc/exsd/pdf/styrene.pdf›). Other carcinogens that were released into the air in large amounts are formaldehyde, acetaldehyde, trichloroethylene and ethylbenzene (**Table 3-2**).

How do the quantities of carcinogens released to air and water compare in terms of toxicity?

Table 3-3 summarizes the data on total releases and then applies the toxic equivalency potentials (TEPs) for releases of carcinogens to the air and water. As shown, the relative ranking of the chemicals changes when TEPs are applied. When **amounts released to air** are weighted for toxicity using the TEPs:

- Carbon tetrachloride is ranked #18 for amounts of on-site air releases, whereas it ranked #1 in terms of tonnes of air releases when weighted by TEP.
- Lead and its compounds is ranked #11 for amounts of on-site air releases, while it ranked #2 based on tonnes of air releases when weighted by TEP.
- Styrene is ranked #1 for amounts of on-site air releases, whereas it ranked #23 when weighted by TEP, because of its relatively lower potency.

For releases to water under the TEP ranking:

- Lead and its compounds is ranked #4 for amounts released to water, while ranked #1 when weighted by TEP.
- Carbon tetrachloride is ranked #28 for amounts released to water, while ranked #2 when weighted by TEP.
- Formaldehyde is ranked #1 for amounts released to water, whereas it is ranked #19 when weighted by TEP.

Thus we find that in the case of carcinogens, the application of TEPs helps to focus attention not only on quantities of releases to the environment but also on the potential for toxicity. It can be seen that this analysis is limited by a number of missing TEPs for carcinogens, including two of the top ten air carcinogens (vinyl acetate and ethylbenzene) and two of the top ten water carcinogen releases (nickel and cobalt).

Where were the largest quantities of carcinogens released?

Five jurisdictions led Canada and the United States in total releases (on- and off-site) of known and suspected carcinogens in 2002 (**Table 3-4**):

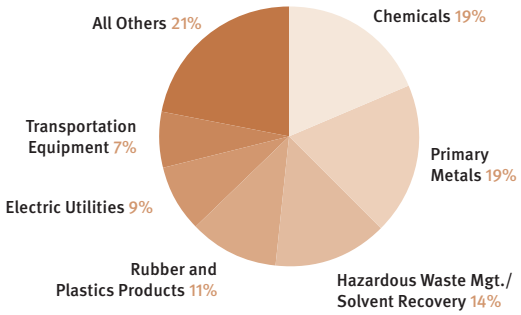
- Texas, with 16,900 tonnes
- Ohio, with 9,000 tonnes
- Indiana, with 9,000 tonnes
- Louisiana, with 8,700 tonnes
- Ontario, with 6,700 tonnes

Texas, Indiana and Ontario also ranked as the top three jurisdictions in North America for releases of carcinogens to air.

Which industrial sectors released the largest quantities of carcinogens?

Three sectors were responsible for over half of carcinogens released (on- and off-site) in Canada and the United States in 2002 (**Figure 3-2**):

Figure 3-2 Industrial Sectors with the Largest Releases (On- and Off-site) of Carcinogens Reported to North American PRTRs, 2002
(2002 Matched Chemicals and Industries)



Total Releases On- and Off-site: 153,274 tonnes

Note: Canada and US data only. Mexico data not available for 2002. Data include 55 chemicals common to both NPRI and TRI lists from selected industrial and other sources. A chemical is considered a carcinogen for the purposes of this report if it is so classified by the International Agency for Research on Cancer (IARC) **‹http://www.iarc.fr/›** or the US National Toxicology Program (NTP) **‹http://ntp-server.niehs.nih.gov /›**. Substances classified under IARC as carcinogenic to humans (1), probably carcinogenic to humans (2A), and possibly carcinogenic to humans (2B) are included. Under the US National Toxicology Program, substances classified as known to be carcinogenic (K) or may reasonably be anticipated to be carcinogenic (P) are included.

- Chemicals sector (includes chemical manufacturing and processing), with 28,800 tonnes
- Primary metals sector (includes steel mills, etc.), with 28,700 tonnes
- Hazardous waste management/solvent recovery, with 21,700 tonnes

Three sectors were responsible for well over half of the carcinogens released to the air in Canada and the United States in 2002 (**Figure 3-3**):

- Rubber and plastics products, accounting for more than one-quarter of the total carcinogens reported released to the air, with 16,200 tonnes
- Chemical manufacturing, with 10,500 tonnes
- Transportation equipment, with 9,400 tonnes

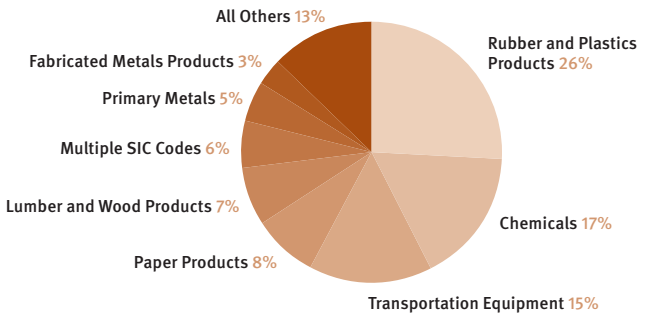
The facilities reporting the largest air releases in both the United States and Canada manufacture rubber and plastics products (US SIC code 30). Such facilities can release large quantities of carcinogens to the air, mainly the result of large emissions of one chemical, dichloromethane, also known as methylene chloride.

Have releases of carcinogens increased or decreased over time?

The quantity of known carcinogens released decreased 26 percent from 1998 to 2002. Over the same time period, releases of all the matched chemicals in the TRI-NPRI matched data set decreased by 11 percent. The decrease in carcinogens released to the air at the facility site was 30 percent (26,400 tonnes). Carcinogens disposed of mainly in landfills on-site decreased by 31 percent (6,900 tonnes) and off-site decreased by 37 percent (7,300 tonnes). Carcinogens discharged into surface waters decreased by 27 percent (235 tonnes). However, underground injection on-site increased by 31 percent (3,500 tonnes) (**Figure 3-4**). Although it can be argued that such disposal is safer than release to air and water, it is generally agreed that it is preferable to prevent pollution rather than control it. The data indicate, particularly for carcinogens, that there have been reductions in environmental releases and disposal from these sectors in Canada and the United States over this period beyond those for chemicals as a whole.

These trends are based on the chemicals that were commonly reported over this time period. They do not include lead and its compounds because the reporting threshold was lowered during this time period.

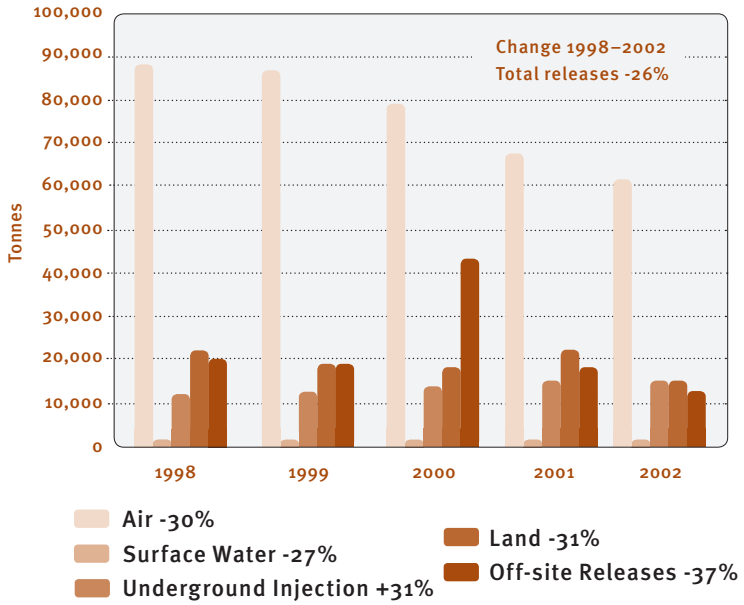
Figure 3-3 Industrial Sectors with the Largest On-site Air Emissions of Carcinogens Reported to North American PRTRs, 2002
(2002 Matched Chemicals and Industries)



Total On-site Air Emissions: 62,297 tonnes

Note: Canada and US data only. Mexico data not available for 2002. Data include 55 chemicals common to both NPRI and TRI lists from selected industrial and other sources. A chemical is considered a carcinogen for the purposes of this report if it is so classified by the International Agency for Research on Cancer (IARC) **<http://www.iarc.fr/>** or the US National Toxicology Program (NTP) **<http://ntp-server.niehs.nih.gov />**. Substances classified under IARC as carcinogenic to humans (1), probably carcinogenic to humans (2A), and possibly carcinogenic to humans (2B) are included. Under the US National Toxicology Program, substances classified as known to be carcinogenic (K) or may reasonably be anticipated to be carcinogenic (P) are included.

Figure 3-4 Releases (On- and Off-site) of Carcinogens in North America, 1998–2002
(1998–2002 Matched Chemicals and Industries)



Note: Canada and US data only. Mexico data not available for 1998–2002. Does not include lead and its compounds and polychlorinated alkanes. A chemical is considered a carcinogen for the purposes of this report if it is so classified by the International Agency for Research on Cancer (IARC) **<http://www.iarc.fr/>** or the US National Toxicology Program (NTP) **<http://ntp-server.niehs.nih.gov />**. Substances classified under IARC as carcinogenic to humans (1), probably carcinogenic to humans (2A), and possibly carcinogenic to humans (2B) are included. Under the US National Toxicology Program, substances classified as known to be carcinogenic (K) or may reasonably be anticipated to be carcinogenic (P) are included.

3.3.2 Releases and Transfers of Recognized Developmental and Reproductive Toxicants

Developmental and reproductive toxicants are those substances that can produce detrimental effects during fetal development. Some of these effects include structural abnormalities and other birth defects, low birth weight, growth retardation, fetal death, metabolic or biological dysfunction and psychological and behavioral defects that manifest as the child grows (Goldman and Koduru 2000, Scorecard 2002). PRTR data provide one source of

information on releases and transfers of known developmental and reproductive toxicants from larger industrial facilities.

What quantities of recognized developmental and reproductive toxicants were released and transferred in Canada and the US in 2002?

In Canada and the United States, almost half a million tonnes (482,600 tonnes) of chemicals that are recognized developmental and reproductive toxicants were released and transferred in 2002. Almost 95,500 tonnes of this total amount of recognized developmental and reproductive toxicants were released at the site of the facility, directly into the air, land, or water or injected underground. Of particular concern are the 58,600 tonnes of chemicals recognized as developmental and reproductive toxicants that were directly released into the air from facilities (**Table 3-5**).

Recognized developmental and reproductive toxicants made up approximately 15 percent of the total amount of matched chemicals released and transferred in Canada and the United States (3.25 million tonnes). Eighty-seven percent of the Canadian and United States total load of recognized developmental and reproductive toxicants originated from US facilities that reported to the TRI, and 13 percent came from Canadian facilities that reported to NPRI.

Which recognized developmental and reproductive toxicants were released and transferred in the largest quantities?

In 2002, the recognized developmental and reproductive toxicants released or transferred in the five largest quantities were:

- Lead and its compounds (211,200 tonnes)
- Toluene (134,800 tonnes)
- Nickel and its compounds (82,900 tonnes)
- Carbon disulfide (13,800 tonnes)
- N-Methyl-2-pyrrolidone (13,400 tonnes)

Of special concern are the recognized developmental and reproductive toxicants with the highest air releases: toluene, carbon disulfide, and benzene (**Table 3-6**).

How do the quantities of recognized developmental and reproductive toxicants released to air and water compare in terms of toxicity?

Table 3-7 applies the toxic equivalency potentials (TEPs) for air and water releases to the air and water releases of the recognized developmental and reproductive toxicants, in addition to showing the data on total releases. As shown, the relative ranking of the chemicals changes when TEPs are applied. For releases to air under the TEP ranking:

- Mercury and its compounds is ranked #1 (ranked #14 in terms of amounts of air releases)
- Lead and its compounds is ranked #2 (ranked #7 in terms of amounts of air releases)
- Toluene is ranked #6, whereas it has the largest air releases in terms of amounts of the recognized developmental and reproductive toxicants

For releases to water under the TEP ranking:

- Mercury and its compounds is again ranked #1 (ranked #14 in terms of tonnes of water releases)
- Lead and its compounds is again ranked #2 (also ranked #2 in amounts of water releases)
- Nickel and its compounds is ranked #3, whereas it has the largest water releases, in tonnes, of the recognized developmental and reproductive toxicants

Thus we find that in the case of recognized developmental and reproductive toxicants the application of TEPs helps to focus attention not only on quantities of releases but also on the potential for toxicity. It can be seen that this analysis is limited by a number of missing TEPs. Those with missing TEPs included one of the top ten air developmental and reproductive toxicants (N-methyl-2-pyrrolidone) and two of the top ten water developmental and reproductive toxicants (N-methyl-2-pyrrolidone and lithium carbonate).

Where were the largest quantities of recognized developmental and reproductive toxicants released?

Tennessee, Ontario, Texas and Indiana led the United States and Canada in releasing (on- and off-site) the largest quantities of recognized developmental and reproductive toxicants in 2002:

- Tennessee, with 14,000 tonnes
- Ontario, with 8,600 tonnes
- Texas, with 7,500 tonnes
- Indiana, with 7,100 tonnes

Tennessee led the United States and Canada in releases of recognized developmental and reproductive toxicants to air (12,900 tonnes), followed by Ontario (6,000 tonnes) and Texas (3,800 tonnes) (**Table 3-8**).

Which industrial sectors released the largest quantities of recognized developmental and reproductive toxicants?

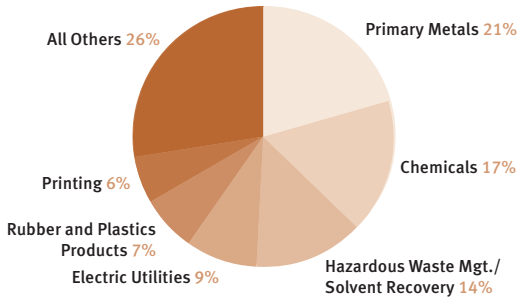
Three sectors released (on- and off-site) the largest quantities of recognized developmental and reproductive toxicants in Canada and the United States in 2002 (**Figure 3-5**):

- Primary metals (includes steel mills, etc.), with 27,500 tonnes
- Chemicals (includes chemical manufacturing and processing), with 22,000 tonnes
- Hazardous waste management/solvent recovery, with 18,500 tonnes

Three sectors were responsible for well over half of the recognized developmental and reproductive toxicants released to the air in the United States and Canada in 2002 (**Figure 3-6**):

- Chemical manufacturing, with 17,400 tonnes
- Rubber and plastics products, with 8,300 tonnes
- Printing and publishing, with 7,400 tonnes

Figure 3-5 Industrial Sectors with the Largest Releases (On- and Off-site) of Recognized Developmental and Reproductive Toxicants, 2002 (2002 Matched Chemicals and Industries)



Total Releases On- and Off-site: 128,673 tonnes

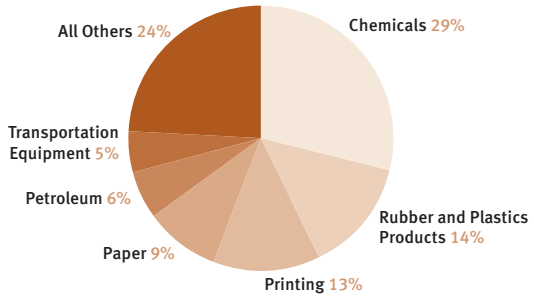
Note: Canada and US data only. Mexico data not available for 2002. Data include chemicals common to both NPRI and TRI lists from selected industrial and other sources. A chemical is included as a developmental or reproductive toxicant if it is listed as a recognized developmental or reproductive toxicant on the California Proposition 65 list <www.oehha.ca.gov/prop65/prop65_list/files/070904list.html >.

Have the releases of recognized developmental and reproductive toxicants increased or decreased over time?

In the United States and Canada, the amount of recognized developmental and reproductive toxicants released decreased by 28 percent from 1998 to 2002. Releases of all the matched chemicals in the TRI-NPRI matched data set decreased by 11 percent over the same time period. On-site air releases of recognized developmental and reproductive toxicants represent about three-quarters of all releases of these chemicals. From 1998 to 2002 air releases of recognized developmental and reproductive toxicants fell by 31 percent. In contrast, on-site land releases increased by 4 percent (368 tonnes) from 1998 to 2002. This was due to reporting by one primary metals facility, BHP Copper in San Manuel, Arizona, which reported an increase of 3,200 tonnes in on-site land releases. The facility indicated that this was a one-time release due to discontinued operations related to mining. Both Canada and the United States showed decreases in releases of recognized developmental and reproductive toxicants from 1998 to 2002 (**Figure 3-7**). These data show that there have been successful efforts to prevent pollution, particularly for recognized developmental and reproductive toxicants, by reducing or eliminating environmental releases and disposal from these sectors in Canada and the United States over this period.

These trends do not include lead and mercury and their compounds because the reporting thresholds for these chemicals were lowered between 1998 and 2002 in order to better capture these releases, which are of concern at lower levels because of the persistence and toxicity of these substances.

Figure 3-6 Industrial Sectors with the Largest On-site Air Emissions of Recognized Developmental and Reproductive Toxicants, 2002
(2002 Matched Chemicals and Industries)



Total On-site Air Emissions: 58,591 tonnes

Note: Canada and US data only. Mexico data not available for 2002. Data include 21 chemicals common to both NPRI and TRI lists from selected industrial and other sources. A chemical is included as a developmental or reproductive toxicant if it is listed as a recognized developmental or reproductive toxicant on the California Proposition 65 list <www.oehha.ca.gov/prop65/prop65_list/files/070904list.html>.

3.3.3 Releases and Transfers of Suspected Developmental and Reproductive Toxicants

Those chemicals with less weight of evidence of developmental or reproductive effects are considered *suspected* development and reproductive toxicants (Scorecard 2002).

What quantities of suspected developmental and reproductive toxicants were released and transferred in Canada and the US?

In Canada and the United States, over two and a quarter million tonnes of suspected developmental and reproductive toxicants were released and transferred in 2002. Almost one million tonnes (974,700 tonnes) were released on- and off-site. Of particular concern is the 273,900 tonnes of suspected developmental and reproductive toxicants that were directly released into the air from facilities (**Table 3-9**).

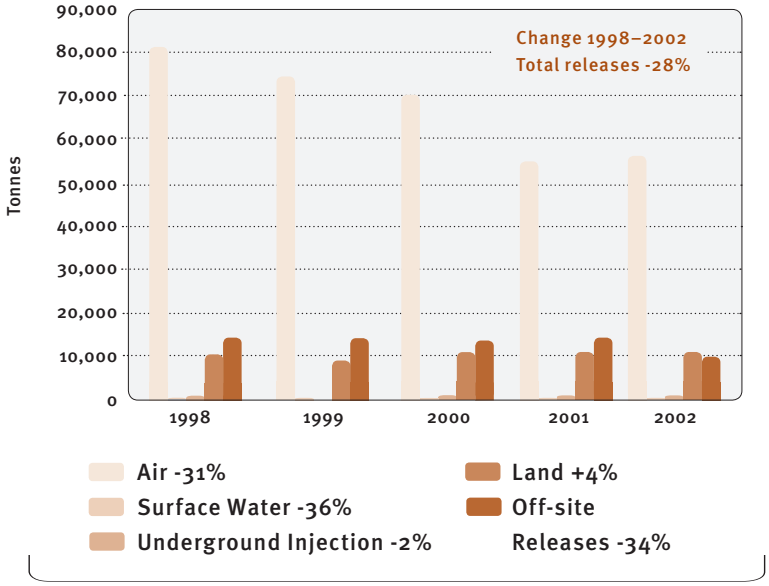
Suspected developmental and reproductive toxicants made up more than two-thirds of the total amount of matched chemicals released and transferred in Canada and the United States (2.25 million of 3.25 million tonnes). Eighty-nine percent of the Canada/US total load of suspected developmental and reproductive toxicants originated from US TRI facilities, and 11 percent came from Canadian NPRI facilities. Chemicals suspected of having developmental and reproductive effects represent a large proportion of total reported amounts, indicating that a closer examination of these chemicals' potential for causing developmental and reproductive toxicity would be warranted.

Which suspected developmental and reproductive toxicants were released and transferred in the largest quantities?

In 2002, the five chemicals suspected to be developmental and reproductive toxicants released or transferred in the largest quantities were (**Table 3-10**):

- Copper and its compounds (457,400 tonnes)
- Zinc and its compounds (406,300 tonnes)
- Methanol (244,900 tonnes)

Figure 3-7 **Releases** (On- and Off-site) **of Recognized Developmental and Reproductive Toxicants in North America, 1998–2002**
(1998–2002 Matched Chemicals and Industries)



Note: Canada and US data only. Mexico data not available for 1998–2002. Does not include lead and its compounds and mercury and its compounds. A chemical is included as a developmental or reproductive toxicant if it is listed as a recognized developmental or reproductive toxicant on the California Proposition 65 list ‹www.oehha.ca.gov/prop65/prop65_list/files/070904list.html›.

- Nitric acid and nitrate compounds (244,100 tonnes)
- Manganese and its compounds (191,700 tonnes)

It is of note that some of these compounds (certain forms of copper and zinc) are essential trace nutrients at lower levels of exposures; moreover, over-exposure to these substances is quite unusual. While the releases of these substances may be of limited concern to the public, other types of exposures, such as to industrial workers, may confer serious health risks. The suspected developmental and reproductive toxicants released into the air in the largest amounts were (**Table 3-10**):

- Methanol (88,600 tonnes)
- Hydrogen fluoride (35,100 tonnes)
- Xylenes (26,100 tonnes)
- Styrene (23,500 tonnes)
- *n*-Hexane (23,100 tonnes)
- Methyl ethyl ketone (16,200 tonnes)

Of note is that the methanol released to the environment from such facilities may be insignificant as a source of exposure, compared to other sources, such as formation in food.

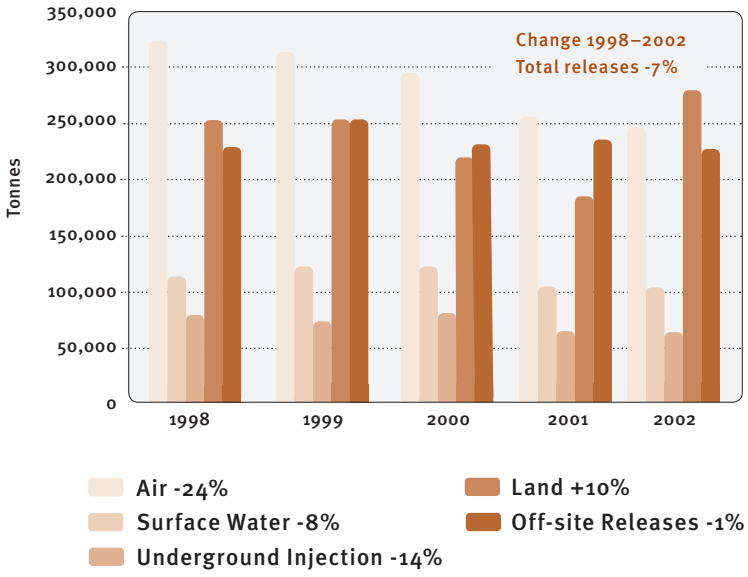
Has the quantity of suspected developmental and reproductive toxicants released increased or decreased over time?

The release of all suspected developmental and reproductive toxicants decreased by 7 percent from 1998 to 2002 in Canada and the United States (**Figure 3-8**). Air releases of suspected developmental and reproductive toxicants fell by 24 percent. On-site releases to land (mainly disposal in landfills) of suspected developmental and reproductive toxicants increased by 10 percent during this time period. This was due to reporting by one primary metals facility, BHP Copper in San Manuel, Arizona, which reported an increase of 109,100 tonnes in on-site land releases of copper, manganese and zinc compounds. The facility indicated that this was a one-time release due to discontinued operations related to mining. Without reporting by this one facility, on-site land releases of suspected developmental and reproductive toxicants would have decreased by 32 percent and total releases by 18 percent from 1998 to 2002. Releases of all the matched chemicals in the TRI-NPRI matched data set decreased by 11 percent over the same time period. These data support the conclusion that, for suspected developmental and reproductive toxicants, there have been successful efforts to prevent pollution by reducing or eliminating environmental releases and disposal from these sectors in Canada and the United States over this period.

3.3.4 Releases and Transfers of Suspected Neurotoxicants

Neurotoxicants are chemicals that alter the structure or functioning of the central and/or peripheral nervous system (Scorecard 2002).

Figure 3-8 **Releases** (On- and Off-site) **of Suspected Developmental and Reproductive Toxicants in North America, 1998–2002**
(1998–2002 Matched Chemicals and Industries)



Note: Canada and US data only. Mexico data not available for 1998–2002. A chemical is included as a developmental or reproductive toxicant if it is listed as a suspected developmental or reproductive toxicant on the Scorecard list ‹www.scorecard.org›.

What quantities of suspected neurotoxicants were released and transferred in Canada and the US?

In Canada and the United States, over two and a half million tonnes of suspected neurotoxicants were released and transferred in 2002. One million tonnes were sent for recycling and almost one million tonnes were released on- and off-site. Of particular concern are the 378,300 tonnes of suspected neurotoxicants that were directly released into the air from facilities (**Table 3-11**).

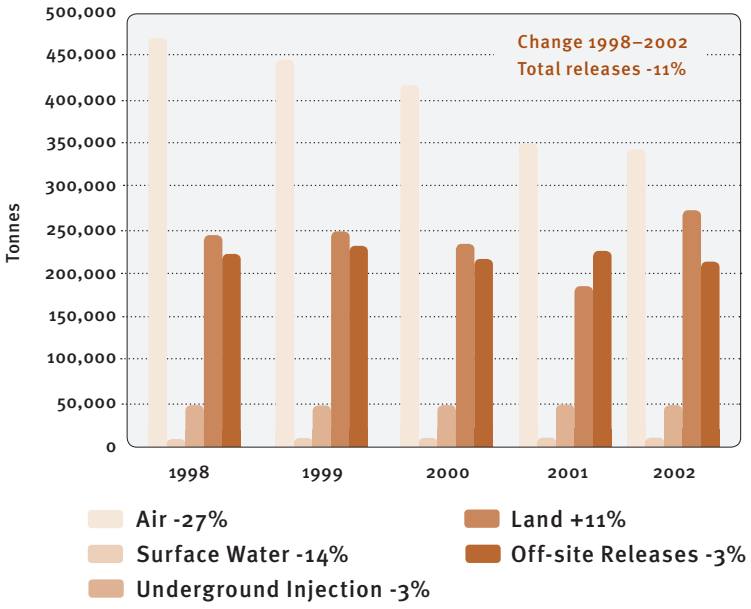
Suspected neurotoxicants made up more than three-quarters (77 percent) of the total amount of matched chemicals released and transferred in Canada and the United States (3.25 million tonnes). Eighty-eight percent of the North American total load of suspected neurotoxicants originated from US facilities that reported to TRI, and 12 percent came from Canadian facilities that reported to NPRI. Suspected neurotoxicants are a large proportion of the emissions, indicating that a closer examination of these chemicals' potential to cause neurotoxicity would be warranted.

Has the quantity of suspected neurotoxicants released increased or decreased over time?

Suspected neurotoxicants decreased by 11 percent from 1998 to 2002 in Canada and the United States (**Figure 3-9**). However, air releases of suspected neurotoxicants fell by 27 percent. On-site releases of suspected neurotoxicants to land (mainly disposal in landfills) increased by 11 percent during this time period. This was due to reporting by one primary metals facility, BHP Copper in San Manuel, Arizona, which reported an increase of 109,100 tonnes in one-time, on-site land releases of copper, manganese, nickel and zinc compounds. Without reporting by this single facility, on-site land releases of suspected neurotoxicants would have been 34 percent less and total releases 22 percent less from 1998 to 2002. Releases of all the matched chemicals in the TRI-NPRI matched data set decreased by 11 percent over the same time period. These data support the conclusion that, for suspected neurotoxicants, there have been successful efforts to prevent pollution by reducing or eliminating environmental releases and disposal from these sectors in Canada and the United States over this period.

These trends do not include lead and mercury and their compounds because the reporting thresholds for these chemicals were lowered between 1998 and 2002.

Figure 3-9 **Releases** (On- and Off-site) **of Suspected Neurotoxicants in North America, 1998–2002**
(1998–2002 Matched Chemicals and Industries)



Note: Canada and US data only. Mexico data not available for 1998–2002. A chemical is included as a neurotoxicant if it is listed as a suspected neurotoxicant on the Scorecard list <www.scorecard.org>.

3.4 Chemicals of Concern to Children's Health

In addition to analyzing releases and transfers of carcinogens, developmental and reproductive toxicants, and neurotoxicants reported to PRTRs, we can look at individual chemicals widely recognized to be of concern to children's health. Some of these chemicals are:

- Lead
- Mercury
- PCBs
- Dioxins and furans
- Phthalates
- Manganese

This chemical list is illustrative of some of the chemicals that are recognized to have adverse effects on children's health at sufficient levels of exposure. Many more chemicals, some just being recognized and others not traditionally monitored, are also likely to affect children's health.

3.4.1 Lead and its Compounds

Uses of lead

Lead is produced by mining and smelting of ores. In North America, a major use is in the lead acid batteries used in automobiles. The second-largest use of lead is in pigments and compounds (9 percent of Western world demand in 1999). Other uses of lead are in PVC stabilizers, in color pigments, and in the manufacture of glass (crystal, light bulbs, insulators and television/computer screens). The US and Canada have taken regulatory action and Mexico voluntary steps to curtail use of lead solder in plumbing (CEC 2004a); regulatory action on lead solder in electronic equipment in Europe and Japan is causing manufacturers to switch to non-lead solders (Li *et al.* 2005).

Elemental lead and lead alloys are also used for the production of steel and brass, in rolled sheet and strip roofing applications, in power and communication cable sheathing (especially underground and submarine), as a sound barrier in construction, and as shielding around X-ray equipment and at nuclear installations. Lead also is used as a weight in the keels of boats and to balance tires. It has a number of other consumer uses as well, including glazing for pottery, and has been found at hazardous levels in a long list of consumer products in recent years, including some imported crayons, plastic mini-blinds, a wide range of inexpensive jewelry and toy figu-

rines, and even in some candle wicks. It has also been used in folk remedies (Flattery *et al.* 1993).

Health effects of lead

Lead as a metal and in its compounds behaves as a neurotoxicant and developmental toxicant and inorganic lead compounds may behave as carcinogens (IARC 2004, Bellinger 2005). Lead can damage a child's developing brain, kidneys and reproductive system. Even low levels of lead are associated with learning disabilities, hyperactivity, behavioral problems, impaired growth and hearing loss (Needleman and Bellinger 1991). Low-level exposure stunts the growth of children, both *in utero* and as they grow to adolescence. Lead may be related to the onset of puberty in adolescent girls (Denham *et al.* 2005, Selevan *et al.* 2003). As our knowledge of lead effects increases, many researchers have come to realize that there may not be any safety threshold for lead's impact on human health (Federal/Provincial Committee on Environmental and Occupational Health 1994). Recent research suggests a relationship between impaired IQ and blood lead levels even below the intervention level of 10 micrograms of lead per deciliter (µg/dL) of blood (Canfield *et al.* 2003).

Given the same exposure dosage of lead, children will absorb more than adults. An infant may absorb up to 50 percent of the lead dose through the intestine, while an adult may absorb only 10 percent of the same lead dose (Plunkett *et al.* 1992). Infants also have an immature blood-brain barrier, which allows lead to pass more easily into brain tissue (Rodier 1995).

Moreover, the effects of lead may be irreversible. Adolescents who, as children, had high lead levels in their teeth in Grades 1 and 2 were seven times more likely to be high school dropouts and six times more likely to read at least two grade levels below expectation. They also showed higher rates of absenteeism in their final year of school, along with a lower class rank, poorer vocabulary, lower grammatical scores, longer reaction times and poorer hand-eye coordination (Needleman *et al.* 1990).

Unlike most organic chemicals, lead, a metal, does not break down in the environment. Lead released into the air is a concern not only for direct exposure via inhalation but also for indirect routes of exposure, such as falling onto agricultural land and entering the food supply, or falling onto dust and soil, where it becomes accessible to children.

Lead levels and exposures in North America

Health Canada states that Canadian children are most likely to be exposed to lead from food, then air, then drinking water. Estimates of daily lead exposure for preschoolers (ages 1 to 4) are 1.1 g/kg body weight from food, 2–10 micrograms from air, and 2.9 micrograms for drinking water. Soils and household dust can also be significant sources of lead exposure for young children (Health Canada 1998b). A recent study (Rasmussen *et al.* 2001) found that indoor sources, unrelated to outdoor soil lead levels, can contribute significantly to lead exposures. There are no national data on blood lead levels for Canadian children below age six years and only one national survey (1979–1980) of blood lead levels in older children has been conducted.

Children also may be exposed to lead from a number of other sources, including mobile sources (now much reduced due to removal of lead in gasoline in North America), deteriorating lead-based paint in the home, mining, pottery glazes, a parent or sibling working in a lead-related industry or in a cottage industry as a hobbyist or artist. The importance of a particular source of lead will vary with the amount of lead, the type and the extent of exposure.

The removal of lead from gasoline has reduced atmospheric concentrations of lead and is reflected in the lower levels of lead in children's blood. Blood screening surveys in Ontario from 1983 to 1992 indicate a steady decline in lead levels: 1.04 micrograms of lead per deciliter (µg/dL) of blood each year (Wang *et al.* 1997). In 1992, blood lead levels of children (ages 1 to 5) in Ontario averaged 3.11 µg/dL. This was similar to the US mean of 3.52 µg/dL. Averages, however, can cloak children with high blood levels who require treatment. The distribution of Ontario's blood lead levels indicates that a portion of children have blood lead levels at or above the intervention level.

In the early 1990s, between 40 and 88 percent of Mexican children (from various studies) were reported to have blood lead levels that exceeded the US Centers for Disease Control and Prevention (CDC) intervention levels of 10 µg/dL (Romieu *et al.* 1994). Several studies found that Mexican children with higher lead levels had reduced IQ, increased frequency of crying, lower birth weight, and were shorter at birth and at three years old. Mexican mothers with high lead levels had increased risk of miscarriage and a three-fold increase in the frequency of premature babies (less than 37 weeks) (Romieu *et al.* 1994).

In 1991, Mexico phased out the use of lead in gasoline, decreasing airborne lead concentrations in Mexico City by 90 percent (Rothenberg *et al.* 1998) and contributing to lower blood lead levels locally. More recently, blood lead levels of full-term babies born in three Mexico City hospitals have averaged 8 µg/dL (Torres-Sanchez *et al.* 1999). However, the use of lead pigment in pottery glazes is still common in parts of Mexico, as well as lead emissions from battery recycling and vehicle repair shops and smelters. These exposures cause many children living in the vicinity of these facilities in Mexico to have blood lead levels exceeding the US Centers for Disease Control and Prevention (CDC) intervention levels of 10 µg/dL. For example, blood lead levels of children living within one kilometer of a smelter in Torreón averaged 17 µg/dL, compared to those of children living approximately five kilometers from the smelter, which averaged approximately 5 µg/dL (Calderon-Salinas *et al.* 1996). Children with parents who are exposed to lead at work are also reported to have higher blood lead levels. For instance, children of radiator repairmen with home-based workshops had average blood lead levels around 22 µg/dL, while children of repairmen with external workshops had average blood lead levels of approximately 14 µg/dL; these compare to average blood lead levels of 5.6 µg/dL among children in the control group (Aguilar-Garduno *et al.* 2003).

Lead levels in bone can be used as a longer-term indicator of lead exposure than blood lead levels. In pregnancy, lead stored in the bone is rapidly turned over, which can expose the developing child to lead even if the mother is not currently exposed. This means that fetal exposure to lead, not just daily exposure in a child's environment, can cause mental impairment in infants. A recent groundbreaking study conducted in Mexico City, with a team that included researchers from the Harvard School of Public Health, showed that mothers with higher levels of lead in their bones produced infants with impaired mental development (Gomaa *et al.* 2002). Cognitive development was more affected than motor skill development. It is therefore not only important to lower the amount of lead a mother is exposed to during pregnancy, but also in the years before pregnancy. This finding suggests that lead is an intergenerational problem. A mother's exposure to lead many years before pregnancy can significantly affect the mental functioning of her infant.

Blood lead levels in US children have decreased over the last twenty years. The current blood lead level in children which triggers intervention is 10 µg/dL. Between 1976 and 1980, the average blood lead level was between 14.1 and 15.8 µg/dL, which decreased to between 3.3 and 4.0 µg/dL between 1988 and 1991, and then fell to between 2.0 and 2.5 µg/dL in 1999–2000 (CDC 2003a) and to 1.4 for 2001–2002 (CDC 2005a). Approximately two million US children under the age of six live in homes with flaking or deteriorating lead paint (CDC 1997).

There has been cooperation between Mexico and the United States to address problems with lead contamination of children's candy that previously entered commerce along the border between these two countries (US FDA 2004). Additionally, there is accelerated international action to remove lead from gasoline and other uses worldwide (UNEP 2001).

What can PRTR data tell us about releases and transfers of lead and its compounds?

PRTR data provide information on one source of lead releases and transfers: those from larger industrial and other facilities. For children, PRTR data capture some important sources of lead, such as smelters and hazardous waste facilities. PRTR data can also help identify potential areas, facilities and sectors that may be important starting points for reducing lead exposure to children. However, for children in other areas, the most important sources of lead exposure may be from old lead paint, lead pottery, consumer products, and items such as folk remedies, which are not captured by PRTR data. This, of course, is because PRTRs were designed to gather data on industrial releases.

Based on the matched TRI and NPRI data for 2002, 211,200 tonnes of lead and its compounds were released and transferred (**Table 3-12**). Of this total amount, more than three-quarters (162,800 tonnes) was sent for recycling.

Over 960 tonnes of lead and its compounds were released into the air from matched TRI and NPRI facilities. Canadian NPRI facilities reported 400 tonnes of lead and its compounds released into the air, comprising over 40 percent of the total in Canada and the US.

The large amounts of lead and its compounds released into the air from Canadian facilities that reported to NPRI were driven by two Canadian smelters, which released the largest amounts of lead and its compounds into the air in North America in 2002. Indeed, the primary metals sector, which includes smelters, reported the largest releases, including the largest air releases, on-site land releases, and off-site releases (mainly transfers to disposal) (**Table 3-13**). The electronic/electrical equipment sector reported the largest transfers to recycling, accounting for over half of the lead and its compounds transferred to recycling in 2002.

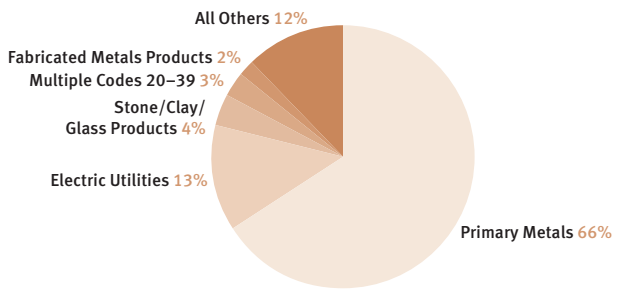
Three sectors in Canada and the US released (on- and off-site) the largest amounts of lead and its compounds in 2002:

- Primary metals (includes smelters) (20,500 tonnes)
- Hazardous waste management/solvent recovery (14,600 tonnes)
- Electric utilities (includes power plants burning oil and/or coal) (4,100 tonnes)

The primary metals sector was also the sector with the largest air releases, accounting for 66 percent of the total in 2002. Electric utilities (power plants burning oil and/or coal) accounted for 13 percent (**Figure 3-10**). The stone/clay/glass sector, which includes facilities making cement, accounted for 4 percent of air releases of lead and its compounds in 2002.

From 1998 to 2000, total releases (on- and off-site) of lead and its compounds decreased by 19 percent in Canada and the US. Air releases of lead and its compounds decreased by 6 percent (71 tonnes) (**Figure 3-11**). The decrease in releases of lead to the air from some facilities is encouraging, as this kind of release has been found to be an important source of lead exposure for children in some areas.

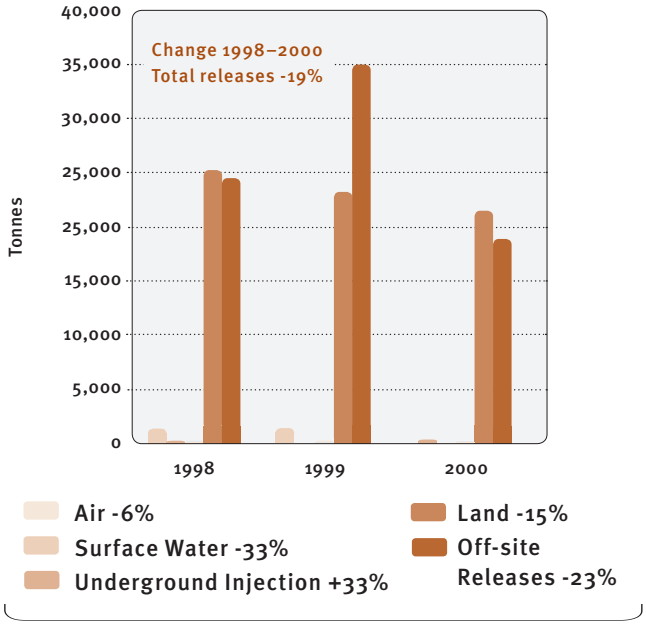
Figure 3-10 Industrial Sectors with the Largest On-site Air Releases of Lead and its Compounds, 2002
(2002 Matched Chemicals and Industries)



Total On-site Air Releases: 960,623 kg

Note: Canada and US data only. Mexico data not available for 2002.

Figure 3-11 **Releases** (On- and Off-site) **of Lead and its Compounds in North America, 1998–2000**
(1998–2000 Matched Chemicals and Industries)



Note: Canada and US data only. Mexico data not available for 1998–2000.

Comparisons between data from 2001 and 2002 cannot be made easily because the thresholds for reporting lead and its compounds have been lowered (for 2001 in TRI and for 2002 in NPRI). However, during the time period from 1995 to 2000, air releases of lead and its compounds decreased by more than 500 tonnes, or 33 percent, and total releases decreased by 2 percent. These trends are based on industries that consistently reported over this time period, thus electric utilities and hazardous waste/solvent recovery facilities are not included.

3.4.2 Mercury

Uses of mercury

Mercury is a naturally occurring metallic element, found throughout the environment. Because of its unique physical and chemical properties, this dense, fluid metal and its compounds have a wide spectrum of uses, from medical applications (medical instruments, dental amalgams and disinfectants) to pesticides (fungicides), industrial thermometers, switches in thermostats, pressure-measuring devices and fluorescent lamps (CEC 2000). The use of mercury in batteries, once very common, is declining. However, people are generally exposed to mercury through diet and via dental amalgam fillings (Clarkson 2002). The burning of coal for power generation is an important source of mercury in the environment. For example, it has been estimated that, within the United States, mercury from power plant emissions constitutes 41 percent of US anthropogenic (human origin) sources of mercury to the environment, but as little as 1 percent of the total "global pool," which includes natural as well as anthropogenic sources (Trasande *et al.* 2005).

Health effects of mercury

Mercury exists in three different forms (Health Canada 2002b):

- Elemental mercury—a silvery, shiny, volatile liquid, which slowly transmutes to a colorless, odorless vapor at room temperatures. Elemental mercury can remain in atmospheric circulation for up to one year and readily converts to other forms.
- Inorganic mercury—formed when elemental mercury combines with other elements, such as sulfur, chlorine, or oxygen to create mercury salts.
- Organic mercury (one form is called methylmercury)—a compound formed when elemental mercury combines with carbon and hydrogen in nature. Airborne elemental and inorganic mercury can be deposited into water, where it can be converted into organic methylmercury that accumulates in fish, wildlife and humans.

A variety of health conditions has been found to be due to mercury exposure, with the severity of effects depending on the amount and timing of exposure. Health impairment from high levels of exposure to elemental mercury includes damage to the stomach and large intestine, as well as permanent damage to the brain and kidneys, (US EPA 2002b).

Inorganic mercury salts may also cause health problems, such as kidney failure and gastrointestinal damage. Highly irritating at high levels of exposure, these salts can cause blisters and ulcers on the lips and tongue, or rashes, excessive sweating, irritability, muscle twitching, and high blood pressure (Health Canada 2002b). Sources of mercury exposure to children include consumer and industrial products such as broken thermometers and mercury switches, *in utero* exposures, breast milk and proximity to a source of mercury, such as certain hazardous waste facilities, utilities, smelters, mines and steel mills.

Children are primarily exposed to the most bioavailable form of mercury, methylmercury, from food, mainly fish and other seafood, where it can bioaccumulate to levels up to 100,000 times greater than in the surrounding water (Health Canada 2002b). Releases of mercury to the air from industrial and combustion sources contribute to levels of mercury in fish and other seafood. Methylmercury is a developmental neurotoxicant. When pregnant women eat fish contaminated with mercury, the methylmercury can cross the placenta and enter the body of the developing child. It readily accumulates in the brain. Depending on how much is absorbed, infants exposed to methylmercury can appear normal at birth but later show impairment of attention focus, fine motor function, language, drawing ability and memory. Such effects have been shown to occur with levels of exposure that can result from the consumption of contaminated fish and other seafood from locations such as the Great Lakes, the Faroe Islands and New Zealand (NAS 2000, Goldman and Shannon 2001, Stewart *et al.* 2003); however, not all studies have demonstrated such effects (Myers *et al.* 2003). Methylmercury

has other toxic effects as well, including on the cardiovascular and immune systems, although children have not been found to be particularly sensitive to such effects (National Academy of Sciences 2000). More recently, studies have found new evidence for a relationship between exposure to methylmercury and cardiovascular disease (Stern 2005).

Fish is an excellent source of high-quality protein, and is low in saturated fat, which makes it a healthy food choice. Because of nutritional value, fish continue to be an important food source available to consumers, with advice to limit consumption to avoid exposure to hazardous levels of mercury. Specifically, pregnant women, women of childbearing age and young children are advised to limit their consumption of shark, swordfish and fresh and frozen tuna to no more than one meal per month. For others in the population, a consumption level of no more than one meal per week is recommended for these species (Health Canada 2002c). There are currently no data available for fish consumption advisories in Mexico and advisories are not issued by Mexican authorities at the national or state level.

What can PRTR data tell us about releases and transfers of mercury?

Mercury has historically been emitted in large quantities from chlor-alkali plants (manufacturing plants that make chlorine and caustic soda, using mercury during the process), Portland cement production, incineration of medical and municipal wastes, and fossil fuel (especially coal) combustion in utility boilers (US EPA 1997b).

PRTR data provide information on mercury releases to the environment from certain industrial and combustion sources. PRTR data can help identify potential areas, facilities and sectors that may be important starting points for reducing mercury exposure to children. However, the matched NPRI and TRI data do not include municipal incinerators, which are often a significant source of mercury emissions. Also missing are a number of smaller anthropogenic sources as well as natural releases of mercury (e.g., from volcanic eruptions) or sources from outside Canada, Mexico and the United States.

In Canada and the US in 2002, approximately 453,300 kg of mercury and its compounds were released and transferred from matched TRI and NPRI facilities. PRTR facilities reported that approximately 65,900 kg were released to the air, and 608 kg to the water. Large amounts of mercury and its compounds (almost 91,400 kilograms) were sent off site for disposal, while a similar amount, 82,000 kg, was disposed of on-site in landfills (**Table 3-14**).

Over half of all releases of mercury and its compounds were contributed by two industry sectors (**Table 3-15**):

- Hazardous waste management/solvent recovery (73,200 kg)
- Electric utilities (power plants that burn oil and/or coal) (69,300 kg)

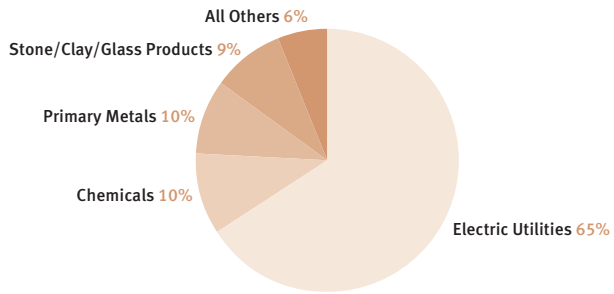
In the case of waste management facilities, because mercury flows and volatilizes, it is more difficult to capture and immobilize it in landfills than other metals. Electric utilities accounted for two-thirds of the air releases of mercury and its compounds in 2002 (**Figure 3-12**). The presence of mercury as a natural constituent of coal has created challenges in controlling releases of mercury from any of the sectors that use coal to generate energy.

From 2000 to 2002, total releases (on- and off-site) of mercury and its compounds decreased by 57 percent in Canada and the United States. This large decrease occurred in off-site releases (transfers to disposal), which decreased from 426,200 kg to 91,400 kg. Air releases of mercury and its compounds decreased by 10 percent (7,000 kg) (**Figure 3-13**). The decrease in mercury releases to the air from some facilities is encouraging, as this has been found to be an important source of mercury exposure for children in some areas. These decreases have been dramatic, indicating that in some PRTR sectors there have been real efforts to reduce not only releases but also generation of wastes that contain mercury.

Comparison of data from 2000 to 2002 with data from the years before 2000 should not be made because the thresholds for reporting mercury and its compounds were lowered, starting with the 2000 reporting year.

Figure 3-12 Industrial Sectors with the Largest On-site Air Releases of Mercury and its Compounds, 2002

(2002 Matched Chemicals and Industries)



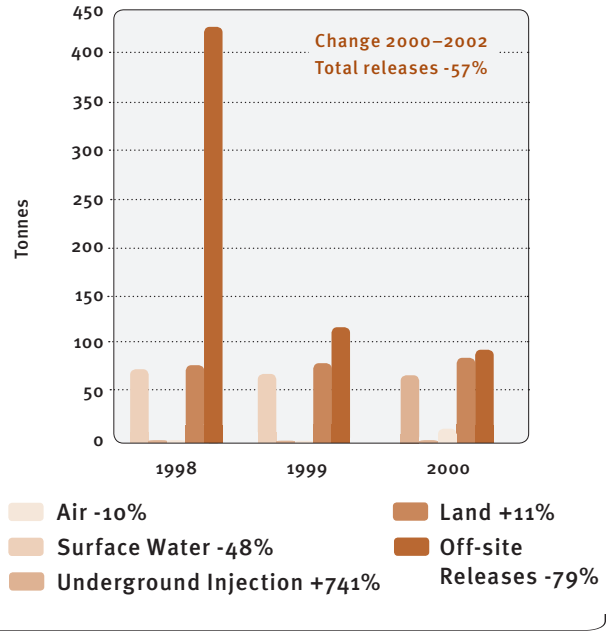
Total On-site Air Releases: 65,901 kg

Note: Canada and US data only. Mexico data not available for 2002.

Mercury levels and exposures in North America

In northern Canada, the Inuit have been particularly affected by mercury and other contaminants. Due to a diet dependent upon fish and mammals, the Inuit have mercury in their blood at levels known to cause developmental toxicity in children (Muckle *et al.* 2001, Dewailly *et al.* 2001). In Ontario, over 95 percent of surveyed lakes had levels of mercury that exceeded the WHO guideline of 0.5–1.0 mg/kg fish body weight, resulting in widespread fish consumption warnings (Environment Canada 2000). Since December 2000, mercury-based antimicrobial pesticides are no longer registered under the Pest Control Products Act by the Pest Management Regulatory Agency

Figure 3-13 **Releases** (On- and Off-site) **of Mercury and its Compounds in North America, 2000–2002**
(2000–2002 Matched Chemicals and Industries)



Note: Canada and US data only. Mexico data not available for 2000–2002.

(PMRA) and are not allowed to be intentionally added to any Canadian-produced paints.

Limited information exists about Mexican children's exposure to mercury. Drinking water studies found mercury in 42 percent of the samples in Sonora (Wyatt *et al.* 1998). A mercury inventory is under development in Mexico, which will help identify sources of mercury to the environment. The first draft results indicate the total amount of mercury air emissions is about 40 tonnes per year, mainly from gold mining and refining (11 tonnes/year), mercury mining and refining (10 tonnes/year), medical waste incinerators (seven tonnes/year), and chlor-alkali plants (five tonnes/year) (CEC 2001).

In the United States, the CDC's national exposure report has provided clear information about the levels of mercury in women of childbearing age (and therefore of the exposure amounts to which children are subjected *in utero*). Depending on the extent to which mercury is transferred from mother to baby via the placenta, between 8 and 16 percent of women of childbearing age in the United States National Health and Nutrition Examination Survey (NHANES) had blood mercury levels above the reference dose level (5.8 µg/L), which is the US EPA's regulatory standard for mercury, and below 58 µg/L, a concentration associated with neurologic effects in the fetus in epidemiology studies (Mahaffey *et al.* 2004). NHANES data for the period 1999–2002, reported in the CDC *Third National Report on Human Exposure to Environmental Chemicals,* show that 5.7 percent of women of childbearing age had levels between EPA's standard of 5.8 and 58 µg/L (National Health and Nutrition Examination Survey, 1999–2002). While defining safe levels of mercury in blood continues to be an active research area, it is encouraging that the percentage of US women with levels in the highest category of exposure appears to have decreased over the last few years.

The CEC's Sound Management of Chemicals program has developed a North American Regional Action Plan on Mercury to facilitate coordination among the three countries in addressing the measurement, monitoring, modeling, research and assessment of the effects of this toxic substance. The goal of this action plan is to significantly reduce mercury in the North American environment to levels attributable to naturally occurring sources. See ‹http://www.cec.org/programs_projects/pollutants_health/smoc/smoc-rap.cfm?varlan=english›.

On a global basis, the (UNEP) Governing Council of the UN Global Ministerial Environment Forum concluded, at its 23rd session in February 2005, that there is sufficient evidence of significant adverse impacts worldwide to warrant further international action to reduce the risks to humans and wildlife from the release of mercury to the environment. The Governing Council summary statement on Chemical Management from the February 2005 meeting reports that the UNEP mercury program should be further developed: that governments, the private sector, and international organizations should take immediate actions to reduce the risks posed by mercury in products and production processes; develop and implement partnerships as one approach to reducing risks to human health and the environment from the release of mercury and its compounds; and assess the need for further action on mercury, including the possibility of a legally-binding instrument, partnerships and other

First Systematic Picture of Chemical Body Burdens in Children Emerges

In 2005, the US National Center for Environmental Health (part of the US Centers for Disease Control and Prevention) continued to fill an important gap in our knowledge of the exposure of children to several common contaminants. The *Third National Report on Human Exposure to Environmental Chemicals* presented data on the body burdens of 148 chemicals, including metals (lead, mercury and cadmium), pesticide metabolites, phthalate metabolites, polycyclic aromatic hydrocarbons (PAHs), dioxins/furans, polychlorinated biphenyls (PCBs), phytoestrogens, and cotinine, which tracks exposure to tobacco smoke.

The CDC assessments have found that overall blood lead levels in children have continued to decline in the US. The CDC assessment also has allowed for an estimation of the percentage of children who are born with concentrations of mercury in blood above the EPA "reference dose" of 5.8 parts per billion (5.8 µg/L) (US EPA 2003).

These results will help improve our understanding of exposure to toxic chemicals. For more information, see **‹www.cdc.gov/exposurereport/3rd/default.htm›**.

actions advised by the Governing Council at its 24th session. At this time, such activities include work to reduce mercury exposures via products, chlor-alkali production, small-scale artisanal gold mining, and burning coal for energy.

3.4.3 PCBs

Uses of PCBs

Polychlorinated biphenyls (PCBs) are a mixture of persistent chlorinated chemicals no longer produced in North America but still found in the environment. PCBs formerly had many industrial uses—especially as heat transfer fluids in transformers, capacitors and fluorescent lamp ballasts. A variety of other uses included industrial applications as plasticizers, hydraulic fluids, vacuum pump and compressor fluids, and in the manufacture of inks, lubricants, flame-retardants, special adhesives and carbonless paper (ATSDR 2000). The estimated cumulative production of PCBs in the United States from 1930 to 1975 was 700,000 metric tonnes. About 44,000 tonnes of PCBs were imported into Canada and 10,000 tonnes into Mexico (CEC 1996).

Health effects of PCBs

PCBs are highly persistent, bioaccumulative and toxic chemicals with subtle yet pervasive health effects that linger long after exposure. They can deleteriously affect birth weight (Rogan *et al.* 1986, Patandin *et al.* 1998, Karmaus and Zhu 2004, Fein *et al.* 1984) and a number of neurological functions in children, including, memory, coordination, IQ and attention span; studies in several regions of the world have demonstrated such effects when exposure takes place at younger ages, with the effects persisting as long as these children have been followed (Winneke *et al.* 1998, Vreugdenhil *et al.* 2004, Stewart *et al.* 2003, Patandin *et al.* 1999b, Jacobson and Jacobson 2003, Grandjean *et al.* 2001). Although a consensus is developing that PCBs have strong developmental toxic effects (Schantz *et al.* 2003, Longnecker *et al.* 1997, Ribas-Fito *et al.* 2001, Mendola *et al.* 2002), some scientists believe that current data do not yet support such conclusions (Kimbrough and Krouskas 2001, 2002).

What can PRTR data tell us about releases and transfers of PCBs?

PRTR data provide information on one source of PCBs released to the environment from certain industrial and combustion sources. PRTR data can help identify potential areas, facilities and sectors that may be starting points for reducing PCB exposure to children. Data on PCBs are available from TRI, but PCBs are not reportable under NPRI.

Using TRI data, the total amount of PCBs released on- and off-site from industrial facilities has decreased over time, from over 187 tonnes in 1988 to less than 5 tonnes in 1999 (US EPA 2002c).

In 2002, the TRI reporting threshold was lowered to 10 pounds, or 4.5 kilograms, which resulted in several facilities newly reporting on PCBs. Hazardous waste facilities landfilled large quantities of PCBs (almost 564 tonnes) in 2002 and facilities sent 192 tonnes of PCBs off-site for treatment in 2002.

According to the most recent 1996 PCB inventory, over 2,800 sites across Canada had PCBs in storage awaiting destruction. One facility, Swan Hills in Alberta, destroyed over 10,000 tonnes of PCBs in 1996 (Environment Canada 2001).

Mexico had approximately 8,800 tonnes of PCBs in storage and in transformers in 1995 (CEC 1996).

PCB levels and exposure in North America

Children's exposure to PCBs can come from a variety of sources, including fish, other food, accidental spills, light ballasts, breast milk, *in utero*, and/or in proximity to a contaminated site or hazardous waste facility.

Canada has monitored levels of a number of persistent organic pollutants in breast milk over the years and has generally found a downward trend. However, it is estimated that exclusively breastfed infants under 6 months of age in the Great Lakes region are likely to be exposed to 81 percent of the daily intake recommended by Health Canada, that is, the provisional tolerable daily intake (PTDI) for PCBs of 1 mg/kg body weight/day. By comparison, the average adult takes in only two percent of the PTDI for PCBs (Haines *et al.* 1998a, 1998b). The concentration of PCBs in breast milk is considered to be an indicator of population exposure to these contaminants by Health Canada 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 the highest of all Canadians and among the highest globally (Haines *et al.* 1998a, 1998b).

Little is known about PCB exposures to children in Mexico. Albert and Aldana (1982) determined the content of PCBs in Mexican cereals and in packaging materials. They concluded that the main source of PCBs in cereals is the transfer from recycled paperboard used for the packaging.

PRTR data demonstrate the decline in releases of PCBs over time, reflecting the utility of bans and phase-outs on uses and production. However, large amounts of PCBs still remain in waste storage sites across North America, in selected uses, and in the large amounts that are sent to landfills and to treatment every year.

PCBs are still commonly found in soil, sediment, fish and people in North America. Because of the highly persistent, bioaccumulative nature of PCBs, it can take many decades for concentrations in the environment to decrease. For some children, such as those in the Arctic, those whose parents eat a lot of contaminated fish, or those who eat contaminated fish themselves, PCBs remain a health threat. Bans and phase-outs work to reduce environmental releases, but many children will still be exposed to harmful levels of PCBs during the time lag between phase-out and reduction in environmental concentrations.

Because PCBs are persistent in the atmosphere and travel long distances, a North American approach to reporting releases and

monitoring PCBs has been considered. The CEC's Sound Management of Chemicals program has developed a North American Regional Action Plan to facilitate coordination among the three countries in addressing the measurement, uses, storage, shipment, and waste reduction and recycling of these toxic substances. See ‹http://www.cec.org/programs_projects/pollutants_health/smoc/pcb.cfm?varlan=english›.

The actions under this plan have been recently completed by the three countries and the CEC's Environmental Monitoring and Assessment Task Force will be incorporating monitoring of PCBs in the environment on a North American scale into their activities.

3.4.4 Chlorinated Dioxins and Furans

Sources of dioxins and furans

Chlorinated dioxins and furans are a family of chemical compounds unintentionally created from a variety of processes, such as incineration, backyard burning, pulp and paper mills, smelters and electric utilities. Dioxins and furans can also be contaminants in some pesticides and chlorinated solvents. Other sources of dioxins include natural sources, such as forest fires and volcanoes, contaminated soils and sediments, and long-range transboundary air pollution.

Health effects of dioxins and furans

The toxicity of the compounds in the chlorinated dioxin and furan family varies, with 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) considered the most toxic. Some members of the dioxins family are considered carcinogens, suspected neurotoxicants, developmental toxicants and endocrine disruptors. Chlorinated dioxins and furans are considered to be persistent, bioaccumulative and toxic compounds (Birnbaum and Fenton 2003, Birnbaum and Staskal *et al.* 2003). Since the toxicity of these compounds is through a similar mode of action, the total amount of dioxins and furans found in environmental samples and humans is often expressed in the form of "dioxin international toxicity equivalence factors," or dioxin iTEQs.[6] Here the amounts of all the compounds are weighted according to their toxicity relative to 2,3,7,8-TCDD using a method developed under the auspices of the WHO (van den Berg *et al.* 1998).

What can PRTR data tell us about releases and transfers of dioxins and furans?

PRTR data provide information on dioxin and furans releases to the environment from some industrial and combustion sources. PRTR data can help identify potential areas, facilities and sectors that may be starting points for reducing dioxin and furan exposure to children.

Facilities began reporting dioxins and furans to both TRI and NPRI with the 2000 reporting year. This provides an improved picture of releases and transfers from some of the sources of dioxins and furans. However, the methods of dioxin and furan reporting differ between NPRI and TRI. The TRI and NPRI numbers are not comparable because they are reported from different industries and the reporting thresholds are different. Also they are in different units that are not readily convertible. For example, only chemical manufacturers producing chlorinated organic solvents must report on dioxins/furans, regardless of amounts and number of employees, to NPRI. In contrast, all chemical manufacturers who otherwise meet the TRI threshold of 0.1 grams per year, as well as TRI's minimum number of employees, must report. Also, NPRI reporting utilizes the iTEQs, so that facilities must report the sum of the amounts of the individual dioxin/furan compounds multiplied by their individual iTEQs. This has the advantage of presenting the data in one number. TRI reporting requires that facilities must report the sum of the dioxin and furan compounds in grams as well as a distribution of the types of dioxins/furans contained in the mixture. This method has the advantage of not being tied to the iTEQ system, which has changed over

Protecting Arctic Children

The image of a clean, untouched wilderness that many of us associate with the Arctic areas in North America is not completely accurate. Unfortunately, the Arctic and Arctic children are on the receiving end of emissions from sources often far to the south.

Elevated concentrations of many persistent toxic substances, such as PCBs, mercury and some pesticides, have been found in such traditional food sources as fish and marine mammals. Arctic women also show high levels of contaminants such as PCBs and mercury from eating this traditional food, as do their children, being nourished by breast milk and from other sources. According to the recent *Canadian Arctic Contaminants Assessment Report II* (Health Canada 2003): "Ten percent of mothers in Baffin region and 16 percent of Nunavik mothers have mercury blood levels that fall within Health Canada's 'increasing risk' category. Nearly 80 percent of Nunavik mothers and 68 percent of Baffin mothers have mercury blood levels that exceed a new guideline based on United States studies. Mercury levels in Yukon First Nations, Dene, Métis, and Inuit from Kivalliq and Kitikmeot regions are much lower and fall within Health Canada's 'acceptable' range."

Although the consumption of traditional foods containing contaminants may be associated with greater exposures and health risks, it is important to recognize that diets containing these foods confer substantial nutritional benefits and are the foundation of the social, cultural and spiritual way of life for Canada's Aboriginal Peoples.

To help protect the children in the Arctic, a series of remedial measures has been undertaken, including improved monitoring and testing, community education, and reduction of emissions from local, national and international sources.

6. TEQ is TEF times grams. TEF is the toxic equivalency factor that indicates the relative toxicity of the particular dioxin/furan congener relative to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (which has a TEF of 1.0). The TEFs used are those developed by international convention and adopted in 1989.

the years as new toxicity information has been developed on individual dioxin/furan compounds. In contrast, the TRI reports are in grams with a percentage distribution of congeners. These different national approaches to dioxin reporting will mean that different types of facilities will report to NPRI and TRI on transfers and releases of dioxins and furans.

NPRI data on dioxins and furans

In 2002, a total of 134.89 grams-iTEQ of dioxins and furans were released on-site from certain Canadian NPRI facilities. This was greater than the amount of dioxins and furans released off-site (103.97 grams-iTEQ) (**Table 3-16**).

Of particular concern are the 90.87 grams of dioxins and furans (iTEQ) released into the air in 2002. The three sectors that released the largest amounts into the air in 2002 were:

- Air, water, solid waste management (includes municipal incinerators, 46.77 grams-iTEQ)
- Primary metals (19.81 grams-iTEQ)
- Electric utilities facilities (15.95 grams-iTEQ)

Five municipal waste incinerators (teepee burners) located in Newfoundland were among the 10 NPRI facilities with the largest air releases of dioxins and furans. Municipal waste teepee burners are the target of proposed actions to reduce releases of dioxins and furans under Canada-wide standards.

From 2000 to 2002, total releases (on- and off-site) of dioxins and furans reported to the NPRI decreased by 32 percent, even though the number of facilities reporting on dioxins and furans increased (**Table 3-17**). The paper products industry reported the largest total releases (in grams-iTEQ) in all three years, but had a 40 percent reduction from 2000 to 2002. Sewer systems reported an overall increase of more than 20 grams-iTEQ from 2000 to 2002. Some of the increase may be due to the change in reporting requirements, which added wastewater collection systems discharging treated or untreated wastewater with an annual discharge of 10,000 m^3 or more per day into surface waters for 2002. However, it is clear that in Canada, dioxin releases to the environment have declined, especially in the paper products industry, which has made such reductions by adopting alternative bleaching technologies.

TRI data on dioxins/furans

TRI-reporting facilities in the United States report transfers and releases of dioxins and furans in grams rather than grams-iTEQ as NPRI-reporting facilities in Canada do. TRI-reporting facilities released 53,147 grams of dioxins and furans on-site in 2002. Over one and one-half times this amount was released off-site (87,146 grams) (**Table 3-18**).

Of particular note are the 3,511 grams of dioxins and furans released into the air.

The three industry sectors that released the largest amounts of dioxins and furans into the air in 2002 were:

- Electric utilities (power plants that combust oil and/or coal) (1,027 grams)
- Chemical manufacturers (976 grams)
- Primary metals (387 grams)

In addition to grams, TRI facilities also report the distribution for 17 dioxin/furan congeners. When the distribution and toxic equivalency factors for the congeners are applied to total releases in grams, the amount in grams-iTEQ can be calculated. Total releases for 2002 from TRI facilities reporting the distribution for dioxin/furan congeners was 928 grams-iTEQ. From 2000 to 2002, TRI facilities reported a decrease of 12.5 percent (from 1,060 to 928 grams-iTEQ) in total releases of dioxins and furans (**Table 3-19**). The chemicals industry had the largest total releases in all three years, and reported a decrease of 12 percent. The primary metals industry had the second-largest total releases (in grams-iTEQ) and reported a 6 percent decrease. Electric utilities reported the third-largest total releases (in grams-iTEQ) in 2000 but, with a 71 percent decrease, were ranked fourth in 2002. There is much uncertainty about the sum total of emissions of dioxins in the United States but the best estimate is by the US EPA; it is under review but the preliminary picture is that some 1,500 grams-iTEQ were released to the US environment in 2000 (US EPA 2005c).

Data for dioxins and furans in both TRI and NPRI reveal that a handful of facilities is responsible for the majority of air releases. In the NPRI, the top ten facilities are responsible for one-half of the total dioxins and furans released to the air (in grams-iTEQ) and, in TRI, the top ten facilities are responsible for over half of the total air releases (in grams).

Levels of dioxins and furans and exposures in North America

Children's exposure to dioxins can come from a variety of sources, including food, such as fish, *in utero* exposure or via breast milk, and from proximity to a contaminated site or hazardous waste facility. Foods that are high in fats, such as beef, pork, dairy products, fish and breast milk, tend to have higher concentrations of dioxins and furans.

Canadian exposure estimates indicate that breastfed infants under six months of age in the Great Lakes region are likely to be exposed to almost six times the tolerable daily intake (TDI) of dioxins (10 picograms-iTEQ/kg of body weight/day for dioxins) (WHO 1998). By comparison, the average adult 20 years of age or older, in this region, takes in only 12 percent of the TDI for dioxin (Haines *et al.* 1998a). 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 picograms/kg of body weight/day (WHO 1998).

Canada has monitored breast milk levels of a number of persistent organic pollutants over the years and has generally found a downward trend. The concentration of dioxins in breast milk is considered an indicator of population exposure to these contaminants by Health Canada (1998a) and is also relevant to determining the exposure of breastfed infants. Breast milk dioxin/furan levels indicate that exposure is relatively uniform geographically

for the general Canadian population. The United States and Mexico do not have such a breast milk monitoring strategy.

The Commission for Environmental Cooperation is developing a draft North American Regional Action Plan on dioxins and furans and hexachlorobenzene as a basis for cooperative work among Canada, Mexico and the United States to improve capacities to reduce exposure of the public and the environment to these substances. See ‹http://www.cec.org/pubs_docs/documents/index.cfm?varlan=english&ID=1220›.

3.5 Emerging Issues

As new chemicals are identified with potential for health hazards for reproduction and/or development, it is important to consider whether PRTRs are capturing information about releases of these substances and how such information can be used to address such emerging issues. In this report, we address two such substances: phthalate esters and manganese.

3.5.1 Phthalate Esters

Uses of phthalates

Phthalates are a class of chemicals widely used to make plastics soft and flexible, and so are found in a broad variety of products (**Table 3-20**). Two phthalate compounds, DEHP (di(2-ethylhexyl) phthalate) and DINP (di-isononyl phthalate) were recently (voluntarily) removed from pacifiers, nipples, and teething toys in Europe and the United States. In Canada (Page and Lacroix 1995) and the United States, a quantity of phthalates is allowed as indirect additives to food via migration from food packages.

Health effects of phthalate esters

Phthalate esters are generally of concern because they are endocrine disruptors in the laboratory and some have demonstrated developmental and reproductive toxicity and cancer risk.

There are seven phthalate compounds that have been evaluated by the US National Toxicology Program (NTP) as developmental and reproductive toxicants. (See **Table 3-20**, which summarizes the chemical names and toxicology data for these.) There are very few studies on humans and there is uncertainty about critical levels of exposure for children. Most phthalates are toxic to the "nurse cells" that nurture developing sperm in laboratory animals and thus are associated with lower sperm counts. In 2000, higher phthalate levels were reported to be associated with early breast development in adolescent girls in Puerto Rico but this finding is yet to be confirmed (Colon *et al.* 2000) and has been disputed (McKee 2004). One study indicated that phthalates in house dust may be associated with increased rates of asthma and allergic symptoms, implying an immune system toxicity; however, this has yet to be confirmed (Bornehag *et al.* 2004). More recently it was reported that prenatal phthalate exposures may have subtle effects on male sexual development, a reduction in the "anogenital distance" (Swan *et al.* 2005).

What can PRTR data tell us about releases and transfers of phthalate esters?

PRTR data provide information on phthalate ester releases and transfers from larger industrial sources and other facilities. As noted above, children may also be exposed to phthalates from a number of other sources. We do not know at this point in time whether localized "point" sources of phthalate release make an important contribution to children's exposure.

Only two of the seven phthalates referenced in **Table 3-20** are included in the matched PRTR—di-n-butyl phthalate (DBP) and di(2-ethylhexyl) phthalate (DEHP)—and can be seen in the matched TRI and NPRI data for 2002. DEHP is identified as a carcinogen (classified as "can reasonably be anticipated to be a human carcinogen" by the NTP and both are recognized as developmental and reproductive toxicants. Although the other five phthalates are not listed, a recent NTP review indicated that these compounds have much in common in terms of toxicity (Kavlock *et al.* 2002a–g). However, they have not been assessed to the same extent.

A total of 6,597 tonnes of these two phthalates was released and transferred in Canada and the US in 2002 (**Table 3-21**). Almost two-thirds (4,298 tonnes) of this total amount was DEHP, found in waste sent for burning for energy recovery. Total releases were 610 tonnes, with almost 139 tonnes of the phthalates released into the air.

The rubber and plastics industry reported the largest releases, including the largest air releases and off-site releases (mainly transfers to disposal) in 2002 (**Table 3-22**). The hazardous waste management sector reported the largest transfers of the phthalates in waste to be burned for energy recovery.

Three sectors in Canada and the United States released (on- and off-site) the largest amounts of the phthalates in 2002:

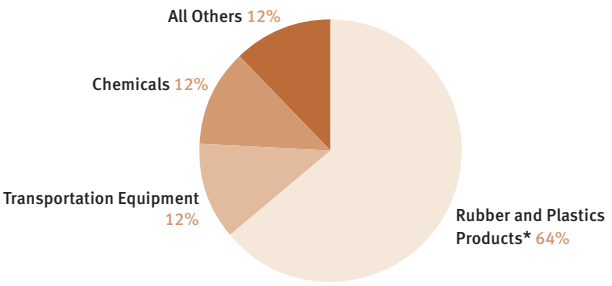
- Rubber and plastics products (398 tonnes)
- Chemical manufacturers (97 tonnes)
- Transportation equipment (41 tonnes)

The rubber and plastics products sector was also the sector with the largest air releases, accounting for 64 percent of the total in 2002. Transportation equipment manufacturers and chemical manufacturers each accounted for 12 percent (**Figure 3-14**).

From 1998 to 2002, total releases (on- and off-site) of the two phthalates, DBP and DEHP, decreased by 28 percent in Canada and the United States. Air releases of the phthalates, however, increased by 11 percent (14 tonnes) over that same time period (**Figure 3-15**). On-site land releases and off-site releases (transfers to disposal) both decreased, as did injection into underground wells. These statistics do not, in and of themselves, provide information about whether there were pollution prevention efforts, switching among phthalates, or some combination of the two.

Figure 3-14 **Industrial Sectors with the Largest Air Releases of Phthalates, 2002**

(2002 Matched Chemicals and Industries)



Total On-site Air Releases: 139 tonnes

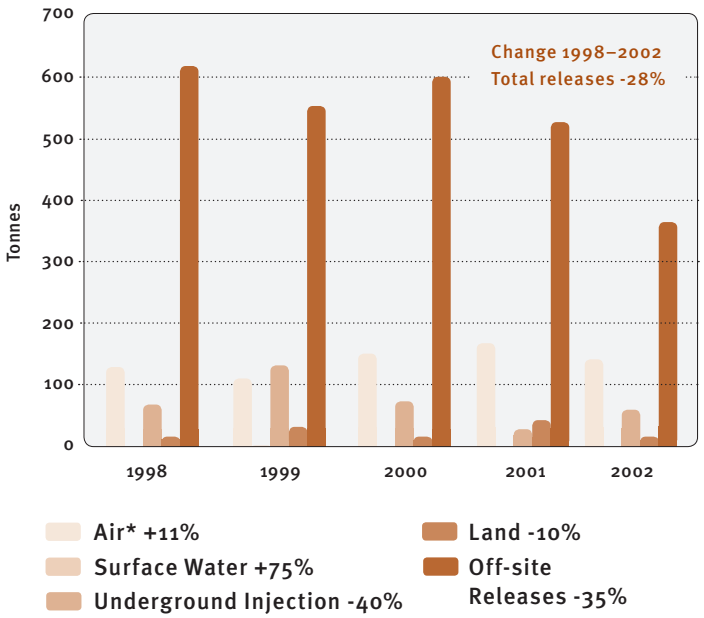
Note: Canada and US data only. Mexico data not available for 2002. *** One TRI facility in the rubber and plastics industry reported incorrect air releases of di(2-ethylhexyl) phthalate for 2002. The correct amount is shown in the figures in this section of the report but was received too late to be included in other sections of the report.**

Phthalate esters levels and exposures in North America

At this time, information about the relative importance of various potential sources of phthalate exposure is unclear (Kohn *et al.* 2000). In addition to the known presence of phthalates in cosmetics and various industrial products (see **Table 3-20**), there also is some migration from food packaging into food (Page and Lacroix 1995). A recent small study of young children found levels of two phthalates (DEHP and butyl benzyl phthalate, or BBP) in indoor and outdoor air, solid foods and on children's hands (Sexton *et al.* 2000). The NTP review of phthalates concluded that the most highly exposed population is likely to be very small newborn babies who receive extensive medical therapy through blood tubing that contains the plasticizer DEHP (for example, exchange transfusions) (Kavlock *et al.* 2002b). In 2000, the US Centers for Disease Control and Prevention (CDC) reported higher exposures (through measure of urine samples of the US population) than had been suspected in women of childbearing age; CDC and others have speculated that cosmetics should be suspected as being among the sources of exposure to women (CDC 2003c). More complete analysis of these nationally representative data indicates that in the United States, patterns of exposure differ by age and gender. Children had significantly higher levels of the metabolites than did adults for: DBP, di-isobutyl phthalate (DIBP), butyl benzyl phthalate (BBP), and DEHP, since levels of urinary metabolites are strongly associated with levels in blood. Of concern for potential *in utero* exposure is that females had significantly higher concentrations of DEHP and BBP than did males.

Figure 3-15 **Releases (On- and Off-site) of Phthalates in North America, 1998–2002**

(1998–2002 Matched Chemicals and Industries)



Note: Canada and US data only. Mexico data not available for 1998–2002. *** One TRI facility reported incorrect air releases of di(2-ethylhexyl) phthalate for 2002. The correct amount is shown in the figures in this section of the report but was received too late to be included in other sections of the report.**

3.5.2 Manganese and its Compounds

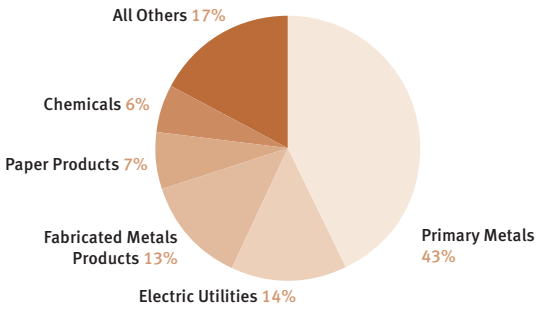
Uses of manganese

Manganese is an abundant mineral in the earth's crust. It is the fourth most widely used metal in the world; 95 percent of manganese production is used to make steel. Other industrial uses of manganese include: welding, manufacture of fungicides, dry alkaline battery manufacturing, manufacture of MMT (methylcyclopentyldienyl manganese tricarbonyl) gasoline additive, and many other uses ranging from catalysts to pigments. MMT gasoline additive has been the most controversial use in North America with, at various times, the United States and Canada making efforts to assess health risks.

Health effects of manganese

Manganese is also an essential trace element in the diet that plays a critical role in many biochemical functions of the body. It is present in the diet in grains, teas and leafy vegetables. In 2002, Health Canada convened a meeting of experts to examine questions of manganese neurotoxicity. At that time it concluded that the strongest epidemiologic evidence for toxicity of manganese was from studies of manganese inhalation exposures in occupational settings. Excessive levels of exposure in occupational settings have been associated with adverse health effects, mainly neurotoxicity (Levy and Nassetta 2003). Manganese has not been well studied, but one study, conducted in Canada, found that excessive manganese exposure was associated with decreased intellectual developmental among young children (Takser *et al.* 2003) but this study has not yet been replicated by other investigators. Manganese has not yet been considered for listing by any authorities for developmental toxicity and has not been identified as a carcinogen.

Figure 3-16 Industrial Sectors with the Largest Air Releases of Manganese and its Compounds, 2002
(2002 Matched Chemicals and Industries)



Total Air Releases On-site: 1,437 tonnes

Note: Canada and US data only. Mexico data not available for 2002.

What can PRTR data tell us about releases and transfers of manganese and its compounds?

PRTR data provide information on manganese releases and transfers from larger industrial sources and other facilities. Children may also be exposed to manganese and its compounds from a number of other sources, such as from air emissions from mobile sources of manganese, which are not reported to the PRTR. The pattern or routes of manganese exposure is unknown but one may use lead as a reasonable model. In the case of manganese, it is likely that where manganese is used as a gasoline additive, mobile sources would be a major contributor to general levels of manganese exposure of the population. However, according to the US ATSDR, communities near facilities with releases of manganese are expected to have higher levels as well. PRTR data may provide valuable information to investigators who may wish to explore this issue further by identifying such communities and assessing their manganese exposure levels.

Based on the matched TRI and NPRI data for 2002, 191,700 tonnes of manganese and its compounds were released and transferred in 2002 (**Table 3-23**). Almost 44 percent (84,200 tonnes) of this total amount was released on-site, including 1,400 tonnes released into the air.

The three sectors in Canada and the US that released (on- and off-site) the largest amounts of manganese and its compounds in 2002 were (**Table 3-24**):

- Primary metals (58,200 tonnes)
- Chemical manufacturers (20,300 tonnes)
- Electric utilities (power plants burning oil and/or coal) (19,400 tonnes)

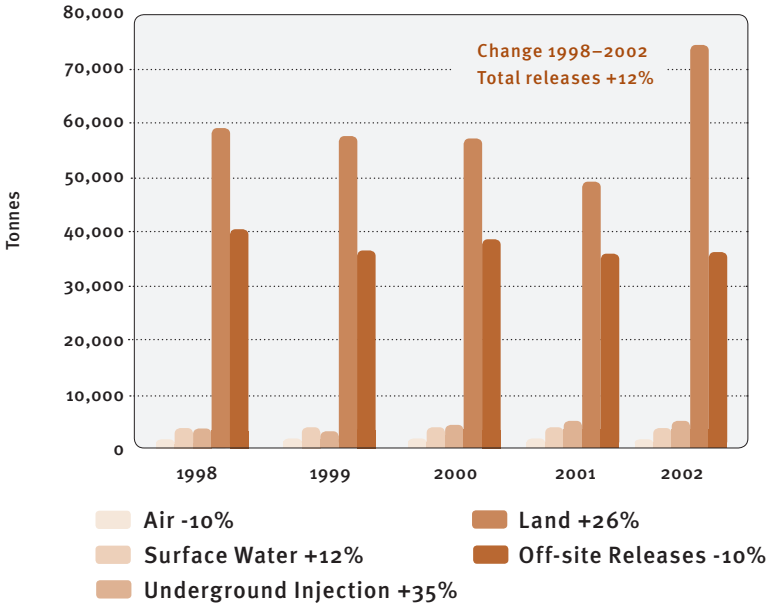
The primary metals sector was also the sector with the largest air releases, accounting for 43 percent of the total in 2002. Electric utilities accounted for 14 percent of air releases and the fabricated metals products sector accounted for 13 percent in 2002 (**Figure 3-16**).

From 1998 to 2002, total releases (on- and off-site) of manganese and its compounds increased by 12 percent in Canada and the US. This was due to an increase reported by one facility, BHP Copper in San Manuel, Arizona. This facility reported that it had a one-time amount of on-site land disposal due to discontinued operations related to mining. Without including the almost 27 tonnes of on-site land releases from this facility in 2002, total releases of manganese and its compounds decreased by 10 percent. Air releases decreased by 10 percent from 1998 to 2002 (**Figure 3-17**). The decrease in releases over time is similar to that observed for other chemicals. The data indicate that some facilities are making efforts to decrease their releases of manganese to the environment.

Manganese levels and exposures in North America

A pilot study in central Mexico found higher exposure levels among persons who lived in proximity to facilities that release manganese, as well as an association between manganese and lead exposures (Santos-Burgoa *et al.* 2001). MMT has been used as a fuel additive in Canada but not much is known about exposure levels. One study demonstrated that manganese in outdoor air is higher in urban than rural areas. The study also found an increase in blood manganese in five persons living in the urban area compared to five rural residents; it was not considered to be a significant difference (Bolte *et al.* 2004). Clearly, more information about manganese exposure levels in North America, as related to both children and the general population, is needed.

Figure 3-17 Releases (On- and Off-site) of Manganese and its Compounds in North America, 1998–2002
(1998–2002 Matched Chemicals and Industries)



Note: Canada and US data only. Mexico data not available for 1998–2002.

4 Actions to Protect Children's Health from Toxic Chemicals

4.1 Overview

Given the need to protect children from environmental hazards, the challenge before us is to intervene whenever possible to prevent or reduce adverse health effects, including those related to exposure to toxic chemicals.

As illustrated by PRTR data, we have made some significant progress over recent decades to reduce releases of toxic chemicals from industrial activities. For example, releases of many carcinogens, neurotoxicants and developmental toxicants to the air from industrial sources decreased from 1995 to 2002. However, there is still much progress to be made.

Actions are underway at the national, regional and international level to improve the environments of children in North America. Each nation has a number of regulations and programs that occur on the national and/or state/provincial levels that will help protect children's health from toxic chemicals. Actions to reduce amounts of toxic substances released into the environment also occur at the local and individual levels. Each of us has an important role to play in the effort to help protect children's health from toxic chemicals.

4.2 International Action to Reduce Children's Exposure to Toxic Chemicals

In the past decade, children's health and the environment has become increasingly more prominent on the international agenda. Several important conventions and agreements have been signed, such as the UN Convention on the Rights of the Child (1989), the Declaration of the Environment Leaders of the Eight on Children's Environmental Health (G7 countries and Russia, 1997), and the Declaration of the Third European Ministerial Conference on Environment and Health (WHO European Delegation, 1999).

The reduction of toxic chemicals into the environment has also become the subject of several international agreements: the Basel Convention on the Transboundary Movement of Hazardous Wastes and Their Disposal, the Montreal Protocol on Ozone-depleting Chemicals, the Convention on Long-range Transboundary Air Pollution, and the Stockholm Convention on Persistent Organic Pollutants. Most recently, the United Nations Environment Programme has agreed to a global goal of phasing lead out of gasoline. It also has completed a global assessment of mercury and is embarking on a series of reduction/elimination partnerships for mercury in areas such as chlor-alkali facilities, artisanal mining, and products.

4.3 National and Trilateral Action to Reduce Children's Exposure to Toxic Chemicals

Across North America, each nation has regulations and programs that will help to protect children's health from toxic chemicals. Although not all are geared specifically to children, many risk reduction programs geared to the general population will benefit children. The details are too numerous to provide in this report, but links to government web sites are available in **Appendix E**.

Action is also being taken at the trinational level. Canada, Mexico and the United States, through the CEC's Sound Management of Chemical (SMOC) initiative, have developed North American Regional Action Plans (NARAPs) for a series of chemicals important to children's health (examples mentioned in Chapter 3 concern mercury, PCBs, and dioxins and furans). Through NARAPs, the three countries have committed themselves to taking specific concrete steps that will reduce these chemicals in the North American environment. In addition to the new NARAPs for lindane, dioxins, furans and hexachlorobenzene, lead is under consideration for future action under SMOC.

The CEC has convened a trilateral community of people interested in the linkages between children's health and the environment. To build the foundation for this initiative, a trilateral symposium on children's health and the environment was held in May 2000, and a background document, entitled *Making the Environment Healthier for Our Kids: An Overview of Environmental Challenges to the Health of North America's Children* (CEC 2002), was developed. These steps formed part of the discussions leading up to the CEC Council's adoption of the Cooperative Agenda for Children's Health and the Environment in

International Action on Persistent Organic Pollutants

Some man-made organic chemicals are slow to break down in the environment. These chemicals are known as persistent organic pollutants (POPs). POPs can travel long distances from their sources. Detectable levels of some of these chemicals, such as DDT, PCBs, and dioxins and furans, can be found in all of our bodies. The chemicals can be passed from one generation to the next through breast milk or placental blood. Several of these chemicals are neurotoxicants and suspected endocrine disruptors.

Faced with the widespread, persistent and toxic nature of these chemicals, over 150 countries, including Canada, Mexico and the United States, have signed the Stockholm Convention on POPs; the Convention became a part of international law on 17 May 2004. The Convention seeks the elimination or phase-out of POPs, with an initial focus on 12 chemicals: aldrin, chlordane, dieldrin, endrin, heptachlor, hexachlorobenzene, mirex, toxaphene, PCBs, DDT, and dioxins and furans. The treaty also encourages cleanup of chemical stockpiles that include POPs. Discussions for the implementation are ongoing. More information about the Stockholm treaty can be found at **‹http://www.pops.int›**. For more information on POPs, see **‹www.chem.unep.ch/pops/›**.

North America in 2002 (Council Resolution 02-06). The initial focus of the Cooperative Agenda was on asthma and other respiratory diseases, the effects of lead, and the effects of exposure to other toxic chemicals. Council added a third priority theme—waterborne diseases—in 2002.

4.4 Looking Forward: Actions to Reduce Toxic Chemicals and Protect Children's Health

The following section provides an overview of some of the types of actions that are being undertaken or are recommended for consideration at different governmental levels. These fall into the following general areas:

1. Monitor and Reduce Releases of Toxic Chemicals to the Environment
2. Monitor and Reduce Exposures to Toxic Chemicals
3. Track Childhood Diseases that May Be Related to the Environment
4. Improve Scientific Knowledge
5. Increase Awareness of the Role of Toxic Chemicals in Children's Health

1. Monitor and Reduce Releases of Toxic Chemicals to the Environment

Preventing or reducing toxic pollution at the source is the best way to ensure that such substances are not released to the environment and do not contaminate the environments of children nor adversely affect their health. Reducing releases can reduce contaminants that children receive from air, water, soil, breast milk, food or *in utero*. A broad range of programs, regulations and actions are intended to reduce releases of chemicals. Traditionally, these programs have either focused on reducing emissions from a specific chemical, from a specific source or to a specific regional area. For an overview of some of these programs, please see Environment Canada's web site at ‹http://www.ec.gc.ca/›, Semarnat's web site at ‹www.semarnat.gob.mx› and the US EPA's web site at ‹www.epa.gov/ttn/airtoxics›.

PRTRs provide information on a number of specific chemicals, industrial sectors and industrial facilities that could be targeted for further reductions of releases. For example, such carcinogens as styrene, dichloromethane, formaldehyde, acetaldehyde and trichloroethylene are released into the air in large quantities in North America, often from the chemical manufacturing, primary metals and electronics sectors. Developmental toxicants and neurotoxicants such as methanol, toluene, hydrogen fluoride and xylenes are released to the air from chemical manufacturing and primary metals production.

It is currently difficult to compile comparable information on sources and amounts of chemical pollutants or ambient concentrations in Canada, Mexico and the United States. Often data are missing or not available to the public. A compounding difficulty is that data often are not directly comparable. For example, data collected using different methods, different reporting thresholds, different time periods or measurement units make comparison difficult. Also, different countries have used different legislation and regulations to compel the reporting of the information. PRTR data can help bridge some of these gaps, especially as reporting under the Mexican program comes online. Other chemical inventories are also being compiled on mercury, dioxins and furans, which will help answer some questions

Working Toward an Improved Picture of North American Releases and Transfers

The three North American national governments have committed themselves to work together to increase the comparability of PRTR data. The *Action Plan to Enhance the Comparability of Pollutant Release and Transfer Registers in North America* provides a framework for the three countries to address and harmonize regulatory requirements that will result in an improved picture of contaminants in North America. The anticipated availability of mandatory data from Mexico's PRTR will be a major step forward. Progress has been made in improving comparability between the US and Canadian PRTRs, including an expanded number of sectors that report in both countries, and a coordinated lowering of reporting thresholds under TRI and NPRI that has improved reporting for substances of concern to children's health such as mercury and lead.

about children's potential exposures. Regional criteria air contaminant inventories in Mexico are increasing in number, and permit a greater understanding of children's potential exposures to chemicals associated with smog and respiratory diseases. Putting together these national and regional inventories will help provide a better picture of releases and environmental levels of chemicals throughout the continent.

Information on pesticides, while limited under current PRTR systems, can be obtained through other programs. For example, through NAFTA, there is increasing harmonization of pesticide reviews and regulatory processes. While the United States already has programs in place to capture information on pesticide use and sales, Canada has revised its national pesticide law and the new legislation contains a number of measures that increase the amount of publicly available information about pesticide sales, use, concentrations, poisonings and exposure. This legislation may help to protect children's health and serve as a model for other countries.

Recommendation 1a: Consideration of children's health should be among the factors that guide the interpretation of PRTR data in order to identify priorities for emissions reduction and pollution prevention. For example, while emissions of lead, a carcinogen, neurotoxicant and development toxicant, from industrial facilities decreased by 19 percent from 1998 to 2000, three facilities in North America, all smelters, released large amounts of lead into the air in 2002. While some facilities have made progress in reducing these emissions, others have not. While the toxicity potency factors do refocus attention on the releases that have the greatest potential for harm, Canada, Mexico and the United States should work together to refine these factors and make them more useful on a North American basis.

Recommendation 1b: PRTR facility reporting and chemicals data need to be combined with other data from monitoring studies, including biomonitoring studies, to provide a more complete picture of children's potential exposures to chemicals from area sources, mobile sources and natural sources in order to establish priorities for action.

Recommendation 1c: PRTR reporting in North America could be enhanced to improve information on key chemicals. Some of the actions needed to improve PRTR efforts within North America include the following:

- Expand the PRTR coverage in North America to give a fuller picture of sources and amounts of industrial chemicals of potential concern for children's health.
- Fully implement the provisions of the CEC Action Plan to Enhance the Comparability of Pollutant Release and Transfer Registers, especially where those provisions can increase the information relevant to children's health available on a North American basis.
- Consider the use of the toxicity weightings, such as those employed in this report, to give a clearer picture of total hazard potential (as opposed to total quantities of releases and transfers of chemical). Also needed is to fill data gaps for key hazard and exposure parameters that are essential to the use of such a weighting system.

Recommendation 1d: National reporting systems for pesticides should be developed. Several alternatives now exist on the federal and state levels in the United States, including pesticide use reporting, pesticide illness reporting and pesticide use surveys. An effort should be initiated to develop a North American approach to national reporting for pesticides that systematically collects this information.

2. Monitor and Reduce Exposures to Toxic Chemicals

Throughout North America, more complete and improved information on hazards and exposures is needed so that we can better assess environmental risks to children. Biomonitoring data on contaminant levels in humans are invaluable for increasing our understanding of exposures and potential links to health. They reflect the amount of chemicals to which a person has been exposed, providing a complement to PRTRs. The generally scanty data now available on levels of contaminants in human cord blood, breast milk and children's bodies make it difficult to obtain a picture of current levels of contaminant burdens in children in North America, hindering exploration of the connections between these levels, the sources of the chemicals and diseases. Biomonitoring data have the potential to provide valuable information for both research and policymaking, about preventing or reducing children's exposures to environmental chemicals, and about the levels at which decisions on public health issues must be made.

Another important source of information about exposure is obtained via monitoring of levels of toxic substances through air, water, soil/dust, food, breast milk, and consumer products. We know that exposures can occur in the environments where children live, play and learn. The challenge is to monitor such exposures and take steps to reduce or prevent those exposures, where appropriate.

Approaches to reducing risks to children need to take into account the diversity of environments in which children find themselves across the continent. For example, the use of biomass fuels for home heating and cooking is exposing children in many homes in Mexico to unacceptable levels of indoor air pollution, including dioxins. Elsewhere, children of Native American/Indian/Indigenous origin may be at elevated risk because of traditional practices like fishing in areas that have become contaminated, sometimes from persistent, toxic compounds that originate from faraway sources.

Recommendation 2a: Expansion of biomonitoring and efforts relevant to the health of children in the North American continent would help our understanding of exposures. Some specific actions that should be undertaken include:

- Trilateral biomonitoring activities under the NARAP on environmental monitoring and assessment should

Biomonitoring

Biomonitoring is a direct measurement of environmental chemicals or their markers (e.g., byproducts of metabolic reactions) in human tissue. Samples are frequently taken from blood or urine specimens (CDC 2005b).

In 2001, the United States CDC National Center for Environmental Health began a biomonitoring program in conjunction with the National Health and Nutrition and Environment Survey (NHANES) to track levels of chemicals in people over time. Results from this program are published in the *Third National Report of Human Exposure to Environmental Chemicals*. This program is examining many of the same chemicals that are monitored by PRTRs. Although biomonitoring information has been collected in individual research studies, this is the first time that a population-based biomonitoring program has been conducted. As such, it could provide valuable experience and lessons learned for Canada and Mexico in the development of their own biomonitoring programs.

More information about biomonitoring can be found at **‹http://www.cdc.gov/biomonitoring›**.

continue in order to generate comparable information for Canada, Mexico and the United States, particularly for exposures relevant to children's health.

- The United States should continue to expand its efforts in the area of biomonitoring and environmental public health tracking, particularly with regard to children's exposures.

Recommendation 2b: Canada, Mexico, and the United States should continue to work together under the North American Regional Action Plan (NARAP) on environmental monitoring and assessment and other mechanisms to monitor toxic contaminants in ambient air, water and soil in North America and increase the cooperative analysis of these results.

Recommendation 2c: As a matter of priority, wherever excessive exposures are found, actions should be taken to protect the populations of North America, especially children. Generally, exposure assessments point to the need to: prevent exposures via maternal and paternal pathways; ensure a clean and safe food and water supply; ensure good air quality both indoors and outdoors, and minimize contamination from consumer products. Although most of these actions need to be taken at the societal level, in some cases governments can provide communities and families with the information and increased awareness they need to better protect their children from exposures and potential risks.

3. Track Childhood Diseases that May Be Related to the Environment

Currently, it is difficult to compare disease and mortality in children. Methods and time frames for data collection and analysis differ. The lack of a comprehensive disease-tracking system handicaps exploring the connections between diseases and environmental exposures. In particular, uniform standards and methods for dealing with morbidity and mortality would increase comparability of data and provide a clearer picture of the health status of children across the entire continent.

Recommendation 3a: The United States should continue and expand its efforts in the area of environmental public health tracking of childhood diseases and other health outcomes that potentially are related to the environment. Likewise, efforts should begin to create and coordinate tools to track relevant health outcomes in children in Canada, Mexico and the United States.

Recommendation 3b: North American nations should strengthen scientific and medical networks to facilitate knowledge dissemination and information exchange about linkages between the environment and children's health.

4. Improve Scientific Knowledge

The United States has demonstrated leadership in research efforts in children's health and the environment. It has established a number of Centers of Excellence in Children's Environmental Health and Disease Prevention Research, funded by the National Institute of Environmental Health Sciences and the US EPA. These centers are producing important research that will help inform decisions about reducing children's risks in the future. The United States also has taken the first steps forward toward the establishment of a national longitudinal study of children's health and the environment called the National Children's Study. As of September 2005, the US government has announced the awarding of the first Vanguard Centers for initiating this study (see **text box**).

The US National Toxicology Program has begun a process of formally assessing the potential for hazard to children, the Center for the Evaluation of Risks to Human Reproduction. To date, this Center has evaluated risks of phthalate derivatives (seven have major industrial uses), methanol, acrylamide, 1-bromopropane and 2-bromopropane, ethylene glycol and propylene glycol, 2-ethylhexanoic acid, and fluoxetine hydrochloride. Reviews are scheduled for styrene, amphetamines, methylphenidate, genistein, and soy formula.

In Mexico, several cohort studies of children are underway, funded by the US National Institute of Health and the Mexican government, under the leadership of Mexico's *Instituto Nacional de Salud Pública* (National Institute of Public Health—INSP). Potentially, these studies could be expanded to provide comparable methods to the National Children's Study.

New Knowledge on the Horizon

The US is designing a major study of children's environmental health and safety called the National Children's Study. As currently proposed, the study would enroll 100,000 children while still *in utero* and assess short- and long-term impacts of prenatal and early childhood risk factors. The US National Children's Study could potentially serve as a basis or starting point for continent-wide, coordinated research. With the CEC having played a role in convening initial meetings about this issue, Canada, Mexico and the US now are exploring possibilities in this regard. Both Canada and Mexico have participated in the international interest group associated with the US National Children's Study.

In November 2004, the National Children's Study released a Study Plan, which outlines the objectives, methodologies and measures related to the first years of the study. Additionally, 98 locations across the United States were identified from which eligible participants will be recruited and enrolled into the study. If work continues according to schedule, preliminary results from the first few years of the study will be available in 2008–2009.

Additional information and updates on the study's progress can be found at the National Children's Study web site at **‹http://www.nationalchildrensstudy.gov/›**. Interested individuals can sign up at this web site to receive periodic e-mail updates about the study's progress. The National Children's Study also has an International Interest Group, allowing for investigators worldwide to exchange information on study design and research results.

Recommendation 4a: Although much effort already is underway, expansion of current research efforts, as well as increased coordination and cooperation, would accelerate the process of identifying the factors in children's environments that are adverse to health and development, and those that are not.

Recommendation 4b: Efforts such as the US National Toxicology Program Center for the Evaluation of Reproduction and Health Risks need to be expanded and to involve scientists from across North America in order to provide more credible, definitive information about which hazards are important to children's health. Having governments make such assessment efforts (as well as scientific analysis) a priority would benefit all citizens of North America.

Recommendation 4c: In particular, Canada and Mexico cooperation on the US National Children's Study is an opportunity for a continent-wide longitudinal study of children, one that would provide an unprecedented wealth of information about the trajectory of the development of children from diverse environments in North America.

5. Increase Awareness of the Role of Toxic Chemicals in Children's Health

Governments, health care providers, parents, teachers, relatives, and neighbors all have a role to play in advising on measures to reduce a child's exposure to toxic chemicals.

Recommendation 5: Governments and others should help to build individual and community awareness of possible sources and pathways of chemicals to children, and the potential for chemicals to harm children. When provided appropriate information, parents and others in the community can take practical actions to reduce potential exposures to chemicals.

Resources

A number of organizations can provide useful information. For information about emissions from mobile, area and other sources in your community, see:

Canada

- Environment Canada's emission inventories, at ‹http://www.ec.gc.ca/pdb/ape/cape_home_e.cfm› or general information, at ‹http://www.ec.gc.ca/pdb/npri/npri_links_e.cfm - ECInv›.

Mexico

- National information, at ‹www.semarnat.gob.mx›.
- Mexico City emission inventories, at ‹www.sma.df.gob.mx/menu.htm›.

United States

- For air toxics, see the National Air Toxics Assessment, at ‹http://www.epa.gov/ttn/atw/nata/›. For criteria air contaminants, see the National Emission Inventory, at ‹http://www.epa.gov/ttn/chief/eiinformation.html›.

For information about practical steps to reduce your child's exposure to chemicals, see:

- Children's Health Environmental Coalition. 2002. *The State of Children's Health and Environment 2002.* See especially Chapter 6: Guidelines for parents and those who manage children's environments, available at: ‹http://www.checnet.org/prodres_sche_enews.asp›.
- A variety of suggestions from the Children's Health Environmental Coalition Healthy House, including "How to Create Better Breathing Space for Asthmatics": ‹http://www.checnet.org/healthehouse/education/top10-detail.asp?Top10_Cat_ID=14›.
- American Academy of Pediatrics. 2003. *Handbook of Pediatric Environmental Health.* See: ‹http://www.aap.org/bst/showdetl.cfm?&DID=15&Product_ID=1697&CatID=132›.

For information about how to watch and monitor for health effects or changes in the environment which could increase exposure:

- You can subscribe to the Children's Health Environmental Coalition (CHEC) Health-eNews, which is sent twice a month, by signing up, at ‹http://checnet.forms.soceco.org/47/›.
- You can learn about emerging research from the Children's Environmental Health Network, at ‹http://www.cehn.org/cehn/About.html - listserv›.

For general information about children's health and the environment:

- **Health Canada's** Office of Children's Environmental Health serves as a focal point within Health Canada to focus on the special sensitivity of children to environmental exposures. With national and international partners, the Office of Children's Environmental Health (within Health Canada) aims to monitor and analyze scientific evidence regarding environmental exposures and children's health; identify knowledge gaps in this area and sponsor research to address the gaps; coordinate policy and strategies to reduce environmental health threats to children; and develop public education materials on means to reduce environmental health threats to children. For more information, see: ‹http://www.hc-sc.gc.ca/hecs-sesc/oceh/index.htm›.

- **Environment Canada** at ‹www.ec.gc.ca›.
- The Children's Health project of the Canadian Environmental Law Association, at ‹http://www.cela.ca/›.
- The Canadian Institute of Child Health, at ‹http://www.cich.ca/›.
- Canadian Partnership for Children's Health and Environment, at ‹www.healthyenvironmentforkids.ca›.
- Pollution Probe, at ‹www.pollutionprobe.org/›.
- The Canadian Health Network: ‹http://www.canadian-health-network.ca/›.

- **Mexico** has a pediatric environmental health unit within its Department of Environmental Health at the National Institute of Public Health. For more information, see: ‹http://insp.mx/pehsu›.

- **The Presidential Task Force on Environmental Health Risks and Safety Risks to Children**, which involves 16 departments and White House offices, was established by Presidential Executive Order #13045 (1997). The Executive Order recognized the importance of children's environmental health, and directed US governmental agencies to make children's environmental health a high priority. For more information, see: ‹http://yosemite.epa.gov/ochp/ochpweb.nsf/content/Whatwe_fedtask.htm›.

- **EPA's Office of Children's Health Protection** performs several activities related to children's environmental health. Its goals are to provide information on children's environmental health to the general public, support community actions to protect children, increase the ability of health care providers to identify, prevent and reduce environmental threats to children, and work with states to develop programs to address children's environmental health issues. For more information, see: ‹http://yosemite.epa.gov/ochp/ochpweb.nsf/homepage›.

- **CDC NHANES** continues work on biomonitoring, cancer and adverse reproductive outcome registries

- The **Agency for Toxic Substances and Disease Registry** (ATSDR), within the CDC, has a child health program that "emphasizes the ongoing examination of relevant child health issues in all of the agency's activities, and stimulates new projects to benefit children," at ‹http://www.atsdr.cdc.gov/child/ochchildhlth.html›. In addition, the ATSDR web site has a page with links to ATSDR "partners" doing work in children's environmental health, at ‹http://www.atsdr.cdc.gov/child/atsdrochpartners.html›.

- **National Institute of Environmental Health Sciences (NIEHS)**, particularly:
  - Children's Environmental Health Research published in *Environmental Health Perspectives*. For more information, see ‹http://ehp.niehs.nih.gov/children/›.
  - Center for the Evaluation of Risks to Human Reproduction. For more information, see ‹http:///cerhr.niehs.nih.gov›.
  - Children's Environmental Health Research Initiative. For more information, see ‹http://www.niehs.nih.gov/external/resinits/ri-28.htm›.

- Other nongovernmental organizations in the United States:
  - American Academy of Pediatrics,
  - Physicians for Social Responsibility, at ‹www.psr.org/›.
  - Children's Health Environmental Coalition, at ‹www.checnet.org›.
  - Children's Environmental Health Network, at ‹http://www.cehn.org/›.
  - Learning Disabilities Association of America, at ‹http://www.LDAAmerica.org/›.
  - The Center for Children's Health and the Environment, at ‹www.Childenvironment.org›.
  - Partnership for Children's Health and the Environment, at ‹http://www.partnersforchildren.org/›.

For an online directory of **children's environment organizations and links**, see:

- World Health Organization — Children's Environmental Health, at ‹http://www.who.int/ceh/en›.
- The Canadian Institute of Child Health, at ‹http://www.cich.ca/›.
- The Resource Guide on Children's Environmental Health, at ‹http://www.cehn.org/cehn/resourceguide/organizations.html›.

References

–. 1991. Mortality due to intestinal infectious diseases in Latin America and the Caribbean, 1965–1990. *Epidemiological Bulletin* 12(3): 1–6.

Agency for Toxic Substances and Disease Registry (ATSDR). 2000. Toxicological profile for polychlorinated biphenyls (PCBs). Atlanta, GA: US Department of Health and Human Services, Public Health Service.

Aguilar-Garduno C, Lacasana M, Tellez-Rojo MM, Aguilar-Madrid G, Sanin-Aguirre LH, Romieu I, Hernandez-Avila M. 2003. Indirect lead exposure among children of radiator repair workers. *American Journal of Industrial Medicine* 43(6): 662–67.

Albert L, Aldana P. 1982. Polychlorinated biphenyls in Mexican cereals and their packings. *Journal of Environmental Science and Health. Part B.* 17(5): 515–25.

American Academy of Pediatrics. 1999. *Handbook of Pediatric Environmental Health.* See <http://www.aap.org/bst/showdetl.cfm?&DID=15&Product_ID=1697&CatID=132>.

American College of Obstetricians and Gynecologists (ACOG). 2002. ACOG Practice Bulletin. Clinial management guidelines for obstetrician-gynecologists. Number 37, August 2002. (Replaces Practice Bulletin Number 32, November 2001). Thyroid disease in pregnancy. *Obstetrics and Gynecology* 100(2): 387–96.

Anderson LM, Diwan BA, Fear NT, Roman E. 2000. Critical windows of exposure for children's health: Cancer in human epidemiological studies and neoplasms in experimental animal models. *Environmental Health Perspectives* 108 Suppl 3: 573–94.

Arias E, *et al.* 2003. Deaths: final data for 2001. *National Vital Statistics Reports* 52(3): 1–115.

Baldwin RT, Preston-Martin S. 2004. Epidemiology of brain tumors in childhood—A review. *Toxicology & Applied Pharmacology* 199: 118–31.

Baskin LS, Himes K, Colborn T. 2001. Hypospadias and endocrine disruption: Is there a connection? *Environmental Health Perspectives* 109 (11).

Beckman DA, Brent RL. 1984. Mechanisms of teratogenesis. *Annual Review of Pharmacology and Toxicology* 24: 483–500.

Bellinger DC. 2004. What is an adverse effect? A possible resolution of clinical and epidemiological perspectives on neurobehavioral toxicity. *Environmental Research* 95: 394–405.

Bellinger D. 2005. Teratogen update: Lead and pregnancy. *Birth Defects Research (Part A)* 73: 409–420.

Birnbaum LS, Fenton SE. 2003. Cancer and developmental exposure to endocrine disruptors. *Environmental Health Perspectives* 111(4): 389–94.

Birnbaum, LS, Staskal DF, *et al.* (2003). Health effects of polybrominated dibenzo-*p*-dioxins (PBDDs) and dibenzofurans (PBDFs). *Environ Int* 29(6): 855–60.

Bishop JB, Witt KL, Sloane RA. 1997. Genetic toxicities of human teratogens. *Mutation Research* 396: 9–43.

Black RE, Morris SS, Bryce J. 2003. Where and why are 10 million children dying every year? *Lancet* 361: 2226–34.

Bolte SL, Normandin L, Kennedy G, Zayed J. 2004. Human exposure to respirable manganese in outdoor and indoor air in urban and rural areas. *Journal of Toxicology and Environmental Health. Part A.* 67(6): 459–67.

Bornehag CG, Sundell J, Weschler CJ, Sigsgaard T, Lundgren B, Hasselgren M, Hagerhed-Engman L. 2004. The association between asthma and allergic symptoms in children and phthalates in house dust: A nested case-control study. *Environmental Health Perspectives* 112(14): 1393–97.

Bradman A, Eskenazi B, Sutton P, Athanasoulis M, Goldman LR 2001. Iron deficiency associated with higher blood lead in children living in contaminated environments. *Environmental Health Perspectives* 109: 1079–84.

Brouwer A, Ahlborg UG, van Leeuwen FX, Feeley MM. 1998. Report of the WHO working group on the assessment of health risks for human infants from exposure to PCDDs PCDFs and PCBs. *Chemosphere* 37(9-12): 1627–43.

Bucher JR, Lucier G. 1998. Current approaches toward chemical mixture studies at the National Institute of Environmental Health Sciences and the US National Toxicology Program. *Environmental Health Perspectives* 106(Suppl 6): 1295–98.

Calderon J, Navarro ME, Jimenez-Capdeville ME, Santos-Diaz MA, Golden A, Rodriguez-Leyva I, Borja-Aburto V, Diaz-Barriga F. 2001. Exposure to arsenic and lead and neuropsychological development in Mexican children. *Environmental Research* 85: 69–76.

Calderon-Salinas JV, Valdez-Anaya B, Mazuniga-Charles, Albores- Medina A. 1996. Lead exposure in a population of Mexican children. *Human and Experimental Toxicology* 15: 305–311.

Caldwell JC, Woodruff TJ, Morello-Frosch R, Axelrad DA. 1998. Application of health information to hazardous air pollutants modeled in EPA's cumulative exposure project. *Toxicology and Industrial Health* 14(3): 429–54.

Calvert GM, Plate DK, Das R, Rosales R, Shafey O, Thomsen C, Male D, Beckman J, Arvizu E, Lackovic M. 2004. Acute occupational pesticide-related illness in the US, 1998–1999: Surveillance findings from the SENSOR-pesticides program. *Am J Ind Med* 45(1): 14–23.

Canadian Cancer Statistics 2002. Media release 18 April 2002. See ‹http://www.cancer.ca/cos/internet/mediareleaselist/0,3208,3172_15232_333099_langId-en,00.html›.

Canfield RL, Henderson CR Jr, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. 2003. Intellectual impairment in children with blood lead concentrations below 10 µg per deciliter. *The New England Journal of Medicine* 348(16): 1517–26. April 17.

Centers for Disease Control and Prevention (CDC). 1997. Update: Blood lead levels—United States, 1991–1994. *Morbidity and Mortality Weekly Report (MMWR)* 46(7): 141–46.

Centers for Disease Control and Prevention (CDC). 2003a. Children's Blood Lead Levels in the United States. See ‹http://www.cdc.gov/nceh/lead/lead.htm›. Atlanta.

CDC. 2003b. National Center on Birth Defects and Developmental Disabilities. ‹http://www.cdc.gov/ncbddd/adhd/what.htm›. Atlanta, GA: CDC. Updated 23 October 2003; Accessed 25 October 2003.

CDC. 2005a. Third National Report on Human Exposure to Environmental Chemicals. Atlanta, GA: Centers for Disease Control and Prevention. Available at ‹http://www.cdc.gov/exposurereport/›.

CDC. 2005b. National Biomonitoring Program. ‹http://www.cdc.gov/nceh/dls/national_biomonitoring_program.htm›. Accessed 9 February 2005.

Chaudhuri N. 1998. Child health, poverty and the environment. The Canadian context. *Canadian Journal of Public Health* 89(Suppl 1): S26-S30.

Clarkson TW. 2002. The three modern faces of mercury. *Environmental Health Perspectives* 110(Suppl 1):11-23.

Cofepris. 2005. Pesticide catalog. ‹http://www.cofepris.gob.mx›.

Colon I, Caro D, Bourdony CJ, Rosario O. 2000. Identification of phthalate esters in the serum of young Puerto Rican girls with premature breast development. *Environmental Health Perspectives* 108(9): 895-900.

Commission for Environmental Cooperation (CEC). 1996. *Status of PCB Management in North America*. Available at ‹http://www.cec.org›.

CEC. 2000. North American Regional Action Plan on Mercury. Phase II. Available at ‹http://www.cec.org›.

CEC. 2001. *Preliminary Atmospheric Emission Inventory of Mercury in Mexico.* Acosta y Asociados.

CEC. 2001. *Taking Stock 2001.* Commission for Environmental Cooperation.

CEC. 2002. *Making the Environment Healthier for Our Kids: An Overview of Environmental Challenges to the Health of North America's Children*. Available at ‹http://www.cec.org/pubs_docs/documents/index.cfm?varlan=english&ID=840›.

CEC. 2002. *Taking Stock 2002.* Commission for Envirnmenatal Cooperation

CEC. 2004a. Decision Document on Lead under the Process for Identifying Candidate Substances for Regional Action by the Sound Management of Chemicals Initiative. Commission for Environmental Cooperation, Montreal, Quebec.

CEC. 2004b. Honing the methods: Assessing population exposures to motor vehicle exhaust. Commission for Environmental Cooperation, Montreal, Quebec. Available at: ‹http://www.cec.org/files/PDF/POLLUTANTS/Honing-the-Methods_en.pdf›.

Croen LA, Grether JK, Hoogstrate J, Selvin S. 2002. The changing prevalence of autism in California. *Journal of Autism and Developmental Disorders* 32(3): 207–215.

Daston G, Faustman E, Ginsberg G, Fenner-Crisp P, Olin S, Sonawane B, Bruckner J, Breslin W, McLaughlin TJ. 2004. A framework for assessing risks to children from exposure to environmental agents. *Environmental Health Perspectives* 112: 238–56.

Davidson PW, Kost J, Myers GJ, Cox C, Clarkson TW, Shamlaye CF. 2001. Methylmercury and neurodevelopment: Reanalysis of the Seychelles Child Development Study outcomes at 66 months of age. *Journal of the American Medical Association* 285(10): 1291–93.

Denham M, Schell LM, Deane G, Gallo MV, Ravenscroft J, DeCaprio AP. 2005. Relationship of lead, mercury, mirex, dichlorodiphenyldichloroethylene, hexachlorobenzene, and polychlorinated biphenyls to timing of menarche among Akwesasne Mohawk girls. *Pediatrics* 115(2): e127–34.

Denison R. 2004. Orphan Chemicals in the HPV Challenge: A Status Report. New York, NY: Environmental Defense.

DeWailly E, Ayotte P, Bruneau S, Gingras S, Belles-Isles M, Roy R. 2000. Susceptibility to infections and immune status in Inuit infants exposed to organochlorines. *Environmental Health Perspectives* 108: 205–211.

DeWailly E, Ayotte P, Bruneau S, Lebel G, Levallois P, Weber JP. 2001. Exposure of the Inuit population of Nunavik (Arctic Quebec) to lead and mercury. *Archives of Environmental Health* 56: 350–7.

Environment Canada. 2000. *The Status of Mercury in Canada.* Report #2. Background to the CEC North American Task Force on Mercury.

Environment Canada. 2001. *National Inventory of PCBs in Use and PCB Wastes in Storage in Canada: 1996 Annual Report.* Available at ‹http://www.ec.gc.ca/ pcb/nio1/eng/for_e.htm›.

Environment Canada. 2002. Children's Environmental Health. Enviro-zine. Available at ‹http://www.ec.gc.ca/EnviroZine/›.

Federal/Provincial Committee on Environmental and Occupational Health. 1994. The Working Group on Blood Lead Intervention Levels and Strategies for the Federal/Provincial Committee on Environmental and Occupational Health. Health and Welfare Canada. September 1994.

Federal Interagency Forum on Child and Family Statistics (FIRCFS). 2001. *America's Children: Key Indicators of Well-Being.* Washington, DC: National Center for Health Statistics.

Fein GG, Jacobson JL, Jacobson SW, Schwartz PM, Dowler JK. 1984. Prenatal exposure to polychlorinated biphenyls: Effects on birth size and gestational age. *Journal of Pediatrics* 105: 315–20.

Figa-Talamanca I, Tarquini M, Lauria L. 2003. [Is it possible to use sex ratio at birth as indicator of the presence of endocrine disrupters in environmental pollution?] *Giornale Italiano di Medicina del Lavoro ed Ergonomia* 25(Suppl 3): 52–3.

Fenton SE, Hamm JT, Birnbaum LS, Youngblood GL. 2002. Persistent abnormalities in the rat mammary gland following gestational and lactational exposure to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). *Toxicological Sciences* 67(1): 63–74.

Flattery J. *et al.* 1993. Lead poisoning associated with the use of traditional ethnic remedies-California 1991–1992. *Morbidity and Mortality Weekly Report* (MMWR) 42(27): 521–524.

Foster W. 1998. Endocrine disruptors and development of the reproductive system in the fetus and children. Is there cause for concern? *Canadian Journal of Public Health* 89(Suppl 1): S39–41, S52.

Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D, Peters J. 2004. The effect of air pollution on lung development from 10 to 18 years of age. *New England Journal of Medicine* 351: 1057–67.

Gauderman WJ, McConnell R, Gilliland F, London S, Thomas D, Avol E, Vora H, Berhane K, Rappaport EB, Lurmann F, Margolis HG, Peters J. 2000. Association between air pollution and lung function growth in southern California children. *American Journal of Respiratory and Critical Care Medicine* 162(4 Pt 1): 1383–90.

Gilliland FD, Berhane K, McConnell R, Gauderman WJ, Vora H, Rappaport EB, Avol E, Peters JM. 2000. Maternal smoking during pregnancy, environmental tobacco smoke exposure and childhood lung function. *Thorax* 55(4): 271–76.

Ginsberg G, Hattis D, Miller R, Sonawane B. 2004. Pediatric pharmacokinetic data: Implications for environmental risk assessment for children. *Pediatrics* 113: 973–83.

Gladen BC, Ragan NB, Rogan WJ. 2000. Pubertal growth and development and prenatal and lactational exposure to polychlorinated biphenyls and dichlorodiphenyl dichloroethene. *Journal of Pediatrics* 136(4): 490–96.

Goldman LR. 1998. Linking research and policy to ensure children's environmental health. *Environmnetal Health Perspectives* 106(Suppl 3): 857–62.

Goldman LR, Apelberg B, *et al.* 1999. *Healthy From the Start: Why America Needs a Better System to Track and Understand Birth Defects and the Environment.* Baltimore, MD, Pew Environmental Health Commission.

Goldman LR, Koduru S. 2000. Chemicals in the environment and developmental toxicity to children: A public health and policy perspective. *Environmental Health Perspectives* 108(Suppl 3): 443–48.

Goldman LR, Shannon MW. 2001. Technical report: Mercury in the environment: Implications for pediatricians. *Pediatrics* 108: 197–205.

Gomaa A, Hu H, Bellinger D, Schwartz J, Tsaih SW, Gonzalez-Cossio T, Schnaas L, Peterson K, Aro A, Hernandez-Avila M. 2002. Maternal bone lead as an independent risk factor for fetal neurotoxicity: A prospective study. *Pediatrics* 110: 110–18.

Grandjean P, Weihe P, White RF, Debes F, Araki S, Yokoyama K, Murata K, Sorensen N, Dahl R, Jorgensen PJ. 1997. Cognitive deficit in seven-year-old children with prenatal exposure to methylmercury. *Neurotoxicology and Teratology* 19(6): 417–28.

Grandjean P, Weihe P, Burse VW, Needham LL, Storr-Hansen E, Heinzow B, Debes F, Murata K, Simonsen H, Ellefsen P, Budtz-Jorgensen E, Keiding N, White RF. 2001. Neurobehavioral deficits associated with PCB in 7-year-old children prenatally exposed to seafood neurotoxicants. *Neurotoxicology and Teratology* 23: 305–17.

Gray LE Jr and Ostby JS. 1995. *In utero* 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) alters reproductive morphology and function in female rat offspring. *Toxicology & Applied Pharmacology* 133: 285–94.

Guilette LJ Jr., Gunderson MP. 2001. Alterations in development of reproductive and endocrine systems of wildlife populations exposed to endocrine-disrupting contaminants. *Reproduction* 122: 857–64.

Gutierrez G, Tapia-Conyer R, Guiscafre H, Reyes H, Martinez H. and Kumate J. 1996. Impact of oral rehydration and selected public health interventions on reduction of mortality from childhood diarrhoeal diseases in Mexico. *Bulletin of the World Health Organization* 74: 189–97.

Haines M. *et al.* 1998a. Dioxins and furans. Chapter 6 of: *Persistent Environmental Contaminants and the Great Lakes Basin Population: An Exposure Assessment*. Canada: Minister of Public Works and Government Services, Health Canada.

Haines M. *et al.* 1998b. Polychlorinated biphenyls. *Persistent Environmental Contaminants and the Great Lakes Basin Population: An Exposure Assessment.* Canada: Health Canada. Minister of Public Works and Government Services.

Hattis D, Ginsberg G, Sonawane B, Smolenski S, Russ A, Kozlak M, Goble R. 2003. Differences in pharmacokinetics between children and adults—II. Children's variability in drug elimination half-lives and in some parameters needed for physiologically-based pharmacokinetic modeling. *Risk Analysis* 23: 117–42.

Hattis D, Goble R, Russ A, Chu M, Ericson J. 2004. Age-related differences in susceptibility to carcinogenesis: a quantitative analysis of empirical animal bioassay data. *Environmental Health Perspectives* 112: 1152–58.

Health Canada. 1995. Pesticide-related injuries and poisonings to children less than 20 years of age from the entire CHIRP database as of December 1994.Ottawa. Canadian Hospitals Injury reporting and Prevention Program. Laboratory Center for Disease Control, Health Canada.

Health Canada. 1998a. *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.

Health Canada. 1998b. *The Health and Environment Handbook for Health Professionals*. Ministry of Supply & Services. Cat. No. H49-96/2-1995E.

Health Canada. 2002a. *Congenital Anomalies in Canada: A Perinatal Health Report.* Minister of Public Works and Government Services.

Health Canada. 2002b. Mercury and human health. 20 November. Available as <mercury_e.pdf> at Health Canada web site.

Health Canada. 2002c. Information on mercury levels in fish [Advisory]. May 29, 2002. Available at ‹ http://www.hc-sc.gc.ca/ahc-asc/media/advisories-avis/2002/2002_41_e.html›. Accessed 8 September 2005.

Health Canada. 2003. Canadian Perinatal Health Report. Ottawa: Minister of Public Works and Government Services Canada. Published by authority of the Minister of Health. Cat. No. H49-142/200E. ISBN 0-662-35503-2. ‹http://www.phac-aspc.gc.ca/publicat/cphr-rspc-03/pdf/cphr-rspc03_e.pdg›.

Hertwich EG, Pease WS, McKone TE. 1998. Evaluating toxic impact assessment methods: What works best? *Environmental Science & Technology* 32(5): 138A–145A.

Howdeshell, K.L. 2002. A model of the development of the brain as a construct of the thyroid system. *Environ Health Perspect* 100 (suppl 3): 337–348.

Indian and Northern Affairs Canada. 2003. Northern Contaminants Program. *Canadian Arctic Contaminants Assessment Report II*. Ottawa, ON, Canada: Health Canada.

INEGI. 1999. Estadísticas Vitales. Mexico City, INEGI, SSA/DGEI.

INEGI. 2000. Sistemas Nacionales de Estadisticas y de Información Geográphica 2000. ‹http://www.inegi.gob.mx›. Accessed 9/2004.

Institute of Medicine. 1999a. *Clearing the Air: Asthma and Indoor Air Exposures.* Washington, DC: National Academies Press.

Institute of Medicine. Committee on Environmental Justice. 1999b. *Toward Environmental Justice: Research, Education and Health Policy Issues.* Washington, DC: National Academies Press.

International Agency for Research on Cancer (IARC). 2004. Inorganic and Organic Lead Compounds. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. 87(10).

International Program on Chemical Safety (IPCS). 1998. Endocrine disrupters fact sheet. No. 10. Washington, DC: WHO.

IPCS. 2002. *Global Assessment of the State-of-the-Science of Endocrine Disruptors.* Washington, DC: WHO. WHO/PCS/EDC/02.2.

International Study of Asthma and Allergies in Children (ISAAC) Steering Committee. 1998. Worldwide variations in the prevalence of asthma symptoms: The International Study of Asthma and Allergies in Children. *European Respiratory Journal* 12: 315–35.

Jacobson JL. 2001. Contending with contradictory data in a risk assessment context: The case of methylmercury. *Neurotoxicology* 22(5): 667–75.

Jacobson JL and Jacobson SW. 1993. A four-year follow-up study of children born to consumers of Lake Michigan fish. *Journal of Great Lakes Research* 19: 776–783.

Jacobson JL and Jacobson SW. 1996. Dose-response in perinatal exposure to polychlorinated biphenyls (PCBs): The Michigan and North Carolina cohort studies. *Toxicology and Industrial Health* 12: 435–445.

Jacobson JL and Jacobson SW. 1997. Evidence for PCBs as neurodevelopmental toxicants in humans. *Neurotoxicology* 18(2): 415–424.

Jacobson JL and Jacobson SW. 2003. Prenatal exposure to polychlorinated biphenyls and attention at school age. *Journal of Pediatrics* 143: 780–88.

Jahnke GD, Choksi NY, Moore JA and Shelby MD. 2004. Thyroid toxicants: Assessing reproductive health effects. *Environmental Health Perspectives* 112(3): 363–68.

James SJ, Cutler P, Melnyk S, Jernigan S, Janak L, Gaylor DW, Neubrander JA. 2004. Metabolic biomarkers of increased oxidative stress and impaired methylation capacity in children with autism. *The American Journal of Clinical Nutrition* 80: 1611–17.

Karmaus W and Zhu X. 2004. Maternal concentration of polychlorinated biphenyls and dichlorodiphenyl dichlorethylene and birth weight in Michigan fish eaters: A cohort study. *Environmental Health* 3:1.

Kass DE, Thier AL, Leighton J, Cone JE, Jeffrey NL. 2004. Developing a comprehensive pesticide health effects tracking system for an urban setting: New York City's approach. *Environmental Health Perspectives* 112(14): 1419–1423.

Kavlock R, Boekelheide K, Chapin R, Cunningham M, Faustman E, Foster P, Golub M, Henderson R, Hinberg I, Little R, Seed J, Shea K, Tabacova S, Tyl R, Williams P, Zacharewski T. 2002a. NTP Center for the Evaluation of Risks to Human Reproduction: Phthalates expert panel report on the reproductive and developmental toxicity of butyl benzyl phthalate. *Reproductive Toxicology* 16(5): 453–87.

Kavlock R, Boekelheide K, Chapin R, Cunningham M, Faustman E, Foster P, Golub M, Henderson R, Hinberg I, Little R, Seed J, Shea K, Tabacova S, Tyl R, Williams P, Zacharewski T. 2002b. NTP Center for the Evaluation of Risks to Human Reproduction: Phthalates expert panel report on the reproductive and developmental toxicity of di(2-ethylhexyl) phthalate. *Reproductive Toxicology* 16(5): 529–653.

Kavlock R, Boekelheide K, Chapin R, Cunningham M, Faustman E, Foster P, Golub M, Henderson R, Hinberg I, Little R, Seed J, Shea K, Tabacova S, Tyl R, Williams P, Zacharewski T. 2002c. NTP Center for the Evaluation of Risks to Human Reproduction: Phthalates expert panel report on the reproductive and developmental toxicity of di-isodecyl phthalate. *Reproductive Toxicology* 16(5): 655–78.

Kavlock R, Boekelheide K, Chapin R, Cunningham M, Faustman E, Foster P, Golub M, Henderson R, Hinberg I, Little R, Seed J, Shea K, Tabacova S, Tyl R, Williams P, Zacharewski T. 2002d. NTP Center for the Evaluation of Risks to Human Reproduction: Phthalates expert panel report on the reproductive and developmental toxicity of di-isononyl phthalate. *Reproductive Toxicology* 16(5): 679–708.

Kavlock R, Boekelheide K, Chapin R, Cunningham M, Faustman E, Foster P, Golub M, Henderson R, Hinberg I, Little R, Seed J, Shea K, Tabacova S, Tyl R, Williams P, Zacharewski T. 2002e. NTP Center for the Evaluation of Risks to Human Reproduction: Phthalates expert panel report on the reproductive and developmental toxicity of di-*n*-butyl phthalate. *Reproductive Toxicology* 16(5): 489–527.

Kavlock R, Boekelheide K, Chapin R, Cunningham M, Faustman E, Foster P, Golub M, Henderson R, Hinberg I, Little R, Seed J, Shea K, Tabacova S, Tyl R, Williams P, Zacharewski T. 2002f. NTP Center for the Evaluation of Risks to Human Reproduction: Phthalates expert panel report on the reproductive and developmental toxicity of di-*n*-hexyl phthalate. *Reproductive Toxicology* 16(5): 709–19.

Kavlock R, Boekelheide K, Chapin R, Cunningham M, Faustman E, Foster P, Golub M, Henderson R, Hinberg I, Little R, Seed J, Shea K, Tabacova S, Tyl R, Williams P, Zacharewski T. 2002g. NTP Center for the Evaluation of Risks to Human Reproduction: Phthalates expert panel report on the reproductive and developmental toxicity of di-*n*-octyl phthalate. *Reproductive Toxicology* 16(5): 721–34.

Kiely T, David D, Grube AH. 2004. Pesticide Industry Sales and Usage: 2000 and 2001 Market Estimates. Washington, DC: US Environmental Protection Agency. Office of Prevention, Pesticides and Toxic Substances.

Kimbrough RD and Krouskas CA. 2001. Polychlorinated biphenyls, dibenzo-*p*-dioxins, and dibenzofurans and birth weight and immune and thyroid function in children. *Regulatory Toxicology and Pharmacology* 34:42-52.

Kimbrough RD and Krouskas C. 2002. Polychlorinated biphenyls, TEQs, children, and data analysis. *Veterinary and Human Toxicology* 44:354-7.

Kohn MC, Parham F, Masten SA, Portier CJ, Shelby MD, Brock JW, Needham LL. 2000. Human exposure estimates for phthalates. *Environmental Health Perspectives 108(*10): A440–42.

Landrigan PJ. 1998. Environmental hazards for children in USA. *International Journal of Occupational Medicine and Environmental Health* 11(2):189-94.

Landrigan PJ, Halper LA, Silbergeld EK. 1989. Toxic air pollution across a state line: Implications for the siting of resource recovery facilities. *Journal of Public Health Policy* 10(3): 309–23.

Landy S and Tam KK. 1998. Understanding the contribution of multiple risk factors on child development as children grow. National Longitudinal Study in Children and Youth. Workshop paper given at "Investing in Children. A National Research Conference."

Lanphear BP, Dietrich K, Auinger P, Cox C. 2000. Cognitive deficits associated with blood lead concentrations <10 microg/dL in US children and adolescents. *Public Health Reports* 115(6): 521–529.

Learning Disabilities Association of Canada (LDAC). 2002. Official definition of learning disabilities adopted 30 January 2002.

Levy BS, Nassetta WJ. 2003. Neurologic effects of manganese in humans: A review. *International Journal of Occupational and Environmental Health* 9(2): 153–63.

Li Y, Moon KS, Wong CP. 2005. Materials science. Electronics without lead. *Science* 308(5727): 1419–20.

Longnecker MP, Rogan WJ, Lucier G. 1997. The human health effects of DDT (dichlorodiphenyltrichloroethane) and PCBS (polychlorinated biphenyls) and an overview of organochlorines in public health. *Annual Review of Public Health* 18: 211–244.

Longnecker MP, Klebanoff MA, Zhou H, Brock JW. 2001. Association between maternal serum concentration of DDT metabolite DDE and preterm and small-for-gestational age babies at birth. *The Lancet* 358(9276): 110–14.

Mackenzie CA, Lockridge A, Keith M. 2005. Declining sex ratio in a first nation community. *Environmental Health Perspectives* 113: 1295–98. doi:10.1289/ehp.8479.

Mahaffey KR, Clickner RP, Bodurow CC. 2004. Blood organic mercury and dietary mercury intake: National health and nutrition examination survey, 1999 and 2000. *Environmental Health Perspectives* 112: 562–70.

Mahaffey KR, Gartside PS, Glueck CJ. 1986. Blood lead levels and dietary calcium intake in 1- to 11-year-old children: The Second National Health and Nutrition Examination Survey, 1976 to 1980. *Pediatrics* 78: 257–62.

Mannino DM, Homa DM, Akinbami LJ, Moorman JE, Gwynn C, Redd SC. 2002. Surveillance for asthma, United States, 1980–1999. *Morbidity and Mortality Weekly Report* 51(SS-01). March 29.

March of Dimes. 2002. Birth Defects. Available at ‹http://www.modimes.org/›.

McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. 2002. Asthma in exercising children exposed to ozone: A cohort study. *Lancet* 359(9304): 386–91.

McHale CM, and Smith MT. 2004. Prenatal origin of chromosomal translocations in acute childhood leukemia: Implications and future directions. *American Journal of Hematology* 75: 254–57.

McKee RH. 2004. Phthalate exposure and early thelarche. *Environmental Health Perspectives* 112(10): A541–43.

Melnick R, Lucier G, Wolfe M, Hall R, Stancel G, Prins G, Gallo M, Reuhl K, Ho SM, Brown T, Moore J, Leakey J, Haseman J, Kohn M. 2002. Summary of the National Toxicology Program's report of the endocrine disruptors low-dose peer review. *Environvironmnetal Health Perspectives* 110: 427–31.

Mendola P, Selevan SG, Gutter S, Rice D. 2002. Environmental factors associated with a spectrum of neurodevelopmental deficits. *Mental Retardation and Developmental Disabilities Research Reviews* 8: 188–97.Miller MD, Marty MA, Arcus A, Brown J, Morry D, Sandy M. 2002. Differences between children and adults: Implications for risk assessment at California EPA. *International Journal of Toxicology* 21: 403–18.

Mocarelli P, Gerthoux PM, Ferrari E, Patterson DG Jr, Kieszak SM, Brambilla P, Vincoli N, Signorini S, Tramacere P, Carreri V, Sampson EJ, Turner WE, Needham LL. 2000. Paternal concentrations of dioxin and sex ratio of offspring. *Lancet* 355(9218): 1858–63.

Mol NM, Sorensen N, Weihe P, Andersson AM, Jorgensen N, Skakkebaek NE, *et al.* 2002. Spermaturia and serum hormone concentrations at the age of puberty in boys prenatally exposed to polychlorinated biphenyls. *European Journal of Endocrinology* 146(3): 357–363.

Muckle G, Ayotte P, Dewailly EE, Jacobson SW, Jacobson JL. 2001. Prenatal exposure of the northern Quebec Inuit infants to environmental contaminants. *Environental Health Perspectives* 109: 1291–99.

Muhle R, Trentacoste SV, Rapin I. 2004. The genetics of autism. *Pediatrics* 113: e472–86.

Myers GJ, Davidson PW, Cox C, Shamlaye CF, Palumbo D, Cernichiari E, Sloane-Reeves J, Wilding GE, Kost H, Huang LS, *et al.* 2003. Prenatal methylmercury exposure from ocean fish consumption in the Seychelles child development study. *Lancet* 361(9370): 1686–92.

National Academy of Sciences (NAS). 2000. Toxicological Effects of Methylmercury. Washington, DC. Available at ‹http://books.nap.edu/books/0309071402/html/index.html›.

National Birth Defects Prevention Network. 2000. Birth defect surveillance data from selected states, 1989–1996. *Teratology* 61: 86–160.

National Cancer Institute of Canada (NCIC). Canadian Cancer Statistics 2002. Toronto, Canada. Available at ‹https://www.cancer.ca› and ‹https://www.ncic.cancer.ca›.

National Indigenist Institute (*Instituto Nacional Indigenista)*. 2001. XII General Census of Population and Living. Mexico.

National Research Council (NRC). 1993. *Pesticides in the diet of infants and children.* Washington, DC: National Academies Press.

NRC. 1996. *Carcinogens and anticarcinogens in the human diet: A comparison of naturally occurring and synthetic substances.* Washington, DC: National Academies Press.

NRC. 2000. *Toxicological effects of methylmercury*. Washington, DC: National Academies Press.

National Research Council and Institute of Medicine (NRC and IOM). 2004. *Children's Health, the Nation's Wealth: Assessing and Improving Child Health*. Washington, DC: The National Academies Press.

National Toxicology Program (NTP). 2004. *Report on Carcinogens: Eleventh Edition* Research Triangle Park, NC: US Department of Health and Human Services, Public Health Service, National Toxicology Program.

Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. 1990. The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. *New England Journal of Medicine* 322: 83–8.

Needleman HL and Bellinger D. 1991. The health effects of low-level exposure to lead. *Annual Review of Public Health* 12: 111–40.

Nelson K, Holmes LB. 1989. Malformations due to presumed spontaneous mutations in newborn infants. *New England Journal of Medicine* 320(1): 19–23.

Ortega-Cesena J, Espinosa-Torres F, Lopez-Carrillo L. 1994. El controls de los riesgos para la salud generados por los plaguicidas organofosforados en México: Retos ante el Tratado de Libre Comercio. *Salud Pública de México* 36: 624–632.

Page BD, Lacroix GM. 1995. The occurrence of phthalate ester and di-2-ethylhexyl adipate plasticizers in Canadian packaging and food sampled in 1985–1989: A survey. *Food Additives and Contaminants* 12(1): 129–51.

Parent AS, Teilmann G, Juul A, Skakkebaek NE, Toppari J and Bourguignon JP. 2003. The timing of normal puberty and the age limits of sexual precocity: Variations around the world, secular trends, and changes after migration. *Endocrine Reviews* 24(5): 668–93.

Patandin S, Koopman-Esseboom C, de Ridder MA, Weisglas-Kuperus N, Sauer PJ. 1998. Effects of environmental exposure to polychlorinated biphenyls and dioxins on birth size and growth in Dutch children. *Pediatric Research* 44: 538–45.

Patandin S, Dagnelie PC, Mulder PG, Op de Coul E, van der Veen JE, Weisglas-Kuperus N, Sauer PJ. 1999a. Dietary exposure to polychlorinated biphenyls and dioxins from infancy until adulthood: A comparison between breast-feeding, toddler, and long-term exposure. *Environmental Health Perspectives* 107(1): 45–51.

Patandin S, Lanting CI, Mulder PG, Boersma ER, Sauer PJ, Weisglas-Kuperus N. 1999b. Effects of environmental exposure to polychlorinated biphenyls and dioxins on cognitive abilities in Dutch children at 42 months of age [see comments]. *Journal of Pediatrics* 134: 33–41.

Paulozzi LJ, Erickson JD, Jackson RJ. 1997. Hypospadias trends in two US surveillance systems. *Pediatrics* 100(5): 831–834.

Plunkett LM *et al.* 1992. Differences between adults and children affecting exposure assessment. In: Guzelian, P. Henry, C. and S.S.Olin (eds). *Similarities and Differences between Children and Adults: Implications for Risk Assessment.* Washington, DC: ILSI Press.

Porterfield SP. 2000. Thyroidal dysfunctin and environmental chemicals—Potential impact on brain development. *Environmental Health Perspectives* 108(Suppl 3): 433–38.

Public Health Agency of Canada. 1999. *Measuring Up: A Health Surveillance Update on Canadian Children and Youth.* Rusen ID, McCourt C, Eds. Ottawa: Minister of Public Works and Government Services Canada, Cat. H42-2/82-1999E. Available at: ‹http://www.phac-aspc.gc.ca/publicat/meas-haut/index.html›.

Rasmussen, P.E., K.S. Subramanian, B.J. Jessiman. 2001. A multi-element profile of house dust in relation to exterior dust and soils in the city of Ottawa, Canada. *Science of the Total Environment* 267: 125–140.

Ribas-Fito N, Sala M, Kogevinas M, Sunyer J. 2001. Polychlorinated biphenyls (PCBs) and neurological development in children: A systematic review. *Journal of Epidemiology and Community Health* 55: 537–46.

Rice DC. 2000. Parallels between attention deficit hyperactivity disorder and behavioral deficits produced by neurotoxic exposure in monkeys. *Environmental Health Perspectives* 108 (Suppl 3): 405–8.

Ries L, Smith M, Gurney J, Linet M, Tamra T, Young J, Bunin G. 1999. Cancer Incidence and Survival among Children and Adolescents: United States SEER Program 1975–1995.

Ries L, *et al.*, editors. 2001. *SEER Cancer Statistics Review, 1973–1998.* Bethesda, MD: National Cancer Institute.

Rodier PM. 1995. Developing brain as a target of toxicity. *Environmental Health Perspectives* 103(6): 73–76.

Rogan WJ. 1996. Pollutants in breast milk. *Archives of Pediatric & Adolescent Medicine* 150: 981–990.

Rogan WJ, *et al.* 1999. Sex ratio after exposure to dioxin-like chemicals in Taiwan. *Lancet* 353(9148): 206–7.

Rogan WJ and Ragan NB. 2003. Evidence of effects of environmental chemicals on the endocrine system in children. *Pediatrics* 112(1 Pt 2): 247–52.

Romieu I, Palazuelos E, Hernandez Avila M, Rios C, Munoz I, Jimenez C, Cahero G. 1994. Sources of lead exposure in Mexico City. *Environmental Health Perspectives* 102(4): 384–89.

Romieu I, *et al.* 1999. Air pollution and school absenteeism among children in Mexico City. *Epidemiology* 136(12): 1524–31.

Romieu I, *et al*. 2004. Infant mortality and air pollution: Modifying effect by social class. *Journal of Occupational and Environmental Medicine* 46: 1210–16.

Rosales-Castillo JA, Torres-Meza VM, Olaiz-Fernandez G, Borja-Aburto VH. 2001. [Acute effects of air pollution on health: Evidence from epidemiological studies]. *Salud Publica de Mexico* 43: 544–55.

Rothenberg SJ, Schnaas L, Perroni E, Hernandez RM, Karchmer S. 1998. Secular trend in blood lead levels in a cohort of Mexico City children. *Archives of Environmental Health* 53: 231–35.

Rouleau J, Arbuckle TE, Johnson KC, Sherman GJ. 1995. Status report: Description and limitations of the Canadian Congenital Anomalies Surveillance System (CCASS). *Chronic Diseases in Canada* 16.

Ryan JJ, Amirova Z, Carrier G. 2002. Sex ratios of children of Russian pesticide producers exposed to dioxin. *Environmental Health Perspectives* 110(11): A699–701.

Santos-Burgoa C, Rios C, Mercado LA, Arechiga-Serrano R, Cano-Valle F, Eden-Wynter RA, Texcalac-Sangrador JL, Villa-Barragan JP, Rodriguez-Agudelo Y, Montes S. 2001. Exposure to manganese: Health effects on the general population, a pilot study in central Mexico. *Environmental Research* 85(2): 90–104.

Schantz SL, Widholm JJ, Rice DC. 2003. Effects of PCB exposure on neuropsychological function in children. *Environmental Health Perspectives* 111: 357–76.

Schmidt C. 1998. Childhood cancer: A growing problem. *Environmental Health Perspectives* 109(Suppl 6): 813–816.

Schnorr TM, Lawson CC, Whelan EA, Dankovic JA, Deddens JA, Piacitelli LA, Reefhuis J, Sweeney MH, Connally LB, Fingerhut MA. 2001. Spontaneous abortion, sex ratio, and paternal occupational exposure to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. *Environmental Health Perspectives* 109(11): 1127–32.

Scorecard. 2002. Health Effects. Available at ‹www.scorecard.org›.

Secretaría de Salud (SSA), with Instituto Nacional de Salud Pública (INSP). 1997. *Encuesta Nacional de Nutrición.*

SSA. 2001. ‹http://222.ssa.gob.mx.unidades/dgied/sns/vitales›.

Selevan SG, Kimmel CA, Mendola P. 2000. Identifying critical windows of exposure for children's health. *Environmental Health Perspectives* 108 (Suppl 3): 451–55.

Selevan SG, Rice DC, Hogan KA, Euling SY, Pfahles-Hutchens A, and Bethel J. 2003. Blood lead concentration and delayed puberty in girls. *New England Journal of Medicine* 348(16): 1527–36.

Sexton K, Greaves IA, Church TR, Adgate JL, Ramachandran G, Tweedie RL, Fredrickson A, Geisser M, Sikorski M, Fischer G, Jones D, Ellringer P. 2000. A school-based strategy to assess children's environmental exposures and related health effects in economically disadvantaged urban neighborhoods. *Journal of Exposure Analysis and Environmental Epidemiology* 10(6 Pt 2): 682–94.

Siegel BZ, Siegel SM, Correa T, Dagan C, Galvez G, LeeLoy L, Padua A, Yaeger E. 1991. The protection of invertebrates, fish, and vascular plants against inorganic mercury poisoning by sulfur and selenium derivatives. *Archives of Environmental Contamination and Toxicology* 20: 241–46.

Sienra-Monge JJ, Del Rio Navarro B. 1999. Asma aguda. *Boll Medical Hosp. Infantil Mex* 56: 185–194.

Skakkebaek NE, Rajpert-De Meyts E, Main KM. 2001. Testicular dysgenesis syndrome: An increasingly common developmental disorder with environmental aspects. *Human Reproduction* 16(5): 972–978.

Statistics Canada. 2001. CANSUM tables. ‹http:www.statcan.ca›, ‹http://www12.statcan.ca/english/census01/›.

Stern, AH (2005). A review of the studies of the cardiovascular health effects of methylmercury with consideration of their suitability for risk assessment. *Environ Res* 98(1): 133–42.

Stewart PW, Reihman J, Lonky EI, Darvill TJ, Pagano J. 2003. Cognitive development in preschool children prenatally exposed to PCBs and MeHg. *Neurotoxicology and Teratology* 25: 11–22.

Subcomité de Comercio y Fomento Industrial. 2001. *Importación de Productos Regulados por Cicoplafest: 1–4.*

Swan SH, Main KM, Liu F, Stewart SL, Kruse RL, Calafat AM, Mao CS, Redmon JB, Ternand CL, Sullivan S, Teague JL, *et al.* 2005. Decrease in anogenital distance among male infants with prenatal phthalate exposure. *Environmental Health Perspectives* 113(8): 1056–61.

Takser L, Mergler D, Hellier G, Sahuquillo J, Huel G. 2003. Manganese, monoamine metabolite levels at birth, and child psychomotor development. *Neurotoxicology* 24(4-5): 667–74.

Torres-Sanchez LE, Berkowitz G, Lopez-Carrillo L, Torres-Arreola L, Rios C, Lopez-Cervantes M. 1999. Intrauterine lead exposure and preterm birth. *Environmental Research* 81: A 297–301.

Trasande L, Landrigan PJ, Schechter C. 2005. Public health and economic consequences of methylmercury toxicity to the developing brain. *Environmental Health Perspectives* 113(5): 590–6.

United Nations Children's Fund (UNICEF). 2000. A league table of child poverty in rich nations. *Innocenti Report Card No.1*. UNICEF: Innocenti Research Centre, Florence.

UNICEF. 2003. *State of the World's Children: 2003.* UNICEF.

UNICEF. 2005. *State of the World's Children: 2005.* UNICEF. Available at ‹www.unicef.org/sowc05/english/index.html›.

United Nations Environment Programme (UNEP) Governing Council. 2001. Decision 21/6: Lead in gasoline. Nairobi: UNEP.

United Nations Population Division. 2005. Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat, *World Population Prospects: The 2002 Revision* and *World Urbanization Prospects: The 2001 Revision*. ‹http://esa.un.org/unpp›. Accessed 18 February 2005.

United States Environmental Protection Agency (US EPA). 1997a. Pesticide Industry Sales and Usage: 1994 and 1995 Market estimates. Tables 2 and 7. Our Children at Risk: The Five Worst Environmental Threats to Their Health. Washington, DC: National Resources Defense Council.

US EPA. 1997b. *Mercury Study Report to Congress*. EPA/452-R-97-003-009.

US EPA. 1998. *Chemical Hazard Data Availability Study.* Available at ‹www.epa.gov/opptintr/chemtest/hazchem.htm›.

US EPA. 2002a. National Air Toxics Assessment. Summary of results. Available at ‹www.epa.gov/ttn/atw/›.

US EPA. 2002b. *Priority PBTs; Mercury and Compounds.* Persistent, Bioaccumulative and Toxic Chemical Program. Office of Pollution Prevention. Available at ‹epa.gov/pbt/mercury.htm›.

US EPA. 2002c. TRI Explorer results for PCBs. Available at ‹www.epa.gov/triexplorer/›.

US EPA. 2003. *America's Children and the Environment: Measures of Contaminants, Body Burdens and Illnesses.*

US EPA. 2005a. Supplemental guidance for assessing susceptibility from early-life exposure to carcinogens. Washington, DC: Risk Assessment Forum, Environmental Protection Agency. EPA/630/R-03/003F.

US EPA. 2005b. Guidelines for carcinogen risk assessment. Washington, DC: Risk Assessment Forum, Environmental Protection Agency. EPA/630/P-03/001B.

US EPA. 2005c. The Inventory of Sources and Environmental Releases of Dioxin-like Compounds in the United States: The Year 2000 Update (External Review Draft), EPA/600/P-03/002A, March.

United States Food and Drug Administration (US FDA). 2004. Lead contamination in candy. *FDA Consumer* 38: 7.

van den Berg M, *et al.* 1998. Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. *Environmental Health Perspectives* 106(12): 775–92.

van Birgelen AP, Fase KM, van der Kolk J, Poiger H, Brouwer A, Seinen W, van den Berg M. 1996. Synergistic Effect of 2,2′,4,4′,5,5′-hexachlorobiphenyl and 2,3,7,8-tetrachlorodibenzo-*p*-dioxin on hepatic porphyrin levels in the rat. *Environmental Health Perspectives* 104: 550–557.

Vos JG, Dybing E, Greim HA, Ladefoged O, Lambre C, Tarazona JV, Brandt I, Vethaak AD. 2000. Health effects of endocrine-disrupting chemicals on wildlife, with special reference to the European situation. *Critical Reviews in Toxicology* 30: 71–133.

Vreugdenhil HJ, Mulder PG, Emmen HH, Weisglas-Kuperus N. 2004. Effects of perinatal exposure to PCBs on neuropsychological functions in the Rotterdam cohort at 9 years of age. *Neuropsychology* 18: 185–93.

Wang ST, Pizzolato S, Demshar HP, Smith LF. 1997. Decline in blood lead in Ontario children correlated to decreasing consumption of leaded gasoline, 1983–1992. *Clinical Chemistry* 43: 1251–52.

Wargo JW, and Wargo LE. 2002. The State of Children's Health and Environment: Common Sense Solutions for Parents and Policymakers. Children's Health Environmental Coalition.

Watson WA, Litovitz TL, Klein-Schwartz W, Rodgers GC, Youniss J, Reid N, Rouse WG, Rembert RS, Borys D. 2004. 2003 Annual report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. *American Journal of Emergency Medicine* 22: 5, 335–404.

Whyatt RM, Rauh V, Barr DB, Camann DE, Andrews HF, Garfinkel R, Hoepner LA, Diaz D, Dietrich J, Reyes A, Tang D, Kinney PL, Perera FP. 2004. Prenatal insecticide exposures and birth weight and length among an urban minority cohort. *Environmental Health Perspectives* 112(10): 1125–32.

Wiencke JK, Thurston SW, Kelsey KT, Varkonyi A, Wain JC, Mark EJ, Christiani DC. 1999. Early age at smoking initiation and tobacco carcinogen DNA damage in the lung. *Journal of the National Cancer Institute* 91(7): 614–19.

Winneke G, Bucholski A, Heinzow B, Kramer U, Schmidt E, Walkowiak J, Wiener JA, Steingruber HJ. 1998. Developmental neurotoxicity of polychlorinated biphenyls (PCBS): Cognitive and psychomotor functions in 7-month old children. *Toxicology Letters* 102–103: 423–28.

Wood DL. 2003. Increasing immunization coverage. American Academy of Pediatrics Committee on Community Health Services. American Academy of Pediatrics Committee on Practice and Ambulatory Medicine. *Pediatrics* 112: 993–96.

World Health Organization (WHO). 1948. Preamble. Constitution. Geneva: WHO.

WHO. 1997. *Health and Environment in Sustainable Development. Five Years after the Earth Summit.* Geneva: WHO.

WHO. 1998. *Assessment of the Health Risk of Dioxins: Re-evaluation of the Tolerable Daily Intake (TDI).* Executive summary. Geneva: WHO European Centre for Environment and Health International Programme on Chemical Safety.

WHO. 2003. Children in the New Millennium: Environmental Impact on Health. Geneva: WHO.

Wyatt CJ, Fimbres C, Romo L, Mendez RO, Grijalva M. 1998. Incidence of heavy metal contamination in water supplies in northern Mexico. *Environmental Research* 76 (2): 114–19.

Yoshimura T, Kaneko S, Hayabuchi H. 2001. Sex ratio in offspring of those affected by dioxin and dioxin-like compounds: The Yusho, Seveso, and Yucheng incidents. *Occupational and Environmental Medicine* 58(8): 540–41.

Zahm S and Devesa S. 1995. Childhood cancer: An overview of incidence trends and environmental carcinogens. *Environmental Health Perspectives* 103 (Suppl 6): 177–184.

Zahm SH and Ward MH. 1998. Pesticides and childhood cancer. *Environmental Health Perspectives* 106 (Suppl 3): 893–908.

Appendixes

Appendix A: Number of Children in North America

Country	*Number of children under 18 years old*[1]	*Number of children 0–5 years old*[1]	*Total population in 2003*[1]	*Children as a percent of total population*[1]	*0–5 years as percent of total population*[1]	*Urbanized rate*[1]	*Estimated number of urban children*[1]	*Children's "relative poverty" rate*[2]	*Estimated number of children in "relative poverty"*[2]
Canada	6,942,000	1,663,000	31,510,000	22.0	5.3	80	5,553,600	16	1,259,200
Mexico	39,800,000	11,145,000	103,457,000	38.5	10.8	75	29,850,000	26	5,914,700
United States	75,893,000	20,794,000	294,043,000	25.8	7.1	80	60,714,400	22	16,228,000
Total	**122,635,000**	**33,602,000**	**429,010,000**	**28.6**	**7.8**		**96,118,000**		**23,401,900**

Sources: (1) UNICEF. 2005. *State of the World's Children*. See **‹www.unicef.org›**. (2) UNICEF. 2000. *A League Table of Child Poverty in Rich Nations. Innocenti Report Card No.1.* UNICEF Innocenti Research Centre, Florence, 2000. **Relative Poverty** = living in a household where the income is less than half the national median.

Appendix B: List of Chemicals Reported to Both TRI and NPRI That Are Carcinogens, Recognized or Suspected Developmental and Reproductive Toxicants, and/or Suspected Neurotoxicants, and Their Toxic Equivalency Potentials (TEPs)

					Scorecard Lists							
CAS Number	*Chemical*	*Carcinogens*	*C. TEP Air*	*C. TEP Water*	*R. DEV*	*R. REP*	*S. DEV*	*S. REP*	*S. NEU*	*NC. TEP Air*	*NC. TEP Water*	*Not in 98-02 data set*
75-07-0	**Acetaldehyde**	■	0.010000	0.006300			■		■	9.300000	5.100000	
75-05-8	**Acetonitrile**						■	■	■	30.000000	15.000000	
98-86-2	**Acetophenone**									2.500000	0.630000	•
107-02-8	**Acrolein**						■		■	1,600.000000	2,200.000000	•
79-06-1	**Acrylamide**	■	130.000000	1.600000				■	■	2,000.000000	25.000000	
79-10-7	**Acrylic acid**									62.000000	0.220000	
107-13-1	**Acrylonitrile**	■	3.900000	1.600000			■	■	■	38.000000	19.000000	
107-18-6	**Allyl alcohol**								■	4.300000	1.000000	
107-05-1	**Allyl chloride**		0.030000	0.010000			■		■	88.000000	45.000000	
7429-90-5	**Aluminum** (fume or dust)							■	■	61.000000	9.300000	
1344-28-1	**Aluminum oxide** (fibrous forms)								■			
62-53-3	**Aniline**		0.010000	0.006600					■	91.000000	57.000000	
120-12-7	**Anthracene**									0.180000	0.008100	
—	**Antimony** (and its compounds)							■	■	8,100.000000	1,500.000000	
1332-21-4	**Asbestos** (friable)	■										
71-43-2	**Benzene**	■	1.000000	0.760000	■	■			■	8.100000	10.000000	
98-88-4	**Benzoyl chloride**						■					
94-36-0	**Benzoyl peroxide**											
100-44-7	**Benzyl chloride**	■	0.880000	0.070000			■		■	21.000000	1.900000	
92-52-4	**Biphenyl**						■		■	0.980000	3.400000	
7637-07-2	**Boron trifluoride**											•
7726-95-6	**Bromine**								■			•
353-59-3	**Bromochlorodifluoro-methane** (Halon 1211)								■			•

Sources: *Carcinogens*: from US TRI chemicals reported under *de minimus* limit of 0.1 for 2002, **‹www.epa.gov/trichemical/oshacarc.htm›**, based on IARC (1, 2A and 2b), NTP (K or P) and OSHA (Z). Scorecard lists: ‹www.scorecard.org› (recognized developmental and reproductive toxicants based on California Proposition 65 list **‹www.oehha.ca.gov/prop65/prop65_list/files/070904list.html›**. Toxic Equivalency Potentials (TEPs) indicate relative human health risks associated with one unit of chemical, compared to the risk posed by release of a reference chemical (benzene). These TEPs are from **‹www.scorecard.org›**. Abbreviations: **C. TEP Air** = Carcinogen Toxic Equivalency Potential for air; **C. TEP Water** = Carcinogen Toxic Equivalency Potential for water; **R. DEV** = Recognized Developmental Toxicant; **R. REP** = Recognized Reproductive Toxicant; **S. DEV** = Suspected Developmental Toxicant; **S. REP** = Suspected Reproductive Toxicant; **S. NEU** = Suspected Neurotoxicant; **NC. TEP Air** = Noncancer Toxic Equivalency Potential for air; **NC. TEP Water** = Noncancer Toxic Equivalency Potential for water.

Appendix B: continued

CAS Number	Chemical	Carcinogens	C. TEP Air	C. TEP Water	Scorecard Lists: R. DEV	R. REP	S. DEV	S. REP	S. NEU	NC. TEP Air	NC. TEP Water	Not in 98-02 data set
74-83-9	**Bromomethane**				■			■	■	1,600.000000	900.000000	
75-63-8	**Bromotrifluoromethane** (Halon 1301)								■			•
106-99-0	**1,3-Butadiene**	■	0.530000	4.800000	■	■			■	2.200000	7.500000	
141-32-2	**Butyl acrylate**											
71-36-3	**n-Butyl alcohol**								■	0.710000	0.170000	
78-92-2	**sec-Butyl alcohol**								■	0.570000	0.140000	
75-65-0	**tert-Butyl alcohol**						■		■	2.200000	2.200000	
106-88-7	**1,2-Butylene oxide**	■										
123-72-8	**Butyraldehyde**											
156-62-7	**Calcium cyanamide**											
75-15-0	**Carbon disulfide**				■	■			■	1.200000	1.800000	
56-23-5	**Carbon tetrachloride**	■	270.000000	260.000000			■	■	■	2,300.000000	2,300.000000	
120-80-9	**Catechol**	■	0.140000	0.002500					■			
115-28-6	**Chlorendic acid**	■										•
7782-50-5	**Chlorine**								■			
10049-04-4	**Chlorine dioxide**						■	■				
79-11-8	**Chloroacetic acid**								■	190.000000	1.700000	
108-90-7	**Chlorobenzene**						■	■	■	0.950000	5.300000	
75-68-3	**1-Chloro-1,1-difluoroethane** (HCFC-142b)									1.000000	0.008600	•
75-45-6	**Chlorodifluoromethane** (HCFC-22)						■		■	1.400000	0.010000	•
75-00-3	**Chloroethane**		0.006700	0.006900			■		■	0.020000	0.020000	
67-66-3	**Chloroform**	■	1.600000	1.500000			■	■	■	14.000000	16.000000	
74-87-3	**Chloromethane**		0.660000	0.390000	■			■	■	57.000000	34.000000	
563-47-3	**3-Chloro-2-methyl-1-propene**	■										•
542-76-7	**3-Chloropropionitrile**								■			•
63938-10-3	**Chlorotetrafluoroethane** (HCFC-124 and isomers)											•
354-25-6	**Chlorotetrafluoroethane** (HCFC-124 and isomers)											•
2837-89-0	**Chlorotetrafluoroethane** (HCFC-124 and isomers)											•
75-72-9	**Chlorotrifluoromethane** (CFC-13)								■			•
—	**Chromium** (and its compounds)		130.000000					■		2,400.000000	260.000000	
4680-78-8	**C.I. Acid Green 3**											
569-64-2	**C.I. Basic Green 4**											
989-38-8	**C.I. Basic Red 1**											
28407-37-6	**C.I. Direct Blue 218**											•
2832-40-8	**C.I. Disperse Yellow 3**											
81-88-9	**C.I. Food Red 15**											
3118-97-6	**C.I. Solvent Orange 7**											
842-07-9	**C.I. Solvent Yellow 14**											
—	**Cobalt** (and its compounds)	■					■	■	■	31,000.000000	65.000000	
—	**Copper** (and its compounds)						■	■	■	13,000.000000	12,000.000000	
1319-77-3	**Cresols**								■	13.000000	0.770000	
4170-30-3	**Crotonaldehyde**											•
98-82-8	**Cumene**								■	0.410000	0.380000	
80-15-9	**Cumene hydroperoxide**											
—	**Cyanides**						■	■	■			
110-82-7	**Cyclohexane**								■	0.020000	0.120000	
108-93-0	**Cyclohexanol**							■	■			•
1163-19-5	**Decabromodiphenyl oxide**						■					
95-80-7	**2,4-Diaminotoluene**	■	61.000000	1.500000				■	■			
84-74-2	**Dibutyl phthalate**						■	■	■	11.000000	1.800000	
95-50-1	**1,2-Dichlorobenzene**								■	8.200000	10.000000	
106-46-7	**1,4-Dichlorobenzene**	■	1.400000	0.710000			■		■	2.200000	1.300000	
612-83-9	**3,3'-Dichlorobenzidine dihydrochloride**	■										•
75-71-8	**Dichlorodifluoro-methane** (CFC-12)								■	4.600000	3.800000	•
107-06-2	**1,2-Dichloroethane**	■	2.500000	2.900000			■	■	■	4.200000	4.800000	
1717-00-6	**1,1-Dichloro-1-fluoroethane** (HCFC-141b)								■			•
75-09-2	**Dichloromethane**	■	0.200000	0.130000				■	■	7.000000	4.400000	
120-83-2	**2,4-Dichlorophenol**									51.000000	0.150000	
78-87-5	**1,2-Dichloropropane**		0.620000	0.830000				■	■	220.000000	260.000000	
76-14-2	**Dichlorotetrafluoro-ethane (CFC-114)**								■			•

Appendix B: continued

CAS Number	Chemical	Carcinogens	C. TEP Air	C. TEP Water	Scorecard Lists: R. DEV	R. REP	S. DEV	S. REP	S. NEU	NC. TEP Air	NC. TEP Water	Not in 98-02 data set
306-83-2	**Dichlorotrifluoroethane** (HCFC-123 and isomers)											•
77-73-6	**Dicyclopentadiene**								■			•
111-42-2	**Diethanolamine**								■	310.000000	1.700000	
117-81-7	**Di(2-ethylhexyl) phthalate**	■	0.130000	0.030000	■	■				33.000000	9.000000	
64-67-5	**Diethyl sulfate**	■	1.600000	0.020000								
124-40-3	**Dimethylamine**								■	41.000000	10.000000	•
121-69-7	**N,N-Dimethylaniline**								■	12.000000	4.800000	
68-12-2	**N,N-Dimethylformamide**						■	■	■			•
131-11-3	**Dimethyl phthalate**								■	0.020000	0.001700	
77-78-1	**Dimethyl sulfate**	■	190.000000	0.220000					■			
534-52-1	**4,6-Dinitro-o-cresol**								■	1,400.000000	52.000000	
121-14-2	**2,4-Dinitrotoluene**	■	4.400000	0.040000		■			■	100.000000	0.920000	
606-20-2	**2,6-Dinitrotoluene**	■	9.900000	0.040000		■			■	200.000000	0.940000	
25321-14-6	**Dinitrotoluene** (mixed isomers)					■			■			
123-91-1	**1,4-Dioxane**	■	0.080000	0.090000					■	0.050000	0.050000	
122-39-4	**Diphenylamine**								■	14.000000	14.000000	•
106-89-8	**Epichlorohydrin**	■	1.100000	0.450000		■			■	210.000000	83.000000	
110-80-5	**2-Ethoxyethanol**				■	■			■	1.300000	0.080000	
140-88-5	**Ethyl acrylate**	■	0.070000	0.030000			■		■	1.600000	0.710000	
100-41-4	**Ethylbenzene**	■					■	■	■	0.140000	0.280000	
541-41-3	**Ethyl chloroformate**											
74-85-1	**Ethylene**								■			
107-21-1	**Ethylene glycol**						■	■	■	0.250000	0.004200	
75-21-8	**Ethylene oxide**	■	11.000000	5.500000		■	■		■	56.000000	27.000000	
96-45-7	**Ethylene thiourea**	■	1.200000	0.100000	■			■		4,600.000000	400.000000	
7782-41-4	**Fluorine**											•
50-00-0	**Formaldehyde**	■	0.020000	0.000800				■	■	16.000000	0.290000	
64-18-6	**Formic acid**								■	0.060000	0.001800	•
77-47-4	**Hexachlorocyclopentadiene**						■	■	■	130.000000	120.000000	
67-72-1	**Hexachloroethane**	■	260.000000	230.000000			■	■	■	5,500.000000	4,900.000000	
70-30-4	**Hexachlorophene**						■		■			•
110-54-3	**n-Hexane**						■	■	■	0.030000	6.200000	•
302-01-2	**Hydrazine**	■	22.000000	2.400000			■	■	■	390.000000	140.000000	
7647-01-0	**Hydrochloric acid**									12.000000	110.000000	
74-90-8	**Hydrogen cyanide**							■	■	580.000000	530.000000	
7664-39-3	**Hydrogen fluoride**						■	■	■	3.600000		
123-31-9	**Hydroquinone**		1.200000	0.000250				■	■	7.500000	0.001500	
13463-40-6	**Iron pentacarbonyl**								■			•
78-84-2	**Isobutyraldehyde**											
80-05-7	**4,4'-Isopropylidenediphenol**							■	■	7.900000	0.380000	
120-58-1	**Isosafrole**											
—	**Lead** (and its compounds)	■	28.000000	2.000000	■	■			■	580,000.000000	42,000.000000	•
554-13-2	**Lithium carbonate**				■				■			•
108-31-6	**Maleic anhydride**									22.000000	0.000004	
—	**Manganese** (and its compounds)							■	■	780.000000	3.500000	
149-30-4	**2-Mercaptobenzothiazole**								■			•
—	**Mercury** (and its compounds)				■			■	■	14,000,000.000000	13,000,000.000000	•
67-56-1	**Methanol**						■		■	0.090000	0.010000	
109-86-4	**2-Methoxyethanol**				■	■			■	2.000000	15.000000	
96-33-3	**Methyl acrylate**								■	0.800000	0.330000	
1634-04-4	**Methyl tert-butyl ether**		0.004500	0.008300					■	0.030000	0.110000	
101-14-4	**4,4'-Methylenebis** (2-chloroaniline)	■							■			
101-77-9	**4,4'-Methylenedianiline**	■	21.000000	0.430000					■	2.800000	0.040000	
78-93-3	**Methyl ethyl ketone**						■	■	■	0.050000	0.010000	
74-88-4	**Methyl iodide**		100.000000	54.000000					■			
108-10-1	**Methyl isobutyl ketone**						■		■	0.030000	0.040000	
80-62-6	**Methyl methacrylate**						■	■	■	0.530000	0.930000	
924-42-5	**N-Methylolacrylamide**							■	■			•
109-06-8	**2-Methylpyridine**								■			•
872-50-4	**N-Methyl-2-pyrrolidone**				■			■	■			•
90-94-8	**Michler's ketone**	■										
1313-27-5	**Molybdenum trioxide**								■			
76-15-3	**Monochloropentafluoroethane** (CFC-115)								■			•
91-20-3	**Naphthalene**						■		■	18.000000	22.000000	
—	**Nickel** (and its compounds)	■	2.800000		■				■	3,200.000000	26.000000	

Appendix B: continued

CAS Number	Chemical	Carcinogens	C. TEP Air	C. TEP Water	Scorecard Lists: R. DEV	R. REP	S. DEV	S. REP	S. NEU	NC. TEP Air	NC. TEP Water	Not in 98-02 data set
—	**Nitric acid** and nitrate compounds							■		2.100000		
139-13-9	**Nitrilotriacetic acid**	■										
100-01-6	**p-Nitroaniline**								■			•
98-95-3	**Nitrobenzene**	■						■	■	24.000000	110.000000	
55-63-0	**Nitroglycerin**		15.000000	1.500000					■	3.200000	0.330000	
100-02-7	**4-Nitrophenol**								■	21.000000	6.000000	
79-46-9	**2-Nitropropane**	■	22.000000	57.000000			■	■	■	5.800000	15.000000	
86-30-6	**N-Nitroso diphenylamine**		0.010000	0.120000								
123-63-7	**Paraldehyde**								■			•
76-01-7	**Pentachloroethane**								■			•
79-21-0	**Peracetic acid**											
108-95-2	**Phenol**						■	■	■	0.380000	0.004600	
106-50-3	**p-Phenylenediamine**								■	1.400000	0.020000	
90-43-7	**2-Phenylphenol**		0.000710	0.002000			■	■	■	0.260000	0.720000	
75-44-5	**Phosgene**									300,000.000000	82.000000	
7723-14-0	**Phosphorus** (yellow or white)							■	■			
85-44-9	**Phthalic anhydride**								■	3.000000	0.000032	
—	**Polychlorinated alkanes** (C10 to C13)	■					■					•
7758-01-2	**Potassium bromate**	■										•
107-19-7	**Propargyl alcohol**								■			•
123-38-6	**Propionaldehyde**								■			
115-07-1	**Propylene**									0.020000	0.030000	
75-56-9	**Propylene oxide**	■	0.260000	0.420000			■	■	■	29.000000	18.000000	
110-86-1	**Pyridine**								■	74.000000	8.000000	
91-22-5	**Quinoline**		11.000000	2.900000					■			
106-51-4	**Quinone**								■			
94-59-7	**Safrole**	■	0.310000	1.700000				■	■			
—	**Selenium** (and its compounds)						■	■	■	2,400.000000	1,600.000000	
—	**Silver** (and its compounds)								■	1,600.000000	460.000000	
7632-00-0	**Sodium nitrite**						■		■			•
100-42-5	**Styrene**	■	0.002730	0.005280			■	■	■	0.080000	0.340000	
96-09-3	**Styrene oxide**	■	0.580000	0.110000			■	■		30.000000	5.400000	
7664-93-9	**Sulfuric acid**											
630-20-6	**1,1,1,2-Tetrachloroethane**		3.100000	0.280000					■	56.000000	5.000000	•
79-34-5	**1,1,2,2-Tetrachloroethane**		8.900000	6.300000			■		■	0.900000	1.300000	
127-18-4	**Tetrachloroethylene**	■	0.960000	2.300000			■	■	■	65.000000	49.000000	
64-75-5	**Tetracycline hydrochloride**				■							•
62-56-6	**Thiourea**	■	2.300000	0.010000			■	■				
1314-20-1	**Thorium dioxide**											
7550-45-0	**Titanium tetrachloride**											
108-88-3	**Toluene**				■			■	■	1.000000	0.880000	
584-84-9	**Toluene-2,4-diisocyanate**	■							■			
91-08-7	**Toluene-2,6-diisocyanate**	■										
26471-62-5	**Toluenediisocyanate** (mixed isomers)	■							■			
120-82-1	**1,2,4-Trichlorobenzene**		0.120000	0.290000			■		■	9.600000	78.000000	
79-00-5	**1,1,2-Trichloroethane**		2.100000	2.400000					■	4.900000	14.000000	
79-01-6	**Trichloroethylene**	■	0.050000	0.130000			■	■	■	0.630000	1.200000	
75-69-4	**Trichlorofluoromethane** (CFC-11)								■	9.600000	9.100000	•
121-44-8	**Triethylamine**								■	0.400000	0.030000	•
95-63-6	**1,2,4-Trimethylbenzene**								■	11.000000	300.000000	
—	**Vanadium** (and its compounds)							■	■	1,200.000000	710.000000	•
108-05-4	**Vinyl acetate**	■							■	1.500000	0.750000	
75-01-4	**Vinyl chloride**	■	1.900000	4.600000			■	■	■	69.000000	140.000000	
75-35-4	**Vinylidene chloride**		4.600000	11.000000			■	■	■	2.700000	6.300000	
108-38-3	**Xylenes**						■	■	■	0.410000	0.500000	
—	**Zinc** (and its compounds)						■	■	■	190.000000	14.000000	

Appendix C: Methodology for PRTR Data Analyses

The PRTR data used in this report are collected by the national governments under Canada's NPRI program and the US TRI, respectively. Comparable data are not yet available under the Mexican PRTR program, the *Registro de Emisiones y Transferencia de Contaminantes* (RETC). Reporting under Section V of the Mexican reporting form was voluntary for 2002 and, thus, the data are not comparable to the mandatory data collected under TRI and NPRI.

Each country's PRTR has evolved with a different list of chemicals and industries. In order to obtain a North American picture of releases and transfers of chemicals, not all data submitted to the individual countries' PRTR systems can be used, however; only those data common to both systems. This matching process eliminates chemicals reported under one system but not the other. It also eliminates data from industry sectors covered by one PRTR but not the other. Thus, the North American database used in this report consists of a matched data set of industries and chemicals common to NPRI and TRI.

These PRTR reports were submitted by facilities during the summer of 2003. The US EPA released the TRI data to the public in June 2004. The NPRI data used in this report were obtained from the Environment Canada web site in July 2004. At the same time, updated versions of previous years' data for TRI and NPRI were also made available and used in this report.

Matching by Industry

The Canadian NPRI data include both the Standard Industry Classification (SIC) code under the Canadian system as well as the US SIC Code for each facility. Since facilities reporting to the US TRI list only the US SIC code, that industry code is used in this report as an identifier for both NPRI and TRI facilities. Only industry sectors that are common to both TRI and NPRI are part of the matched data set. Which industry sectors are included depends on which years of data are being analyzed, because both TRI and NPRI have added industry sectors over the years.

Tables with just the year 2002 data include the following industry sectors:

- manufacturing (US SIC codes 20–39),
- coal mining,
- electric utilities,
- hazardous waste treatment and solvent recovery facilities,
- chemical wholesalers, and
- petroleum bulk terminals.

NPRI added reporting by petroleum bulk terminals beginning with the 2002 reporting year. Therefore, for the 1998–2002 data set, all of the above industries except petroleum bulk terminals are included.

Matching for Chemicals

The matched data set includes only those substances on both the TRI and NPRI lists. NPRI covers over 260 chemical substances and TRI approximately 650. The matched data set for 2002 includes 203 substances.

Over the years, the PRTRs have added new chemicals and changed reporting requirements. To look at changes over time, it is necessary to select only those chemicals that have been consistently reported over time. The 1998–2002 data set, which looks at changes over the time period 1998–2002, contains 153 chemicals. (See **Appendix B** for the list of chemicals.)

TRI facilities report certain chemicals and their compounds separately, while in NPRI, a chemical and its compounds count as one category. For example, TRI lists both nickel and nickel compounds, counting them as two separate substances, while NPRI lists the single category, nickel and its compounds. Analyses of the PRTR data in this report add the TRI amount reported for the given chemical to the amount reported for its compounds, to correspond with NPRI practice.

Facilities that report to PRTRs are free to revise their previous years' submissions at any time. They may correct previous errors, or they may re-calculate earlier years' data using a different estimation method. Thus, some of the data in this report may, in the future, be revised by the facility. Current databases are available online at ‹www.cec.org/takingstock/›.

Chemicals Grouped by Health Effect

From the list of matched chemicals, four subsets with different health effects are selected and used to analyze PRTR data in this report:

- Carcinogens
- Recognized developmental and reproductive toxicants
- Suspected developmental and reproductive toxicants
- Suspected neurotoxicants

This report considers as **carcinogens** those chemicals reported to NPRI or TRI that are on lists from the International Agency for Research on Cancer (Groups 1, 2A and 2B) ‹www.iarc.fr/› and the US National Toxicology Program ‹http://ntp-server.niehs.nih.gov/›. Of the 203 chemicals in the matched TRI and NPRI 2002 data set, 55 are on these lists. Chromium and its compounds are not included as a carcinogen even though they may be individually listed, because they are no longer reported as a single category under NPRI. NPRI reports on hexavalent chromium (the chromium species that is carcinogenic) separately from other chromium compounds. Under TRI, all chromium compounds are reported as a single amount.

The list of chemicals considered as **recognized or suspected developmental and reproductive toxicants** in this report was compiled by a US nongovernmental group, Environmental Defense, in consultation with other agencies. This list, posted on their Scorecard web site as of July 2004, is a combination of the recognized California Proposition 65 list and chemicals derived

from other government and academic references. It identifies chemicals which are considered recognized developmental toxicants and those chemicals with less weight of evidence which are considered suspected development toxicants. Of the more than 300 chemicals on this list, 21 chemicals matched the TRI and NPRI data and form the basis of the recognized developmental and reproductive toxicant analysis and 74 matching chemicals form the basis of the suspected developmental and reproductive toxicant analysis. The full Scorecard lists of recognized and suspected developmental toxicants is available at ‹http://www.scorecard.org/health-effects/›.

Environmental Defense also compiled a list of chemicals considered to be **suspected neurotoxicants**, as of July 2004, in consultation with other agencies. As there is no recognized authoritative list of neurotoxicants, this Scorecard list of suspected neurotoxicants was compiled from government and academic sources. Of the over 300 chemicals on this list, 146 chemicals matched the TRI and NPRI data and thus form the basis of the suspected neurotoxicant analysis. The full Scorecard list of suspected neurotoxicants is available at ‹http://www.scorecard.org/health-effects/›.

Scorecard lists chemicals and specific chemical compounds on its list of developmental and reproductive toxicants and neurological toxicants. The PRTRs require reporting as one group for the metal and its compounds. The following metals and their compounds were included for purposes of analysis (**Appendix B**) based on the compounds on the Scorecard list (only selected ones could be treated in the main text of the report):

- Antimony and its compounds
- Cobalt and its compounds
- Copper and its compounds
- Lead and its compounds
- Manganese and its compounds
- Mercury and its compounds
- Nickel and its compounds
- Selenium and its compounds
- Vanadium and its compounds
- Zinc and its compounds

Appendix B also provides a list of chemicals reported to both TRI and NPRI in 2002 and indicates whether they are considered to be carcinogens, recognized or suspected developmental and reproductive toxicants, or suspected neurotoxicants for the purposes of this report.

Rankings of Toxic Equivalency Potentials

In addition to grouping chemicals by health effect (i.e., PRTR carcinogens), a further ranking for two of the groups (carcinogens and recognized developmental and reproductive toxicants) is presented based on a system that takes into effect both a chemical's toxicity and its potential for human exposure using toxic equivalency potentials (TEPs). A discussion of TEPs and the methodology for calculating them will be found in **Appendix D**.

Descriptions of Releases and Transfers Used in this Report

Releases On- and Off-site

A release is the entry of a chemical substance into the environment. Facilities report amounts of the listed chemicals they have released to the environment at their own location ("on-site"). Amounts are reported separately for each environmental medium:

- **Air emissions**—Releases to air that occur through identified outlets such as stacks ("smokestacks") or vents are labeled "stack" or "point" emissions. Air releases that occur through of leaks or valves are labeled "fugitive" or "non-point" emissions.
- **Surface water discharges**—Releases to surface water bodies such as rivers and lakes generally occur through discharge pipes. Wastewater is usually treated first, to remove or minimize its pollutant content. Rainwater may also wash pollutants from on-site waste storage areas into surface waters. These releases from run-off are also reportable.
- **Underground injection**—Facilities may inject listed chemicals in waste into deep underground wells, a practice more common in certain parts of the United States than in Canada. Underground injection is regulated, and deep wells that receive toxic waste are intended to isolate the pollutants from groundwater sources. Underground injection is not practiced in Mexico.
- **On-site land releases**—Releases to land at the facility include burying chemical waste in landfills, incorporating it into soil ("land treatment"), holding it in surface impoundments, accumulating it in waste piles, or disposing of it by other methods. NPRI and TRI report on-site land releases differently. NPRI has the separate categories: landfill, land treatment, spills, leaks, and other. TRI has: RCRA Subtitle C landfills, other landfills, land treatment/application farming, RCRA Subtitle C surface impoundments, other surface impoundments, and other disposal. To make the TRI and NPRI data comparable, the separate categories in each of the two PRTR systems are added together for on-site land releases in this report.

Facilities also report transfers off-site that represent releases to the environment at the off-site location. These include:

- **Disposal**—Waste sent off-site to another facility for disposal may be disposed of on land or by underground injection. These methods are the same as on-site land releases and underground injection, although they occur at locations away from the originating facility. For NPRI, the categories containment (landfill and other storage), underground injection and land treatment are included. For TRI, all disposal codes, such as landfills, surface impoundments, land treatment, underground injection, storage, and solidification/stabilization, are included

- **Transfers of Metals**—Transfers of metals to disposal (see above for individual PRTR categories), sewage, treatment, and energy recovery are included in the off-site releases category to make the TRI and NPRI data comparable. TRI classifies all transfers of metals as transfers to disposal because metals sent to energy recovery, treatment, or sewage treatment may be captured and removed from waste and disposed of in landfills or by other disposal methods, but are not destroyed by treatment processes or burned in energy recovery units.

Transfers for Further Management

- **Recycling**—Chemicals in the materials sent off-site for recycling are generally recovered by a variety of recycling methods, including solvent recovery and metals recovery. They can be sent off-site for processing, cleaning, or reclamation and returned to the originating facility or made available for use by other facilities.
- **Energy Recovery**—Chemicals in materials sent off-site for energy recovery are combusted in industrial furnaces (including kilns) or boilers that generate heat or energy for use at the off-site location. Energy recovery is applicable only when the material has a significant heating value and when it is used as an alternate for fossil fuel or other forms of energy.
- **Treatment**—Chemicals can be sent for physical, chemical, or biological treatment. Neutralization is an example of chemical treatment and incineration is an example of physical treatment. Treatment is intended to alter or destroy the chemical. Treatment processes must be appropriate for the particular substance—a chemical that will not burn, for example, cannot be successfully incinerated.
- **Sewage Treatment**—Facilities may send their chemical waste to sewage treatment facilities—municipal sewage treatment plants (MSTPs) in Canada or publicly owned treatment works (POTWs) in the United States. The effectiveness of sewage treatment depends on both the substance and the sewage plant's processes. Volatile chemicals are likely to evaporate (releases to air). Typically, secondary treatment processes apply microorganisms (with aeration or oxygenation) to biodegrade organic compounds.

Please note that this terminology and categorization has been developed to render the data comparable and is specific to this report and may differ from terminology used by the individual PRTR programs.

Appendix D: Methodology for Toxic Equivalency Potentials (TEPs)

(Adapted from ‹www.scorecard.org›)

The toxic equivalency potential (TEP) indicates the relative human health risk associated with a release of one pound of a chemical, compared to the risk posed by release of a reference chemical. Information about the toxicity of a chemical and its exposure potential are used to make this comparison (Hertwich *et al.* 1998). The TEPs for this report were taken from the Scorecard web site in January 2005. One additional set of TEPs not on the web site, for styrene as a carcinogen in air and water releases, was provided by Bill Pease using EPA's risk assessment (Caldwell *et al.* 1998) and the same methodology.

TEPs are calculated using CalTOX, an environmental fate and exposure model developed by California regulatory agencies. The model is basically a screening-level risk assessment that estimates the cancer and/or non-cancer health risks associated with the total dose of a chemical that people receive if one pound of that chemical is released to air or water in a model environment. The CalTOX model has been evaluated by the Integrated Human Exposure Committee of US EPA's Science Advisory Board and described as "potentially the most advanced of all of the models reviewed."

The CalTOX model produces estimates of the health risks posed by a unit release of a chemical to air or water. The TEPs then are the ratio of the risk posed by a one-pound release of the chemical to the risk posed by a one-pound release of a reference chemical. Separate TEPs are calculated for chemical releases to air and water. The reference chemical for carcinogens in benzene and the reference chemical for non-carcinogens is toluene.

The individual TEPs are multiplied times the amount of air or water releases to obtain TEP-weighted releases. The tables in this report show the rank based on the TEP-weighted releases compared to the rank based on the reported releases. For metal compounds, the TEP applied is the TEP for the metal where that is the only one available. As a result of data gaps or modeling problems, not all chemicals possess the information required to weight their mass release by toxicity and exposure potential. Chemicals that lack risk scores should not be assumed to be safe.

Appendix E: Assessment and Management of Industrial Chemicals and Pollution Reporting, by Country

Canada

Overview

In Canada, the federal government, as well as provincial, territorial and Aboriginal governments, share responsibility for protecting the environment—an approach that calls for close collaboration as governments work to support the well-being of Canadians. As a cornerstone of the Government of Canada's environmental legislation, the Canadian Environmental Protection Act of 1999 is aimed at preventing pollution and protecting the environment and human health. The Act is jointly administered by Health Canada and Environment Canada.

One of the major thrusts of the Act is the prevention and management of risks posed by harmful substances. As well, the Act provides for the assessment and/or management of the environmental and human health impacts of new and existing substances. This includes chemicals, products of biotechnology, marine pollution, disposal at sea, vehicle, engine and equipment emissions, fuels, hazardous wastes, environmental emergencies and other sources of pollution.

New Substances

With respect to substances proposed for introduction to Canada, the federal government is committed to ensuring that no new substances (chemicals, polymers or animate products of biotechnology) are manufactured or imported into Canada before an assessment of whether they present potential impacts to human health or the environment has been completed, and any appropriate risk management measures are put in place. The New Substances Notification Regulations (NSNR) pursuant to the Canadian Environmental Protection Act, 1999 (CEPA), an integral component of the federal government's national pollution prevention strategy, ensure that this objective is met. The NSNR are administered jointly by Environment Canada and Health Canada.

The NSNR, however, do not apply to substances or products regulated under other federal Acts that meet CEPA-equivalent requirements for notification and assessment, and that are listed in Schedules 2 or 4 to CEPA. An example of this would be pesticides, which are regulated under the Pest Control Products Act and are administered by the Pest Management Regulatory Agency (PMRA), part of Health Canada.

Existing Substances

Similar regulatory efforts are in place to address the legacy of so-called "existing" substances that have not been previously assessed for risks to human health and the environment. In Canada, "existing" substances include primarily those on the Domestic Substances List (DSL), which was compiled between January 1984 and 31 December 1986, of substances used, imported or manufactured in Canada for commercial purposes in quantities greater than 100 kg per year, as well as contaminants, byproducts, emissions, effluents and wastes. CEPA requires that the Government of Canada examine all substances on the DSL by September 2006, to see whether they possess certain characteristics that require a risk assessment (e.g., greatest potential for human exposure, persistence in the environment, potential to accumulate in living tissue, or whether a substance's very nature makes it harmful).

This examination of the DSL in this fashion, an enormous undertaking that has not been attempted by any other single government in the world, is the first step in a process by which the government will systematically organize information on existing substances in order to identify those chemicals that may need further scientific research, those that could be candidates for early action, and those that should be given priority for risk assessment.

Risk Management

Substances that are found to pose risks to human health or the environment will follow the Toxics Management Process, which is jointly administered by Environment Canada and Health Canada. The Toxics Management Process is used to develop tools for managing toxic substances, including preventive or control instruments. Using this process, Environment Canada and Health Canada develop risk management actions in a manner that ensures stakeholder consultations are effective, and that timelines for managing toxic substances are met.

Central to the Toxics Management Process is the development of a risk management strategy document. The strategy describes how risks to human health and the environment, which are posed by the use and/or release of each substance of concern, will be addressed. Public consultations will be carried out on the proposed risk management objectives, tools or instruments of the risk management strategy.

Further information on risk management activities related to substances of concern can be found at ‹http://www.ec.gc.ca/toxics›.

Right-to-Know

"Public-right-to-know" is firmly established in Canada's environmental legislation. CEPA requires the Minister of the Environment to distribute information on pollution prevention; publish periodic reports on the state of the environment; and to maintain and publish the National Pollutant Release Inventory (NPRI). The NPRI (searchable by postal code or substance) provides Canadians with facility-specific information regarding on-site releases and off-site transfers of over 300 substances listed on the inventory. Companies that manufacture, process or otherwise use a listed substance at or above the reporting threshold must report their releases or transfers to Environment Canada annually. CEPA also requires the Minister of Health to distribute available information to the public about the effects of substances on human health. Together,

these legislative requirements promote public participation and give Canadians access to environmental information related to their communities.

International Conventions

Canada is an active participant in a number of multilateral environmental agreements related to health, pollution and chemicals. As a Party to the Stockholm Convention on POPs and the UNECE POPs Protocol, Canada no longer produces or uses the POP industrial chemicals and pesticides and is taking measures to control and reduce emissions of unintentionally produced POPs, such as dioxins and furans.

Canada has been a leader in the worldwide effort to eliminate the releases of ozone-depleting substances (ODSs) to protect the stratospheric ozone layer and minimize UV radiation exposure levels. Under the1985 Vienna Convention and its 1987 Montreal Protocol on Substances that Deplete the Ozone Layer, production, importation and exportation (consumption) of most ODSs, beginning with CFCs and halons, the worst of the ODSs, has been banned in Canada. Canada has reduced its consumption of ODSs by over 98 percent through implementation of Canada's ozone layer protection program; a program developed in partnership with provincial/territorial governments.

Finally, to meet its obligations under the Convention, Canada has developed the Export of Substances under the Rotterdam Convention Regulations, under the Canadian Environmental Protection Act, 1999. As provided in the Rotterdam Convention, Canadian exporters of a substance that requires Prior Informed Consent (PIC) need to obtain a permit to export the substance to countries that are Parties to the Convention, which is issued if the importing country accepts those imports.

In addition, recognizing that many pollutants, such as POPs, originate from other countries, and that global problems need global solutions, Canada supports capacity building in developing countries and countries with economies in transition to assist them in implementing the provisions of the various Conventions.

Mexico

Overview

The Federal Commission for Sanitary Risk Protection (Cofepris) is a body of the Ministry of Health that oversees, among other responsibilities, the regulation of such substances as pesticides, fertilizers, toxic substances, hazardous materials and drugs. Cofepris is responsible for issuing import and export approval of these substances under the *Reglamento en Materia de Registros, Autorizaciones de Importación, Exportación y Certificados de Exportación de Plaguicidas, Nutrientes Vegetales, Sustancias Tóxicas y Materiales Tóxicos o Peligrosos*.

This regulation provides for consultations with other federal ministries, such as the Ministry of Environment (Semarnat) and the Ministry of Agriculture (Sagarpa).

New Substances

All new substances intended to be registered in Mexico must undergo a registration process mandated by the General Health Law and be incorporated into the official list of registered substances. For the existing substances already in place, Cofepris is responsible for maintaining the official list. The regulation has been published in the Official Gazette (*Diario Oficial* 2004).

Existing Substances

The regulatory process involves a risk assessment analysis for the decision to register or to cancel existing chemical registrations. Cofepris is organized into two main branches: the risk analysis and risk management area, and the authorization and registration area. They interact jointly to decide questions about registrations and import and export certificates.

The results and the status of the process regarding specific substances, as well as the official lists of toxic substances, are posted for public consultation on the Cofepris web site, at ‹http://www.cofepris.gob.mx/›.

Risk Management

The enforcement of all specific and general regulations regarding toxic substances is the responsibility of Cofepris by the General Health Law, and for that purpose, Cofepris has an inspection department that leads inspections and applies penalties in case of any violation.

Right-to-Know

The federal law (*Ley de Transparencia y Acceso Público a la Información*) specifies the public right to request and obtain clear information related to the different types of risks among toxic substances. In addition, there are several mandatory Mexican official norms, related to the labeling of substances in order to communicate possible or probable risks, NOM-045-SSA1-1993 and NOM-046-SSA1-1993.

International Conventions

Mexico, through Cofepris and Semarnat, is actively involved in international protocols, such as the Rotterdam Convention, Stockholm Convention, Basel Convention, and GHS initiative.

United States

Overview

The US Environmental Protection Agency (EPA) is charged with implementing the Toxic Substances Control Act (TSCA), enacted by the US Congress in fall 1976, and subsequent amendments, as well as the Pollution Prevention Act (PPA) of 1990. To accomplish its work, EPA's Office of Pollution Prevention and Toxics (OPPT) has a strategic framework of statutory and regulatory tools as well as voluntary and partnership approaches.

Existing Substances

Under TSCA, EPA is responsible for assuring that chemicals manufactured, imported, processed, or distributed in commerce, or used or disposed of in the United States, do not pose any unreasonable risks to human health or the environment. TSCA provides EPA authority to compile an inventory of existing chemical substances manufactured for commercial purposes. Currently, the TSCA Chemical Substance Inventory lists approximately 81,600 chemical substances as being available for sale and use in the United States at some time since the Inventory was first published in 1979. The inventory grows as new chemicals enter into commerce and are added to the list on an ongoing basis.

Beginning in 1986, OPPT has been updating the inventory at intervals of every four years to obtain basic information about those chemicals that are actively being manufactured, produced, processed or imported during a specified reporting period. The updates are gathered through the Inventory Update Rule (IUR) and include data on the production volume and site location for chemical substances manufactured or imported at levels of 10,000 pounds or more per year per site. In 2003, EPA amended the TSCA IUR, the IUR Amendments (IURA), to modify the reporting threshold from the original 10,000 pounds per year per site to 25,000 pounds per year, and also require reporting of processing and use information for substances above the reporting threshold of 300,000 pound per year. In the 2003 IURA, EPA also added requirements for the reporting of inorganic chemicals and additional exposure-related information, and modified several IUR reporting and record-keeping requirements. The inventory updates provide a more contemporary picture of a smaller subset of the 81,600 inventoried chemicals that are in active commerce and are used by OPPT for priority-setting.

OPPT has implemented TSCA by developing programs addressing existing chemicals with reporting and testing requirements and the Office also manages focused risk-reduction efforts for several toxic chemicals of national concern, including PCBs, lead, and asbestos.

New Substances

Under TSCA, OPPT has addressed new chemicals (that must be reviewed by EPA before they are produced or imported and added to the Inventory) through programs to assess, test, and manage identified potential risks from those chemicals new to commerce, including biotechnology products resulting from industrial processes.

Risk Management

Over the last decade, focus has shifted from individual chemicals to controlling larger numbers of related chemicals, through testing, assessment and risk management efforts. Examples include chemicals produced in high volume, chemicals that exhibit certain behavior characteristics (e.g., persistent, bioaccumulative and toxic (PBT) chemicals, and persistent organic pollutants (POPs), initiated at the domestic and international levels, respectively). Also, EPA has increased its emphasis on pollution prevention (P2) and environmental stewardship by empowering companies to develop and use safer or greener products. The Agency recognizes that a program of integrated voluntary and regulatory actions, with greater emphasis on stakeholder involvement, is necessary to better promote environmental stewardship.

The Pollution Prevention Act (PPA) establishes the national policy that pollution should be prevented or reduced at the source whenever feasible. The PPA includes authority for EPA to facilitate the adoption of source reduction techniques by businesses and by EPA and other federal agencies; to identify opportunities to use federal procurement policies to encourage source reduction; to ensure that the Agency considers the effect of its regulations and its existing and proposed programs on source reduction; to develop improved methods of coordinating, streamlining, and assuring public access to data collected under federal environmental statutes; and to provide grants to States for programs to promote the use of source reduction techniques by businesses.

OPPT is also strongly committed to promoting public understanding of chemical risks by developing and providing scientifically sound, accessible, and comprehensive information to the broadest audience possible.

Right-to-Know

The Emergency Planning and Community Right-to-Know Act of 1986 was passed in response to concerns regarding the environmental and safety hazards posed by the storage and handling of toxic chemicals. These concerns were triggered by the disaster in Bhopal, India, in which more than 2,000 people suffered death or serious injury from the accidental release of methyl isocyanate. To reduce the likelihood of such a disaster in the United States, Congress imposed requirements on both states and regulated facilities.

EPCRA establishes requirements for federal, state and local governments, Indian tribes, and industry regarding emergency planning and "Community Right-to-Know" reporting on hazardous and toxic chemicals. The Community Right-to-Know provisions help increase the public's knowledge and access to information on chemicals at individual facilities, their uses, and releases into the environment. States and communities, working with facilities, can use the information to improve chemical safety and protect public health and the environment.

EPCRA has four major provisions:

- Emergency planning (Sections 301–303)
- Emergency release notification (Section304)
- Hazardous chemical storage reporting requirements (Sections 311–312), and
- Toxic chemical release inventory (Section 313).

For additional information, check the EPA web site, at: ‹http://yosemite.epa.gov/ oswer/ceppoweb.nsf/content/index.html›.

International Conventions

In 2001, the US joined forces with 90 other countries and the European community to sign the groundbreaking United Nations Stockholm Convention on Persistent Organic Pollutants. In 1998, the United States signed the legally binding regional protocol with other member nations (including European countries, Canada, and Russia) of the United Nations Economic Commission for Europe (UNECE) on POPs under the Convention on Long-Range Transboundary Air Pollution (LRTAP). This agreement seeks to eliminate production and reduce emissions of POPs in the UNECE region and addresses the 12 Stockholm Convention POPs and four additional chemicals (hexachlorocyclohexanes, hexabromobiphenyl, chlordecone, and polycyclic aromatic hydrocarbons). Elements from the LRTAP POPs Protocol were used in negotiations for the Stockholm Convention.

Other international work has addressed trade in hazardous substances, some of which are POPs. The United States, along with 71 other countries and the European Community, has signed the Rotterdam Convention on the Prior Informed Consent (PIC) Procedure for Certain Hazardous Chemicals and Pesticides in International Trade, building on a 10-year-old voluntary program. The PIC Convention identifies pesticides and industrial chemicals of concern, facilitates information sharing about their risks, and provides countries with an opportunity to make informed decisions about whether they should be imported. Some of the POP substances are already on the PIC list.

The United States has also provided technical and financial assistance for POPs-related activities to a variety of countries and regions, including Mexico, Central and South America, Russia, Asia, and Africa.

Tables

Table 1-1 Annual Mortality Rates Reflecting Specific Causes of Death for Children in North America (Rate per 100,000), 2001

Cause of Death	*Infant (<1 yr)*	*Pre-schooler (1–4 yrs)*	*School age (5–14 yrs)*
Congenital malformations			
Canada	137.3	3.0	0.9
Mexico	339.5	10.1	2.2
United States	136.7	3.6	0.9
Certain perinatal disorders▲			
Canada	291.4	0.4	0.02
Mexico	872.4	N/A	N/A
United States	340.5	0.5	0.1
Infectious intestinal diseases			
Canada*	0	0.1	0.02
Mexico	75.6	7.4	0.6
United States	4.7	0.4	0.2
Acute (lower) **respiratory infections**			
Canada	N/R	N/R	N/R
Mexico	135.2	6.7	0.8
United States	1.2	N/R	N/R
Septicemia			
Canada	2.5	0.4	0.1
Mexico	29.1	1.7	N/A
United States	7.7	0.7	0.2
Influenza and pneumonia			
Canada	5	0.3	0.05
Mexico	121.7	6.5	0.8
United States	7.4	0.7	0.2
Unintentional injuries			
Canada	11	8.4	5.9
Mexico	74.4	19.3	10.8
United States	24.2	11.2	6.9
Malnutrition, anemia and other nutritional deficiencies			
Canada	0.3	0	0.02
Mexico	38.4	4.6	1.0
United States	N/R	0.1	0.1
Tumors			
Canada	5.3	3.3	2.5
Mexico	N/A	5.5	5.0
United States	2.9	3.1	2.8
Chronic bronchitis, non-specific, and asthma			
Canada	0.8	0.1	0.02
Mexico	3.8	0.9	0.2
United States (1999)	1	0.3	0.3

Sources: Compiled with data from Statistics Canada 2001, INEGI 2000, and Arias et al. 2003. **N/A** – Statistics not available. **N/R** – Statistics not reliable due to sparse numbers.
▲ Certain perinatal disorders are deaths related to short gestation and low birth weight, not otherwise classified. * Rate is tabulated as "Infectious and parasitic diseases."

Table 3-1 Summary of Releases and Transfers of Carcinogens Reported to North American PRTRs, 2002
(2002 Matched Chemicals and Industries)

	North America		Canadian NPRI		United States TRI		NPRI as % of North American Total	TRI as % of North American Total
	(tonnes)	(%)	(tonnes)	(%)	(tonnes)	(%)		
Total On-site Releases*	**117,015**	**4**	**11,530**	**3**	**105,485**	**4**	**10**	**90**
■ Air	62,297	2	9,283	3	53,014	2	15	85
■ Surface Water	691	0.02	113	0.03	578	0.02	16	84
■ Underground Injection	15,043	0.5	99	0.03	14,944	1	1	99
■ Land	38,958	1	2,009	1	36,949	1	5	95
Total Off-site Releases	**36,260**	**1**	**4,281**	**1**	**31,979**	**1**	**12**	**88**
■ Transfers to Disposal (except metals)	4,420	0.1	1,272	0.4	3,148	0.1	29	71
■ Transfers of Metals to Disposal, Energy Recovery, Treatment and Sewage	31,840	1	3,009	1	28,831	1	9	91
Total Releases On- and Off-site	**153,274**	**5**	**15,811**	**4**	**137,463**	**5**	**10**	**90**
Transfers to Recycling	**255,445**	**8**	**42,601**	**12**	**212,844**	**7**	**17**	**83**
■ Transfers to Recycling of Metals	229,787	7	42,060	12	187,727	6	18	82
■ Transfers to Recycling (except metals)	25,658	1	541	0.2	25,117	1	2	98
Other Transfers Off-site for Further Management	**63,856**	**2**	**2,456**	**1**	**61,400**	**2**	**4**	**96**
■ Transfers to Energy Recovery (except metals)	32,343	1	795	0	31,548	1	2	98
■ Transfers to Treatment (except metals)	27,717	1	1,526	0.4	26,191	1	6	94
■ Transfers to Sewage (except metals)	3,795	0.1	134	0.04	3,661	0.1	4	96
Total Reported Amounts of Releases and Transfers of Carcinogens	**472,575**	**15**	**60,868**	**17**	**411,707**	**14**	**13**	**87**
Total Reported Amounts of Releases and Transfers of All Matched Chemicals	**3,250,183**	**100**	**355,883**	**100**	**2,894,300**	**100**	**11**	**89**

Note: Canada and US data only. Mexico data not available for 2002. Data include 55 chemicals common to both NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. A chemical is considered a carcinogen for the purposes of this report if it is so classified by the International Agency for Research on Cancer (IARC) **<http://www.iarc.fr/>** or the US National Toxicology Program (NTP) **<http://ntp-server.niehs.nih.gov />**. Substances classified under IARC as carcinogenic to humans (1), probably carcinogenic to humans (2A), and possibly carcinogenic to humans (2B) are included. Under the US National Toxicology Program, substances classified as known to be carcinogenic (K) or may reasonably be anticipated to be carcinogenic (P) are included. *** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount.**

Table 3-2 Chemicals with Largest Releases and Transfers of Carcinogens Reported to North American PRTRs, 2002
(2002 Matched Chemicals and Industries)

		Total Reported Amounts of Releases and Transfers		On-site Releases				Off-site Releases	Transfers to Recycling	Other Transfers Off-site for Further Management	Canadian NPRI Total Reported Amounts of Releases and Transfers*		United States TRI Total Reported Amounts of Releases and Transfers	
				Air	Surface Water	Underground Injection	Land							
CAS Number	Chemical	(tonnes)	(rank)	(tonnes)	(tonnes)	(tonnes)	(tonnes)	(tonnes)	(tonnes)	(tonnes)	(tonnes)	(rank)	(tonnes)	(rank)
—	**Lead (and its compounds)**	211,157	1	961	67	139	23,645	23,543	162,802	0	37,048	1	174,109	1
—	**Nickel (and its compounds)**	82,850	2	994	124	241	10,426	7,746	63,317	0	10,543	2	72,307	2
100-42-5	**Styrene**	33,067	3	23,511	2	73	91	852	1,538	6,996	2,535	3	30,532	3
75-09-2	**Dichloro-methane**	27,913	4	6,030	2	138	2	84	7,571	14,085	1,431	6	26,483	4
100-41-4	**Ethylbenzene**	13,723	5	3,679	5	431	5	72	2,384	7,140	1,675	5	12,047	5
50-00-0	**Formaldehyde**	13,571	6	6,403	195	3,584	57	285	53	2,991	2,158	4	11,413	6
75-07-0	**Acetaldehyde**	8,792	7	6,716	189	326	7	2	2	1,551	942	9	7,850	8
108-05-4	**Vinyl acetate**	8,147	8	1,568	0	208	4	49	2	6,315	236	12	7,911	7
71-43-2	**Benzene**	7,745	9	3,379	10	374	21	97	1,759	2,103	1,029	8	6,716	10
106-99-0	**1,3-Butadiene**	7,714	10	953	1	17	0	1	6,129	611	91	17	7,623	9
79-01-6	**Trichloroethylene**	6,975	11	4,317	0	64	0	77	1,070	1,444	902	10	6,072	13
117-81-7	**Di(2-ethylhexyl) phthalate**	6,564	12	293	1	0	12	387	1,525	4,346	166	14	6,398	11
—	**Cobalt (and its compounds)**	6,327	13	66	21	20	2,000	551	3,668	0	244	11	6,083	12
127-18-4	**Tetrachloroethylene**	6,108	14	1,078	0	68	66	174	2,237	2,485	226	13	5,883	14
107-13-1	**Acrylonitrile**	5,847	15	314	0	4,941	0	9	2	581	116	16	5,732	15
1332-21-4	**Asbestos (friable)**	4,156	16	0	0	0	2,539	1,617	0	0	1,140	7	3,016	18
79-06-1	**Acrylamide**	3,985	17	6	0	3,917	0	3	0	59	0	35	3,985	16
75-01-4	**Vinyl chloride**	3,357	18	316	0	63	0	0	3	2,974	12	24	3,345	17
107-06-2	**1,2-Dichloroethane**	2,578	19	213	2	97	0	14	1,133	1,119	40	20	2,538	19
64-67-5	**Diethyl sulfate**	2,342	20	8	0	0	0	0	0	2,334	0	—	2,342	20
67-66-3	**Chloroform**	1,982	21	640	9	85	28	14	26	1,180	121	15	1,861	21
75-56-9	**Propylene oxide**	1,484	22	133	9	1	36	6	0	1,300	5	28	1,479	22
123-91-1	**1,4-Dioxane**	1,118	23	49	34	0	1	437	0	597	1	33	1,117	23
140-88-5	**Ethyl acrylate**	772	24	57	0	0	0	60	0	655	0	36	771	24
56-23-5	**Carbon tetrachloride**	698	25	202	0	78	0	4	0	413	28	21	670	25
Subtotal for Top 25		**468,971**		**61,884**	**672**	**14,865**	**38,940**	**36,084**	**255,224**	**61,277**	**60,688**		**408,283**	
All Others		**3,604**		**413**	**19**	**178**	**18**	**176**	**221**	**2,579**	**180**		**3,424**	
Total for Carcinogens		**472,575**		**62,297**	**691**	**15,043**	**38,958**	**36,260**	**255,445**	**63,856**	**60,868**		**411,707**	
Total for All Matched Chemicals		**3,250,183**		**753,310**	**106,557**	**80,719**	**334,154**	**269,421**	**1,065,424**	**641,475**	**355,883**		**2,894,300**	

Note: Canada and US data only. Mexico data not available for 2002. Data include 55 chemicals common to both NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. A chemical is considered a carcinogen for the purposes of this report if it is so classified by the International Agency for Research on Cancer (IARC) **<http://www.iarc.fr/>** or the US National Toxicology Program (NTP) **<http://ntp-server.niehs.nih.gov />**. Substances classified under IARC as carcinogenic to humans (1), probably carcinogenic to humans (2A), and possibly carcinogenic to humans (2B) are included. Under the US National Toxicology Program, substances classified as known to be carcinogenic (K) or may reasonably be anticipated to be carcinogenic (P) are included. *** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount.**

Table 3-3 Carcinogens Reported to North American PRTRs, Ranked by Releases and Toxic Equivalency Potentials (TEPs), 2002 (2002 Matched Chemicals and Industries)

CAS Number	Chemical	Number of Forms	Total Releases On- and Off-site (tonnes)	(rank)	Total Releases On-site (tonnes)	(rank)	On-site Air Releases (tonnes)	(rank)	(TEP)	(TEP rank)	On-site Surface Water Discharges (tonnes)	(rank)	(TEP)	(TEP rank)
—	Lead (and its compounds)	8,783	48,355	1	24,812	1	961	11	28.000	2	67	4	2.000	1
100-42-5	Styrene	1,720	24,532	2	23,680	2	23,511	1	0.003	23	2	17	0.005	26
—	Nickel (and its compounds)	3,809	19,533	3	11,788	3	994	10	2.800	4	124	3		missing
50-00-0	Formaldehyde	938	10,527	4	10,242	4	6,403	3	0.020	17	195	1	0.001	19
75-07-0	Acetaldehyde	363	7,239	5	7,238	5	6,716	2	0.010	22	189	2	0.006	14
75-09-2	Dichloromethane	578	6,258	6	6,173	6	6,030	4	0.200	7	2	13	0.130	18
107-13-1	Acrylonitrile	117	5,265	7	5,256	7	314	15	3.900	6	0.4	21	1.600	16
79-01-6	Trichloroethylene	525	4,460	8	4,383	8	4,317	5	0.050	15	0.3	24	0.130	22
100-41-4	Ethylbenzene	1,775	4,199	9	4,127	9	3,679	6		missing	5	12		missing
1332-21-4	Asbestos (friable)	103	4,156	10	2,539	12	0.3	44		missing	0	—		missing
79-06-1	Acrylamide	86	3,926	11	3,923	10	6	30	130.000	10	0.1	33	1.600	20
71-43-2	Benzene	1,079	3,883	12	3,786	11	3,379	7	1.000	3	10	7	0.760	5
—	Cobalt (and its compounds)	772	2,659	13	2,107	13	66	22	0.000	missing	21	6		missing
108-05-4	Vinyl acetate	195	1,831	14	1,782	14	1,568	8	0.000	missing	0.5	20		missing
127-18-4	Tetrachloroethylene	381	1,387	15	1,212	15	1,078	9	0.960	8	0.4	22	2.300	15
106-99-0	1,3-Butadiene	224	973	16	972	16	953	12	0.530	14	1	18	4.800	9
67-66-3	Chloroform	119	776	17	762	17	640	13	1.600	9	9	9	1.500	3
117-81-7	Di(2-ethylhexyl) phthalate*	364	692	18	306	20	293	16	0.130	24	1	19	0.030	25
123-91-1	1,4-Dioxane	54	521	19	84	25	49	25	0.080	30	34	5	0.090	11
75-01-4	Vinyl chloride	64	380	20	380	18	316	14	1.900	12	0.3	23	4.600	13
107-06-2	1,2-Dichloroethane	92	326	21	312	19	213	17	2.500	13	2	15	2.900	7
56-23-5	Carbon tetrachloride	61	284	22	281	21	202	18	270.000	1	0.1	28	260.000	2
75-21-8	Ethylene oxide	162	202	23	199	22	196	19	11.000	5	2	16	5.500	4
75-56-9	Propylene oxide	114	185	24	178	23	133	20	0.260	26	9	8	0.420	10
98-95-3	Nitrobenzene	27	145	25	138	24	31	26		missing	0.02	36		missing
140-88-5	Ethyl acrylate	109	117	26	57	27	57	23	0.070	29	0.03	35	0.030	30
—	Polychlorinated alkanes (C10 to C13)	61	112	27	4	39	4	35		missing	0.12	31		missing
106-89-8	Epichlorohydrin	73	85	28	83	26	76	21	1.100	20	6	11	0.450	12
106-46-7	1,4-Dichlorobenzene	27	54	29	53	28	49	24	1.400	21	0.15	27	0.710	21
26471-62-5	Toluenediisocyanate (mixed isomers)	196	41	30	26	31	17	27		missing	0.26	25		missing
101-77-9	4,4'-Methylenedianiline	22	34	31	32	30	6	31	21.000	18	0.05	34	0.430	23
302-01-2	Hydrazine	58	33	32	33	29	1	40	22.000	27	2	14	2.400	8
120-80-9	Catechol	127	27	33	13	32	5	32	0.140	33	8	10	0.003	24
139-13-9	Nitrilotriacetic acid	18	13	34	8	35	2	39		missing	0.0005	42		missing
584-84-9	Toluene-2,4-diisocyanate	54	12	35	2	42	2	38		missing	0	—		missing
62-56-6	Thiourea	24	11	36	11	33	1	41	2.300	32	0.2	26	0.010	29
79-46-9	2-Nitropropane	7	9	37	9	34	9	28	22.000	16	0.1	30	57.000	6
64-67-5	Diethyl sulfate	31	8	38	8	36	8	29	1.600	28	0	—	0.020	—
100-44-7	Benzyl chloride	43	6	39	4	37	4	33	0.880	31	0.1	32	0.070	27
77-78-1	Dimethyl sulfate	30	4	40	4	38	4	34	190.000	11	0	—	0.220	—
96-45-7	Ethylene thiourea	14	4	41	0.1	49	0.1	49	1.200	36	0.002	40	0.100	31
563-47-3	3-Chloro-2-methyl-1-propene	3	4	42	4	40	4	36		missing	0	—		missing
106-88-7	1,2-Butylene oxide	18	3	43	3	41	3	37		missing	0.1	29		missing
121-14-2	2,4-Dinitrotoluene	10	1	44	0.2	46	0.1	48	4.400	35	0.003	38	0.040	32
101-14-4	4,4'-Methylenebis (2-chloroaniline)	21	1	45	0.01	51	0.003	52		missing	0	—		missing
91-08-7	Toluene-2,6-diisocyanate	23	1	46	0.3	45	0.3	45		missing	0	—		missing
67-72-1	Hexachloroethane	21	1	47	0.6	44	0.4	43	260.000	19	0.003	37	230.000	17
95-80-7	2,4-Diaminotoluene	8	1	48	0.6	43	0.6	42	61.000	25	0.002	39	1.500	28
606-20-2	2,6-Dinitrotoluene	4	0.4	49	0.1	50	0.1	50	9.900	34	0.0005	41	0.040	33
94-59-7	Safrole	3	0.2	50	0.1	47	0.1	46	0.310	37	0	—	1.700	—
115-28-6	Chlorendic acid	2	0.2	51	0.002	53	0.002	53		missing	0	—		missing
7758-01-2	Potassium bromate	1	0.1	52	0.1	48	0.1	47		missing	0	—		missing
612-83-9	3,3'-Dichlorobenzidine dihydrochloride	13	0.004	53	0.003	52	0.003	51		missing	0	—		missing
96-09-3	Styrene oxide	1	0.002	54	0.002	54	0.002	54	0.580	38	0	—	0.110	—

Note: Canada and US data only. Mexico data not available for 2002. Data include 54 chemicals common to both NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. A chemical is considered a carcinogen for the purposes of this report if it is so classified by the International Agency for Research on Cancer (IARC: Groups 1, 2A and 2B) **‹http://www.iarc.fr/›** or by the US National Toxicology Program (NTP) **‹ http://ntp-server.niehs.nih.gov /›**. Toxic Equivalency Potentials (TEPs) indicate relative human health risks associated with one unit of chemical, compared to the risk posed by release of a reference chemical (benzene). These TEPs are from **‹www.scorecard.org›**. *** One TRI facility reported incorrect air releases of di(2-ethylhexyl) phthalate for 2002. The correct amount is 213 tonnes less than the amount shown.**

Table 3-4 North American States/Provinces with Largest Releases (On- and Off-site) of Carcinogens Reported to North American PRTRs, 2002 (2002 Matched Chemicals and Industries)

State/Province	Total Releases On- and Off-site (tonnes)	(rank)	Air (tonnes)	(rank)	Releases On-site: Surface Water (tonnes)	Underground Injection (tonnes)	Land (tonnes)	Total On-site Releases* (tonnes)	Total Off-site Releases** (tonnes)
Texas	16,880	1	5,444	1	27	8,422	1,173	15,066	1,815
Ohio	9,001	2	2,089	11	18	1,171	2,266	5,543	3,458
Indiana	8,955	3	3,901	2	8	7	1,273	5,189	3,766
Louisiana	8,741	4	1,923	14	48	4,882	1,373	8,226	515
Ontario	6,712	5	3,753	3	11	0	1,430	5,210	1,502
Alabama	6,200	6	1,781	15	34	2	3,641	5,458	742
Pennsylvania	6,137	7	2,121	10	14	0	1,511	3,646	2,491
California	5,404	8	1,448	16	17	0	2,994	4,459	945
Arizona	5,271	9	243	41	0	45	4,928	5,217	54
Illinois	5,010	10	2,250	9	8	0	1,041	3,299	1,711
Missouri	4,913	11	1,018	24	1	0	2,098	3,117	1,796
Tennessee	4,713	12	3,304	4	33	0	584	3,921	792
Florida	4,662	13	2,949	5	33	63	1,426	4,472	190
Kansas	3,587	14	794	30	0	53	111	958	2,629
Georgia	3,423	15	2,658	6	29	0	574	3,261	161
Quebec	3,415	16	2,464	7	20	0	182	2,670	745
South Carolina	3,217	17	2,397	8	46	0	201	2,644	572
Utah	3,172	18	74	53	1	0	2,749	2,824	348
Michigan	3,134	19	1,945	13	5	28	373	2,352	782
Connecticut	3,081	20	353	38	1	0	0	354	2,727
North Carolina	2,960	21	1,974	12	38	0	544	2,556	404
Kentucky	2,557	22	1,132	18	18	1	952	2,104	454
Alberta	1,928	23	1,043	21	3	99	215	1,362	566
Virginia	1,908	24	1,245	17	7	0	288	1,540	368
Oregon	1,817	25	1,061	19	10	0	474	1,544	273
Arkansas	1,755	26	858	27	22	92	392	1,365	389
Idaho	1,697	27	175	45	20	0	1,478	1,673	24
New York	1,694	28	814	29	36	0	532	1,382	312
West Virginia	1,691	29	396	35	10	0	1,059	1,465	226
Iowa	1,679	30	1,039	22	21	0	122	1,182	497
Nevada***	1,609	31	298	39	0	0	1,296	1,593	16
Minnesota	1,509	32	1,046	20	2	0	132	1,180	328
Wisconsin	1,465	33	1,027	23	7	0	22	1,055	410
Nebraska	1,418	34	226	43	0	0	102	327	1,091
British Columbia	1,353	35	971	25	22	0	6	1,002	351
Washington	1,199	36	930	26	11	0	210	1,152	47
Mississippi	1,155	37	855	28	13	177	41	1,086	69
New Brunswick	988	38	397	34	30	0	19	448	540
New Jersey	915	39	367	37	3	0	19	388	527
Oklahoma	839	40	462	32	4	0	254	720	118
Maryland	636	41	431	33	2	0	45	478	158
Puerto Rico	632	42	611	31	1	0	5	617	15
Manitoba	571	43	394	36	22	0	0	417	154
Saskatchewan	537	44	143	46	3	0	15	161	375
Massachusetts	400	45	132	47	3	0	12	147	253
Montana	374	46	274	40	1	0	83	358	16
North Dakota	348	47	88	48	0	0	132	220	128
Delaware	314	48	175	44	5	0	52	232	82
Maine	271	49	232	42	18	0	3	253	18
Colorado	250	50	86	49	0	0	82	168	82
New Mexico	247	51	45	56	0	0	129	173	74
Nova Scotia	240	52	75	52	1	0	132	208	32
Wyoming	218	53	53	55	0	0	152	205	13
New Hampshire	134	54	78	50	0	0	1	79	55
South Dakota	95	55	76	51	0	0	18	94	1
Rhode Island	74	56	59	54	0	0	0	59	14
Newfoundland and Labrador	59	57	41	58	0	0	9	50	10
Hawaii	44	58	41	57	0	0	0	41	4
Vermont	22	59	5	61	0	0	0	5	17
Virgin Islands	20	60	17	59	0	0	1	18	2
Alaska	14	61	13	60	0	0	0	13	0
Prince Edward Island	7	62	1	63	0	0	0	1	6
Guam	2	63	2	62	0	0	0	2	0
Northern Marianas	0.4	64	0	64	0	0	0	0	0
District of Columbia	0.1	65	0	65	0	0	0	0	0
Total for Carcinogens	**153,274**		**62,297**		**691**	**15,043**	**38,958**	**117,015**	**36,260**
Total for All Matched Chemicals	**3,250,183**		**752,310**		**106,557**	**80,719**	**334,154**	**1,543,284**	**269,421**

Note: Canada and US data only. Mexico data not available for 2002. Data include 55 chemicals common to both NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. A chemical is considered a carcinogen for the purposes of this report if it is so classified by the International Agency for Research on Cancer (IARC) **‹http://www.iarc.fr/›** or the US National Toxicology Program (NTP) **‹http://ntp-server.niehs.nih.gov /›**. Substances classified under IARC as carcinogenic to humans (1), probably carcinogenic to humans (2A), and possibly carcinogenic to humans (2B) are included. Under the US National Toxicology Program, substances classified as known to be carcinogenic (K) or may reasonably be anticipated to be carcinogenic (P) are included. *** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount. ** Includes transfers of metals and metal compounds to energy recovery, treatment, sewage and disposal. *** One TRI facility located in Nevada reported incorrect air releases of di(2-ethylhexyl) phthalate for 2002. The correct amount is 213 tonnes less than the amount shown.**

Table 3-5 Summary of Releases and Transfers of Recognized Developmental and Reproductive Toxicants, 2002 (2002 Matched Chemicals and Industries)

	North America		*Canadian NPRI*		*United States TRI*		*NPRI as % of North American Total*	*TRI as % of North American Total*
	(tonnes)	*(%)*	*(tonnes)*	*(%)*	*(tonnes)*	*(%)*		
Total On-site Releases*	**95,486**	**3**	**10,625**	**3**	**84,861**	**3**	**11**	**89**
■ Air	58,591	2	8,562	2	50,029	2	15	85
■ Surface Water	255	0.01	35	0.01	220	0.01	14	86
■ Underground Injection	2,355	0.1	125	0.04	2,230	0.08	5	95
■ Land	34,271	1	1,888	1	32,382	1	6	94
Total Off-site Releases	**33,187**	**1**	**3,398**	**1**	**29,789**	**1**	**10**	**90**
■ Transfers to Disposal (except metals)	1,807	0.1	410	0.1	1,397	0.05	23	77
■ Transfers of Metals to Disposal, Energy Recovery, Treatment and Sewage	31,380	1	2,988	1	28,392	1	10	90
Total Releases On- and Off-site	**128,673**	**4**	**14,023**	**4**	**114,650**	**4**	**11**	**89**
Transfers to Recycling	**257,738**	**8**	**45,215**	**13**	**212,524**	**7**	**18**	**82**
■ Transfers to Recycling of Metals	226,323	7	41,979	12	184,344	6	19	81
■ Transfers to Recycling (except metals)	31,415	1	3,236	0.9	28,179	1	10	90
Other Transfers Off-site for Further Management	**96,178**	**3**	**5,657**	**2**	**90,521**	**3**	**6**	**94**
■ Transfers to Energy Recovery (except metals)	76,144	2	2,292	1	73,852	3	3	97
■ Transfers to Treatment (except metals)	19,104	1	3,352	0.9	15,752	1	18	82
■ Transfers to Sewage (except metals)	930	0.03	13	0.004	917	0.03	1	99
Total Reported Amounts of Releases and Transfers of Recognized Developmental and Reproductive Toxicants	**482,589**	**15**	**64,894**	**18**	**417,695**	**14**	**13**	**87**
Total Reported Amounts of Releases and Transfers of All Matched Chemicals	**3,250,183**	**100**	**355,883**	**100**	**2,894,300**	**100**	**11**	**89**

Note: Canada and US data only. Mexico data not available for 2002. Data include 21 chemicals common to both NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. A chemical is included as a developmental or reproductive toxicant if it is listed as a recognized developmental or reproductive toxicant on the California Proposition 65 list **‹www.oehha.ca.gov/prop65/prop65_list/files/070904list.html›**. *** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount.**

Table 3-6 Chemicals with Largest Releases and Transfers of Recognized Developmental and Reproductive Toxicants, 2002 (2002 Matched Chemicals and Industries)

					Releases On- and Off-site									Canadian NPRI		United States TRI	
					On-site Releases												
CAS Number	Chemical	Number of Forms	Total Reported Amounts of Releases and Transfers (tonnes)	(rank)	Air (tonnes)	Surface Water (tonnes)	Underground Injection (tonnes)	Land (tonnes)	Total On-site Releases* (tonnes)	Off-site Releases** (tonnes)	Total Releases On- and Off-site (tonnes)	Transfers to Recycling (tonnes)	Other Transfers Off-site for Further Management (tonnes)	Total Reported Amounts of Releases and Transfers* (tonnes)	(rank)	Total Reported Amounts of Releases and Transfers (tonnes)	(rank)
—	Lead (and its compounds)	8,783	211,157	1	961	67	139	23,645	24,812	23,543	48,355	162,802	0	37,048	1	174,109	1
108-88-3	Toluene	3,529	134,758	2	34,833	12	397	54	35,304	804	36,109	18,594	80,056	14,852	2	119,906	2
—	Nickel (and its compounds)	3,809	82,850	3	994	124	241	10,426	11,788	7,746	19,533	63,317	0	10,543	3	72,307	3
75-15-0	Carbon disulfide	131	13,795	4	13,543	4	2	1	13,551	1	13,552	0	243	53	10	13,742	4
872-50-4	N-Methyl -2-pyrrolidone	502	13,448	5	1,330	6	1,112	17	2,464	376	2,840	3,350	7,258	234	6	13,214	5
106-99-0	1,3-Butadiene	224	7,714	7	953	1	17	0	972	1	973	6,129	611	91	8	7,623	6
71-43-2	Benzene	1,079	7,745	6	3,379	10	374	21	3,786	97	3,883	1,759	2,103	1,029	4	6,716	7
117-81-7	Di(2-ethylhexyl) phthalate***	364	6,564	8	293	1	0	12	306	387	692	1,525	4,346	166	7	6,398	8
74-87-3	Chloromethane	96	1,634	9	1,499	1	60	0	1,561	0	1,561	0	73	754	5	881	9
106-89-8	Epichlorohydrin	73	646	10	76	6	0	1	83	2	85	0	561	1	15	645	10
25321-14-6	Dinitrotoluene (mixed isomers)	12	574	11	4	0	0	0	4	1	5	0	569	0	19	574	11
109-86-4	2-Methoxy-ethanol	38	512	12	200	15	0	0	215	9	224	33	255	11	12	500	12
—	Mercury(and its compounds)	1,808	453	13	66	1	9	82	158	91	249	204	0	84	9	369	13
75-21-8	Ethylene oxide	162	293	14	196	2	0	0	199	3	202	21	70	26	11	267	14
74-83-9	Bromomethane	39	236	15	233	0	2	0	235	0	236	0	0	0	18	236	15
554-13-2	Lithium carbonate	49	147	16	6	6	0	11	23	119	141	3	3	0	16	147	16
110-80-5	2-Ethoxyethanol	27	50	17	26	0	0	0	27	0	27	0	23	1	14	48	17
96-45-7	Ethylene thiourea	14	7	18	0	0	0	0	0	4	4	1	3	2	13	5	18
64-75-5	Tetracycline hydrochloride	6	3	19	0	0	0	0	0	2	2	0	1	0	20	3	19
121-14-2	2,4-Dinitro-toluene	10	3	20	0	0	0	0	0	1	1	0	1	0	17	3	20
606-20-2	2,6-Dinitro-toluene	4	1	21	0	0	0	0	0	0	0	0	1	0	21	1	21
Total for Recognized Developmental and Reproductive Toxicants		**20,759**	**482,589**		**58,591**	**255**	**2,355**	**34,271**	**95,486**	**33,187**	**128,673**	**257,738**	**96,178**	**64,894**		**417,695**	
Total for All Matched Chemicals		**84,654**	**3,250,183**		**752,310**	**106,557**	**80,719**	**334,154**	**1,273,863**	**269,421**	**1,543,284**	**1,065,424**	**641,475**	**355,883**		**2,894,300**	

Note: Canada and US data only. Mexico data not available for 2002. Data include chemicals common to both NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. A chemical is included as a developmental or reproductive toxicant if it is listed as a recognized developmental or reproductive toxicant on the California Proposition 65 list ‹www.oehha.ca.gov/prop65/prop65_list/files/070904list.html›. *** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount. ** Includes transfers of metals and metal compounds to energy recovery, treatment, sewage and disposal. *** One TRI facility reported incorrect air releases of di(2-ethylhexyl) phthalate for 2002. The correct amount is 213 tonnes less than the amount shown.**

Table 3-7 Recognized Developmental and Reproductive Toxicants, Ranked by Releases and Toxic Equivalency Potentials (TEPs), 2002 (2002 Matched Chemicals and Industries)

CAS Number	Chemical	Number of Forms	Total Releases On- and Off-site (tonnes)	(rank)	Total Releases On-site (tonnes)	(rank)	On-site Air Releases (tonnes)	(rank)	(TEP)	(TEP rank)	On-site Surface Water Discharges (tonnes)	(rank)	(TEP)	(TEP rank)
—	**Lead (and its compounds)**	8,783	48,355	1	24,812	2	961	7	580,000.0	2*	67	2	42,000.00	2
108-88-3	**Toluene**	3,529	36,109	2	35,304	1	34,833	1	1.0	6	12	4	0.88	10
—	**Nickel (and its compounds)**	3,809	19,533	3	11,788	4	994	6	3,200.0	3	124	1	26.00	3
75-15-0	**Carbon disulfide**	131	13,552	4	13,551	3	13,543	2	1.2	8	4	9	1.80	11
71-43-2	**Benzene**	1,079	3,883	5	3,786	5	3,379	3	8.1	7	10	5	10.00	6
872-50-4	**N-Methyl -2-pyrrolidone**	502	2,840	6	2,464	6	1,330	5		missing	6	7		missing
74-87-3	**Chloromethane**	96	1,561	7	1,561	7	1,499	4	57.0	5	1	12	34.00	9
106-99-0	**1,3-Butadiene**	224	973	8	972	8	953	8	2.2	12	1	11	7.50	12
117-81-7	**Di(2-ethylhexyl) phthalate****	364	692	9	306	9	293	9	33.0	11	1	13	9.00	13
—	**Mercury (and its compounds)**	1,808	249	10	158	13	66	14	14,000,000.0	1*	1	14	13,000,000.0	1
74-83-9	**Bromomethane**	39	236	11	235	10	233	10	1,600.0	4	0.05	16	900.00	8
109-86-4	**2-Methoxyethanol**	38	224	12	215	11	200	11	2.0	13	15	3	15.00	5
75-21-8	**Ethylene oxide**	162	202	13	199	12	196	12	56.0	10	2	10	27.00	7
554-13-2	**Lithium carbonate**	49	141	14	23	16	6	16		missing	6	8		missing
106-89-8	**Epichlorohydrin**	73	85	15	83	14	76	13	210.0	9	6	6	83.00	4
110-80-5	**2-Ethoxyethanol**	27	27	16	27	15	26	15	1.3	15	0.4	15	0.08	15
25321-14-6	**Dinitrotoluene (mixed isomers)**	12	5	17	4	17	4	17		missing	0.03	17		missing
96-45-7	**Ethylene thiourea**	14	4	18	0.06	19	0.05	19	4,600.0	14	0.002	19	400.00	14
64-75-5	**Tetracycline hydrochloride**	6	2	19	0.005	21	0.005	21		missing	0	—		missing
121-14-2	**2,4-Dinitrotoluene**	10	1	20	0.20	18	0.09	18	100.0	17	0.003	18	0.92	16
606-20-2	**2,6-Dinitrotoluene**	4	0.4	21	0.05	20	0.05	20	200.0	16	0.0005	20	0.94	17

Note: Canada and US data only. Mexico data not available for 2002. Data include chemicals common to both NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. A chemical is included as a developmental or reproductive toxicant if it is listed as a recognized developmental or reproductive toxicant on the California Proposition 65 list ‹**www.oehha.ca.gov/prop65/prop65_list/files/070904list.html**›. Toxic Equivalency Potentials (TEPs) indicate relative human health risks associated with one unit of chemical, compared to the risk posed by release of a reference chemical (toluene). These TEPs are from ‹**www.scorecard.org**›. *** TEPs for mercury as shown. TEPs for mercury compounds are 5,000,000 (air) and 5,400,000 (water). Air TEP Rank would change to 2 behind lead and compounds if TEP for mercury compounds is applied. ** One TRI facility reported incorrect air releases of di(2-ethylhexyl) phthalate for 2002. The correct amount is 213 tonnes less than the amount shown.**

Table 3-8 North American States/Provinces with Largest Releases (On- and Off-site) of Recognized Developmental and Reproductive Toxicants, 2002 (2002 Matched Chemicals and Industries)

State/Province	Total Releases On- and Off-site (tonnes)	(rank)	On-site Releases: Air (tonnes)	(rank)	Surface Water (tonnes)	Underground Injection (tonnes)	Land (tonnes)	Total On-site Releases* (tonnes)	Total Off-site Releases** (tonnes)
Tennessee	13,969	1	12,857	1	8	0	504	13,370	599
Ontario	8,614	2	6,000	2	5	0	1,347	7,362	1,253
Texas	7,526	3	3,827	3	15	1,608	1,001	6,451	1,075
Indiana	7,082	4	2,235	5	7	7	1,145	3,394	3,688
Ohio	6,813	5	1,354	14	7	128	2,069	3,559	3,254
Pennsylvania	5,215	6	1,400	13	15	0	1,489	2,904	2,311
Illinois	5,141	7	2,683	4	9	0	1,013	3,705	1,437
Alabama	5,060	8	806	20	7	2	3,514	4,329	731
Arizona	5,048	9	61	48	0	45	4,912	5,018	30
Missouri	4,546	10	786	21	1	0	1,961	2,748	1,798
Kansas	3,748	11	996	19	4	28	101	1,129	2,619
Utah	3,068	12	75	43	1	0	2,676	2,752	316
Louisiana	3,055	13	1,855	9	13	224	562	2,654	401
North Carolina	2,999	14	2,008	7	3	0	445	2,456	543
California	2,971	15	322	31	2	5	1,734	2,064	907
Connecticut	2,961	16	265	33	1	0	0	266	2,695
Kentucky	2,959	17	1,680	10	7	1	859	2,547	412
Virginia	2,692	18	2,195	6	7	0	248	2,450	242
South Carolina	2,591	19	1,883	8	17	0	170	2,069	522
Michigan	2,420	20	1,442	12	2	4	256	1,704	716
Florida	2,200	21	653	24	3	2	1,380	2,037	163
Quebec	1,992	22	1,288	16	4	0	187	1,480	512
Arkansas	1,934	23	1,142	18	2	12	392	1,547	388
New York	1,902	24	1,303	15	33	0	272	1,607	295
West Virginia	1,888	25	717	22	9	0	929	1,655	233
Mississippi	1,748	26	1,473	11	1	163	45	1,682	65
Georgia	1,747	27	1,159	17	8	0	422	1,589	157
Nevada***	1,656	28	403	30	0	0	1,239	1,643	13
Idaho	1,330	29	74	44	0	0	1,231	1,305	24
Nebraska	1,323	30	139	39	0	0	96	235	1,088
Iowa	1,244	31	615	26	16	0	119	750	494
Wisconsin	1,067	32	644	25	3	0	21	669	398
New Jersey	997	33	445	29	2	0	19	466	532
Minnesota	991	34	533	27	2	0	133	667	324
Oregon	899	35	154	36	2	0	476	632	267
Alberta	881	36	149	37	0	125	187	463	418
Manitoba	840	37	667	23	22	0	0	689	151
Oklahoma	804	38	477	28	1	0	210	688	116
New Brunswick	567	39	31	56	1	0	19	53	514
Massachusetts	512	40	257	34	0	0	12	270	242
British Columbia	451	41	268	32	2	0	6	279	172
Saskatchewan	412	42	55	51	0	0	4	59	353
Washington	377	43	155	35	1	0	138	295	82
Maryland	297	44	111	40	2	0	46	158	139
Colorado	248	45	99	42	0	0	70	169	79
New Mexico	246	46	72	45	0	0	107	179	67
North Dakota	229	47	37	54	0	0	83	120	110
Delaware	178	48	56	49	7	0	52	114	64
Wyoming	177	49	30	57	0	0	134	164	13
Nova Scotia	173	50	29	58	0	0	133	161	11
Puerto Rico	160	51	143	38	1	0	5	149	11
Rhode Island	124	52	105	41	0	0	0	105	19
Montana	119	53	33	55	0	0	70	104	15
New Hampshire	106	54	50	52	0	0	1	51	55
South Dakota	82	55	63	47	0	0	18	81	1
Newfoundland and Labrador	79	56	66	46	0	0	4	70	8
Virgin Islands	58	57	55	50	0	0	1	56	2
Hawaii	54	58	50	53	0	0	0	50	4
Maine	35	59	16	60	1	0	3	19	16
Alaska	22	60	21	59	0	0	0	22	0
Vermont	21	61	6	63	0	0	0	7	14
Prince Edward Island	14	62	9	61	0	0	0	9	6
Guam	7	63	7	62	0	0	0	7	0
Northern Marianas	1	64	1	64	0	0	0	1	0
District of Columbia	0.1	65	0	65	0	0	0	0	0.1
Total for Recognized Developmental and Reproductive Toxicants	**128,673**		**58,591**		**255**	**2,355**	**34,271**	**95,486**	**33,187**
Total for All Matched Chemicals	**3,250,183**		**752,310**		**106,557**	**80,719**	**334,154**	**1,543,284**	**269,421**

Note: Canada and US data only. Mexico data not available for 2002. Data include 21 chemicals common to both NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. A chemical is included as a developmental or reproductive toxicant if it is listed as a recognized developmental or reproductive toxicant on the California Proposition 65 list ‹www.oehha.ca.gov/prop65/prop65_list/files/070904list.html›. *** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount. ** Includes transfers of metals and metal compounds to energy recovery, treatment, sewage and disposal. *** One TRI facility located in Nevada reported incorrect air releases of di(2-ethylhexyl) phthalate for 2002. The correct amount is 213 tonnes less than the amount shown.**

Table 3-9 Summary of Releases and Transfers of Suspected Developmental and Reproductive Toxicants, 2002
(2002 Matched Chemicals and Industries)

	North America (tonnes)	North America (%)	Canadian NPRI (tonnes)	Canadian NPRI (%)	United States TRI (tonnes)	United States TRI (%)	NPRI as % of North American Total	TRI as % of North American Total
Total On-site Releases*	**744,423**	**23**	**72,193**	**20**	**672,230**	**23**	**10**	**90**
■ Air	273,927	8	51,040	14	222,887	8	19	81
■ Surface Water	105,841	3	6,252	2	99,588	3	6	94
■ Underground Injection	69,507	2	993	0.3	68,514	2	1	99
■ Land	295,060	9	13,819	4	281,241	10	5	95
Total Off-site Releases	**230,297**	**7**	**25,696**	**7**	**204,602**	**7**	**11**	**89**
■ Transfers to Disposal (except metals)	17,000	0.5	2,411	0.7	14,590	0.5	14	86
■ Transfers of Metals to Disposal, Energy Recovery, Treatment and Sewage	213,297	7	23,285	7	190,012	7	11	89
Total Releases On- and Off-site	**974,720**	**30**	**97,889**	**28**	**876,831**	**30**	**10**	**90**
Transfers to Recycling	**796,049**	**24**	**129,578**	**36**	**666,471**	**23**	**16**	**84**
■ Transfers to Recycling of Metals	695,379	21	119,572	34	575,807	20	17	83
■ Transfers to Recycling (except metals)	100,670	3	10,006	3	90,664	3	10	90
Other Transfers Off-site for Further Management	**481,425**	**15**	**24,621**	**7**	**456,805**	**16**	**5**	**95**
■ Transfers to Energy Recovery (except metals)	243,921	8	5,712	2	238,209	8	2	98
■ Transfers to Treatment (except metals)	90,466	3	10,685	3	79,781	3	12	88
■ Transfers to Sewage (except metals)	147,038	5	8,224	2	138,815	5	6	94
Total Reported Amounts of Releases and Transfers of Suspected Developmental and Reproductive Toxicants	**2,252,195**	**69**	**252,088**	**71**	**2,000,107**	**69**	**11**	**89**
Total Reported Amounts of Releases and Transfers of All Matched Chemicals	**3,250,183**	**100**	**355,883**	**100**	**2,894,300**	**100**	**11**	**89**

Note: Canada and US data only. Mexico data not available for 2002. Data include 74 chemicals common to both NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. A chemical is included as a developmental or reproductive toxicant if it is listed as a suspected developmental or reproductive toxicant on the Scorecard list ‹www.scorecard.org›. *** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount.**

Table 3-10 Chemicals with Largest Releases and Transfers of Suspected Developmental and Reproductive Toxicants, 2002 (2002 Matched Chemicals and Industries)

CAS Number	Chemical	Total Reported Amounts of Releases and Transfers (tonnes)	(rank)	On-site Releases: Air (tonnes)	Surface Water (tonnes)	Underground Injection (tonnes)	Land (tonnes)	Off-site Releases* (tonnes)	Transfers to Recycling (tonnes)	Other Transfers Off-site for Further Management (tonnes)	Canadian NPRI: Total Reported Amounts of Releases and Transfers** (tonnes)	(rank)	United States TRI: Total Reported Amounts of Releases and Transfers (tonnes)	(rank)
—	Copper (and its compounds)	457,393	1	1,147	200	450	100,885	13,242	341,464	0	46,559	2	410,834	1
—	Zinc (and its compounds)	406,251	2	4,053	570	305	67,690	137,290	196,334	0	60,651	1	345,600	2
67-56-1	Methanol	244,855	3	88,618	2,530	6,341	548	897	8,838	137,074	28,965	3	215,889	4
—	Nitric acid and nitrate compounds	244,102	4	908	95,546	24,039	7,315	9,132	1,283	105,877	14,172	7	229,929	3
—	Manganese (and its compounds)	191,725	5	1,437	3,968	4,850	73,894	36,117	71,451	0	28,744	4	162,980	5
—	Xylenes	121,945	6	26,131	15	373	48	848	17,814	76,708	14,882	5	107,063	6
—	Chromium (and its compounds)	92,362	7	549	100	929	12,658	16,148	61,974	0	14,669	6	77,693	7
78-93-3	Methyl ethyl ketone	65,461	8	16,172	44	211	56	331	8,720	39,920	9,523	8	55,939	8
107-21-1	Ethylene glycol	50,695	9	2,134	727	113	332	1,102	28,259	18,024	2,520	13	48,175	9
7664-39-3	Hydrogen fluoride	39,739	10	35,067	17	2,404	859	368	123	903	3,597	10	36,142	10
110-54-3	n-Hexane	35,274	11	23,070	3	28	64	35	2,633	9,435	3,392	11	31,881	11
100-42-5	Styrene	33,067	12	23,511	2	73	91	852	1,538	6,996	2,535	12	30,532	12
75-09-2	Dichloromethane	27,913	13	6,030	2	138	2	84	7,571	14,085	1,431	18	26,483	13
—	Vanadium (and its compounds)	26,408	14	894	270	493	17,062	3,371	4,317	0	2,244	14	24,164	14
7429-90-5	Aluminum (fume or dust)	25,982	15	657	6	0	7,782	4,585	12,951	0	5,934	9	20,048	15
75-05-8	Acetonitrile	18,841	16	339	5	8,043	0	4	607	9,844	7	49	18,834	16
108-10-1	Methyl isobutyl ketone	18,015	17	5,019	8	54	19	50	4,624	8,238	1,805	16	16,210	17
100-41-4	Ethylbenzene	13,723	18	3,679	5	431	5	72	2,384	7,140	1,675	17	12,047	19
50-00-0	Formaldehyde	13,571	19	6,403	195	3,584	57	285	53	2,991	2,158	15	11,413	20
91-20-3	Naphthalene	13,027	20	1,285	13	76	295	158	9,232	1,967	354	24	12,673	18
108-95-2	Phenol	11,877	21	3,312	46	679	92	767	721	6,258	1,408	19	10,469	21
75-07-0	Acetaldehyde	8,792	22	6,716	189	326	7	2	2	1,551	942	20	7,850	23
68-12-2	N,N-Dimethylformamide	8,099	23	268	12	25	9	170	411	7,204	201	28	7,898	22
79-01-6	Trichloroethylene	6,975	24	4,317	0	64	0	77	1,070	1,444	902	21	6,072	26
—	Antimony (and its compounds)	6,439	25	52	23	2	1,712	1,751	2,898	0	282	25	6,157	24
Subtotal for Top 25		**2,182,527**		**261,766**	**104,496**	**54,032**	**291,482**	**227,737**	**787,272**	**455,659**	**249,552**		**1,932,975**	
All Others		**69,667**		**12,161**	**1,345**	**15,474**	**3,578**	**2,560**	**8,777**	**25,766**	**2,535**		**67,132**	
Total for Suspected Developmental and Reproductive Toxicants		**2,252,195**		**273,927**	**105,841**	**69,507**	**295,060**	**230,297**	**796,049**	**481,425**	**252,088**		**2,000,107**	
Total for All Matched Chemicals		**3,250,183**		**752,310**	**106,557**	**80,719**	**334,154**	**269,421**	**1,065,424**	**641,475**	**355,883**		**2,894,300**	

Note: Canada and US data only. Mexico data not available for 2002. Data include 74 chemicals common to both NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. A chemical is included as a developmental or reproductive toxicant if it is is listed as a suspected developmental or reproductive toxicant on the Scorecard list <www.scorecard.org>. *** Includes transfers of metals and metal compounds to energy recovery, treatment, sewage and disposal. ** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount.**

Table 3-11 Summary of Releases and Transfers of Suspected Neurotoxicants, 2002
(2002 Matched Chemicals and Industries)

	North America (tonnes)	(%)	*Canadian NPRI* (tonnes)	(%)	*United States TRI* (tonnes)	(%)	*NPRI as % of North American Total*	*TRI as % of North American Total*
Total On-site Releases*	**753,799**	**23**	**82,692**	**23**	**671,108**	**23**	**11**	**89**
■ Air	378,340	12	64,510	18	313,829	11	17	83
■ Surface Water	10,883	0.33	1,924	0.54	8,959	0.31	18	82
■ Underground Injection	53,111	1.6	845	0.24	52,266	1.81	2	98
■ Land	311,351	10	15,298	4	296,053	10	5	95
Total Off-site Releases	**241,377**	**7**	**25,808**	**7**	**215,569**	**7**	**11**	**89**
■ Transfers to Disposal (except metals)	12,820	0.4	1,541	0.4	11,279	0.39	12	88
■ Transfers of Metals to Disposal, Energy Recovery, Treatment and Sewage	228,557	7	24,267	7	204,290	7	11	89
Total Releases On- and Off-site	**995,176**	**31**	**108,500**	**30**	**886,676**	**31**	**11**	**89**
Transfers to Recycling	**1,000,379**	**31**	**165,300**	**46**	**835,078**	**29**	**17**	**83**
■ Transfers to Recycling of Metals	860,830	26	150,067	42	710,764	25	17	83
■ Transfers to Recycling (except metals)	139,548	4	15,234	4.3	124,315	4	11	89
Other Transfers Off-site for Further Management	**513,753**	**16**	**23,843**	**7**	**489,910**	**17**	**5**	**95**
■ Transfers to Energy Recovery (except metals)	345,036	11	8,310	2	336,726	12	2	98
■ Transfers to Treatment (except metals)	112,744	3	12,980	3.6	99,763	3	12	88
■ Transfers to Sewage (except metals)	55,973	1.72	2,553	0.717	53,420	1.85	5	95
Total Reported Amounts of Releases and Transfers of Suspected Neurotoxins	**2,509,307**	**77**	**297,643**	**84**	**2,211,664**	**76**	**12**	**88**
Total Reported Amounts of Releases and Transfers of All Matched Chemicals	**3,250,183**	**100**	**355,883**	**100**	**2,894,300**	**100**	**11**	**89**

Note: Canada and US data only. Mexico data not available for 2002. Data include 146 chemicals common to both NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. A chemical is included as a neurotoxicant if it is listed as a suspected neurotoxicant on the Scorecard list ‹www.scorecard.org›. *** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount.**

Table 3-12 Summary of Total Reported Releases and Transfers of Lead and its Compounds, 2002 (2002 Matched Chemicals and Industries)

	North America (tonnes)	*Canadian NPRI (tonnes)*	*United States TRI (tonnes)*
On-site Releases	**24,812**	**2,016**	**22,796**
■ Air	961	402	559
■ Surface Water	67	8	60
■ Underground Injection	139	0	139
■ Land	23,645	1,607	22,038
Total Off-site Releases*	**23,543**	**2,123**	**21,420**
Total Releases On- and Off-site	**48,355**	**4,139**	**44,216**
Transfers to Recycling	**162,802**	**32,909**	**129,893**
Other Transfers Off-site for Further Management	**0**	**0**	**0**
Total Reported Amounts of Releases and Transfers of Lead and its Compounds	**211,157**	**37,048**	**174,109**

Note: Canada and US data only. Mexico data not available for 2002. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. *** Includes transfers of metals and metal compounds to energy recovery, treatment, sewage and disposal.**

Table 3-13 North American Industries with Largest Total Reported Amounts of Releases and Transfers of Lead and its Compounds, 2002 (2002 Matched Chemicals and Industries)

				On-site Releases							
US SIC Code	*Industry*	*Total Reported Amounts of Releases and Transfers (tonnes)*	*(rank)*	*Air (tonnes)*	*Surface Water (tonnes)*	*Underground Injection (tonnes)*	*Land (tonnes)*	*Total On-site Releases (tonnes)*	*Total Off-site Releases* (tonnes)*	*Total On- and Off-site Releases (tonnes)*	*Transfers Off-site to Recycling (tonnes)*
36	Electronic/Electrical Equipment	89,782	1	18	1	0	35	54	666	720	89,063
33	Primary Metals	52,693	2	632	13	1	9,240	9,886	10,643	20, 529	32,164
39	Misc. Manufacturing Industries	24,897	3	1	0	0	25	26	106	132	24,766
495/738	Hazardous Waste Mgt./ Solvent Recovery	15,246	4	6	0	5	8,989	9,000	5,603	14,603	642
—	Multiple SIC Codes	7,374	5	26	10	0	146	182	3,075	3, 257	4,117
34	Fabricated Metals Products	5,216	6	20	1	0	19	39	312	351	4,865
28	Chemicals	4,829	7	15	7	130	750	901	1,110	2, 011	2,817
491/493	Electric Utilities	4,336	8	122	17	0	3,210	3,349	733	4, 082	253
37	Transportation Equipment	1,923	9	13	1	0	5	19	154	173	1,750
35	Industrial Machinery	1,466	10	10	0	0	0	11	24	35	1,431
32	Stone/Clay/Glass Products	1,355	11	42	0	1	333	377	680	1,057	298
12	Coal Mining	703	12	0	0	2	698	701	2	703	0
30	Rubber and Plastics Products	417	13	12	0	0	28	40	154	194	223
26	Paper Products	251	14	17	14	0	122	153	87	240	10
38	Measurement/Photographic Instruments	249	15	1	0	0	0	1	29	30	219
29	Petroleum and Coal Products	183	16	6	2	0	5	12	102	114	69
27	Printing and Publishing	75	17	0	0	0	0	0	4	4	71
24	Lumber and Wood Products	60	18	9	0	0	20	29	13	42	18
20	Food Products	33	19	7	0	0	2	10	16	26	7
25	Furniture and Fixtures	23	20	2	0	0	0	2	9	11	13
5171	Petroleum Bulk Terminals	20	21	0	0	0	16	16	4	20	0
22	Textile Mill Products	18	22	2	0	0	1	3	11	14	4
5169	Chemical Wholesalers	4	23	0	0	0	0	0	2	2	2
21	Tobacco Products	3	24	0	0	0	0	0	3	3	0
31	Leather Products	0	25	0	0	0	0	0	0	0	0
23	Apparel and Other Textile Products	0	26	0	0	0	0	0	0	0	0
Total for Lead and its Compounds		**211,157**		**961**	**67**	**139**	**23,645**	**24,812**	**23,543**	**48,355**	**162,802**

Note: Canada and US data only. Mexico data not available for 2002. *** Includes transfers of metals and metal compounds to energy recovery, treatment, sewage and disposal.**

Table 3-14 Summary of Total Reported Releases and Transfers of Mercury and its Compounds, 2002 (2002 Matched Chemicals and Industries)

	North America (kg)	*Canadian NPRI (kg)*	*United States TRI (kg)*
On-site Releases	**157,693**	**6,778**	**150,915**
■ Air	65,901	4,966	60,935
■ Surface Water	608	59	549
■ Underground Injection	9,163	0	9,163
■ Land	82,020	1,752	80,268
Total Off-site Releases*	**91,361**	**13,422**	**77,938**
Total Releases On- and Off-site	**249,053**	**20,200**	**228,853**
Transfers to Recycling	**204,217**	**63,579**	**140,639**
Other Transfers Off-site for Further Management	**0**	**0**	**0**
Total Reported Amounts of Releases and Transfers of Mercury and its Compunds	**453,271**	**83,779**	**369,492**

Note: Canada and US data only. Mexico data not available for 2002. Data are NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. *** Includes transfers of metals and metal compounds to energy recovery, treatment, sewage and disposal.**

Table 3-15 North American Industries with Largest Total Reported Amounts of Releases and Transfers of Mercury and its Compounds, 2002 (2002 Matched Chemicals and Industries)

US SIC Code	*Industry*	*Total Reported Amounts of Releases and Transfers (kg)*	*(rank)*	*On-site Releases: Air (kg)*	*Surface Water (kg)*	*Underground Injection (kg)*	*Land (kg)*	*Total On-site Releases (kg)*	*Total Off-site Releases* (kg)*	*Total On- and Off-site Releases (kg)*	*Transfers Off-site to Recycling (kg)*
495/738	Hazardous Waste Mgt./ Solvent Recovery	182,237	1	875	1	8,388	21,570	30,834	42,322	73,156	109,080
33	Primary Metals	87,870	2	6,208	78	0	26,206	32,492	6,633	39,125	48,745
491/493	Electric Utilities	72,911	3	42,986	229	0	18,493	61,708	7,553	69,261	3,650
28	Chemicals	57,689	4	6,722	165	681	9,595	17,164	24,846	42,010	15,680
38	Measurement/Photographic Instruments	14,852	5	48	2	0	0	49	100	149	14,702
36	Electronic/Electrical Equipment	11,102	6	211	0	0	0	212	2,438	2,650	8,452
32	Stone/Clay/Glass Products	8,107	7	6,021	2	8	1,439	7,469	100	7,569	538
34	Fabricated Metals Products	5,379	8	22	0	0	0	22	5,248	5,270	108
12	Coal Mining	4,129	9	4	1	39	4,078	4,122	6	4,128	0.05
26	Paper Products	2,481	10	1,176	59	0	474	1,709	394	2,103	378
29	Petroleum and Coal Products	2,424	11	711	33	45	79	868	953	1,821	603
—	Multiple Codes 20–39*	1,787	12	703	36	0	78	817	506	1,323	464
37	Transportation Equipment	887	13	14	1	0	0	15	26	41	847
35	Industrial Machinery	573	14	0	0	0	0	0	0	0	573
39	Misc. Manufacturing Industries	313	15	7	0	0	0	7	53	60	253
20	Food Products	274	16	121	0	0	9	130	91	221	52
30	Rubber and Plastics Products	112	17	6	0	0	0	6	26	32	80
21	Tobacco Products	85	18	43	1	0	0	44	41	85	0
5169	Chemical Wholesalers	21	19	0	0	0	0	0	20	20	1
5171	Petroleum Bulk Terminals	18	20	11	2	0	0	12	4	16	2
24	Lumber and Wood Products	12	21	2	0	0	0	2	0	2	10
22	Textile Mill Products	9	22	9	0	0	0	9	0	9	0
25	Furniture and Fixtures	0	23	0	0	0	0	0	0	0	0
Total for Mercury and its Compounds		**453,271**		**65,901**	**608**	**9,163**	**82,020**	**157,693**	**91,361**	**249,053**	**204,217**

Note: Canada and US data only. Mexico data not available for 2002. *** Includes transfers of metals and metal compounds to energy recovery, treatment, sewage and disposal.**

Table 3-16 Canadian NPRI Releases (On- and Off-site) of Dioxins/Furans, by Industry, 2002 (2002 Matched Chemicals and Industries)

US SIC Code	Industry	Canadian NPRI: Air (grams-iTEQ*)	Water (grams-iTEQ*)	Underground Injection (grams-iTEQ*)	Land (grams-iTEQ*)	Total On-site Releases (grams-iTEQ*)	Off-site Releases (grams-iTEQ*)	Total Releases On- and Off-site (grams-iTEQ*)	(rank)
26	Paper Products	3.81	0.22	0.00	35.52	39.55	32.70	72.26	1
33	Primary Metals	19.81	0.02	0.00	1.38	21.21	40.14	61.35	2
95	Air, Water & Solid Waste Management***	46.77	0.00	0.00	0.00	46.77	0.00	46.77	3
49	Sewage Systems***	0.12	0.00	0.00	0.00	0.12	28.93	29.05	4
491/493	Electric Utilities	15.95	0.00	0.00	0.20	16.15	0.00	16.16	5
28	Chemicals**	1.04	0.01	4.79	1.69	7.53	0.06	7.58	6
39	Misc. Manufacturing Industries	0.17	0.00	0.00	0.00	0.17	1.33	1.50	7
24	Lumber and Wood Products	1.08	0.04	0.00	0.15	1.27	0.07	1.34	8
32	Stone/Clay/Glass Products	1.08	0.00	0.00	0.00	1.08	0.00	1.08	9
495/738	Hazardous Waste Mgt./Solvent Recovery	0.32	0.00	0.00	0.00	0.32	0.74	1.06	10
25	Furniture and Fixtures	0.30	0.00	0.00	0.00	0.30	0.00	0.30	11
36	Electronic/Electrical Equipment	0.19	0.00	0.00	0.00	0.19	0.00	0.19	12
80	Health and Allied Services***	0.17	0.00	0.00	0.00	0.17	0.00	0.17	13
34	Fabricated Metals Products	0.04	0.00	0.00	0.00	0.04	0.00	0.04	14
1094	Uranium Mines***	0.01	0.00	0.00	0.00	0.01	0.00	0.01	15
37	Transportation Equipment	0.01	0.00	0.00	0.00	0.01	0.00	0.01	16
Total for Dioxins/Furans		**90.87**	**0.29**	**4.79**	**38.94**	**134.89**	**103.97**	**238.87**	

Note: Only certain activities within these industries must be reported under NPRI. *** Grams-iTEQ, as reported, are based on toxic equivalency factors developed by international convention adopted in 1989. ** Only manufacturers of chlorinated organic solvents or chlorinated monomers are required to report dioxins/furans to NPRI. *** Industry not required to report to TRI.**

Table 3-17 Canadian NPRI Releases (On- and Off-site) of Dioxins/Furans, by Industry, 2000–2002 (ordered by total grams-iTEQ, 2002) (2000–2002 All Industries)

US SIC Code	Industry	Canadian NPRI: 2000 Number of Facilities	2000 Total Reported Releases On- and Off-site (grams-iTEQ*)	2001 Number of Facilities	2001 Total Reported Releases On- and Off-site (grams-iTEQ*)	2002 Number of Facilities	2002 Total Reported Releases On- and Off-site (grams-iTEQ*)	2002 (% of total)	Change 2000–2001 Number of Facilities	Change 2000–2001 Total Reported Releases On- and Off-site (grams-iTEQ*)	Change 2000–2001 (%)
26	Paper Products	52	120.65	55	140.49	52	72.26	30	0	-48.39	-40
33	Primary Metals	52	119.06	55	56.32	53	61.35	26	1	-57.71	-48
95	Air, Water & Solid Waste Management***	41	53.10	38	47.47	38	46.77	20	-3	-6.33	-12
49	Sewage Systems***	10	8.64	12	34.69	5	29.05	12	-5	20.41	236
491/493	Electric Utilities	31	4.46	31	4.83	34	16.16	7	3	11.70	262
28	Chemicals**	9	35.67	10	66.91	10	7.58	3	1	-28.09	-79
39	Misc. Manufacturing Industries	2	0.00	3	0.00	5	1.50	0.6	3	1.50	—
24	Lumber and Wood Products	66	4.62	89	4.99	96	1.34	0.6	30	-3.28	-71
32	Stone/Clay/Glass Products	14	1.85	15	0.91	17	1.08	0.5	3	-0.77	-42
495/738	Hazardous Waste Mgt./ Solvent Recovery	4	1.26	6	1.23	8	1.06	0.4	4	-0.20	-16
25	Furniture and Fixtures	0	0.00	2	0.00	4	0.30	0.1	4	0.30	—
36	Electronic/Electrical Equipment	1	0.00	1	0.00	1	0.19	0.1	0	0.19	—
80	Health and Allied Services***	2	0.00	3	0.33	6	0.17	0.1	4	0.17	—
34	Fabricated Metals Products	3	0.05	3	0.04	3	0.04	0.02	0	-0.01	-20
1094	Uranium Mines***	1	0.00	1	0.00	3	0.01	0.004	2	0.01	—
37	Transportation Equipment	2	0.00	0	0.00	1	0.01	0.004	-1	0.01	—
02	Agricultural Production***	0	0.00	0	0.00	1	0.00	0	1	0.00	—
07	Agricultural Services***	0	0.00	0	0.00	1	0.00	0	1	0.00	—
09	Fishing, Hunting, Trapping***	1	0.00	1	0.00	1	0.00	0	0	0.00	—
10	Metal Mining	2	0.00	2	0.00	2	0.00	0	0	0.00	—
13	Oil and Gas Extraction***	2	0.00	2	0.00	1	0.00	0	-1	0.00	—
14	Nonmetallic Minerals Mining***	0	0.00	1	0.00	1	0.00	0	1	0.00	—
16	Heavy Construction, except building***	1	0.00	1	0.00	0	0.00	0	-1	0.00	—
20	Food Products	1	0.00	1	0.00	0	0.00	0	-1	0.00	—
35	Industrial Machinery	1	0.00	0	0.00	0	0.00	0	-1	0.00	—
47	Transportation Services***	1	0.00	1	0.00	0	0.00	0	-1	0.00	—
82	Educational Services***	0	0.00	0	0.00	1	0.00	0	1	0.00	—
89	Other Scientific & Technical Services***	1	0.01	1	0.00	1	0.00	0	0	-0.01	-100
97	National Security and International Affairs***	0	0.00	0	0.00	1	0.00	0	1	0.00	—
Total for Dioxins/Furans		**300**	**349.37**	**334**	**358.21**	**346**	**238.87**	**100**	**46**	**-110.50**	**-32**

Note: Only certain activities within these industries must be reported under NPRI. *** Grams-iTEQ, as reported, are based on toxic equivalency factors developed by international convention adopted in 1989. ** Only manufacturers of chlorinated organic solvents or chlorinated monomers are required to report dioxins/furans to NPRI. *** Industry not required to report to TRI.**

Table 3-18 United States TRI Releases (On- and Off-site) of Dioxins/Furans, by Industry, 2002 (2002 All Industries)

		United States TRI									
US SIC Code	Industry	Air (grams)	Water (grams)	Underground Injection (grams)	Land (grams)	Total On-site Releases (grams)	Total Off-site Releases (grams)	Total Releases On- and Off-site (grams)	(rank)	Total Releases On- and Off-site (grams iTEQ*)	(rank)
28	Chemicals	976.13	692.62	489.65	43,135.77	45,294.17	79,889.36	125,183.53	1	605.96	1
33	Primary Metals	386.83	0.03	0.00	2,968.08	3,354.94	1,380.47	4,735.41	3	200.30	2
26	Paper Products	102.06	58.03	0.00	307.09	467.18	873.09	1,340.27	5	41.36	3
491/493	Electric Utilities	1,027.10	0.02	0.00	804.45	1,831.57	7.85	1,839.42	4	26.98	4
25	Furniture and Fixtures	157.15	0.00	0.00	0.00	157.15	0.00	157.15	10	15.70	5
7389/4953	Hazardous Waste Mgt./Solvent Recovery	24.71	0.01	421.00	211.38	657.09	35.77	692.86	6	12.98	6
32	Stone/Clay/Glass Products	244.49	0.04	0.28	21.29	266.11	0.00	266.11	7	10.05	7
24	Lumber and Wood Products	293.91	344.73	0.00	135.79	774.42	4,735.45	5,509.88	2	8.30	8
—	Multiple Codes 20–39	31.94	6.64	0.00	6.45	45.03	204.84	249.87	8	3.30	9
29	Petroleum and Coal Products	23.69	4.79	0.00	6.20	34.68	17.49	52.17	11	1.55	10
10	Metal Mining	11.18	0.05	0.00	1.94	13.17	0.00	13.17	13	0.95	11
20	Food Products	8.71	0.00	0.00	0.10	8.81	0.36	9.17	14	0.40	12
38	Measurement/Photographic Instruments	3.03	3.00	0.00	0.00	6.03	1.01	7.04	15	0.37	13
37	Transportation Equipment	0.67	0.00	0.00	0.00	0.67	0.00	0.67	19	0.05	14
—	No Codes	2.34	0.00	0.00	0.00	2.34	0.00	2.34	17	0.05	15
5169	Chemical Wholesalers	5.56	0.00	0.00	0.00	5.56	0.00	5.56	16	0.01	16
22	Textile Mill Products	0.69	0.00	0.00	0.00	0.69	0.00	0.69	18	0.01	17
30	Rubber and Plastics Products	210.09	0.00	0.00	0.14	210.23	0.00	210.23	9	0.00	18
12	Coal Mining	0.00	0.00	0.00	16.26	16.26	0.00	16.26	12	0.00	19
5171	Petroleum Bulk Terminals	0.43	0.00	0.00	0.00	0.43	0.00	0.43	20	0.00	20
21	Tobacco Products	0.28	0.00	0.00	0.00	0.28	0.00	0.28	21	0.00	21
Total for Dioxins/Furans		**3,510.99**	**1,109.95**	**910.93**	**47,614.94**	**53,146.81**	**87,145.69**	**140,292.50**		**928.33**	

*** Grams-iTEQ calculated from reported weight, congener distribution, and toxic equivalency factors developed by international convention adopted in 1989.**

Table 3-19 Total Releases (On-site and Off-site) of Dioxins/Furans, TRI, 2000–2002 (ordered by grams-iTEQ, 2002) (2000–2002 All Chemicals and Industries)

		United States TRI									
		2000 Forms with Dioxin/Furan Distribution		2001 Forms with Dioxin/Furan Distribution		2002 Forms with Dioxin/Furan Distribution			Change 2000–2002 Forms with Dioxin/Furan Distribution		
US SIC Code	Industry	Number of Facilities	Total Reported Releases On- and Off-site (grams-iTEQ*)	Number of Facilities	Total Reported Releases On- and Off-site (grams-iTEQ*)	Number of Facilities	Total Reported Releases On- and Off-site (grams-iTEQ*)	(% of total)	Number of Facilities	Total Reported Releases On- and Off-site (grams-iTEQ*)	(%)
28	Chemicals	99	689.34	100	738.35	97	605.96	65.3	-2	-83.37	-12.1
33	Primary Metals	85	212.18	80	201.02	78	200.30	21.6	-7	-11.88	-5.6
26	Paper Products	141	15.00	145	28.17	142	41.36	4.5	1	26.36	175.7
491/493	Electric Utilities	318	91.94	364	105.87	350	26.98	2.9	32	-64.96	-70.7
25	Furniture and Fixtures	0	0	6	11.53	3	15.70	1.7	3	15.70	—
495/738	Hazardous Waste Mgt./ Solvent Recovery	10	12.03	9	10.78	10	12.98	1.4	0	0.95	7.9
32	Stone/Clay/Glass Products	57	17.53	50	11.24	56	10.05	1.1	-1	-7.48	-42.7
24	Lumber and Wood Products	68	1.97	81	6.93	82	8.30	0.9	14	6.33	320.6
—	Multiple Codes 20–39**	31	13.35	30	4.56	31	3.30	0.4	0	-10.05	-75.3
29	Petroleum and Coal Products	23	2.93	24	1.03	24	1.55	0.2	1	-1.38	-47.1
10	Metal Mining	11	0.91	10	0.95	9	0.95	0.1	-2	0.04	4.6
20	Food Products	16	0.42	16	0.34	17	0.40	0.04	1	-0.02	-3.9
38	Measurement/Photographic Instruments	1	0.18	1	0.42	1	0.37	0.04	0	0.19	102.8
37	Transportation Equipment	3	0.12	2	0.04	3	0.05	0.01	0	-0.07	-60.3
—	No Codes	2	0.05	1	0.03	1	0.05	0.01	-1	-0.01	-13.6
5169	Chemical Wholesalers	1	0.01	1	0.02	1	0.01	0.001	0	0.00	24.4
22	Textile Mill Products	0	0.00	0	0.00	1	0.01	0.001	1	0.01	—
34	Fabricated Metals Products	1	0.03	0	0.00	0	0.00	0.0	-1	-0.03	-100.0
5171	Petroleum Bulk Terminals	1	2.69	0	0.00	0	0.00	0.0	-1	-2.69	-100.0
Total for Dioxins/Furans		**868**	**1,060.69**	**920**	**1,121.29**	**906**	**928.33**	**100.0**	**38**	**-132.36**	**-12.5**

*** Grams-iTEQ calculated from reported weight, congener distribution, and toxic equivalency factors developed by international convention adopted in 1989. ** Multiple SIC codes reported only in TRI.**

Table 3-20 Phthalate Esters in Use in North America: Known Uses and Toxicities

Phthalate Compound	*Major Uses*	*Cancer Listings*	*Developmental and Reproductive Toxicity Listings*
Di(2-ethylhexyl) phthalate (DEHP)	Building products (flooring, roof coverings, wallpaper, coatings, wire insulation), car products (upholstery, car seats, underbody coating, trim), clothing (footwear, raincoats), food packaging, children's products (toys, crib bumpers), and medical devices and tubing. In the United States, not in nipples, teethers, pacifiers, rattles, but used in toys for older children	• NTP: "reasonably anticipated to be a human carginogen" • IARC: "not classifiable" • Prop 65: carcinogen	• NTP CERHR: developmental and reproductive (animals) • Prop 65: developmental
Di-n-butyl phthalate (DBP) and di-isobutyl phthalate (DIBP)	Latex adhesives, cosmetics and other personal care products, cellulose plastics (used in food packaging), and in dyes	None	• DBP: Prop 65 - male and female reproductive and developmental (proposed March 2005) • DBP; NTP CHRHR: developmental and reproductive (animals) • DIPB: Not reviewed
Butyl benzyl phthalate (BBP)	Vinyl tiles, food conveyor belts, artificial leather, automotive trim, and traffic cones	• IARC: "not classifiable"	• Prop 65: developmental (proposed March 2005) • NTP CERHR: developmental (animals)
Di-isodecyl phthalate (DIDP)	Coverings on wires and cables, artificial leather, toys, carpet backing, and pool liners	None	• Prop 65: developmental (proposed March 2005) • NTP CERHR: developmental (animals)
Di-isononyl phthalate (DINP)	Garden hoses, pool liners, flooring tiles, tarps, and toys, including baby toys and teething rings	None	• NTP CERHR: developmental (animals)
Di-n-hexyl phthalate (DnHP)	Tool handles, dishwasher baskets, flooring, gloves, flea collars and food processing conveyer belts	None	• Prop 65: male and female reproductive (proposed March 2005) • NTP CHRHR: reproductive (animals)
Di-n-octyl phthalate (DnOP)	Flooring, carpet tiles, tarps, pool liners, and garden hoses. FDA-approved as a food additive and in food containers and conveyor belts	None	• NTP CERHR: inadequate evidence

Sources: Kavlock *et al.* 2002a–g. Abbreviations: **IARC** = International Agency for Research on Cancer; **Prop 65** = California Proposition 65; **NTP** = National Toxicology Program; **NTP CERHR** = National Toxicology Program Center for the Evaluation of Risks to Human Reproduction.

Table 3-21 Summary of Total Reported Releases and Transfers of Phthalates, 2002 (2002 Matched Chemicals and Industries)

	North America				
	All phthalates (number)	*Dibutyl phthalate (number)*	*Di(2-ethylhexyl) phthalate (number)*	*Canadian NPRI (number)*	*United States TRI (number)*
Facilities	**495**			**64**	**431**
Forms	**543**	**179**	**364**	**68**	**475**
	(kg)	*(kg)*	*(kg)*	*(kg)*	*(kg)*
On-site Releases*	**208,911**	**117,188**	**91,723**	**14,536**	**194,375**
■ Air	138,627	59,829	78,797	13,900	124,727
■ Surface Water	692	78	614	0	692
■ Underground Injection	56,689	56,689	0	0	56,689
■ Land	12,267	127	12,140	0	12,267
Total Off-site Releases	**401,416**	**14,661**	**386,755**	**47,013**	**354,403**
Total Reported Releases On- and Off-site	**610,328**	**131,849**	**478,479**	**61,549**	**548,779**
Off-site Transfers to Recycling	**1,529,578**	**4,394**	**1,525,185**	**105,090**	**1,424,488**
Other Transfers Off-site for Further Management	**4,456,939**	**110,557**	**4,346,382**	**9,022**	**4,447,917**
■ Transfers to Energy Recovery	4,374,993	76,806	4,298,187	195	4,374,798
■ Transfers to Treatment	78,870	32,672	46,199	8,825	70,045
■ Transfers to Sewage	3,075	1,079	1,996	2	3,073
Total Reported Amounts of Releases and Transfers	**6,596,845**	**246,799**	**6,350,045**	**175,661**	**6,421,184**

Note: Canada and US data only. Mexico data not available for 2002. Data are NPRI and TRI lists from selected industrial and other sources. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. *** One TRI facility reported incorrect air releases of di(2-ethylhexyl) phthalate for 2002. The correct amount is shown in the tables in this section of the report but was received too late to be included in other tables of the report.**

Table 3-22 Releases and Transfers of Phthalates, by Industry, 2002 (2002 Matched Chemicals and Industries)

		Releases On- and Off-site											
				On-site Releases									
US SIC Code	Industry	Total Releases On- and Off-site (kg)	(rank)	Air (kg)	Surface Water (kg)	Underground Injection (kg)	Land (kg)	Total On-site Releases* (kg)	Total Off-site Releases** (kg)	Transfers Off-site to Recycling (kg)	Other Transfers for Further Management (kg)	Total Reported Amounts of Releases and Transfers (kg)	(rank)
30	Rubber and Plastics Products***	397,997	1	88,032	187	0	2,364	90,582	307,415	680,615	94,498	1,173,111	2
28	Chemicals	97,186	2	16,514	438	56,689	141	73,908	23,278	361,214	69,314	527,713	3
37	Transportation Equipment	40,716	3	17,067	0	0	340	17,570	23,146	0	842	41,558	7
—	Multiple SIC Codes	28,389	4	3,331	13	0	0	3,343	25,045	142,551	13,011	183,951	4
33	Primary Metals	10,083	5	7	0	0	0	7	10,076	156,060	0	166,143	6
29	Petroleum and Coal Products	7,891	6	944	0	0	2,376	3,320	4,571	374	0	8,265	10
495/738	Hazardous Waste Mgt./Solvent Recovery	7,644	7	355	5	0	7,046	7,405	238	0	4,249,173	4,256,816	1
39	Misc. Manufacturing Industries	5,717	8	5,717	0	0	0	5,717	0	0	0	5,717	13
27	Printing and Publishing	4,618	9	1,328	0	0	0	1,328	3,290	1,674	928	7,220	11
26	Paper Products	2,906	10	2,906	0	0	0	2,906	0	0	1,380	4,286	14
22	Textile Mill Products	2,470	11	187	0	0	0	187	2,283	395	12,019	14,885	8
34	Fabricated Metals Products	2,458	12	877	0	0	0	1,217	1,240	7,496	2,511	12,465	9
5169	Chemical Wholesalers	791	13	509	0	0	0	509	281	0	6,297	7,088	12
36	Electronic/Electrical Equipment	719	14	471	2	0	0	473	245	1,474	1,418	3,610	15
38	Measurement/Photographic Instruments	408	15	120	48	0	0	168	239	177,725	2,830	180,963	5
32	Stone/Clay/Glass Products	326	16	258	0	0	0	258	68	0	2,716	3,042	16
35	Industrial Machinery	11	17	2	0	0	0	11	0	0	0	11	17
12	Coal Mining	0	18	0	0	0	0	0	0	0	0	0	18
24	Lumber and Wood Products	0	19	0	0	0	0	0	0	0	0	0	19
25	Furniture and Fixtures	0	20	0	0	0	0	0	0	0	0	0	20
Total for Phthalates		**610,328**		**138,627**	**692**	**56,689**	**12,267**	**208,911**	**401,416**	**1,529,578**	**4,456,939**	**6,596,845**	

Note: Canada and US data only. Mexico data not available for 2002. *** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount. ** Includes transfers of metals and metal compounds to energy recovery, treatment, sewage and disposal. *** One TRI facility reported incorrect air releases of di(2-ethylhexyl) phthalate for 2002. The correct amount is shown in the tables in this section of the report but was received too late to be included in other tables of the report.**

Table 3-23 Summary of Total Reported Releases and Transfers of Manganese and its Compounds, 2002 (2000–2002 All Chemicals and Industries)

	North America (tonnes)	*Canadian NPRI (tonnes)*	*US TRI (tonnes)*
On-site Releases*	**84,157**	**6,087**	**78,069**
■ Air	1,437	295	1,142
■ Surface Water	3,968	1,055	2,913
■ Underground Injection	4,850	0	4,850
■ Land	73,894	4,730	69,164
Total Off-site Releases**	**36,117**	**5,432**	**30,685**
Total Releases On- and Off-site	**120,274**	**11,519**	**108,755**
Transfers to Recycling	**71,451**	**17,225**	**54,226**
Other Transfers Off-site for Further Management	**0**	**0**	**0**
Total Reported Amounts of Releases and Transfers of Manganese and its Compounds	**191,725**	**28,744**	**162,980**

Note: Canada and US data only. Mexico data not available for 2002. The data reflect estimates of releases and transfers of chemicals, not exposures of the public to those chemicals. The data, in combination with other information, can be used as a starting point in evaluating exposures that may result from releases and other management activities which involve these chemicals. *** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount. ** Includes transfers of metals and metal compounds to energy recovery, treatment, sewage and disposal.**

Table 3-24 North American Industries with Largest Total Reported Amounts of Releases and Transfers of Manganese and its Compounds, 2002 (2002 Matched Chemicals and Industries)

				Releases On- and Off-site								
				On-site Releases								
US SIC Code	*Industry*	*Total Reported Amounts of Releases and Transfers (tonnes)*	*(rank)*	*Air (tonnes)*	*Surface Water (tonnes)*	*Underground Injection (tonnes)*	*Land (tonnes)*	*Total On-site Releases* (tonnes)*	*Total Off-site Releases** (tonnes)*	*Total Releases On- and Off-site (tonnes)*	*(rank)*	*Transfers Off-site to Recycling (tonnes)*
33	Primary Metals	86,674	1	617	152	592	36,054	37,417	20,767	58,184	1	28,489
28	Chemicals	21,105	2	88	545	4,222	10,297	15,151	5,138	20,289	2	816
491/493	Electric Utilities	19,927	3	202	224	0	15,246	15,672	3,738	19,410	3	517
34	Fabricated Metals Products	17,451	4	185	2	0	65	255	610	865	8	16,586
26	Paper Products	10,817	5	103	2,800	0	5,870	8,773	1,962	10,735	4	82
37	Transportation Equipment	10,718	6	31	4	0	3	39	627	665	10	10,053
35	Industrial Machinery	7,440	7	55	2	0	2	60	344	404	12	7,037
36	Electronic/Electrical Equipment	5,732	8	33	1	0	0	34	449	483	11	5,249
7389/4953	Hazardous Waste Mgt./Solvent Recovery	4,499	9	1	0	18	4,117	4,136	362	4,498	5	0
—	Multiple Codes 20–39*	3,674	10	20	225	0	475	720	1,255	1,975	6	1,699
12	Coal Mining	1,093	11	0	7	19	1,067	1,093	0	1,093	7	0
32	Stone/Clay/Glass Products	1,031	12	30	0	0	420	451	303	755	9	277
24	Lumber and Wood Products	376	13	21	0	0	235	256	108	364	13	12
39	Misc. Manufacturing Industries	349	14	45	0	0	0	45	78	123	16	226
25	Furniture and Fixtures	267	15	1	0	0	0	1	0	1	20	266
20	Food Products	215	16	2	0	0	42	44	166	210	14	5
38	Measurement/Photographic Instruments	153	17	0	0	0	0	0	29	29	18	124
31	Leather Products	132	18	0	0	0	0	0	131	132	15	0
29	Petroleum and Coal Products	62	19	2	5	0	2	9	41	51	17	12
30	Rubber and Plastics Products	10	20	0	0	0	0	0	8	8	19	2
5169	Chemical Wholesalers	0	21	0	0	0	0	0	0	0.1	21	0
Total for Manganese and its Compounds		**191,725**		**1,437**	**3,968**	**4,850**	**73,894**	**84,157**	**36,117**	**120,274**		**71,451**

Note: Canada and US data only. Mexico data not available for 2002. *** The sum of air, surface water, underground injection and land releases in NPRI does not equal the total on-site releases because in NPRI on-site releases of less than 1 tonne may be reported as an aggregate amount. ** Includes transfers of metals and metal compounds to energy recovery, treatment, sewage and disposal.**

Commission for Environmental Cooperation
393, rue St-Jacques Ouest, Bureau 200
Montréal (Québec) Canada H2Y 1N9
t (514) 350-4300 *f* (514) 350-4314
info@cec.org / www.cec.org