

**Ten Key Carcinogens
in Toronto Workplaces and Environment:
Assessing the Potential for Exposure**

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March 2002



Reference: Toronto Public Health, *Ten Key Carcinogens in Toronto Workplaces and Environment: Assessing the Potential for Exposure*. Toronto, Ontario: March 2002.

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Acknowledgements: This report incorporates information and highlights from the technical study prepared for Toronto Public Health by Dr. Pavel Muller of ToxProbe Incorporated entitled *Potential for Occupational and Environmental Exposure to Ten Carcinogens in Toronto*. The technical review is being released concurrently with this summary report.

The views presented in this summary report are solely the views of the authors and Toronto Public Health. However, we would like to thank the Project Advisory Committee to the technical study, *Potential for Occupational and Environmental Exposure to Ten Carcinogens in Toronto*, who offered valuable advice and technical comments at several important points in this project.

This Project Advisory Committee included representatives from the Ontario Ministry of the Environment, the Ontario Ministry of Labour, the Occupational Health Clinic for Ontario Workers (Toronto), the Toronto Environmental Alliance (TEA), the Ontario Cancer Institute/Princess Margaret Hospital and Toronto Public Health.

We thank Dr. Lynn Marshall, Medical Director, Environmental Health Clinic, Sunnybrook and Women's College Hospital of Toronto for her participation in the project and expert review of this report.

We would also like to thank Dr. Monica Campbell of Toronto Public Health for comments and advice offered throughout this project.

Distribution: Both reports are available at: www.city.toronto.on.ca/health/

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EXECUTIVE SUMMARY

Background

This report has been prepared in response to recommendations contained in both Toronto's Environmental Plan approved by City Council in May 2000, and in the Toronto Cancer Prevention Coalition Action Plan approved by the Board of Health in May 2001. This assessment was conducted to determine what is known about the potential for occupational and environmental exposure to ten key known and probable carcinogens in Toronto's workplaces and environment. This report draws information from a background report entitled, *Potential For Exposure to Ten Carcinogens in Toronto's Environment and Workplaces* that was prepared on contract by Dr. Pavel Muller of ToxProbe Incorporated.

The ten carcinogens were chosen because of their potential to cause cancer and because they are frequently found in the workplace and in the environment. They are asbestos, benzene, 1,3-butadiene, cadmium, chromium (VI), dioxins, formaldehyde, polycyclic aromatic hydrocarbons (PAHs), tetrachloroethylene and trichloroethylene. For nine of the ten, there is strong evidence to indicate that they induce cancer in humans and strong agreement among regulatory agencies that they should be treated as known or probable human carcinogens. For the tenth contaminant, tetrachloroethylene, there is more contradictory evidence respecting its ability to induce cancer in humans, and less agreement among regulatory agencies about its classification as a human carcinogen.

Emissions and Exposure Data

The review of available emissions release data suggests that there are huge gaps in our knowledge about the sources of toxics released within the City's boundaries. Environment Canada's National Pollutant Release Inventory (NPRI) focuses primarily on large point sources only, when in fact, many of the emission sources within Toronto are mobile sources such as cars and trucks, area sources such as residential heating, and small and medium-sized commercial, industrial and institutional point sources.

Relatively good data are available on the levels of the selected carcinogens present in outdoor air although Toronto-specific data is missing for several contaminants. Two carcinogens – asbestos and chromium (VI) – could be present in Toronto's air at levels that are one or two orders of magnitude greater than those associated with a one in a million cancer risk. For these two carcinogens, Toronto-specific data were not available and a range of air levels from urban centres in Ontario/Canada was used. Toronto Public Health recommends that the Ontario Ministry of the Environment and Environment Canada should provide Toronto-specific monitoring results for these two contaminants so they can be prioritized for source reduction.

Information about contaminant levels in indoor air is very scarce. Air sampling conducted for formaldehyde indicates that it may be present in indoor air at levels that are an order of magnitude greater than the air levels deemed tolerable (i.e. those that correspond to a one in a million cancer risk). For formaldehyde and several other contaminants, indoor air can be the pathway of greatest exposure. For a number of the carcinogens examined in this report such as PAHs, 1,3-butadiene, tetrachloroethylene and trichloroethylene, exposure levels in indoor environments can vary significantly depending upon the activities undertaken, and the building materials, furnishings and consumer products used. For these

reasons, indoor air quality should be considered as a research priority for Toronto Public Health in the coming year.

The knowledge gaps for occupational exposures are wider and even more severe than for environmental exposures. Currently, there is no publicly available information on occupational exposure levels experienced in Ontario workplaces. The Ontario Ministry of Labour used to systematically collect exposure information on selected chemical and physical agents for Ontario workplaces but this data has not been collected for several years. The older data are currently being transferred into a new database and were not available for this study. Without proper exposure data, it is not possible to estimate the burden of illness associated with occupational exposure to carcinogens. Nor is it possible to ensure that workers are being adequately protected from carcinogens in the workplace. Toronto Public Health recommends that the Ontario Ministries of Labour and Health and Long-term Care, and Cancer Care Ontario prioritize carcinogens in Ontario workplaces for further investigation and exposure assessment.

Health Assessment and Conclusions

While the contaminant levels in Toronto's outdoor air are not higher than those found in other large urban centres, the data suggests that nine of the ten carcinogens (all 10 contaminants except tetrachloroethylene) examined in this report tend to be present in Toronto's outdoor air at levels that approach or exceed the air levels deemed "tolerable" (i.e. the level associated with a one in a million excess cancer risk). A number of the carcinogens examined can also be found in indoor air at levels approaching or exceeding the levels considered tolerable (i.e. formaldehyde, benzene, 1,3-butadiene, trichloroethylene and polycyclic aromatic hydrocarbons or PAHs). Several can also be found in food products, soil, and lake sediments.

Two of the ten carcinogens – benzene and polycyclic aromatic hydrocarbons (PAHs) – are present in outdoor air at levels that are ten times higher than the levels considered tolerable and should be given high priority by the City for actions that will reduce emissions. The transportation sector is likely the most significant source of emissions for both these contaminants within the City. Toronto Public Health also recommends that the Ontario Ministry of the Environment and Environment Canada move quickly to establish air standards for PAHs to drive improvements in air quality.

Estimates of the levels of dioxins taken into the body indicate that most residents of the Great Lakes Basin (which includes residents of Toronto) are being exposed to levels of dioxins that are within or above the range at which adverse health effects may be expected. For dioxins, which are released as unintentional by-products from the incineration of medical and municipal wastes, the burning of diesel fuel in vehicles, and to a lesser extent, the burning of wood in fireplaces, food is the most significant pathway for exposure. Dioxins have been identified for "virtual elimination" by a number of national and international agreements. For these reasons, the City needs to give high priority to the development of a strategy that supports their virtual elimination.

Estimates derived with the CAREX model indicate that a significant number of Toronto workers may be exposed to PAHs in the transportation sector, tetrachloroethylene in clothing manufacturing and dry-cleaning, formaldehyde in clothing and furniture manufacturing, chromium in a variety of sectors, benzene in whole/retail/restaurants/hotels and the personal services sectors, and asbestos in wholesale/retail/restaurants/ hotels, above typical outdoor air levels. Very little is known about the level of exposure in many of these situations. Toronto Public Health recommends that the Ontario Ministries of Labour and Health and Long-term Care and Cancer Care Ontario prioritize these contaminants and industrial sectors for investigation to determine the extent and level of worker exposure to carcinogens.

The published occupational exposure data that is available (which is based on workplaces in the United States) indicates that workers can be exposed to the selected contaminants at levels that are several orders of magnitude (i.e. 100 to 10,000 times) greater than the exposure levels deemed “tolerable” for environmental exposures in terms of cancer risk. These exposure levels suggest that workers may be exposed to the selected contaminants at levels that correspond to a cancer risk of one in a hundred to one in ten thousand. While workers are potentially exposed for fewer hours per day (8 hours instead of 24 hours), fewer days per week (5 instead of 7) and fewer years (45 instead of 70 years) than is assumed by regulatory agencies for environmental exposures, this difference would only justify a 5 to 10-fold increase in the exposure levels deemed “tolerable” for workers. It is also true that the working population does not include many of the more vulnerable members of society such as young children, the sick, and the elderly.

However, the working population does include men and women who intend to become parents and women who are pregnant or breast-feeding, and the scientific literature does suggest childhood cancer is linked to occupational exposure of both parents before conception and of mothers during pregnancy.

Many of the selected carcinogens present a health concern to both workers and members of the public. For example, formaldehyde, trichloroethylene and tetrachloroethylene, that can be detected in relatively high levels in workplaces where they are used to manufacture or treat products, can be found in lower levels in homes, offices and public spaces when they off-gas or vapourize from the products in which they have been used. The carcinogens present in outdoor air because of vehicle exhaust such as benzene, PAHs, formaldehyde and 1,3-butadiene, can present a hazard to people who work in and around vehicles operated on gasoline and diesel (eg. trucking, loading docks, bus stations, taxis). In some situations, workers are exposed to chemicals such as chromium or cadmium in the workplace before they are emitted into the larger environment where they may present a hazard to members of the general population.

This study indicates that Toronto residents are routinely exposed to a variety of carcinogenic chemicals in their environment, and that these exposures are associated with a lifetime cancer risk greater than 1 in a million excess cancers. This level of cancer risk exceeds the level considered “tolerable” by Toronto Public Health and many other health agencies throughout the world. Based on the 10 key chemicals assessed in this study, both the outdoor and indoor air exposure pathways appear to present the greatest cancer risk for many of the carcinogens examined. For exposures related to the food pathway, dioxins are flagged as being of priority concern. For dioxins, ingestion from food is the pathway of greatest concern because dioxins released in the air and water will bioaccumulate through the food chain. Toronto’s municipally treated drinking water is a negligible source of cancer risk from the ten contaminants assessed in this study.

This study indicates that in addition to the routine exposure of Toronto residents to a variety of environmental carcinogens, many of these people are exposed to additional and even higher levels of carcinogens at work, depending on the type of work they do. It is the multiplicity of exposures to known and probable carcinogens in Toronto’s indoor and outdoor air, in some food sources, and in many workplaces that gives rise to Toronto Public Health’s concerns about the contribution of workplace and environmental carcinogens to cancer rates in Toronto. It is only through better assessment of exposure to contaminants that we can prioritize other carcinogens for priority reduction through implementation of pollution prevention strategies.

Pollution prevention strategies that aim to reduce or eliminate the contaminant at source can produce public health benefits for people whether they are at work, at home or outdoors. Given that people in industrialized societies are exposed to low levels of thousands of different chemical and physical agents simultaneously, that the toxicity of many of those substances are not known, and that very little is known about the interactions between contaminants on the human body, it is important to target those

contaminants with both high exposure potential and high cancer risk, in workplaces and the environment, such as those selected for this report, for priority emissions reduction wherever possible.

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1.0 BACKGROUND

The Board of Health, at its meeting of July 27, 1998, supported the formation of the Toronto Cancer Prevention Coalition and requested that it report back to the Board with a proposed action plan for cancer prevention. At its meeting of May 22, 2001, the Board of Health received a report entitled, "Toronto Cancer Prevention Coalition Action Plan" and endorsed, in principle, the 20-point action plan developed by the Toronto Cancer Prevention Coalition. Several points in that plan are directed towards chemical and physical carcinogens in Toronto's workplaces and environment.

In May 2000, Toronto City Council approved, in principle, the plan prepared by Toronto's Environmental Task Force entitled, *Environmental Plan, Clean, Green and Healthy, A Plan for an Environmentally Sustainable Toronto* (February 2000). Several of the recommendations contained in this plan are directed towards toxic air pollutants. Recommendation 20a indicates that the City should work to make Toronto's air clean and free of harmful levels of pollutants. Recommendation 22a) indicates that the City should assess the need to improve City, provincial and federal emissions inventories, while recommendation 22c) indicates that the City should explore the feasibility of introducing a by-law to restrict point source emissions.

This report has been prepared to provide background information to support the on-going work of the Toronto Cancer Prevention Coalition, and to support City staff with the implementation of the recommendations contained in the Coalition's May 2001 report and in the Environmental Task Force's May 2000 report. In large part, this report is based on the technical report entitled, *Potential for Occupational and Environmental Exposure to Ten Carcinogens in Toronto* that was prepared for Toronto Public Health by Dr. Pavel Muller of ToxProbe Incorporated. The technical report benefited from the direction and advice of a Project Advisory Committee that included staff from the Ontario Cancer Institute, the Ontario Ministries of Labour and the Environment, the Occupational Health Clinic for Ontario Workers, the Toronto Environmental Alliance (TEA) and Toronto Public Health.

The ten contaminants included in this report – asbestos, benzene, 1,3-butadiene, cadmium, chromium, dioxins, formaldehyde, polycyclic aromatic hydrocarbons (PAHs), tetrachloroethylene, and trichloroethylene – were selected for their potential to be present in both Toronto's indoor and outdoor environments as well as for their carcinogenic potential. Pesticides and electromagnetic fields (EMFs) related to power lines and radio towers were not included because their health effects have been the subject of previous reports prepared by Toronto Public Health, and because these topics were considered too complex for the budget allocated to this project.

All material in this report that has not been referenced has been drawn from the technical review prepared by ToxProbe. The context, interpretation of the technical information, and recommendations contained in this report reflect the positions of Toronto Public Health and do not necessarily reflect those of ToxProbe or the members of the Project Advisory Committee.

2.0 INTRODUCTION

Cancer Incidence and Mortality in Toronto

Toronto is the largest city in Canada with a population of 2.4 million. Toronto accounts for about 22% of Ontario's population and 8% of Canada's population. Cancer is second only to circulatory disease as the leading cause of death in Toronto. It causes more deaths than respiratory disease, diabetes, accidents, injuries, and AIDS combined. Between 1991 and 1995, cancer was responsible for an average of 4,620 deaths each year in Toronto. While increased cancer incidence and mortality are associated with aging, approximately 40% of new cancer cases and 30% of cancer deaths occur in Toronto residents who are between the ages of 20 and 64 (TPH, June 2000).

For women in Toronto, breast cancer (about 29 per 100,000) is the leading cause of cancer fatalities followed by lung cancer (about 25 per 100,000) and then colorectal cancer (about 15 per 100,000). Between 1986 and 1995, the incidence of breast cancer increased although mortality decreased. For Toronto men, lung cancer is the leading cause of cancer fatalities (about 62 per 100,000) followed by colorectal cancer (about 24 per 100,000) and prostate cancer (about 26 per 100,000). Between 1986 and 1995, the incidence of lung cancer decreased while the incidence of prostate cancer increased (TPH, June 2000).

Between 1991 and 1995, approximately 90 children per year (0 to 19 years of age) in Toronto were diagnosed with cancer. Leukemia was the leading type of cancer (about 27% of cases) for this age group followed by brain cancer (about 15% of cases), Hodgkin's Lymphoma (about 11% of cases) and Non-Hodgkin's Lymphoma (about 7% of cases). While the numbers in Toronto are too small to discern a trend, on a national level, there has been a gradual increase in all childhood cancers combined (TPH, June 2000).

Childhood Cancer

Cancer in childhood is rare but it is the most common cause of death due to disease in the age range of 1 to 14 years (McBride, 1998). While there has been a significant improvement in the survival rate of children with cancer in the last twenty years (NCI, 1999), there has been no decrease in the incidence of childhood cancers in Canada (NCIC, 2001). Some scientists have suggested that exposure to carcinogens in the environment is one factor that may contribute to childhood cancer. However, the study of childhood cancers is complicated by the rarity of the disease and by the poor exposure profiles that exist for many of the risk factors being examined (McBride, 1999), making it difficult to establish definitive links between environmental exposures and childhood cancer at this time.

Environmental factors proposed as risk factors for childhood cancer include exposure to ionizing radiation, electromagnetic fields, chemicals in drugs and food products, second-hand smoke, and parental occupational exposures (McBride, 1998). Children can be exposed to carcinogens directly and indirectly, and at different developmental points (i.e. before conception, during pregnancy and after birth). A number of studies have examined the link between childhood cancers such as leukemia, brain tumors, kidney cancer (particularly Wilm's tumor), liver cancer and maternal and paternal occupations that involve

exposure to hydrocarbons such as solvents, metals, pesticides and occupations in the manufacturing, medical, dental, personal service, agricultural and forestry sectors. While the results of these studies have been inconsistent and contradictory, a number have demonstrated an association between parental occupational exposures and increased rates of childhood cancer (McBride, 1998). Contradictory findings have been found as well among studies that have examined childhood cancers among children with direct exposure to pesticides, electromagnetic fields, and smoking in their homes.

Cancer and the Environment

It is very difficult to prove that environmental exposures to chemical and physical agents are causing cancer because of the difficulties involved in estimating personal exposures and because of confounding exposures such as second-hand smoke. There are, however, many reasons to believe that environmental exposures contribute to the incidence of cancer in the general population. A number of “known and probable human carcinogens” are present in outdoor air and in other media to which the general population is regularly exposed. Among urban residents, gradients of air pollution levels frequently correspond to area differences in the risk of lung cancer. And in communities with large point sources of carcinogens, where adjustments have been made for tobacco and occupational exposures, the risk of lung cancer is proportional to the nearness of the household to the point sources (Shy, 1996).

Several studies have examined associations between proximity to streets with high density traffic and the occurrence of childhood cancers and childhood leukemia. It is believed that the streets with high traffic density reflect chronic exposure to the carcinogenic components of vehicle exhaust such as PAHs, benzene and 1,3-butadiene. Several of these studies, which took into account exposures such as parental smoking, have demonstrated a strong and statistically significant association between high traffic neighborhoods ($\geq 20,000$ vehicles per day) and childhood cancers including leukemia (Pearson, 2000).

The results of the ten-year Harvard Six Cities epidemiological study conducted by Dockery and colleagues suggest a much higher relative risk for lung cancer from air pollution than most previous studies. This study, which had the benefit of air monitoring results for the entire period in which the population was followed as well as detailed information about confounding factors such as smoking habits, estimated that lung cancer mortality could increase by 14 to 20% for every $10 \mu\text{g}/\text{m}^3$ increase in long-term exposure to respirable particulates ($\text{PM}_{2.5}$) or inhalable particulates (PM_{10}). These estimates correspond to a relative risk of about 1.4. The air levels of inhalable particulates in the six cities studied ranged from a low of $18.2 \mu\text{g}/\text{m}^3$ to a high of $46.5 \mu\text{g}/\text{m}^3$ (Shy, 1996) while the average daily levels of inhalable particulates ranges from 19.7 to $23.9 \mu\text{g}/\text{m}^3$ at the three monitoring stations in Toronto. This suggests that small particles could be contributing to cancer risk in Toronto.

In order to calculate the proportion of cancers attributed to ambient air pollution, two parameters must be estimated, the average relative risk associated with exposure to air pollution and the proportion of the population exposed (Shy, 1996). Currently, there is too much uncertainty associated with estimates of relative risk and too little information about individual exposure levels to produce an accurate estimate of air pollution’s impact on cancer rates.

Cancer and the Workplace

“Industrial workers have long served as the sentinels for the general population with regard to environmental hazards” according to Dr. Aaron Blair of the Occupational Studies Section of the National Cancer Institute (NCI). Many of the well-established chemical carcinogens such as benzene and asbestos have been identified through studies of workers occupationally exposed. Workers can experience prolonged and/or intense exposures to chemical and physical agents that are found in low levels in non-occupational environments.

The International Agency for Research on Cancer (IARC), an agency of the World Health Organization (WHO), recognizes 87 chemical, physical and biological agents as “known human carcinogens” including asbestos, benzene, chromium (VI) compounds and dioxins, which are discussed in this report. It recognizes another 63 agents as “probably carcinogenic to humans” including 1,3-butadiene, polycyclic aromatic hydrocarbons (PAHs), the dry-cleaning solvent, tetrachloroethylene, and the degreasing agent, trichloroethylene, that are discussed in this report. It also identifies another 233 agents or mixtures as “possibly carcinogenic to humans” including gasoline engine exhaust, fibreglass, and methylene chloride (IARC, 2000).

Estimating the contribution of occupational exposures to cancer incidence is controversial and complex because of the dearth of information related to occupational exposures. Estimates range from a low of 4% to a high of 20% of all cancers being attributable to carcinogen exposures in the workplace. Cancer Care Ontario (CCO) has been citing 9% as the proportion of cancer deaths attributable to occupation on the basis of a report published by A.B. Miller in 1992. This suggests that approximately 400 cancer deaths per year in Toronto could be the result of workplace exposures. This assumes that about 9% of the approximately 4,620 cancers deaths in Toronto each year arise from workplace exposures to carcinogens.

3.0 CARCINOGENIC POTENTIAL OF SELECTED CONTAMINANTS

Assessing the Carcinogenic Potential

The International Agency for Research on Cancer (IARC) was the first organisation to develop a system for the classification of contaminants for their carcinogenicity (potential to produce cancer) in humans. IARC convenes a panel of international experts to systematically evaluate the evidence of carcinogenicity for contaminants. Other agencies have developed similar ranking schemes. Of these, the one published by the United States Environmental Protection Agency (US EPA) in 1986 is probably the most influential.

In 1996, the US EPA replaced its ranking scheme based on letter ranks with a new descriptive scheme which takes into account a wider range of data. However, the number of agents that has been ranked by the 1996 scheme is relatively small, so the 1986 scheme is still widely used. The ranking schemes by IARC and the US EPA (1986) are quite similar. Health Canada has developed a carcinogen ranking scheme under the Canadian Environmental Protection Act (CEPA). The CEPA scheme consists of more categories and sub-categories than those produced by IARC and the US EPA. Health Canada distinguishes between carcinogens that initiate cancer and those that promote cancer and tends to give the latter group a lower cancer ranking when epidemiological evidence is inadequate.

Cancer Classification of the Ten Contaminants

There is a strong agreement among IARC, the US EPA and Health Canada that nine of the ten contaminants selected for this assessment are known or probable human carcinogens (see Table 1 below). The one exception is tetrachloroethylene (also known as perchloroethylene) which is classified as a “probable human carcinogen” by IARC, a “probable or possible human carcinogen” by the US EPA, and “unlikely to be carcinogenic to humans” by CEPA. Until there is greater clarity on the carcinogenic potential of tetrachloroethylene, Toronto Public Health will treat it as a possible or probable human carcinogen.

A number of the ten contaminants selected also present a cancer concern when ingested. This is particularly clear for dioxins, some PAHs and benzene.

Table 1: Cancer Classifications for Ten Contaminants by US EPA, IARC and CEPA

	Classification – Inhalation			Oral
	US EPA ¹	IARC (WHO) ²	CEPA ³	ToxProbe ¹ (Using US EPA data)
1,3-Butadiene	Probable human carcinogen ⁷	Probable human carcinogen	Highly likely human carcinogen	Probable human carcinogen ⁴
Asbestos	Human carcinogen	Human carcinogen	NA	Possible human carcinogen ⁵
Benzene	Human carcinogen	Human carcinogen	Human carcinogen	Human carcinogen ⁴
Cadmium	Probable human carcinogen	Human carcinogen	Probable human carcinogen	Possible carcinogen or not classifiable ⁵
Chromium (VI)	Human carcinogen	Human carcinogen	Human carcinogen	Not classifiable ⁵
Dioxins	Probable human carcinogen ⁸	Human carcinogen	NA	Probable human carcinogen ⁸
Formaldehyde	Probable human carcinogen	Probable human carcinogen	NA	Not Classifiable ⁵
PAHs (B[a]P)	Probable human carcinogen	Probable human carcinogen	Probable human carcinogen	Probable carcinogen ⁴
Tetrachloroethylene	Probably to possible human carcinogen ⁶	Probable human carcinogen	Unlikely human carcinogen	NA
Trichloroethylene	Withdrawn (was probable human carcinogen)	Probable human carcinogen	Probable human carcinogen	Withdrawn

1. *Iris Database (US EPA 1986)*

2. *IARC Monographs*

3. *Priority Substances List Reports*

4. *Not differentiated from inhalation*

5. *ToxProbe Inc interpretation from IRIS*

6. *ATSDR, 1995*

7. *US EPA (1998a)*

8. *US EPA (2000)*

Cancer Initiators and Promoters

Some carcinogens are capable of inducing cancer by producing an irreversible mutation in the DNA of the cells in the body. These carcinogens are called genotoxic because of their ability to affect the gene of the cell. They are also called initiators because they can begin or initiate the cancer process. These cancer-inducing compounds are considered non-threshold carcinogens because it is believed that there is no threshold below which there is no risk of excess cancer. There is a strong consensus among IARC, the US EPA and Health Canada that 1,3-butadiene, benzene, chromium (VI), formaldehyde and PAHs are genotoxic carcinogens capable of initiating cancer. There are conflicting views about whether asbestos, cadmium and trichloroethylene are genotoxic.

Other carcinogens are capable of accelerating the development of cancer. These carcinogens are called promoters because, while they do not initiate the cancer process, they promote its development and progress. These carcinogens are also called threshold carcinogens because it is believed that there is a threshold of exposure below which exposure does not present a cancer risk. Above the threshold, the risk of cancer promotion increases as the exposure level increases. The evidence suggests that asbestos, formaldehyde and at least one of the PAHs (benzo[a]pyrene) are promoters of cancer. Dioxins and similar compounds are promoters that induce the cancer effect through activation of the Ah receptor. The carcinogens that are capable of both initiation and promotion such as formaldehyde, some PAHs, and perhaps asbestos, are called complete carcinogens.

Some regulatory agencies build greater margins of safety into the calculation of exposure limits for cancer initiators than they do for cancer promoters on the assumption that promoters will not be found in the environment¹ at the higher levels required to exert their cancer effect. It must be recognized however that some cancer promoters, such as dioxins, can exert their cancer promoting effects at levels of exposure that are less than those commonly experienced in the environment.

In occupational settings, cancer promoters may present as much risk as cancer initiators given that exposure levels tend to be higher than in the general environment.

¹ Throughout this report, the word “environment” will be used to mean all non-occupational environments including residential indoor air, outdoor air, drinking water, surface water, soil, dust, food and consumer products.

Cancers Linked to the Contaminants

Different carcinogens have a tendency to induce different types of cancer at different sites in the body. Table 2 below summarizes the types of cancers most clearly associated with each of the ten contaminants. It is important to bear in mind that, besides the cancer concerns, all of the selected agents present other toxic effects that may play an important role in their overall toxicity. The non-cancer effects may be particularly important in occupational settings where exposures tend to be higher than environmental exposures.

Table 2: Sites and Types of Cancers in Humans for the Ten Contaminants

	Cancer types	Reference
1,3-butadiene	Cancer of lymph and blood systems (leukemia, lymphosarcoma and reticulum cell sarcoma)	ATSDR (1992)
Asbestos	Lung cancer and mesothelioma (cancer of the membrane surrounding the lungs and other organs)	ATSDR (1995)
Benzene	Acute myeloid leukemia	ATSDR (1997)
Cadmium	Lung cancer	ATSDR (1993a)
Chromium(VI)	Lung cancer	ATSDR (1993b)
Dioxins	All cancers combined, lung cancer and soft tissue sarcoma, liver cancers in animals	USEPA (1994b)
Formaldehyde	Nasal and nasopharyngeal tumours	IPCS (1989a) ATSDR (1999)
PAHs (B[a]P)	Ingestion: mainly stomach tumors; Inhalation: mainly lung tumors; skin absorption: mainly skin tumours	ATSDR (1995d)
Tetrachloroethylene	Weak evidence for cancer in humans	CEPA (1993d)
Trichloroethylene	No consistent pattern as to the type of cancer	CEPA (1993a)

Cancer Potency of the Contaminants

The distinction between threshold and non-threshold effects is used by regulating agencies when establishing exposure limits for chemical contaminants. For chemicals with a threshold, a dose-response assessment is used to identify the threshold level at which no adverse effect is expected (i.e. the no observable adverse effect level [NOAEL]). By applying an appropriate safety factor that accounts for the uncertainties in the estimation of the threshold, the reference dose (RfD) or tolerable daily intake (TDI) can be calculated. The TDI or RfD can be used as an indicator of cancer potency.

Since there is no “safe” level of exposure for non-threshold carcinogens, it is deemed necessary by regulatory agencies to establish a level of exposure for each chemical that is deemed “tolerable” or “acceptable”. Such a level is called a risk-specific dose (RsD). Generally for environmental exposures, most organisations define “tolerable” risk as one excess cancer case per million people exposed daily over a life-time. The risk-specific dose (RsD), which reflects both the potency of the contaminant being assessed as well as the risk level deemed tolerable, can be used to calculate the unit risk which can be used as an estimate of cancer potency.

The number of cancer cases expected with any carcinogen increases as the exposure levels increases. With more potent carcinogens, the number of people affected will increase more quickly as the exposure level increases. In other words, a more potent carcinogen is expected to induce cancer at lower exposure levels and to induce a greater number of cancer cases as exposure increases.

Among the initiators, carcinogenic PAHs and chromium (VI) are considered the most potent carcinogens by inhalation, followed by asbestos and cadmium. 1,3-butadiene and benzene are about three to four orders of magnitude (i.e. 1,000 to 10,000 times) less potent than PAHs and chromium (VI). Formaldehyde is a weak initiator but a strong promoter. Dioxins and related compounds are considered very potent promoters of cancer.

4.0 ENVIRONMENTAL EXPOSURE TO THE TEN CARCINOGENS

Sources of Emissions to the Environment

It was not possible to obtain accurate estimates of environmental emissions for the ten contaminants selected for study within Toronto. When Environment Canada's National Pollutant Release Inventory (NPRI) database was checked for emission sources within Toronto for 1999 (i.e. the most recent year for which data were available) there were no entries for asbestos or 1,3-butadiene, only one facility for benzene, three facilities for both cadmium and chromium, and four for formaldehyde, tetrachloroethylene and trichloroethylene. There were no entries for PAHs or dioxins because they were not reportable substances before 2000.

NPRI focuses primarily on large point sources while many of the emission sources within Toronto are mobile sources such as cars and trucks, area sources such as residential heating, and small commercial and industrial point sources such as autobody and printing shops. Facilities only have to report emissions to NPRI if they employ 10 or more employees (or an equivalent of 20,000 worker hours per year), manufactured, processed or used more than 10,000 kilograms (i.e. 10 tonnes) of the reportable substance in the reporting year, and the reportable substance was manufactured, processed or used at a concentration of 1% or more. Dioxins and PAHs (17 individual PAHs) were added to the list of reportable substances in 2000. The quantity cut-off for PAHs is 50 kilograms or more while there will be no concentration cut-off for dioxins (Environment Canada, 2000a).

The Ontario Ministry of the Environment (MOE) has recently established a mandatory monitoring and reporting requirement (i.e. the Airborne Contaminant Discharge Monitoring and Reporting Regulation 127/01) as the basis for a provincial inventory of pollutant emissions within Ontario. This Regulation lists all of the NPRI substances at the threshold levels required under NPRI, as well as some additional substances. It appears to target large point sources as well.

The technical report includes emissions data from the US EPA's Toxic Release Inventory (TRI) that can be used to identify the potential emission sources for the ten contaminants within and around the City of Toronto. U.S. emissions inventories were used because they include a wider range of data than the NPRI. However, these inventories will include emission sources that may not be relevant to Toronto while excluding emissions sources, such as vehicles, hospitals, airports, and service businesses such as auto service stations, that are relevant to Toronto.

This assessment suggests that there are huge gaps regarding the sources of air toxics released within the City's boundaries. The Air Quality Improvements Branch (AQIB) within Toronto Works and Emergency Services plans to supplement the emissions inventories kept by the provincial and federal governments by targeting emissions from mobile, area and small point sources that are excluded from provincial and federal emission inventories. The AQIB plans to target the criteria contaminants as well as a few prioritized air toxics. The AQIB should proceed on a sector by sector basis giving priority to the "air toxics" identified in this report. Toronto Public Health recommends that the provincial and federal levels of government expand their proposed and existing inventories to capture a greater percentage of small and medium sized point sources.

Contaminant Levels – Indoor and Outdoor Air

For most of the ten contaminants reviewed, inhalation is the most important route of exposure because people tend to be more sensitive to the selected contaminants when inhaled than when ingested or absorbed through the skin. Exposures encountered in indoor environments usually have a greater impact on health than outdoor exposures because most people in northern climates spend more time indoors than outdoors. However, many of the contaminants present in the outdoor air readily penetrate into indoor air through ventilation systems, windows and doors. As a result, outdoor air levels can significantly influence the total exposure by inhalation, even though most of that exposure takes place indoors. Most, if not all, of the contaminants considered in this report are expected to readily enter the indoor environment when present in outdoor air.

Compared to estimates of quantities released, relatively good data are available on the levels of the selected contaminants present in outdoor air although Toronto-specific data are missing for several contaminants (see Table 3 below). Information about contaminant levels in indoor air in Toronto homes and public spaces is much more scarce. Indoor air quality can be affected by the construction materials, furnishings and consumer products used as well as by the activities undertaken indoors. Asbestos may be found in the flooring, ceiling tiles, shingles and heating system insulation in older buildings. Formaldehyde can be released from building materials such as plywood, as well as from furniture, permanent press fabrics, draperies, carpets and mattress ticking. Fireplaces and cigarette smoking can emit 1,3-butadiene, benzene, cadmium, chromium and PAHs into the indoor environment. Contaminants such as trichloroethylene and tetrachloroethylene can be released from household products such as paints, paint strippers, wood preservatives, aerosol sprays, cleansers and dry-cleaned clothing.

Table 3: Outdoor Air Levels of Ten Contaminants and Air Levels that Correspond to a One in a Million Cancer Risk

Contaminants	Air Level that Corresponds to One in a Million Cancer Risk	Ambient Air Levels	Data Location
Asbestos (PCM fibres/mL)	0.000004	0.000003 - 0.003	Urban areas
Benzene ($\mu\text{g}/\text{m}^3$)	0.24	1.3 – 3.1 2.2 average	Toronto
1,3-Butadiene ($\mu\text{g}/\text{m}^3$)	0.16	0.32 average 0.07-0.11 average	5 stations: Toronto 2 stations: Toronto
Cadmium ($\mu\text{g}/\text{m}^3$)	0.00056	0.00024 - 0.00072 0.00042 average	Southern Ontario
Chromium ($\mu\text{g}/\text{m}^3$)	0.000083 (Chromium VI)	0.003 - 0.009 (Total Chromium)	12 Ontario Cities
Dioxins (pg/m^3 TEQ)	0.01 (US EPA)	0.4-36.7 0.090-0.26 average	North America Canada: urban sites
Formaldehyde ($\mu\text{g}/\text{m}^3$)	3.6	3.3 average 2 – 4 1.8 to 6.1 average	Canada: 4 urban/4 suburban Canada: Large Cities Toronto, Windsor & Ottawa
PAHs ($\mu\text{g}/\text{m}^3$ B[a]P)	0.000043	0.0003 average 0.00014 summer 0.00036 winter	Toronto Toronto Toronto
Tetrachloroethylene ($\mu\text{g}/\text{m}^3$)	250 (WHO)	5 average	Toronto
Trichloroethylene ($\mu\text{g}/\text{m}^3$)	1.6	0.32-2.8	Toronto

* Data provided in CCME or CEPA reports or by Environment Canada

Potential Routes of Exposure

Toronto is a city with a strong industrial past. As a result, some of the lands in downtown Toronto and many low-lying areas were filled partly with industrial wastes which could contain PAHs, cadmium or chromium. Soils in Toronto may also be contaminated directly from past or present industrial operations. In Toronto, high PAH levels are found on sites that used or produced coal tar, such as coal gasification sites or roofing operations. Benzene is most often found as a component of gasoline or diesel fuel contamination. Trichloroethylene and tetrachloroethylene are found where they have been used as solvents or degreasing agents on site. Chromium may be found on metal-finishing sites. 1,3-butadiene, which is a gas at room temperature, is not found in contaminated soils

People can be exposed to contaminants in the soil from skin contact with the soil when gardening or playing on the site; inadvertent ingestion of soil or dust blown into a house through hand to mouth activities; inhalation of vapours that enter buildings through cracks in the foundation; and ingestion of food grown in the contaminated soil. Soil contamination in Toronto is highly variable from one site to the next and is not well documented.

Food can be an important route of exposure both in terms of the quantity of the contaminant that can be taken into the body, and in terms of the toxic effects associated with that route of exposure. The level of chemical contamination in food products depends upon the environmental conditions in which they are grown, the farming practices employed, the processes to which they are exposed, and the practices used in their preparation. For example, smoked or barbecued meat can have very high levels of PAHs.

Environmental exposure can be heavily influenced by lifestyle factors. For example, families that depend solely upon vegetables grown in the backyard can be at increased risk of cancer if their soil is contaminated with carcinogens. Likewise, families that consume large quantities of fish caught in the Great Lakes can be at increased risk of cancer from exposure to dioxins. In order to better understand the pattern and extent of exposures by these routes, it is important to gather information about lifestyle factors related to food growing, processing and consumption patterns.

Reports on Toronto's drinking water indicate that drinking water is a negligible source of exposure to these ten contaminants in Toronto.

Intake Levels from Environmental Sources

Intake levels are estimates of the quantity of a contaminant that may actually contact the body's lungs, gastro-intestinal tract and skin. They can be calculated for the different exposure pathways (i.e. water, food, air) and for the different ages and circumstances of the general population. Intake levels can be used to compare the relative importance of different routes and pathways of exposure. They must be interpreted carefully however, because they do not always identify the route or pathway that is associated with the greatest harm. Because carcinogens can be more potent by one route of exposure than another, a less significant path of exposure could still present the greatest level of risk. The significant intake levels are highlighted in the contaminant-specific summaries provided later in this report. (See Section 6.0).

5.0 OCCUPATIONAL EXPOSURE TO THE TEN CARCINOGENS

Industrial Exposures

The Ontario Ministry of Labour used to systematically collect exposure information on selected chemicals and physical agents for Ontario workplaces but this data has not been collected for several years. The older data are currently being transferred into a new database and were not available for this report. Although some data may be available from the Ministry of Labour in the future, it may not reflect current working conditions and it may not reflect exposures to all of the carcinogens currently being used in Ontario workplaces.

In the absence of current exposure data, an attempt has been made to estimate the number of Toronto workers who may be occupationally exposed to the ten contaminants and to identify the industrial sectors in which those exposures may occur. These estimates have been derived using the CAREX model developed by the Finnish Institute of Occupational Health in collaboration with IARC. CAREX contains estimates of the number of workers occupationally exposed to 139 carcinogens by different industrial sectors in 15 countries of the European Union (EU) and in four non-EU countries including the United States. With this model, workers are considered “exposed” if they are exposed to the contaminants at levels exceeding those typical of outdoor air in Europe. A weakness of this approach is that it assumes that occupational exposures in Toronto in 2001 are comparable to those in the United States in the early 1980s and to those in Finland in the late 1980s and early 1990s.

Number of Workers Potentially Exposed

While the estimates derived from CAREX do not indicate the levels of exposure that may be encountered in Toronto workplaces, they do identify the contaminants to which the largest number of workers may be exposed and the industrial sectors in which the largest number of workers may be exposed (see Table 4 below). The estimates indicate that the largest number of Toronto workers are potentially exposed to: PAHs in the land transport sector (e.g. trucking); tetrachloroethylene in the clothing manufacturing sector and in dry-cleaning establishments; formaldehyde in the clothing manufacturing and furniture and fixtures manufacturing sectors; chromium (VI) in a number of industrial sectors; benzene in the personal and household services (e.g. dry cleaners, hair dressers) sector and in the wholesale and retail trade and restaurants and hotels sector; and asbestos in the wholesale and retail trade and restaurants and hotels.

These sectors and contaminants should be given priority for further investigation. Future investigations should focus on determining the extent and level of worker exposures in each sector, the sources of those exposures, and the actions that could be taken to reduce or eliminate exposures where warranted.

The high estimates derived for potential exposure to benzene in the personal and household services sector and in the wholesale and retail trade and restaurant and hotels sector likely reflect the potential to be exposed to second-hand smoke in these sectors. Given that smoking is now prohibited in most Toronto workplaces, these numbers likely over-estimate the potential for benzene exposure in these two sectors in

Toronto. These estimates do indicate, however, the exposure reduction benefit that can occur with full implementation of policies to restrict smoking in the workplace.

Table 4: Major Industrial Sectors with Greatest Number of Workers Potentially Exposed to the Ten Contaminants in Toronto Based on CAREX Modelling

Contaminant	Sector with Greatest Potential for Exposure	Number of Toronto Workers Potentially Exposed to Specific Contaminants
Asbestos	Construction	1240
	Wholesale and retail trade and restaurants and hotels	4846
1,3-Butadiene	Manufacture of plastic products	116
Benzene	Wholesale and retail trade and restaurants and hotels	6059*
	Personal and household services (includes dry cleaners)	8123*
	Land transport (e.g. trucks, buses, taxis)	247
Cadmium	Manufacture of plastic products	298
	Manufacture of other non-metallic mineral products	638
	Non-ferrous metal basics industries	374
	Other manufacturing industries	230
Chromium	Manufacture of textiles	1060
	Printing, publishing and allied industries	365
	Manufacture of plastic products	326
	Manufacture of fabricated metal products	617
	Manufacture of machinery except electrical	494
	Manufacture of transport equipment	287
Formaldehyde	Personal and household services	734
	Manufacture of clothing apparel, except footwear	1794
PAHs	Manufacture of furniture and fixtures	4846
	Manufacture of clothing apparel, except footwear	842
PAHs	Iron and steel basic industries	295
	Wholesale and retail trade and restaurants and hotels	569
	Land Transport (e.g. trucks, buses, taxis)	29469
	Personal and household services (includes dry cleaners)	861
	Manufacture of clothing apparel, except footwear	41148
Tetrachloroethylene	Printing, publishing and allied industries	284
	Construction	406
	Personal and household services (includes dry cleaners)	1957
	Personal and household services (includes dry cleaners)	196

* Given that smoking is now prohibited in Toronto workplaces, these estimates likely over-estimate the potential for benzene exposure in these two sectors at this time.

6.0 SUMMARIES FOR EACH CONTAMINANT

Asbestos

Asbestos has been used at one time or another in more than 5,000 products. It has been used in the production of textiles, electrical insulation, pharmaceutical and beverage filters, asbestos-cement pipes and sheets, clutch facings, break linings, asbestos paper, packaging, gaskets and pipe coverings. For many years, asbestos-based products were used in buildings because of their fire resistant qualities. Although asbestos has been eliminated from many of these products in Canada, its use continues unabated in a number of “developing” countries while its use in selected products continues in Canada as well.

Asbestos is a very hazardous material that has been the cause of thousands of deaths among Canadians occupationally exposed. It has also been linked to deaths and disease experienced by the families of workers occupationally exposed. Asbestos is known to produce lung cancer and mesothelioma (i.e. cancer of the membrane that surrounds the lungs and other internal organs) in people occupationally exposed. Gastro-intestinal cancer has been associated with exposure by both inhalation and ingestion of asbestos, although the risk is generally lower. Asbestos exposure also leads to asbestosis, a progressive disease of the lungs, cardiovascular disease, and depression of the immune system.

Inhalation is the most significant route of exposure for asbestos. The US EPA has estimated the cancer potency of asbestos by inhalation to be 0.23 per fibre/mL (fibres per milliliter of air) which corresponds to a one in a million cancer risk for a lifetime exposure to daily air levels of 0.000004 fibres/mL. (In this report, the exposure levels that correspond to a one in a million cancer risk are referred to as the “tolerable” exposure levels.)

While there is no Toronto-specific information on the levels of asbestos expected in outdoor air, asbestos has been reported in outdoor air in urban areas in North America at levels ranging from 0.000003 to 0.003 fibres/mL. While the lower end of this range hovers around the air levels deemed tolerable, the high end of the range is three orders of magnitude (i.e. 1,000 times) higher. Environmentally, additional levels of exposure can occur when asbestos-containing products are disturbed or removed without proper precautions in homes or public buildings or when structures containing asbestos are demolished without proper precautions.

Occupational exposure can occur in any industrial sector in which asbestos-containing products are manufactured, used or handled. It can also occur in situations in which old asbestos-containing products such as ceiling tiles or insulation are removed or disturbed without proper precautions. Within Toronto, worker exposure would be expected to occur during demolition, renovation or repair work done on structures in which asbestos-products have been used, where proper precautions are not taken.

With the CAREX model, it was estimated that approximately 7,000 Toronto workers may be potentially exposed to asbestos at levels greater than background. Most of these people are expected to work in the wholesale and retail trade and restaurants and hotels sector and the construction industry. While it is clear how workers in the construction industry may be exposed to asbestos, the numbers estimated for the wholesale and retail trade sector require further investigation, first to verify that it is a sector of concern for asbestos and secondly, to identify the sources and levels of exposure that may be encountered.

Benzene

Benzene is used extensively in industry as a volatile solvent and as an intermediate in the production of many chemicals. It is a natural component of petroleum and is added to gasoline as an octane-enhancer and as an anti-knock agent. Vehicle exhaust from gasoline operated vehicles is the most significant source of benzene in the environment. Emissions estimates produced by Environment Canada suggest that gasoline-powered vehicles were responsible for about 65% of the 11,500 tonnes of benzene emitted in Ontario's air each year in the mid 1990s. Other vehicles in the transportation sector (15%), the steel sector (8%), petroleum production and distribution (3%), residential wood-burning fireplaces (3%), and chemical production (3%) are important contributors of benzene in Ontario as well (CHEMinfo., 1997).

Benzene is a cancer initiator that has been clearly linked to acute myeloid leukemia (i.e. a cancer of the blood system). Long-term exposure to low levels of benzene has also been associated with other blood disorders, reproductive effects, and depression of the immune system. Benzene can enter the body by inhalation, ingestion and absorption through the skin.

The US EPA has estimated that the cancer potency for benzene by inhalation is 0.0000041 per $\mu\text{g}/\text{m}^3$ and by ingestion is 0.029 per mg/kg body weight/day. These estimates correspond to a one in a million cancer risk with lifetime environmental exposures to air levels of 0.24 $\mu\text{g}/\text{m}^3$ or ingestion of 0034 $\mu\text{g}/\text{kg}/\text{body}$ weight per day.

The levels of benzene in the outdoor air in Toronto range from 1.3 to 3.1 $\mu\text{g}/\text{m}^3$ with an average of 2.2 $\mu\text{g}/\text{m}^3$. The average level is one order of magnitude (i.e. 10 times) greater than the air levels deemed tolerable for lifetime environmental exposures. Airborne concentrations of benzene at the perimeter of gasoline service stations in five Canadian cities averaged 439 $\mu\text{g}/\text{m}^3$ in the summer and 1383 $\mu\text{g}/\text{m}^3$ in the winter of 1986 (Canada, 1993c). These air levels suggest that actions to reduce emissions from gas stations could be a municipal priority.

Outdoor air is the main source of exposure to benzene for the general population in Canada, contributing 1.3 to 3.0 $\mu\text{g}/\text{kg}/\text{day}$ to the estimated daily intake. Vehicle-related activities contribute an additional 0.7 to 0.9 $\mu\text{g}/\text{kg}/\text{day}$ and indoor air contributes another 0.05 to 0.6 $\mu\text{g}/\text{kg}/\text{day}$ to the estimated daily intake.

With the CAREX model, it was estimated that approximately 15,000 Toronto workers are potentially exposed to benzene at levels greater than background in two sectors: the wholesale, retail trade, restaurants and hotels sector (about 6,000) and in the personal and household services sector (about 8,000). It is likely that second-hand cigarette smoke was the source of benzene exposure for many of the workers in these two sectors. Given that Toronto's new smoking by-law prohibits smoking in most establishments in Toronto, it is expected that these numbers represent over-estimates of the number of workers potentially exposed to benzene in Toronto.

Occupational exposure data published by IARC, the National Toxicology Program (NTP) and ATSDR (American Toxic Substances and Disease Registry) indicate that people can be exposed to substantial levels of benzene in a number of different industrial sectors including car repair, rubber manufacturing, petroleum refining, paint manufacturing and shoe production. These data indicate that even among those workers whose occupational exposures are relatively low, such as car mechanics (140 $\mu\text{g}/\text{m}^3$), tanker truck drivers (680 $\mu\text{g}/\text{m}^3$), and tanker truck loaders (1,400 to 6,100 $\mu\text{g}/\text{m}^3$), exposures are quite high when

compared to those experienced environmentally. The exposure of the car mechanic for example, is three orders of magnitude (i.e. 1,000 times) greater than the air levels deemed tolerable for a lifetime environmental exposure.

1, 3-Butadiene

1, 3-Butadiene is a combustion by-product that results from natural processes and human activities. It is also an industrial chemical used in the production of polymers including polybutadiene, styrene-butadiene rubbers, latex, and nitrile-butadiene rubbers. Gasoline and diesel powered vehicles are significant sources of 1, 3-butadiene emitted to the environment as is fuel combustion for home heating and industrial activities (Canada, 2000a).

1, 3-Butadiene is a human carcinogen that has been linked to cancers of the blood and lymph systems. It has also been linked to disorders of the heart, blood and lungs, and to reproductive and developmental effects. Inhalation is the predominant route of exposure. The US EPA and Health Canada have recently revised their cancer potency estimates for 1, 3-butadiene and they are quite similar. The US EPA estimates that 1, 3-butadiene has a cancer potency of 0.0000063 per $\mu\text{g}/\text{m}^3$ by inhalation, which corresponds to a one in a million cancer risk with lifetime environmental exposures to daily air levels of $0.16 \mu\text{g}/\text{m}^3$.

1, 3-Butadiene can be found in outdoor air at levels that average $0.32 \mu\text{g}/\text{m}^3$ (range 0.03 - $2.20 \mu\text{g}/\text{m}^3$) in Toronto. The average is slightly higher than the air levels deemed tolerable. Air levels are expected to be higher at gasoline filling stations and in enclosed structures such as parking garages and urban road tunnels (4 - $49 \mu\text{g}/\text{m}^3$ in parking garages). Estimated daily intakes for 1, 3-Butadiene suggest that indoor air can be the main source of environmental exposure with air levels ranging from 0.04 to $1.0 \mu\text{g}/\text{m}^3$ in homes of non-smokers and 0.3 to $19.2 \mu\text{g}/\text{m}^3$ in the homes of smokers.

With the CAREX model, it was estimated that 183 Toronto workers may be occupationally exposed to 1, 3-butadiene at levels greater than background. These numbers are surprising and likely represent an underestimate of the number of workers potentially exposed in Toronto. Given that 1, 3-butadiene would be expected in any workplace where cigarette smoking was allowed and in any workplace exposed to vehicle exhaust, the estimated number of workers should have been closer to those estimated for benzene and PAHs.

Occupational exposure data published by the NTP and ATSDR indicate the potential for workers to be exposed to substantial levels of 1, 3-butadiene in the rubber and other manufacturing plants, but do not provide any exposure estimates for workers in the transport, retail or personal services sectors.

Cadmium

Cadmium is present in the environment as a result of both natural processes and human activities. Cadmium is used in paints, pigments, plastics, batteries and in some metal alloys. Base metal smelting and refining operations are considered the most significant sources of cadmium from human activities; responsible for approximately 80% of the cadmium released into the air and water. Other sources include coal-fired electrical generation, space heating, vehicles, solid waste incineration and sewage sludge application (Canada, 1994a).

Cadmium is most clearly linked to lung cancer by inhalation. Other health effects, such as kidney disease, have been associated with exposure by ingestion as well as inhalation. The US EPA has estimated cadmium's cancer potency to be 0.0018 per $\mu\text{g}/\text{m}^3$ for exposure by inhalation. This estimate corresponds to one additional cancer per million people exposed for a lifetime to daily air levels of 0.00056 $\mu\text{g}/\text{m}^3$. The average outdoor air concentration of cadmium in southern Ontario has been reported to be 0.00042 $\mu\text{g}/\text{m}^3$.

This concentration hovers around the exposure level deemed "tolerable" for lifetime environmental exposures.

With the CAREX model, it has been estimated that approximately 2,500 Toronto workers may be exposed to cadmium at levels greater than background. These people work in a variety of industrial sectors with the largest numbers in the plastics production, non-metallic mineral products, non-ferrous metal basic, and other manufacturing sectors. Occupational exposure data reported by NTP and IARC indicate that exposure to cadmium can vary greatly depending on the industry and the job.

Chromium

Chromium is used in the metallurgical industry to produce stainless steel and alloys. In the chemical industry, it is used in the production of pigments. Smaller amounts of chromium are also used in leather tanning, textiles manufacturing, wood preservation, toners for copying machines, magnetic tapes, and as a catalyst. It is also released when fossil fuels such as coal, oil and gas are burned as fuels. Emissions estimates developed in the early 1990s indicate that about 84 tonnes of total chromium are released into the air in Canada each year, while about 27 tonnes are discharged into water, and another 5,000 tonnes to land (i.e. this includes landfill sites). Industrial processes are responsible for about 29% of the chromium emitted into the air, while fuel consumption in stationary sources and from the transportation sector is responsible for about 51% and 12% respectively. Electrical generating stations are the most significant contributors of chromium to air; in 1991, they were responsible for about 24% of all emissions to air in Canada (Canada, 1994b).

Chromium exists in three forms: metallic chromium, chromium (III) and chromium (VI). Metallic chromium and chromium (III) are not considered carcinogenic, while chromium (VI) is. Chromium (VI) has been most clearly linked to lung cancer by inhalation. High level occupational exposures have also been associated with nasal irritation, nosebleeds, holes in the nasal septum and other respiratory effects. Exposure to low levels of chromium of any form can induce allergic skin reactions in sensitive people. Chromium (VI) may also cause adverse effects on reproduction.

There is general agreement among regulators regarding the inhalation cancer potency of chromium (VI). The US EPA estimates a cancer potency of 0.012 per $\mu\text{g}/\text{m}^3$ by inhalation which corresponds to one additional cancer case per million exposed for a lifetime to daily air levels of 0.000083 $\mu\text{g}/\text{m}^3$.

Total chromium levels in outdoor air in Ontario range between 0.003 to 0.009 $\mu\text{g}/\text{m}^3$, which corresponds to chromium (VI) air levels of about 0.00009 $\mu\text{g}/\text{m}^3$ to 0.00072 $\mu\text{g}/\text{m}^3$. The high range is one order of magnitude (10 times) greater than the air levels deemed “tolerable”.

Food is the most important pathway of exposure for the general population. However, given that chromium (VI) is known to be carcinogenic by inhalation and not by ingestion, indoor and outdoor air would be of greatest concern for cancer. Cigarette smoking can significantly increase total daily intake of chromium.

With the CAREX model, it was estimated that approximately 6,000 Toronto workers may be exposed to chromium (VI) at levels greater than background. These workers are spread across many different industrial sectors. Occupational exposure levels reported by ATSDR suggest that workers can be exposed to substantial levels of chromium in operations that involve plating, alloy production, and stainless steel welding.

Dioxins and Furans

Polychlorinated dibenzo-p-dioxins (or dioxins) and dibenzofurans (furans) are not manufactured intentionally. They are by-products of many processes including incineration of municipal and medical wastes, production of chlorinated compounds, pulp and paper bleaching with chlorinated compounds, and some smelting operations. The primary pathway for exposure to dioxins is ingestion from food. 2,3,7,8-tetrachloro-dibenzo-p-dioxins (TCDD) is the most toxic member of the dioxin family and all other dioxins are expressed as toxic equivalents (TEQ) of TCDD.

Environment Canada has released a quantitative inventory of environmental releases of dioxins and furans in Canada for the year 1999. It indicates that in Ontario, medical waste incinerators are the most significant source of dioxins, accounting for 14 g TEQ per year, followed by hazardous waste incineration (7.4 g TEQ per year), iron sintering (6 g TEQ per year), waste burning in backyard barrels (5 g TEQ per year), steel manufacturing (3.66 g TEQ per year), diesel fuel combustion in vehicles (3.11 g TEQ per year), base metal smelting (2.9 g TEQ per year), municipal waste incineration (2.15 g TEQ per year), residential wood burning (0.84 g TEQ per year) and electrical power generation (0.69 g TEQ per year). Within the City of Toronto’s limits, diesel fuel combustion, residential wood burning, and to a lesser extent, medical waste incineration, are expected to be important emission sources of dioxins (Environment Canada, 2000).

Dioxins and related compounds (including dibenzofurans and coplanar PCBs) are possible inducers of a wide spectrum of responses in humans and animals including cancer at multiple sites, a severe acne-like condition, reproductive and developmental effects, suppression of the immune system, and hormonal disruption.

Although dioxins and related compounds are not cancer initiators, they are considered potent cancer promoters. Estimation of the cancer potency for dioxins is a controversial issue. The US EPA, which treats all carcinogens as non-threshold contaminants, has estimated a cancer potency of 0.001 per picogram (pg) TEQ/kg/day. With this estimate, it is assumed that daily exposure to 0.001 pg TEQ/kg/day will result in one additional cancer per million people exposed. The US EPA cancer potency estimates differs significantly from the estimates developed by WHO and Health Canada of 10 per pg TEQ/kg/day.

(Note that 1 gram is equivalent to 1,000,000,000,000 picograms.) The WHO has established a lower tolerable daily intake level for dioxins of 1-4 per pg TEQ/kg/day on the basis of their reproductive and developmental effects. While the controversy surrounding the cancer potency of dioxins is extremely important to standard setting processes, for the purposes of this report, it is sufficient to say that dioxins are contaminants recognized as human carcinogens by IARC, the US EPA and Health Canada.

Food is the major source of exposure to dioxins and furans because they bioaccumulate in the food chain. Age-specific estimates of average intakes of dioxins and furans for Great Lakes basin residents range from 1.20 pg TEQ/kg/day in adults 20 years and older, to 4.25 pg TEQ/kg/day in adults who eat sports fish, to 12.56 pg TEQ/kg/day in non-breast fed infants to 57.05 pg TEQ/kg/day in breast-fed infants.² While the interpretation of these intake levels varies depending on which cancer potency estimate is used, they indicate that people are being exposed to levels within and above the range at which adverse health effects may be expected. The potentially high intake levels in breast-fed infants is a concern. These levels should not be used to discourage women from breast-feeding because of its many nutritional, immunological and psychological benefits. However, they should motivate all levels of government to take action to eliminate the release of dioxins from all human activities wherever possible.

It was not possible to estimate the number of workers exposed to dioxins in the workplace using the CAREX model because the database does not include dioxins. Nor were any occupational exposure data for dioxins found in documents published by NTP, IARC or ATDSR.

²The estimated intake levels for breast-fed infants are higher than for non-breast-fed infants because dioxins, which are fat soluble and which accumulate in the food chain, are expected to be present in greater quantities in the breast milk of humans than in the milk of cows (i.e. that are lower on the food chain) or in soya products (i.e. that are at the bottom of the food chain).

Formaldehyde

Formaldehyde enters the environment from natural processes such as forest fires and from human activities such as the combustion of fuel in vehicles and industrial applications. Formaldehyde is used in the production of resins, plastics and as an intermediate in the production of other chemicals. Formaldehyde resins are used as adhesives for particleboard, fibreboard, plywood, and mouldings. Formaldehyde can also be used for the treatment of paper and textiles, in surface coatings, foam insulation, and medicines for humans and animals.

It has been estimated that approximately 12,700 tonnes of formaldehyde were released directly into the Canadian environment in 1997. Motor vehicle exhaust is by far the most significant source of emissions, responsible for about 88% of those emissions (Canada, 2000b). People can also be exposed to formaldehyde that “off-gases” from materials and products used in indoor environments.

Formaldehyde is considered to be a weak initiator of cancer and a strong promoter of cancer. It is a highly reactive substance that can be irritating to the nose, eyes, skin and lungs at fairly low levels of exposure. Formaldehyde is considered a probable human carcinogen. The US EPA has recently proposed a cancer potency estimate of 0.00000028 per $\mu\text{g}/\text{m}^3$, which corresponds to a one in a million cancer risk with a lifetime exposures to daily air levels of $3.6 \mu\text{g}/\text{m}^3$.

Formaldehyde levels measured in outdoor air in Canada between 1989 and 1995 average about $3.3 \mu\text{g}/\text{m}^3$ while the indoor air levels in residential settings were found to be about $36 \mu\text{g}/\text{m}^3$. The outdoor air levels that are common in Canada hover around the air level deemed tolerable for lifetime environmental exposures, while indoor air levels can be an order of magnitude (ie.10 times) higher.

With the CAREX model, it was estimated that approximately 8,000 Toronto workers are potentially exposed to formaldehyde at levels above background. Occupational exposure data reported by IARC indicate that workers can be exposed to formaldehyde in a number of industrial operations including textile (0.68 ppm) and paper production, hospitals (0.7 ppm), garment (0.9-2.7 ppm) and furniture (0.4 to 5.4 ppm) manufacturing. These exposure levels, which when converted to $\mu\text{g}/\text{m}^3$ range from a low of $492 \mu\text{g}/\text{m}^3$ to a high of $6,642 \mu\text{g}/\text{m}^3$, are two to three orders of magnitude (i.e. 100 to 1,000 times) higher than the air levels deemed tolerable for lifetime environmental exposures.

Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs are present in the environment as complex mixtures that are difficult to measure and identify. They are produced whenever organic materials such as wood, paper, coal, oil or gasoline are burned as fuel so they are ubiquitous in the environment of industrialized societies. In nature, forest fires are the most significant source of PAHs, while gasoline and diesel-operated vehicles, residential fireplaces and industrial combustion are the human sources that contribute the most to emissions (Canada, 1994c).

Some PAH-rich mixtures are carcinogenic to both humans and animals. Some individual PAHs are carcinogenic in animals, and some are not. Some PAHs are genotoxic while others are not. Other health effects include suppression of the immune system, adverse effects on the female and male reproductive systems, and impairment of fetal development. The doses required to induce developmental effects are generally similar or somewhat higher than those required for a carcinogenic response.

There are generally two approaches used to estimate the cancer potency of a PAH-rich mixture. One approach involves summing up the risk from exposure to individual PAHs in the mixture. This is the approach that has been used by Health Canada and the US EPA. This approach can underestimate the cancer risk because it considers only about a dozen PAHs when a typical mixture contains hundreds of PAHs. Another approach, which has been adopted by the WHO, is to assess PAHs as a whole. The Ontario Ministry of the Environment (MOE), which has thoroughly evaluated the two approaches, recommends that PAH-rich mixtures be evaluated on a whole mixture basis. It has recommended that cancer potency for PAH-rich mixtures be expressed in terms of benzo[a]pyrene (B[a]P), the most toxic member of the PAH family of compounds, and recommends a cancer potency estimate of 0.023 per $\mu\text{g B[a]P/m}^3$ for inhalation, 2.9 per mg/kg/day for ingestion, and 95 per mg/kg/day for dermal exposures. These values correspond to a lifetime cancer risk of one in a million when individuals are exposed to a PAH-rich mixture that contains $0.000043 \mu\text{g B[a]P/m}^3$ by inhalation, or yields an intake of $0.00034 \mu\text{g B[a]P/kg/day}$ by ingestion or $0.00001 \mu\text{g B[a]P/kg/day}$ by dermal absorption. (Note that 1 mg is equal to 1000 μg).

The average concentration of PAHs in outdoor air in Toronto is approximately $0.0003 \mu\text{g/m}^3 \text{ B[a]P}$, an order of magnitude (i.e. 10 times) greater than the exposure level deemed tolerable. In general, due to winter heating, outdoor air levels of PAHs are about ten times higher in the winter (i.e. $0.00036 \mu\text{g B[a]P/m}^3$) than in the summer months (i.e. $0.000014 \mu\text{g B[a]P/m}^3$). Because people spend more time indoors, the indoor air contributes more to the total daily intake even though exposure levels tend to be about one half of those outside. Indoor exposure is further increased in situations where the residents supplement home heating with a fireplace or where cigarette smoking takes place in the home. Although food is the major source of exposure to B[a]P, since B[a]P is a more potent carcinogen when inhaled than ingested, the risk of stomach cancer from oral intake may not be higher than the risk of lung cancer due to inhalation exposure.

With the CAREX model, it was estimated that approximately 33,500 workers in Toronto may be exposed to PAHs at levels greater than background. About 88% of these people work in the land transport sector (eg. trucking). Exposure levels reported in IARC and ATSDR publications indicate that workers can be exposed to substantial levels of PAHs in the production of aluminum, near coke ovens, in bitumen processing operations and in roofing operations.

Tetrachloroethylene

Tetrachloroethylene is used primarily for dry cleaning and textile processing. It is also used as a chemical intermediate and for metal degreasing. It can be found in a number of different products including adhesives, aerosols, paints, printing inks, glues, sealants, polishes, lubricants, paint removers, rug and upholstery cleaners, and stain, spot and rust removers. In Toronto, it is expected that dry cleaning and textile processing are important sources of environmental emissions.

While tetrachloroethylene is recognized as an animal carcinogen by IARC, the US EPA and Health Canada, there is no consensus between the three agencies on its potential to produce cancer in humans. IARC has classified it as probably carcinogenic to humans, the US EPA has classified it as a probably or possibly carcinogenic to humans, and Health Canada has revised its classification downwards to “unlikely to be carcinogenic to humans”. WHO has established a tolerable daily concentration of 250 $\mu\text{g}/\text{m}^3$ for inhalation exposure to tetrachloroethylene, while the US EPA has developed a reference dose of 10 $\mu\text{g}/\text{kg}/\text{day}$ for exposure by ingestion (i.e. exposure levels deemed “tolerable”). Until there is greater agreement among three agencies, tetrachloroethylene will be treated by Toronto Public Health as a contaminant that can produce cancer in humans. Tetrachloroethylene has also been associated with other non-cancer health effects including neurological and kidney effects in people occupationally exposed and liver effects in animals exposed experimentally.

Tetrachloroethylene levels in the outdoor air in Toronto range from 2 to 5 $\mu\text{g}/\text{m}^3$ while the average indoor air levels are about 5.1 $\mu\text{g}/\text{m}^3$. These levels are well below those deemed “tolerable” for lifetime environmental exposures. The use of household products that contain tetrachloroethylene are the likely sources of tetrachloroethylene in indoor environments.

With the CAREX model, it was estimated that approximately 45,000 in Toronto may be occupationally exposed to tetrachloroethylene at levels above background. Most of those work in the manufacture of clothing apparel sector while another 4% work in the personal and household services sector, probably in dry-cleaning establishments. Published exposure data indicate that workers can be exposed to high levels of tetrachloroethylene when involved in degreasing, dry-cleaning, printing, spray painting, film processing, and electroplating. For example, workers operating dry-cleaning machinery can be exposed to air levels (146,000 $\mu\text{g}/\text{m}^3$) that are almost three orders of magnitude (i.e. 1,000 times) higher than those deemed tolerable for lifetime environmental exposures.

Trichloroethylene

Trichloroethylene is used primarily as a solvent for vapour degreasing and cold cleaning metal parts in industry. To a lesser extent, it is used in dry-cleaning operations, paints, paint removers and various household products such as adhesives, rug-cleaning fluids, and spot removers. With the phasing out of 1,1,1-trichloroethane as an ozone depleting substance, the use of trichloroethylene is expected to increase in industrial applications (Canada, 1993a). Degreasing operations present the greatest potential for occupational exposure in the workplace and are the largest source of emissions to the general environment.

Trichloroethylene has been classified as a probable human carcinogen by both IARC and CEPA, and until recently, by the US EPA. (The US EPA recently withdrew this classification upon re-examination of the data.) Trichloroethylene exposure has been associated with other health effects besides cancer, including depression of the central nervous system, liver and kidney damage, and developmental effects in animals exposed experimentally.

Health Canada has developed a cancer potency estimate of 0.00000061 per $\mu\text{g}/\text{m}^3$ by inhalation and 0.0001 per mg/kg/day by ingestion for trichloroethylene. These values correspond to a lifetime cancer risk of one in a million when individuals are exposed to daily air levels of $1.6 \mu\text{g}/\text{m}^3$ or by ingestion to 0.0067 mg/kg/day.

In Toronto, outdoor air levels of trichloroethylene range from 0.32 to $2.8 \mu\text{g}/\text{m}^3$, while indoor air levels average about $1.4 \mu\text{g}/\text{m}^3$. These exposure levels hover around the levels deemed tolerable for lifetime environmental exposures. As a rule, indoor air is expected to be the major source of exposure to trichloroethylene for Canadians in the general population, while outdoor air, drinking water and food are expected to make only minor contributions

Using the CAREX model, it was estimated that approximately 500 people in Toronto could be occupationally exposed to trichloroethylene at levels that exceed background levels. Published exposure data indicate that occupational exposure to trichloroethylene can be quite high when it is being used as a degreasing agent (i.e. 4,000 to $43,000 \mu\text{g}/\text{m}^3$). These occupational exposure levels, while encountered for less time than environmental exposures, are three to five orders of magnitude (i.e. 1,000 to 10,000 times) higher than those deemed tolerable for lifetime environmental exposures.

7.0 CONCLUSIONS

The assessment conducted by ToxProbe has demonstrated that there is insufficient data available on environmental emissions of toxic substances from the sources that contribute most to Toronto's airshed (i.e. mobile and area sources and small commercial and industrial point sources).

It has also shown that Toronto residents can be exposed to low levels of these ten contaminants from a number of different environmental media. Inhalation of pollutants present in indoor and outdoor air was found to be the most important route of exposure for most of the ten carcinogens.

Nine of the ten carcinogens are present in outdoor air at levels that closely approach or exceed the air levels deemed "tolerable" by regulatory agencies. Two of the carcinogens – PAHs and benzene – are present in outdoor air at levels that are an order of magnitude (i.e. 10 times) higher than the air levels that correspond to a one in a million cancer risk (i.e. air levels deemed tolerable), and should be given priority for action by the City. Another two – chromium (VI) and asbestos – may be present in outdoor air at levels that exceed those deemed tolerable and should be targeted for further investigation.

At least one of the carcinogens – formaldehyde – can be detected in indoor air at levels that exceed the air levels deemed tolerable. For several others, indoor air is the medium of greatest exposure. Given the scarcity of data on indoor exposure levels and the potential for exposure from this medium, it is recommended that Toronto Public Health consider indoor air quality as a research priority in the next calendar year.

Intake levels estimated for dioxins indicate that exposure by ingestion from food products hover around the intake levels at which adverse effects may be expected. Therefore, it is recommended that the City give high priority to actions that support their virtual elimination.

The technical report has documented that there is no systematically collected publicly available information on current occupational exposure levels to contaminants in Ontario workplaces. Modeling has estimated that a significant number of workers may be exposed to polycyclic aromatic hydrocarbons (PAHs), tetrachloroethylene, formaldehyde, chromium (VI), asbestos and benzene in Toronto workplaces. A review of occupational exposure data published in the United States suggests that workers can be exposed to the selected carcinogens at levels that are hundreds or thousands of times higher than those air levels deemed tolerable for lifetime environmental exposures. The City should liaise with Cancer Care Ontario and the Ontario Ministries of Labour and Health and Long-term Care to ensure that occupational exposure to carcinogens gains greater attention and action.

8.0 RECOMMENDATIONS

It is recommended that the Board of Health:

- (1) request the Medical Officer of Health to:
 - (a) identify strategies that can be taken to reduce the release of the ten key carcinogens, giving priority to benzene, dioxins and PAHs;
 - (b) report back to the Board of Health on these strategies; and
 - (c) continue to liaise with the Ontario Ministries of Labour and Health and Long-term Care, and with Cancer Care Ontario, to ensure that Toronto workers are adequately protected from occupational exposures to carcinogens in Toronto workplaces;
- (2) request the Ontario Minister of the Environment and the Federal Ministers of Health and the Environment to:
 - (a) provide Toronto-specific data on the levels of asbestos and chromium (VI) in Toronto's outdoor air;
 - (b) expand their respective emission release inventories to include a greater percentage of small and medium-sized point sources; and
 - (c) move quickly to establish a health-protective air standard for polycyclic aromatic hydrocarbons (PAHs) as a whole;
- (3) request that Cancer Care Ontario and the Ontario Minister of Labour give priority to the investigation and assessment of occupational exposure to the following known and probable carcinogens in industrial sectors in Toronto:
 - (a) PAHs in the land transport sector;
 - (b) tetrachloroethylene in the clothing apparel manufacturing sector and personal and household services sector;
 - (c) formaldehyde in the furniture and fixtures manufacturing and clothing apparel manufacturing sectors;
 - (d) chromium (VI) in a number of manufacturing sectors;
 - (e) benzene in the personal and household services sector and wholesale, retail trade, restaurants and hotels sector; and
 - (f) asbestos in the wholesale, retail trade, restaurants and hotels sector;

- (4) encourage Cancer Care Ontario and the Ontario Ministers of Labour, of the Environment, and of Health and Long-term Care to prioritize all occupational and environmental carcinogens for further assessment and toxics reduction where appropriate;
- (5) send a copy of this report to the Ontario Ministers of Labour, the Environment, and Health and Long-Term Care, the Federal Ministers of Health and the Environment and to Cancer Care Ontario, the Toronto Cancer Prevention Coalition, and the Commissioners of Works and Emergency Services and Corporate Services; and
- (6) request that the appropriate city officials be authorized and directed to take the necessary action to give effect thereto.

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