

# Cancer

IN NEW BRUNSWICK COMMUNITIES

Investigating the environmental connection

**PART 1**

Moncton, Saint John and Fredericton (1991-2005)

Inka Milewski and Lily Liu



Conservation Council of New Brunswick  
Conseil de conservation *du* Nouveau-Brunswick

## **Cancer in New Brunswick Communities:**

*Investigating the environmental connection*

Part 1: Moncton, Saint John and Fredericton (1991-2005)

Inka Milewski and Lily Liu

## **Acknowledgments**

The authors of this report would like to thank the New Brunswick Department of Health and the New Brunswick Cancer Network, especially Bin Zhang and Mallory Fowler, for providing cancer counts from the NB Provincial Cancer Registry Data Base. We would like to thank Patricia Griffith and Jackson McGaw from Statistics Canada (Halifax) for providing current and historic community profile census data. We are also grateful to our colleagues at the Conservation Council and to Dr. Paula Tippet, former Medical Officer of Health for Health Region 2 (Saint John area) and long-time Conservation Council Board member, for their professional advice, review and support throughout this research project.

**This publication was translated by André Laurion.**

**Report Design: Imprint Communications**

Financial support for the Conservation Council's Health Watch Program and this research was provided by the EJLB Foundation and the Salamander Foundation.



Conservation Council of New Brunswick  
Conseil de conservation *du* Nouveau-Brunswick

**Conservation Council of New Brunswick Inc.**

180 St. John Street

Fredericton, NB E3B 4A9

Tel (506) 458-8747

Fax (506) 458-1047

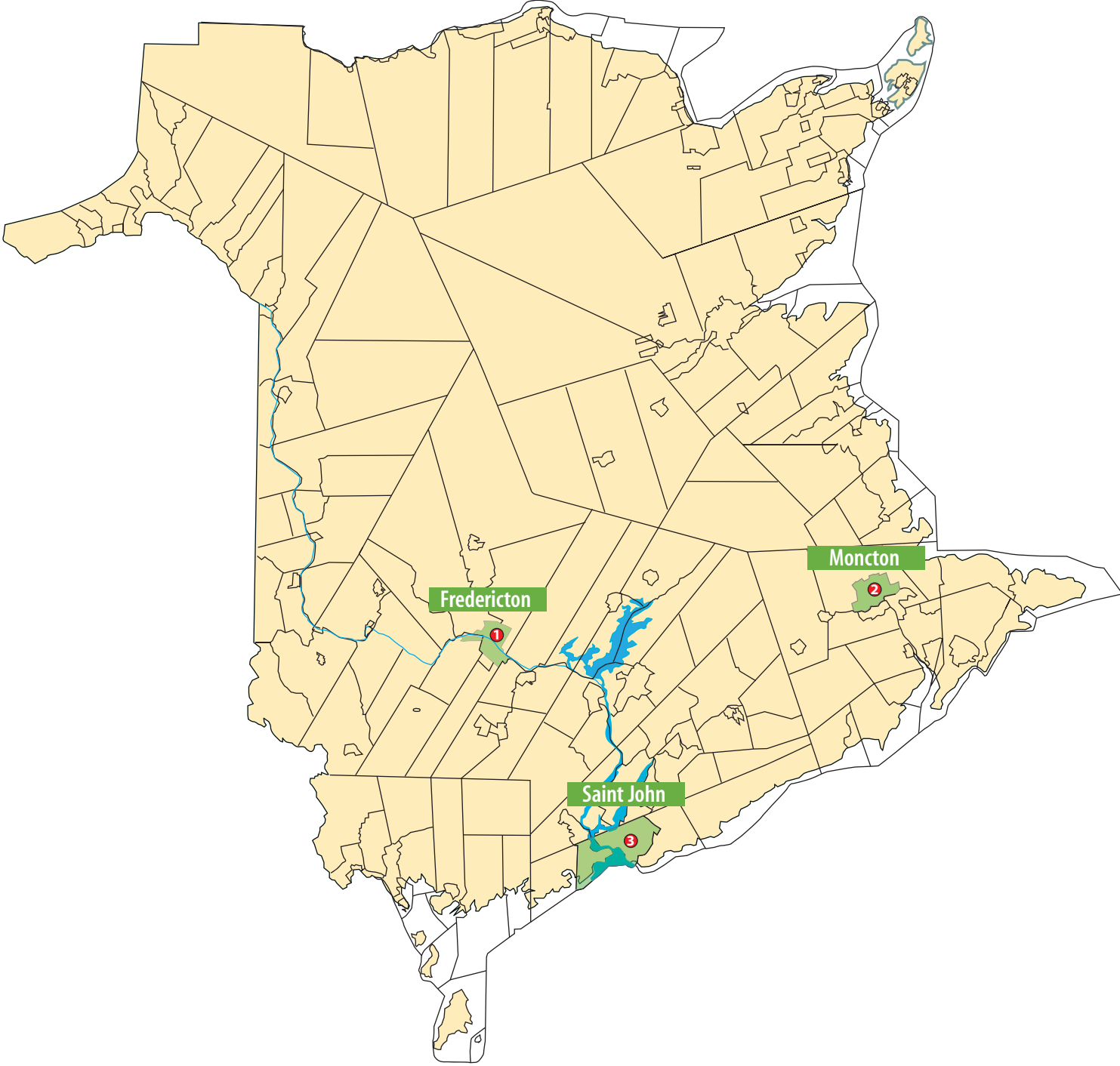
E-mail: [info@conservationcouncil.ca](mailto:info@conservationcouncil.ca)

[www.conservationcouncil.ca](http://www.conservationcouncil.ca)

# Contents

<b>Executive Summary</b>	<b>3</b>
<b>Introduction</b>	<b>5</b>
<b>Cancer Incidence Rates Among Three New Brunswick Communities</b>	<b>7</b>
How are cancers recorded and cancer rates calculated?	7
Lung Cancer Rates	8
Males	8
Females	9
Colorectal Cancer Rates	10
Males	10
Females	10
Breast Cancer Rates	11
Prostate Cancer Rates	12
<b>Cancer Risk Factors: What causes cancer?</b>	<b>14</b>
Lung Cancer Risk Factors	14
Smoking	15
Occupational Exposure	16
Air Pollution	19
Colorectal Cancer Risk Factors	21
Breast Cancer Risk Factors	22
Prostate Cancer Risk Factors	25
<b>Conclusions</b>	<b>27</b>
<b>Recommendations</b>	<b>28</b>
<b>Appendix A</b> – Cancer counts and age-standardized cancer incidence rates per 100,000 population for males and females in Saint John, Moncton, Fredericton, New Brunswick and Health Regions 1, 2 and 3 (1999-2003).	29
<b>Appendix B</b> – Age-standardized prostate, breast, colorectal and lung cancer incidence rates per 100,000 population for males and females in Saint John, Moncton, Fredericton, New Brunswick and Canada for 1991, 1996, 2001 and 2005.	30
<b>Appendix C</b> – Age-standardized colorectal cancer incidence rates per 100,000 population for males and females, by age groups, in Saint John, Moncton and Fredericton for 1991 and 2005.	31
<b>Appendix D</b> – Percent employment by major occupation categories for males and females in Saint John, Moncton and Fredericton for 1991, 1996, 2001 and 2006.	33
<b>References</b>	<b>34</b>

# Statistics Canada's Census Subdivisions for New Brunswick: Fredericton, Moncton and Saint John Study Areas



# Executive Summary

**M**ost health information in New Brunswick and across Canada is reported by large geographic areas. The New Brunswick Health Department reports cancer and other health statistics for seven health regions and for the entire province. Statistics reported over such large geographic areas do not provide information about health and disease conditions at the community level, where people live and work and where environmental exposures and industrial emissions are concentrated. Consequently, the identification, development and implementation of cancer and other disease prevention programs appropriate to the community does not occur.

**The Conservation Council of New Brunswick's *Health Watch* program undertook a study to examine cancer incidence rates in fourteen urban and rural areas in New Brunswick.**

The Conservation Council of New Brunswick's *Health Watch* program undertook a study to examine cancer incidence rates in fourteen urban and rural areas in New Brunswick. This report, the first of two reports, presents the incidence rates of four cancer types for males and females in the province's three largest cities: Saint John; Moncton and Fredericton. These rates were then compared to incidence rates

reported for health regions, the provincial and Canada between 1991-2005. Key risk factors for each cancer type were examined where data was available.

The major findings of this study are:

- reporting cancer incidence rates by large geographic areas obscures important information about the health of New Brunswickers at the community level;
- lung cancer incidence rates (1991-2005) among males and females in Saint John were consistently and significantly higher than rates reported for Saint John's health region (Health Region 2), Fredericton, Moncton, New Brunswick and Canada;
- In 2005, lung cancer incidence rates for males and females in Saint John were higher than provincial (49% and 78% respectively) and national (82% and 98% respectively) rates;
- occupational exposure and air pollution are key risk factors for lung cancer in Saint John;
- depending on the year and city, colorectal cancer incidence rates were slightly above or below provincial and national rates (1991-2005) and, in 2005, incidence rates were highest in Moncton for men and women;
- from 1991 to 2005, breast cancer incidence rates rose in Saint John and Moncton and declined in Fredericton and, in 2005, incidence rates in

Saint John and Moncton were significantly above provincial and national rates.

- from 1991 to 2005, prostate cancer incidence rates rose in Moncton and declined in Fredericton and, in 2005, incidence rates in Saint John, Moncton and the province were significantly higher than the national rate.
- from 1991 to 2005, the age group at which colorectal, breast and prostate cancer incidence rates peak has shifted downward to younger age categories in all three cities and cancer incidence rates are increasing among younger men and women; and
- community-level data on cancer risk factors (e.g. behaviour/lifestyle, occupation and environmental quality) are virtually non-existent.

**In 2003, the New Brunswick Department of Health established the *New Brunswick Cancer Network*. The Network does not monitor cancer incidence at the community level nor does it gather information on cancer risk factors at any geographic level.**

In 2003, the New Brunswick Department of Health established the New Brunswick Cancer Network. To date, the focus of the Network's activities has been on cancer treatment and monitoring. The Network does not monitor cancer incidence at the community level nor does it gather information on cancer risk factors at any geographic level.

Based on the results of this study, the Conservation Council of New Brunswick recommends that:

- the Minister of Health work with the Minister of Environment to improve air quality standards and eliminate the release of carcinogens from industrial sources in communities.

In addition, the Conservation Council recommends that the Minister of Health direct the New Brunswick Cancer Network to:

- undertake an appropriate epidemiological study to determine the cause of high lung cancer rates in Saint John;
- undertake detailed individual- and community-level epidemiological studies to determine why prostate cancer rates are high in Saint John and Moncton and why rates are rising among younger men in Saint John;
- undertake detailed individual- and community-level epidemiological studies to determine why breast cancer rates are high in Saint John and Moncton;
- begin public reporting of cancer rates at the community-level;
- expand cancer prevention messaging and programs to include occupational and environmental risk factors like exposure to pesticides, household and industrial chemicals and air pollution; and
- conduct an epidemiological study to examine the relationship between cancer incidence and occupations in the province.

# Introduction

**M**ost health information, including cancer incidence, is reported by large geographic areas. The Canadian Cancer Society and National Cancer Institute of Canada report cancer statistics by province and territory. The New Brunswick Health Department reports cancer and other health statistics by health regions. Health information reported at these larger geographic scales offer no insight into disease conditions at the community level, where people live and work and where environmental exposure and industrial emissions are concentrated.

**Health information reported at these larger geographic scales offer no insight into disease conditions at the community level, where people live and work and where environmental exposure and industrial emissions are concentrated.**

It is widely acknowledged that developing effective and efficient intervention policies to protect human health begins with gathering information at the appropriate geographic scale (e.g. individual, neighbourhood, community).<sup>1</sup> The first step in examining the connection between human health and pollution involves breaking down disease patterns into smaller geographic units. In the study of diseases this is referred to as *spatial epidemiology* which has been defined as “the description and analysis of geographic variation in diseases with respect to demographic, environmental, behavioural, socioeconomic, genetic and infectious risk factors.”<sup>2</sup> This approach has its roots in ecology where making observation at the right geographic scale (e.g. individual-level, population-level and community-level) matters greatly from a management and policy perspective.

The results of the 2005 Belledune Area Health Study underscored the need to gather health information at the appropriate geographic scale. The Belledune area as defined by the study included the villages of Jacquet River, Belledune, Pointe-Verte and Petit-Rocher in northern New Brunswick.<sup>3</sup> The village of

Belledune has a lead smelter which has been operating in the community since 1967 and a coal-fired power plant operating since 1993. Acid and fertilizer plants, a gypsum plant and battery recycling plant also operate in the village, some of which have closed in recent years.

The Belledune Area Study found that levels of known (arsenic and cadmium) and probable (lead) carcinogens released from industrial facilities were high enough in the environment to pose health risks (above provincial health guidelines) for residents for

**The results of the 2005 *Belledune Area Health Study* underscored the need to gather health information at the appropriate geographic scale.**

more than thirty years.<sup>4</sup> The study also found that rates of several cancers, as well as mortality and other disease rates, were higher in the Belledune area than in either of its health regions and higher

than provincial rates.<sup>5</sup> Had the cancer clusters been identified years earlier, appropriate epidemiological studies could have identified key risk factors in the area and appropriate community-level prevention programs and mitigation measures could have been implemented.

The Conservation Council of New Brunswick's (CCNB) *Health Watch* program began a two-year project in April 2007 to raise public awareness about the importance of environmental quality in human health and, through research, contribute to environmental health policy development in the province. As part of the Conservation Council's contribution to the province's policy development process for cancer prevention, *Health Watch* undertook a study to examine cancer incidence rates and their risk factors in fourteen urban and rural areas in New Brunswick. This report, the first of two reports, presents the incidence rates (1991-2005) of four cancer types in the province's three largest cities (Saint John, Moncton and Fredericton) and compares them to rates at the national, provincial and health region level. Where data were available, major risk factors for each cancer type were examined.

# Cancer Incidence Rates Among Three New Brunswick Communities

**C**ancer is the second leading cause of death among New Brunswick adults and children.<sup>6</sup> Between 1999-2003 lung, colorectal and prostate cancers accounted for 57% of all cancer incidences among New Brunswick male adults.<sup>7</sup> For females, lung, colorectal and breast cancers account for 55% of all cancers reported. According to the 2008 national Cancer Statistics Report, these cancers account for the majority of new cases in Canada.<sup>8</sup> Lung and colorectal cancer are the first and second leading cause of cancer death for Canadian males and females.<sup>9</sup>

Every five years the NB Department of Health reports on cancer rates for the province and in each health region. Their last report covered the period 1997-2001. The department also reports on the health status of New Brunswickers every five years. The most recent report covers the period 1999-2003 and includes data on cancer incidence rates by health region and province.

**Cancer is the second leading cause of death among New Brunswick adults and children.**

## How are cancers recorded and incidence rates calculated?

Each new case or incidence of cancer is identified by a pathologist using an international classification system. The gender, age, year of diagnosis, type of cancer and geographic location for every new case of cancer is recorded with the *New Brunswick Cancer Registry* and shared with the *National Cancer Registry*.

The geographic location of each cancer diagnosis is recorded using Statistics Canada's census subdivision (CSD) codes. It is recorded based on where an individual lives and not the location where the diagnosis or treatment occurred. For example, if a cancer diagnosis was made by a physician in Saint John but the patient lived in Rothesay (CSD 05 045) or Quispamsis (CSD 05 057), the cancer incidence or death would be recorded by the Rothesay or Quispamsis code and not the code for Saint John (CSD 01 006).

Cancer incidence rates are generally reported as age-standardized incidence rates (ASIR) per 100,000 population. Age standardization involves adjusting the population age structure (demographics) of a province, health region or community to the age structure of the Canadian population in 1991. Standardization allows populations with different age demographics to be more accurately compared.

This study calculated ASIR per 100,000 population for lung, colorectal, breast and prostate cancer for males and females in Saint John (CSD 01 006), Moncton (CSD 07 022) and Fredericton (CSD 10 032) for various years between 1991-2005. Calculations were based on cancer counts obtained from the NB Provincial Cancer Registry Database.

The mathematical formula used to calculate age-standardized incidence rates was the same formula used by the New Brunswick Department of Health,

the Canadian Cancer Society/National Cancer Institute of Canada and Statistics Canada to calculate provincial and national rates. Census data by age group were obtained from Statistics Canada. Census data from 2001 were used to calculate ASIR for 1999-2003. Statistics Canada community census data for 1991, 1996 and 2001 were used to calculate community ASIR for 1991, 1996 and 2001 respectively. ASIR for 2005 were calculated based on 2006 Statistics Canada census data.

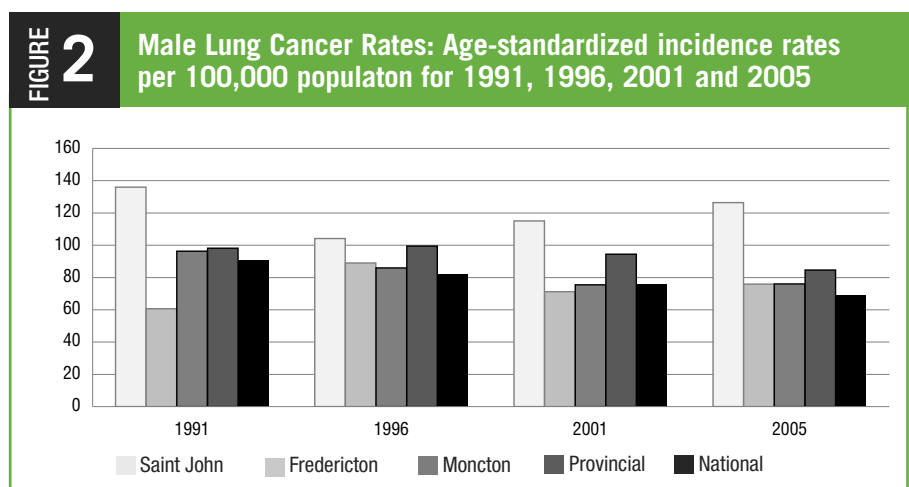
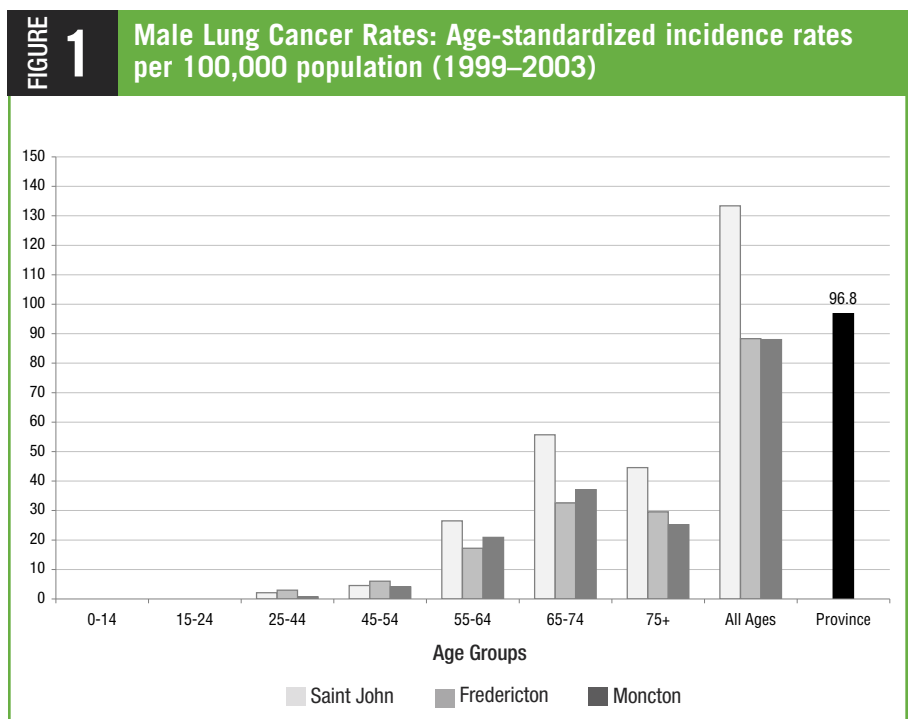
Between 1991 and 2005, lung cancer rates among males in Saint John decreased by 5.5% but rates were consistently and significantly higher than national and provincial rates (Figure 2; Appendix B). In 2005, the lung cancer rate among Saint John males was 82% higher than national rate and 49% above the provincial rate. Rates in Fredericton and Moncton were below the provincial rate between 1991 and 2005 (Appendix B).

## Lung Cancer Rates

### Males

The overall lung cancer rate among Saint John males between 1999-2003 was 51% higher than rates for males in Fredericton and Moncton and 38% higher than the lung cancer rate for New Brunswick males for the same time period (Figure 1; Appendix A). For Moncton and Fredericton, lung cancer rates were lower (9%) than the provincial average for males. Rates for all three cities were highest among males in the 65-74 age category.

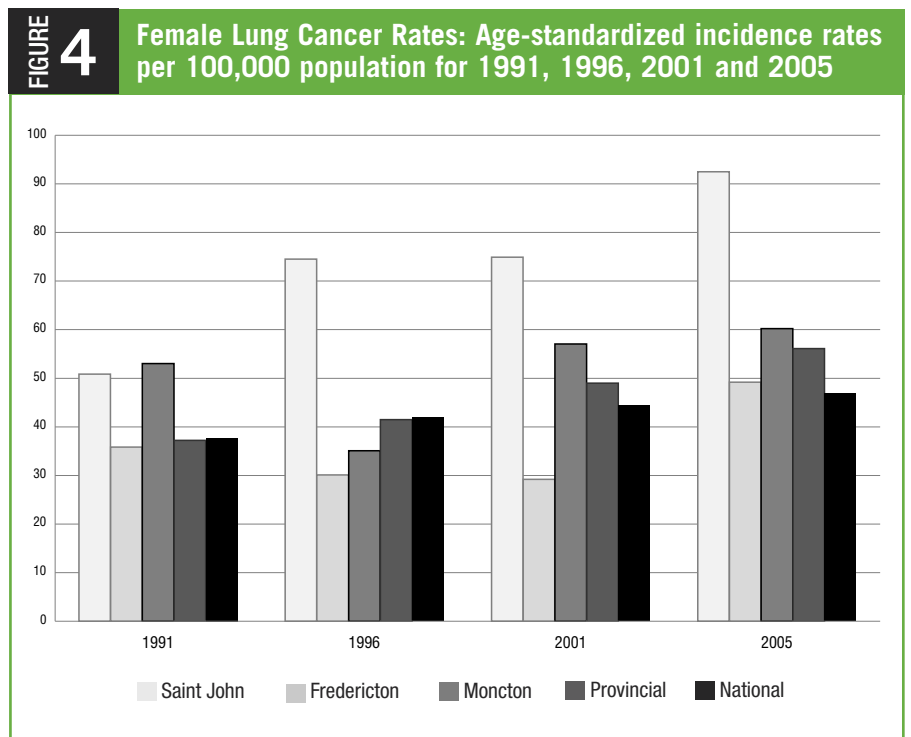
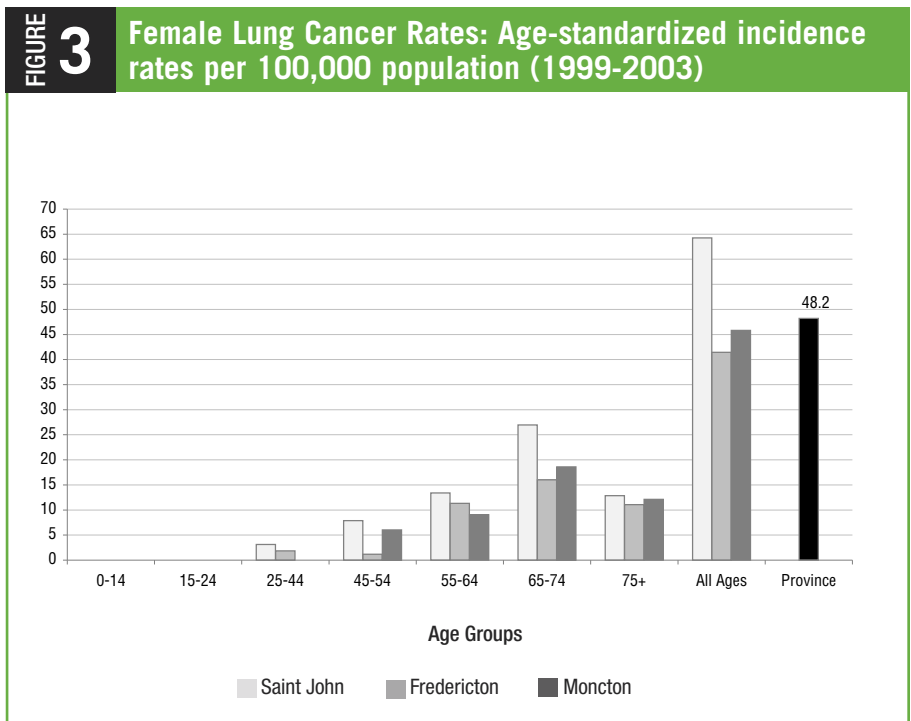
Saint John is located in Health Region 2 and the lung cancer rate for Saint John males was 23% higher than reported for Health Region 2 for 1999-2003 (Appendix A).<sup>10</sup> Moncton is located in Health Region 1 and the lung cancer rate for males was slightly higher than those for Health Region 1 (Appendix A). Fredericton is in Health Region 3 and the rate of lung cancer for males was 5% lower than those for the Health Region (Appendix A).



## Females

The overall lung cancer rate (1999-2003) among Saint John females was significantly higher than rates in Fredericton and Moncton (51% and 40% respectively) and 33% higher than the provincial rate (Figure 2; Appendix A). Lung cancer rates for Saint John females were higher than Fredericton and Moncton in all age categories (Figure 3). Lung cancer rates in Moncton and Fredericton were lower (5% and 14% respectively) than the provincial rate and at or below the rates in their respective Health Regions (Appendix A). Rates for all communities were highest among females in the 65-74 age category.

From 1991 to 2005, incidence rates increased in all three cities with Saint John experiencing the largest increase (82%). Rates in Saint John were also consistently and significantly higher than those for Moncton, Fredericton, New Brunswick and Canada in 1996, 2001 and 2005 (Figure 4; Appendix B). In 2005, the lung cancer rate for Saint John females was 98% higher than the national average and 78% above the provincial average. Moncton rates were significantly (28%) above the national rate in 2005 and rates in Fredericton were slightly above (5%) the national rate.



## Colorectal Cancer Rates

### Males

Overall, Fredericton had the highest rate of colorectal cancer for 1999-2003 and the rate was 16.7% higher than the provincial rate (Figure 5; Appendix A). For all cities, rates were highest in the 65-74 age category.

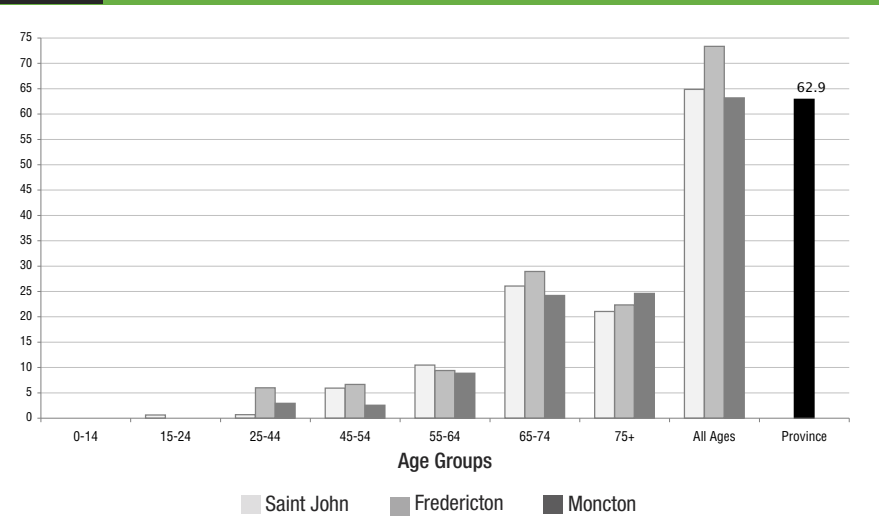
Males colorectal cancer rates in Fredericton were 18% higher than the rate reported for their health region (Health Region 3) (Appendix A).

Colorectal cancer rates rose in all three cities from 1991 to 2005, with the greatest increase occurring in Saint John (13%) followed by Moncton (7.5%) (Figure 6; Appendix B). Fredericton rates rose 1.5%. Between 1991 and 2005, colorectal rates increased in all age categories and the age at which rates peak appears to be shifting downward to younger ages (Appendix C, Figures A and B).

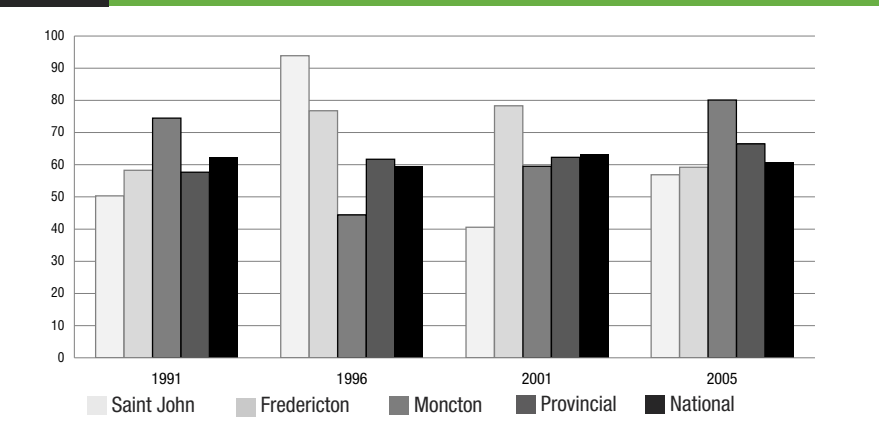
### Females

The overall colorectal cancer rate (1999-2003) was slightly higher in Moncton than in Fredericton and Saint John and only slightly above the provincial rate (Figure 7). Colorectal cancer rates among the three cities were slightly above those reported for their respective health regions (Appendix A).

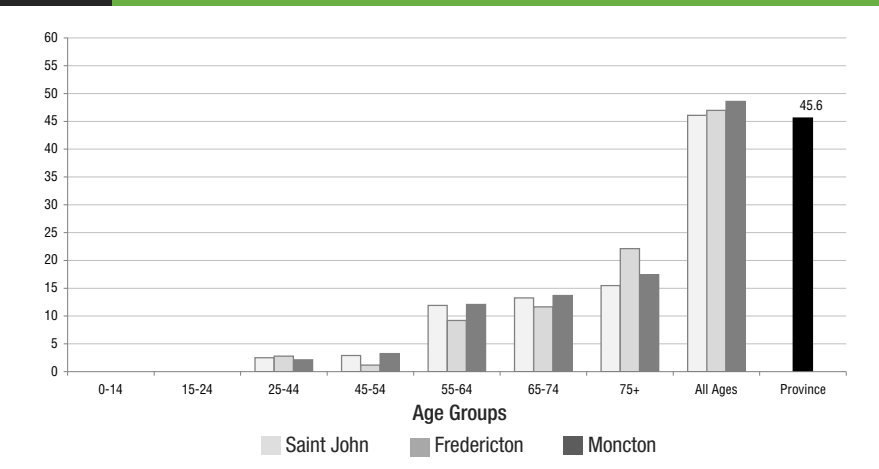
**FIGURE 5** Male Colorectal Cancer Rates: Age-standardized incidence rates per 100,000 population (1999 -2003)



**FIGURE 6** Male Colorectal Cancer Rates: Age-standardized incidence rates per 100,000 population for 1991, 1996, 2001 and 2005



**FIGURE 7** Female Colorectal Cancer Rates: Age-standardized incidence rate per 100,000 population (1999-2003)



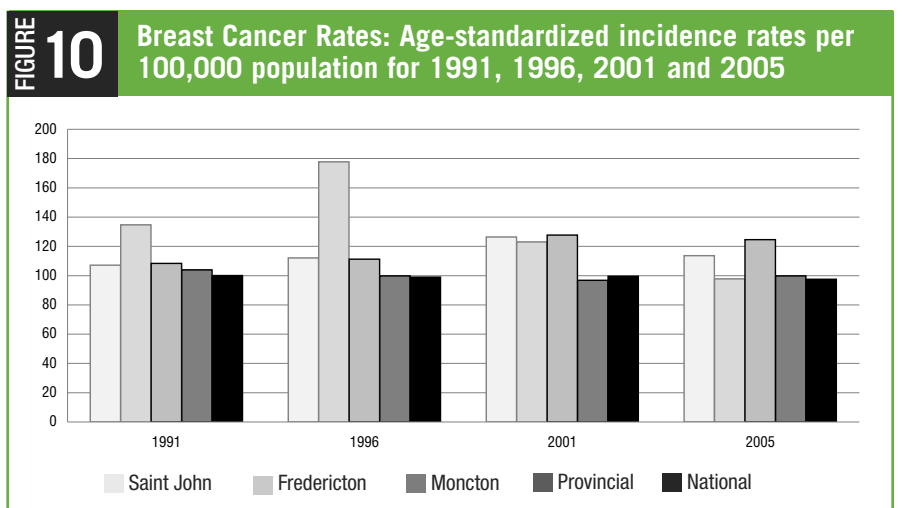
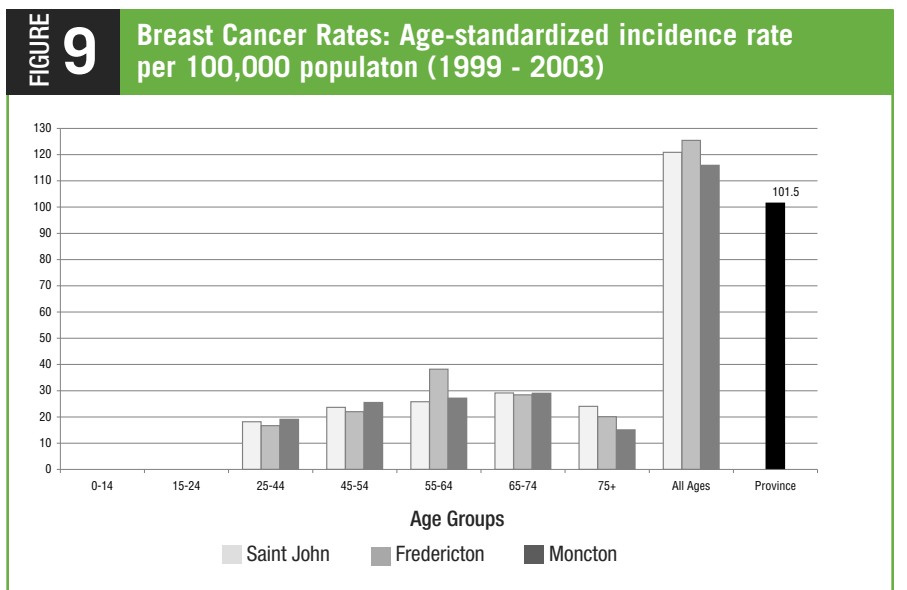
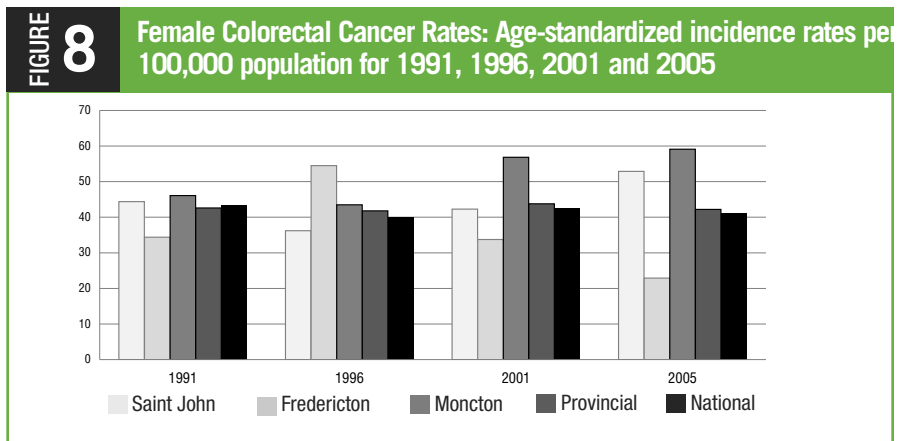
From 1991 to 2005, colorectal cancer rates in Saint John and Moncton rose 19% and 28% respectively and fell 33% in Fredericton (Figure 8; Appendix B). In 2005, the rate in Fredericton was significantly below provincial and national rates and Moncton and Saint John were significantly above national and provincial rates. Colorectal cancer rates have increased in all age categories from 1991 to 2005 and the age at which rates peak appears to be shifting downward to younger ages (Appendix C, Figures C and D).

## Breast Cancer Rates

Fredericton had the highest rate of breast cancer (1999-2003) among the three cities and all three cities had rates significantly higher than the provincial rate (Figure 9; Appendix A).

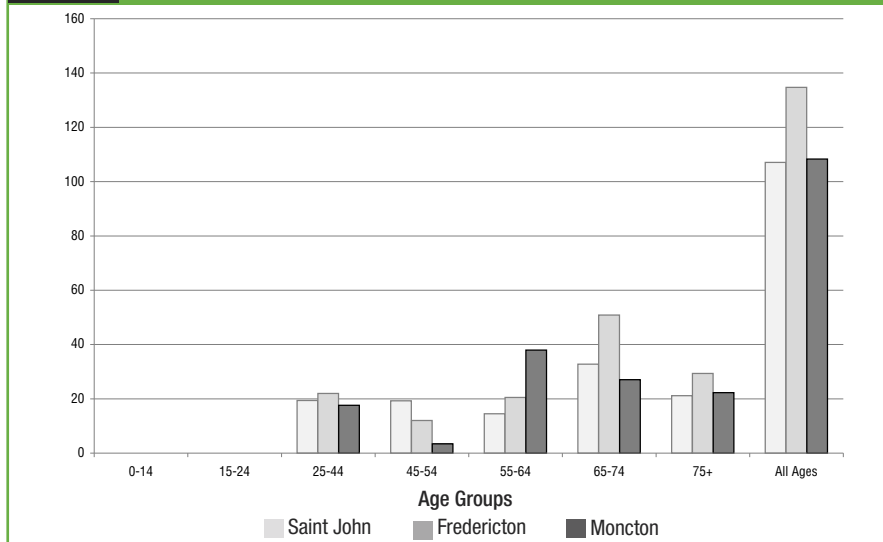
Breast cancer rates in all three cities were significantly higher than rates reported for their respective health regions (Appendix A).

From 1991 to 2005, breast cancer rates rose in Saint John and Moncton (6% and 15% respectively) while Fredericton rates fell significantly (27%) (Figure 10; Appendix B). Provincial and national rates also fell between 1991 and 2005. In 2005, breast cancer rates in Saint John and Moncton were significantly higher (17% and 28% respectively) than the provincial rate and 17% and 28% higher than the national rate (Figure 10; Appendix B).

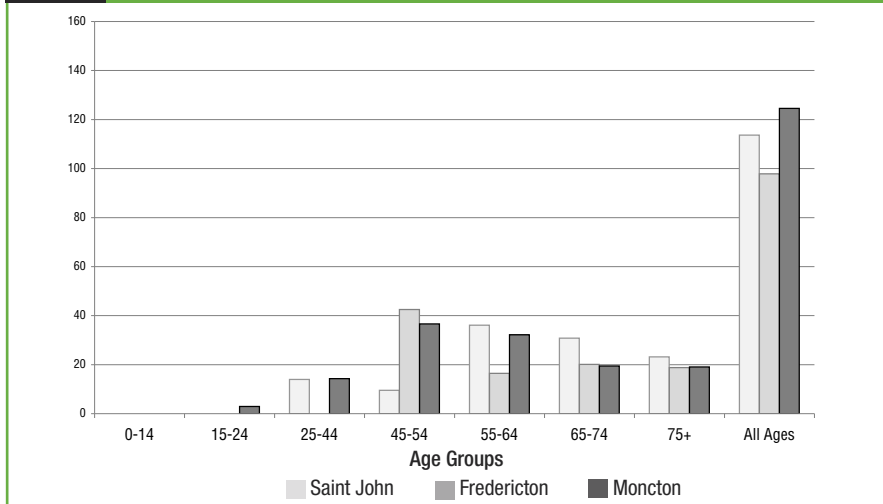


From 1991 to 2005, the age group at which breast cancer rates peak has shifted downward indicating women are developing cancer at a younger age (Figures 11 and 12).

**FIGURE 11** 1991 Breast Cancer Rates by Age Groups  
Age-standardized incidence rates per 100,000 population



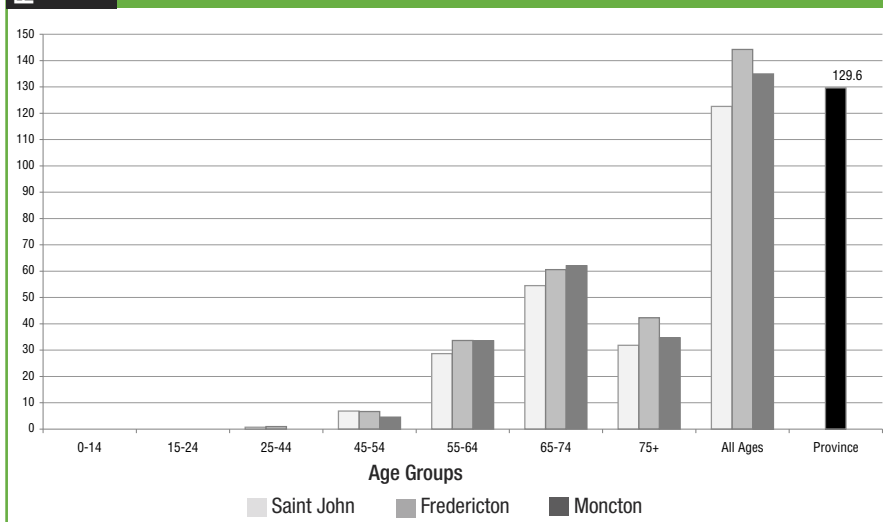
**FIGURE 12** 2005 Breast Cancer Rates by Age Groups  
Age-standardized incidence rates per 100,000 population



## Prostate Cancer Rates

Among the three cities, the prostate cancer rate (1999-2003) was highest in Fredericton. The rate was 11% above the provincial rate and 9.5% above the rate in Fredericton's health region (Figure 13; Appendix A). The rate in Saint John was below (5.4%) the provincial rate and slightly above (2.7%) the rate in Health Region 2. The rate in Moncton was above the provincial and Health Region rates (4.1 % and 2% respectively).

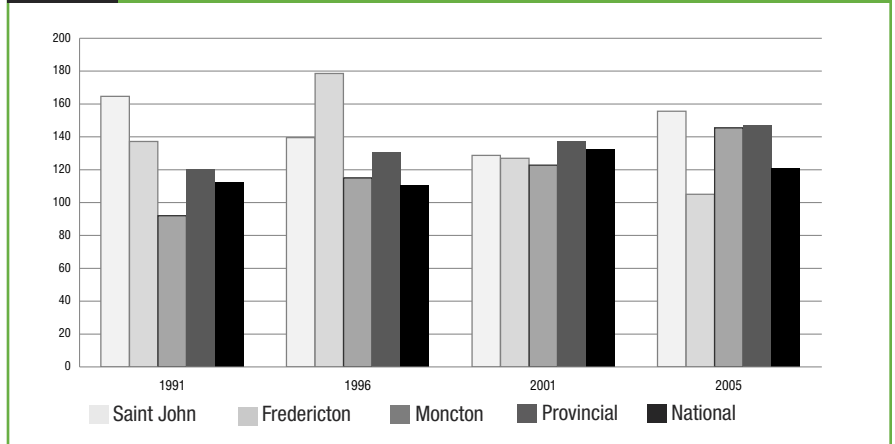
**FIGURE 13** Prostate Cancer Rates: Age-standardized incidence rate per 100,000 population (1999 -2003)



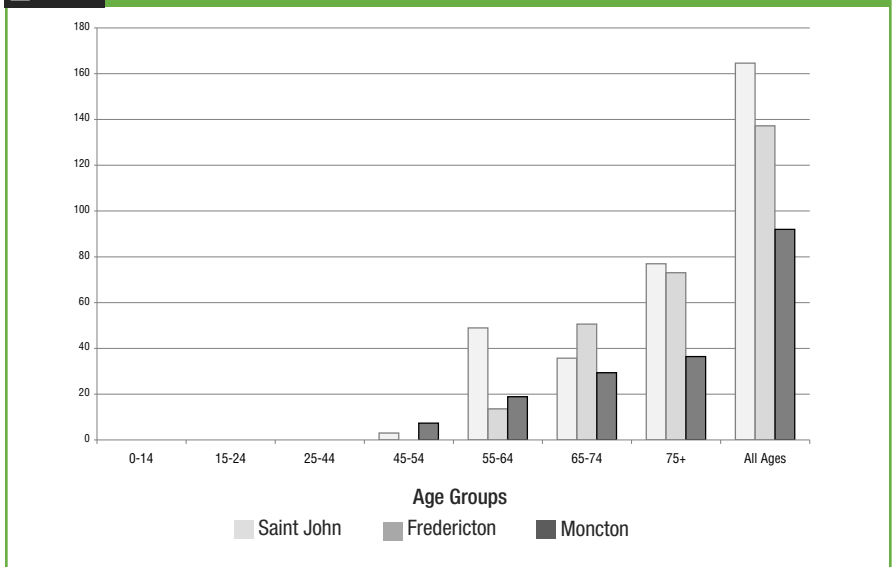
From 1991 to 2005, prostate cancer rates fell significantly (23%) in Fredericton and slightly (6%) in Saint John. (Figure 14; Appendix B). Rates have been steadily rising in Moncton and provincially. In 2005, rates in Saint John and Moncton were significantly higher than national rates (29% and 20% respectively) and the Fredericton rate was significantly below (13%) the national rate (Appendix B).

Prostate cancer rates from 1991 to 2005 also showed a downward trend in the age at which rates peaked. In 1991, the age at which rates were highest was 75+ years (Figure 15). In 2005, the peak age at diagnosis for prostate cancer among the three cities was the 65-74 age category except in Saint John where peak rates were shared with an even younger age category (55-64 years) (Figure 16).

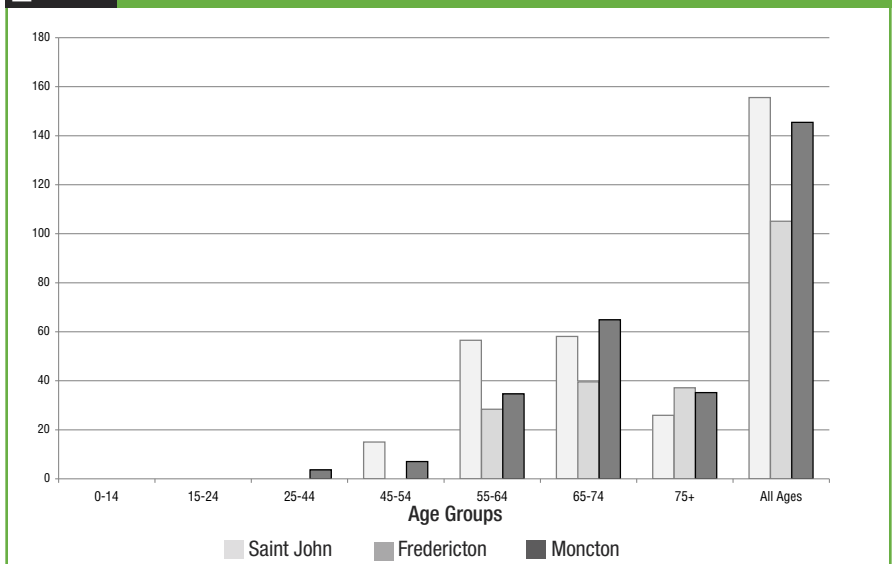
**FIGURE 14** Prostate Cancer Rates: Age-standardized incidence rates per 10,000 population for 1991, 1996, 2001 and 2005



**FIGURE 15** 1991 Prostate Cancer Rates by Age Groups Age-standardized incidence rates per 100,000 population



**FIGURE 16** 2005 Prostate Cancer Rates by Age Groups. Age-standardized incidence rates per 100,000 population



# Cancer Risk Factors: What causes cancer?

**C**ancer is a disorder in cell growth. It is generally recognized as a multistage disease involving the accumulation of a critical number of mutations within a stem cell (a cell that can undergo division to become another cell type).<sup>11</sup> When cells lose the ability to replicate without errors or the DNA repair mechanism does not work properly, abnormal growth occurs. Cell replication is under the control of genes and there is a consensus among cancer researchers that most cancers are not inherited but acquired over the course of a lifetime. Depending on the cancer type, only 2-10% of cancers are the result of a mutation in, or the operation of, a particular gene.<sup>12</sup>

**Depending on the cancer type, only 2-10% of cancers are the result of a mutation in, or the operation of, a particular gene.**

Recent studies on identical twins have demonstrated that environmental rather than genetic factors predominate in the causes of cancers.<sup>13</sup> Since so few cancers are linked to genetic mutations, the consensus among cancer experts is that the majority of cancers are preventable.

The traditional definition of 'environment' among healthcare professionals and policy makers covers a wide range of factors such as lifestyle (smoking, alcohol consumption, physical inactivity, obesity and overweight), pollution, viruses, bacteria, sunlight, medicine (e.g. estrogen replacement therapy) and medical procedures (e.g. chemotherapy/radiation). This list of factors are a mixture of initiating or

causative factors and risk factors, a distinction that is important to cancer experts.<sup>14</sup>

Cancer initiating agents are those physical (e.g. ionizing radiation and particles like asbestos and silica), chemicals (e.g. arsenic, benzene, chlorinated compounds) and biological (e.g. viruses) substances that cause mutations. Risk factors, on the other hand, refer to activities such as smoking and alcohol consumption, air pollution or occupations that enhance or promote exposure to cancer-causing agents. The proportion of cancers deaths that have been attributed to so-called classical lifestyle factors (smoking, alcohol consumption, obesity/overweight, diet) range from 25-45% with smoking being the most significant lifestyle factor.<sup>15</sup>

## Lung Cancer Risk Factors

### Smoking

Smoking is a leading risk factor for lung cancer followed by occupational exposure and outdoor (and increasingly indoor) air pollution. In high income countries, smoking accounts for 25-35% of the overall cancer incidence in a population.<sup>16</sup> The question being examined by many researchers is what factors contribute to the approximately 65-75% of cancers not related to smoking.

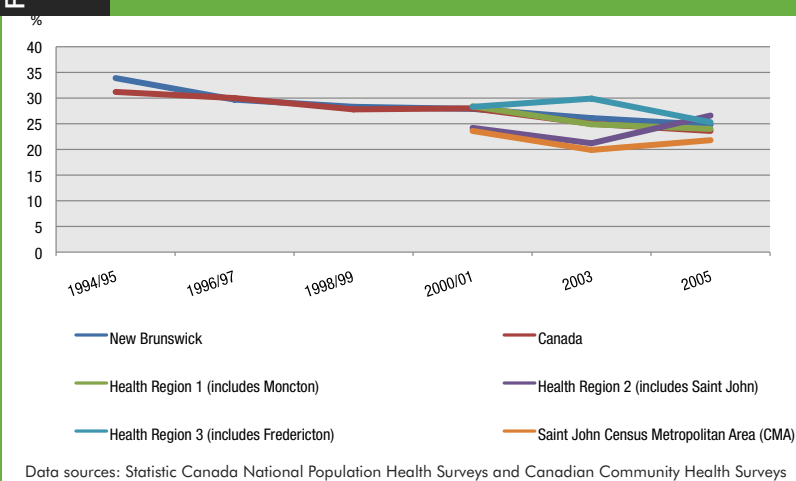
Worldwide and across Canada, smoking rates have declined dramatically since the mid-1960s. In 1966, 45% of Canadians (56% males and 34% females) were smokers.<sup>17</sup> By 2005, the rate had dropped by more than 40% to 23.6% males and 19.8% females.<sup>18</sup>

Smoking rates among New Brunswickers have also dropped significantly.<sup>19</sup> Rates among New Brunswick males have not been significantly different from national rates since the mid 1990s (Figure 17). For females, smoking rates briefly rose above the national rate in the mid-1990s, but by 2000/2001 smoking rates had dropped to meet the national rates for females (Figure 18).

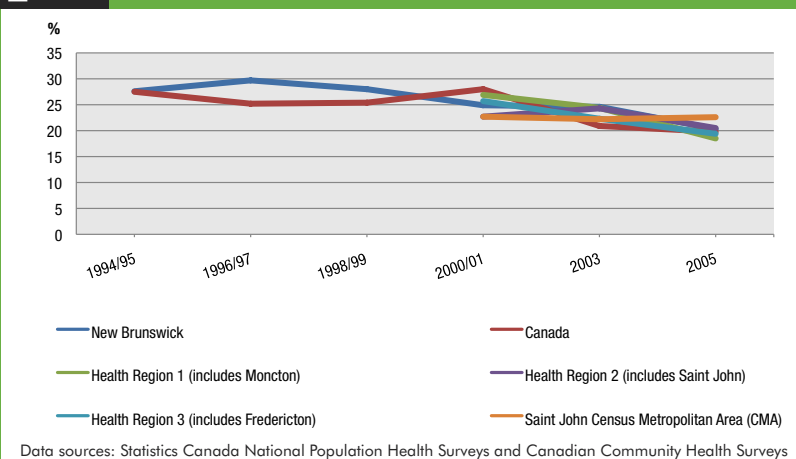
Statistics Canada reports smoking rates by province. As part of its Canadian Community Health Survey initiated in 2000/2001, Statistics Canada began

reporting smoking and other factors (e.g. physical activity, alcohol consumption, body mass index, etc.) affecting health by health region and census metropolitan areas (CMAs). The only CMA identified in New Brunswick is the Saint John CMA. This area includes the population of Saint John plus the population from the surrounding municipalities that are connected to the city through the workforce. According to Statistics Canada, municipalities included in CMAs must have a high degree of integration with the central urban area (e.g. Saint John) as measured by commuting flows derived from census place of work data. Figures 17 and 18 compare the rates of smokers classified as current daily or occasional smokers (males and females) for Health Regions 1, 2 and 3 and the Saint John CMA to national and provincial rates.<sup>20</sup>

**FIGURE 17** Male smoking rates for current daily or occasional smokers (1994-2005)



**FIGURE 18** Female smoking rates for current daily or occasional smokers (1994-2005)

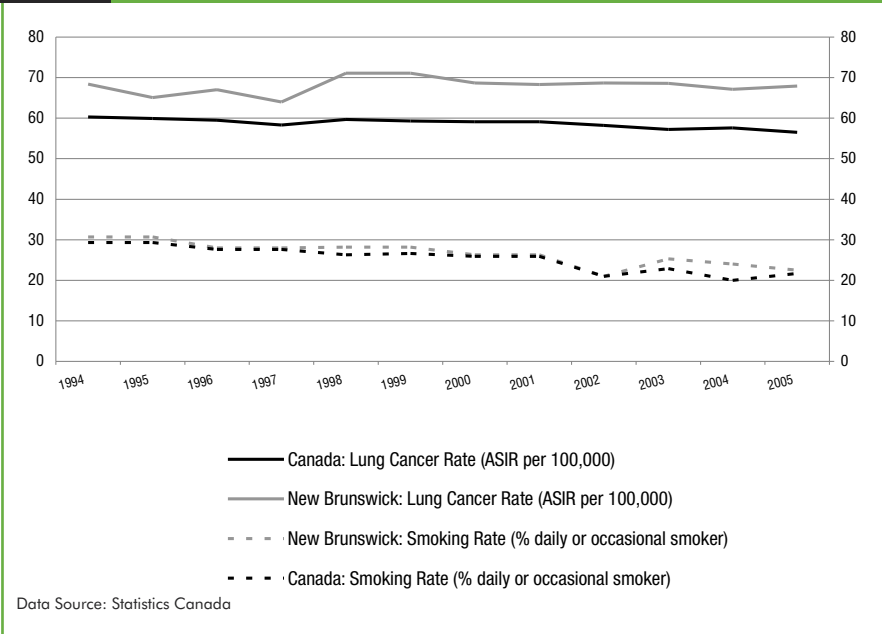


The data indicates (at least for the period between 2000/2001 and 2005) that smoking rates for males in Health Regions 1 and 2 and the Saint John CMA were at or below provincial and national rates. Male smoking rates in Health Region 3 were above the provincial and national rates. Males in the Saint John CMA had consistently lower smoking rates than all three health regions, the province and the national rate (Figure 17). In 2000/01, females smoking rates in all three health regions and the Saint John CMA were below the provincial and national rates and in 2005, smoking rates in Health Region 2 and the Saint John CMA were slightly above the provincial and national rate. Female smoking rates in Health Region 1 and 3 were at or slightly below national and provincial rates in 2005 (Figure 18).

These results raise important questions that require further study. Since smoking rates in New Brunswick have dropped as significantly as the rates in the rest of Canada, why are lung cancer

# 19

## Lung Cancer and Smoking Rates (both sexes) 1994-2005



rates among males and females in New Brunswick significantly and stubbornly higher than national rates (Figure 19)?

Since smoking rates in New Brunswick have dropped as significantly as the rates in the rest of Canada, why are lung cancer rates among males and females in New Brunswick significantly and stubbornly higher than national rates?

The same question applies to lung cancer rates in Saint John which in 2005 were significantly higher than provincial (49% and 65% for males and females respectively) and national (82% and 98% for males and females respectively) rates (Appendix B). Although smoking rates for Saint John alone are not available, smoking rates for the Saint John CMA

could be viewed as a reasonable indicator of smoking rates in the city. Smoking rates in the Saint John CMA would have to be exceptionally higher than the provincial or national rate to account for the significantly higher rates of lung cancer in Saint John. Yet, at least for 2000-2005, smoking rates in the Saint John CMA were below (males) or at (females) the provincial and national average for the same period. Clearly other risk factors are influencing the rates of lung cancer in Saint John.

### Occupational Exposure

Much of what is known about the cancer-causing effect of chemicals comes from studies based on occupational exposures. By the 1950s, the concept that workers

who were directly exposed to cancer-causing chemicals were at highest risk and that the risk did not stop at the factory gates was well understood by cancer epidemiologists.<sup>21</sup>

By the 1950s, the concept that workers who were directly exposed to cancer-causing chemicals were at highest risk and that the risk did not stop at the factory gates was well understood by cancer epidemiologists.

The number of cancers attributed to occupational exposure has increased from estimates of 2-10% in 1981 to 15-20% in 2007 because the number of agents/chemical considered to be definite occupational carcinogens has increased from 16 in 1981 to 28 in 2007, with an additional 140 chemicals listed as probable or possible industrial carcinogens.<sup>22</sup> The risk for some cancers increases even further for workers who smoke. Researchers at

Boston University School of Public Health recently published a review of new evidence linking occupational as well as environmental exposure to various cancers.<sup>23</sup> Table 1 summarizes the evidence for occupational exposure based on the strength of scientific research.

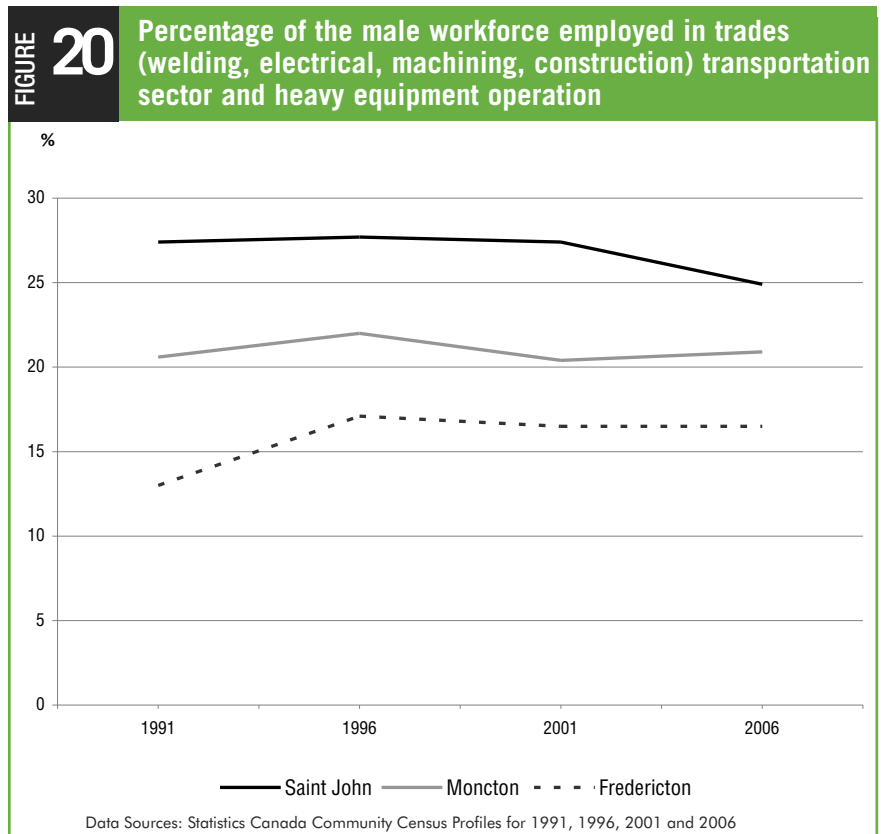
In Canada, several peer-reviewed scientific studies have examined the mortality, incidence and risks of various cancers and their link to occupational exposure in Canada. Primarily these studies have been done in Quebec, Ontario, Saskatchewan, Alberta and British Columbia.<sup>24</sup> No published, peer-reviewed scientific studies could be found for New Brunswick.

Occupational exposure to cancer-causing agents play a major role in lung cancer. Studies done in British Columbia have observed excess risk for all lung cancers for men employed in primary metal and mining, machining/welding, transportation, carpentry/wood processing, ship building, agriculture, electrical/utility and protective services (e.g. military) industries.<sup>25</sup> The risks were associated with exposure to metals, chlorinated pesticides and compounds such as PCBs and dioxins, asbestos, radon, wood dust and polyaromatic hydrocarbons (PAHs) such as benzene. All these agents/compounds have been classified as carcinogens by the International Agency for Research on Cancer (IARC). Workers who smoked added to their risk of lung cancer.

According to Statistics Canada census data, Saint John has a greater percentage of males working in occupations known to be associated with increased risks of lung cancer such as trades (e.g. welding, electrical, machining, construction) transportation and heavy equipment

operation (Figure 20; Appendix D).<sup>26</sup> These occupations also pose increased risks of other cancers (see Table 1, page 18). In addition, a higher percentage of Saint John males worked in manufacturing, processing and utilities sectors than males in Fredericton and Moncton (Appendix D).<sup>27</sup>

Between 40-51% of males in all three cities work in so-called sedentary or white collar occupations which include management, administration, and sales and services (Appendix D).<sup>28</sup> Fredericton and Moncton are in the upper end (47% and 51% respectively) of this range while Saint John is in the lower end (44%). Lung cancer has not been linked to sedentary employment, but office-type occupations have been linked to physical inactivity which has been identified in some studies as a risk factor for colorectal cancer.<sup>29</sup>



**Table 1. Selected carcinogenic agents and their occupational links with cancer.<sup>1</sup>**

Carcinogenic Agent	Occupation	Cancer site and strength of evidence <sup>2</sup>		Latency Period in years <sup>3</sup>
		Strong	Probable and Suspected	
<b>Arsenic</b>	metal mining and smelting; coal mining and burning; oil refineries; wood preserving operations	bladder; lung; skin; soft tissue sarcoma (angiosarcoma of the liver)	brain/central nervous system; liver; prostate; soft tissue sarcoma	10+
<b>Asbestos</b>	mining; insulation and shipyard workers;	lung; laryngeal; mesothelioma		4-40
<b>Benzene</b>	oil and petrochemical industries; transportation; manufacturing of plastics, resins, some types of rubbers and lubricants	leukemia; non-Hodgkin's Lymphoma	brain/central nervous system; lung; nasal & nasopharynx; multiple myeloma	6-14
<b>Butadiene</b>	oil refineries; petrochemical industries	lung	leukemia	
<b>Cadmium</b>	metal mining and smelting; electrical workers; battery plant and alloy workers; painters		pancreatic; kidney; prostate	
<b>Chromium</b>	steel and alloy producers; chrome plating operations; wood preserving operations	lung; nasal and nasopharynx		5-15
<b>Creosotes; Coal tars</b>	roofing; road paving; aluminum smelting and coking	bladder (coal tars); lung; skin		
<b>Ethylene oxide</b>	laboratory workers; hospital workers; fumigators	leukemia	breast	
<b>Formaldehyde</b>	plywood and oriented strand board manufacturing; appliance, telephone and electrical control manufacturing		nasal and nasopharynx	
<b>Ionizing radiation</b>	high-voltage equipment operators; nuclear reactors; uranium mining	bone; brain & CNS; nervous system; breast; leukemia; liver & biliary; lung; multiple myeloma; soft tissue sarcoma; skin; thyroid	bladder; colon; nasal and nasopharynx; ovarian; stomach	
<b>Lead</b>	metal smelting and mining; battery manufacturing/recyclers		brain/central nervous system; lung; kidney; stomach	
<b>Nickel</b>	nickel smelters, mixers and roasters; electrolysis workers	lung, nasal and sinuses;	laryngeal; pancreatic; stomach	3-30
<b>Pesticides</b>	agriculture and forestry workers; landscapers		brain/ central nervous system, breast; kidney; prostate; lung; leukemia; NHL; colon; Hodgkin's; multiple myeloma; ovarian; pancreatic; soft tissue sarcoma; stomach; testicular	
<b>Silica</b>	mining; foundries, brickmaking and sandblasting; solar panel manufacturing	lung		
<b>Straight oils, soluble oils, synthetic and semi-synthetic fluids</b>	metal machining; print press operations	bladder; laryngeal; lung nasal and nasopharynx; rectal; skin; stomach	esophageal; pancreatic; prostate	
<b>toluene</b>	manufacturing of paint, thinners, adhesives and rubber; oil refineries		brain/central nervous system; lung; rectal	
<b>wood dust</b>	carpentry; furniture and cabinetry making	lung; nasal and nasopharynx	laryngeal	

<sup>1</sup> Source: Adapted from Clapp RW, Jacobs MM and Loechler EL. 2007. Environmental and Occupational Causes of Cancer: New Evidence, 2005-2007. Prepared for: Cancer Working Group of the Collaborative on Health and the Environment. Lowell Center for Sustainable Production. University of Massachusetts. 45 p.

<sup>2</sup> Strong causal evidence of a link is based primarily on a Group 1 (known carcinogen) designation by the International Agency for Research on Cancer. Suspected evidence of a link is based on Clapp et al. 2007 assessment of existing epidemiologic studies.

<sup>3</sup> Source: Adapted from Davis DL. 2007. *Secret History of the War on Cancer*. Basic Books. New York, NY. p. 258-261.

Among males, occupational exposure is more likely a significant risk factor for lung cancer in Saint John than in Fredericton or Moncton.

Few women in Moncton, Fredericton and Saint John work in trade and transportation related occupations. Between 1991 and 2006, the majority (70%) of females in Moncton, Fredericton and Saint John worked in management, administrative, clerical, sales and services occupations (Appendix D).<sup>30</sup> An additional 20% were employed in social science, teaching, government and health care professions.

Occupational exposure is not likely a significant risk factor for lung cancer among women in Moncton, Saint John and Fredericton.

## Air pollution

Over the past two decades hundreds of studies have highlighted the role of airborne particulate matter (dust) in cardiovascular diseases and lung cancer,<sup>31</sup> two leading causes of death in New Brunswick and Canada. Major sources of particulate matter are vehicle exhaust, industrial smoke, fossil fuel combustion and waste incinerators. The negative health effects of particulate air pollution are known to increase as the particle size decreases. Fine particles penetrate deeper into the respiratory tract and have a high retention rate (i.e., not coughed up).

**Over the past two decades hundreds of studies have highlighted the role of airborne particulate matter (dust) in cardiovascular diseases and lung cancer, two leading causes of death in New Brunswick and Canada.**

Fine (less than 2.5 microns in diameter) and ultrafine (less than 1.0 microns) particulate matter can be laced with a range of contaminants including metals (e.g. arsenic and lead), ions (e.g. nitrates), organic compounds (e.g. dioxins, PAHs, benzene, butadiene), reactive gases (e.g. radon) and material of biologic origin (e.g. wood dust), all of which have been classified as known or probable carcinogens by the International Agency on Cancer Research (IARC). Considerable research has been done to determine the underlying mechanism of how air pollution causes cancer. There is a consensus among researchers that the cancer-causing effect of particulate matter is a combination of DNA repair suppression and enhancement of DNA replication errors.<sup>32</sup> When cells lose their ability to replicate without error or the DNA repair mechanism does not work properly, abnormal cell growth (cancer) can occur.

Scientific studies report that each 10 µg/m<sup>3</sup> (microgram per cubic meter) increase in fine particulate pollution (PM<sub>2.5</sub>) significantly increases the risk of cardiopulmonary and lung cancer deaths.<sup>33</sup> After controlling for smoking effects, the incremental increase in risk is as high as 4% for cardiopulmonary diseases and 8% for lung cancer.<sup>34</sup> Scientists with the World Health Organization (2004) have estimated that a yearly average PM<sub>2.5</sub> concentration of 7.5 µg/m<sup>3</sup> is the theoretical minimum-risk exposure for cancer.<sup>35</sup> For coarse particulate matter (PM<sub>10</sub>), the minimum risk-exposure value is estimated at 15 µg/m<sup>3</sup>.<sup>36</sup>

The Canada-wide 24-hour air quality standard for PM<sub>2.5</sub> is 30 µg/m<sup>3</sup>. There is no nation-wide annual average standard. The New Brunswick Department of Environment has not established air quality standards for PM<sub>2.5</sub> under the *Clean Air Act*. The department references the Canada-wide 24-hour air quality standard (30 µg/m<sup>3</sup>) as well as other standards in their annual reports.

Since 1992, the department has been monitoring PM<sub>2.5</sub> (by various methods) at several sites in Saint John. Sampling for PM<sub>2.5</sub> in Moncton and Fredericton, as well as other communities, began in 1999. Monitoring methods (e.g. dichotomous sampler, continuous unattended electronic monitors and Beta Attenuation Monitor), particularly in Saint John, have changed several times over the past 15 years and, therefore, it is difficult to compare long-term trends within and between communities.

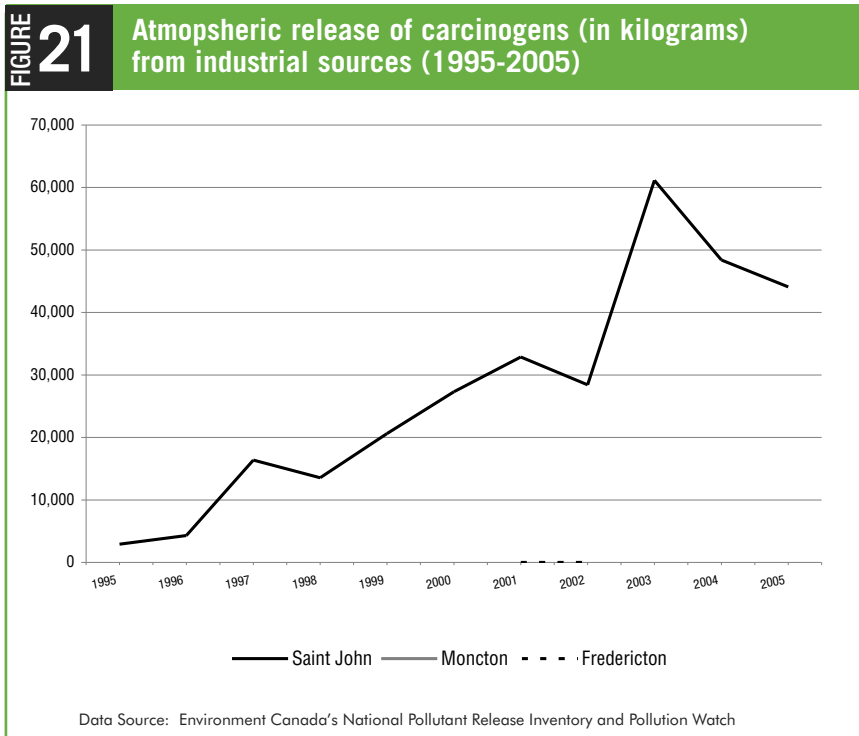
Provincial air quality monitoring reports indicate that between 1999 and 2006, monitoring stations in Saint John recorded a greater number of violations of the Canada-wide 24-hour air quality standard (30 µg/m<sup>3</sup>) than in Moncton or Fredericton.<sup>37</sup> Monitoring stations in Saint John also recorded higher monthly maximum 24-hour values of PM<sub>2.5</sub>, depending on the location and type of monitor, than those in Moncton and Fredericton. Annual average 24-hour PM<sub>2.5</sub> values between 1999-2006 in Saint John have also been over the World Health Organization's estimated PM<sub>2.5</sub> minimum-risk exposure threshold for cancer risk (7.5 µg/m<sup>3</sup>).

These results are not surprising given that Saint John has a greater number of industries releasing fine particulates and other pollutants into the atmosphere. According to Environment Canada's National Pollutant Release Inventory (NPRI), 23 companies in Saint John reported to the NPRI in 2007 compared to eight in Moncton and three in Fredericton.<sup>38</sup> The NPRI is a database containing information on the annual on-site releases of 367 substances and groups of substances to the air, water and land from industrial sources. Companies are legally obligated to report to the NPRI if they release one or more of the listed substances and they employ approximately 10 full-time employees. If a facility or

operation is involved in waste or sewage sludge incineration, wood preservation, fuel terminal operations, municipal waste water collection and treatment, stationary combustion equipment or quarrying, a report may be required regardless of the number of employees.

According to the NPRI database, the average amount of PM<sub>2.5</sub> released from three major facilities in Saint John (NB Power's Coleson Cove electrical generating station, Irving Oil Refinery and Irving Tissue) between 2002 and 2007 was 796 metric tonnes (mt).<sup>39</sup> In Moncton and Fredericton for the same time period, PM<sub>2.5</sub> releases were 43 mt and 1.4 mt respectively.

As indicated above, fine particulates can be laced with carcinogens, making the "dust" a more potent carcinogen. The amount of carcinogens released into the air by all industries in Saint John have been rising steadily since 1995 and are orders of magnitude greater than those released in Moncton and Fredericton where virtually no releases of carcinogens



**The amount of carcinogens released into the air by all industries in Saint John have been rising steadily since 1995 and are orders of magnitude greater than those released in Moncton and Fredericton where virtually no releases of carcinogens were reported.**

were reported (Figure 21). (Data prior to 1995 is not available as the NPRI was established in 1994.)

The high volume of fine particulates and carcinogen releases from Saint John industries and long-term exposure to air pollution levels above minimum risk-exposure levels combine to make air quality a leading cancer, particularly lung cancer, risk factor for Saint John residents.

## Colorectal Cancer Risk Factors

Like most cancers, a very small percentage (3%) of colorectal cancer cases have been linked to genetic syndromes, specifically familial adenomatous polyposis and hereditary nonpolyposis colon cancer.<sup>40</sup> A family history of colorectal cancer in first-degree relatives has been estimated to occur in 12-15% of colon cancer cases.<sup>41</sup> Since genetic factors play such a small role in colorectal cancer, the contribution of shared family lifestyle and environmental risk factors are key to understanding the incidence of colorectal (and other) cancers. For example, a US population-based case-control study found that individuals who shared a family history of colorectal cancer also shared certain risk factors such as smoking and a low fruit and

**Genetic factors play a small role in colorectal cancer. The contribution of shared family lifestyle and environmental risk factors are key to understanding the incidence of colorectal (and other) cancers.**

vegetable and high meat, fat and fast food diet.<sup>42</sup> The study found no link between physical inactivity and increased cancer risk.

A wide range of diet and lifestyle factors (e.g. obesity, excessive fat consumption, high meat - low fibre intake, lack of physical activity, smoking and alcohol consumption) have been identified as potential risks for colorectal cancer.<sup>43</sup> Physical inactivity and white collar jobs or sedentary jobs have been frequently (although not consistently or conclusively) linked to increased risk of colorectal cancer.<sup>44</sup> Similarly, the evidence for high dietary intake of red meat, low fruit and vegetable consumption and obesity as colorectal risk factors is mixed.<sup>45</sup> Researchers believe that fat and red meat *per se* are not carcinogens but rather the process of cooking meat at high temperatures forms carcinogenic compounds (e.g. N-nitrosos compounds and polycyclic aromatic hydrocarbons) and fatty diets can activate specific liver enzymes that enhance the metabolism and toxicity of

environmental chemicals in the body.<sup>46</sup> Similarly, obesity or over-weight *per se* has never been shown to cause or initiate cancer.<sup>47</sup>

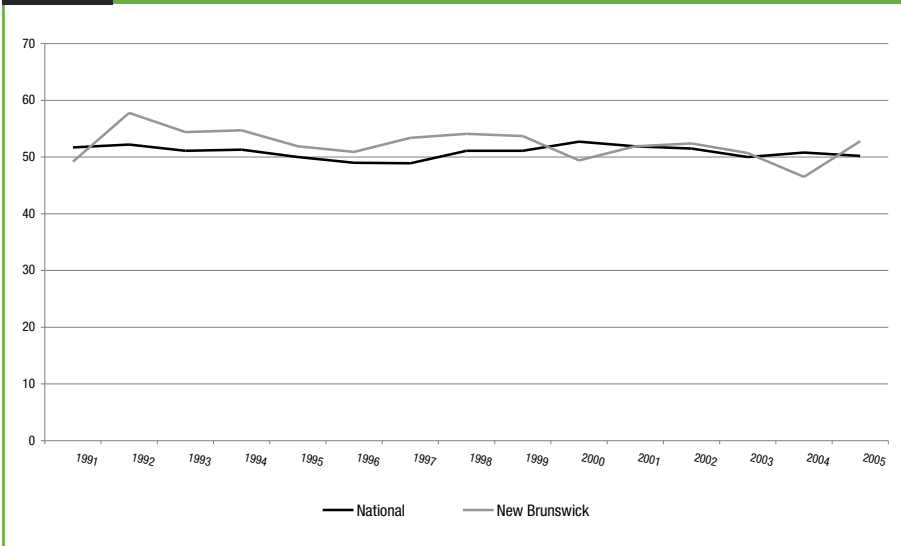
Increasingly, researchers believe that fat tissues are reservoirs for lipophilic (fat-loving)

environmental chemical carcinogens that eventually are released into the blood stream.<sup>48</sup>

Provincial-level data for potential diet and lifestyle risk factors associated with colorectal (or other cancers) is very limited. Statistics Canada data for 1994/95 to 2005 indicates New Brunswickers had lower physical activity rates and higher obesity rates.<sup>49</sup> Based on these potential risk factors, New

**FIGURE 22**

**Colorectal Cancer Rates (both sexes) 1991-2005.**  
Age-standardized incidence rates per 100,000 population



Brunswickers should have higher colorectal cancer rates than the national rate. In fact, colorectal cancer rates among New Brunswickers are similar to national rates (Figure 22).

Comparable long-term community-level data on the lifestyle risk factors associated with colorectal cancer (e.g. physical activity, diet, smoking) are not available for New Brunswick communities and therefore risk factor comparisons among communities were not possible in this study.

## Breast Cancer Risk Factors

It is widely accepted that genetic mutations account for a very small percentage (2-10%) of all breast cancers.<sup>50</sup> Approximately 20 genes are known to contribute to inherited breast cancers. Mutations in the so-called breast cancer genes, BRCA 1 and BRCA 2, are the most common and account for a small fraction (10%) of all breast cancer diagnoses. While the probability of developing breast cancer is higher among women with a mutation of BRCA 1 or BRCA 2, it does not imply the disease will develop. US and European studies have shown that the incidence of

breast cancer among women with BRCA 1 mutation born after 1940 were nearly twice that of women born earlier.<sup>51</sup> For women who test negative for the BRCA 1 and BRCA 2 gene mutation but have a family history of breast cancer, the lifetime incidence of breast cancer increases 5.5% with one affected relative and 13.3% with two affected relatives.<sup>52</sup>

Since so few breast cancers are linked to genetic mutations, the key to understanding why rates are higher among women with a family history of breast cancer is to examine shared family characteristics. For example,

family members are more likely to have similar or shared lifestyles, diets and environmental histories, as well as similar reproductive and hormonal patterns. Studies on identical twins provide some of the most compelling evidence that environmental, not genetic, factors contribute significantly to the development of cancer. In the largest study of twins ever conducted, researchers found that, among twins in which at least one woman developed breast cancer, environmental exposures unique to that woman made the most significant contribution (67%) to the development of cancer.<sup>53</sup> The risk of breast cancer in identical twins was 13% and the

risk in non-identical twins was 9%.

**One of the most significant risk factors for breast cancer is life-time exposure to synthetic estrogens.**

One of the most significant risk factors for breast cancer is life-time exposure to synthetic estrogens.<sup>54</sup>

Since 1987, the International Agency for Research on Cancer (IARC) has listed steroidal and non-steroidal estrogens as known human carcinogens. A decade

earlier, the IARC listed a man-made estrogen mimic, diethylstilbestrol (DES), as a carcinogen. Synthetic estrogens are found in oral contraceptives and hormone replacement therapies (HRT).

In 2003, large-scale studies investigating the benefits and risks of HRT in the US and Europe were halted before the end of the studies because women taking HRT had three times the rate of recurrence of new tumors compared to women who received other treatments for menopausal symptoms.<sup>55</sup> These findings were supported by the largest-ever study of breast cancer, the *Million Women Study* in the UK (2003). The study found that the risk of breast cancer was four times greater among users of estrogen-progestin combination therapies versus estrogen-only therapies.<sup>56</sup> Numerous studies have shown oral contraceptives increase the risk of breast cancer.<sup>57</sup>

A substantial (and growing) body of peer-reviewed scientific literature has demonstrated that many classified carcinogens are also estrogen mimics (xenoestrogens) which disrupt estrogen pathways and are risk factors for breast cancer (Table 2). For example, known carcinogens like organochlorinated pesticides (e.g. DDT, hexachlorobenzene) and polychlorinated biphenyls (e.g. PCBs, dioxins) are also estrogen mimics. These compounds are lipophilic (fat loving) and deposit in fatty tissue like breasts where they have been found to generate estrogenic micro-environments that influence the growth, shape and behaviour of breast tumours.<sup>58</sup> These compounds also cross the placental barrier and affect the developing fetus. Numerous studies have linked increased risks of breast and other cancers to pesticide exposure.<sup>59</sup> Exposures to various forms of radiation are also risk factors for breast cancer.<sup>60</sup>

Conservatively, half of all breast cancer risks are attributed to established risk factors such as genetics, family history, alcohol intake, obesity,

hormonal exposure, menopause and increased breast density.<sup>61</sup> Factors with unknown or no apparent consistent effects on breast cancer incidence include diet (coffee/tea consumption, high fat intake, low fruit and vegetable consumption) and lifestyle factors (smoking and physical exercise).<sup>62</sup> Many researchers now believe the rise in breast cancer is associated with lifestyle modification linked to hormone treatments and changes in reproductive behaviour (e.g. age at first pregnancy, low

**Many researchers now believe the rise in breast cancer is associated with lifestyle modification linked to hormone treatments and changes in reproductive behaviour (e.g. age at first pregnancy, low birthrates) and to increased levels of estrogen mimics in the environment.**

birthrates) and to increased levels of estrogen mimics in the environment.<sup>63</sup>

Between 1991 and 2005, average provincial breast cancer rates in New Brunswick were similar to average Canadian rates (Figure 10, page 11). Average breast cancer rates in Saint John, Fredericton and Moncton for the same time periods were 15-30% higher than provincial and national rates. When an epidemiological study found breast cancer rates in nine of 15 towns on Cape Cod were 20% above the average rate for the state of Massachusetts, researchers raised questions about possible environmental exposure.<sup>64</sup> Follow-up studies on indoor air and dust in 120 homes found 52 different hormonally active agents and mammary carcinogen compounds in air and 66 in dust.<sup>65</sup> The number of compounds detected per home ranged from 13-28 in air and from 6-42 in dust. The most abundant were plasticizers, disinfectants and flame retardants (banned in 1977). Twenty-three

**Table 2. Some compounds linked to breast cancer<sup>1</sup>**

Compound	International Agency for Research on Cancer (IARC) Classification			Endocrine Disrupting Compounds
	Known	Probable	Possible	
<b>Dioxins</b> - by-product of incinerating chlorinated compounds and industrial process that use chlorine (e.g. pesticide manufacturing)	■			■
<b>PCBs</b> - insulation fluids, plastics, inks, paints, dyes		■		■
<b>DDT/DDE</b> - insecticide			■	■
<b>Hexachlorobenzene</b> - herbicide				■
<b>Atrazine</b> - herbicide				■
<b>Heptachlor</b> - insecticide				■
<b>Dieldrin and Aldrin</b> - insecticides				■
<b>Other pesticides</b>				■
<b>Polycyclic aromatic hydrocarbons (PAHs)</b> - fossil fuel combustion, industrial air pollution, oil refining				■
<b>Bisphenol A (BPA)</b> - hard/soft plastic containers labelled with a triangle and the numbers 3, 6 or 7				■
<b>Alkylphenols</b> - surfactants, detergents, some pesticides				■
<b>Some metals</b> - smelters, oil refineries, battery recycling	■			■
<b>Phthalates</b> -plasticizer for PVC polymers				■
<b>Benzene</b> - solvents, fossil fuel combustion, oil refineries	■			
<b>Vinyl chloride</b> - resins for production of plastic pipes, floor covering, food packaging, appliances, credit cards	■			
<b>Organic solvents</b> - (e.g. styrene, formaldehyde, toluene, methylene chloride, trichlorethylene) used in manufacturing computer components, cleaning products and cosmetics	■	■		
<b>1,3 - Butadiene</b> - oil refining and fossil fuel combustion; production of polymers for paints, carpet backing, tires and other rubber products		■		
<b>Ethylene oxide</b> - disinfectant and pesticide; used in making resins, films and antifreeze	■			
<b>Aromatic amines</b> - manufacture of polyurethane foams, dyes, pharmaceuticals; diesel exhaust		■		■

<sup>1</sup> Source: Adapted from Gray et al. 2009. State of the Evidence: The connection between breast cancer and the environment. International Journal of Occupational and Environmental Health 15:43-78.

pesticides, including those long-banned such as DDT, heptachlor, and chlordane, were detected in air and 27 in dust. Detected concentrations exceeded government health-based guidelines for 15 compounds, but no guidelines were available for 28 compounds. In a related study, researchers found synthetic estrogens in septic tanks, groundwater and private wells.<sup>66</sup>

Researchers also examined whether there was an association between high breast cancer rates and length of residence on Cape Cod<sup>67</sup> and community-level versus individual-level socioeconomic status (income, education, unemployment).<sup>68</sup> They found that the longer a woman lived on Cape Cod, the greater her risk of breast cancer and the risk was not associated with her socio-economic status. However, when the risk was calculated using community-level socioeconomic data, the study found that breast cancer risks were higher in communities with higher socio-economic status. In other words, there was something about living in higher socio-economic communities that conferred a higher risk of breast cancer. One possible explanation is that community-level analysis may be encompassing (although not measuring) the collective effect of community-wide exposure to environmental contaminants which are not captured when examining individual-level data.

As for breast cancer rates in Fredericton, Moncton and Saint John, more detailed individual-level and community-level epidemiologic studies need to be done to determine why breast cancer rates are significantly higher in Saint John and Moncton.

## Prostate Risk Factors

Like breast cancer, genetic susceptibility and family occurrence explain a very small portion of the incidence of prostate cancers. Genetic mutations accounts for less than 2% and family history accounts for 5-20%.<sup>69</sup> There is little or inconclusive

evidence that high vegetable and fruit diets reduce the risk or that animal fat, meat, coffee and smoking increase the risk of prostate cancer.<sup>70</sup> Like breast cancer, prostate cancer is a hormone-related cancer and there is strong and growing evidence that synthetic hormone (endocrine) disrupting compounds affect prostate cancer development and progression.<sup>71</sup> In addition, researchers believe that male infants and children exposed to endocrine disrupting compounds may be at increased risk of prostate cancer as they age. These compounds can pass through the placenta into the developing fetus and the prostate appears to be more sensitive to these compounds during critical period of development (e.g. *in utero* and early childhood).<sup>72</sup>

Occupational exposure to compounds that mimic hormones (e.g. pesticides, metals, PAHs, chlorinated compounds) have been linked to increased risks of prostate cancer (See Table 1, page 18).<sup>73</sup> Studies consistently show that farmers and men with other occupational pesticide exposure are at risk of prostate cancer.<sup>74</sup>

**Occupational exposure to compounds that mimic hormones (e.g. pesticides, metals, PAHs, chlorinated compounds) have been linked to increased risks of prostate cancer.**

Between 1991 and 2005, average prostate cancer rates in Saint John and Fredericton were above the average provincial rate and significantly above the average national rate (Figure 14, page 13). Canadian and US studies report that prostate cancer rates are highest among men 75 years and older and that few men aged 50 and younger have prostate cancer.<sup>75</sup> This study found that prostate cancer rates in Saint John, Moncton and Fredericton were highest among men in the 75+ category in 1991 but by 2005 this

pattern had changed. Prostate cancer rates are now higher in the 65-74 category and there has been a general increase in the rate of cancer in the 55-64 age category (Figures 15 and 16, page 13).

The higher incidence of prostate cancer in younger men could be a function of higher rates of PSA (prostate-specific antigen) testing among men 40 years and older. A 2003 Statistics Canada study found that the introduction of PSA testing in the early 1990s did lead to more diagnoses among men younger than 80, but the national incidence rate in 1996-1998 was still highest among men 80 years and older. In New Brunswick, prostate cancer rates in Saint John, Moncton and Fredericton have been higher in the 65-74 age category since 1996 and, according to this study, incidence rates in Saint John have increased significantly in the 55-64 and 45-54 age categories between 1991 and 2005 (Figures 15 and 16, page 13).

More detailed individual-level and community-level epidemiologic studies need to be done to determine why prostate cancer rates are significantly higher in Saint John and Moncton and why prostate cancer rates in Saint John are rising among younger and younger men. Occupational exposure should be a significant component of these studies since a higher percentage of males in Saint John are employed in industries where their potential exposure to chemicals implicated in prostate cancer are also higher.

## Conclusions

**T**his study examined cancer incidence rates for four major cancers among three cities in New Brunswick (Saint John, Moncton and Fredericton) and compared them to national, provincial and health region-level rates. The results confirm that reporting cancer incidence rates by large geographic areas obscures important information on the health of New Brunswickers at the community level. For example, this study found that lung cancer incidence rates among males and females in Saint John were consistently and significantly higher than rates reported for Saint John's health region (Health Region 2), Fredericton, Moncton, New Brunswick and Canada. An examination of the major risk factors for lung cancer revealed that occupational exposure and air pollution were more significant risk factors for lung cancer in Saint John than in Fredericton or Moncton.

This study identified community-level changes in cancer incidence rates. The long-term trend for the age at which cancer rates peak in Saint John, Moncton and Fredericton for colorectal, prostate and breast cancer has shifted to younger age categories. These results contradict a common explanation given by health officials that increasing cancer rates are a function of an aging population and that a diagnosis of cancer is inevitable as one gets older.

With the exception of smoking, national and provincial data-gathering on so-called classical cancer risk factors (e.g. diet, physical activity, smoking, alcohol consumption, obesity) are inadequate. Furthermore, community-level data for these risk factors are virtually non-existent.

This study found no peer-reviewed scientific publications examining cancer risks associated with various occupations in New Brunswick. Long-term data on community-level exposure to environmental and industrial pollutants are also non-existent. Limited air quality monitoring indicates industrial releases of carcinogens and fine particulate matter are significantly higher in Saint John than in Moncton or Fredericton.

Cancer is largely a preventable disease. The majority of all cancers are associated with environmental exposures which occur at the individual and community level. Cancer prevention strategies based on disease and risk factor information gathered at the provincial or national level fail to: 1) identify cancers hotspots and their risk factors in communities; and 2) delay the development and implementation of risk intervention programs for those communities.

**An examination of the major risk factors for lung cancer revealed that occupational exposure and air pollution were more significant risk factors for lung cancer in Saint John than in Fredericton or Moncton.**

# Recommendations

In 2003, the New Brunswick Department of Health established the New Brunswick Cancer Network. To date, the focus of the Network's activity has been on cancer treatment and monitoring. The Network does not monitor cancer incidence at the community level nor does it gather information on cancer risk factors at any geographic scale.

Based on the results of this study, the Conservation Council of New Brunswick recommends that:

- the Minister of Health work with the Minister of Environment to improve air quality standards and eliminate the release of carcinogens from industrial sources in communities.

In addition, the Conservation Council recommends that the Minister of Health direct the New Brunswick Cancer Network to:

- undertake an appropriate epidemiological study to determine the cause of high lung cancer rates in Saint John;
- undertake detailed individual- and community-level epidemiological studies to determine why prostate cancer rates are high in Saint John and Moncton and why rates are rising among younger men in Saint John;

- undertake detailed individual- and community-level epidemiological studies to determine why breast cancer rates are high in Saint John and Moncton;
- begin public reporting of cancer rates at the community-level;
- expand cancer prevention messaging and programs to include occupational and environmental risk factors like exposure to pesticides, household and industrial chemicals and air pollution; and
- conduct an epidemiological study to examine the relationship between cancer incidence and occupations in the province.

## Appendix A

Cancer counts (N) and age -standardized incidence rates (ASIR) per 100,000 population for males and females in Saint John, Moncton, Fredericton, New Brunswick<sup>1</sup> and Health Regions 1, 2 and 3 (1999-2003)<sup>1</sup>

MALES	New Brunswick		Health Region 1		Moncton		% above (+) or below (-) Provincial Rate	% above (+) or below (-) Health Region Rate
	N	ASIR	N	ASIR	N	ASIR		
	<b>Prostate</b>	2513	129.6	655	132.3	206	134.9	+4.1
<b>Lung</b>	1886	96.8	436	87.2	136	88.0	-9.1	+0.9
<b>Colorectal</b>	1240	62.9	330	65.7	99	63.2	+0.5	-3.8

MALES	Health Region 2		Saint John		% above (+) or below (-) Provincial Rate	% above (+) or below (-) Health Region Rate	Health Region 3		Fredericton		% above (+) or below (-) Provincial Rate	% above (+) or below (-) Health Region Rate
	N	ASIR	N	ASIR			N	ASIR	N	ASIR		
	<b>Prostate</b>	529	119.4	225	122.6	-5.4	+2.7	548	131.8	174	144.3	+11.3
<b>Lung</b>	475	108.2	246	133.4	+37.8	+23.3	387	92.6	107	88.3	-8.8	-4.6
<b>Colorectal</b>	272	60.1	121	64.9	+3.2	+8.0	263	62.1	88	73.4	16.7	+18.2

FEMALES	New Brunswick		Health Region 1		Moncton		% above (+) or below (-) Provincial Rate	% above (+) or below (-) Health Region Rate
	N	ASIR	N	ASIR	N	ASIR		
	<b>Breast</b>	2420	101.5	638	104.6	231	115.9	+14.2
<b>Lung</b>	1135	48.2	276	45.5	103	45.8	-5.0	+0.7
<b>Colorectal</b>	1158	45.6	323	48.4	114	48.6	+6.6	+0.4

FEMALES	Health Region 2		Saint John		% above (+) or below (-) Provincial Rate	% above (+) or below (-) Health Region Rate	Health Region 3		Fredericton		% above (+) or below (-) Provincial Rate	% above (+) or below (-) Health Region Rate
	N	ASIR	N	ASIR			N	ASIR	N	ASIR		
	<b>Breast</b>	597	105.8	+19.1	120.9	-5.4	+14.3	544	107.7	197	125.4	+23.5
<b>Lung</b>	335	59.1	+33.4	64.3	+37.8	+8.8	218	44.0	69	41.5	-13.9	-5.7
<b>Colorectal</b>	273	45.4	+1.1	46.1	+3.2	+1.5	250	46.2	88	47.0	+3.1	+1.7

<sup>1</sup> Data source for New Brunswick and Health Region cancer rates. New Brunswick Department of Health. New Brunswick Health Status Report: 1999-2003. ISBN 978-1-55396-863-4.

## Appendix B

Age-standardized prostate, breast, colorectal and lung cancer incidence rates (ASIR) per 100,000 population for Saint John, Moncton, Fredericton, New Brunswick<sup>1</sup> and Canada for 1991, 1996, 2001 and 2005<sup>2</sup>

MALES	Prostate				% change 1991 to 2005	Colorectal				% change 1991 to 2005	Lung				% change 1991 to 2005
	1991	1996	2001	2005		1991	1996	2001	2005		1991	1996	2001	2005	
	<b>Saint John</b>	164.7	139.5	128.7	155.6	-5.5	50.3	93.9	40.6	56.9	+13.1	136.0	104.2	115.1	126.4
<b>Fredericton</b>	137.2	178.6	127.0	105.1	-23.4	58.3	76.8	78.3	59.2	+1.5	60.6	89.0	71.2	75.9	+25.2
<b>Moncton</b>	92.0	115.0	122.8	145.5	+58.2	74.5	44.4	59.5	80.1	+7.5	96.3	85.9	75.5	76.0	-21.1
<b>Provincial</b>	120.2	130.5	137.3	146.8	+22.1	57.7	61.7	62.3	66.5	+15.3	98.2	99.5	94.5	84.6	-13.8
<b>National</b>	112.3	110.6	132.5	121.0	+7.7	62.3	59.4	63.4	60.9	-2.2	90.7	82.2	75.8	69.4	-23.5

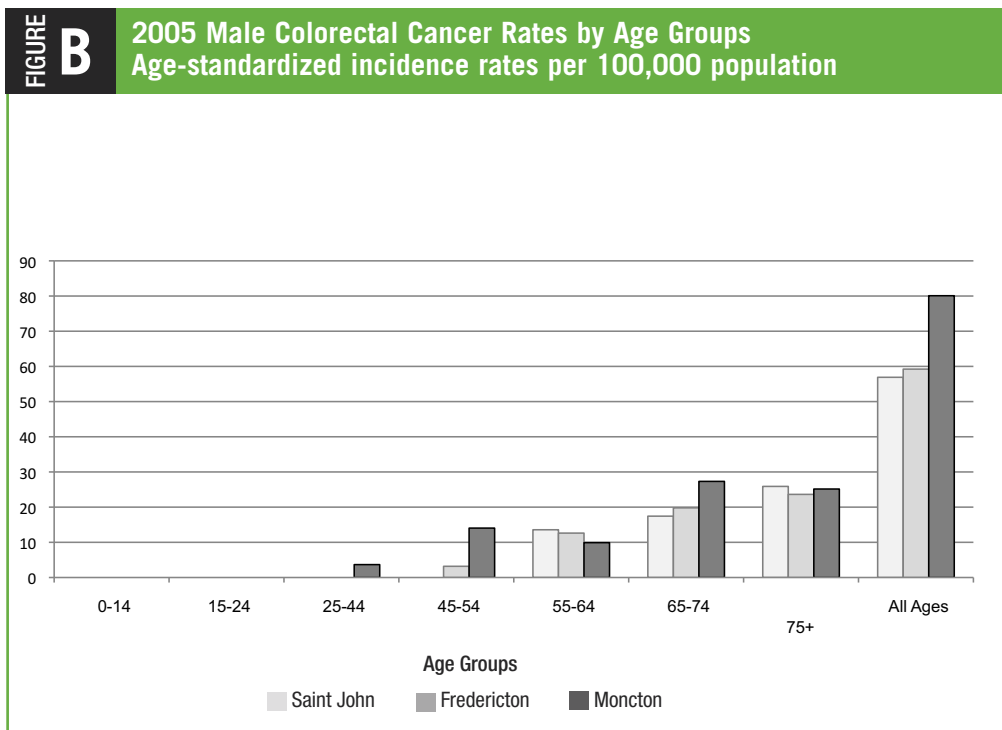
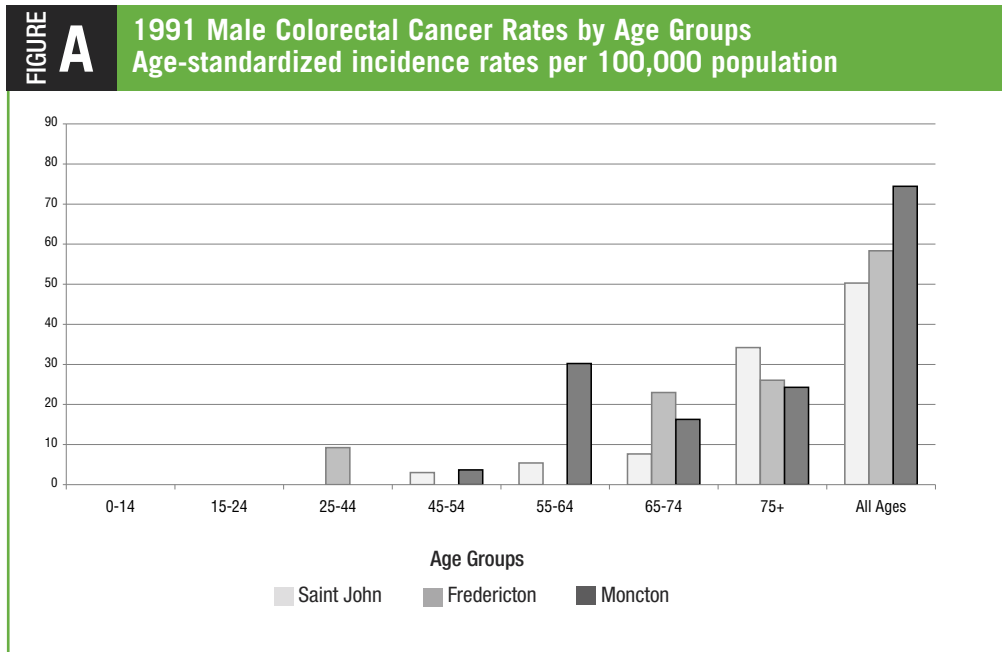
FEMALES	Breast				% change 1991 to 2005	Colorectal				% change 1991 to 2005	Lung				% change 1991 to 2005
	1991	1996	2001	2005		1991	1996	2001	2005		1991	1996	2001	2005	
	<b>Saint John</b>	107.1	112.1	126.4	113.7	+6.2	44.4	36.2	42.3	52.9	+19.1	50.9	74.5	74.9	92.5
<b>Fredericton</b>	134.7	177.8	123.0	97.8	-27.4	34.4	54.5	33.7	22.9	-33.4	35.8	30.1	29.2	49.2	+37.4
<b>Moncton</b>	108.3	111.2	127.7	124.6	+15.1	46.1	43.5	56.8	59.1	+28.2	53.0	35.1	57.1	60.2	+13.5
<b>Provincial</b>	103.9	99.8	96.8	99.8	-3.9	42.6	42.0	43.8	42.2	-0.9	37.2	41.5	49.0	56.1	+50.8
<b>National</b>	100.1	99.0	99.6	97.5	-2.6	43.5	40.3	42.5	41.2	-5.3	37.7	41.9	44.4	46.8	+24.1

<sup>1</sup> Data source for New Brunswick cancer rates. NB Provincial Cancer Registry Data Base.

<sup>2</sup> Data source for Canadian cancer rates. Statistics Canada. Table 103-0550- New cases for ICD-O-3 primary sites of cancer, by age group and sex, Canada, provinces and territories, annual CANSIM (database).

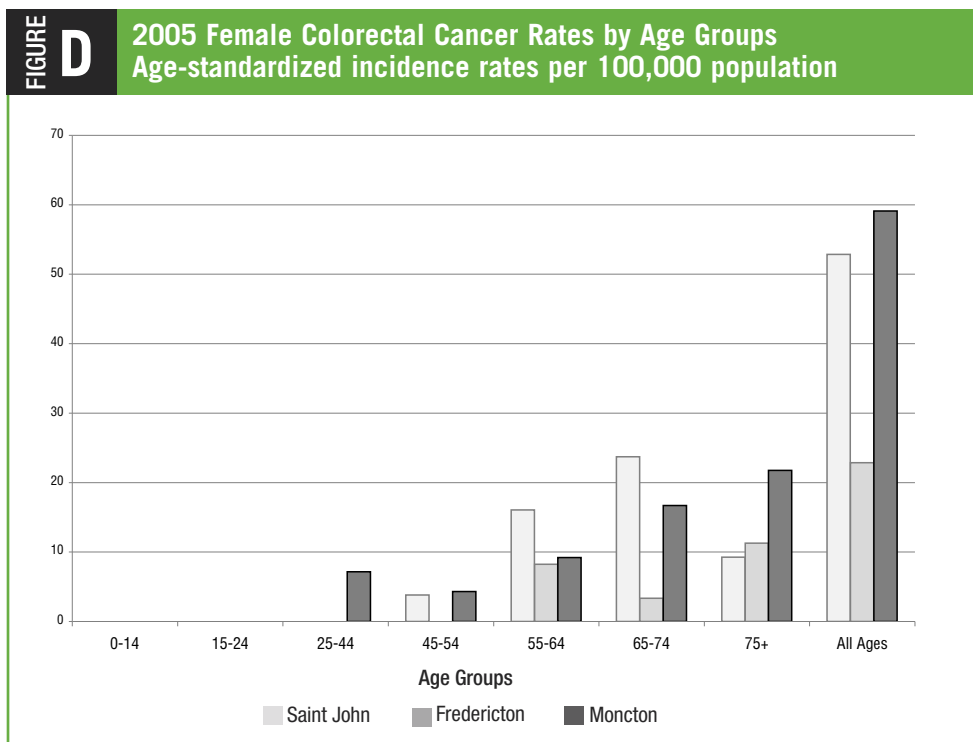
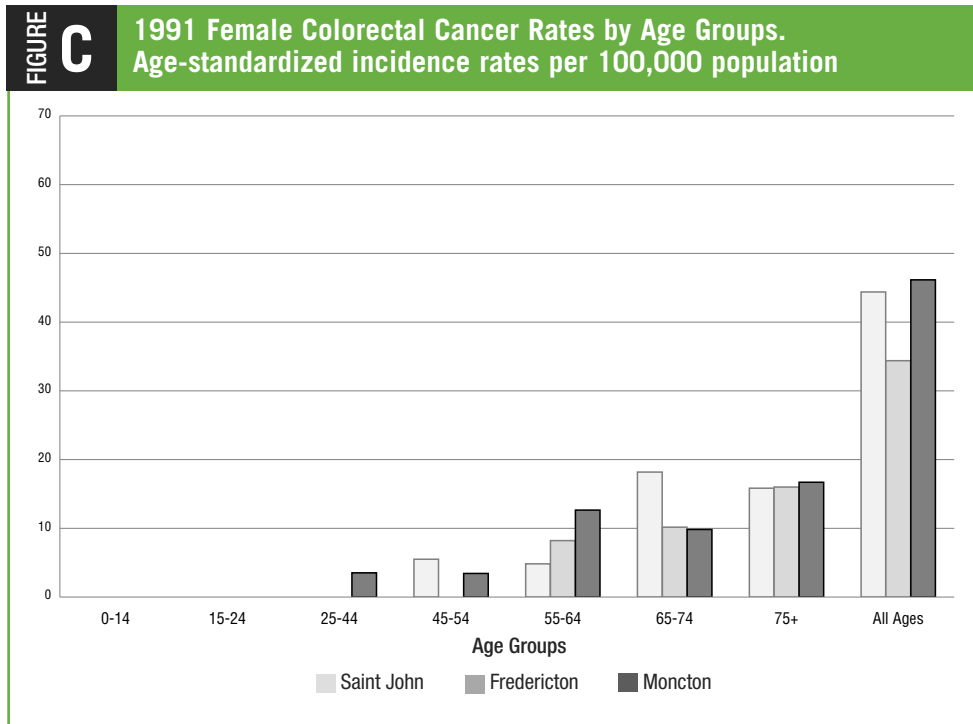
## Appendix C

Male age-standardized colorectal cancer incidence rates per 100,000 population by age groups for Saint John, Moncton and Fredericton for 1991 and 2005.



# Appendix C

Female age-standardized colorectal cancer incidence rates per 100,000 population by age groups for Saint John, Moncton and Fredericton for 1991 and 2005.



## Appendix D

Percent employment by major occupation categories for males and females in Saint John, Moncton and Fredericton for 1991, 1996, 2001, 2006<sup>1</sup>

MALES													
Major occupation categories	Saint John 1991	Saint John 1996	Saint John 2001	Saint John 2006	Moncton 1991	Moncton 1996	Moncton 2001	Moncton 2006	Fredericton 1991	Fredericton 1996	Fredericton 2001	Fredericton 2006	
Management/administration/clerical	18.5	17.0	21.8	21.6	24.8	24.8	27.7	27.4	23.1	22.7	25.2	25.4	
Sales/service	22.3	25.4	25.0	26.4	24.4	25.3	23.3	25.3	22.4	23.4	21.7	23.4	
Trades/transport/heavy equipment operation	27.4	27.7	27.4	24.9	20.7	22.0	20.4	20.9	18.5	17.1	16.5	16.5	
Manufacturing/processing/utilities	14.4	7.7	5.3	4.4	9.3	4.9	6.3	3.5	6.6	3.1	1.9	1.7	
Agriculture	1.4	0.5	0.5	0.4	0.9	0.6	0.1	0.3	1.4	1.0	1.0	0.7	
Forestry/mining/oil and gas/fishing	0.6	0.8	0.5	0.5	0.6	0.2	0.2	0.4	1.2	0.7	0.7	0.4	

FEMALES													
Major occupation categories	Saint John 1991	Saint John 1996	Saint John 2001	Saint John 2006	Moncton 1991	Moncton 1996	Moncton 2001	Moncton 2006	Fredericton 1991	Fredericton 1996	Fredericton 2001	Fredericton 2006	
Medicine/health	10.9	9.6	9.7	10.6	11.4	11.7	10.5	12.8	8.3	7.4	8.1	9.1	
Management/administration/clerical	43.1	33.6	34.3	37.0	42.6	37.1	37.3	37.0	44.9	36.6	38.2	34.6	
Sales/service	30.7	37.3	38.5	33.6	28.7	32.2	32.9	30.7	26.7	32.4	28.5	30.7	
Social science/teaching/government	8.3	6.9	7.3	9.9	10.4	8.9	8.9	10.0	11.6	12.0	13.2	14.1	
Agriculture	0.1	0.3	0.2	0.1	0.2	0.2	0.0	0.2	0.2	0.2	0.4	0.2	
Forestry/mining/oil and gas/fishing	0.0	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.4	0.0	0.1	0.0	

<sup>1</sup>Data Source: Statistics Canada. Community Profiles for 1991, 1996, 2001 and 2006.

## References

1. Sexton K, Waller LA, McMaster RB, Maldonado B and Adgate JL. 2002. The importance of spatial effects for environmental health policy and research. *Human and Ecological Risk Assessment* 8(1):109-25.
- Walter SD. 2000. Disease mapping: a historical perspective. In *Spatial Epidemiology: methods and applications*. Elliott P, Wakefield J, Best N, Briggs DM (eds). Oxford: Oxford University Press 223-252.
- Elliot SJ, Eyels J, and DeLuca P. 2001. Mapping health in the Great Lakes Areas of Concern: A user-friendly tool for policy and decision makers. *Environmental Health Perspectives* 109(s.6): 817-826.
2. Elliot, P and Wartenberg P. 2004. Spatial epidemiology: current approaches and future challenges. *Environmental Health Perspectives* 112(9):998-1006.
3. Goss Gilroy Inc, Environmental & Occupational Health+Plus, SENES Consultants Ltd. 2005. Belledune Area Health Study. Summary Report.
4. Goss Gilroy Inc, Environmental & Occupational Health+Plus, SENES Consultants Ltd. 2005. Belledune Area Health Study. Appendix A - Human Health Risk Assessment.
5. Goss Gilroy Inc, Environmental & Occupational Health+Plus, SENES Consultants Ltd. 2005. Belledune Area Health Study. Appendix D - Community Health Status Assessment Technical Report.
6. New Brunswick Department of Health. *New Brunswick Health Status Report: 1999-2003*. Report Number: ISBN 978-1-55396-863-4.
7. Ibid.
8. Canadian Cancer Society/National Cancer Institute of Canada. 2008. *Canadian Cancer Statistics 2008*. Toronto, Canada.
9. Ibid.
10. New Brunswick Department of Health. See note 6.
11. Loeb KR and Loeb LA. 2000. Significance of multiple mutations in cancer. *Carcinogenesis* 21:379-85.
12. Davis DL, Donovan M, Herberman R, Gaynor M, Axelrod D, van Larebecke, N and Sasco AJ. 2007. The need to develop centers for environmental oncology. *Biomedicine & Pharmacotherapy* 61:614-622.
- Peto J. 2001. Cancer epidemiology in the last century and the next decade. *Nature* 411:390-395.
- Perera FP. 1997. Environment and cancer: who are susceptible? *Science* 278(7):1068-1073.
13. Czene K, Lichtenstein P and Hemminki K. 2002. Environment and heritable causes of cancer among 9.6 million individuals in Swedish family-cancer database. *International Journal of Cancer* 99:260-266.
- Lichtenstein P, Holm NV, Verkasalo PK, et al. 2000. Environmental and heritable factors in the causation of cancer - analyses of cohorts of twins from Sweden, Denmark and Finland. *New England Journal of Medicine* 342:78-85.
14. Belpomme D, Irigaray P, Sasco AJ, Newby JA, Howard V, Clapp R and Hardell L. 2007. The growing incidence of cancer: role of lifestyle and screening detection (Review). *International Journal of Oncology* 30:1037-49.
15. Irigaray P, Newby JA, Clapp R et al. 2007. Lifestyle-related factors and environmental agents causing cancer: an overview. *Biomedicine and Pharmacotherapy* 61:640-58.
- Danaei G, Vander Hoorn S, Lopez AD, Murray CJL and Ezzati M. 2005. Causes of cancer in the world: comparative risk assessment of nine behavioural and environmental risk factors. *Lancet* 366: 1784-1793.
- Peto 2001. See note 12.
16. Danaei et al. 2005. See note 15.
- Levi F. 1999. Cancer prevention: epidemiology and perspectives. *European Journal of Cancer* 35:1912-1924.
17. Stephens M and Siroonian J. 1998. Smoking prevalence, quit attempts and successes. (Statistics Canada, Catalogue 82-003-XPB) *Health Reports* 9(4):31-37.
18. Statistics Canada, Canadian Community Health Survey (CCHS), 2000/2001, 2003 and 2005; National Population Health Survey (NPHS), 1994/1995, 1996/1997 and 1998/1999. Table 104-0027 Smoking status, by age group and sex, household population aged 12 and over, Canada, provinces, territories, health regions.
19. Ibid.
20. Ibid.

21. Davis DL. 2007. *The secret history of the war on cancer*. New York (New York), Basic Books. p. 100.
22. Belpomme D, Irigaray P, Hardell L, Clapp R, Montagnier L, Epstein S and Sasco AJ. 2007. The multitude and diversity of environmental carcinogens. *Environmental Research* 105:414-429.
- Rushton L, Hutchings S and Brown T. 2008. The burden of cancer at work: estimation as the first step to prevention. *Occupational and Environmental Medicine* 65(12): 789-800.
- Doll R and Peto R. 1981. The causes of cancer: quantitative estimates of avoidable/risk of cancer in the United States today. *Journal of the National Cancer Institute* 66:1191-1308.
23. Clapp RW, Jacobs MM and Loechler EL. 2007. *Environmental and Occupational Causes of Cancer: New Evidence, 2005-2007*. Prepared for: Cancer Working Group of the Collaborative on Health and the Environment. Lowell Center for Sustainable Production. University of Massachusetts. 45 p.
24. Karunanayake CP, McDuffie HH, Dosman JA, Spinelli JJ and Pahwa P. 2008. Occupational exposures and non-Hodgkin's lymphoma: Canadian case-control study. *Environmental Health* 7: 44-52.
- Pahwa P and McDuffie HH. 2008. Cancer among potash workers in Saskatchewan. *Journal of Occupational and Environmental Medicine* 50(9): 1035-1041.
- Ramanahumar AV, Nadon L and Siemiatycki J. 2007. Exposure in painting related occupations and risk of selected cancer: results from a case-control study in Montreal. *American Journal of Industrial Medicine* 51:419-427.
- McDuffie HH, Quail J, Ghosh S, and Pahwa P. 2007. Host factors, occupation and testicular cancer in Saskatchewan, Canada: 1979-2002. *J. Agriculture, Safety and Health* 13(3):247-58.
- Rouseau MC, Parent ME, Nadon L, Latreille B and Siemiatycki J. 2007. Occupational exposure to lead compounds and risk of cancer among men: a population-based case-control study. *American Journal of Epidemiology* 166(9): 1005-14.
- Brophy et al. 2006. Occupation and breast cancer: A Canadian case-control study. *Annals of the New York Academy of Science* 1076:765-77.
- Gaertner RR, Trpeski L, and Johnson KC. 2004. A case-control study of occupational risk factors for bladder in Canada. *Cancer Causes and Control* 15(10):1007-19.
25. MacArthur AC, Le ND, Fang R and Band PR. 2009. Identification of occupational cancer risk in British Columbia: A population-based case-control study of 2,998 lung cancers by histopathological type. *American Journal of Industrial Medicine*, published online at DOI 10.1002/ajim.20663.
26. Statistics Canada. Community Census Profiles for 1991, 1996, 2001 and 2005.
27. Ibid.
28. Ibid.
29. Moradi T, Gridley G, Bjork J, Dosemeci M, Ji BT, Berkel HJ and Lemeshow S. 2008. Occupational physical activity and risk for cancer of the colon and rectum in Sweden among men and women by anatomic subsite. *European Journal of Cancer Prevention* 17(3):201-8.
- Zhang Y, Cantor KP, Dosemeci M, Lynch F, Shu Y and Zheng T. 2006. Occupational and leisure-time physical activity and risk of colon cancer by subsite. *Journal of Occupational and Environmental Medicine* 48(3):236-43.
- Hsing AW, McLaughlin JK, Chow WH, Schuman LM, CO Chien HT, Gridley G, Bjelke E, Wacholder S and Blot WL. 1998. Risk factors for colorectal cancer in a prospective study among U.S. white men. *International Journal of Cancer* 77(4):549-53.
- Fraser G and Pearce N. 1993. Occupational physical activity and risk of cancer of the colon and rectum in New Zealand males. *Cancer Causes and Control* 4(1):45-50.6
30. Statistics Canada. See note 26
31. Beelen R, Hoek G, van den Brandt PA et al. 2008. Long-term exposure to traffic-related air pollution and lung cancer risk. *Epidemiology* 19(5): 702-10.
- Edwards R, Pless-Molloli T, Howel D, Chadwick T, Bhopal R, Harrison R and Gribbin H. 2006. Does living near heavy industry cause lung cancer in women? A case-control study using life grid interviews. *Thorax* 61(12):1076-82.

- Oyana TJ, Rogerson P and Lwebuga-Mukassa JA. 2004. Geographic clustering of adult asthma hospitalization and residential exposure to pollution at a United States-Canada border crossing. *American Journal of Public Health* 94(7): 1250-57.
- Kampa M and Castanas E. 2008. Human health effects of air pollution. *Environmental Pollution* 151(2):362-7.
- Chen TM, Gokhale J, Shofter S and Kuschner WG. 2007. Outdoor air pollution: particulate matter health effects. *American Journal of Medical Science* 333(4): 235-43.
- Laden F, Hart JE, Smith TJ, Davis ME and Garshick E. 2007. Cause-specific mortality in the unionized trucking industry. *Environmental Health Perspectives* 115(8): 1192-6.
- Miller KA, Siscovick DS, Sheppard L et al. 2007. Long-term exposure to air pollution and incidence of cardiovascular events in women. *New England Journal of Medicine* 356(5):447-58.
- Naess Ø, Nafstad P, Aamodt G, Clauseen B and Rosland P. 2007. Relation between concentration of air pollution and cause-specific mortality: four-year exposures to nitrogen dioxide and particulate pollution in 470 neighborhoods in Oslo, Norway. *American Journal of Epidemiology* 165(4): 435-43.
- Coyle Y, Minahjuddin AT, Hynan LS and Minna JD. 2006. An ecological study of the association of metal air pollutants with lung cancer incidence in Texas. *Journal of Thoracic Oncology* 1(7): 654-61.
32. Mehta M, Chen LC, Gordon T, Rom W and Tang MS. 2008. Particulate matter inhibits DNA repair and enhance mutagenesis. *Mutagenesis Research* 657(2): 116-21.
- Møller P, Folkmann JK, Forchhammer L, Bruäuner EV, Danielsen PH, Risom L and Loft S. 2008. Air pollution, oxidative damage to DNA, and carcinogenesis. *Cancer Letters* 266(1):84-97.
- Valavanidis A, Fiotakis K and Vlachogianni T. 2008. Airborne particulate matter and human health: toxicological assessment and importance of size and composition of particulates for oxidative damage and carcinogenic mechanism. *Journal of Environmental Science and Health C. Environmental Carcinogenicity and Ecotoxicologic Review* 26(4): 339-62.
33. Valavanidis et al., 2008. See note 32.
- Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K and Thurston GD. 2002. Lung cancer, cardiopulmonary mortality, and long term exposure to fine particulate air pollution. *Journal of the American Medical Association* 287(9):1132-41.
34. Laden F, Schwartz J, Speizer FE and Dockery DW. 2006. Reduction in fine particulate air pollution and mortality: Extended follow-up of the Harvard Six Cities Study. *American Journal of Respiratory and Critical Care Medicine* 173(6):667-72.
- Cohen AJ. 2003. Air pollution and lung cancer: what more do we need to know? *Thorax* 58:1010-2.
- Pope et al., 2002. see Note 33.
- Dockery DW, Pope CA, Xu X et al. 1993. An association between air pollution and mortality in six U.S. cities. *New England Journal of Medicine* 329:1753-9.
35. Cohen AJ, Anderson HR, Ostro B, et al. 2004. Urban air pollution .1353-433. In: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors*. Geneva: World Health Organization. Cited in Danaei et al., 2005, note 15.
36. Ibid.
37. New Brunswick Department of Environment: Air Quality Monitoring Results in New Brunswick for 2000 to 2006.
38. Environment Canada. 2009. National Pollutant Release Inventory online data base.
39. Ibid.
40. Samowitz WS, Curtin K, and Lin HH. 2001. The colon cancer burden of genetically-defined hereditary nonpolyposis colon cancer. *Gastroenterology* 121(4):830-838.
- Jass JR. 2000. Familial colorectal cancer: pathology and molecular characteristics. *Lancet Oncology* 1:220-226.
41. Kerber RA, Slattery ML, Potter JD, Caan BJ and Edwards SL. 1998. Risk of colon cancer associated with family history of cancer or colorectal polyps: the diet, activity, and reproduction in colon cancer study. *International Journal of Cancer* 78:157-160.

- Fuchs C, Giovannucci E, Colditz G, Hunter DJ, Speizer F and Willett W. 1994. A prospective study of family history and the risk of colorectal cancer. *New England Journal of Medicine* 331:1669-1674.
42. Slattery ML, Levin TR, Ma K, Goldgar K, Holubkov R and Edwards S. 2003. Family history and colorectal cancer predictors of risk. *Cancer Causes and Control* 14:879-887.
43. Gibbons L, Waters C, Mao Y and Elliston L. 2001. Trends in colorectal cancer incidence and mortality. *Health Reports* 12(2): 41-55.
44. Harriss DJ, Atkinson G, Batterham A, George K, Tim Cable N, Reilly T, Haboubi N and Renehan AG. 2009. Lifestyle factors and colorectal cancer risk (2): a systematic review and meta-analysis of associations with leisure-time physical activity. *Colorectal Disease* [e-published January 17, 2009 ahead of print].
- Moradi T, Gridley G, Bjork J, Dosemeci M, Ji BT, Berkel HJ and Lemeshow S. 2008. Occupational physical activity and risk for cancer of the colon and rectum in Sweden among men and women by anatomic subsite. *European Journal of Cancer* 17(3): 2001-8.
- Zhang Y, Cantor KP, Dosemeci M, Lynch CF, Zhu Y and Zheng T. 2006. Occupational and leisure-time activity and risk of colon cancer by subsite. *Journal of Occupation and Environmental Medicine* 48(3):236-43.
- Hsing AW, McLaughlin JK, Chow WH, Schuman LM, CoChien Ht, Gridley G, Bjeike E, Wacholder S and Blot WJ. 1998. Risk factors for colorectal cancer in a prospective study among U.S. white men. *International Journal of Cancer* 77(4): 549-53.
- Martinez ME, Giovannucci E, Spiegelman D, Hunter DJ, Willett WC and Colditz GA. 1997. Leisure-time physical activity, body size and colon cancer in women. Nurses's Health Study Research Group. *Journal of the National Cancer Institute* 89(13): 948-55.
45. Wilkins T and Reynolds PL. 2008. Colorectal cancer: a summary of the evidence for screening and prevention. *American Family Physician* 78(12):1385-92.
- Hu J, La Vecchia C, DesMeules M, Negri E and Mery L. 2008. Meat and fish consumption in Canada. *Nutrition and Cancer* 60(3):313-24.
- Ryan-Harshman M and Aldoori W. 2007. Diet and colorectal cancer: review of the evidence. *Canadian Family Physician* 53(11):1913-20.
- Weisburger JH. 2002. Lifestyle, health and disease prevention: the underlying mechanisms. *European Journal of Cancer Prevention* 11:S1-7.
46. Santarelli RL, Pierre F and Corpet DE. 2008. Processed meat and colorectal cancer: a review of epidemiologic and experimental evidence. *Nutrition and Cancer* 60(2):131-44.
- Ryan-Harshman and Aldoori 2007. See note 45.
- Gonzalez GA. 2006. The European prospective investigation into cancer and nutrition (EPIC). *Public Health and Nutrition* 259:629-42.
- Kato J, Ikemoto A and Mizutani T. 2003. The effect of dietary fatty acids on the expression levels and activities of hepatic drug metabolizing enzymes. *Journal of Health Science* 49:105-14.
- Potter JD. 1996. Nutrition and colorectal cancer. *Cancer Causes and Control*. 7:127-46.
47. Irigaray P, Newby JA, Clapp R, Hardell L, Howard V, Montagnier L, Epstein S and Belpomme D. 2007. Lifestyle-related factors and environmental agents causing cancer: an overview. *Biomedicine and Pharmacotherapy* 61:640-658.
48. Irigaray P, Newby JA, Lacomme S and Belpomme D. 2007. Overweight/obesity and cancer genesis: more than a biological link. *Biomedicine and Pharmacotherapy* 61:665-678.
49. Statistics Canada, Canadian Community Health Survey (CCHS), 2000/2001, 2003 and 2005; National Population Health Survey (NPHS), 1994/1995, 1996/1997 and 1998/1999. Table 105-4009. Body mass index (BMI), by sex, household population aged 18 and over excluding pregnant females, Canada, provinces and territories, occasional. Table 105-4033. Leisure-time physical activity, by sex, household population aged 12 and over, Canada, provinces and territories, occasional.
50. Gray J, Evans N, Taylor B, Rizzo J and Walker M. 2009. State of the Evidence: The connection between breast cancer and the environment. *International Journal of Occupational and Environmental Health* 15(1):43-78

Steiner E, Klubert D and Knuston D. 2008. Assessing breast cancer risk in women. *American Family Physician* 78(12): 1361-1366.

51. King MC, Marks JH, Mandell JB and New York Breast cancer Study Group. 2003. Breast and ovarian cancer risk due to inherited mutations in BRCA 1 and BRCA 2. *Science* 302:643-46.

52. Collaborative Group on Hormonal Factors in Breast Cancer. 2001. Familial breast cancer: collaborative reanalysis of individual data from 52 epidemiological studies including 58,209 women with breast cancer and 101,986 women without the disease. *Lancet* 358 (9291):1389-1399.

53. Lichtenstein P, Niels V and Pia K. 2000. Environmental and heritable factors in the causation of cancer: analyses of cohorts of twins from Sweden, Denmark and Finland. *New England Journal of Medicine* 343(2):78-85.

54. Gray et al. 2009. See note 50.

Steiner et al. 2008. See note 50.

55. Writing Group for the Women's Health Initiative Investigators. 2002. Risks and Benefits of estrogen plus progestin in healthy postmenopausal women. *Journal of the American Medical Association* 288(3); 321-33

Holmberg L and Anderson H. 2004. HABITS (hormonal replacement therapy after breast cancer - is it safe?): trial stopped. *Lancet* 363:453-5.

56. Million Women Study Collaborators. 2003. Breast cancer and hormone-replacement therapy in the Million Women Study. *Lancet* 362:419-27.

57. Newcomer LM, Newcomb PA, Trentham-Dietz A et al. 2003. Oral contraceptive use and risk of breast cancer by histologic type. *International Journal of Cancer* 106:961-4

Althuis MD, Brogan DD, Coates RJ et al. 2003. Breast cancers among very young premenopausal women (United States). *Cancer Causes Control* 14:151-160.

Kumle M, Weiderpass, Braaten T et al. 2002. Use of oral contraceptives and breast cancer risk: the Norwegian-Swedish women's Lifestyle and Health Cohort Study. *Cancer Epidemiology Biomarkers and Prevention* 11:1375-81

58. Munoz-de-Toro M, Durando M, Beldomenico PM et al. 2006. Estrogenic microenvironments generated by organochlorine residues in adipose mammary tissue modulates biomarker expression in ERalpha-positive breast carcinoma. *Breast Cancer Research* 8: 47-56.

Muscat JE, Britton JA, Djordjevic MV et al. 2003. Adipose concentrations of organochlorine compounds and breast cancer recurrence in Long Island, New York. *Cancer Epidemiology Biomarkers and Prevention* 12:1474-8.

Woolcott CG, Aronson KJ, Hanna WM et al. 2001. Organochlorines and breast cancer risk by receptor status, tumor size and grade (Canada). *Cancer Causes Control* 12:395-404.

59. Bassil KL, Vakil C, Sanborn M, Cole DC, Kaur JS and Kerr KJ. 2007. Cancer health effects of pesticides: Systematic review. *Canadian Family Physician* 53:1704-1711.

Brophy JT, Keith MM, Gorey KM et al. 2006. Occupation and breast cancer: a Canadian case-control study. *Annals of the New York Academy of Sciences* 1076: 765-777.

Teitelbaum SL, Gammon MD, Britton JA et al. 2006. Reported residential pesticide use and breast cancer risk on Long Island New York. *American Journal of Epidemiology* 165: 643-51.

60. Andrieu N, Easton DF, Chang -Claud J et al. 2006. Effect of chest X-rays on the risk of breast cancer among BRAC1/2 mutation carriers in the International BRAC 1/2 Carrier Cohort Study: a report from EMBRACE, GEGNNEPSO, GEO-HEBON and IBCCS Collaborators' Group. 2006. *Journal of Clinical Oncology* 24:3361-6.

Tsai KK, Chuang EY, Little JB, Yuan ZM, 2005. Cellular mechanisms for low-dose ionizing radiation-induced perturbation of the breast tissue microenvironment. *Cancer Research* 6:6734-44.

Barcellos-Hoff MH, Park C and Wright EG. 2005. Radiation and the microenvironment: tumorigenesis and therapy. *National Review of Cancer* 5:867-75.

Morgan WF. 2003. Non-targeted and delayed effects of exposure to ionizing radiation: II. Radiation-induced genomic instability and bystander effects in vivo, castogenic factors and transgenerational effects. *Radiation Research* 159:581-96.

61. Gaudette LA, Silberberger C, Altmayer CA and Gao R. 1996. Trends in breast cancer incidence and mortality. *Health Reports* 8(2): 29-37.
- Madigan MP, Zielger RG, Benichou J, Byrne C and Hoover RN. 1995. Proportion of breast cancer cases in the United States explained by well-established risk factors. *Journal of the National Cancer Institute* 87:1681-5.
62. Pirie K, Beral V, Peto R et al., for the Million Women Study Collaborators. 2008. Passive smoking and breast cancer in never smokers; prospective study and meta-analysis. *International Journal of Epidemiology* 37(5):1069-79.
- Steiner E, Kulbert D, Hayes M, Hamilton A and Kolasa K. 2007. Clinical inquiries. Does a low-fat diet help prevent breast cancer? *Journal of Family Practice* 56(7): 583-584.
- Prentice RL, Caan B, Chlebowski RT, et al. 2006. Low-dietary pattern and risk of invasive breast cancer: the Women's Health Initiative Randomized Controlled Dietary Modification Trial *Journal of the American Medical Association* 296(6):629-642.
- van Gils CH, Peeters PH, Bueno-de-Mesquita HB et al. 2005. Consumption of vegetables and fruits and risk of breast cancer. *Journal of the American Medical Association* 293(2):183-193.
- Hamajima N, Hirose K, Tajima K et al., for the Collaborative Group on Hormonal Factors in Breast Cancer. 2002. Alcohol, tobacco and breast cancer - collaborative re-analysis of individual data from 53 epidemiological studies, including 58,515 women with breast cancer and 95,067 women without the disease. *British Journal of Cancer* 87(11): 1234-1245.
63. Nudelman J, Taylor B, Evans N et al. 2009. Policy and research recommendations emerging from the scientific evidence connecting environmental factors and breast cancer. *International Journal of Occupational and Environmental Health* 15(1):79-101.
- Reeves GK, Pirie K, Green J, Bull D and Beral V for the Million Women Study Collaborators. 2009. Reproductive factors and specific histological types of breast cancer: prospective study and meta-analysis. *British Journal of Cancer* 100(3):538-544.
64. Brody JG, Rudel RA, Melly SJ and Maxwell NI. 1998. Endocrine disruptors and breast cancer. *Forum for Applied Research and Public Policy* 13(3): 24-31.
65. Rudel RA, Camann DE, Spengler JD, Korn LR and Brody JG. 2003. Phthalates, alkylphenols, pesticides, polybrominated diphenyl ethers, and other endocrine-disrupting compounds in indoor air and dust. *Environmental Science and Technology* 37(20):4543-4553.
66. Rudel RA, Geno P, Melly SJ et al. 1998. Identification of alkylphenols and other estrogenic phenolic compounds in wastewater, septage, and groundwater on Cape Cod, Massachusetts. *Environmental Science and Technology* 32(7): 861-9.
67. McKelvey W, Brody JG, Aschengrau A and Swartz CH. 2004. Association between residence on Cape Cod, Massachusetts, and breast cancer. *Annals of Epidemiology* 14:89-94.
68. Webster TF, Hoffman K, Weinberg, J, Vieira J and Aschengrau A. 2008. Community- and individual-level socioeconomic status and breast cancer risk: multilevel modeling on Cape Cod, Massachusetts. *Environmental Health Perspectives* 116(3): 1125-1129.
69. Wigle DT, Turner MC, Gomes J and Parent M. 2008. Role of hormonal and other factors in human prostate cancer. *Journal of Toxicology and Environmental Health Part B* 11:242-259.
- Grönberg H. 2003. Prostate cancer epidemiology. *Lancet* 361:859-64.
70. Wigle et al. 2008. See note 69.
- Darlington GA, Kreiger N, Lightfoot N, Purdham, and Sass-Kortsak A. 2007. Prostate cancer risk and diet, recreational physical activity and cigarette smoking. *Chronic Disease in Canada* 27(4):145-53.
71. Prins GS. 2008. Endocrine disruptors and prostate cancer risk. *Endocrine Related Cancer* 15(3):649-56.
- Wigle et al. 2008. See note 69.
- Hess-Wilson JK and Knudsen KE. 2006. Endocrine disrupting compounds and prostate cancer. *Cancer Letters* 241:1-12.
72. Prins 2008. See note 71.
73. Ramanakumar AV, Nadon L and Siemiatycki J. 2008. Exposure in painting related occupations and risk of selected cancers: results from a case-control study in Montreal. *American Journal of Industrial Medicine* 51:419-427.

Clapp et al 2007. See note 23.

Band PR, Le ND, Fang R, Threlfall WJ and Gallagher RP. 1999. Identification of occupational cancer risks in British Columbia, Part II: A population-based case-control study of 1516 prostate cancer cases. *Journal of Occupational and Environmental Medicine* 41(4):223-47.

Aronson KJ, Siemiatycke J, Dewar R and Gérin M. 1996. Occupational risk factors for prostate cancer: results from a case-control study in Montréal, Québec, Canada. *American Journal of Epidemiology* 143(4): 363-73.

74. Bassil et al. 2007 see note 59.

Hardell L, Andersson SO, Carlberg M et al. 2006. Prostate cancer and exposure to pesticides in agricultural settings. *Journal of Occupational and Environmental Medicine* 48:700-7.

Van Maele-Fabry G, Libotte V, Willems J and Lison D. 2006. Review and meta-analysis of risk estimates for prostate cancer in pesticide manufacturing workers. *Cancer Causes Control* 17:353-373.

Van Maele-Fabry G and Willems J. 2004. Prostate cancer among pesticide applicators: a meta-analysis. *International Archives of Occupation and Environmental Health* 77: 559-570.

Alavanja MC, Samanic C, Dosemeci M et al. 2003. Use of agricultural pesticides and prostate cancer risk in the Agricultural Health Study cohort. *American Journal of Epidemiology* 157:800-14.

75. Gibbons L and Waters C. 2003. Prostate cancer - testing, incidence, surgery and mortality. *Health Reports* 14(3):9-20.

Grönberg 2003. See note 69.