Cancer in Young Adults in Canada

www.cancercare.on.ca www.cancer.ca

cancer care ontario | action cancer ontario

Public Health Agency of Canada | Agence de santé publique du Canada

Canadian Cancer Society | Société canadienne du cancer

The information in this report was analyzed and interpreted by:

Beth Theis[1], Diane Nishri[1], Saira Bahl[1], Anne-Marie Ugnat[2] and Loraine Marrett[1]
[1]Cancer Care Ontario and [2]Public Health Agency of Canada (formerly part of Health Canada)

with support from the Cancer in Young Adults in Canada (CYAC) Working Group:

Christofer Balram, Provincial Epidemiology Service, New Brunswick
Sharon Buehler, Memorial University of Newfoundland
Ron Dewar, Cancer Care Nova Scotia
Douglas C. Dover, Alberta Cancer Board
Dagny Dryer, Prince Edward Island Cancer Registry
Juanita Hatcher, Alberta Cancer Board (currently at Aga Khan University, Pakistan)
Eric Holowaty, Cancer Care Ontario
Claire Infante-Rivard, McGill University, Quebec
Yang Mao, Public Health Agency of Canada
Mary McBride, British Columbia Cancer Agency
Nazeem Muhajarine, University of Saskatchewan
Donna Turner, CancerCare Manitoba
Hannah K. Weir, Centers for Disease Control and Prevention, USA

and with the editorial assistance of Shirley A. Huchcroft.

This report is dedicated to the memory of Sir Richard Doll, who had a long and distinguished career as an epidemiologist.
Richard Doll died on July 24 2005, at the age of 92.
His talk "Progress against cancer: an epidemiologic assessment" at the 1991 Meeting of the Society for Epidemiologic Research in Buffalo, New York, subsequently published in American Journal of Epidemiology (volume 134, 1991) was the inspiration for the work presented in this report.

"Cancer Care Ontario: **Cancer in Young Adults in Canada**, Toronto, Canada, 2006." May 2006, ISBN 0-921325-10-X (print), ISBN 0-921325-11-8 (pdf)

On the Internet this report is available at http://www.cancercare.on.ca and http://www.cancer.ca

Additional copies may be requested from Divisions of the Canadian Cancer Society or by calling Cancer Information Service 1 888 939-3333 (see *For Further Information*).

La version française de cette publication est disponible sur demande.

- More than 150,000 Canadians are survivors of a cancer diagnosed since 1980 in their young adult years.
- Approximately 10,000 cancers are diagnosed in young adults every year.
- Cancer mortality rates in general fell for young men and women between 1983 and 1999.
- Cancer is the main cause of early death among young adult women.
- Almost two-thirds of young adult cancers occur in young women. Of these, breast cancer is the most common.
- More young women than young men are now diagnosed with, and die from, lung cancer.
- Testicular cancer is the most common cancer in young men, and its incidence rose between 1983 and 1999.
- Melanoma is the second most common cancer among young adults.
- Increases in incidence are most striking for thyroid cancer in young men and women, lymphoma in young women and testicular cancer.
- Incidence is decreasing among young adults for many preventable cancers. Young Canadians appear to be increasingly following three main recommendations — avoiding smoking, minimizing sun exposure and having regular Pap tests.

CALL TO ACTION

While the increasing trends in incidence for a number of the cancers common in young adults support the need for more research into the reasons for these increases, and for continued surveillance, enough is known about the causes of some common cancers, such as melanoma and lung cancer, that risk reduction should be a priority. For these, exposure reduction strategies and supporting policies aimed at children and youth, their custodians and the organizations with a special responsibility for their wellbeing should be developed, implemented and vigorously promoted. While tobacco reduction strategies provide a good example of what can be achieved, smoking rates are still unacceptably high, especially in adolescent girls. Other factors that may increase cancer risk among young adults, such as use of tanning equipment, poor diet and lack of physical activity, need equal attention.

ACKNOWLEDGEMENTS

This report could not have been produced without the technical support and graphic skills of Sandrene Chin Cheong and the administrative support of Yen Borrego, both at Cancer Care Ontario.

The authors acknowledge the contributions of Jennifer Frood (Cancer Care Ontario) during the early development of this work, and the assistance and support of colleagues, in particular Eric Holowaty, Terry Sullivan, Bronwen Waller and Erin Pichora (Cancer Care Ontario) and Yang Mao, Robert Semenciw, Chris Waters, Wei Luo and Wendy Thompson (Public Health Agency of Canada). Mary Hodges and Catherine McGowan assisted Shirley Huchcroft.

Drs. Max Parkin (Oxford University), Anthony Miller (University of Toronto) and Don Wigle (University of Ottawa) critically reviewed a draft of this report. Their insightful comments resulted in important improvements.

Bo Miedema, Ryan Hamilton and Julie Easley (Dalhousie University Family Medicine Teaching Unit, New Brunswick) provided a personal perspective, with quotations from survivors themselves. These were collected as part of a qualitative study of the impact of cancer in young adults.

The following agencies provided the data used in this report:

- The Public Health Agency of Canada (cancer mortality and health survey data)
  - The mortality data were provided to the Public Health Agency of Canada from the Canadian Vital Statistics databases at Statistics Canada with the knowledge and consent of the provincial and territorial vital statistics registries which supply the data to Statistics Canada. Their cooperation is gratefully acknowledged.
  - Parts of this analysis are based on the Statistics Canada micro-data which contains anonymized data collected in the National Population Health Survey 1994-1998. All computations on these micro-data were prepared by the Public Health Agency of Canada and the responsibility for the use and interpretation of these data is entirely that of authors.
- The provincial and territorial cancer registries (cancer incidence data).
- The International Agency for Research on Cancer (pre-publication international cancer incidence data for 1993-1997).

Monika Dixon, Canadian Cancer Society, provided excellent administrative support to produce, print, translate and distribute this report.

TABLE OF CONTENTS

TABLE OF CONTENTS

Tables*

Figures*

* See individual cancer chapters for additional tables and figures.

FOREWORD

There are few countries with populations the size of Canada's that are covered entirely by population-based cancer registries, and even fewer in which such coverage extends far enough back in time to permit examination of trends in cancer incidence for two decades. This precious resource is what has allowed the preparation of this volume in which the patterns and trends in cancer incidence (and mortality) in young adults in Canada are presented. There is no standard international definition of "young adult". The age range chosen by the authors (ages 20–44) has a sound rationale: it stretches from "adolescence" (generally defined as ages 15–19) to the time of life around which, in women, menopause induces quite marked changes in the cancer profile. The value of studying cancer in young adults has been clearly stated by Sir Richard Doll, in his now-classic article published in 1991. He pointed out that trends in this age group are particularly valuable in identifying the causes of cancer, since they must reflect exposures to carcinogens that have occurred in the relatively recent past, and that when such exposures vary according to specific generations ("birth cohorts"), then the consequent changes in disease risk will be first observed in the young. The authors of this monograph add a third reason—the relative importance of inherited cancer syndromes among young cancer cases—which provides the opportunity to identify the responsible genes and their interaction with risk factor exposures.

The monograph is an example of what is often defined as "descriptive epidemiology", presenting the risk of different types of cancer (defined in terms of anatomical site, and often by histological features too) according to sex, age, geography (within Canada, and internationally), and over time. But, as in all good scientific enquiry, the observations are used to make deductions about the possible reasons underlying them, artefactual or causal. Patterns and trends of the different disease entities can be compared with what is known of the prevalence of known or suspected risk factors, and their changes over time. It is in this type of comparison that incidence data from cancer registries are particularly valuable, in giving a direct measure of the risk of disease. Mortality rates are a poor substitute, particularly in this age group, where survival varies between different populations, and, with major advances in chemotherapy for several cancers in the last decades, is changing rapidly over time. These changes in survival mean that variations in mortality cannot be interpreted in terms of differential exposure to possible risk factors. A further obvious limitation is that the deaths occurring in a particular time period and age group relate to cases diagnosed several years earlier, and at younger ages.

It is manifestly not pleasant to develop cancer at any age, even in extreme old age, but in the most active and productive years of life, with maximum family and social responsibilities, it is a particular tragedy. Simple statistics such as the number of new cases of different cancers, the numbers of deaths, and the numbers of years of life lost that these deaths represent are invaluable in setting priorities for cancer control activities. The comprehensive review of numbers and causes in this monograph provides an invaluable planning resource, and is a useful basis from which to evaluate how the occurrence of cancer in this age group should be weighed when considering strategies for cancer control in general. Future projections of incidence and mortality have been used to set targets against which to evaluate progress in prevention and treatment, the monograph incorporates some short-term projections of incidence, and the importance of extending this work is included among the recommendations.

FOREWORD

In his keynote address to the 1985 meeting of the International Association of Cancer Registries, Dr. Peter Greenwald of the US NCI stated that cancer registries could only justify their existence if they spent as much time and effort on analyzing, interpreting and presenting information as they did on collection, coding and data management. This volume is an admirable example of how cancer registry data can, and should, be made available, for the benefit of planners, researchers, and the public, with the ultimate aim of reducing the burden of this important cause of death and disability among the young.

Dr. D. Maxwell Parkin
University of Oxford
President, International Association of Cancer Registries

"I think the low points have been… that you question whether or not that the dreams that you did have will ever come true, and how much longer should you have to wait to have them? That you're not ready for your life to end, in fact you're just starting to realize that you had a life." ***Female, Breast Cancer, 32***

"…what can we tell patients with testicular cancer when they ask, 'Doctor … will I still be able to have children?' "
Editorial by Scott Saxman, MD, FACP, National Cancer Institute
(Journal of the National Cancer Institute 2005)

INTRODUCTION

Approximately 10,000 Canadians aged 20–44 were expected to be diagnosed with cancer in 2005 and 2,000 of them were expected to die from it. Based on data from 2003, it is estimated that more than 150,000 Canadians are survivors, with all its attendant difficulties, of a cancer diagnosed since 1980 in their young adult years.

Society has a "duty of care" to protect its younger and more vulnerable members, including children, adolescents and to some extent young adults. A diagnosis of cancer in a young adult has far-reaching consequences. Affected individuals may either spend decades living with the effects of cancer and its treatment (physical, reproductive, social, emotional and spiritual), or have tragically shortened lives with major repercussions on their families and society in general. They may still be completing their education, establishing their economic independence and building their own families when they are diagnosed and treated. The short- and long-term economic consequences of their cancer may be substantial.

The importance of studying cancers in young adults

Although cancer can affect individuals at any age, including infants, it is largely a disease associated with older age; 44% of all cancers are diagnosed after the age of 70.[25] Consequently, overall cancer patterns reflect cancers that are most prevalent in middle and old age, especially breast, prostate, lung, and colorectal. Cancer patterns at younger ages (childhood, adolescence and young adulthood) differ, however, from patterns at older ages and even among themselves.

There are several reasons to examine cancer patterns specifically in young adults. First, cancers occurring during young adulthood result from exposures relatively early in life. While it is generally believed that many childhood cancers arise as a result of events prior to birth, cancers in young adults are more likely to be related to post-natal exposures. Since the duration of time for relevant exposure is relatively short, young adults will have had fewer accumulated exposures. Potential etiologic factors may therefore be easier to identify from systematic examination of cancer patterns in this age group. Furthermore, the developing tissues of children and adolescents may be more sensitive to some carcinogenic events, which may manifest as a cancer diagnosed in early adulthood. Some of these cancers may therefore represent "sentinel events" providing a warning of "new" or changing exposures. For example, excess sun exposure in childhood appears to be particularly important in the etiology of cutaneous malignant melanoma, one of the most common cancers in the young adult age group. There is some evidence that this is due to enhanced sensitivity to the carcinogenic effects of sunlight early in life.[8]

Second, lifetime cancer risks tend to be shared to some extent by individuals who are born about the same time (a "birth cohort"). For example, today's adolescent boys have a much lower prevalence of smoking than their grandfathers did, and these boys will likely continue, through their lives, to smoke less. Thus, as they age they will have lower rates of lung cancer compared to their grandfathers at comparable ages. This "cohort effect" will first be evident during young adulthood, when lung cancer begins to appear. The incidence of lung cancer in today's young adults will therefore be important for forecasting future lung cancer rates and for planning preventive strategies and cancer services.

Third, cancers that occur in this age group may reflect heightened genetic susceptibility to the disease, for example, in individuals who inherit a cancer predisposition gene (e.g., BRCA 1 or BRCA 2 mutation).[6] Cancer patterns in young adults may therefore

suggest directions for investigation of genetic factors and interactions between genetic factors and risk factor exposures.

Sir Richard Doll argued strongly for surveillance of cancer trends in young adults in 1991, during his address to the Society for Epidemiologic Research. He challenged epidemiologists by saying:

> The trends in young adults are, I suggest, by far the most important for assessing our progress against cancer for two reasons. First, because the trends can reflect only relatively recent changes in the prevalence of carcinogenic agents and are not confused by the effect of changes in the distant past and, second, because young people tend to adopt new habits before the old.[41]

In addition to the theoretical reasons noted above, systematic surveillance can serve a number of more immediate purposes. First, it can highlight etiologic research priorities that would not otherwise be evident. For example, increasing trends in the incidence of testicular cancer are alarming and should be stimulating research aimed at identifying exposure changes in the early lives of males. Surveillance could also help to identify areas where prevention should be focussed in attempts to stem both the current and future tides of cancer. For example, the increasing incidence of melanoma, for which the primary risk factor is known to be overexposure to the sun, would highlight the need for emphasis on sun safety early in life.

Finally, knowing more about the size and characteristics of the affected population should stimulate research into the unknown consequences of cancer in this age group, and thence to efforts to reduce harmful late effects. It can also serve as a foundation for related health system planning.

Canada's unique contribution

Canada is fortunate to have a mature nationwide system of cancer registration that covers the entire population. A registry exists in every province and territory in Canada, and all have good quality data from at least the early 1980s.[10] Canada has a history of using its cancer registry data for monitoring the occurrence of cancer and for projecting the future burden of cancer for Canada and its regions. *Canadian Cancer Statistics*, an annual publication of data from the Canadian cancer registries, has been produced since 1987.[25] Data from all the registries combined permits the study of relatively rare events, such as cancer in young adults, not only currently but also over time. No one jurisdiction within Canada and few around the world could carry out this work for a wide range of cancer types.

Specific objectives

The primary objectives of this monograph are to:

1. identify and describe the most important forms of cancer in young men and young women aged 20–44 years in Canada in a recent decade, 1990–1999;
2. document time trends in incidence and mortality between 1983 and 1999 for these cancers and for important sub groupings thereof;
3. interpret the patterns and trends; and
4. recommend priority areas for research, surveillance and policy stemming from the results.

More than 9% of the 1.2 million cancers diagnosed in adults aged 20 and over in Canada are diagnosed in young adults aged 20–44 years. Almost two-thirds of the cancers in this age group occur among women. The higher cancer incidence rate among young adult women is attributable to the sex-specific cancers, particularly breast and cervix, that tend to occur at younger ages than other cancers and represent over 50% of all cancers in women of this age group. If cancers of the breast, cervix, ovary and uterus are excluded for women and cancer of the testis for men, the number of incident cancers and the incidence rate are slightly higher for men (33,033 and 53.8 per 100,000, respectively) than for women (31,380 and 49.3 per 100,000, respectively).

Table 1

Summary statistics, adults aged 20–44, Canada, 1990–1999

	Males	Females
Diagnoses of cancer*	38,339	62,035
Percentage of all cancers in adults aged 20 & over	6%	11%
Age-standardized incidence rate per 100,000	63.5	102.6
Deaths from cancer	10,499	13,529
Age-standardized mortality rate per 100,000	17.2	22.1
Percentage of total potential years of life† that are lost due to cancer	11%	32%
Five-year relative survival percent (Ontario) 1998–2002	72%	81%
Average annual percent change in incidence 1983–1999	-1.5%‡	0.1%
Average annual percent change in mortality 1983–1999	-1.4%	-1.3%
Most common cancer (%)	testis (14%)	breast (34%)
Most common cancer cause of death (%)	lung (15%)	breast (31%)

* Excludes basal and squamous cell skin cancers; † See *Appendix* for definition; ‡ After 1992

The male:female incidence rate ratio in this age group (0.6:1) differs from the sex ratio in other periods of life: in adults aged 45+ and in children and adolescents, there is a slight male excess.[24] The reasons for the predominance of female-specific cancers in the reproductive age range are not known, although sex hormones, which begin to exert their effects early in life, likely have a role. Young women also have more cancers of the thyroid and adrenal glands. Because of the interrelatedness of the endocrine system, it may be that endogenous sex hormones play a role for these cancers as well; there is some evidence of this for thyroid cancer.[168]

Young adulthood has been described as a "crossover" age range for cancer types. Distinct patterns emerge when incidence and mortality are examined according to whether cancers are epithelial or non-epithelial. Epithelial cancers arise in the cells lining the inside or outside of organs and the body, and account for almost 90% of cancers in adults aged 45 and older. Non-epithelial cancers arise in other types of cells, such as melanocytes, stem cells and lymphatic tissue, and account for over 90% of cancers diagnosed in childhood and adolescence. Using this morphology-based classification,[192] 37% of the tumours in young adults in Canada are non-epithelial in origin; excluding breast cancer, 47% are non-epithelial. The common non-epithelial cancers are melanoma, lymphoma (both Hodgkin and non-Hodgkin), testicular cancer,

brain cancer, the sarcomas and leukemia. The proportion of non-epithelial cancer is lower in young women (25%) than in young men (64%). The incidence rates for non-epithelial cancer are also lower in women; the higher incidence of all cancer in young women is thus entirely due to epithelial cancers.

Age-specific rates

The incidence of cancer rises with age, from about 30 per 100,000 in both men and women at ages 20–24 to 118.5 in men and 224.3 in women per 100,000 at ages 40–44 (Figure 1). The higher incidence for women in the 40–44 age group is due to the sex-specific cancers. When these are excluded, age-at-incidence curves are similar in men and women, although by ages 40–44 they are slightly higher in men. Mortality rates also increase steadily with age, reaching 42.5 and 59.0 per 100,000 in males and females, respectively.

The proportion of non-epithelial cancers decreases with age, from 76% at ages 20–24, to 26% at ages 40–44. While age-specific incidence rates for epithelial cancers increase twenty-fold between ages 20–24 and 40–44, incidence rates for non-epithelial cancers rise only slightly. The age-specific patterns are similar (on a semi-log scale, not shown) in the two sexes, although females have a much higher incidence of epithelial cancers at every age and males have slightly higher incidence of non-epithelial cancers across the age range (Figure 2).

Figure 1
All cancers
Age-specific rates for young adults
Canada, 1990-1999

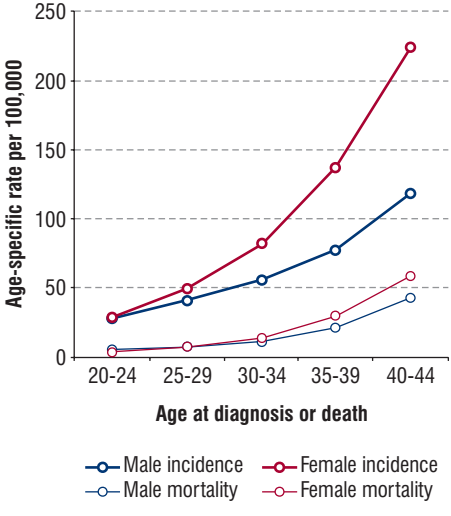
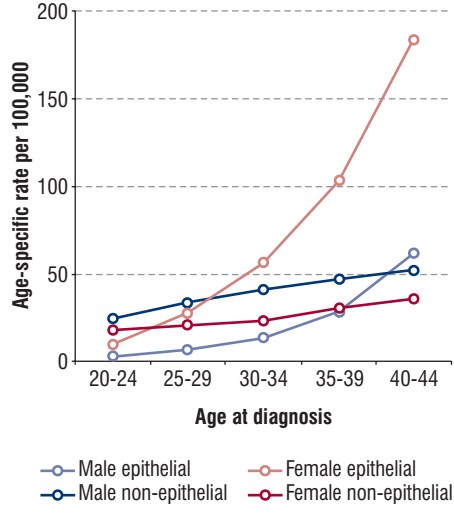


Figure 2
Epithelial and non-epithelial cancers
Age-specific incidence rates for young adults, Canada, 1990-1999

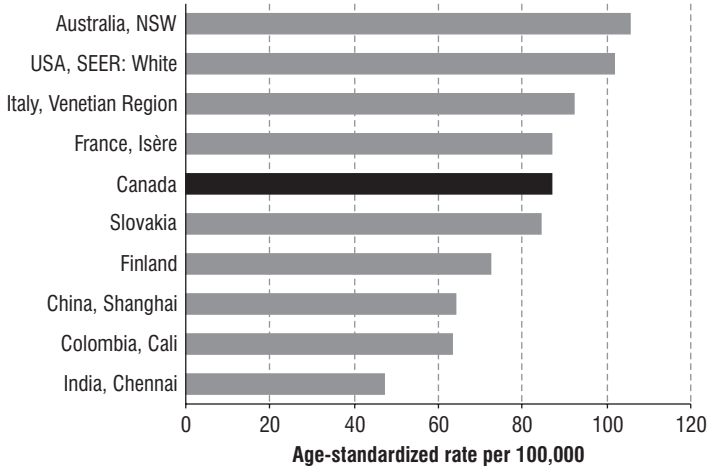


International and regional variation

Canada's incidence rate for all cancers in this age group is intermediate among nine selected international registries (Figure 3). It is similar to the incidence rates of many European countries, but substantially lower than in Australia and the US SEER registries and higher than in Shanghai, China; Cali, Colombia; and Chennai, India. Canada has a high breast cancer incidence rate typical of western developed nations; this may partly explain why our all-cancer incidence appears high when compared to Asian countries where breast cancer is less common. Possible reasons for Canada's lower incidence compared with the US and Australia include differences in risk factors, differences in uptake of screening behaviours and differences in cancer registration practices. Canadian and Australian incidence rates are similar when melanoma, which is much more common in Australia, is excluded. Excesses in several cancers, such as melanoma, testis and Kaposi sarcoma, appear to contribute to the overall higher rate in the US white SEER population.

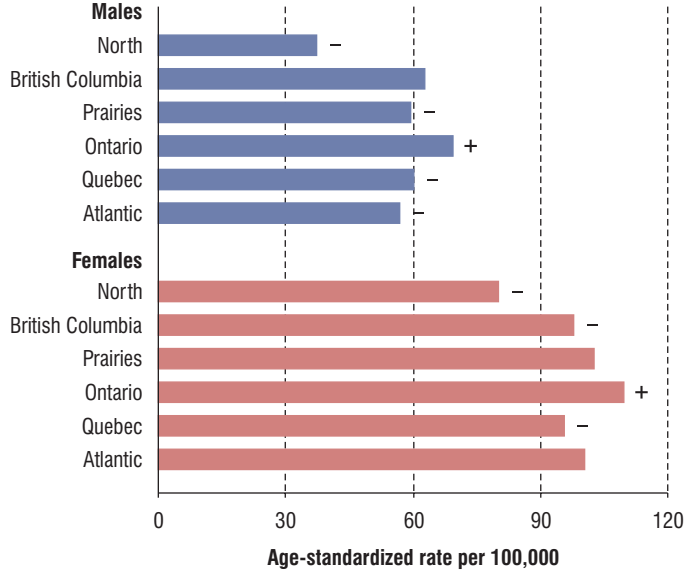
Incidence is significantly higher in Ontario than in Canada as a whole, and is significantly lower in the North (Figure 4). Within Canada, differences in registry practices that lead to differences in completeness and accuracy of data may explain some regional variation in the incidence of all cancers combined. Ontario, where incidence is significantly higher overall, has the highest percentage of cancers without microscopic confirmation. Since Ontario uses passive registration methods, some unconfirmed cancers may actually not be cancer, and their inclusion could result in artefactually high cancer incidence rates.[72] If one-half of unconfirmed cases were not truly cancer (likely an overestimate), Ontario's incidence would be only slightly elevated. Cancer incidence is lowest in the North, where 18% of the population belongs to one of the First Nation groups, who historically have experienced lower rates of cancer than the general population.[112] Another 29% are Inuit, who have similar overall rates of cancer to the non-Aboriginal population.[126]

Figure 3
All cancers
Age-standardized incidence rates* for adults aged 20-44
Selected international regions, 1993-1997



Source: Parkin et al., 2002
* Both sexes combined

Figure 4
All cancers
Age-standardized incidence rates for adults aged 20-44
Canadian regions, 1990-1999



+/– Significantly higher/lower than the Canadian rate

Most common cancers in young adults

Two criteria were used to determine which cancers should be analyzed and discussed in this report. The first criterion was based on frequency: a cancer must have had at least 1,000 cases diagnosed in Canada during the period 1990–1999 to be considered a major cancer. (See Tables A1 and A2 for "CYAC" cancer definitions.) In this decade, at least 1,000 diagnoses were registered for 18 types of cancer (14 in men; 13 in women). These cancers collectively accounted for 87% of all cancers in young men and 90% in women.

The second criterion stressed the relative importance of the 20–44 age group: 25% or more of the cases aged 20 or older at diagnosis must have been diagnosed within the 20–44 age group. Three cancers met only the second criterion and were so rare that detailed analysis was not possible: cancer of the thymus (74 males, 57 females), cancer of the adrenal gland (64 males, 81 females) and cancer of endocrine organs other than the thyroid or adrenal glands (e.g., the pituitary and pineal glands) (67 males, 53 females). Little is known about the causes of these cancers or why a relatively high percentage of them occur in this age group.

Ten cancers qualified under only the first criterion: breast, non-Hodgkin lymphoma, colorectal, lung, leukemia, ovary, kidney, bladder, uterus, and lip, oral cavity and pharynx. A further eight cancers—melanoma, thyroid, cervix, testis, Hodgkin lymphoma, brain, sarcoma and Kaposi sarcoma—qualified under both criteria. Of these eight cancers, all but cervix and thyroid are non-epithelial in origin.

The three most common cancers in young men, which account for 33% of cancers in this age group, are all non-epithelial: testis (14%, 5,306 cases between 1990 and 1999), non-Hodgkin lymphoma (NHL) (11%, 4,043 cases) and melanoma (9%, 3,399 cases) (Figure 5). In contrast, the three most common types of cancer in men aged 45 and over are prostate, lung and colorectal; these cancers are all epithelial and represent 60% of the cases diagnosed in the 1990s (Figure 6). Eighty percent of cases of testicular cancer in adult men are diagnosed before age 45.

The three most common cancers in young women, accounting for 53% of cancers in this age group, are breast cancer (34%, 21,308 cases diagnosed in the 1990s), cervical cancer (10%, 6,277 cases) and thyroid cancer (9%, 5,296 cases) (Figure 7). In women aged 45 and over, the three most common cancers occur in the breast, colon and rectum, and lung, and represent 56% of cases (Figure 8). In females, the top three cancers in both age groups are all epithelial. Nearly half of cervical and thyroid cancers in women aged 20 and over occur in the 20–44 age group.

Just over half of cancer deaths in young men are due to five cancers: lung (1,578 deaths), brain (1,312), NHL (911), leukemia (901), and colorectal cancer (796) (Figure 9). In young women, almost two-thirds of cancer deaths are accounted for by breast (4,181 deaths), lung (1,770), cervical (940), brain (772) and colorectal (729) cancers (Figure 10).

Figure 5
Most common cancer diagnoses
Males aged 20-44, 1990-1999
N=38,339

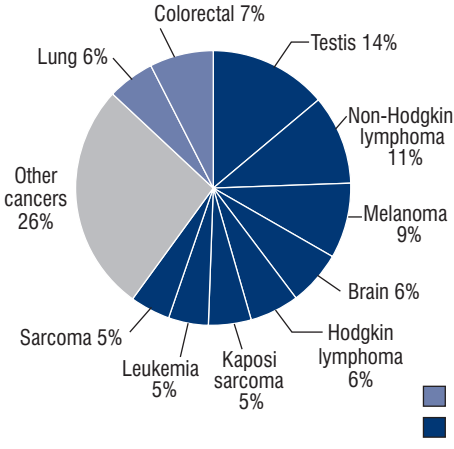


Figure 6
Most common cancer diagnoses
Males aged 45 & older, 1990-1999
N=574,630

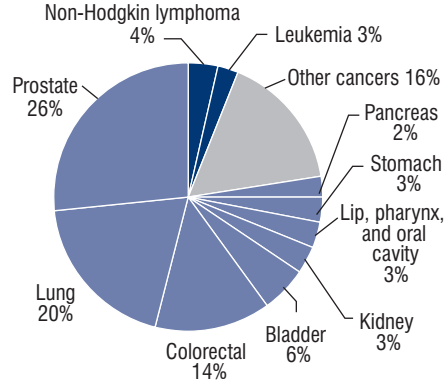


Figure 7
Most common cancer diagnoses
Females aged 20-44, 1990-1999
N=62,035

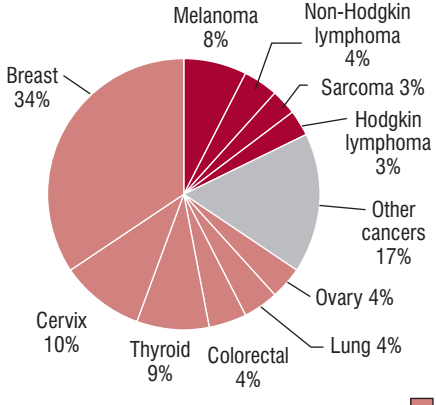


Figure 8
Most common cancer diagnoses
Females aged 45 & older, 1990-1999
N=491,527

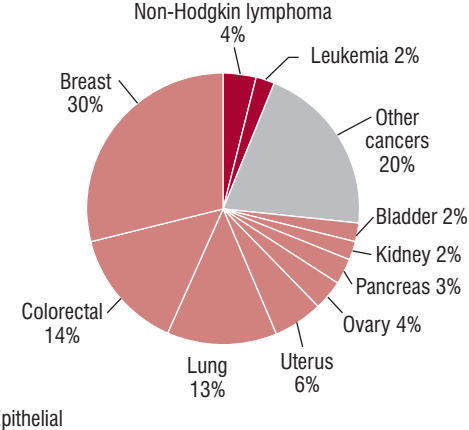


Figure 9
Most common cancer deaths
Males aged 20-44, 1990-1999
N=10,499

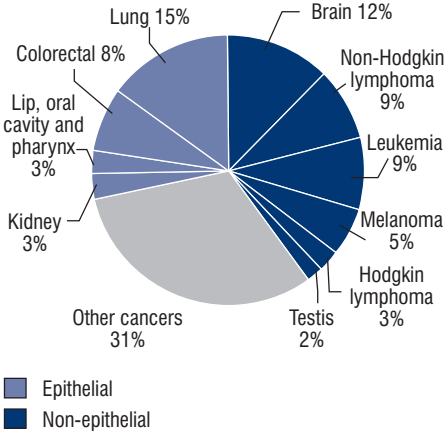
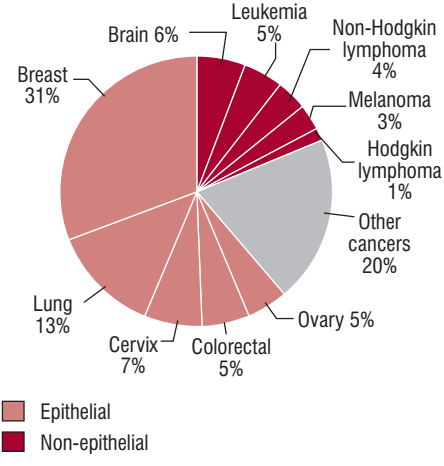


Figure 10
Most common cancer deaths
Females aged 20-44, 1990-1999
N=13,529



Time trends

Between 1983 and 1999, incidence rates were stable for young women, while mortality fell significantly by 1.3% per year (Figure 11). In young men, incidence rates increased significantly by 1.6% per year until 1992, then fell significantly by 1.5% per year. Male mortality rates declined significantly by 1.4% per year over the entire period. Recent trends in both incidence and mortality were projected to continue through 2005. If sex-specific cancers are excluded, incidence rose significantly in women (1.0% per year) and declined significantly after 1992 in men (-2.0%) (Figure 12).

Examining time trends in epithelial and non-epithelial cancers by sex confirms the existence of different patterns. Epithelial cancer incidence in young women was stable, while non-epithelial cancer incidence increased significantly by 0.5% per year (Figure 13). In young men, epithelial cancer rates fell significantly by 0.8% per year. Non-epithelial cancers in males increased significantly between 1983 and 1990 at 4.1% per year, then started a significant decline at 1.2% per year.

The trends for the different cancer groupings are influenced by the increases and decreases of individual cancers. The rise and fall of incidence rates in males seem most affected by the trends in non-Hodgkin lymphoma and testicular cancer, while the impact of thyroid cancer in females can be seen when the sex-specific cancers are removed in Figure 12. Table 2 summarizes the average annual percent change (AAPC) in incidence rates for all CYAC cancers; they are divided into epithelial and non-epithelial, and then ordered by frequency. Table 3 presents the AAPCs in mortality rates for the nine cancers in which the criterion for trend analysis, described in the *Appendix*, was met; the cancers have been ordered by frequency for both sexes combined. Figures 14 and 15 highlight the incidence trends for selected cancers for males and females respectively. Breast cancer trends, both incidence and mortality, are presented in Figure 16 to allow the scale in Figures 14, 15, 17 and 18 to better illustrate the incidence and mortality trends in less common cancers.

Figure 11
All cancers
Age-standardized rates for adults aged 20-44, Canada, 1983-2005

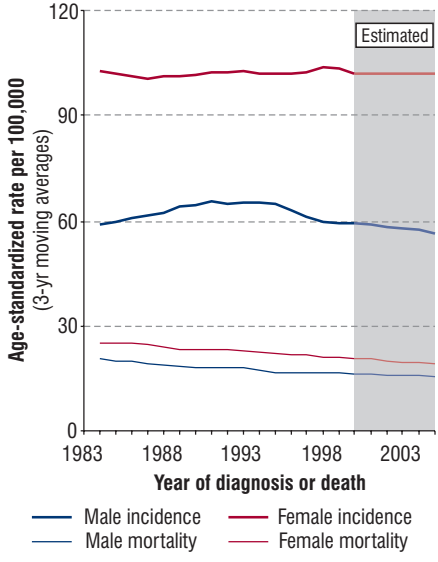


Figure 12
All except sex-specific cancers
Age-standardized rates for adults aged 20-44, Canada, 1983-2005

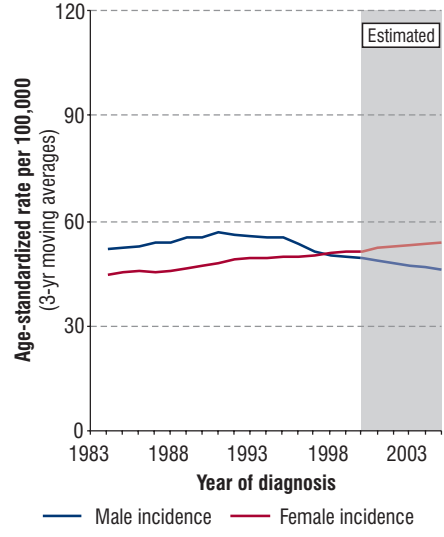


Figure 13
Epithelial and non-epithelial cancers
Age-standardized incidence rates for adults aged 20-44, Canada, 1983-2005

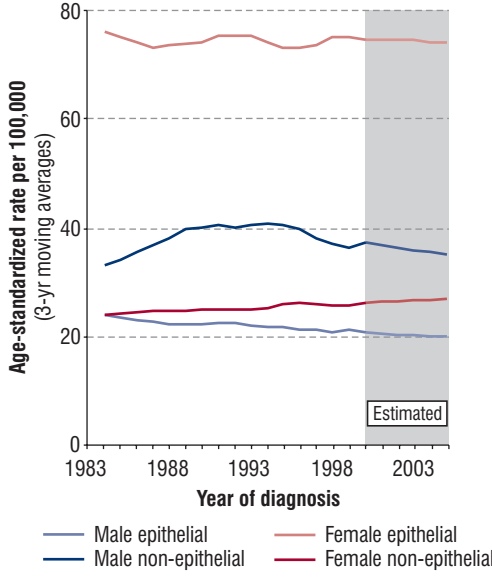


Figure 14
Incidence for selected cancers
Age-standardized rates for males aged 20-44
Canada, 1983-2005

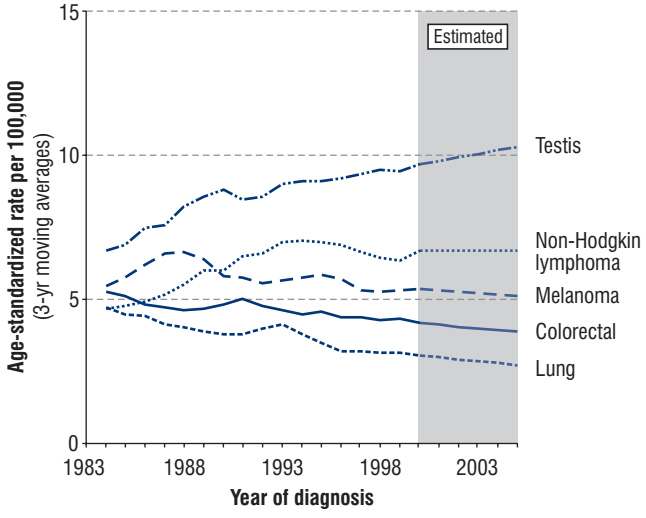
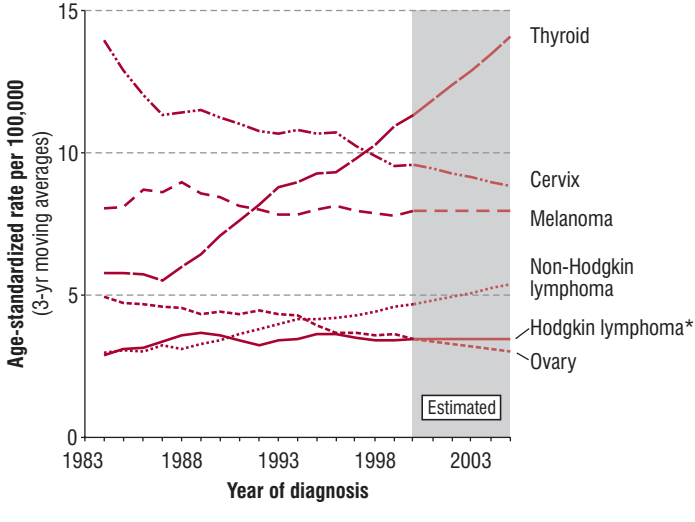


Figure 15
Incidence for selected cancers
Age-standardized rates for females aged 20-44
Canada, 1983-2005



* Ages 15-44

Table 2

Average annual percent change (AAPC) in age-standardized incidence rates and 95% confidence intervals (CI), CYAC cancers, ages 20-44, 1983–1999

	Males			Females		
Cancer	**Period**	**AAPC %**	**95% CI**	**Period**	**AAPC %**	**95% CI**
All cancers	1983–1992	1.6	(1.0, 2.2)	1983–1999	0.1	(-0.0, 0.3)
	1992–1999	-1.5	(-2.4, -0.7)			
All except sex-specific*	1983–1992	1.3	(0.5, 2.1)	1983–1999	1.0	(0.8, 1.2)
	1992–1999	-2.0	(-3.0, -0.9)			
Non-epithelial	1983–1990	4.1	(2.6, 5.7)	1983–1999	0.5	(0.3, 0.7)
	1990–1999	-1.2	(-2.1, -0.3)			
Non–Hodgkin lymphoma	1983–1994	4.9	(3.3, 6.5)	1983–1999	2.9	(2.1, 3.7)
	1994–1999	-3.3	(-7.4, 0.9)			
Hodgkin lymphoma†	1983–1999	-0.3	(-1.1, 0.5)	1983–1999	1.0	(0.1, 1.9)
Melanoma	1983–1987	6.4	(-1.4, 14.7)	1983–1999	-0.4	(-1.1, 0.3)
	1987–1999	-1.8	(-3.0, -0.6)			
Sarcoma	1983–1999	0.4	(-0.4, 1.3)	1983–1999	0.6	(-0.5, 1.7)
Kaposi sarcoma	1983–1985	135.4	(-2.4, 467.8)	Rates too low to interpret		
	1985–1990	21.7	(9.6, 35.2)			
	1990–1995	-0.5	(-7.8, 7.5)			
	1995–1999	-43.6	(-51.8, -34.1)			
Testis	1983–1999	2.2	(1.4, 3.0)	–	–	
Brain	1983–1999	0.2	(-0.5, 0.9)	1983–1999	0.5	(-0.2, 1.2)
Leukemia	1983–1999	-0.1	(-1.2, 0.9)	1983–1999	-0.7	(-1.8, 0.5)
Epithelial	1983–1999	-0.8	(-1.1, -0.5)	1983–1999	-0.0	(-0.2, 0.2)
Breast‡	–	–		1983–1999	0.3	(0.0, 0.5)
Thyroid	1983–1999	2.9	(2.0, 3.9)	1983–1999	4.9	(4.1, 5.6)
Cervix	–	–		1983–1986	-7.0	(-11.8, -2.1)
				1986–1999	-1.4	(-2.0, -0.8)
Colorectal	1983–1999	-1.2	(-1.8, -0.5)	1983–1999	-1.3	(-2.1, -0.3)
Lung	1983–1999	-2.6	(-3.5, -1.8)	1983–1999	0.7	(-0.0, 1.5)
Ovary	–	–		1983–1999	-2.0	(-2.8, -1.3)
Kidney	1983–1999	1.4	(0.2, 2.5)	1983–1989	9.2	(2.8, 15.9)
				1989–1999	-2.1	(-4.2, 0.0)
Lip, oral cavity and pharynx	1983–1999	-0.3	(-1.3, 0.7)	1983–1999	1.5	(0.0, 3.1)
Bladder§	1990–1999	-3.6	(-5.4, -1.8)	1990–1999	-3.4	(-5.3, -1.5)
Body of uterus	–	–		1983–1999	-1.3	(-2.3, -0.4)

* Excludes cancers of the testis, female breast, cervix, ovary and body of uterus
† Ages 15-44
‡ Ages 20-49
§ For bladder cancer, period 1990–1999 was used because earlier data included non-invasive papillomas

Table 3

Average annual percent change (AAPC) in age-standardized mortality rates and 95% confidence intervals (CI), CYAC cancers, ages 20-44, 1983–1999

	Males			Females		
Cancer	Period	AAPC %	95% CI	Period	AAPC %	95% CI
All cancers	1983–1999	-1.4	(-1.7, -1.1)	1983–1999	-1.3	(-1.5, -1.0)
Breast*	–	–		1983–1986	3.5	(-2.6, 9.9)
				1986–1995	-1.9	(-3.0, -0.7)
				1995–1999	-5.3	(-8.5, -1.9)
Lung	1983–1999	-2.8	(-3.7, -1.9)	1983–1999	1.1	(0.4, 1.8)
Brain	1983–1999	-0.3	(-1.0, 0.5)	1983–1999	-0.2	(-1.3, 0.8)
Leukemia	1983–1995	-4.6	(-6.2, -3.0)	1983–1999	-3.8	(-5.1, -2.4)
	1995–1999	6.1	(-3.4, 16.6)			
Colorectal	1983–1999	-3.0	(-4.1, -1.9)	1983–1999	-2.5	(-3.8, -1.1)
Non-Hodgkin lymphoma	1983–1999	0.4	(-1.0, 1.7)	1983–1999	0.4	(-1.5, 2.4)
Melanoma	1983–1999	-2.2	(-3.2, -1.1)	Rates too low to interpret		
Cervix	–	–		1983–1999	-1.3	(-2.4, -0.2)
Ovary	–	–		1983–1999	-1.8	(-3.2, -0.3)

* Ages 20-49

Figure 16
Breast cancer
Age-standardized rates for females aged 20-49, Canada, 1983-2005

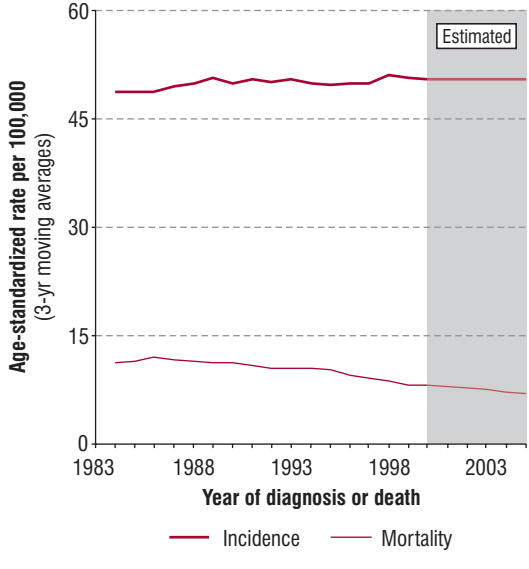


Figure 17
Mortality for selected cancers
Age-standardized rates for males aged 20-44
Canada, 1983-2005

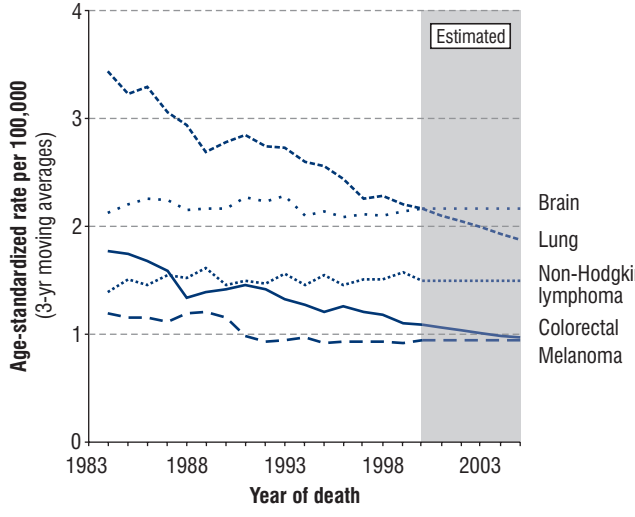
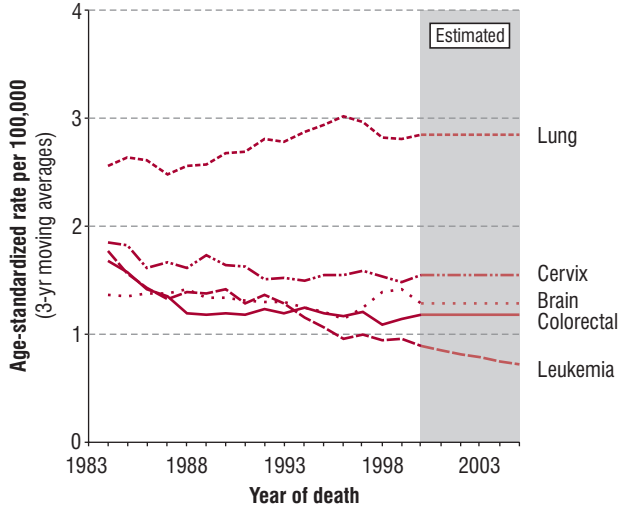


Figure 18
Mortality for selected cancers
Age-standardized rates for females aged 20-44
Canada, 1983-2005

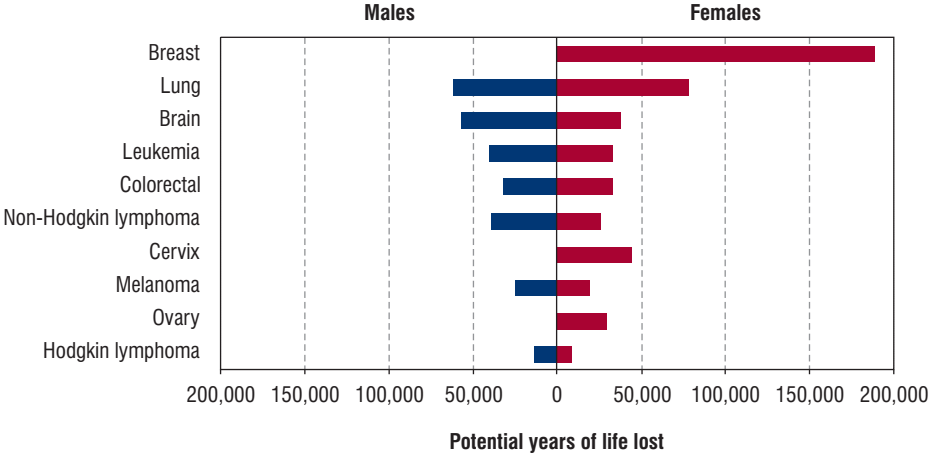


Potential years of life lost and survival

Cancer is the main contributor to early death among young women, accounting for nearly one-third (32%) of the potential years of life lost (PYLL). In men, cancer ranks third after accidental deaths and suicides, accounting for 11% of PYLL.

Even when both sexes are combined, the potential years of life lost are greatest for female breast cancer (190,000 years for women diagnosed in the 1990s) (Figure 19). Lung cancer, brain cancer, leukemia, colorectal cancer and NHL each account for more than 60,000 estimated lost years.

Figure 19
Cancers with 20,000 or more potential years of life lost
Adults aged 20-44, Canada, 1990-1999



The differences between the rankings for cancer deaths or PYLL and the rankings for cancer diagnoses can be partially explained by differences in survival. Five-year relative survival in Ontario, used as a surrogate for Canada (see *Appendix*), is 72% for young men and 81% for young women for all cancers combined. The poorer survival for young men persists when sex-specific cancers are excluded (67% and 79% for men and women, respectively).

Lung cancer survival is poor, with 5-year relative survival estimates of 23% and 29% in Ontario men and women, respectively (Table 4). Survival estimates for brain and colorectal cancers and leukemia vary, but are all between 50 and 65%. Some common cancers such as testis and thyroid have excellent survival (>95% 5-year relative survival) and others such as breast, cervix and kidney cancers have intermediate survival around 80%.

While survival for all non-epithelial cancers combined is better than for epithelial cancers, particularly for males, both groups include cancers with good (testis and body of uterus) and poor (Kaposi sarcoma and lung) survival.

Table 4

Five-year relative survival* (%) and 95% confidence intervals (CI), CYAC cancers, ages 20–44

Cancer	Males	Females
Non-epithelial	77 (76, 79)	82 (81, 83)
Non-Hodgkin lymphoma	75 (71, 79)	83 (80, 87)
Hodgkin lymphoma	91 (88, 93)	94 (92, 96)
Melanoma	86 (84, 89)	95 (94, 97)
Sarcoma	66 (61, 72)	74 (70, 79)
Kaposi sarcoma	45 (32, 59)	
Testis	97 (96, 98)	
Brain	53 (48, 57)	63 (58, 68)
Leukemia	54 (49, 59)	59 (53, 64)
Epithelial	63 (61, 65)	80 (79, 81)
Breast		83 (82, 85)
Thyroid	98 (97,100)	100
Cervix		86 (84, 88)
Colorectal	64 (60, 68)	65 (61, 69)
Lung	23 (18, 27)	29 (25, 33)
Ovary		69 (65, 73)
Kidney	80 (75, 84)	89 (84, 93)
Lip, oral cavity and pharynx	79 (75, 84)	75 (69, 82)
Bladder	80 (73, 86)	82 (73, 91)
Body of uterus		94 (91, 97)

Source: Ontario Cancer Registry, 2004

* Period method, 1998–2002 (see *Appendix*)

Cancer chapters

Detailed information is provided about each of the 18 CYAC cancers in the cancer chapters. The order of these chapters is the same as Tables 2 and 4: divided into non-epithelial and epithelial cancer, then ranked by incidence frequency. This arrangement attempts to provide a different structure for emphasizing the most important cancers, while also trying to stimulate the discovery of new patterns and the generation of etiologic hypotheses for future research.

> "…you can never imagine it happening to yourself …but, it's amazing how much strength you can pull out that you didn't know you ever had." ***Male, Osteosarcoma, 26***

LYMPHOMA

Lymphoma encompasses the cancers of the lymphatic system. When the two categories of lymphoma—non-Hodgkin lymphoma (NHL) and Hodgkin lymphoma—are combined, they are the most common non-epithelial cancer in young adults. To emphasize their importance and common features, these two cancers have been placed together; since they also have distinctly different patterns, they have been analyzed and discussed separately.

Non-Hodgkin lymphoma is the fourth most common cancer in young adults and ranks sixth in terms of cancer mortality. It is slightly more prominent in this age group (6% of all cancers) than among all ages combined (4%).[25] Hodgkin lymphoma is the ninth most common cancer in young adults and ranks twelfth in terms of mortality. It is more common in young adults (4%) than for all ages combined (< 1%).[25] Survival from this cancer is very good and mortality low.

Non-Hodgkin lymphoma

Age-specific rates

For both males and females, a constant rate of increase is observed, with the incidence rate in each age group 1.4 times the rate in the previous age group. From the youngest to the oldest age group, incidence more than quadruples for both sexes. The age-specific mortality pattern is very similar, with an increase of 1.5 times with each successive age group, and a 4.4-fold increase from the youngest to the oldest age group.

Non-Hodgkin lymphoma
Summary statistics, 1990-1999

	Males	Females
Cases	4,043	2,449
Incidence rate	6.7	4.1
% of all cancers	11%	4%
Incidence rank	2nd	7th
Deaths	911	529
Mortality rate	1.5	0.9
5-yr survival	75%	83%
PYLL rank	4th	8th

Geographic variation

Worldwide, rates are high in the US, Italy and Canada, intermediate for most other European countries, and lowest in China and India.[134] Canada ranks third among the ten countries chosen for comparison. Within Canada, NHL incidence rates in males are highest in Ontario, followed by Quebec and British Columbia. For females, incidence is high in Ontario only.

Trends

Incidence rates are considerably higher in young men than young women for the period examined here. NHL incidence (all ages combined) has been increasing over the past 30 years, especially in males.[25] Between the early 1980s and 1990s incidence among young adult males rose significantly at 4.9% per year from 4.2 per 100,000 in 1983 to a peak of 7.2 in 1994. Thereafter, incidence fell non-significantly. Female incidence did not follow these patterns, as rates continued to rise significantly between 1983 and 1999 at an overall rate of 2.9% per year. While incidence rates in males were projected to stabilize between 2000 and 2005, they were expected to continue to rise in females through 2005. These short-term projections were based on 1990–1999 data. Mortality was stable over the 1980s and 1990s for both sexes, with rates below 2.0 per 100,000. These trends were projected to continue through 2005.

Subgroups

AIDS accounts for a small proportion of NHL, particularly among males. To assess the impact of the AIDS epidemic on incidence patterns of AIDS-defining lymphomas, immunoblastic and Burkitt lymphomas were examined. Morphology information for Quebec cases was not specific enough for inclusion in subgroup analyses. Among males aged 20–44, rates of these lymphomas rose between the 1980s and mid 1990s, then fell in the late 1990s (years were combined due to small numbers). Central nervous system lymphomas could not be analyzed because they were not consistently coded through the entire time period. Not surprising, high-grade NHL, which includes AIDS-defining immunoblastic and Burkitt lymphomas, increased significantly among young adult males by 14.4% per year until 1992 and then fell significantly by 10.1% per year. Incidence rates for these subgroups in females were too low to interpret.

Trends also varied by subgroup within the standard classification scheme, the Working Formulation.[60, 122] Most NHLs are diffuse (38%) followed by follicular (24%), not specified (14%) and high-grade (11%). Diffuse lymphoma incidence increased significantly in both males (3.3% per year from 1.5 per 100,000 in 1983 to 2.4 in 1999) and females (3.6% per year from 1.2 per 100,000 in 1983 to 2.0 in 1999). Follicular NHL incidence in females increased significantly at 1.8% per year, but was stable in males.

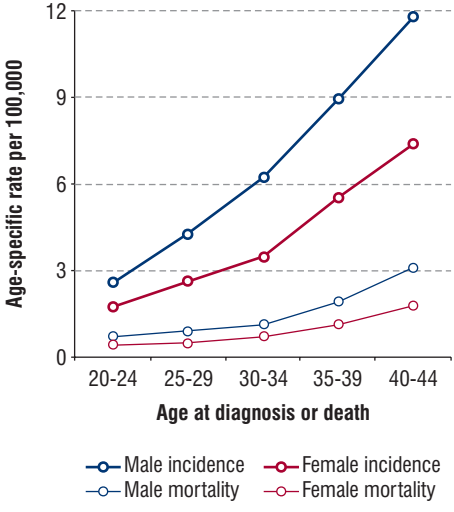
Interpretation of patterns and trends

The rise and fall in NHL incidence among young men mirrors the AIDS epidemic that began with a sudden rise in AIDS cases in 1981 and was followed by a decline in the mid 1990s to levels of the pre-AIDS era—a pattern seen also in young men in the US.[32, 45] In Canada, the numbers of AIDS cases diagnosed in young men between 1991 and 1999 also show the characteristic decline in the mid 1990s (data prior to 1991 were not available for this age group).[65] The overall increase in NHL cases in Canada since the 1970s in both sexes is, however, difficult to explain because the major risk factor, immunosuppression (related to drugs, disease, viruses, etc.), accounts for only a small fraction of cases, and information on other potential risk factors is limited.[153]

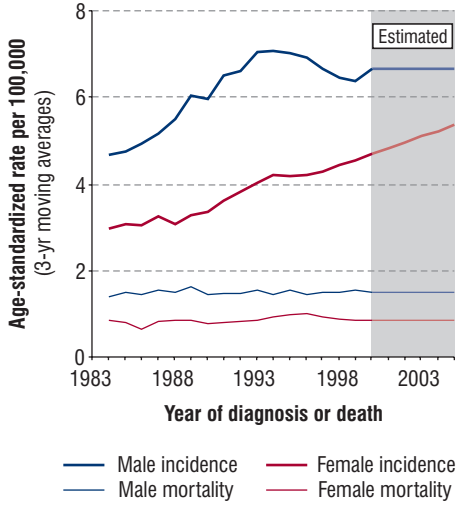
Provincial differences in male rates may relate to differences in the numbers of homosexual young adult males who are considered at high risk for AIDS.[66]

> "…since I was in the hospital for so long I was missing out on a whole bunch of school work and [I was] really worried that I was not going to be able to finish the courses… I ended up dropping one of my courses because I was too far behind… I had to stay in school [full time] because otherwise the insurance wouldn't cover me…."
>
> ***Male, Hodgkin lymphoma, 20***

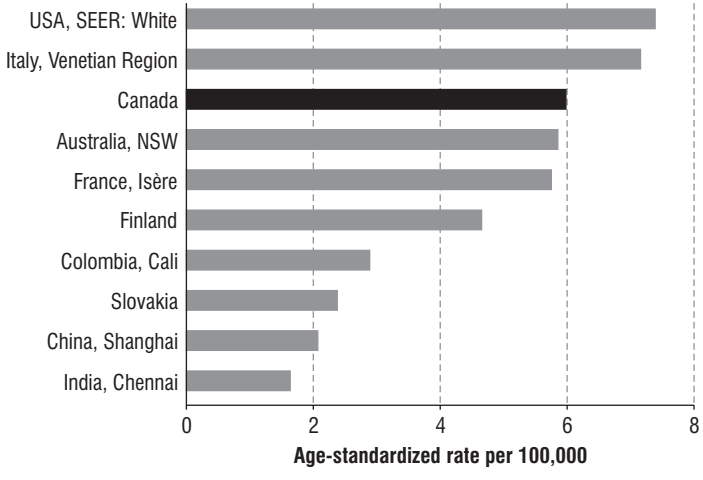
Non-Hodgkin lymphoma
Age-specific rates for young adults
Canada, 1990–1999



Non-Hodgkin lymphoma
Age-standardized rates for adults
aged 20–44, Canada, 1983–2005

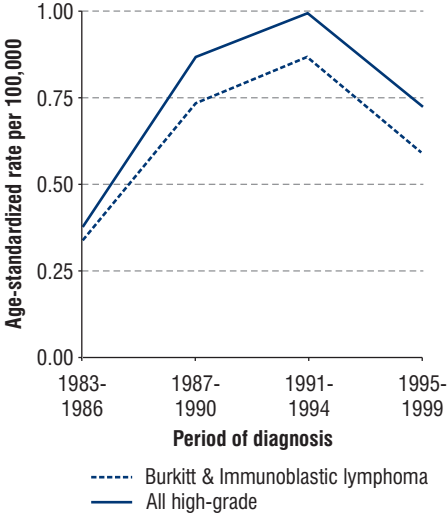


Non-Hodgkin lymphoma
Age-standardized incidence rates* for adults aged 20–44
Selected international regions, 1993–1997



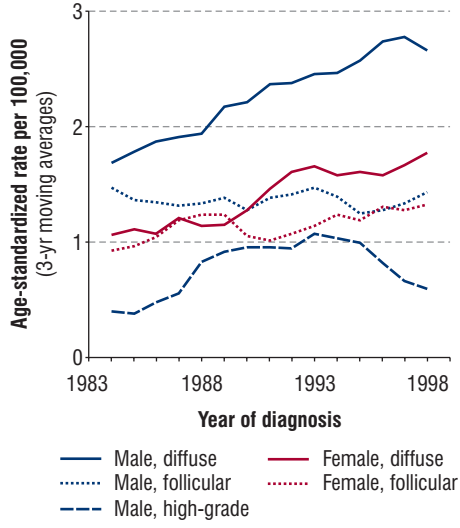
Source: Parkin et al., 2002
* Both sexes combined

High-grade lymphoma
Age-standardized incidence rates for males aged 20–44, Canada*, 1983–1999



* Quebec excluded as morphologic data were largely incomplete

Non-Hodgkin lymphoma morphologic subgroups (Working Formulation)
Age-standardized incidence rates for adults aged 20–44, Canada*, 1983–1999



* Quebec excluded as morphologic data were largely incomplete

Hodgkin lymphoma

Age-specific rates

Hodgkin lymphoma is unusual among cancers in displaying a bimodal age-incidence curve in industrialized countries, with the first peak occurring in the 20–24 year age group and the second in old age.[105] In adolescence, incidence is higher for females than males; after the peak in incidence, male rates remain higher than female rates even though both decline with increasing age.[84]

Hodgkin lymphoma
Summary statistics, 1990-1999

	Males	Females
Cases	2,301	1,847
Incidence rate	3.9	3.3
% of all cancers	6%	3%
Incidence rank	6th	10th
Deaths	288	175
Mortality rate	0.5	0.3
5-yr survival	91%	94%
PYLL rank	7th	10th

Geographic variation

In the 15–44 age group, incidence rates are high in Italy, the US and Canada and other major populations of Europe, and low in India and China.[134] Canada ranks third among the ten countries chosen for comparison. Throughout the 1990s, rates in Canada were significantly high in Ontario, and low in the Prairies (males only) and British Columbia compared to the rest of the country.

Trends

Hodgkin lymphoma incidence rates for ages 15–44 remained stable in males throughout the 1980s and 1990s, at 4–5 per 100,000. Female rates increased significantly by 1.0% per year over the two decades, with rates ranging from 2.6 per 100,000 in 1983 to 3.5 per 100,000 in 1999. Incidence rates in males were projected to decline between 2000 and 2005 while female rates were expected to stabilize. These short-term projections were based on 1990–1999 data.

Subgroups

Hodgkin lymphoma is classified into four categories based on morphological features. The most common subgroups are nodular sclerosis, representing 76% of all Hodgkin lymphomas in this age group, followed by the mixed cellularity subgroup at 13%. The remaining two subgroups, lymphocyte predominant and lymphocyte depletion, are infrequent, comprising 5% of all Hodgkin lymphomas. Nodular sclerosis incidence is essentially unimodal and represents the age 20–24 peak seen in developed countries.[84] The mixed cellularity subgroup, which has been described as bimodal with the highest incidence in the oldest age groups, is more common in developing nations.[117] Nodular sclerosis incidence is higher for females than males under about age 25 and similar to males from age 25 to 44.

The incidence of nodular sclerosis increased dramatically and significantly in males and females during the 1980s (10.7% and 18.1% per year, respectively), while during the 1990s the rate of increase was much less, non-significant and similar for both sexes. Incidence of the mixed cellularity subgroup, which was much lower than for nodular sclerosis, fell significantly at 5.0% per year over the two decades for males. Data from the US also show dramatic increases in nodular sclerosis and decreases in the mixed cellularity subgroup from the 1970s to the 1980s.[117]

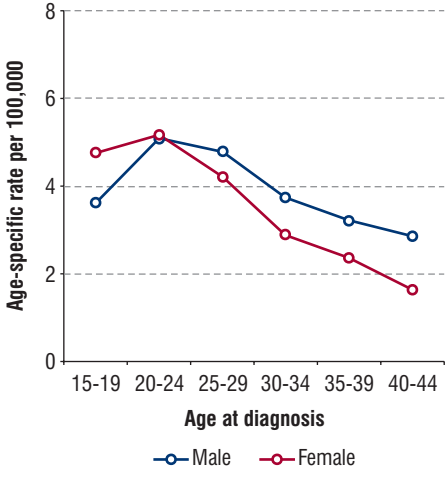
Morphology information for Quebec cases was not specific enough for inclusion in subgroup analyses.

Interpretation of patterns and trends

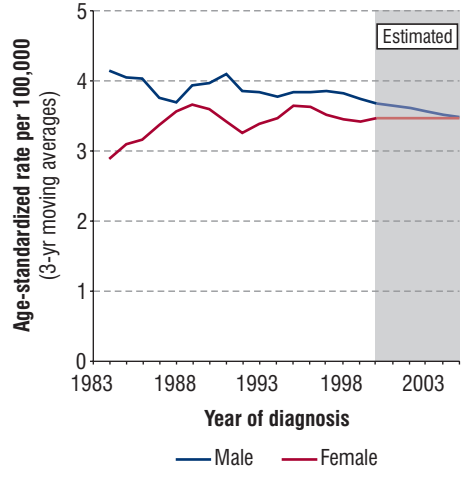
Increases in Hodgkin lymphoma incidence have been reported among young adults in several industrialized countries and are primarily explained by marked increases in the nodular sclerosis type.[117]

The reasons for the high incidence in Ontario and low incidence in the Prairies (males only) and British Columbia remain unknown. Incidence patterns worldwide appear to vary in relation to economic development.[109, 84] In developed countries Hodgkin lymphoma is more common in young adults and is primarily of the nodular sclerosis subgroup, which is likely related to a delayed infection. The major viral candidate has been Epstein-Barr virus, although it is found less often in young adults than in the extreme age groups. Incidence rates in Canada and the US are among the highest in the world. Intermediate rates are seen in populations moving towards greater economic development—rural regions undergoing urbanization.[121] Hodgkin lymphoma in less economically advantaged populations typically displays a peak in childhood with a late peak in old age and is predominantly of the mixed cellularity subgroup. The bimodality of histological subgroups is consistent with the hypothesis that the etiology of Hodgkin lymphoma in young adults differs from that of children and older adults.[117]

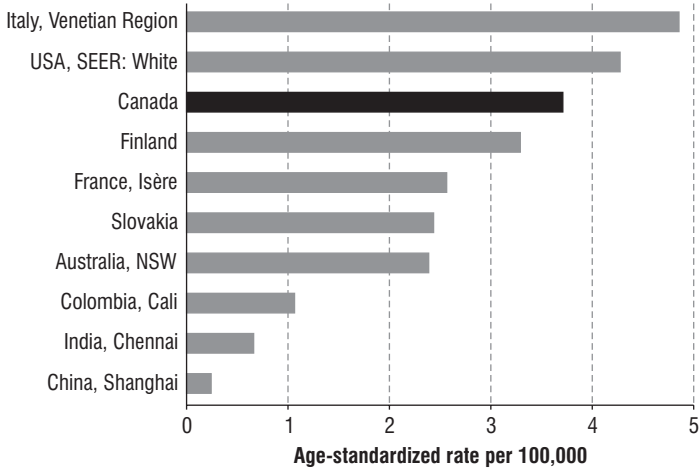
Hodgkin lymphoma
Age-specific incidence rates for young adults, Canada, 1990–1999



Hodgkin lymphoma
Age-standardized incidence rates for adults aged 15–44, Canada, 1983–2005

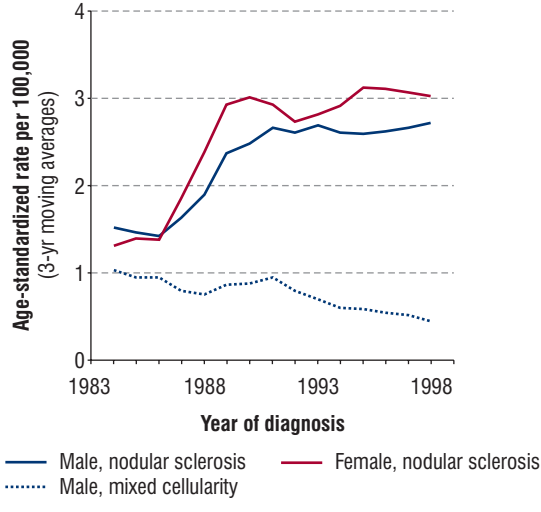


Hodgkin lymphoma
Age-standardized incidence rates* for adults aged 15–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Both sexes combined

Hodgkin lymphoma morphologic subgroups
Age-standardized incidence rates for adults aged 15–44
Canada*, 1983–1999



* Quebec excluded as morphologic data were largely incomplete

Melanoma of the skin is the second most common cancer in young adults and ranks eighth in terms of cancer mortality. It accounts for 8% of cancers in this age group compared to 3% for all ages combined.[25]

Melanoma
Summary statistics, 1990-1999

	Males	Females
Cases	3,399	4,724
Incidence rate	5.6	7.9
% of all cancers	9%	8%
Incidence rank	3rd	4th
Deaths	577	408
Mortality rate	0.9	0.7
5-yr survival	86%	95%
PYLL rank	6th	9th

Age-specific rates

Incidence rates are higher and mortality rates lower in females compared to males in each 5-year age group. Among females, age-specific incidence rises 1.3 times with each successive age group and incidence among males rises similarly. The result is an approximate 6-fold increase in incidence for males between the youngest and oldest age groups, and a tripling in incidence for females. Mortality rises by 1.7 times in each age group for an overall 11-fold increase in both sexes.

Geographic variation

Incidence of melanoma varies dramatically worldwide. Incidence is lowest in Asians, US blacks (0.2 per 100,000) and Latin Americans and highest in fair-skinned populations living near the equator, such as Australians and New Zealanders (25.3 per 100,000). Canada is intermediate, along with European countries.[134] Canada ranks fourth among the ten countries chosen for comparison. Although Quebec has much lower incidence than other provinces, this may be due in large part to incomplete registration of melanoma; Brisson et al.[19] estimated that 35% of melanomas are not reported to the Quebec tumour registry.

Trends

After increasing non-significantly between 1983 and 1987, the incidence of melanoma in young men fell significantly by an average of 1.8% per year. Incidence was stable in females from 1983 to 1999. Prior to 1983, large annual increases in melanoma incidence and mortality (all ages combined) had been noted in Canada for both males and females.[22] Incidence was projected to continue to fall slightly in males and remain stable in females. Mortality fell significantly for males between 1983 and 1999 by 2.2% per year and was projected to stabilize through 2005. Mortality rates in females were too low to analyze.

Subgroups

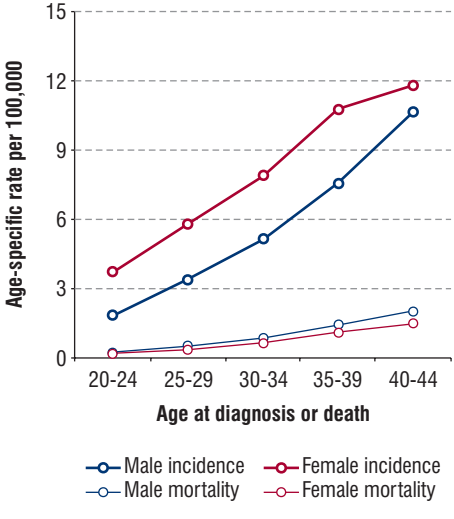
The most informative subgroupings of melanoma relate to body site. Melanoma occurs most often on the trunk (back and chest, not including shoulders) in men and on the legs in women. Nearly half of the melanomas in young men occur on the trunk, compared to 29% in women; 37% of melanomas in young women are on the legs, compared to 15% in young men. Twelve percent of melanomas in men and 7% in women are on the face, head and neck.

Interpretation of patterns and trends

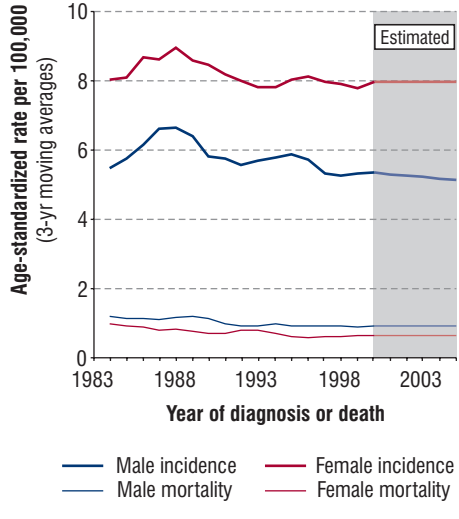
The long-term rise in incidence that preceded the study period is presumed related to increased sun exposure, probably due to both more time outdoors and less use of protective clothing, that occurred with successive cohorts of people born in the 1920s, 1930s and 1940s. Some of the increase may also be due to a shift in *type* of exposure from more continuous to more intermittent, as the number of people with outdoor jobs declined and outdoor leisure time and sunny winter vacations became more common. The subsequent stabilization or decline in incidence may reflect improved sun protection of later-born cohorts, perhaps beginning in early life. Since there are no sun behaviour data available in Canada prior to 1996, this is speculation. Sunscreens have been widely available since the late 1960s or 1970s but there is no evidence that their use reduced the risk of melanoma.[81] Improved sunscreen formulations with higher SPF and broad spectrum (UVA and UVB) protection have not been in use for long enough to determine their effect on melanoma risk. Since sunlamps and sunbeds have been commonly used only in recent years, it is likely that any major impact they may have on incidence rates has not yet been seen. Although ultraviolet (UV) radiation exposure from sunlamps and sunbeds accounts for only a small proportion of total exposure,[40] use tends to be concentrated in young people (adolescents and those in their 20s),[144, 54] so that any effect on melanoma incidence would be expected to be seen first in the young adult age group.

The international and regional variation in melanoma incidence reflects differences in amount of UV radiation exposure and between-population variation in pigmentation. The former explains why rates are much higher in Australia compared to the UK and the latter explains why New Zealand Maoris have much lower incidence than New Zealand non-Maoris, for example.

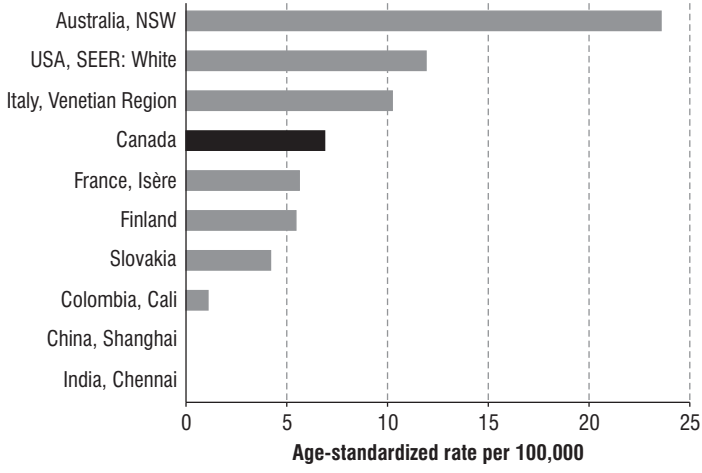
Melanoma
Age-specific rates for young adults
Canada, 1990–1999



Melanoma
Age-standardized rates for adults
aged 20–44, Canada, 1983–2005



Melanoma
Age-standardized incidence rates* for adults aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Both sexes combined

Because of its unique features and high incidence among young men for the period examined here, Kaposi sarcoma has been considered separately. If it were combined with the other sarcomas, they would be the third most common non-epithelial cancer in young adults. To emphasize their importance, these two cancers have been placed together.

Sarcoma (except Kaposi sarcoma)

Sarcomas are rare neoplasms of muscle, bone, and other connective tissue. They include a wide range of morphologies and anatomic sites; Table A2 in the *Appendix* lists the ICD-O-2 morphologic codes used to define sarcoma for this analysis. Sarcoma ranks eleventh in terms of incidence among young adults. Since sarcoma is defined morphologically, mortality data are not available; its mortality rank is estimated to be seventh.

Sarcoma
Summary statistics, 1990-1999

	Males	Females
Cases	1,742	1,990
Incidence rate	2.9	3.4
% of all cancers	5%	3%
Incidence rank	10th	9th
Deaths	-	-
Mortality rate	-	-
5-yr survival	66%	74%
PYLL rank	-	-

Age-specific rates

Age-specific incidence is similar for both sexes, increasing from one age group to the next by a factor of 1.1 in men and 1.3 in women. From the youngest age group to the oldest, incidence increases 1.5-fold in men and 2.5-fold in women.

Geographic variation

International data are not available for this morphologically defined group of malignancies.

Trends

Sarcoma incidence for ages 20 through 44 was stable for males between 1983 and 1999, and rose slightly (but not significantly) for females. Rates for both sexes are around 3 per 100,000, with female rates slightly higher because of breast and uterine sarcomas. Incidence was projected to be stable through to 2005 for both sexes.

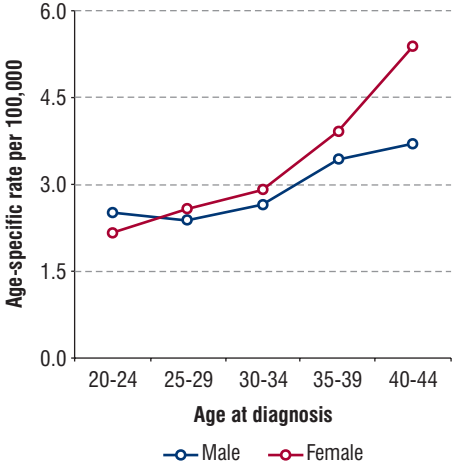
Subgroups

Approximately one-quarter of sarcomas are fibromatous tumours, mostly dermatofibromas of the skin. Some sarcoma subgroups occur either more frequently or exclusively among women. Myomatous (muscle) tumours are slightly more common among females largely because of uterine leiomyosarcoma; otherwise they occur mostly in the anatomic category "connective, subcutaneous and other soft tissues" (ICD-O C49) and in the digestive system. This monograph has followed some authors in grouping malignant phyllodes tumours of breast with sarcomas.[127, 1] Osseous and chondromatous and Ewing's sarcoma, mostly in bones and joints, account for a higher proportion of sarcomas in males than females for both young adults and other age groups.[195] Lipomatous tumours, soft tissue sarcomas not otherwise specified, and "other sarcomas" are mostly in connective, subcutaneous and other soft tissues. Most (approximately 60%) of sarcomas in bones and joints, connective tissue and specified skin locations occur in the limbs.

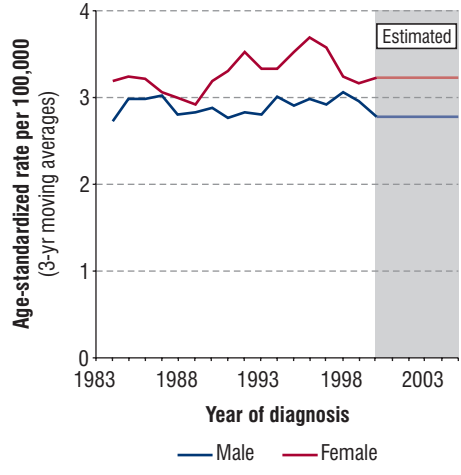
Interpretation of patterns and trends

Sarcomas are a heterogeneous group of cancers. Comparison of the periods 1983–1989 and 1990–1999 showed little change in the proportions of the various types over time (data not shown). Much of the higher incidence at ages 40–44 in females is because of an increase in uterine sarcomas toward the upper end of this age range.

Sarcoma
Age-specific incidence rates for young adults, Canada, 1990–1999



Sarcoma
Age-standardized incidence rates for adults aged 20–44, Canada, 1983–2005



Distribution of sarcoma morphologic subgroups for adults aged 20-44, Canada, 1990-1999

	Females (%)	Males (%)
Fibromatous (881-883)	23	28
Myomatous (889-892)	17	12
Osseous and chondromatous (918-924), Ewing's (92603)	14	21
Lipomatous (885-888)	8	10
Endometrial stromal (89303)	7	
Malignant phyllodes tumour (breast) (90203)	6	
Soft tissue tumours and sarcomas not otherwise specified (880)	5	7
Other sarcomas	20	23

KAPOSI SARCOMA

Kaposi sarcoma was the eighth most common cancer among young males for the period examined here. Few cases of Kaposi sarcoma occur in females and, consequently, incidence rates could not be estimated. Since Kaposi sarcoma is defined morphologically, data on the number of deaths are not available; mortality estimates place it ninth as a cancer cause of death in young adults.

Kaposi sarcoma
Summary statistics, 1990-1999

	Males	Females
Cases	1,915	59
Incidence rate	3.2	-
% of all cancers	5%	0.1%
Incidence rank	8th	17th
Deaths	-	-
Mortality rate	-	-
5-yr survival	45%	-
PYLL rank	-	-

Age-specific rates

Kaposi sarcoma is rare among men in their early 20s. Incidence then rises steeply, peaks at 4.5 per 100,000 in the 35–39 age group and falls thereafter.

Geographic variation

The frequency of this cancer varies dramatically worldwide. The highest reported rates in males are in Africans of Zimbabwe (90.2 per 100,000). Rates are also high in San Francisco, CA (29.7 per 100,000; one of the SEER registries included in the US white rates), but much lower in Australia and Canada. Asian and most European countries have the lowest reported rates.[134] However, rates in Canada are third among rates in the ten countries selected for comparison. Compared to the overall incidence rate in Canada, rates in British Columbia, Quebec and Ontario are significantly higher.

Trends

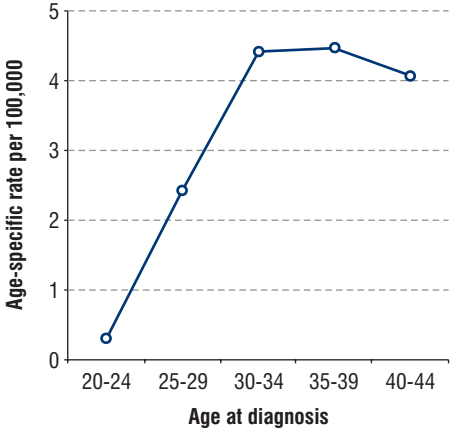
The overall age-standardized incidence rate in males of 3.2 in the 1990s obscures dramatic changes in incidence during the 1980s and 1990s. Prior to the onset of the AIDS epidemic in 1981, Kaposi sarcoma incidence was low to negligible. From 1985 to 1990, incidence was rising significantly at 21.7% per year. Incidence peaked in 1990 at over 4 per 100,000, remained stable until 1995 and then fell significantly by 43.6% per year. By 1999, incidence was back to the levels of the early 1980s (approximately 0.6 per 100,000). Projected incidence rates were expected to remain low and continue to fall to 2005. These short-term projections were based on 1990–1999 data.

Interpretation of patterns and trends

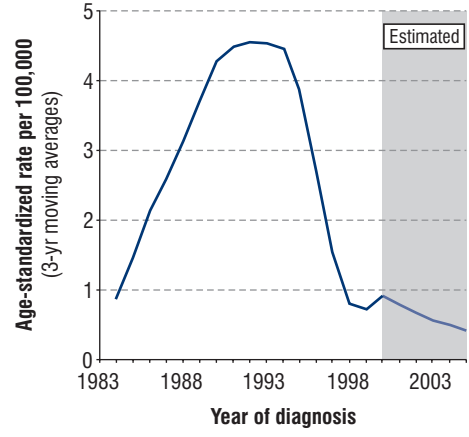
Historically, Kaposi sarcoma was a rare cancer known to occur in patients with suppressed immune systems. By 1982, it was one of the diseases considered "AIDS-defining". Increases in incidence, which continued until the early 1990s, thus occurred most notably among homosexual men with AIDS.[5] Since then, Kaposi sarcoma incidence rates have decreased to levels of the pre-AIDS era, probably because of aggressive prevention methods to decrease the rate of sexually transmitted HIV infections and the introduction of effective antiretroviral therapies between the late 1980s and mid-1990s. Treatment advances, especially combination Highly Active Antiretroviral Therapy (HAART) introduced in 1996, improved immune function among HIV-immunocompromised individuals and thus led to a dramatic decrease in Kaposi sarcoma rates.[15]

Within Canada, Kaposi sarcoma incidence in males was highest in regions where the incidence of AIDS or groups at highest risk for developing AIDS (homosexuals and injection drug users) was high.[66] Kaposi sarcoma incidence was higher than previously reported for Ontario; see the *Appendix* for details.

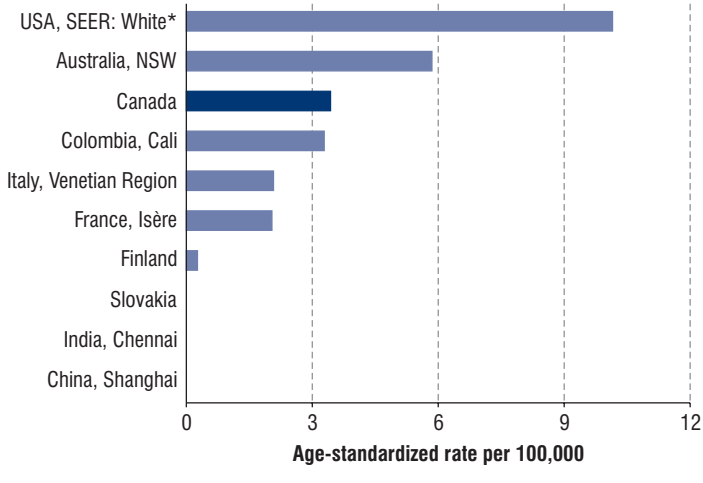
Kaposi sarcoma
Age-specific incidence rates for young males, Canada, 1990–1999



Kaposi sarcoma
Age-standardized incidence rates for males aged 20–44, Canada, 1983–2005



Kaposi sarcoma
Age-standardized incidence rates for males aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Includes San Francisco, CA

TESTIS

Cancer of the testis is the seventh most common cancer in young adults, and represents a larger portion of cancers in young adults (5%) than in all ages combined (< 1%).[25] It is the most common cancer in young men and one of the few in this age group that is increasing in incidence. The high survival from this cancer is reflected in its tenth place in terms of potential years of life lost from cancer in young men and fifteenth place in terms of mortality.

Testis
Summary statistics, 1990-1999

Cases	5,306
Incidence rate	9.0
% of all male cancers	14%
Incidence rank	1st
Deaths	217
Mortality rate	0.4
5-yr survival	97%
PYLL rank	10th

Age-specific rates

The age-specific incidence rate for testicular cancer peaks at 11.4 per 100,000 in the 30–34 age group, and then begins to decline. The incidence in the 40–44 age group is lower than in the 20–24 age group.

Geographic variation

Rates are highest in Denmark (22.8 per 100,000), with intermediate incidence in the eastern European countries, the UK, US whites and Canada. Incidence rates are lowest in Asian and South and Central American populations.[134] Canada ranks fifth among the ten countries chosen for comparison. Within Canada, rates are lowest in the Atlantic provinces and Quebec, at around 7.5 per 100,000, compared with rates over 9.5 per 100,000 in the other regions.

Trends

In Canada, incidence rates increased significantly by 2.2% per year, from 6.7 per 100,000 in 1983 to 9.6 per 100,000 in 1999. Incidence was expected to continue to increase through to 2005.

Subgroups

The two main morphological subgroups occur with similar frequencies: 54% are seminoma and 41% non-seminoma germ cell tumours; 5% are other types. Non-seminoma germ cell cancer of the testis is more common in men younger than about 30 and seminoma is more common thereafter.

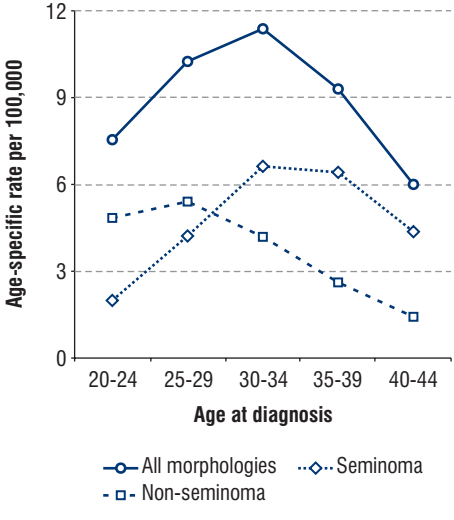
The incidence of non-seminoma testicular cancer increased consistently and significantly between 1983 and 1999 (2.6% per year). Seminoma incidence rose significantly between 1983 and 1989 (5.9% per year) and increased non-significantly from 1990 onwards. There is some evidence that the rate of increase for seminoma may be slowing in the US population also.[116]

Interpretation of patterns and trends

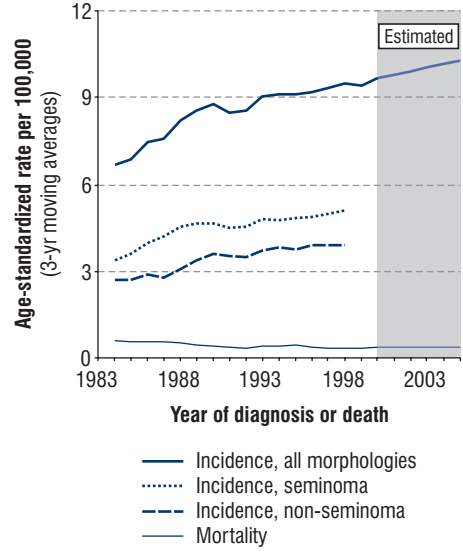
Testicular cancer incidence rates have also risen in other populations of European ancestry.[141] The increasing incidence is likely due to a birth cohort effect, with more recently born men at greatest risk.[104] This suggests that diagnostic changes are not a major factor, and there is no evidence of changes in diagnostic criteria or increased surveillance for early cancers. Since the risk factors for testicular cancer are not well defined, apart from undescended testicle, there is no accepted explanation for the increasing trend. A shift towards earlier age at puberty may be one explanation if early age at puberty is indeed a risk factor, as some studies suggest. Age at puberty has

declined in females in the US and many other developed countries, although the rate of decline appears to be less since the 1960s.[132] It is possible that a parallel decline has occurred in males, although supportive data are lacking in part because there is no obvious, reproducible indicator of onset of puberty in boys, analogous to menarche in girls. Reasons for the variation in incidence within Canada are unknown.

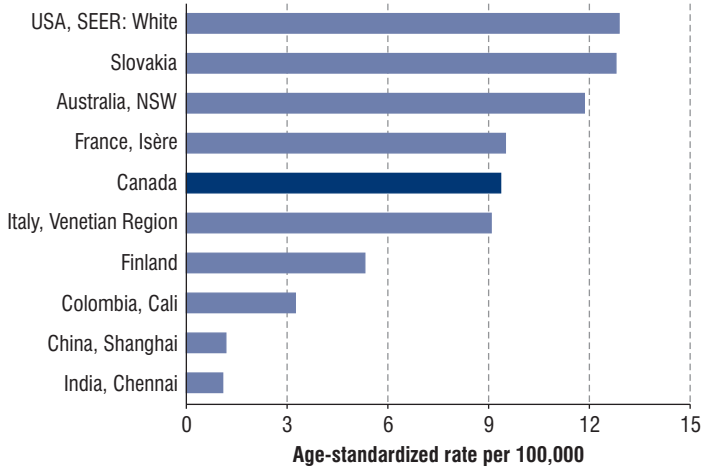
Testicular cancer
Age-specific incidence rates for young males, Canada, 1990–1999



Testicular cancer
Age-standardized rates for males aged 20–44, Canada, 1983–2005



Testicular cancer
Age-standardized incidence rates for males aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002

BRAIN

This group of cancers includes cancers of the brain and other parts of the central nervous system (CNS), including meninges. Since cancer registries typically register only invasive tumours, the clinically important, but histologically benign, brain tumours are not included. Brain cancer is the third most common cancer cause of death in young adults and ranks tenth in incidence. This cancer is more prominent among young adults (4% of all cancers) than for all ages combined (2%).[25]

Brain
Summary statistics, 1990-1999

	Males	Females
Cases	2,434	1,695
Incidence rate	4.1	2.9
% of all cancers	6%	3%
Incidence rank	5th	11th
Deaths	1,312	772
Mortality rate	2.2	1.3
5-yr survival	53%	63%
PYLL rank	2nd	4th

Age-specific rates

Age-specific incidence of brain and other CNS cancer increases approximately 1.2-fold in each successive age group. From the youngest to the oldest age group, the incidence for males increases 2.6-fold, while the incidence for females doubles. The age-specific mortality curves increase slightly faster with increasing age, with the rate in each age group being 1.4 times the rate in the previous age group. From the youngest to the oldest age group, mortality rates quadruple for both males and females.

Geographic variation

Rates are highest in some northern and western European countries, Canada and other western developed nations. Some eastern European countries have intermediate rates, while Japan (1.2 per 100,000) and some less developed Asian countries, and countries of Central America, have the lowest rates.[134] Canada ranks fifth among the ten countries chosen for comparison.

Trends

Incidence and mortality were stable through the 1980s and 1990s for this age group for both sexes and were projected to remain stable through 2005.

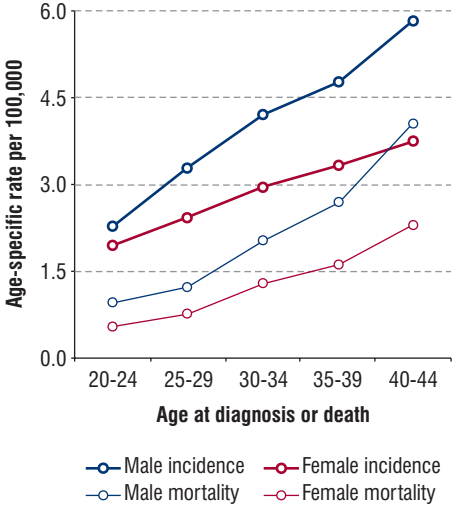
Subgroups

Brain cancer represents a heterogeneous group that can be categorized according to site and morphology. Most (95%) of the cancers in this group arise in the brain, another 4% in the spinal cord. Morphologically, most are gliomas (85%), two-thirds of which are astrocytomas (mostly not otherwise specified) or glioblastomas.

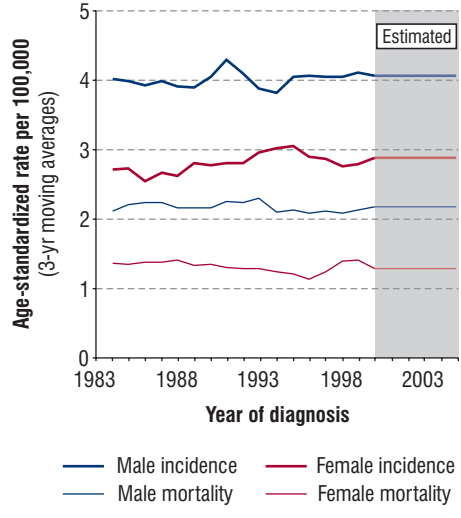
Interpretation of patterns and trends

The stable rates over time and male to female ratio are consistent with the pattern seen in the US SEER registries for gliomas diagnosed in men and women in their early 30s.[70] Little is known about the causes of most of the heterogeneous group of brain and other central nervous system cancers.

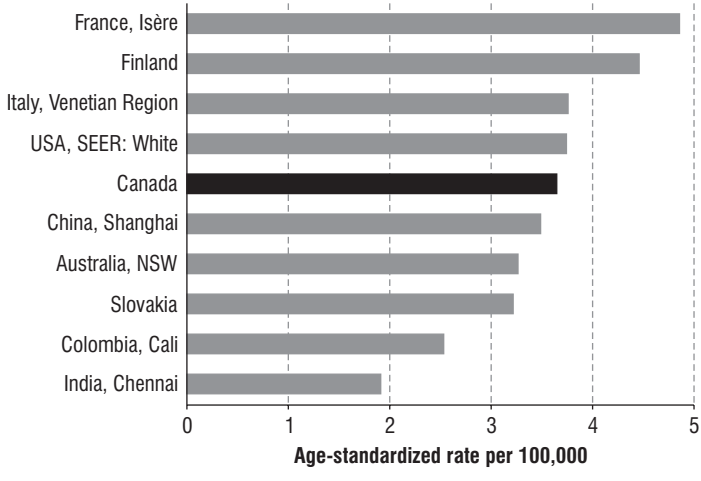
Brain cancer
Age-specific rates for young adults
Canada, 1990–1999



Brain cancer
Age-standardized rates for adults
aged 20–44, Canada, 1983–2005



Brain cancer
Age-standardized incidence rates* for adults aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Both sexes combined

LEUKEMIA

Leukemia is a heterogeneous group of hematopoietic cancers. In young adults, leukemia ranks fourth in terms of cancer mortality and twelfth in terms of incidence.

Leukemia
Summary statistics, 1990-1999

	Males	Females
Cases	1,823	1,364
Incidence rate	3.0	2.3
% of all cancers	5%	2%
Incidence rank	9th	12th
Deaths	901	669
Mortality rate	1.5	1.1
5-yr survival	54%	59%
PYLL rank	3rd	5th

Age-specific rates

Age-specific leukemia incidence is higher for men than women for all age groups, but both curves are very similar and show a sharp change in the rate of increase at age 30–34. For all leukemias combined, from the youngest age group to the oldest, incidence in males increases 2.5 times, while female incidence doubles. The age-specific incidence curves for all leukemias mask the different age-specific incidence curves characteristic of the leukemia subgroups. Although the incidence of myeloid leukemia (acute or chronic) and chronic lymphoid leukemia (CLL) increases with age (from 20–29 to 30–44 age groups), the reverse is observed for acute lymphoid leukemia (ALL), the main pediatric subgroup. Across the age range, leukemia mortality nearly doubles for males, and more than doubles for females.

Geographic variation

Leukemia incidence varies relatively little worldwide (1.8 to 3.5 per 100,000), with no particular pattern evident. The highest reported rates are in South and Central America, UK, US and Australia while the lowest rates are reported for India and some Central and Northern European countries.[134] Canada ranks fifth among the ten countries chosen for comparison. Within Canada, leukemia rates are highest in Ontario.

Trends

Between 1983 and 1999, leukemia incidence was stable in young men and declined non-significantly in young women. Rates remained between 2.6 and 3.6 per 100,000 in men and between 1.9 and 2.9 per 100,000 in women over the two decades. This continues a trend for all ages combined that was first observed in the 1970s.[23] Incidence was projected to be stable for both sexes between 2000 and 2005. Mortality for all ages combined and in both sexes has been falling since the 1970s.[23] In young adults, mortality for males fell significantly by 4.6% per year until 1995 at which point there was a slight and non-significant rise. Mortality for males was projected to be stable between 2000 and 2005. For females, a significant decline in mortality of 3.8% per year throughout the 1980s and 1990s was projected to continue to 2005. These short-term projections were based on 1990–1999 data.

Subgroups

In young adults, acute myeloid leukemia (AML) comprises 37% of leukemias, chronic myeloid leukemia (CML) 23%, ALL 16% and CLL 8%. The remaining 16% are primarily hairy cell and unspecified leukemias. ALL, CLL and CML account for a larger proportion of leukemia in young men than in young women; AML proportions are similar in both sexes.

The distribution of these subgroups varies by age. AML is dominant for both the 20–29 (42%) and 30–44 (36%) age groups. In the 30–44 age group the proportion of CLL is higher and ALL is lower than in the 20–29 age group. It is not surprising that AML and CML are the most common types in young adults, since ALL typically occurs in children and CLL in the elderly.[136]

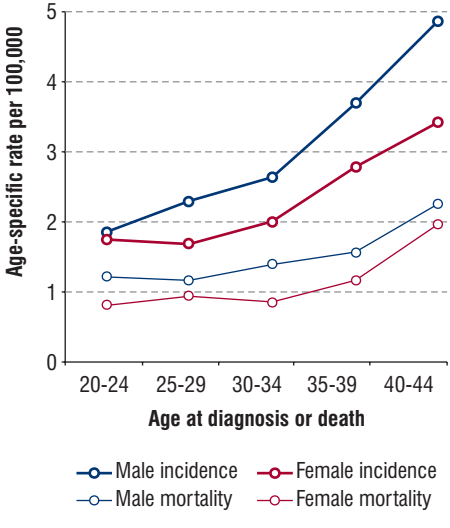
AML incidence was stable between 1983 and 1999 for both sexes combined; numbers for the other three subgroups were too low to estimate incidence trends. Morphology information for Quebec cases was not specific enough for inclusion in subgroup analyses.

Interpretation of patterns and trends

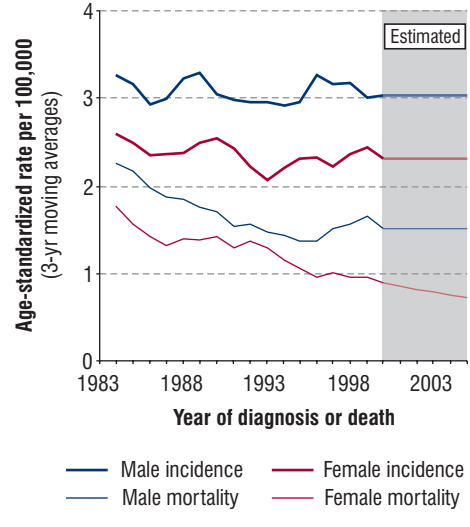
The different leukemia subgroups have distinct age and trend patterns. Combined, they demonstrate stable rates and little international variation. Little can be said to explain the high rates reported in Ontario. There is evidence that incidence rates have been stable or slowly increasing worldwide.[168] Low mortality rates reflect continuing improvement in the treatment of these previously uniformly fatal diseases.[97]

Known risk factors, such as tobacco, occupational exposures and treatment with chemotherapy or radiation, explain only a small proportion of AML, the most common type in young adults.[100, 136] Stable patterns for the AML subgroup also have been observed in the US for young adults.[193]

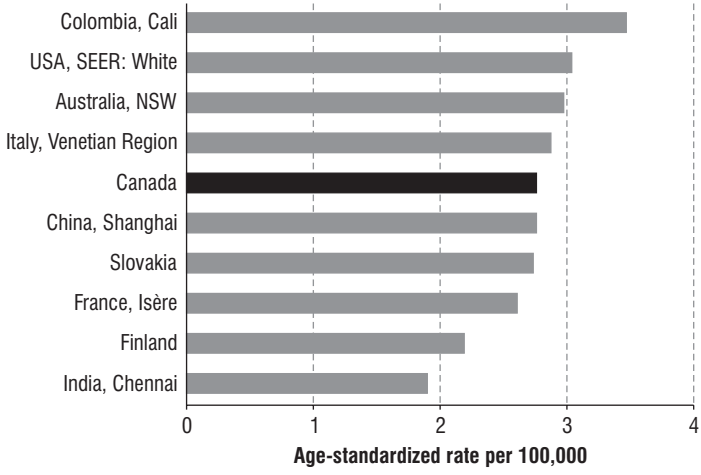
Leukemia
Age-specific rates for young adults
Canada, 1990–1999



Leukemia
Age-standardized rates for adults
aged 20–44, Canada, 1983–2005

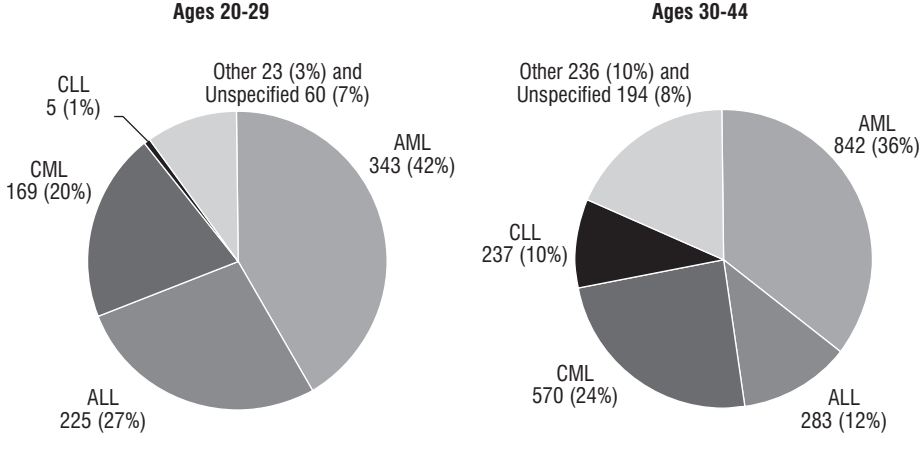


Leukemia
Age-standardized incidence rates* for adults aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Both sexes combined

Distribution of leukemia morphologic subgroups*, Canada, 1990–1999



* Both sexes combined

"For a long time, I didn't think I had a future. To this day, I still don't know, but I can't picture myself growing old with my husband…I bargained with Him [God], to give me ten extra years. So, I'm at five…. even if I do make it beyond five more years, I don't know if I'll have much more beyond that." ***Female, Breast Cancer, 34***

"So, workwise it set me back… because I didn't already have the secure full-time job with a few years there…I didn't want to go into the interviews and say, well, I had cancer twice, because, who is going to want to hire me?"
Female, Breast Cancer, 28

"So, luckily I had my grandfather that helped with money and now I have a husband that looks after me, but it is completely funny that I went from independent to completely having to rely on him, which is good, but I had to do that…. At 25, you are just a kid." ***Female, Breast Cancer, 28***

Breast cancer is by far the most common cancer in young adults, and the leading cancer cause of death. Breast cancer also represents a larger proportion of cancers in young adults (21%) than in all ages combined (14%).[25] Breast cancer rarely occurs in young men (47 cases during the decade 1990–1999); therefore, this discussion is restricted to female breast cancer. For the following analyses, the age range is extended to 49 to better represent the population at risk for pre-menopausal breast cancer.

Breast
Summary statistics, 1990-1999

Cases	21,308
Incidence rate	34.5
% of all female cancers	34%
Incidence rank	1st
Deaths	4,181
Mortality rate	6.7
5-yr survival	85%
PYLL rank	1st

Age-specific rates

Age-specific incidence increases sharply with age, with incidence in each age group 25–29 through 40–44 nearly 2.5 times the rate of the previous age group. This rate of increase drops to 1.6 for the 45–49 age group. Age-specific mortality rates follow a similar pattern. Across the extended age range, incidence increases 200-fold, while mortality increases 400-fold.

Geographic variation

Canada is among several western developed countries with the highest breast cancer incidence rates for women aged 20–49. Incidence is lower in Asian and eastern European countries.[134] This pattern is similar to the pattern for breast cancer at all ages. Canada occupies sixth place among the ten countries that were selected for comparison.

Trends

After a significant rise in incidence at 0.3% per year between 1983 and 1999, projections indicated that the rate for ages 20–49 would likely stabilize in 2000–2005. This rise was actually confined to women in their 40s, while rates for younger women declined slightly. The slight decline in breast cancer among women younger than 40 is consistent with declines in incidence for Canadian, Scottish and US white women in birth cohorts after 1950.[171]

Mortality has declined since 1986, with a steeper decline (-5.3%) since the mid 1990s. This trend was projected to continue through 2005. The drop in mortality across the late 1980s and the 1990s is similar to that seen for women of all ages in several western developed countries.[85]

Subgroups

Most breast cancers in women aged 20–49 are infiltrating duct carcinoma (71%). Another 9% are either lobular carcinoma or infiltrating duct plus lobular. A steady increase in both infiltrating duct carcinoma and cancers with a lobular component has been accompanied by a decrease in other types. Other types are infiltrating ductular carcinoma (5%), comedocarcinomas (3%), medullary carcinoma (2%), mucinous adenocarcinoma (1%), Paget's disease (1%) and other carcinomas (7%). The increase in breast cancers with a lobular component is similar to that seen for young women in other jurisdictions.[98, 180] This increase in lobular carcinoma relative to ductal carcinoma is not as large among young women as in older women.

Morphology is non-specific for fewer than 1% of breast cancers in the data set. Malignant phyllodes tumours of the breast are discussed with the sarcomas.

Interpretation of patterns and trends

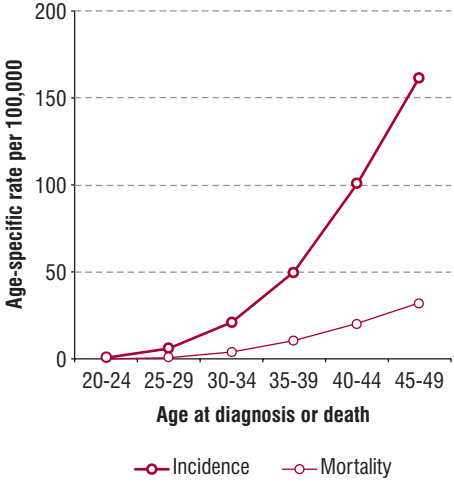
The overall increase in breast cancer incidence reflects the high number of cases diagnosed at ages 40 and over. In women diagnosed with breast cancer at ages 20 through 39 there was a non-significant decrease in incidence from 1983 to 1999.

A continued rise in age at first birth may contribute most to the increase in breast cancer incidence in Canadian women aged 20–49 between 1983 and 1999. Another contributing factor may have been more widespread use of breast cancer screening by women in their 40s. Most other known risk factors for premenopausal breast cancer are modest in magnitude, and some show opposing trends over the relevant periods in terms of possibly increasing or decreasing risk.

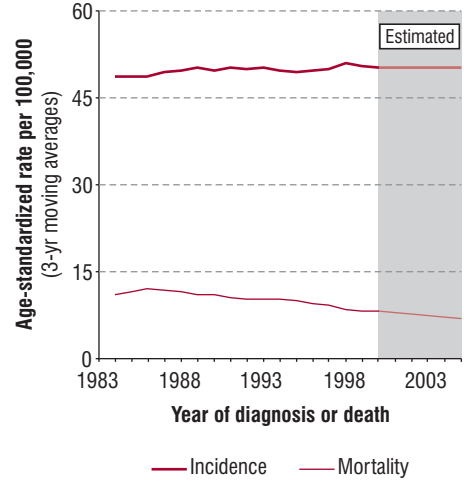
A proposed explanation for the rise in lobular breast cancer in the US and in Switzerland is the use of hormone replacement therapy.[98, 180] This may apply to Canadian women, since the increase in lobular breast cancer was greater for women in their 40s than for younger women, and there is some evidence that Canadian women aged 45–49 increased their use of hormone replacement therapy across the 1980s and early 1990s.[39]

The drop in mortality across the late 1980s and the 1990s is similar to that seen for women of all ages in several western developed countries.[85] This decrease in mortality rates may be mainly attributable to improvements in treatment (notably adjuvant systemic therapy) and earlier detection of palpable tumours. Earlier detection by mammographic screening is less common and less effective in this age group than in older women.[85]

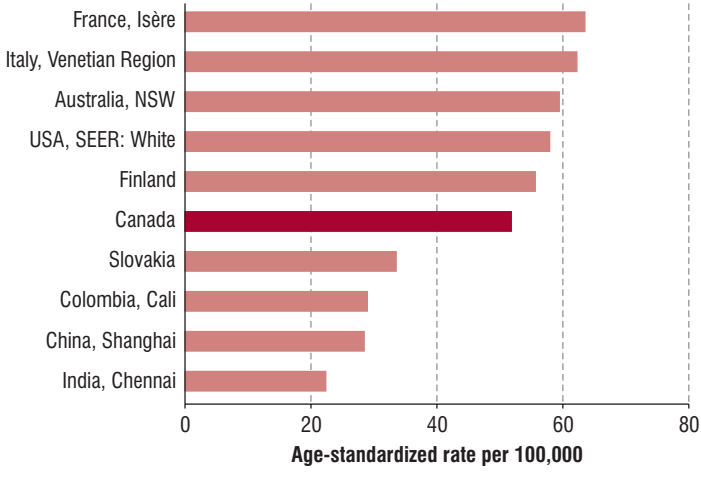
Breast cancer
Age-specific rates for young females
Canada, 1990–1999



Breast cancer
Age-standardized rates for females
aged 20–49, Canada, 1983–2005

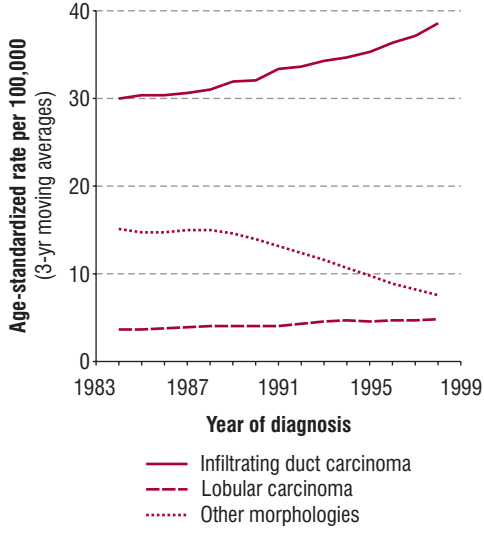


Breast cancer
Age-standardized incidence rates for females aged 20–49
Selected international regions, 1993–1997



Source: Parkin et al., 2002

Breast cancer morphologic subgroups
Age-standardized incidence rates for females aged 20–49
Canada, 1983–1999



Thyroid cancer is the third most common cancer in young adults. It is more prominent in young adults (7% of all cancers) than all ages combined (2% of all cancers)[25] and its incidence in young adults is rising more rapidly than that of any other cancer. Five-year survival from thyroid cancer is high, resulting in very low mortality and potential years of life lost.

Thyroid
Summary statistics, 1990-1999

	Males	Females
Cases	1,295	5,296
Incidence rate	2.2	9.0
% of all cancers	3%	9%
Incidence rank	13th	3rd
Deaths	28	21
Mortality rate	<0.1	<0.1
5-yr survival	98%	100%
PYLL rank	12th	15th

Age-specific rates

The age-specific incidence curves for thyroid cancer are unusual because the pattern is strikingly different between males and females. For males, incidence increases slowly with increasing age, with the incidence rate in each age group 1.2 times the rate in the previous age group. For females, the rate of increase slows from the 20–24 age group to the 35–39 age group, and then is almost zero between that and the 40–44 age group. From the youngest to the oldest age group, incidence doubles among females and more than doubles among males.

Geographic variation

Incidence rates for thyroid cancer vary considerably around the world. The lowest rates are found in Asian populations and the highest in the US (in whites only), Australia, Finland and Canada; most European countries have intermediate rates.[134] Canada ranks fourth among the ten countries chosen for comparison. Within Canada, Ontario has significantly higher incidence rates than the rest of the country.

Trends

Increasing incidence in young adults is part of a trend for all ages that began at least 30 years ago.[24] Incidence in young women rose significantly at a rate of 4.9% per year, from 5.5 per 100,000 in 1983 to 11.2 per 100,000 in 1999. In young men, the increase was also significant, but slower, averaging 2.9% per year between 1983 (1.7 per 100,000) and 1999 (2.7 per 100,000). These trends were expected to continue through 2000–2005.

Subgroups

Almost all thyroid cancers in the young adult age group are well-differentiated carcinomas of the papillary (83%), follicular (9%) and medullary (2%) types. The rate of increase is greatest for the papillary carcinomas, and significant for both young women (6.0%) and young men (3.9%).

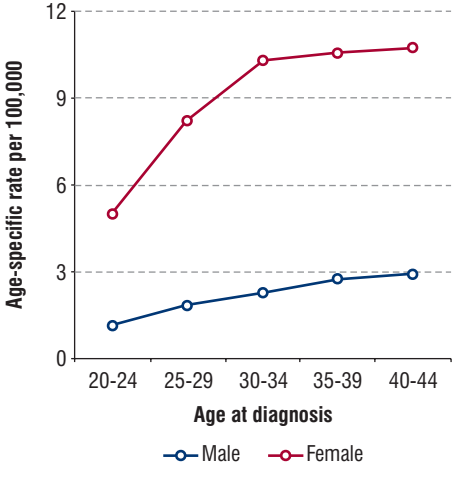
Interpretation of patterns and trends

Of the 18 cancer types examined in this analysis, thyroid cancer has been the most rapidly increasing cancer in young men and women. An increasing trend in thyroid cancer incidence has been noted in most developed countries[168] and is generally strongest in young and middle-aged women.[103] Possible reasons include changes in risk factor prevalence and changes in diagnosis, in terms of either disease definition or application of diagnostic methods.

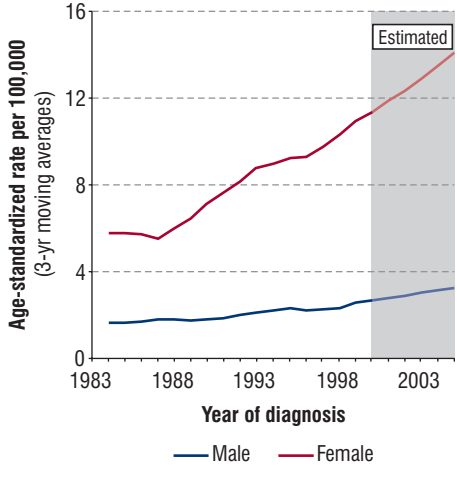
Ionizing radiation is the clearest known risk factor for thyroid cancer. Age-period-cohort analysis of Canadian thyroid cancer incidence revealed strong birth cohort effects, perhaps reflecting the use of ionizing radiation to treat childhood head and neck conditions between the 1930s and 1960s.[103] The stronger effects in women may reflect the increasing use of oral contraception in successive cohorts of young women. Oral contraceptive use is not, however, regarded as a strong risk factor. Changes in the prevalence of other possible risk factors, such as iodine deficiency/excess or radioactive fallout, while unknown, are unlikely explanations for the increasing incidence in Canada, although the latter certainly explains increasing incidence in those living close to Chernobyl.[111]

Period effects were also found in the analysis of Canadian data, suggesting changes in diagnosis. Increasing numbers of diagnostic investigations with fine needle aspiration biopsy and ultrasonography, coupled with increased vigilance, particularly in young women, have likely resulted in the diagnosis of asymptomatic tumours.[38] Changes in diagnostic definitions or criteria have probably occurred also.[38] No obvious explanation accounts for Ontario's higher incidence.

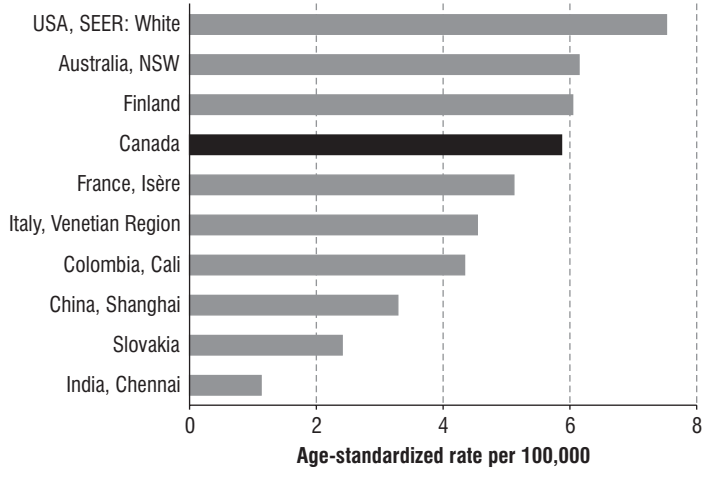
Thyroid cancer
Age-specific incidence rates for young adults, Canada, 1990–1999



Thyroid cancer
Age-standardized incidence rates for adults aged 20–44, Canada, 1983–2005



Thyroid cancer
Age-standardized incidence rates* for adults aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Both sexes combined

"And the worst thing I think about all this is that thyroid cancer wasn't a popular cancer. …a lot of the stuff on the Internet was American …and there are actually no [support group] meetings for thyroid cancer. … even if I did want to go somewhere for a meeting, there was nothing for thyroid cancer..."

Female, Thyroid, 33

Cancer of the cervix is the fifth most common cancer in young adults (second among young women), and ranks tenth in terms of mortality. Cervical cancer is more prominent in young adults (6% of cancers) than in all ages combined (1%).[25]

Cervix
Summary statistics, 1990-1999

Cases	6,277
Incidence rate	10.5
% of all female cancers	10%
Incidence rank	2nd
Deaths	940
Mortality rate	1.5
5-yr survival	86%
PYLL rank	3rd

Age-specific rates

Unlike most epithelial cancers, the age-specific incidence rates for cervical cancer do not show a constant rate of increase with increasing age. Incidence peaks in the 35–39 age group and falls slightly thereafter. Although much lower than incidence, the mortality rate rises almost 19-fold between the youngest and oldest age groups.

Geographic variation

Incidence for cervical cancer varies considerably around the world. Rates are highest in some of the less developed regions that do not have cancer registries, such as eastern, southern and central Africa, the Caribbean, Central and South America, and parts of south and south-eastern Asia.[168] Among countries with cancer registries, rates for young adults are high in Latin America, parts of Europe and in India, and low in China and other European regions. The incidence in Canada is intermediate, as it is in the US, Sweden and Australia.[134] Canada ranks fifth among the ten countries chosen for comparison.

Trends

Between 1983 and 1986, incidence for cancer of the cervix fell significantly by 7.0% per year; this represented the continuation of a steep downward trend that started much earlier.[22] Between 1986 and 1999, rates declined more slowly but significantly at 1.4% per year, from 11.2 to 9.3 per 100,000. Mortality also fell significantly (-1.3% per year), from 1.8 to 1.3 per 100,000 between 1983 and 1999. As with incidence, mortality fell more steeply in earlier years.[22] While incidence rates were expected to continue their slight decline through 2005, mortality rates were projected to level off.

Subgroups

Squamous cell carcinoma accounts for 71% of cancers of the cervix; adenocarcinoma (including adenosquamous carcinoma) accounts for most of the remainder (22%). Morphology is not specified for 5% of cancers of the cervix.

While the incidence of squamous cell carcinoma declined significantly over the period of study, adenocarcinoma incidence rose significantly between 1983 and 1995, then fell (at a non-significant annual rate). Adenocarcinoma incidence in young Canadian women had been increasing before 1983; rates for 20–34 and 35–49 year old women increased 3- and 2-fold respectively between 1970–1972 and 1994–1996.[101]

Interpretation of patterns and trends

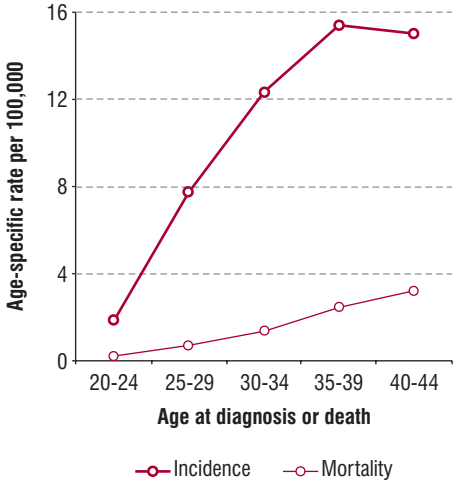
The long-term downward trends in cervical cancer incidence and mortality are largely the results of the introduction and uptake of the Pap test. Participation rates, laboratory quality assurance and the extent of organized screening have all continued to improve

over time in Canada. The slowing of the overall rate of decline in incidence in the past 15–20 years may be due to increases in the true underlying incidence of cervical cancer, including adenocarcinoma, not completely counteracted by screening. We may also be approaching the maximum achievable benefit of screening as currently practised. In the most recent decade, the proportion of Canadian women aged 20–44 who reported ever having had a Pap test improved only slightly from 88% in 1990 to 91% in 1998/99.[161–164]

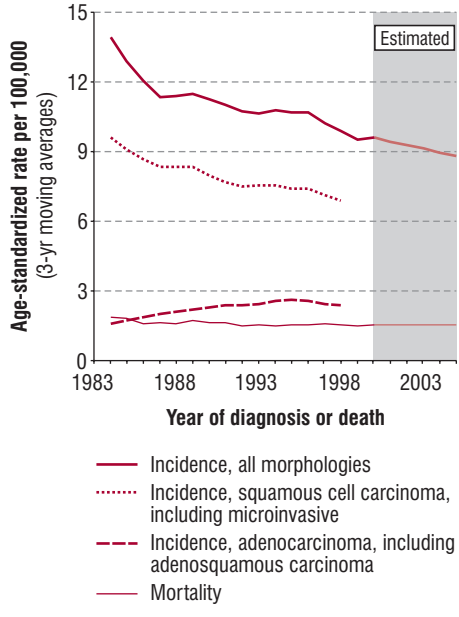
Since no major changes have been documented in morphological classification of cervix tumours, trends are not likely due to shifts of diagnosis between the two main tumour types.[188] The rising incidence of adenocarcinoma of the cervix through the early 1990s has been noted among young women in many parts of the world.[181] It appears to be the result of a cohort effect, starting with women born around the mid-1920s[197] or 1930s.[181] More recent data from the UK suggest that, as in Canada, rates began to decline in the mid 1990s, and that women born since 1960 are at lower risk than those born earlier.[151] The long-term rise in adenocarcinoma incidence may reflect increased human papilloma virus (HPV) infection rates with changes in sexual behaviour during the 1960s in many countries. Alternatively, it may be due to increased prevalence of co-factors, coupled with relatively poor detection of its precursors on Pap tests. There is, however, no evidence that the prevalence of oral contraceptive use or current smoking—two co-factors on which data are available—increased significantly in this age group over the 1990s.[162–164] Women born more recently may have reduced risk because of improvements in Pap testing that increase the likelihood that early adenocarcinoma and its precursors will be identified.[181] The changing patterns of morphology-specific cervical cancer incidence emphasizes the importance of cancer surveillance according to histopathological subgroup.

International variation in incidence is related to differences in rates of infection with oncogenic forms of HPV, in the effectiveness of Pap test screening, and in the prevalence of co-factors such as smoking. Within developed parts of the world, countries with a long history of organized Pap test screening, such as Finland, tend to have lower incidence rates.

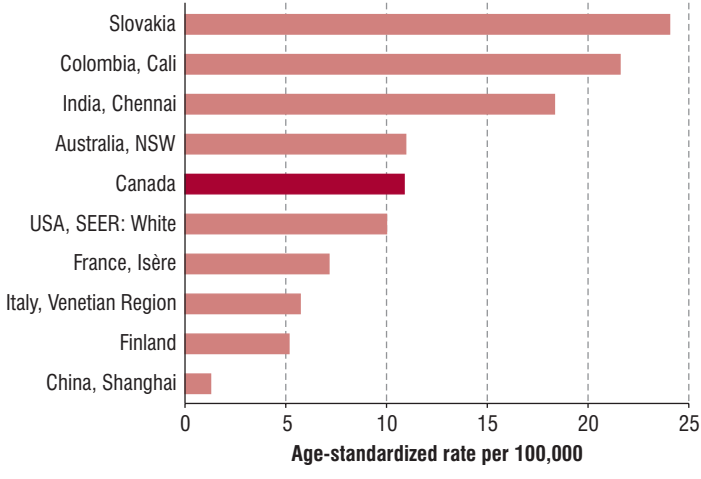
Cervical cancer
Age-specific rates for young females
Canada, 1990–1999



Cervical cancer
Age-standardized rates for females
aged 20–44, Canada, 1983–2005



Cervical cancer
Age-standardized incidence rates for females aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002

Cancer of the colon and rectum is the sixth most common cause of cancer incidence among young adults and ranks fifth in mortality.

Colon and rectum
Summary statistics, 1990-1999

	Males	Females
Cases	2,831	2,717
Incidence rate	4.6	4.4
% of all cancers	7%	4%
Incidence rank	4th	5th
Deaths	796	729
Mortality rate	1.3	1.2
5-yr survival	64%	65%
PYLL rank	5th	6th

Age-specific rates

Age-specific incidence and mortality rates are similar for both sexes. In each successive age group, the incidence rates are more than double the rates in the previous age group. From the youngest age group to the oldest, colorectal cancer incidence increases over 30-fold for both males and females. Mortality rates increase at the same rate for both sexes, with each age group having rates 2.3 times higher than the previous age group. Across the age range, both male and female colorectal cancer mortality increase more than 35-fold. Mortality rates are just over one-quarter the incidence rates for each age group.

Geographic variation

The highest reported rates worldwide are in US blacks (6.9 per 100,000) and Hong Kong (7.1 per 100,000), and the lowest in India and some South and Central American nations. Canadian rates are intermediate for this age group, along with several European countries.[134] Canada ranks fourth among the ten countries chosen for comparison. Within Canada, colorectal cancer incidence rates are highest in the Atlantic region and Ontario.

Trends

Colorectal cancer incidence in young adults fell significantly at about 1.2% per year, from 5 per 100,000 in 1983 to 4 per 100,000 in 1999, for both sexes. Mortality rates fell significantly at 3.0% per year for males and at 2.5% for females. Incidence and mortality rates were expected to continue to decline in males and to stabilize in females between 2000 and 2005. These short-term projections were based on 1990–1999 data.

Subgroups

The colon and rectum together may be divided into anatomic subgroups: the right colon (caecum, ascending colon, hepatic flexure and transverse colon), the left colon (splenic flexure, descending colon and sigmoid colon) and the rectum and rectosigmoid junction. Right colon cancer proportions are similar in both sexes (29% in males, 26% in females), left colon cancers are more common in females (31%) than males (24%), and rectosigmoid, rectal and anal tumours are more prevalent in males (39%) than females (34%). Anal tumours comprise 18% of this last subgroup. Eight percent of colorectal cancers in males and 9% in females cannot be assigned to a subgroup because either they arise in overlapping sites, or a specific location has not been specified.

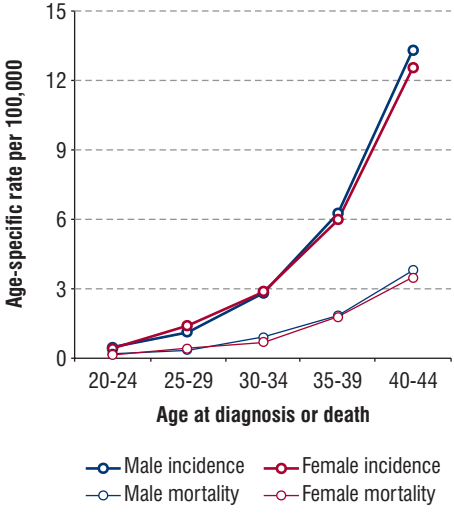
Incidence increases with age for all subgroups, but most abruptly for cancers of the rectosigmoid, rectum or anus. The most common morphology for cancer of the colon and rectum is adenocarcinoma, representing 91% of microscopically confirmed cases.

Interpretation of patterns and trends

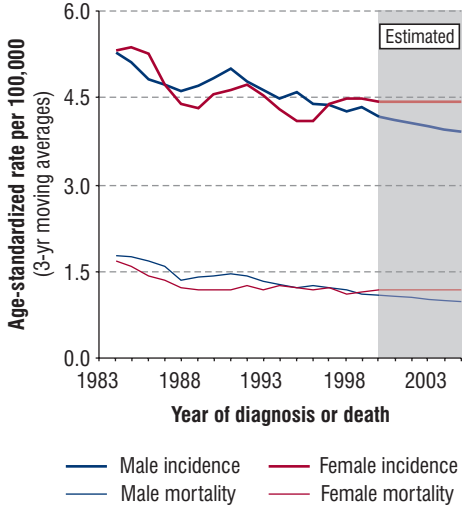
The decline in incidence of colon cancer may be attributable to increased physical activity in young adult Canadians, although obesity, another risk modifier, has also risen for this age range. Within-Canada regional variation for this age group is consistent with the east-west gradient for all ages, with higher rates in Atlantic Canada and lower rates in the western provinces.[25] This pattern is consistent with rates of physical inactivity across Canada, although there may be other explanations for the variation.[21]

The decline in mortality may be due to earlier detection when treatment may be more effective, improvements in access to treatment, and to the treatment itself.

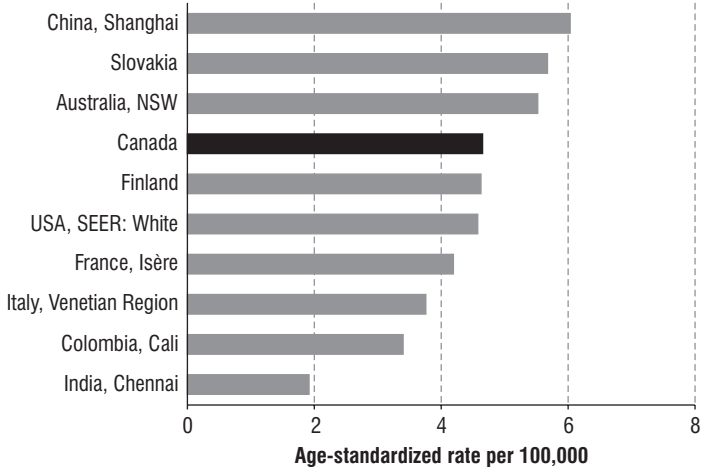
Colorectal cancer
Age-specific rates for young adults
Canada, 1990–1999



Colorectal cancer
Age-standardized rates for adults
aged 20–44, Canada, 1983–2005

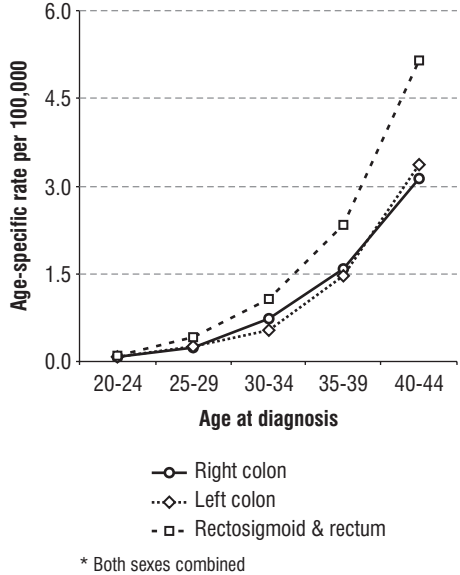


Colorectal cancer
Age-standardized incidence rates* for adults aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Both sexes combined

Colorectal cancer subgroups
Age-specific incidence rates* for young adults
Canada, 1990–1999



* Both sexes combined

Lung cancer is the second most common cancer cause of death in young adults and ranks eighth in incidence. During the 1990s deaths from lung cancer became more common in young women than in young men.

Lung
Summary statistics, 1990-1999

	Males	Females
Cases	2,201	2,613
Incidence rate	3.5	4.2
% of all cancers	6%	4%
Incidence rank	7th	6th
Deaths	1,578	1,770
Mortality rate	2.5	2.8
5-yr survival	23%	29%
PYLL rank	1st	2nd

Age-specific rates

Age-specific incidence and mortality rates are similar for both sexes. In each successive age group, incidence rates are nearly triple the rates in the previous age group. From the youngest age group to the oldest, both male and female lung cancer incidence increase nearly 50-fold. The age-specific mortality rates more than triple from one age group to the next. Across the age range, male and female mortality increases approximately 120-fold.

Geographic variation

Canadian lung cancer incidence rates for this age group are high compared to many other nations. The highest reported incidence rates are found in US blacks and in Thailand (7.0 per 100,000), while the lowest rates are found in South and Central America.[134] Canada ranks third among the ten countries selected for comparison. Rates within Canada vary, with high rates in Quebec and low rates in the Prairie provinces and British Columbia.

Trends

Lung cancer incidence for males fell by an average of 2.6% annually during the period 1983–1999, from 5.2 to 3.1 per 100,000. Female lung cancer incidence, however, rose non-significantly during the same period. Because survival is poor for lung cancer, the national mortality patterns were similar, with male mortality declining significantly by 2.8% per year and female mortality increasing significantly by 1.1%. Incidence and mortality were projected to continue declining in males and to stabilize in females through 2005. These short-term projections were based on 1990–1999 data.

The national time trends for lung cancer incidence conceal regional trends. While incidence decreased for males across the country, the decline was not significant in Atlantic Canada (-2.4%), less steep but significant in Quebec (-2.0%) and steepest in the other provinces and territories (-2.9%). The regional trends for females reveal more dramatic differences. While incidence rates fell significantly in the rest of Canada (-0.7%), rates increased significantly among females in Quebec (3.3%); in Atlantic Canada, the increase was not significant.

Subgroups

To better describe morphologic subgroups for lung cancer, only microscopically confirmed cases were examined. Adenocarcinoma is the most common morphology, representing 47% of histologically confirmed cases among young adults. Large cell carcinoma (13%), squamous cell carcinoma (12%) and small cell carcinoma (11%) are the other main morphologies. The distribution of morphologies differs between the

sexes. Females have a significantly higher proportion of adenocarcinomas (52% for females; 40% for males); squamous cell and small cell carcinomas are more common in males.

Incidence trends differ between the sexes by morphologic type. The incidence of adenocarcinoma of the lung rose significantly for women (1.5%), while declining significantly for men (-1.7%). For all other morphologies combined, rates for both sexes fell significantly, but more steeply for males (-5.1%) than for females (-1.5%).

Regional differences also exist within morphologic subgroups. Adenocarcinoma is increasing significantly among women in Quebec and Atlantic Canada (3.9%), while rates are stable in the rest of the country.

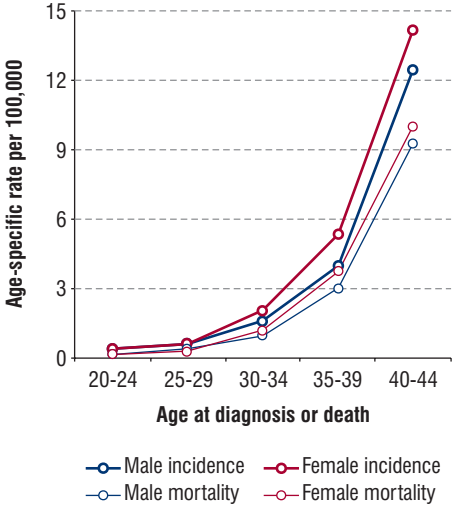
Interpretation of patterns and trends

Lung cancer mortality rates among males aged 20–44 peaked in the early 1970s, 20 years after smoking rates started to fall. Lung cancer incidence in young women surpassed that in young males in 1989, while the mortality rates crossed in 1992. Because smoking among females aged 20–24 peaked in the mid 1970s, it is possible that lung cancer rates among women aged 20–44 may have started declining, although it will be several more years before a change in trend can be detected.

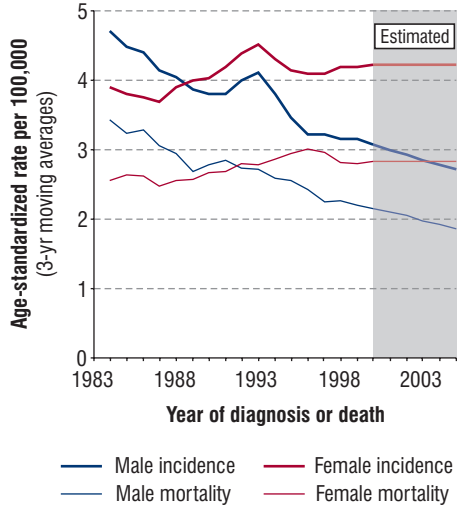
The tendency for Quebec to have the highest lung cancer rates and British Columbia the lowest is likely to continue; in 1999, Quebec had the highest teen smoking rate and British Columbia the lowest.[55, 68] More than half of all 10–14-year-old smokers live in Quebec.[167] The decline in smoking rates appears to have slowed, particularly among women aged 20–24. Such measures as the enforcement of new anti-smoking laws and the efforts of the Federal Tobacco Control Strategy will, it is hoped, lead to further reductions in smoking rates.

Further research into the relative importance of other risk factors could help to explain the regional differences in female lung cancer morphologies. Several studies have found that adenocarcinoma is the most common lung cancer morphology among women, young adults and non-smokers, and that the incidence of adenocarcinoma has been increasing among women.[53] Thun et al.[173] concluded that while the relationship between cigarette smoking and adenocarcinoma is weaker than for other morphologies, the increases are consistent with changes in cigarette design and smoking behaviour, and are not the result of diagnostic advances.

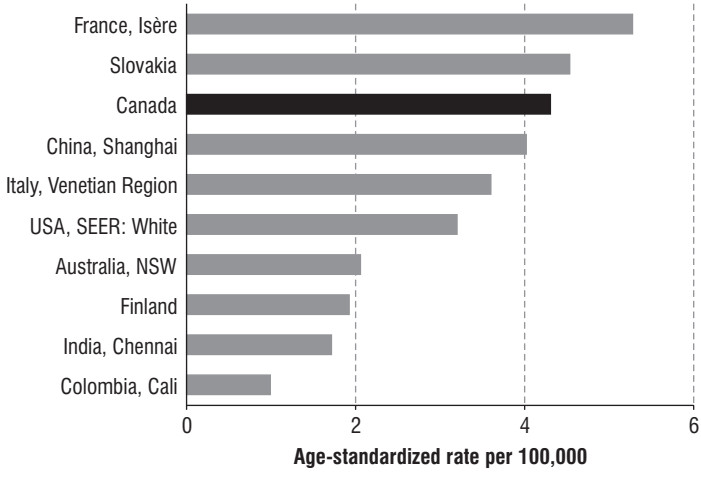
Lung cancer
Age-specific rates for young adults
Canada, 1990–1999



Lung cancer
Age-standardized rates for adults
aged 20–44, Canada, 1983–2005

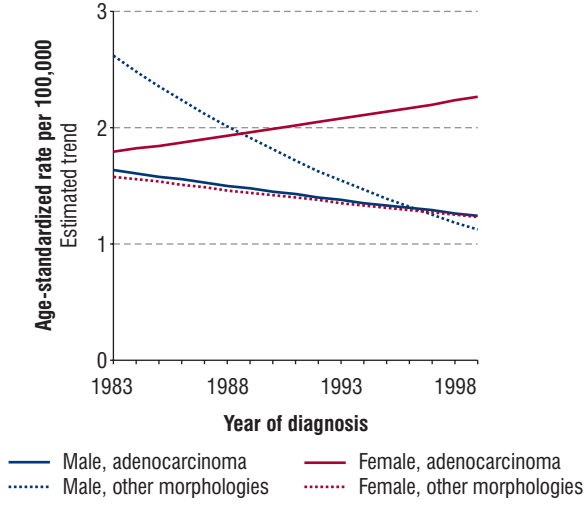


Lung cancer
Age-standardized incidence rates* for adults aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Both sexes combined

Lung cancer morphologic subgroups
Trend in age-standardized incidence rates for adults aged 20–44
Canada, 1983–1999



OVARY

Cancer of the ovary ranks eleventh in terms of mortality among young adults, and thirteenth in terms of incidence.

Ovary
Summary statistics, 1990-1999

Cases	2,433
Incidence rate	4.0
% of all cancers	4%
Incidence rank	8th
Deaths	649
Mortality rate	1.1
5-yr survival	69%
PYLL rank	7th

Age-specific rates

The age-specific incidence curve for ovarian cancer differs from most epithelial cancers; instead of a constant rate of increase with each successive age group, the increase is greater at older than younger ages (1.3-fold between the two youngest age groups and 1.7 between the two oldest age groups). From the youngest age group to the oldest, incidence for ovarian cancer increases nearly 6-fold. Age-specific mortality rates, on the other hand, do demonstrate a constant rate of increase, with the rates in each age group 2.2 times higher than in the previous age group. Across the age range, mortality for ovarian cancer increases nearly 20-fold.

Geographic variation

Incidence from several regions show, to some extent, the international pattern noted in the past for epithelial cancers of the ovary at all ages: highest rates in Europe, particularly in Scandinavian countries, and in North America, with lower rates in South America and Asia.[134] Canada ranks third among the ten countries chosen for comparison.

Trends

Incidence for ovarian cancer fell significantly at an average of 2.0% per year during the period 1983–1999, from around 5.0 per 100,000 to 3.6 per 100,000, and was projected to continue falling through 2005. Mortality fell significantly at the same rate, from about 1.3 per 100,000 to just 1.0 per 100,000, and was projected to continue declining through 2005.

Subgroups

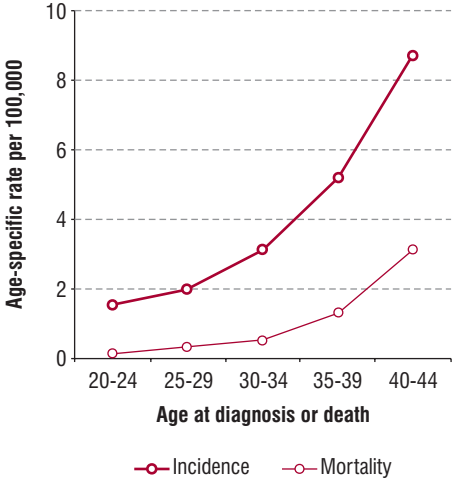
Carcinomas account for 81% of ovarian cancers in this age group (serous 32%, mucinous 18%, endometrioid 14%). A further 12% are germ cell tumours, 2% are sex-cord stromal tumours, 1% are other specified tumours and 4% have unspecified morphology. Median age at diagnosis among young women is 39 for carcinomas, 35 for sex-cord stromal, and 29 for germ cell tumours. Germ cell tumour incidence remained stable between the periods 1983–1989 and 1990–1999, while the incidence of serous and mucinous carcinoma and unspecified adenocarcinoma declined. Serous carcinoma showed a significant decline of 3% per year over the two decades; this is consistent with a decline in this morphological type noted earlier for 1950–1960 birth cohorts.[196]

Interpretation of patterns and trends

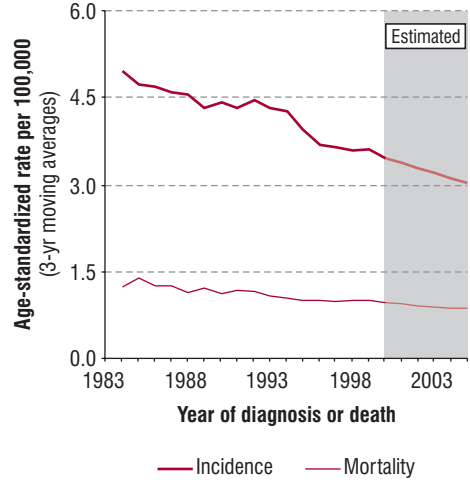
The falling incidence of ovarian cancer continues a long-term trend. The decline for young women was proportionately larger than a decline observed for women of all ages in Canada over the same period.[25] Declines in incidence and mortality were also noted through the 1990s in the US.[59] Decreasing incidence and mortality among young women in the US and UK coincide with the introduction and use of oral contraceptives in those countries; the protection appears stronger for this age range than any

increase in risk associated with declining fertility, similar to the Canadian pattern, over the same period.[43, 58] Oral contraceptive use appeared to remain at levels of 30% or more of women younger than 30 between the late 1970s and the 1990s and may explain falling trends of ovarian cancer in young Canadian women.[196, 114]

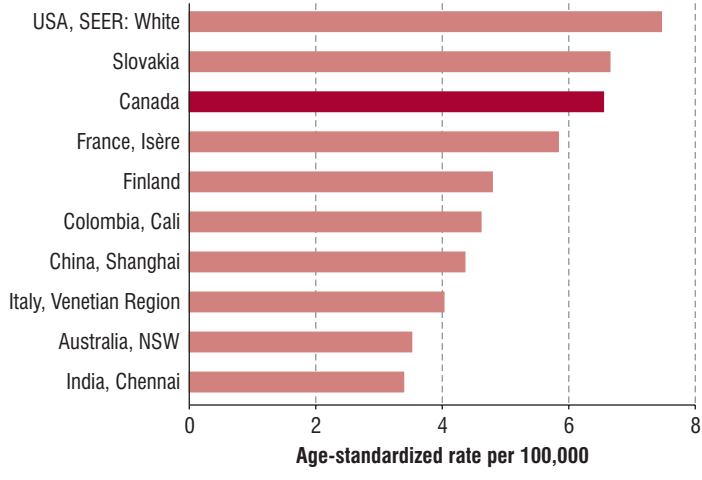
Ovarian cancer
Age-specific rates for young females
Canada, 1990–1999



Ovarian cancer
Age-standardized rates for females
aged 20–44, Canada, 1983–2005



Ovarian cancer*
Age-standardized incidence rates for females aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Tumours of borderline malignancy were included

Kidney cancer in young adults ranks fourteenth in terms of incidence and thirteenth in terms of cancer mortality.

Kidney
Summary statistics, 1990-1999

	Males	Females
Cases	1,406	907
Incidence rate	2.3	1.5
% of all cancers	4%	1%
Incidence rank	12th	14th
Deaths	302	155
Mortality rate	0.5	0.3
5-yr survival	80%	89%
PYLL rank	9th	12th

Age-specific rates

Age-specific incidence rates for kidney are typical of most epithelial cancers, with a constant rate of increase with increasing age. For males, kidney cancer more than doubles with each successive age group, while for females, the rate of increase is slightly less than twice. From the youngest age group to the oldest, male kidney cancer incidence increases 31-fold, while the increase for females is 13-fold. The age-specific mortality pattern for males is similar to the incidence pattern, showing an increase of 2.5 times with each successive age group. Across the age range, kidney cancer mortality increases 20-fold.

Geographic variation

The highest reported incidence rates are in Italy and some eastern European countries, and the lowest in Asia.[134] Canada, along with some northern European countries, is in the top third of the ten countries with similar cancer registration methods selected for comparison.

Trends

Kidney cancer incidence in young males increased significantly at 1.4% per year from about 1.7 per 100,000 in 1983 to 2.6 per 100,000 in 1999. Incidence was expected to stabilize through 2005. Incidence in females rose significantly, at 9.2% per year, during the 1980s, and then fell, but not significantly, through the 1990s, a decline which was projected to continue through 2005.

Subgroups

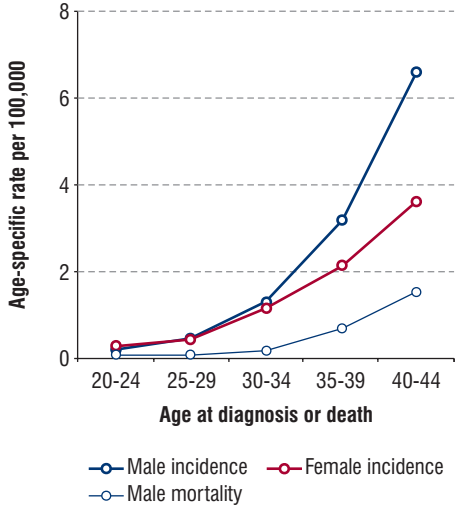
Almost all kidney tumours (96%) arise in the tubular epithelium of the renal parenchyma; the remainder (4%) arise in the renal pelvis or ureter. Tumours arising in the parenchyma are generally referred to as "renal cell carcinoma" and are predominantly adenocarcinomas, while those originating in the renal pelvis or ureter are most commonly transitional cell carcinoma (this is also the most common morphology for bladder cancers). More detailed examination by morphology is limited by the fact that, in some jurisdictions, the proportion of kidney cancers with unspecified morphology is relatively high (e.g., over 12% in this age group in Ontario).

Interpretation of patterns and trends

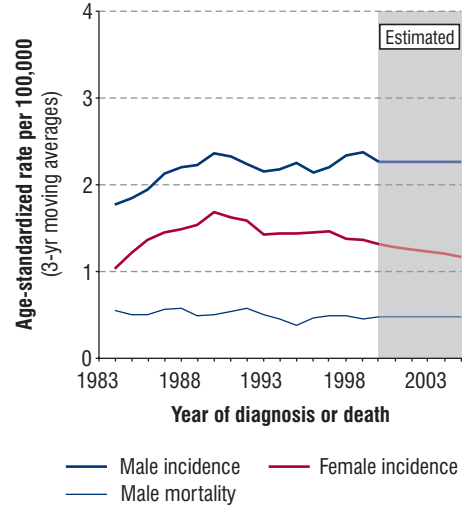
Rising incidence rates in the 1980s were due in part to increased early diagnosis of tumours, both intentional and incidental, reflecting the wider availability of imaging procedures such as ultrasonography and computed tomography beginning in the 1970s.[102] However, increases in the US have not been confined to early tumours,[34] indicating the importance of other factors such as obesity or smoking. The rising prevalence of obesity, and increases in body mass generally, may have played a role. It is unlikely that smoking is a major explanatory factor because the incidence trends do not mirror smoking patterns.

The rise and subsequent stabilization or decline in incidence rates may reflect a common phenomenon when a new method of early diagnosis is introduced: pre-existing tumours are diagnosed earlier, causing incidence to rise. Once this pool of pre-existing tumours has been exhausted, incidence falls temporarily.

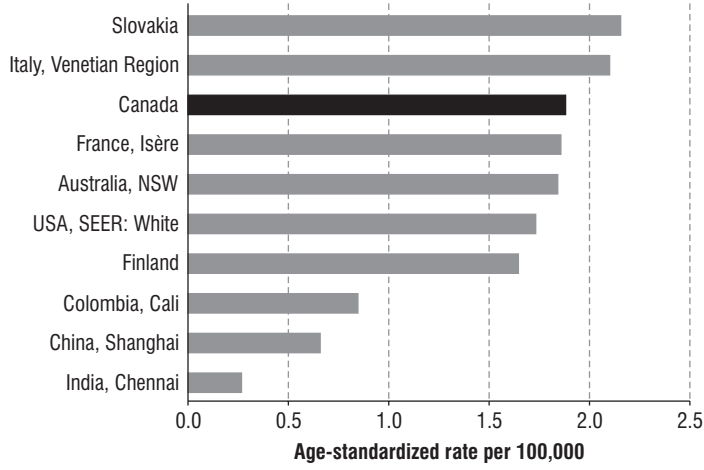
Kidney cancer
Age-specific rates for young adults
Canada, 1990–1999



Kidney cancer
Age-standardized rates for adults
aged 20–44, Canada, 1983–2005



Kidney cancer
Age-standardized incidence rates* for adults aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Both sexes combined

LIP, ORAL CAVITY AND PHARYNX

Cancers discussed here include lip (excluding skin of lip), tongue, gum, mouth, and pharynx (including nasopharynx). Cancers of the salivary glands are not included in this grouping. Lip, oral cavity and pharyngeal cancer in young adults rank fifteenth in terms of incidence and fourteenth in terms of mortality. Mortality rates are substantially lower than incidence rates, reflecting the reasonably high survival for this cancer.

Lip, oral cavity and pharynx
Summary statistics, 1990-1999

	Males	Females
Cases	1,598	681
Incidence rate	2.6	1.1
% of all cancers	4%	1%
Incidence rank	11th	15th
Deaths	315	124
Mortality rate	0.5	0.2
5-yr survival	79%	75%
PYLL rank	8th	13th

Age-specific rates

Incidence rates for males double with each successive age group, while for females the rates in each age group are 1.6 times the rate in the previous age group. From the youngest age group to the oldest, male incidence increases more than 20-fold, while female incidence increases 10-fold. Male age-specific mortality rates also increase at a constant rate of 2.3 times between successive age groups; again, across the age range, mortality increases 20-fold.

Geographic variation

The highest reported rates worldwide for this group as a whole are in Hong Kong (16.8 per 100,000), and the lowest in Scandinavia and South and Central America.[134] Along with the US, Canada ranks in the lower half among the ten countries selected for comparison.

Trends

Incidence and mortality for young men remained stable over two decades and were expected to remain stable through 2005. Incidence in females rose significantly over the same period, at an average of 1.5% per year, but was projected to stabilize through 2005.

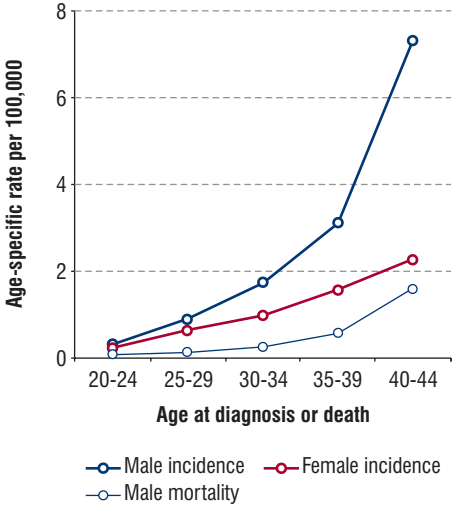
Subgroups

Most of these cancers are squamous cell carcinoma (83% of microscopically confirmed cases). The most common locations are the tongue (26%) and nasopharynx (24%), followed by the lip (18%), mouth and gums (18%), oropharynx (10%), hypopharynx (2%) and ill-defined locations (2%). The precise site of origin may be difficult to determine for cancers in junctional areas, such as between lip and skin or between lip and oral mucosa, and some misclassification may occur between tumours of the hypopharynx and the supraglottic part of the larynx.

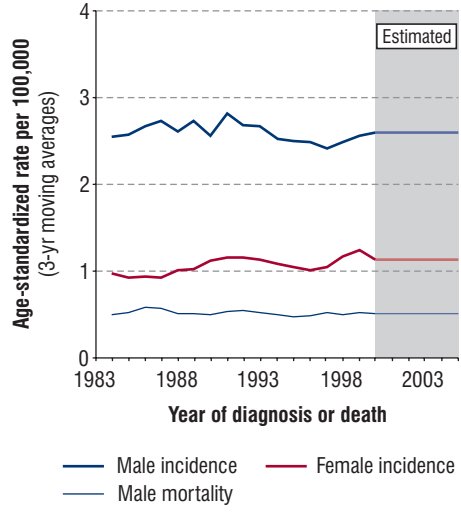
Interpretation of patterns and trends

Although alcohol consumption is considered an important risk factor for lip, oral cavity and pharyngeal cancer, trends in this cancer relate more closely to trends in smoking than to alcohol consumption. Stable incidence in males and rising incidence in females may reflect the later decline in smoking rates in females compared with males. Excluding nasopharynx from the trends (not shown) changes the male trend to a non-significant decrease and the female increase to non-significant, which may demonstrate more clearly the effect of previous declines in smoking rates. The very high rates in Hong Kong are explained by the high incidence of nasopharyngeal cancer.

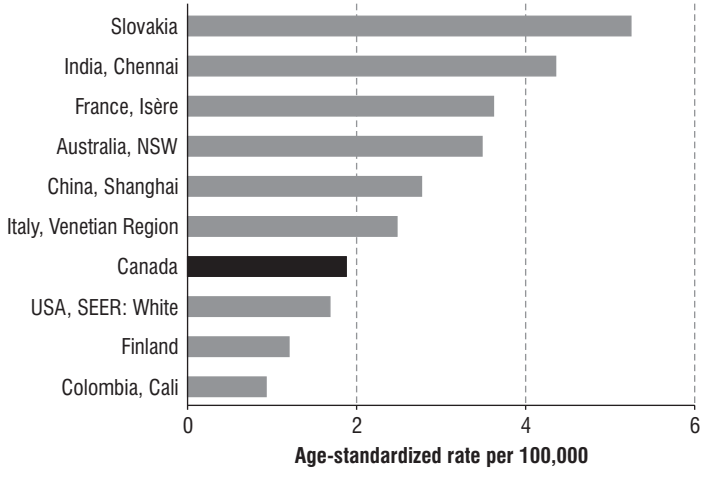
Lip, oral cavity and pharyngeal cancer
Age-specific rates for young adults
Canada, 1990–1999



Lip, oral cavity and pharyngeal cancer
Age-standardized rates for adults
aged 20–44, Canada, 1983–2005



Lip, oral cavity and pharyngeal cancer
Age-standardized incidence rates* for adults aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Both sexes combined

BLADDER

Bladder cancer among young adults ranks seventeenth in terms of both incidence and mortality. Mortality rates for bladder cancer are substantially lower than incidence rates, reflecting the reasonably high survival for this cancer.

Bladder
Summary statistics, 1990-1999

	Males	Females
Cases	1,102	516
Incidence rate	1.8	0.8
% of all cancers	3%	1%
Incidence rank	14th	16th
Deaths	97	49
Mortality rate	0.2	0.1
5-yr survival	80%	82%
PYLL rank	11th	14th

Age-specific rates

Age-specific incidence rates for bladder cancer are typical of most epithelial cancers, with a constant rate of increase with increasing age. For males, bladder cancer more than doubles with each successive age group, while for females, the rate of increase is slightly less than twice. From the youngest age group to the oldest, male bladder cancer incidence increases 18-fold, while the increase for females is 13-fold.

Geographic variation

Canadian rates are relatively high for this age group, consistent with the evidence that rates are generally high in western Europe and North America.[134] Canada ranks fourth among the ten countries chosen for comparison. Rates are appreciably lower in most of Asia.

Trends

Trends in the incidence of bladder cancer over the 1980s could not be estimated with confidence because non-invasive tumours were included with invasive cancers for Ontario and Alberta.[108] During the 1990s, however, bladder cancer incidence in males and females fell significantly by 3.6% and 3.4% per year. Incidence was projected to continue falling between 2000 and 2005.

Subgroups

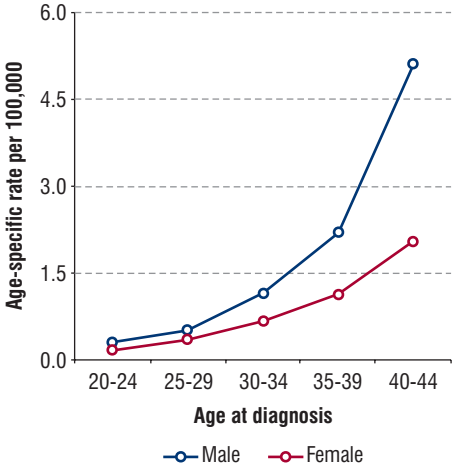
Most bladder cancers are transitional cell carcinoma (91% of microscopically confirmed cases). The remaining morphologies include squamous cell carcinoma, adenocarcinoma, undifferentiated carcinoma and rare morphological types.

Interpretation of patterns and trends

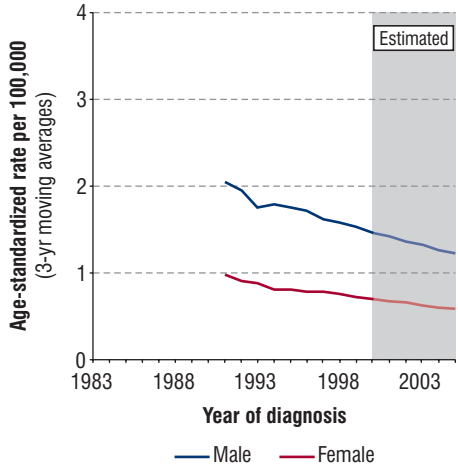
The most important risk factor for bladder cancer is smoking, followed by certain occupational exposures. The male preponderance of bladder cancer in young adults, although not as striking as that reported for all ages combined,[25] may be attributable to the higher rate of smoking among males before 1985 and to differences in occupation. The male preponderance has been reported, however, to persist in the absence of tobacco smoke and exposure to occupational hazards, leading to suggestions that unidentified environmental, dietary exposures or hormonal factors may play a role.[64, 91]

Much of the variation in bladder cancer incidence internationally may partly be due to different registration practices for low-grade, or non-invasive, papillomas of the bladder.[91, 159]

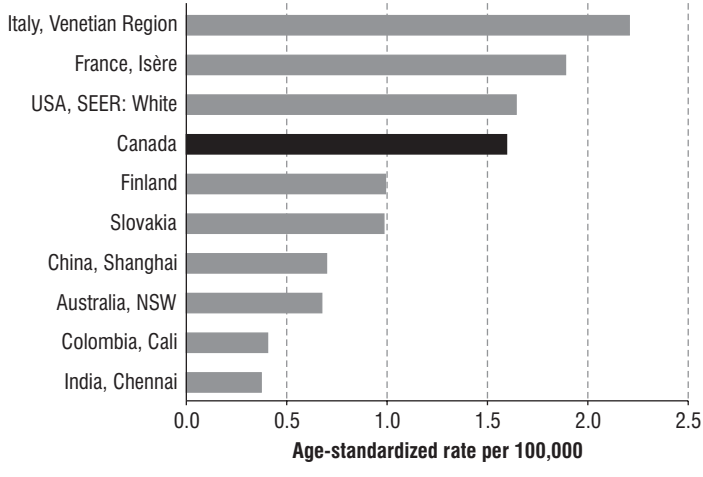
Bladder cancer
Age-specific incidence rates for young adults, Canada, 1990–1999



Bladder cancer
Age-standardized incidence rates for adults aged 20–44, Canada, 1990–2005



Bladder cancer
Age-standardized incidence rates* for adults aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002
* Both sexes combined

BODY OF UTERUS

Cancer of the body of the uterus (excluding cervix) ranks eighteenth in terms of incidence among young adults, and sixteenth in terms of mortality. The very low mortality rate for this cancer reflects the high survival rate.

Body of uterus
Summary statistics, 1990-1999

Cases	1,362
Incidence rate	2.2
% of all female cancers	2%
Incidence rank	13th
Deaths	156
Mortality rate	0.3
5-yr survival	94%
PYLL rank	11th

Age-specific rates

Cancer of the body of the uterus is rare among women in their early 20s. Thereafter, incidence rises 2.6 times with each successive age group. From the youngest age group to the oldest, incidence of uterine cancer increases over 100-fold.

Geographic variation

Canada is among several developed countries with relatively high incidence, including the US and some areas of eastern and western Europe.[134] Canada ranks fourth among the ten countries chosen for comparison. Within Canada, rates are significantly higher in Atlantic Canada than in other regions.

Trends

Incidence of uterine cancer fell significantly, at an average of 1.3% per year during the period 1983–1999, from 3.0 to 2.4 per 100,000. Incidence was expected to stabilize for the years up to 2005.

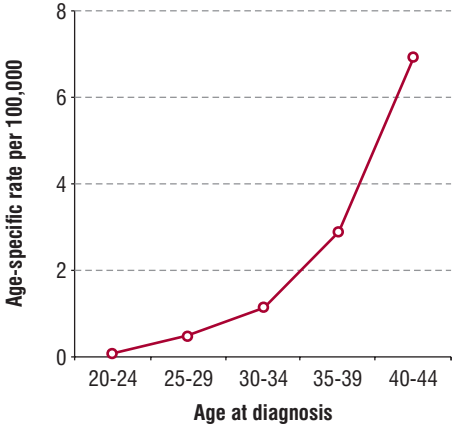
Subgroups

Most uterine cancers are adenocarcinoma (93% of morphologies); a further 3% are other and unspecified carcinomas, while morphology is unspecified for the remaining 4%. Of the adenocarcinomas, 28% are endometrioid and 18% are other specified adenocarcinomas, while 54% are unspecified adenocarcinomas. As only 3% of uterine cancers were coded as unspecified as to anatomic subsite, the possibility that this group contains some cervical cancers is unlikely to affect conclusions about trends in cancer of the uterine corpus. Smooth muscle tumours of the uterus are included with other sarcomas in this publication.

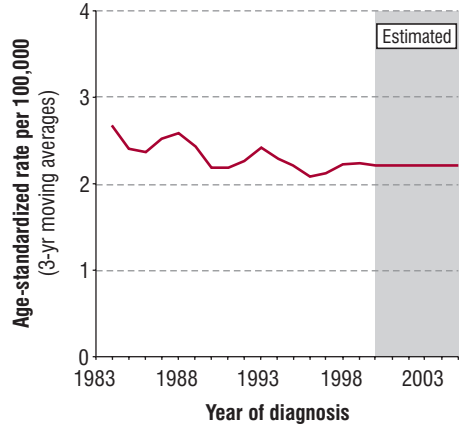
Interpretation of patterns and trends

The decrease in incidence of uterine cancer resembles a decline for this cancer in all ages for the same period, and declines in younger women in the US and northern Europe.[25, 168] A change from increasing incidence in earlier decades may be most related to changing oral contraceptive formulations. The sequential oral contraceptives marketed in the 1960s and 1970s increased the risk of endometrial cancer in the late 1970s and early 1980s.[140] Although survey data on oral contraceptive use show little change for the 20–44 age group as a whole between the mid 1980s and the mid 1990s, usage appeared to increase for women in their late 20s and particularly in their early 30s. These would have been combined oral contraceptives, which provide a protective effect.[114] Higher rates of uterine cancer for the Atlantic region are unlikely to be related to oral contraceptive use, which was actually higher in Atlantic Canada than in other regions in the late 1990s.[48] They may be related to the high levels of obesity and low levels of physical activity in Atlantic Canada as compared with other regions.[28, 21]

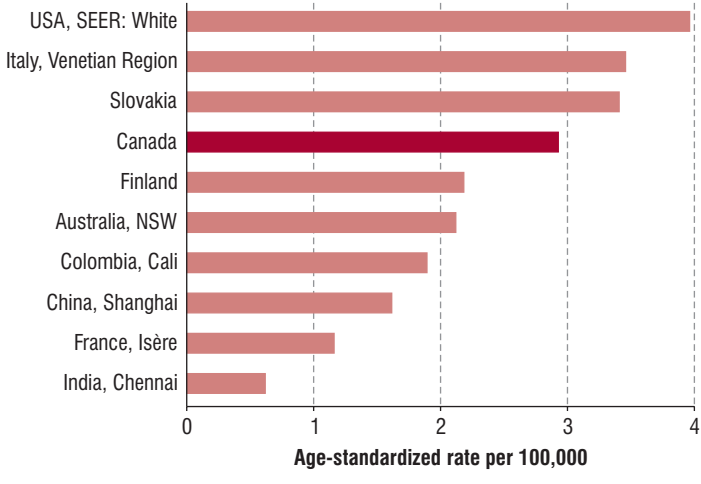
Uterine cancer
Age-specific incidence rates for young females, Canada, 1990–1999



Uterine cancer
Age-standardized incidence rates for females aged 20–44, Canada, 1983–2005



Uterine cancer
Age-standardized incidence rates for females aged 20–44
Selected international regions, 1993–1997



Source: Parkin et al., 2002

RISK MODIFIERS IN RELATION TO YOUNG ADULT CANCER PATTERNS

With some exceptions, cancer results from a complex interplay of factors rather than from a single factor. For cancers that occur in both younger and older adults, individual risk modifiers may vary in their strength or relative importance at younger and older ages. Risk modifiers for pre- and post-menopausal breast cancer, for instance, appear to differ somewhat.[36] A difficulty in determining risk modifiers for cancer in young adults is that studies of cancers over a broad age range often do not report risk modifiers by age, and are dominated by the fact that most cancers occur at age 50 or older. Genetic factors may have a far greater effect in younger than in older adults.

The 18 cancers discussed in this publication are of two general cell types and show several distinct age-specific incidence patterns. Eight of the 18 cancers are non-epithelial; that is, they originate in such tissues as smooth muscle, melanocytes, stem cells and lymphatic tissue. Two types, testis and Hodgkin lymphoma, peak in the young adult age group and then decline. Cancer of the testis peaks in the 30–34 age group, and Hodgkin lymphoma at ages 20–24. Kaposi sarcoma peaked abruptly in the early 30s for the time period considered here, a pattern which suggests a cohort effect when considered with the incidence time trend in the 1980s and 1990s. For the other non-epithelial cancers described here, incidence is at least 1.0 per 100,000 population in the 20–24 age group and rises only gradually across the young adult age range. This is the age-specific pattern for non-Hodgkin lymphoma (NHL), brain and other central nervous system cancers, bone and soft tissue sarcomas, leukemia and melanoma. Melanoma, Kaposi sarcoma, Hodgkin lymphoma and cancer of the testis show more geographic variation in incidence than the epithelial cancers among the various national or regional cancer registries compared in this document.

Epithelial cancers arise in the cells that line the internal and external surfaces of the body. Breast, colorectal, kidney, oral, bladder, lung, thyroid and cancers of the ovary, uterus and cervix are the epithelial cancers important among young adults. All are more common at older ages than in the young adult range. Incidence in most of the epithelial cancers is very low in the 20–24 age group and rises across the young adult age range, abruptly for some cancers. The two exceptions are cancer of the cervix and of the thyroid in females, both of which show a pattern suggesting a cohort effect in the CYAC age range during the 1980s and 1990s. Cervical cancer shows more geographic variation than the other epithelial cancers considered here.

Risk modifiers, as far as evidence to date has established, are to some extent distributed in accordance with these cell types and age distributions, as Table 5 demonstrates. Some behaviours—physical inactivity, obesity, high alcohol consumption, various aspects of diet, and tobacco use—are established risk factors for epithelial but not, in general, for non-epithelial cancers. Appearing most important for cancers that occur most often in the second half of the life span, they may depend on longer duration of exposure or late effects of early exposure. Reproductive and hormonal factors and various medical conditions are associated more with epithelial than non-epithelial cancers. Radiation and other occupational or environmental exposure, and viruses and/or immunosuppression are established risk factors for some cancers in both groups. It must be remembered that this risk modifier pattern may merely reflect the fact that

studies of cancers that occur at older ages will reflect risk modifiers at those ages. Studies of the same cancers in young adults may reveal different risk modifiers.

Several cancer risk modifiers have shown marked changes in prevalence in Canada over at least the past 20 years: smoking rates have declined, but not in a linear fashion and much more recently for females; sun exposure likely has changed with the introduction of sunscreens and changes in jobs and outdoor leisure pursuits; Pap test screening has increased; obesity has become increasingly prevalent despite an increase in the self-reported leisure activity level of Canadians; alcohol consumption dropped during the 1970s and 1980s but rose in the 1990s; fertility rates have fallen dramatically and age at first birth has risen; and oral contraceptive use has remained constant, but the formulations have changed.

A rationale for studying cancers in young adults is that the associations with risk modifiers are likely to be clearer because young people have fewer accumulated exposures, and that cancers arising during young adulthood may be considered "sentinel events" providing a warning of "new" or changing exposures. For these two reasons, one might expect trends in some cancers in young adults to best reflect trends in risk modifiers.

The most recent trends in the period 1983–1999 showed incidence increasing in at least one sex for 7 of the 18 cancers and falling in 8 of the 18. In large part, cancers decreasing in incidence are those for which risk modifiers are best understood and some prevention strategies possible. In particular, there is suggestive evidence that the three main risk reduction recommendations of avoiding smoking, minimizing sun exposure and regular Pap smear screening are being increasingly followed by young Canadians and that these trends are paying off. For several cancers with increasing trends—testis, thyroid and female lymphomas in particular—reasons for the increase are not understood.

Table 5

Summary of epithelial and non-epithelial cancer patterns in young adults: risk modifiers*, time trends, incidence across age range

		Risk modifiers										Trends			
												Incidence		Mortality	
		Physical inactivity	Obesity/ height	Diet	Alcohol	Tobacco smoke	Reproductive/ hormonal	Medical conditions	Radiation (ionizing/ ultraviolet)	Other occupational/ environmental	Viruses/ altered immunity	M	F	M	F
Epithelial	Breast	✓	✓		✓		✓	✓	✓				↑		↓
	Colorectal	✓	✓	✓	✓	✓		✓				↓	↓	↓	↓
	Uterus	✓	✓				✓						↓		
	Kidney		✓			✓		✓	✓			↑	↔		
	Lip, etc.			✓	✓	✓					✓	↔	↑		
	Bladder			✓		✓		✓	✓	✓		↓	↓		
	Lung			✓		✓		✓	✓	✓		↓	↔	↓	↑
	Thyroid						✓	✓	✓			↑	↑		
	Ovary						✓		✓				↓		↓
	Cervix					✓	✓				✓		↓		↓
Non-epithelial	Sarcoma						✓	✓	✓	✓		↔	↔		
	Leukemia					✓			✓	✓		↔	↔	↔	↓
	Brain								✓			↔	↔	↔	↔
	Melanoma								UV			↓	↔	↓	
	NHL										✓	↔	↑	↔	↔
	Hodgkin										✓	↔	↑		
	Testis						✓	✓				↑			
	Kaposi										✓	↓			

Incidence low in young adults, rises exponentially across CYAC age range

Incidence rises gradually across young adult age range

Incidence rises, then flattens across CYAC age range (for females only, in thyroid cancer)

Incidence peaks in young adults, then falls

↑ / ↓ Significant increase/decrease in average annual percent change in most recent trend within period 1983–1999

↔ Non-significant increase/decrease in average annual percent change in most recent trend within period 1983–1999

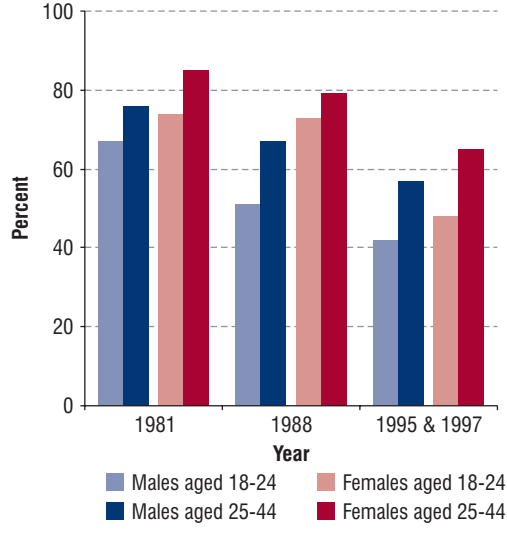
* Supporting references in risk modifier sections of text

Physical inactivity, obesity and diet

Physical inactivity is established as a convincing risk factor with a moderate contribution to the risk of colon cancer,[75, 82] a small contribution to the risk of breast cancer,[75] and as a probable, small-impact contributor to the risk of cancer of the uterus.[51, 140, 75] The proportion of Canadians in the CYAC age group reporting low levels of leisure-time physical activity declined between 1981 and the mid- to late 1990s (Figure 20).[21] The decline occurred earlier for males than females and physical inactivity is more common in females. Canadians reporting more leisure-time physical activities tend also to report more physical activity incorporated into daily chores and commuting.[21, 67] Although reported leisure-time physical inactivity shows a general east-west gradient, with higher proportions of the population inactive in the Atlantic provinces, proportions of physically inactive individuals have declined in all regions of Canada.[21]

Figure 20

Percentage of Canadians aged 18–44 who are physically inactive*



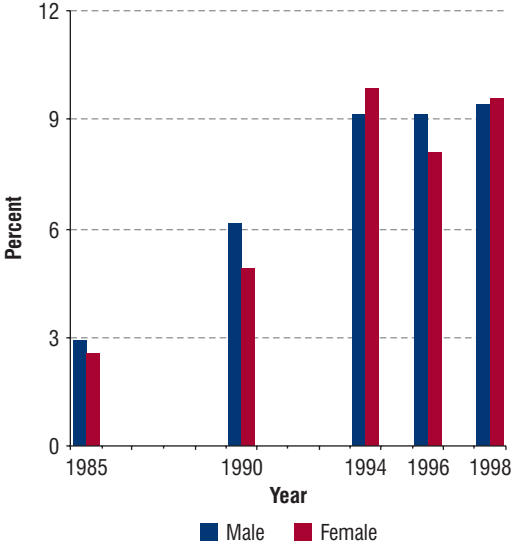
Source: Cameron et al., 2000

* Energy expenditure <3 KKD (kilocalories/kilogram of body weight/day); 3 KKD is roughly equivalent to walking one hour every day

Obesity (usually measured by body mass index) has been designated a convincing risk factor with a large impact on the risk of kidney and endometrial (uterine) cancer and a moderate impact on the risk of colon cancer, by a review panel for the US Institute of Medicine.[75] Central adiposity (usually measured as a high waist:hip ratio) also increases the risk of colon cancer.[82] Weight and body mass index were positively associated in some studies with cancer of the ovary in premenopausal, but not postmenopausal, women.[46, 94] Premenopausal breast cancer risk increases with increasing height, but decreases with higher weight or body mass index, and there is no association with increased central adiposity.[50] A woman's own high birth weight is associated with increased risk of breast cancer at young ages.[74]

Figure 21 shows that the proportion of obese men and women aged 18–34 increased from almost 3% in 1985 to approaching 10% in 1998. These figures, from body mass index (BMI, weight in kilograms/(height in metres)^2) ≥ 30, are based on self-reported height and weight and are likely to be underestimates. The trend continues an increase in obesity seen in Canadian adults from at least as far back as the early 1970s. The prevalence of obesity in the 1990s was highest in the Atlantic region, Saskatchewan

Figure 21
Percentage of Canadians aged 18–34 with BMI* ≥30



Source: Data provided by the Public Health Agency of Canada (HPS 1985; HPS 1990; NPHS 1994; NPHS 1996; NPHS 1998)
* Body mass index: weight in kilograms/(height in metres)^2

and Manitoba.[28] There has also been a general shift towards higher body mass in young men and women.[88]

Diet has not been studied specifically in younger adults. From studies across wider age ranges, the consistency of findings across a variety of cancer sites argues in favour of considering it an important etiologic factor. Diet may be more important in relation to cancers that appear later in life and may operate indirectly via body size (e.g. obesity),[89] metabolism, or hormones. Phytoestrogens, for example, present in many foods, may be protective themselves through their hormonal effects, or may be biomarkers of a healthy diet.[2] The general tendency suggested by research on diet is that high consumption of fresh fruits, cruciferous vegetables, complex carbohydrates, fibre and some vitamins and minerals (e.g. vitamins A and E, calcium, selenium and carotenoids) is protective, while high consumption of sugars and animal fats and protein (particularly from red meat), cured or smoked meat or fish and salted fish confers risk.

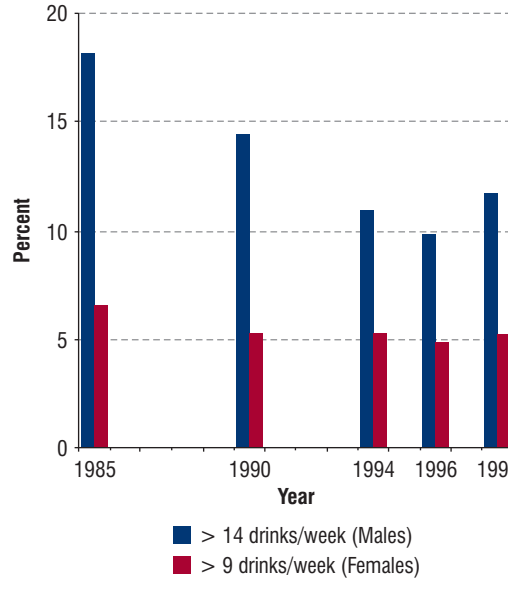
Some or all of these associations have been noted for approximately half of the cancers examined here: brain,[152] ovarian,[95, 148] colorectal,[77, 75] lung,[75] kidney,[143] oral and pharyngeal,[106, 146] nasopharynx[120] and uterine[140] cancers. Dietary factors have been proposed as a possible explanation for the persistence of the excess risk of bladder cancer in men in the absence of known risk factors (tobacco smoke and occupational hazards).[64, 91] Thyroid cancer risk appears to be associated with iodine intake, although the relationship is complex. On the basis of ecologic data, iodine deficiency appears to be involved in the development of thyroid cancer, especially follicular carcinoma, although iodine excess also appears to play a role, particularly for papillary carcinoma.[168, 20] Associations have been harder to identify in case-control studies,[138] perhaps because of relatively small variation in iodine intakes within, as opposed to between, populations.

Alcohol

Alcohol consumption is established as having a large impact on the risk of oropharyngeal cancers[75, 106, 146] and a moderate impact on the risk of breast cancer.[75, 51] It is a probable risk factor, with a moderate impact, for colorectal cancer.[75] There is suggestive evidence that high alcohol intake is a risk factor for endometrial cancer among younger women in particular.[183] Canadian low-risk drinking guidelines give an upper limit of 14 drinks per week for men and 9 for women.[30] Survey data on reported number of drinks over the preceding week show a decline between the mid-1980s and

the mid-1990s in proportions of males and females aged 18–44 exceeding the weekly upper limit (Figure 22). The proportion of males exceeding the guideline is consistently higher than the proportion of women, and shows a slight increase toward the end of the 1990s. This pattern is consistent with estimates of per capita pure alcohol consumption for Canadians aged 15 and over derived from sales data, which increased from the 1950s to the mid-1970s and then declined, but showed a slight increase in the late 1990s.[142] Survey data for ages 18–29 (not shown) show a slightly higher proportion of both sexes exceeding the weekly guideline for every survey year than for the whole 18–44 age group.

Figure 22
Percentage of Canadians aged 18–44 exceeding alcohol guidelines



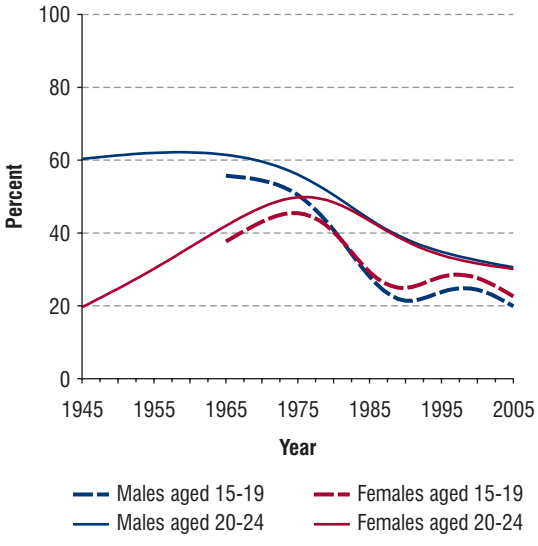
Source: Data provided by the Public Health Agency of Canada (HPS 1985; HPS 1990; NPHS 1994; NPHS 1996; NPHS 1998)

Tobacco

As with cancer in adults at all ages, cigarette smoking is thought to account for a substantial proportion of several cancer types. The earliest studies linking tobacco smoke to cancer were published in the 1950s, when case-control studies demonstrated that smoking was an important risk factor for lung cancer.[42] Since then, the evidence has been building to connect smoking to a wide variety of other cancers. Among the cancer types evaluated by the International Agency for Research on Cancer (IARC) as associated with tobacco smoking,[150] six are important cancers in the 20–44 age group: lung, bladder, upper aerodigestive (similar to the "Lip, oral cavity & pharynx" group in this monograph), kidney, cervix and myeloid leukemia. Smoking in cancer of the cervix appears to be a co-factor with human papilloma virus (HPV) infection (see below). Although a recent review by the US Institute of Medicine regarded the evidence as convincing for smoking and colon cancer, an apparent long latency between exposure and disease makes it a less likely risk factor for colorectal cancer in the CYAC age range.[75, 56] While studies of smoking and breast cancer have shown mixed results, there is some evidence that cigarette smoking increases risk for premenopausal women. In particular, increased risk is suggested for smoking commenced in the mid-teens or earlier, of long duration or before a first full-term pregnancy, and in women with certain genotypes, or with exposure to environmental tobacco smoke.[11, 172, 87] Exposure to environmental tobacco smoke (second-hand smoke) has been categorized as a carcinogen and increases the risk of lung cancer.[75, 168]

Current trends in tobacco-related cancers reflect smoking patterns from earlier decades due to the long latency period, and current smoking patterns foreshadow cancer trends in the next 20 years.[3] The smoking habits of Canadians aged 15–24 from the 1940s to the

Figure 23
Percentage of Canadians aged 15–24 currently smoking, 1945–2005



Sources: Ferrence 1988, Statistics Canada 2004, Canadian Council for Tobacco Control 2002, Health Canada 2000

present are, therefore, of particular interest when trying to understand the relationship between tobacco and cancers diagnosed in the 20–44 age group.

Estimates of the proportion of Canadians aged 15–19 and 20–24 who smoke cigarettes were obtained from a variety of sources.[47, 27, 68] Cubic splines have been fit to the data to produce smoother curves. Figure 23 shows that smoking among males aged 20–24 peaked at 62% in the late 1950s and has been declining since. Currently, around 31% of males 20–24 smoke, the lowest proportion in 60 years. Smoking among females peaked at 50% in the mid 1970s and is now approximately 30%.

Smoking trends among teens aged 15–19 are much more complex. Over 50% of boys smoked in the late 1960s and early 1970s, but rates declined sharply to 20% around 1990. Smoking among girls increased from 1965–1975, peaking at 45% before falling to 25% in 1990. Smoking rates among teenage girls began to outstrip rates in males in the late 1970s and early 1980s. Currently, approximately 22% of girls aged 15–19 and 20% of boys are current smokers.

Smoking among teens increased through the 1990s before falling again. Teenagers are more sensitive to the pricing of tobacco products, and this rise has been linked to the availability of cheaper cigarettes through smuggling and the 1994 federal tobacco tax cuts.[182]

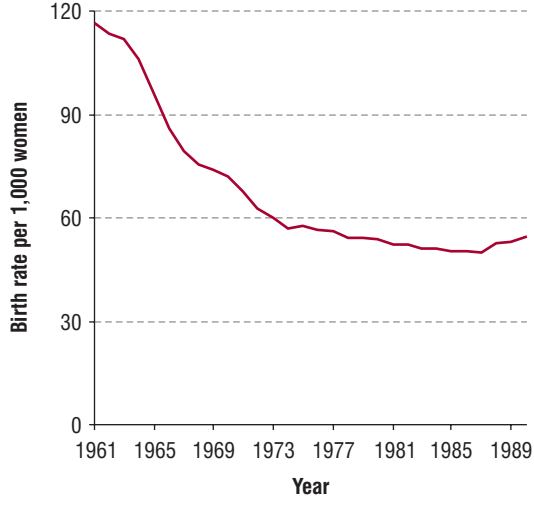
Reproduction and hormonal factors

Hormonal factors operate before birth and throughout the life span. They appear to be risk modifiers for several cancers, most notably cancers of the sex organs. Generally speaking, high exposure to estrogen appears to confer risk, while use of oral contraceptives tends to lower risk. Some hormonal factors are likely co-factors rather than causal factors for cancer of the cervix, in that they are associated with greater exposure to the causative agent, HPV infection.

Prenatal and early childhood exposures. Exposures in early childhood, perhaps *in utero*, are believed to play a role in the etiology of testicular cancer.[186, 104] Elevated levels of maternal estrogen during pregnancy, as indicated by low parity and higher maternal age, have been associated with increased risk.[185] Early age at puberty may increase the risk of testicular cancer.[184, 175] It is also possible that greater exposure to estrogen-like compounds in the environment increases risk, although supporting epidemiologic evidence is scant.[149]

Figure 24

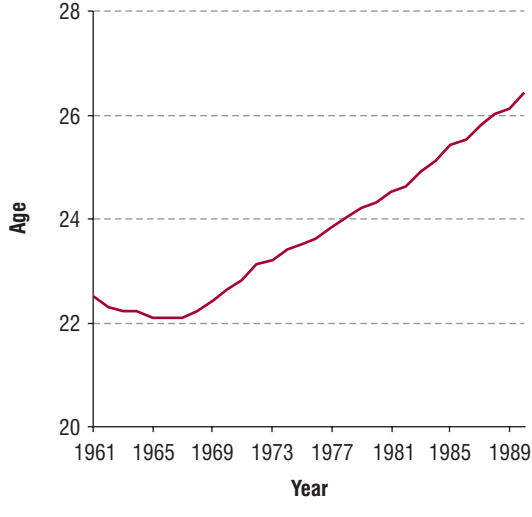
General fertility rates* for women aged 15–49 Canada, 1961–1990



Source: Statistics Canada 1993
* Standardized to the Canadian 1971 female population

Figure 25

Median age of mother at first live birth Canada*, 1961–1990



Source: Statistics Canada 1993
* Newfoundland and Labrador not included

Factors that contribute to high levels of unopposed estrogen. For women, one theory holds that high levels of unopposed estrogen increase cancer risk. Early age at menarche, late menopause, later age at first birth, lower parity and use of exogenous estrogen result in high levels of unopposed estrogen. One or all of these factors is associated with cancers of the breast, uterus (endometrial and sarcoma) and ovary (Table 6). Endometrial cancer in younger women is associated with proliferative endometrium, depending on continuous estrogen stimulation, and may have different causes from the atrophy-associated endometrial cancers occurring in older women.

Table 6
Risk modifiers related to high levels of unopposed estrogen

	Breast cancer	Uterine cancer		Ovarian cancer
		Endometrial	Sarcoma	
Early age at menarche or irregular menses	✓* [36]	✓* [187, 128, 183, 63, 140]	✓ [155]	
Lower parity	✓† [36]	✓ [140]		✓ [148]
Later age at first full-term pregnancy	✓* [36, 4]			
Late menopause			✓ [155]	
Induced abortion			✓ [155]	
Use of exogenous estrogen	✓‡ [37]	✓‡ [125]	✓ [154]	

* Association stronger among younger women

† Association weaker among younger women

‡ May be a risk factor for some of the oldest women in this young adult range; use of hormone replacement therapy even for less than five years has been shown to increase the risk of endometrial cancer[13], while risk for breast cancer may not be raised until duration of use is five years or more and time since first use is even longer[37]

Although pregnancy temporarily increases the risk of breast cancer in premenopausal women, parity does lower risk, although not so much as for older women.[36] Parity also lowers the risk of cancer of the ovary, with a reduction of 13–19% per pregnancy,[148] while either infertility itself, or the use of fertility drugs, may increase the risk.[95] High parity is likely a co-factor for cancer of the cervix, operating only in the presence of oncogenic HPV infection. Figure 24 shows a dramatic fall in fertility rates in Canadian women across the 1960s, and a continued decline until the mid 1980s.

Later age at first birth, like early menarche, is associated with a higher risk of premenopausal than of postmenopausal breast cancer.[36] Median age at first live birth, shown in Figure 25, increased steady from age 22 in the early 1960s to age 26 in 1990.

Breastfeeding protects against breast cancer, and may counteract the adverse effect of late age at first birth.[85] It may lower the risk of cancer of the ovary and sarcoma of the uterus by suppressing ovulation.[59, 155]

Oral contraceptive use is protective for ovarian cancer and (as combined estrogen-progesterone oral contraceptives) for uterine cancer. Its protective role has been important for ovarian cancer, estimated at 40% in ever users, increasing with duration of use, and persisting for at least 10–15 years after cessation.[95, 148] (It is, however,

associated with an increased risk of germ cell ovarian tumours.)[196] It may be protective for soft tissue sarcoma.[154, 130] However, recent or current oral contraceptive use is a risk factor for breast cancer.[4] Oral contraceptive use as a risk factor for cancer of the cervix is thought, like high parity, to operate only in the presence of oncogenic HPV infection.[49] Data from the Saskatchewan Drug plan show a constant prevalence of oral contraceptive use by 35% of women aged 15–29 from 1977 to 1992.[196] Data in Table 7, from the 1984 Canadian Fertility Survey and the 1995 General Social Survey, suggest that this is probably true for Canada as a whole.[114] National Population Health Surveys show constant proportions of users among women aged 20–44, at 19%, 20% and 20% for the 1994/95, 1996/97, and 1998/99 surveys, respectively.[162-164]

Table 7
Oral contraceptive use by Canadian women[114]

Age group	1984	1995
18-24	44%	38%
25-29	27%	30%
30-34	13%	21%
35-39	5%	6%
40-44	2%	1%

Because thyroid cancer is so much more common in women than in men, particularly in the reproductive age group, hormonal factors have been examined. Epidemiological studies have generally shown only weak or no associations with various menstrual and reproductive characteristics.[124] A small increased risk has been found for current use of oral contraceptives in relation to papillary cancer in particular.[96]

Medical conditions and treatment

Certain medical conditions other than cancer sometimes predispose an individual to cancer. Some of these conditions are inherited while others are acquired, and the degree of cancer risk associated with them varies. The mechanisms by which some medical conditions predispose an individual to cancer are often unknown.

Although undescended testicle is often mentioned as a strong risk factor for testicular cancer, the two conditions may have a common etiology, involving genetic or lifestyle factors or environmental exposures.[185, 137, 17] Other medical conditions associated with specific cancers are: goitre and benign thyroid nodules/adenomas with thyroid cancer;[138] benign breast disease with breast cancer;[62] inflammatory bowel disease (Crohn's disease or ulcerative colitis) with colorectal cancer;[14] several pre-existing bone defects, including Paget's disease (osteitis deformans), with osteosarcoma and chondrosarcoma;[119] urinary tract infections with bladder cancer;[168] type 2 diabetes and, among premenopausal women, polycystic ovary syndrome with cancer of the uterus.[140] Medication use appears to modify the risk of some cancers; reduced risk of colorectal cancer, for instance, is associated in observational studies with the use of nonsteroidal anti-inflammatory drugs, including aspirin.[176]

Radiation

Ionizing (high energy) radiation is a known human carcinogen.[80] Approximately 80% of our exposure to ionizing radiation is from natural sources, usually at very low dose rates, such as cosmic rays and naturally-occurring radioactive elements in the earth's crust and air. Artificial (man-made) sources of ionizing radiation include nuclear weapons tests, nuclear facilities, uranium mines, mills and plants, and X ray devices. Although ultraviolet radiation is traditionally considered a non-ionizing form of radiation, a portion of the ultraviolet spectrum has energy similar to some ionizing

radiation. Radiation is an important causal factor for two of the most common cancers in young adults—malignant melanoma and thyroid cancer.

External exposure to ionizing radiation from medical diagnosis or treatment, or from occupational or environmental sources, such as nuclear weapons use or nuclear accidents, increases the risk of thyroid cancer.[147, 111] Risk increases with dose, more steeply so for exposure occurring in childhood compared to later life.[147, 157] Studies of internal exposure to ionizing radiation in the form of I-131 treatment for hyperthyroidism have been negative,[44] although cases of thyroid cancer subsequent to treatment have been reported.[71] There have been fewer studies of thyroid cancer following the use of I-131 for diagnostic purposes. No excess risk was found in three cohort studies.[57, 61]

Although ionizing radiation, often from medical exposures or occupational exposures, is linked to several other cancers (breast, kidney, bladder, lung, ovary, bone and soft tissue sarcoma, leukemia, and cancers of the brain and other central nervous system), it accounts for a relatively small proportion of them. Exposure to ionizing radiation during adolescence or earlier is a potent risk factor for breast cancer in adulthood.[62] External radiation therapy for other cancers induces a small proportion of bone and soft tissue sarcoma, with latency between two and 40 years (median eight years)[194] and leukemia, primarily acute myeloid leukemia.[136] Radon, a naturally occurring radioactive element present in geographic areas where rock and soil contain uranium, can accumulate to high levels in buildings. It now appears to be a risk factor for lung cancer in members of the general population exposed to residential radiation, in addition to the long-established link for underground miners.[93]

The major risk factor for cutaneous malignant melanoma (CMM) is exposure to ultraviolet (UV) radiation, particularly from the sun. Over 90% of melanoma at mid-latitudes is probably related to over-exposure to sunlight.[9] Although most human exposure to UV is solar in origin, non-solar sources include tanning equipment and some special-purpose lights (e.g., UV lights used to treat psoriasis; special photographic lights).

Current epidemiologic data suggest that both the amount and the degree of intermittency of UV radiation exposure determine risk of melanoma.[7] This "intermittent exposure" hypothesis posits that an increased amount of exposure with a fixed degree of intermittency increases risk and vice versa. Amount of exposure is a function of the intensity of UV radiation and time exposed. There is some evidence that use of tanning equipment also increases risk of melanoma.[179]

The fact that CMM is a common cancer in the young adult age group suggests a role for UV radiation exposure early in life. It is not clear whether this is simply because everyone is exposed essentially from birth (in contrast to many other carcinogens), in some cases to large doses, or because children and adolescents are particularly susceptible to the carcinogenic effects of UV radiation. There is some evidence to support the latter,[8] although it is not strong or direct.

Other occupational and/or environmental exposures

Occupational carcinogens affect workers often chronically exposed at high levels of a given agent or mixture of agents. Reported research does not usually subdivide exposed workers by age, and the importance of established workplace carcinogens for young adults is unknown. The upper end of the 20–44 age range does, however, allow for at least two decades of exposure for some workers. As for ages below 20, some

young adult cancers have been linked to parental occupational exposures. Research on relationships between cancer and the either widespread, or else usually far lower, exposures to chemical or physical agents in the general population is more difficult and has led to fewer established links. It is possible that such environmental agents are more of a risk than current evidence suggests.

Sarcoma. Risk factors for soft tissue sarcomas are only partially known, because their rarity and heterogeneity make for difficulty in etiological research. Various occupational exposures, especially in farming, forestry, and manufacturing, have been associated with specific soft tissue sarcomas. Risks are not uniform across subtypes. Exposures include phenoxyacetic acid herbicides and chlorophenols (found in some wood preservatives), and their contaminants such as dioxin.[194] Fibrous and myomatous sarcomas have been associated with insecticides used prior to the mid-1950s.[194] Arsenical insecticides and medications, and vinyl chloride exposure during plastics manufacture, are well established causes of hepatic angiosarcomas.[194] Exposure to cutting oil and wood dust are possible occupational risks.[130] Data are limited for lower exposures experienced by the general population.

Separate analysis for young adults from studies of Ewing's sarcoma have found positive associations for those diagnosed at ages 20 and over for the mother working as a labourer, machine operator or driver and for parental handling of insecticides or pesticides, solvents and glues, or car-related chemicals.[177, 178]

Lung cancer. Occupational exposures associated with lung cancer include metals (arsenic, beryllium, cadmium and chromium), silicon minerals (asbestos and silica) and polycyclic aromatic hydrocarbons (PAHs).[168] At the general population level, there is some evidence for a relationship between air pollution (a complex and mix of gaseous and particulate components, varying with time and location) and lung cancer.[29, 168]

Bladder cancer. Specific occupational exposures are probably the second most important risk factor (after tobacco smoking), for bladder cancer in both men and women. Aromatic amines and PAHs are the exposures most consistently found to increase risk.[91] Occupational groups linked most consistently with bladder cancer are painters, machinists, mechanics, textile workers, leather workers, transportation workers, hairdressers and dry cleaners.[91, 160, 86] These known occupational associations may, however, reflect past exposures to chemicals not currently used.[91]

Chlorination by-products in drinking water have been associated with an increased risk of bladder cancer.[29] Arsenic, a contaminant of ground water from both natural sources and mining waste, is an established cause of skin, lung and bladder cancer when present at the high levels found in some parts of the world.[29] Information is incomplete about its carcinogenic potential at low levels, and about the actual range of levels in Canada.

Other cancers. There is limited, and sometimes conflicting, evidence for an association between some other cancers and chemical and other occupational exposures. There is strong evidence linking leukemias to occupational exposure to benzene.[158] Lymphomas have been linked with several agents and occupations. Organic solvents have been linked to both Hodgkin lymphoma[115] and NHL,[153] occupational exposure to wood has been associated with Hodgkin lymphoma[115] and agricultural and industrial workers exposed to phenoxyacetic acid herbicides, organophosphate insecticides, chlorophenols and dioxins may have increased risk of NHL.[153] Some occupational

exposures or groups have been associated with brain cancers: vinyl chloride, petrochemical workers, electrical workers, polychlorinated biphenyls, health professions, agriculture and pesticides, and the rubber and tire industries.[152]

Viruses and altered immunity

Viruses and altered immunity (suppression, deficiency or other factors such as allergies) appear to be particularly important in relation to cancers that tend to arise at younger ages.

Human papilloma virus and cancer of the cervix. The clearest association between a virus and cancer is exhibited in relation to cancer of the cervix. Oncogenic forms of the sexually transmitted HPV, particularly types 16 and 18, are the main cause of cervical cancer.[78, 49] Sexual behaviours traditionally considered risk factors, such as early age at first intercourse and multiple sexual partners, are probably surrogates for persistent infection with oncogenic HPV.[49] HPV is a necessary cause for both squamous cell carcinoma and adenocarcinoma.[78]

Epstein-Barr virus (EBV) and lymphomas and cancer of the nasopharynx. EBV is the major viral candidate in relation to Hodgkin lymphoma, although it is found less often in young adults than in the extreme age groups. For young adults, a still unidentified virus may be involved in the pathogenesis of Hodgkin lymphoma.[84] Chronic exposure to EBV is a suspected causal agent in relation to cancer of the nasopharynx.[120] EBV or HPV have also been proposed to explain why subsets of young individuals with oral and pharyngeal cancer report no history of the known risk factors (smoking or alcohol use).[106]

Human herpesvirus 8 (HHV-8) and Kaposi sarcoma. HHV-8 was found in 1994 to be associated with all forms of Kaposi sarcoma.[33, 5]

Other virus-cancer associations. Considering the role of viruses in early onset cancers, some viruses, possibly EBV or cytomegalovirus (CMV), may be involved in the etiology of testicular cancer.[170] Given their role in some adult cancers rare in Canada, there is speculation that viruses may explain many cases of leukemia.[136]

Immunosuppression and lymphomas and Kaposi sarcoma. Immunosuppression can result from inherited or acquired immune disorders, immunosuppressive therapy during cancer treatment or organ transplantation, and the human immunodeficiency virus (HIV). Immunosuppression is the clearest risk factor for NHL.[153, 52] HIV-induced immunodeficiency accounts for a small proportion of NHL cases and is also suspect in relation to Hodgkin lymphoma.[35, 117, 118] Kaposi sarcoma is the most common AIDS-associated malignancy in North America and Africa. In individuals with AIDS, Kaposi sarcoma is caused by an interaction between HIV, immune system suppression, and HHV-8.[5] AIDS-associated Kaposi sarcoma (there are other types) is much more common in male homosexuals than in other groups with AIDS, such as intravenous drug users, heterosexual women, and hemophiliacs.[13, 73, 83, 5] Therapeutic immunosuppression for transplants and other conditions may cause soft tissue sarcoma and NHL.[194, 153, 52]

The AIDS epidemic in Canada began in the 1980s and ebbed in the early 1990s. It is the clearest example of a viral trend that can be linked to cancer.

Familial aggregation

Aggregations of cancers in families are a result of hereditary factors, shared risk factors, or both, including complex interactions of exposures and inherited susceptibility. Because of this complex interplay, familial aggregation has not been included as a separate column in Table 5. Malignancies important in young adults and occurring more often in persons with a positive family history include sarcoma,[194] melanoma,[113] Hodgkin lymphoma[117, 121] and cancers of the breast,[6] ovary,[59] medullary thyroid cancer,[168] colon and rectum,[168] lung (before age 50),[156, 92, 99] central nervous system,[191] and kidney.[110]

A predisposition to cancer may be inherited either directly via a germ line mutation or indirectly via another risk factor, such as an inherited immune disorder (Hodgkin lymphoma[121] and NHL [153, 52]), skin colour (melanoma[113]), a genetic syndrome (sarcoma,[194] kidney[110]) or an inherited susceptibility to certain exposures (brain[191]). Germ line mutations in some genes identified to date are associated with earlier onset of several cancers. Examples are BRCA1 and BRCA2 mutations with early-onset breast and ovarian cancer,[6] and at least some of the cancers associated with hereditary non-polyposis colorectal cancer (colorectal, endometrial, small bowel, ureter, kidney, ovarian, pancreatic, gastric, brain and hepatobiliary cancer).[26] Hereditary ovarian cancers occur an average of 10 years earlier than sporadic cancers, and are often serous adenocarcinomas.[148]

Genetic factors are implicated in cancers of the nasopharynx[120] and may explain why subsets of young individuals with oral and pharyngeal cancer report no history of the known risk factors (smoking or alcohol use).[106]

> "One thing that's really important is to never ask 'Why me?' That's such a self-destructive question when you have cancer because you start blaming yourself…I do blame some of my life habits…I've done some pretty foolish things that may or may not have contributed to my cancer…there are people who have put way more energy into destroying their lives than I ever have, and they end up with no cancer and they're fine…"
>
> ***Male, Colon Cancer, 31***

Etiologic research

There is relatively little awareness about the unique pattern of cancer in this age group and little research into the reasons for it. Increased research focused on the etiology of cancers in this age group will make prevention and risk reduction possible.

The picture of cancer in the young adult years differs in a number of ways from that of other stages of life. First, tumours are split almost equally between epithelial and non-epithelial; in later life, most cancers are of the former type while in childhood most are of the latter type. Second, cancer is nearly 50% more common in young adult women than men; at all other stages of life there is a male excess. Third, some of the most common cancers are almost unique to this age group—testicular cancer and Hodgkin lymphoma being primary examples. Fourth, a number of the most common cancers are increasing in incidence without any real understanding of why. These unique features strongly suggest some differences in either relevant risk factors or biological mechanisms, or both.

Genetic susceptibility is likely to be a particularly important determinant of cancer risk in young adults, although genetic traits cannot, on their own, explain the recent incidence trends. This population may therefore be ideal for exploring the role of genetic factors, particularly gene-environment interactions, to explain why, with similar environmental exposures, some people get cancer and others do not. Such research may identify subgroups at particularly high risk that can be targeted for preventive actions.

Research and surveillance on the effects of cancer diagnosis and treatment

There is a dearth of information about the effects of a diagnosis of cancer and its treatment on people in this age group. Additional research and surveillance are urgently needed to identify major issues and concerns and therefore priorities for action.

A diagnosis of cancer in young adulthood can have particularly devastating consequences for the individual, even though prognosis is very good for many of the cancers common in this group. On the cusp of their most healthy and productive years, young adults with cancer may be crushed economically, psychologically, emotionally and socially as well as physically. A cancer diagnosis shatters their youthful belief in their own invincibility. They may feel that they face a future of uncertainty: will they achieve their dreams? will they be able to have children? will they be able to find partners willing to accept this uncertainty? do they have an enhanced risk of other serious conditions, due either to aggressive cancer treatment or personal susceptibility?

Because survival is good and mortality rates are dropping, while incidence rates and young adult population size are projected to be either stable or declining only slightly,[165] the number of Canadians living with a cancer diagnosed during young adulthood is only going to increase over at least the next 15 years. Furthermore, enhanced risk of cancer-related adverse health events (such as a second cancer) may become apparent only many years after diagnosis. These add to the urgency of establishing research and surveillance protocols focused on short-, medium- and long-term effects. Only after issues are identified can young adults expect to get the support they need, and can research to reduce long-term treatment risks be undertaken. A very recent example comes from a study that estimated an astonishingly high cumulative risk of breast cancer in women who received

radiation treatment for Hodgkin disease as young adults.[174] The accompanying editorial[107] urges evaluation of the evidence for benefit of treatment modalities involved in producing these risks, as well as research and surveillance into other late effects of treatment.

Ongoing surveillance of incidence and mortality

"Trends [in cancer in this age group] can reflect only relatively recent changes in prevalence of carcinogenic agents…and young people tend to adopt new habits before the old." [41, p 679] Sir Richard Doll suggested that monitoring of cancer trends in this age group could serve as an early warning system, or in the case of a declining trend, early indication of effectiveness of a preventive strategy or risk reducing agent. Ongoing surveillance can therefore direct us in searching for new exposures, both beneficial and harmful.

The first premise behind this recommendation is that there is a relatively short period of time during which young adults could have had exposures leading to enhanced or reduced cancer risk. As a result, it may be easier to identify such exposures than in older adults. For example, the fact that melanoma is relatively common in this age group suggests that excessive sun exposure early in life is particularly important (in a way that early dietary exposures, for example, are not; the two are similar in that they begin soon after birth).

The second premise, that young people tend to be earlier adopters of change, may mean that the effectiveness of preventive efforts can be detected sooner in this age group. For example, the positive changes in melanoma incidence in recent years for this age group, but not older adults,[139] suggests that preventive efforts may be having a positive impact. In 1996, over 80% of Ontario parents reported that their children under age 12 always or often used sunscreen and 74% wore a hat when out in the sun.[131] Will ongoing monitoring of melanoma incidence in young adults reflect the benefit of these behaviours or will it reflect the rising rate of tanning equipment use by this same cohort in its adolescent years?

Monitoring of trends may lead to recommendations for preventive or risk reduction strategies, which would be expected to manifest their impact relatively quickly in the young adult age group. For example, if melanoma incidence begins to increase in the young adult population, it would support development of regulations prohibiting use of tanning equipment by adolescents, as has been recommended by the World Health Organization.[189]

Collaboration, tumour classification and age group

Given the mix of epithelial and non-epithelial cancers common in young adults, careful consideration should be given to the most appropriate classification system and age groupings for monitoring trends and researching etiology. Further, because cancer in this age group is relatively rare, collaborations across jurisdictions will be required.

A large proportion of cancers in young adults are of non-epithelial origins, particularly among men. This proportion gradually decreases with increasing age for both men and women. Non-epithelial cancers are generally defined through morphology, rather than topography, although the standard classification system[190] most commonly used for surveillance is largely organized according to the latter. This is partly because this classification works well enough for the

predominant cancers of older adults. Furthermore, little is known about the etiology of non-epithelial cancers, and the risk factors that are most important for many of the epithelial cancers appear to be much less relevant to non-epithelial cancers.

Different age groups have been used to study cancers in the adolescent/young adult population. For example, Birch et al.[16] use a narrow definition (15–24 years of age), while Wu et al.[192] use 15–49. Such variation creates difficulties for making comparisons or identifying clues to etiology. It might be useful to estimate the ages at which meaningful shifts in predominant tumour types occur (at both the lower and upper ends) to empirically define an appropriate age range. The range 15–34 may be defensible on these grounds: the common non-epithelial cancers of early adulthood begin to appear in the teen years; in men, non-epithelial cancers have the higher incidence through age 35–39; and in women, non-epithelial cancers are more common until sometime in the late 20s. Use of this age group may also be helpful in studying epithelial cancers. For example, there is some evidence that breast cancer in these very young women may be different than that occurring in older age groups, where increasing proportions of women are becoming peri- or post-menopausal.

It is therefore recommended that consideration be given to what constitutes the most appropriate age group, and to developing an alternative classification scheme for the common non-epithelial cancers; one such system has been suggested by Birch et al.[16] Surveillance and research focused on a more homogenous age group and using a more considered system of classification is likely to be more informative. This has certainly proved true for the study of tumours common in childhood, where an alternative classification has become well-accepted.[79]

The number of cancers diagnosed each year in young adults across Canada is about 10,000. For only a few specific types of cancer do the annual numbers exceed 500. Even in the largest single jurisdiction within Canada (Ontario), numbers of diagnoses are large enough for study of only a few cancer types. In order to present a comprehensive picture, this report had to be national in scope and based on a decade or more of data. For etiologic or surveillance studies of individual types of cancer, it will be necessary in many instances to join forces across provinces and territories, and countries. This will be particularly so for studies of genetic traits and gene-environment interactions, or if a narrower age group and finer classification system is adopted.

Ongoing risk factor surveillance

The prevalence of known cancer risk modifiers in this age group will be a very important determinant of cancer burden at older ages. Therefore, risk factor surveillance in this age group should be a priority.

This may be a good age group at which to target cancer prevention strategies, as behaviour change may have double benefit: it will reduce future cancer risk in the main cancer age group and it may result in benefits to their children who represent the next generation of cancer patients. It is important to ensure that young adults are well represented in routine surveillance of important cancer risk factors so that priorities for action in this age group can be identified and the results of such actions monitored. This could include establishment of registries

of workers exposed to known carcinogens; currently, such a registry exists at the national level only for workers exposed to ionizing radiation.

Furthermore, routine risk factor surveillance should be enhanced to periodically include collection of biological samples such as blood, buccal cells, etc. (biomonitoring), so that body burdens of potential carcinogens can be monitored on a population basis. Biomonitoring of young adults will be particularly informative, because it will reflect only relatively recent exposures. In future, data from biological analyses can be used in conjunction with disease data to assess cancer risks associated with measured substances and genetic traits. As noted earlier, this type of research (i.e., of gene-environment interactions) is likely to be particularly informative in this age group.

Projection of future cancer burden

Young adults will grow into older adults, taking the cancer risks accumulated through young adulthood with them. Therefore, study of cancer trends in young adults is useful for projecting future cancer burden.

Study of cancer patterns according to birth cohort has proven a useful technique in epidemiology. A birth cohort represents a group of people born around the same time who therefore have certain types of experiences or exposures in common as they move through life; in some cases these will be very different from their parents and grandparents. To use melanoma as an example yet again, those who were young adults in Canada during the 1960s and 1970s grew up wearing much less clothing in the summer time, spending much less time working out of doors and getting much higher doses, relatively speaking, of "intermittent" as compared to "chronic" exposure to the sun than did their grandparents at the same age; this is reflected in the long term increases in melanoma incidence in this age group up to the mid 1980s. More recent cohorts of young adults would in addition have received much more education about the dangers of overexposure to the sun and had fair quality sunscreens regularly available. These may explain the most recent leveling or decline in melanoma incidence. As these young adults get older, they will carry their lower risk of melanoma with them so that we will expect to see a declining burden of melanoma at older ages in future.

The fact that the incidence of lung cancer in now considerably higher in young women compared to young men should be raising concerns about the future epidemic of lung cancer in women: as today's young women age they will carry their high risk of lung cancer with them into the age groups where this disease is much more common.

Sophisticated forecasting methods already incorporate cohort patterns. However, simple graphical displays and analyses of cancer trends in this age group may provide a straightforward tool for planners.

REFERENCES

1. Adem C, Reynolds C, Ingle JN, et al. Primary breast sarcoma: clinicopathologic series from the Mayo Clinic and review of the literature. Br J Cancer 2004;91:237-41.
2. Adlercreutz H. Phytoestrogens and breast cancer. J Steroid Biochem Mol Biol 2002;83:113-8.
3. Alberg AJ, Samet JM. Epidemiology of lung cancer. Chest 2003;123:21S-49S.
4. Althuis MD, Broan DD, Coates RJ, et al. Breast cancers among very young premenopausal women (United States). Cancer Causes Control 2003;14:151-60.
5. Antman K, Chang Y. Kaposi's sarcoma. N Engl J Med 2000;342:1027-38.
6. Antoniou A, Pharoah PD, Narod S, et al. Average risks of breast and ovarian cancer associated with BRCA1 or BRCA2 mutations detected in case series unselected for family history: a combined analysis of 22 studies. Am J Hum Genet 2003;72:1117-30.
7. Armstrong BK. How sun exposure causes skin cancer: an epidemiological perspective. In: Hill D, Elwood JM, English DR, editors. Prevention of skin cancer. Boston: Kluwer Academic Publishers; 2004. p. 89-116.
8. Armstrong BK. Melanoma: childhood or lifelong sun exposure. In: Grob JJ, Stern RS, Mackie RM, et al, editors. Epidemiology, causes and prevention of skin diseases. Oxford: Blackwell Science; 1997. p. 63-6.
9. Armstrong BK, Kricker A. How much melanoma is caused by sun exposure? Melanoma Res 1993;3:395-401.
10. Band PR, Gaudette LA, Hill GB, et al. The making of the Canadian Cancer Registry: cancer incidence in Canada and its regions, 1969 to 1988. Ottawa: Minister of Supply and Services Canada; 1993. Catalogue Number C52-42/1992.
11. Band PR, Le ND, Fang R, Deschamps M. Carcinogenic and endocrine disrupting effects of cigarette smoke and risk of breast cancer. Lancet 2002 Oct 5;360(9339):1044-9.
12. Beaupré M. Surveillance du cancer au Québec: Nouveaux cas déclarés au Fichier des tumeurs et mortalité par cancer de 1992 à 1998. Québec: Santé et Services sociaux Québec; 2002.
13. Beral V, Peterman TA, Berkelman RL, et al. Kaposi's sarcoma among persons with AIDS: a sexually transmitted infection? Lancet 1990;335:123-8.
14. Bernstein CN, Blanchard JF, Kliewer E, et al. Cancer risk in patients with inflammatory bowel disease: a population-based study. Cancer 2001;91:854-62.
15. Biggar RJ. AIDS-related cancers in the era of highly active antiretroviral therapy. Oncology 2001;15:439-49.
16. Birch JM, Alston RD, Kelsey AM, et al. Classification and incidence of cancers in adolescents and young adults in England 1979-1997. Br J Cancer 2002;87:1267-76.
17. Boisen KA, Kaleva M, Main KM, et al. Difference in prevalence of congenital cryptorchidism in infants between two Nordic countries. Lancet 2004;363:1264-9.

18. Brenner H, Gefeller O. An alternative approach to monitoring cancer patient survival. Cancer 1996;78:2004-10.

19. Brisson J, Major D, Pelletier E. Évaluation de l'exhaustivité du fichier des tumeurs du Québec. Quebec: Institut national de la santé publique du Québec; 2003.

20. Burgess J. Temporal trends for thyroid carcinoma in Australia: an increasing incidence of papillary thyroid carcinoma (1982-1997). Thyroid 2002;12:141-9.

21. Cameron C, Craig CL, Russell SJ, et al. Increasing physical activity: creating effective communications. Ottawa: Canadian Fitness and Lifestyle Research Institute; 2000. http://www.cflri.ca/cflri/resources/pub.php#98pam. Accessed October 15, 2005.

22. Canadian Cancer Society/National Cancer Institute of Canada. Canadian Cancer Statistics 1988, Toronto, Canada. 1988.

23. Canadian Cancer Society/National Cancer Institute of Canada. Canadian Cancer Statistics 1991, Toronto, Canada. 1991.

24. Canadian Cancer Society/National Cancer Institute of Canada. Canadian Cancer Statistics 2002, Toronto, Canada. 2002.

25. Canadian Cancer Society/National Cancer Institute of Canada. Canadian Cancer Statistics 2005, Toronto, Canada. 2005.

26. Canadian Cancer Society—Ontario Division. Colorectal Cancer Risk Management Recommendations/Risk Triage. June 2004.

27. Canadian Council for Tobacco Control. How have smoking rates changed in the last 30 years? National Clearinghouse on Tobacco and Health Program, 2002. http://www.ncth.ca/NCTHweb.nsf. Accessed February 9, 2005.

28. Canadian Institute for Health Information. Improving the Health of Canadians. Ottawa: CIHI; 2004.

29. Cancer Care Ontario. Insight on cancer: Environmental exposures and cancer. Toronto: Canadian Cancer Society (Ontario Division); 2005.

30. Centre for Addiction and Mental Health. Low-risk drinking guidelines. http://www.camh.net/about_addiction_mental_health/low_risk_drinking_guidelines.html. Accessed April 14, 2005.

31. Centers for Disease Control and Prevention. NPCR EDITS tools. http://www.cdc.gov/cancer/npcr/edits/edittool.htm. Accessed April 29, 2005.

32. Centers for Disease Control and Prevention. HIV and AIDS—United States, 1981-2000. MMWR Morb Mortal Wkly Rep 2001;50(21):430-4.

33. Chang Y, Cesarman E, Pessin MS, et al. Identification of herpesvirus-like DNA sequences in AIDS-associated Kaposi's sarcoma. Science 1994;266:1865-9.

34. Chow W-H, Devesa SS, Warren JL, et al. Rising incidence of renal cell cancer in the United States. JAMA 1999;281:1628-31.

35. Clarke CA, Glaser SL. Epidemiologic trends in HIV-associated lymphomas. Curr Opin Oncol 2001;13:354-9.

36. Clavel-Chapelon F, Gerber M. Reproductive factors and breast cancer risk. Do they differ according to age at diagnosis? Breast Cancer Res Treat 2002;72:107-15.

37. Collaborative Group on Hormonal Factors in Breast Cancer. Breast cancer and hormone replacement therapy: collaborative reanalysis of data from 51 epidemiological studies of 52,705 women with breast cancer and 108,411 women without breast cancer. Lancet 1997 Oct 11;350(9084):1047-59.

38. Colonna M, Grosclaude P, Romontet L, et al. Incidence of thyroid cancer in adults recorded by French cancer registries (1978–1997). Eur J Cancer 2002;38:1762-8.

39. Csizmadi I, Benedetti A, Boivin JF, et al. Use of postmenopausal estrogen replacement therapy from 1981 to 1997. CMAJ 2002 Jan 22;166(2):187-8.

40. Diffey BL. A quantitative estimate of melanoma mortality from ultraviolet: a sunbed use in the U.K. Br J Dermatol 2003;149:578-81.

41. Doll R. Progress against cancer: an epidemiologic assessment. The 1991 John C. Cassel Memorial Lecture. Am J Epidemiol 1991;134:675-88.

42. Doll R, Hill AB. Smoking and carcinoma of the lung. Preliminary report. BMJ 1950;2:739-48.

43. Dos Santos Silva I, Swerdlow AJ. Recent trends in incidence of and mortality from breast, ovarian and endometrial cancers in England and Wales and their relation to changing fertility and oral contraceptive use. Br J Cancer 1995;72:485-92.

44. Edmonds CJ, Smith T. The long-term hazards of the treatment of thyroid cancer with radioiodine. Br J Radiol 1986;59:45-51.

45. Eltom MA, Jemal A, Mbulaiteye SM, et al. Trends in Kaposi's sarcoma and non-Hodgkin's lymphoma incidence in the United States from 1973 through 1998. J Natl Cancer Inst 2002;94:1204-10.

46. Fairfield KM, Willett WC, Rosner BA, et al. Obesity, weight gain, and ovarian cancer. Obstet Gynecol 2002;100:288-96.

47. Ferrence RG. Sex differences in cigarette smoking in Canada, 1900-1978: a reconstructed cohort study. Can J Public Health 1988;79:160-5.

48. Fisher WA, Boroditsky R, Bridges ML. The 1998 Canadian Contraception Study Part 4: Oral contraceptive use among Canadian women: practices and opinions. Can J Hum Sex 1999;8:183-8.

49. Franco EL, Duarte-Franco E, Ferenczy A. Cervical cancer: epidemiology, prevention and the role of human papillomavirus infection. CMAJ 2001;164:1017-25.

50. Friedenreich CM. Review of anthropometric factors and breast cancer risk. Eur J Cancer Prev 2001;10:15-32.

51. Friedenreich CM, Orenstein MR. Physical activity and cancer prevention: etiologic evidence and biological mechanisms. J Nutr 2002;132:3456S-3464S.

52. Garber K. Lymphoma rate rise continues to baffle researchers. J Natl Cancer Inst 2001;93:494-6.

53. Gazdar AF, Minna JD. Cigarettes, sex and lung adenocarcinoma. J Natl Cancer Inst 1997;89:1563-5.

54. Geller AC, Colditz G, Oliveria S, et al. Use of sunscreen, sunburning rates, and tanning bed use among more than 10 000 U.S. children and adolescents. Pediatrics 2002;109:1009-14.

55. Gilmore J. Report on smoking prevalence in Canada, 1985 to 1999. Ottawa: Statistics Canada; 2000. Catalogue 82F0077XIE.

56. Giovannucci E. An updated review of the epidemiological evidence that cigarette smoking increases risk of colorectal cancer. Cancer Epidemiol Biomarkers Prev 2001;10:725-31.

57. Globel B, Globel H, Oberhausen E. Epidemiological studies on patients with iodine-131 diagnostic and therapy. In: Kaul A, Neider R, Pensko J, et al, editors. Radiation-Risk-Protection, Vol II. International Radiation Protection Association. Koln: Fachverbund fur Strahlenscutz e.V; 1984. p. 565-8.

58. Gnagy S, Ming EE, Devesa SS, et al. Declining ovarian cancer rates in U.S. women in relation to parity and oral contraceptive use. Epidemiology 2000;11:102-5.

59. Goodman MT, Howe HL. Descriptive epidemiology of ovarian cancer in the United States, 1992–1997. Cancer 2003;97:2615-30.

60. Groves FD, Linet MS, Travis LB, et al. Cancer surveillance series: non-Hodgkin's lymphoma incidence by histologic subtype in the United States from 1978 through 1995. J Natl Cancer Inst 2000;92:1240-51.

61. Hamilton PM, Chiacchierini RP, Kaczmarek RG. A follow-up study of persons who had iodine-131 and other diagnostic procedures during childhood and adolescence. Washington (DC): U.S. Department of Health and Human Services; 1989. FDA Publication 89-8276.

62. Hankinson S, Hunter D. Breast Cancer. In: Adami H-O, Hunter D, Trichopoulos D, editors. Textbook of cancer epidemiology. New York: Oxford University Press; 2002. p. 301-39.

63. Hardiman P, Pillay OC, Atiomo W. Polycystic ovary syndrome and endometrial carcinoma. Lancet 2003;361:1810-2.

64. Hartge P, Harvey EB, Linehan WM, et al. Unexplained excess risk of bladder cancer in men. J Natl Cancer Inst 1990;82:1636-40.

65. Health Canada. HIV and AIDS in Canada. Surveillance Report to December 31, 2000. Division of HIV/AIDS Epidemiology and Surveillance, Bureau of HIV/AIDS, STD and TB, Center for Infectious Disease Prevention and Control: Health Canada; 2001. p. 26.

66. Health Canada. HIV and AIDS in Canada. Surveillance Report to December 31, 2003. Division of HIV/AIDS Epidemiology and Surveillance, Center for Infectious Disease Prevention and Control: Health Canada; 2004. p. 37-43.

67. Health Canada. Non-leisure physical activity. (National Population Health Survey Highlights, Physical Activity of Canadians, Cycle 2, 1996/97, No. 2),1999. http://www.phac-aspc.gc.ca/ccdpc-cpcmc/cancer/publications/pdf/nphs_pae.pdf. Accessed November 24, 2005.

68. Health Canada. Youth smoking in Canada. CTUMS (Canadian Tobacco Use Monitoring Survey), Annual, February-December 2000. Aussi disponible en français.

69. Health Canada Tobacco Control Program. Canadian Tobacco Use Monitoring Survey (CTUMS). Supplementary tables, 2000. http://www.hc-sc.gc.ca/hl-vs/alt_formats/hecs-sesc/pdf/tobac-tabac/research-recherche/stat/ctums-esutc/1999/supp-1999_e.pdf. Accessed March 4, 2005.

70. Hess KR, Broglio KR, Bondy ML. Adult glioma incidence trends in the United States, 1977-2000. Cancer 2004;101:2293-9.

71. Holm L-E, Hall P, Wiklund K, et al. Cancer risk after iodine-131 therapy for hyperthyroidism. J Natl Cancer Inst 1991;83:1072-7.

72. Holowaty EJ. The Ontario cancer registry. In: Black RJ, Simonato L, Storm HH, et al, editors. Automated data collection in cancer registration. Lyon, France: International Agency for Research on Cancer; 1998. IARC Technical Report No. 32. p. 39-44.

73. Hymes KB, Cheung T, Greene JB, et al. Kaposi's sarcoma in homosexual men–a report of eight cases. Lancet 1981;2:598-600.

74. Innes K, Byers T, Schymura M. Birth characteristics and subsequent risk for breast cancer in very young women. Am J Epidemiol 2000;152:1121-8.

75. Institute of Medicine (U.S.). Fulfilling the potential of cancer prevention and early detection. Curry SJ, Byers T, Hewitt M, editors. Washington (DC): The National Academies Press; 2003.

76. International Agency for Research on Cancer. CONVERT software. http://www.iacr.com.fr/convert.htm. Accessed April 29, 2005.

77. International Agency for Research on Cancer. Fruit and vegetables. (IARC handbooks of cancer prevention, Volume 8). Lyon, France: IARC; 2003.

78. International Agency for Research on Cancer. Human papillomaviruses (IARC monographs on the evaluation of carcinogenic risks to humans, Volume 64). Lyon, France: IARC; 1995.

79. International Agency for Research on Cancer. International incidence of childhood cancer (IARC Scientific Publication No. 144). Lyon, France: IARC; 1999.

80. International Agency for Research on Cancer. Solar and ultraviolet radiation (IARC monographs on the evaluation of carcinogenic risks to humans, Volume 55). Lyon, France: IARC; 1992.

81. International Agency for Research on Cancer. Sunscreens (IARC handbooks of cancer prevention, Volume 5). Lyon, France: IARC; 2001.

82. International Agency for Research on Cancer. Weight control and physical activity (IARC handbooks of cancer prevention, Volume 6). Lyon, France: IARC; 2002.

83. Jaffe HW, Bregman DJ, Selik RM. Acquired immune deficiency syndrome in the United States: the first 1000 cases. J Infect Dis 1983;148:339-45.

84. Jarrett RF, MacKenzie J. Epstein-Barr virus and other candidate viruses in the pathogenesis of Hodgkin's disease. Semin Hematol 1999;36:260-9.

85. Jatoi I, Miller AB. Why is breast-cancer mortality declining? Lancet Oncol 2003;4:251-4.

86. Johansson SL, Cohen SM. Epidemiology and etiology of bladder cancer. Semin Surg Oncol 1997;13:291-8.

87. Johnson KC. Accumulating evidence on passive and active smoking and breast cancer risk.
Int J Cancer 2005 Nov 20;117(4):619-28.

88. Katzmarzyk PT. The Canadian obesity epidemic: an historical perspective. Obes Res 2002;10:666-74.

89. Key TJ, Allen NE, Spencer EA, et al. The effect of diet on risk of cancer. Lancet 2002;360:861-8.

90. Kim HJ, Fay MP, Feuer EJ, et al. Permutation tests for joinpoint regression with applications to cancer rates. Stat Med 2000;19:335-51.

91. Kogevinas M, Trichopoulos D. Urinary bladder cancer. In: Adami H-O, Hunter D, Trichopoulos D, editors. Textbook of cancer epidemiology. New York: Oxford University Press; 2002. p. 446-66.

92. Kreuzer M, Kreienbrock L, Gerken M, et al. Risk factors for lung cancer in young adults. Am J Epidemiol 1998;147:1028-37.

93. Krewski D, Lubin JH, Zielinski JM, et al. Residential radon and risk of lung cancer: a combined analysis of 7 North American case-control studies. Epidemiology 2005 Mar;16(2):137-45.

94. Kuper H, Cramer DW, Titus-Ernstoff L. Risk of ovarian cancer in the United States in relation to anthropometric measures: does the association depend on menopausal status? Cancer Causes Control 2002;13:455-63.

95. La Vecchia C. Epidemiology of ovarian cancer: a summary review. Eur J Cancer Prev 2001;10:125-9.

96. La Vecchia C, Ron E, Franceschi S, et al. A pooled analysis of case-control studies of thyroid cancer. III. Oral contraceptives, menopausal replacement therapy and other female hormones. Cancer Causes Control 1999;10:157-66.

97. Levi F, Lucchini F, Negri E, et al. Trends in mortality from leukemia in subsequent age groups. Leukemia 2000;14:1980-5.

98. Li CI, Anderson BO, Daling JR, et al. Trends in incidence rates of invasive lobular and ductal breast carcinoma. JAMA 2003;289:1421-4.

99. Li X, Hemminki K. Inherited predisposition to early onset lung cancer according to histologic type. Int J Cancer 2004;112:451-7.

100. Linet MS, Cartwright RA. The leukemias. In: Schottenfeld D, Fraumeni D, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press; 1996. p. 847-69.

101. Liu S, Semenciw R, Mao Y. Cervical cancer: the increasing incidence of adenocarcinoma and adenosquamous carcinoma in younger women. CMAJ 2001a;164:1151-2.

102. Liu S, Semenciw R, Morrison H, et al. Kidney cancer in Canada: the rapidly increasing incidence of adenocarcinoma in adults and seniors. Can J Public Health 1997;88:99-104.

103. Liu S, Semenciw R, Ugnat AM, et al. Increasing thyroid cancer incidence in Canada, 1970-1996: time trends and age-period-cohort effects. Br J Cancer 2001b;85:1335-9.

104. Liu S, Semenciw R, Waters C, et al. Clues to the aetiological heterogeneity of testicular seminomas and non-seminomas: time trends and age-period-cohort effects. Int J Epidemiol 2000b;29:826-31.

105. Liu S, Semenciw R, Waters C, et al. Time trends and sex patterns in Hodgkin's disease incidence in Canada, 1970-1995. Can J Public Health 2000a;91:188-92.

106. Llewellyn CD, Johnson NW, Warnakulasuriya KA. Risk factors for squamous cell carcinoma of the oral cavity in young people—a comprehensive literature review. Oral Oncol 2001;37:401-18.

107. Longo DL. Radiation therapy in Hodgkin disease: why risk a Pyrrhic victory? J Natl Cancer Inst 2005;97:1394-5.

108. Lynch CF, Platz CE, Jones MP, et al. Cancer registry problems in classifying in invasive bladder cancer. J Natl Cancer Inst 1991;83:429-33.

109. Macfarlane GJ, Evstifeeva T, Boyle P, et al. International patterns in the occurrence of Hodgkin's disease in children and young adult males. Int J Cancer 1995;61:165-9.

110. Maher ER. Inherited renal cell carcinoma. Br J Urol 1996;354:93-9.

111. Mahoney MC, Lawvere S, Falkner KL, et al. Thyroid cancer incidence trends in Belarus: examining the impact of Chernobyl. Int J Epidemiol 2004;33:1025-33.

112. Marrett LD, Chaudhry M. Cancer incidence and mortality in Ontario First Nations, 1968-1991. Cancer Causes Control 2003;14:259-68.

113. Marrett LD, King WD, Walter SD, et al. Use of host factors to identify people at high risk for cutaneous malignant melanoma. CMAJ 1992;147:445-53.

114. Martin K, Wu Z. Contraceptive use in Canada: 1984-1995. Fam Plann Perspect 2000;32:65-73.

115. McCunney RJ. Hodgkin's disease, work and the environment. A review. J Occup Environ Med 1999;41:36-46.

116. McGlynn KA, Devesa SS, Sigurdson AJ, et al. Trends in the incidence of testicular germ cell tumors in the United States. Cancer 2003;97:63-70.

117. Melbye M, Adami H-O. Hodgkin's lymphoma. In: Adami H-O, Hunter D, Trichopoulos D, editors. Textbook of cancer epidemiology. New York: Oxford University Press; 2002a. p. 520-34.

118. Melbye M, Trichopoulos D. Non-Hodgkin's lymphomas. In: Adami H-O, Hunter D, Trichopoulos D, editors. Textbook of cancer epidemiology. New York: Oxford University Press; 2002b. p. 535-55.

119. Miller RW, Boice JD Jr, Curtis RE. Bone. In: Schottenfeld D, Fraumeni JF Jr, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press; 1996. p. 971-83.

120. Mucci L, Adami H-O. Oral and pharyngeal cancer. In: Adami H-O, Hunter D, Trichopoulos D, editors. Textbook of cancer epidemiology. New York: Oxford University Press; 2002. p. 115-36.

121. Mueller NE. Hodgkin's disease. In: Schottenfeld D, Fraumeni D, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press; 1996. p. 897-914.

122. National Cancer Institute sponsored study of classifications of non-Hodgkin's lymphomas: summary and description of a working formulation for clinical usage. The Non-Hodgkin's Lymphoma Pathologic Classification Project. Cancer 1982;49:2112-35.

123. National Cancer Institute Statistical Research and Applications Branch. Joinpoint regression program, Version 2.7. September 2003. http://srab.cancer.gov/joinpoint/. Accessed April 29, 2005.

124. Negri E, DalMaso L, Ron E, et al. A pooled analysis of case-control studies of thyroid cancer. II. Menstrual and reproductive factors. Cancer Causes Control 1999;10:143-55.

125. Newcomb PA, Trentham-Dietz A. Patterns of postmenopausal progestin use with estrogen in relation to endometrial cancer (United States). Cancer Causes Control 2003;14:195-201.

126. Nielsen NH, Storm HH, Gaudette LA, et al. Cancer in Circumpolar Inuit 1969-1988—A summary. Acta Oncol 1996;35:621-8.

127. Nijhuis PH, Schaapveld M, Otter R, et al. Epidemiological aspects of soft tissue sarcomas (STS)—consequences for the design of clinical STS trials. Eur J Cancer 1999;35:1705-10.

128. Niwa K, Imai A, Hashimoto M, et al. A case-control study of uterine endometrial cancer of pre- and post-menopausal women. Oncol Rep 2000;7:89-93.

129. North American Association of Central Cancer Registries. Standards for Cancer Registries, (Vol. IV) NAACCR Standard Edits. http://www.naaccr.org/index.asp?Col_SectionKey=7&Col_ContentID=136. Accessed April 29, 2005.

130. Olsson H. An updated review of the epidemiology of soft tissue sarcoma. Acta Orthop Scand Suppl 2004;75:16-20.

131. Ontario Sun Safety Working Group. Sun exposure and protective behaviours: Ontario report 1998. Toronto: Canadian Cancer Society (Ontario Division); 1998.

132. Parent A-S, Teilmann G, Anders J, et al. The timing of normal puberty and the age limits of sexual precocity: Variations around the world, secular trends, and changes after migration. Endocr Rev 2003;24:668-93.

133. Parkin DM, Shanmugaratnam K, Sobin L, et al. Histological groups for comparative studies. Lyon, France: IARC; 1998. IARC Technical Report No. 31.

134. Parkin DM, Whelan SL, Ferlay J, et al, editors. Cancer incidence in five continents. Volume VIII. Lyon, France: IARC; 2002.

135. Percy C, Van Holten V, Muir C, editors. International Classification of Diseases for Oncology, Second Edition. Geneva, Switzerland: WHO; 1990.

136. Petridou E, Trichopoulos D. Leukemias. In: Adami H-O, Hunter D, Trichopoulos D, editors. Textbook of cancer epidemiology. New York: Oxford University Press; 2002. p. 556-72.

137. Prener A, Engholm G, Jensen OM. Genital anomalies and risk for testicular cancer in Danish men. Epidemiology 1996;7:14-9.

138. Preston-Martin S, Francheschi S, Ron E, et al. Thyroid cancer pooled analysis from 14 case-control studies: what have we learned? Cancer Causes Control 2003;14:787-9.

139. Public Health Agency of Canada. Cancer Surveillance On-Line. http://dsol-smed.hc-sc.gc.ca/dsol-smed/cancer/index_e.html. Accessed April 29, 2005.

140. Purdie DM, Green AC. Epidemiology of endometrial cancer. Best Pract Res Clin Obstet Gynaecol 2001;15:341-54.

141. Purdue MP, Devesa SS, Sigurdson AJ, et al. International patterns and trends in testis cancer incidence. Int J Cancer 2005;115:822-7.

142. Ramstedt M. Alcohol consumption and alcohol-related mortality in Canada, 1950-2000. Can J Public Health 2004;95:121-6.

143. Rashidkhani B, Lindblad P, Wolk A. Fruits, vegetables and risk of renal cell carcinoma: a prospective study of Swedish women. Int J Cancer 2005;113:51-5.

144. Rhainds M, DeGuire L, Claveau J. A population-based survey on the use of artificial tanning devices in the Province of Québec, Canada. J Am Acad Dermatol 1999;40:572-6.

145. Ries LAG, Eisner MP, Kosary CL, et al, editors. SEER Cancer Statistics Review 1973-1999. Bethesda: National Cancer Institute; 2002. http://seer.cancer.gov/csr/1973_1999/overview.pdf. Accessed April 29, 2005.

146. Rodriguez T, Altieri A, Chatenoud L, et al. Risk factors for oral and pharyngeal cancer in young adults. Oral Oncol 2004;40:207-13.

147. Ron E. Thyroid cancer. In: Schottenfeld D, Fraumeni JF Jr., editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press; 1996. p. 1000-21.

148. Runnebaum IB, Stickeler E. Epidemiological and molecular aspects of ovarian cancer risk. J Cancer Res Clin Oncol 2001;127:73-9.

149. Safe S. Environmental estrogens: roles in male reproductive tract problems and in breast cancer. Rev Environ Health 2002;17:253-62.

150. Sasco AJ, Secretan MB, Straif K. Tobacco smoking and cancer: a brief review of recent epidemiological evidence. Lung Cancer 2004;45:S3-S9.

151. Sasieni P, Adams J. Changing rates of adenocarcinoma and adenosquamous carcinoma of the cervix in England. Lancet 2001;357:1490-3.

152. Savitz D, Trichopoulos D. Brain cancer. In: Adami H-O, Hunter D, Trichopoulos D, editors. Textbook of cancer epidemiology. New York: Oxford University Press; 2002. p. 486-503.

153. Scherr PA, Mueller NE. Non-Hodgkin's lymphoma. In: Schottenfeld D, Fraumeni D, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press; 1996. p. 920-45.

154. Schwartz SM, Weiss NS, Daling JR, et al. Exogenous sex hormone use, correlates of endogenous hormone levels, and the incidence of histologic types of sarcoma of the uterus. Cancer 1996;77:717-24.

155. Schwartz SM, Weiss NS, Daling JR, et al. Incidence of histologic types of uterine sarcoma in relation to menstrual and reproductive history. Int J Cancer 1991;49:362-7.

156. Sellers TA, Bailey-Wilson JE, Elston RC, et al. Evidence for Mendelian inheritance in the pathogenesis of lung cancer. J Natl Cancer Inst 1990;82:1272-9.

157. Shore RE. Issues and epidemiological evidence regarding radiation-induced thyroid cancer. Radiat Res 1992;131:98-111.

158. Siemiatycki J, Richardson L, Straif K, et al. Listing occupational carcinogens. Environ Health Perspect 2004;112:1447-59.

159. Silverman DT. Bladder cancer. In: Schottenfeld D, Fraumeni JF Jr, editors. Cancer epidemiology and prevention. 2nd ed. New York: Oxford University Press; 1996. p. 1156-79.

160. Silverman DT, Hartge P, Morrison AS, et al. Epidemiology of bladder cancer. Hematol Oncol Clin North Am 1992;6:1-30.

161. Statistics Canada. Health Promotion Survey (1985, 1990) microdata file. Ottawa: Statistics Canada, Special Surveys Division; 1995a.

162. Statistics Canada. National Population Health Survey (NPHS) 1994/1995, public use microdata file. Ottawa: Statistics Canada, Health Statistics Division; 1995b.

163. Statistics Canada. National Population Health Survey (NPHS) 1996/1997, public use microdata file. Ottawa: Statistics Canada, Health Statistics Division; 1998.

164. Statistics Canada. National Population Health Survey (NPHS) 1998/1999, public use microdata file. Ottawa: Statistics Canada, Health Statistics Division; 2000.

165. Statistics Canada. Population projections for 2001, 2006, 2011, 2016, 2021 and 2026, at July 1. http://www40.statcan.ca/l01/cst01/demo23a.htm, http://www40.statcan.ca/l01/cst01/demo23b.htm, http://www40.statcan.ca/l01/cst01/demo23c.htm Accessed November 2, 2005.

166. Statistics Canada. Selected Birth and Fertility Statistics, Canada, 1921-1990. Ottawa, ON: Minister of Supply and Services; 1993. Catalogue 82-553. pp. 34, 41.

167. Statistics Canada. Youth Smoking Survey 2002. The Daily, catalogue number 11-001-XIE, June 14, 2004. http://www.statcan.ca/Daily/English/040614/d040614b.htm. Accessed November 4, 2005.

168. Stewart BW, Kleihues P, editors. World Cancer Report. Lyon, France: International Agency for Research on Cancer; 2003.

169. Surveillance, Epidemiology, and End Results (SEER) Program. SEER*Stat Database: Incidence – SEER 9 Registries, August 2000 Submission (1973-1998). National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch. 2001.

170. Tamimi R, Adami H-O. Testicular cancer. In: Adami H-O, Hunter D, Trichopoulos D, editors. Textbook of cancer epidemiology. New York: Oxford University Press; 2002. p. 429-45.

171. Tarone RE, Chu KC, Gaudette LA. Birth cohort and calendar period trends in breast cancer mortality in the United States and Canada. J Natl Cancer Inst 1997;89:251-6.

172. Terry PD, Rohan TE. Cigarette smoking and the risk of breast cancer in women: a review of the literature. Cancer Epidemiol Biomarkers Prev. 2002 Oct;11(10 Pt 1):953-71.

173. Thun MJ, Lally CA, Flannery JT, et al. Cigarette smoking and changes in the histopathology of lung cancer. J Natl Cancer Inst 1997;89:1580-6.

174. Travis LB, Hill D, Dores GM, et al. Cumulative absolute breast cancer risk for young women treated for Hodgkin lymphoma. J Natl Cancer Inst 2005;97: 1428-37.

175. United Kingdom Testicular Cancer Study Group. Aetiology of testicular cancer: association with congenital abnormalities, age at puberty, infertility, and exercise. BMJ 1994;308:1393-9.

176. Vainio H, Miller AB. Primary and secondary prevention in colorectal cancer. Acta Oncol 2003;42:809-15.

177. Valery PC, McWhirter W, Sleigh A, et al. Farm exposures, parental occupation, and risk of Ewing's sarcoma in Australia: a national case-control study. Cancer Causes Control 2002;13:263-70.

178. Valery PC, Williams G, Sleigh AC, et al. Parental occupation and Ewing's sarcoma: Pooled and meta-analysis. Int J Cancer 2005 (in press).

179. Veierød MB, Weiderpass E, Thörn M, et al. A prospective study of pigmentation, sun exposure, and risk of cutaneous malignant melanoma in women. J Natl Cancer Inst 2003;95:1530-8.

180. Verkooijen HM, Fioretta G, Vlastos G, et al. Important increase of invasive lobular breast cancer incidence in Geneva, Switzerland. Int J Cancer 2003;104:778-81.

181. Vizcaino AP, Moreno V, Bosch FX, et al. International trends in the incidence of cervical cancer: I. Adenocarcinoma and adenosquamous cell carcinomas. Int J Cancer 1998:75:536-45.

182. Waller BJ, Cohen JE, Ferrence R, et al. The early 1990s cigarette price decrease and trends in youth smoking in Ontario. Can J Public Health 2003;94:31-5.

183. Weiderpass E, Ye W, Mucci LA, et al. Alcoholism and risk for endometrial cancer. Int J Cancer 2001;93:299-301.

184. Weir HK, Kreiger N, Marrett LD. Age at puberty and risk of testicular germ cell cancer. Cancer Causes Control 1998;9:253-8.

185. Weir HK, Marrett LD, Kreiger N, et al. Prenatal and peri-natal exposures and risk of testicular germ cell cancer. Int J Cancer 2000;87:438-43.

186. Weir HK, Marrett LD, Morovan V. Trends in the incidence of testicular germ cell cancer in Ontario by histologic subgroups: 1964-1996. CMAJ 1999;160:201-5.

187. Westhoff C, Heller D, Drosinos S, et al. Risk factors for hyperplasia-associated versus atrophy-associated endometrial carcinoma. Am J Obstet Gynecol 2000;182:506-8.

188. Wharton V. Neoplasms of the cervix. In: Holland JF, Frei E III, Bast RC Jr, et al, editors. Cancer medicine. Vol. II. 4th ed. Toronto: Williams & Wilkins; 1995. p. 2227-61.

189. World Health Organization. Artificial tanning sunbeds: risks and guidance. Geneva, Switzerland: WHO; 2003.

190. World Health Organization. International statistical classification of diseases and related health problems, 10th revision,Volumes 1 to 3. Geneva, Switzerland: WHO; 1992.

191. Wrensch M, Minn Y, Chew T, et al. Epidemiology of primary brain tumors: current concepts and review of the literature. Neuro-oncol 2002;4:278-99.

192. Wu X, Groves FD, McLaughlin CC, et al. Cancer patterns among adolescents and young adults in the United States. Cancer Causes Control 2005;225:309-20.

193. Xie Y, Davies SM, Xiang Y, et al. Trends in leukemia incidence and survival in the United States (1973-1998). Cancer 2003;97:2229-35.

194. Zahm SH, Fraumeni JF Jr. The epidemiology of soft tissue sarcoma. Semin Oncol 1997;24:504-14.

195. Zahm SH, Tucker MA, Fraumeni JF Jr. Soft tissue sarcomas. In: Schottenfeld D, Fraumeni JF Jr, editors. Cancer epidemiology and prevention. 2nd ed. Oxford: Oxford University Press; 1996. p. 984-99.

196. Zhang J, Ugnat AM, Clarke K, et al. Ovarian cancer histology-specific incidence trends in Canada 1969-1993: age-period-cohort analyses. Br J Cancer 1999;81:152-8.

197. Zheng T, Holford TR, Ma Z, et al. The continuing increase in adenocarcinoma of the uterine cervix: a birth cohort phenomenon. Int J Epidemiol 1996;25:252-8.

APPENDIX

MATERIALS AND METHODS

Data sources

Data from the provincial/territorial cancer registries form the foundation for this work. Each provincial/territorial cancer registry provided a standardized data set to Cancer Care Ontario, under a data agreement that permitted use only for this project. Registry staff were asked to identify specific aspects of their own data that would warrant special attention during analysis and interpretation. These steps maximized comparability across registries and ensured that local data artefacts could be accounted for to the extent possible. Other data sets included the Canadian cancer mortality file and surveys containing risk factor information.

Incidence data

Data from each provincial/territorial cancer registry contained cases meeting the following criteria:

- aged 20–44* at diagnosis;
- all invasive cancer except squamous and basal cell skin cancer;
- first diagnosis of an invasive primary cancer;
- from 1969, or earliest date with acceptable quality;
- to the most recent year with complete data;
- resident in the province at diagnosis.

Data for the Yukon territory were provided by the British Columbia Cancer Agency, while Nunavut and the Northwest Territories authorized the release of their data from Statistics Canada. The earliest year for which data were available varied by province, from 1969 for several registries to 1983 for Quebec. To present a consistent national picture, 1983 was chosen as the earliest date for trend analysis. At the time of the data request, 1999 was the most recent year for which all provinces had complete incidence data.

The fact that non-melanoma skin cancers are not registered in the Ontario Cancer Registry (OCR) meant that most of Ontario's Kaposi sarcoma would be missed. With the insertion of a rule that all skin cancers should have ICD-9 (International Classification of Diseases, 9th revision) site code 172 (usually reserved for cutaneous malignant melanoma), the OCR's automated programs were forced to treat all skin cancer records in a consistent manner and additional cases were identified for inclusion in this study.

Because no personal identifying information other than birth year and sex were requested, data sets could not be merged to identify cases registered in multiple provinces. However, investigations of the available data suggested that few duplicate cases exist.

* For some analyses, the age range was extended to 15–44 for Hodgkin lymphoma, since incidence is already high by age 20, and to 20–49 for breast cancer to better represent the population at risk for pre-menopausal breast cancer.

Mortality data

The Public Health Agency of Canada supplied cancer mortality data in the form of frequencies by province or territory, sex, 5-year age group and year (1950–1999) for most of the cancers. National mortality data can be examined across five versions of the International Classification of Diseases (ICD) used for coding cause of death. Because sarcoma and Kaposi sarcoma were not part of ICD coding, no comparable mortality data for these cancers could be obtained.

International data

The International Agency for Research on Cancer (IARC) provided international incidence and population data for the 5-year age groups in the years 1993–1997 for 27 registries. Registries that represent various world regions and have registration methods reasonably similar to those used in Canada were selected.[134] These criteria omit Africa and much of South America. Incidence reported by the selected registries was standardized to the 1991 Canadian population (sexes combined). From these 27 registries, nine registries were then chosen whose incidence rates effectively represented the observed international variation: New South Wales (NSW), Australia; Surveillance, Epidemiology and End Results (SEER) data for the US White population; Venetian region, Italy; Isère, France; Slovakia; Finland; Shanghai, China; Cali, Colombia; and Chennai, India.

Other data

The Public Health Agency of Canada provided several other data sets essential to the project: population counts for calculating rates, life expectancies for calculating potential years of life lost (PYLLs), and projected incidence and mortality data to 2005. The projection methods used are described in *Canadian Cancer Statistics 2005*, but were based on data for the years 1990–1999. Estimates of survey-based risk factor prevalences were also supplied.[161–164] Canadian cancer incidence data from Cancer Surveillance On-line were used to determine the proportions of individual cancers that are diagnosed in the young adult age group.[139]

Data preparation

Cancer topology and morphology coding

To minimize artefacts arising from differences in coding conversions, registries were encouraged to provide data in the original coding system. Some provinces had converted all data to the latest version of the International Classification of Diseases for Oncology (ICD-O), while others were able to send data in the original coding system. Provincial data included topologies coded in four different systems (ICD 8, ICD 9, ICD-O-1, ICD-O-2) and morphologies coded in four systems (SNOP, ICD-O-1, ICD-O-2, ICD-O-3). ICD-O-2 was chosen as the standard coding system because it was the original coding system for the majority of cases and software was easily available for conversions.[135]

Six cancers—melanoma, Hodgkin lymphoma, non-Hodgkin lymphoma, leukemia, sarcoma and Kaposi sarcoma—were defined by morphology. Cases meeting the definitions for these cancers were identified first; all remaining cancers were then classified based on topology. Detailed cancer definitions are documented in Tables A1 and A2. Epithelial cancers were defined as those cases with a morphology in the range 8010 to 8580; non-epithelial cancers had morphologies in the range 8590 to 9941.

Morphologic information is not available for cancer mortality data. ICD topology codes can be, and were, used to identify lymphomas and leukemias from the mortality file, although the two methods yield slightly different cases. It is also not possible to examine mortality trends in epithelial and non-epithelial cancers.

Conversion and edit checks

SAS® code was written for the simple site and morphology code conversions (ICD 8 to ICD 9; ICD-O-3 to ICD-O-2), and CONVERT was used for the more complicated conversion (ICD 9 & ICD-O-1 to ICD-O-2). CONVERT, a specialized software package using internationally accepted standard rules, is available free from IARC.[76]

Once converted, data from each province and territory were checked for invalid data and inter-field inconsistencies such as impossible topology and morphology combinations, using the National Program of Cancer Registries' GenEDITS program[31] and the North American Association of Central Cancer Registries (NAACCR) Version 9 metafile.[129] All problems were forwarded to the appropriate registries for resolution, and the resulting corrections were returned to Cancer Care Ontario.

Data quality

Each provincial/territorial registry has its own methods for ascertaining cancer cases and verifying the essential information about each patient and tumour. As a result, the standard cancer data quality indicators vary greatly across the country. To assess the data set's quality, values for these indicators were calculated and compared to values for the high quality incidence data collected through the US National Cancer Institute's SEER program.

Microscopic confirmation

A cancer case is considered to be microscopically confirmed (MC) if a pathology or cytology report confirmed the cancer diagnosis. If the %MC is low, the poor verification of diagnoses may result in over-registration of some types of cancer and under-registration of others. For example, melanoma may be under-registered, as it is frequently registered only on the basis of histopathologic information. SEER reported that between 1990 and 1998, 96.8% of its cases aged 20–44 were microscopically confirmed.[169] British Columbia, Prairie and Atlantic regions all reported over 97% MC. In the other regions, the %MC was lower: 93.3% in the North, 89.3% in Ontario and 86.4% in Quebec. Pathology reports are not available to the Quebec registry for case ascertainment and it has been estimated that the Ontario Cancer Registry receives 75–80% of the required pathology reports.[72]

Microscopic confirmation is over 95% for Hodgkin lymphoma and testicular, breast, and colorectal cancers. Three cancers had %MC less than 85%: leukemia (84.0%), brain (82.8%) and Kaposi sarcoma (60.0%). The %MC for leukemia and brain were low for Ontario and Quebec because of the unavailability to the provincial cancer registry of hematology reports and CT scans. Kaposi sarcoma had the poorest %MC in each region, ranging from 86.8% to 34.2%.

Mortality-to-incidence ratio

The mortality-to-incidence ratio (M/I%) compares the number of deaths from a specific cancer in a given population with the number of incident cancers diagnosed in the same population during the same time period. The M/I% is heavily influenced by

survival; thyroid cancer has an extremely low value (0.7%), while lung cancer has the highest (70%). The mortality to incidence ratio can also be used as a crude indicator of completeness of registration. If registry procedures are somehow flawed, too many cases may be reported on the basis of death certificate information and the M/I ratio will be inflated. A crude estimate of M/I% for ages 20–44 using SEER data is 20%.[145] Three regions—Ontario, British Columbia and Prairie—all have M/I% less than 23%. Higher M/I ratios are found in the Atlantic region (27%), Quebec (29%) and the North (34%). Quebec's high M/I% for some cancers may be due to incomplete ascertainment of cases living in the Outaouais region who are frequently treated in Ontario.[12]

Analyses

Age-specific incidence and mortality rates

For each selected cancer, age-specific rates were calculated by 5-year age group (20–24, 25–29, …, 40–44) as the number of incident cases or deaths occurring in Canada between 1990 and 1999, divided by the person-years occurring in the same period. Age-specific rates were plotted if the rate was greater than or equal to 1.0 per 100,000 in at least one age group.

Age-standardized incidence and mortality rates

An age-standardized rate is the incidence or mortality rate that would be observed in a population with a given set of age-specific rates if its age distribution were the same as that of a standard population. For this publication, the 1991 Canadian population (Table A3) was used as the standard. Age-standardized annual rates were plotted if at least one rate between 1990 and 1999 was equal to or greater than 1.0 per 100,000 or the criterion for an age-specific graph was met.

Time trend analyses

The National Cancer Institute's Joinpoint software was used for trend analyses.[90, 123] Joinpoint fits one to four lines connected at 'joinpoints' to the trend data and selects the simplest model that fits the data best. Monte Carlo methods are used for tests of significance. Time trends were analyzed only for cancers with at least one annual age-standardized rate in the 1990s greater than or equal to 1.0 per 100,000.

In this publication, trends with average annual percent changes (AAPCs) of greater than or equal to +/- 0.5 are described as rising or falling. Estimated AAPCs between -0.5 and 0.5 which are not significant are described as stable, while significant estimates in this range are described as rising or falling. Actual values are noted in the text only for statistically significant AAPC estimates. Three-year moving averages smooth the fluctuations caused by random variation in the time trend graphs; numbers in the text thus may not match graphs exactly.

Regional analyses

While variation in cancer incidence rates across Canada can provide insight into etiology, caution must be used when interpreting regional patterns. Observed differences may be due to registry data quality or random variation. In this publication, at least one of two conditions had to be met to warrant discussion of regional rates that differed significantly from the national rate: either the regional incidence pattern mirrored the pattern of known risk factors (e.g., Kaposi sarcoma) or known registry practices (e.g., melanoma), or the regional pattern was consistent between the sexes

and could not be explained by data quality factors (e.g., non-Hodgkin lymphoma). Three regions were created in order to increase the number of cases and produce more stable results. The Prairie region includes Manitoba, Saskatchewan and Alberta, while Newfoundland and Labrador, Prince Edward Island, Nova Scotia and New Brunswick make up the Atlantic region and the three territories are the North region.

Subgroup analyses

For some cancers, subgroup analyses by either morphology (e.g., testis, lung) or anatomic site (e.g., colorectal) were of interest. Data availability and quality were often deciding factors in determining the techniques that could be applied to subgroup analyses. Where rates were sufficiently high (>1.0 per 100,000), annual age-standardized rates could be calculated for subgroups and time trends examined (e.g., cervix). When the numbers were too small, several years were combined to produce stable rates for analysis (e.g., non-Hodgkin lymphoma). Subgroup definitions are documented in Table A4. The histological groups defined by Parkin et al.[133] were used extensively.

Potential Years of Life Lost (PYLL)

Potential years of life lost is the sum of the number of years of life that individual Canadians "lost"—that is, did not live—due to premature death. Calculating potential years of life lost provides an indication of the impact of premature death on society. PYLL is calculated by obtaining the number of cancer deaths in the 5-year age groups 20–24, 25–29, …, 40–44 for each province/territory for 1990–1999, multiplying these counts by the corresponding provincial life expectancy and summing the results. Cancers that are more common, have a younger age at death, and are more fatal will rank higher when ordered by PYLL.

Survival

Because follow-up information was not requested from the provincial/territorial registries, survival could not be calculated for all of Canada. Estimates were calculated using the OCR to approximate national survival. The generalizability of the Ontario estimates was confirmed by comparing traditional (cohort method) 5-year relative survival estimates for 1992–1997 with Canadian survival estimates requested from Statistics Canada. So that survival estimates would better represent current treatment, the period method was used to produce 5-year relative survival estimates for 1998–2002.[18]

Table A1
Definitions of CYAC cancers and criteria for inclusion

	Cancer definitions		Criteria for inclusion	
Cancer	ICD-9	ICD-10	N ≥ 1000 in 10 years	≥ 25% of all cases in adults occur in young adults
Lip, oral cavity, pharynx (including nasopharynx, excluding salivary glands)	140, 141, 143–149	C00–C06, C09–C14	✔	
Colorectal (including anus)	153, 154	C18–C21	✔	
Lung (trachea, bronchus, lung)	162	C33, C34	✔	
Thymus	164.0	C37		✔
Sarcoma		See Table A2	✔	✔
Melanoma (cutaneous)	172	C44 with morphology codes 8720–8790	✔	✔
Breast (female)	174	C50	✔	
Uterus (corpus and NOS)	179, 182	C54, C55	✔	
Cervix uteri	180	C53	✔	✔
Ovary*	183.0	C56	✔	
Testis	186	C62	✔	✔
Bladder	188	C67	✔	
Kidney (including ureter)	189.0–189.2	C64–C66	✔	
Brain (and other CNS)	191, 192	C70–C72	✔	✔
Thyroid	193	C73	✔	✔
Adrenal	194.0	C74		✔
Other endocrine	194.1–194.9	C75		✔
Non-Hodgkin lymphoma	200, 202	959, 967–971	✔	
Hodgkin lymphoma	201	965, 966	✔	✔
Leukemia	204–208	980–994	✔	
Kaposi sarcoma	-	9140	✔	✔
All cancers	140–208, excluding 173 SCC and BCC	C00–C80, excluding C44 & C80 with morphology codes 8090–8110	✔	

* Ovarian tumours of borderline malignancy were excluded

NOS = not otherwise specified; CNS = central nervous system; SCC = squamous cell cancer; BCC = basal cell cancer

Table A2
CYAC sarcoma definition

ICD-O-2 code	Morphology term
880	Soft tissue tumors and sarcomas, NOS
881–883	Fibromatous neoplasms
884	Myxomatous neoplasms
885–888	Lipomatous neoplasms
889–892	Myomatous neoplasms
8930	Endometrial stromal sarcoma
8933	Adenosarcoma
8950	Mullerian mixed tumor
8951	Mesodermal mixed tumor
8963	Rhabdoid sarcoma
8964	Clear cell sarcoma of kidney
8982	Malignant myoepithelioma
8990	Mesenchymoma, malignant
8991	Embryonal sarcoma
9020	Phyllodes tumor, malignant
904	Synovial-like neoplasms
9120	Hemangiosarcoma
9124	Kupffer cell sarcoma
9130	Hemangioendothelioma, malignant
9134	Intravascular bronchial alveolar tumor
9150	Hemangiopericytoma, malignant
9170	Lymphangiosarcoma
918–924	Osseous and chondromatous neoplasms
925	Giant cell tumors
926	Miscellaneous bone tumors
9270	Ondontogenic tumor, malignant
9273	Cementoblastoma
9330	Ameloblastic fibrosarcoma
9363	Melanotic neuroectodermal tumor
9364	Peripheral neuroectodermal tumor
9370	Chordoma
9490	Ganglioneuroblastoma
9500	Neuroblastoma, NOS
9530	Meningioma, malignant
9539	Meningeal sarcomatosis
9540	Neurofibrosarcoma
9560	Neurilemmoma, malignant
9561	Triton tumor, malignant
958	Granular cell tumors and alveolar soft part sarcoma

Table A3

Weights* used for age-standardization in CYAC age groups

Age group	20–44 Weights	15–44 Weights	20–49 Weights
15–19		0.14114	
20–24	0.17998	0.15457	0.15748
25–29	0.21579	0.18533	0.18882
30–34	0.22168	0.19040	0.19398
35–39	0.20006	0.17183	0.17506
40–44	0.18249	0.15673	0.15968
45–49			0.12498
Total	1.00000	1.00000	1.00000

* Based on 1991 Canadian population[25]

Table A4
Subgroup definitions for CYAC cancers

Cancer	Subgroup	Morphology/Topology (ICD-O-2)
Lip, oral cavity and pharynx	Lip	C00.0–C00.9
	Tongue	C01.9–C02.9
	Mouth and Gums	C03.0–C06.9
	Oral pharynx	C09.0–C09.9, C10.0–C10.9
	Nasopharynx	C11.0–C11.9
	Hypopharynx	C12.9, C13.0–C13.9
	Ill-defined	C14.0–C14.9
	Squamous cell	8050–8083
	Other cancer	All other morphologies, excluding 8000, 8010
Colon and rectum	Right colon	C18.0–C18.4
	Left colon	C18.5–C18.7
	Rectosigmoid junction, rectum, anus and anal canal	C19.9, C20.9, C21.0–21.8
	Colon, NOS and overlapping lesion of colon	C18.8–C18.9
	Adenocarcinoma	8050, 8140–8510
	Other and unspecified cancer	All other morphologies
Lung	Squamous	8050–8076
	Adenocarcinoma	8140, 8211, 8230–8231, 8250–8260, 8323, 8480–8490, 8550–8560, 8570–8572
	Small-cell carcinoma	8040–8045
	Large-cell carcinoma *(include giant cell, clear-cell and large-cell undifferentiated carcinoma)*	8012–8031, 8310
	Unspecified carcinoma	8010–8011, 8032–8034
	Other specified carcinoma *(include adenoid cystic, mucoepidermoid, and large-cell neuroendocrine carcinomas, and carcinoid tumours)*	All other morphologies in the range 8010–8572
	Other and unspecified cancer	All other morphologies
Melanoma	Skin of lip, NOS	C44.0
	Eyelid	C44.1
	External ear	C44.2
	Skin of other and unspecified parts of face	C44.3
	Skin of scalp and neck	C44.4
	Skin of trunk	C44.5
	Skin of upper limb and shoulder	C44.6
	Skin of lower limb and hip	C44.7
	Overlapping lesion of skin	C44.8
	Skin, NOS	C44.9

Table A4 *(cont'd)*
Subgroup definitions for CYAC cancers

Cancer	Subgroup	Morphology/Topology (ICD-O-2)
Breast	Infiltrating duct carcinoma	8500
	Infiltrating ductular carcinoma	8521
	Mucinous adenocarcinoma	8480–8481
	Comedocarcinoma	8501
	Lobular carcinoma	8520
	Infiltrating duct and lobular carcinoma	8522
	Medullary carcinoma	8510, 8512
	Paget's disease	8540–8543
	Other and unspecified carcinoma	All other morphologies in the range 8010–8573
	Unspecified cancer	8000–8004
	Other specified cancer	All other morphologies
Body of the uterus	Adenocarcinoma *(include adenosquamous carcinoma and adenocarcinoma with squamous differentiation)*	8050, 8140–8141, 8190–8211, 8230–8231, 8260–8263, 8310, 8380, 8430, 8440–8490, 8510, 8560, 8570–8572
	Unspecified carcinoma	8010–8034
	Unspecified cancer	8000–8004
	Other specified cancer	All other morphologies
Cervix	Squamous cell carcinoma (excluding microinvasive)	8051–8075
	Microinvasive squamous cell carcinoma	8076
	Adenocarcinoma	8050, 8140–8510
	Adenosquamous carcinoma	8560, 8570
	Unspecified carcinoma	8010–8034
	Other and unspecified cancer	All other morphologies
Ovary	Carcinomas:	
	Serous carcinoma	8441–8462, 9014
	Mucinous carcinoma	8470–8490, 9015
	Endometrioid carcinoma	8380–8381, 8560, 8570
	Clear cell carcinoma	8310–8313, 9110
	Adenocarcinoma NOS	8140–8190, 8211–8231, 8260, 8440
	Unspecified carcinomas	8010–8034
	Other specified carcinomas	All other morphologies in range 8010–8570, 9014–9015, 9110, excluding 8240–8245
	Sex cord-stromal tumours	8590–8671
	Germ cell tumours	8240–8245, 9060–9102
	Unspecified cancer	8000–8004
	Other specified cancer	All other morphologies
Testis	Seminoma	906
	Non-seminoma	907–908
	Other	All other morphologies
Bladder	Transitional cell carcinoma	8050, 8120–8122, 8130
	Squamous cell carcinoma	8051–8076
	Adenocarcinoma	8140–8145, 8190–8231, 8260–8263, 8310, 8480–8490, 8560, 8570
	Other and unspecified cancer	All other morphologies
Kidney	Kidney, NOS	C64.9
	Renal pelvis	C65.9
	Ureter	C66.9

Table A4 *(cont'd)*
Subgroup definitions for CYAC cancers

Cancer	Subgroup	Morphology/Topology (ICD-O-2)
Brain	Astrocytomas	9384, 9400–9421, 9424, 9481
	Glioblastomas	9440–9442
	Oligodendroglial & mixed gliomas	9382, 9450–9451
	Ependymal tumours	9383, 9391–9394
	Gliomas of uncertain origin	9380, 9381, 9422, 9423, 9430, 9443, 9460, 9480
	Medulloblastoma	9470–9472; C71.6–C71.7 and morphology 9364, 9473
	Other embryonal tumours	9490, 9500–9504; 9364, 9473, excluding topology C75.3
	Other neuroepithelial tumours	8680, 9390, 9491, 9505, 9506, 9520–9523
	Unspecified cancer	8000–8045
	Other specified cancer	All other morphologies
	Brain	C71.0–C71.9
	Meninges	C70.0–C70.9
	Spinal cord	C72.0
	Cauda equina	C72.1
	Cranial nerve	C72.2–C72.5
	Other	C72.8–C72.9
Thyroid	Follicular carcinoma	8290, 8330–8334
	Papillary carcinoma	8050, 8260, 8340, 8350, 8450
	Medullary carcinoma	8510–8511
	Anaplastic carcinoma	8020–8034
	Other and unspecified cancer	All other morphologies
Non-Hodgkin lymphoma	*AIDS-defining and -related:*	
	Immunoblastic and Burkitt (including small cell, noncleaved) lymphoma	9684, 9686–9687
	Intermediate grade large cell diffuse lymphoma	9593, 9680–9683
	Working Formula classification:	
	Small lymphocytic	9670–9671
	Follicular	9690–9698
	Diffuse	9593, 9595, 9672–9677, 9680–9683, 9688, 9710, 9711, 9715
	High-grade	9684–9687, 9594
	Other specified T/NK-cell	9700–9709, 9712–9714, 9716–9719
	Lymphoma NOS	9590–9592
Hodgkin lymphoma	Lymphocytic predominance	9657–9659, 9660
	Nodular sclerosis	9663–9667
	Mixed cellularity	9652
	Lymphocytic depletion	9653–9655
	Unspecified Hodgkin lymphoma	9650, 9661, 9662
Leukemia	Chronic myeloid leukemia	9863
	Acute myeloid leukemia	9861, 9866, 9867, 9871, 9891, 9910
	Chronic lymphoid leukemia	9823
	Acute lymphoid leukemia	9821, 9826
	Other leukemia	All other morphologies in the range 9800–9941

1. For further information of the contents of this report, please contact

Surveillance Unit, Division of Preventive Oncology
Cancer Care Ontario
620 University Avenue
Toronto, Ontario, M5G 2L7
Tel: (416) 971-9800
Fax: (416) 971-6888
E-mail: surveillanceunit@cancercare.on.ca

2. Cancer data

Provincial and Territorial Cancer Registries

NEWFOUNDLAND AND LABRADOR

Sharon Smith
Director, Cancer Care Program
Eastern Health
Dr. H. Bliss Murphy Cancer Centre
300 Prince Philip Drive
St. John's, Newfoundland, A1B 3V6
Tel: (709) 777-7588
Fax: (709) 753-0927
www.nctrf.nf.ca

PRINCE EDWARD ISLAND

Dagny E. Dryer
Director
PEI Cancer Registry
PEI Cancer Treatment Centre
Riverside Drive
Charlottetown, Prince Edward Island
C1A 8T5
Tel: (902) 894-2167
Fax: (902) 894-2187

NOVA SCOTIA

Maureen MacIntyre
Director
Surveillance and Epidemiology Unit
Cancer Care Nova Scotia
Bethune Building, Room 571
1278 Tower Road
Halifax, Nova Scotia, B3H 2Y9
Tel: (902) 473-5172
Fax: (902) 473-4425
www.cancercare.ns.ca

NEW BRUNSWICK

Christofer Balram
Provincial Epidemiologist
Director of Provincial Epidemiology Service
Department of Health and Community Services
P.O. Box 5100
520 King Street, 2nd Floor, Carleton Place
Fredericton, New Brunswick, E3B 5G8
Tel: (506) 453-3092
Fax: (506) 456-2780

QUEBEC

Michel Beaupré
Fichier des tumeurs du Québec
Ministère de la Santé et des Services sociaux
Direction générale de la santé publique
1075, Chemin Ste-Foy, 11ième étage
Québec, Québec G1S 2M1
Tel: (418) 266-6739
Fax: (418) 266-4609
www.msss.gouv.qc.ca/statistiques/tumeurs.html

ONTARIO

Kamini Milnes
Director, Informatics
Cancer Care Ontario
620 University Avenue
Toronto, Ontario, M5G 2L7
Tel: (416) 217-1260
Fax: (416) 217-1304
www.cancercare.on.ca

FOR FURTHER INFORMATION

MANITOBA

Jeri Kostyra
Manager
Epidemiology and Cancer Registry
CancerCare Manitoba
675 McDermot Ave.
Winnipeg, Manitoba, R3E 0V9
Tel: (204) 787-2174
Fax: (204) 786-0628
www.cancercare.mb.ca

SASKATCHEWAN

Heather Stuart
Director, Cancer Registry
Saskatchewan Cancer Agency
Allan Blair Cancer Centre
4101 Dewdney Avenue
Regina, Saskatchewan, S4T 7T1
Tel: (306) 766-2695
Fax: (306) 766-2179
www.scf.sk.ca

ALBERTA

Heather Bryant
Director
Population and Health Information
Alberta Cancer Board
1331 29th Street North West
Calgary, Alberta, T2N 4N2
Tel: (403) 521-3901
Fax: (403) 270-3898
www.cancerboard.ab.ca

YUKON

Sherri Wright
Director of Insured Health Services
Yukon Cancer Registry
Health Services Branch
Yukon Government
Box 2703 (H-2)
Whitehorse, Yukon, Y1A 2C6
Tel: (867) 667-5202
Fax: (867) 393-6486

BRITISH COLUMBIA

Mary McBride
Director, Cancer Registry
BC Cancer Agency
Cancer Control Research Program
600 West 10th Avenue
Vancouver, British Columbia, V5Z 4E6
Tel: (604) 707-5925
Fax: (604) 877-1868
www.bccancer.bc.ca

NUNAVUT

Sylvia Healy
Director of Registry
Department of Health and Social Services
Box 1000, Station 1000
Iqaluit, Nunavut, X0A 0H0
Tel: (867) 975-5700
Fax: (867) 975-5780

NORTHWEST TERRITORIES

Dr. André Corriveau
Chief Medical Health Officer and
Registrar, Disease Registries
Department of Health and Social Services
Government of the N.W.T.
Box 1320, 5022 49th Street
Centre Square Tower, 6th Floor
Yellowknife, N.W.T., X1A 2L9
Tel: (867) 920-8646
Fax: (867) 873-0442
www.hlthss.gov.nt.ca

Public Health Agency of Canada

Cancer Surveillance On-Line
http://www.phac-aspc.gc.ca/ (select surveillance button)

3. General Cancer Information

For information regarding any aspects of cancer (such as cancer prevention, screening, diagnosis, treatment and care), or to obtain additional copies of this publication, please contact the **Canadian Cancer Society's (CCS) Cancer Information Service** at **1 888 939-3333** or your local CCS office (see below)**.**

CANADIAN CANCER SOCIETY

National Office

Canadian Cancer Society &
National Cancer Institute of Canada
10 Alcorn Avenue, Suite 200
Toronto, Ontario M4V 3B1
Tel. (416) 961-7223
Fax. (416) 961-4189
www.cancer.ca and
www.ncic.cancer.ca

Newfoundland & Labrador Division

Canadian Cancer Society
Viking Building, 2nd Floor
P.O. Box 8921
136 Crosbie Road
St. John's, Newfoundland A1B 3K3
Tel. (709) 753-6520
Fax. (709) 753-9314

Prince Edward Island Division

Canadian Cancer Society
1 Rochford Street, Suite #1
Charlottetown, Prince Edward Island
C1A 9L2
Tel. (902) 566-4007
Fax. (902) 628-8281

Nova Scotia Division

Canadian Cancer Society
5826 South Street, Suite 1
Halifax, Nova Scotia B3H 1S6
Tel. (902) 423-6183
Fax. (902) 429-6563

New Brunswick Division

Canadian Cancer Society
133 Prince William Street
P.O. Box 2089
Saint John, New Brunswick E2L 3T5
Tel. (506) 634-6272
Fax. (506) 634-3808

Quebec Division

Maison de la Société canadienne du cancer
5151 boul. de l'Assomption
Montréal, Québec H1T 4A9
Tel. (514) 255-5151
Fax. (514) 255-2808

Ontario Division

Canadian Cancer Society
1639 Yonge Street
Toronto, Ontario M4T 2W6
Tel. (416) 488-5400
Fax. (416) 488-2872

Manitoba Division

Canadian Cancer Society
193 Sherbrook Street
Winnipeg, Manitoba R3C 2B7
Tel. (204) 774-7483
Fax. (204) 774-7500

Saskatchewan Division

Canadian Cancer Society
1910 McIntyre Street
Regina, Saskatchewan S4P 2R3
Tel. (306) 790-5822
Fax. (306) 569-2133

Alberta & N.W.T. Division

Canadian Cancer Society
#200, 2424-4th Street S.W.
Calgary, Alberta T2S 2T4
Tel. (403) 228-4487
Fax. (403) 228-4506

British Columbia & Yukon Division

Canadian Cancer Society
565 West 10th Avenue
Vancouver, British Columbia V5Z 4J4
Tel. (604) 872-4400
Fax. (604) 879-4533

cancer care ontario | action cancer ontario

Working together to create the best cancer system in the world

Questions about Cancer?

When you want to know more about cancer
call the Canadian Cancer Society's Cancer Information Service

1 888 939-3333 Monday to Friday: 9 a.m. – 6 p.m.

Distributed by:

Canadian Cancer Society | Société canadienne du cancer