Tobacco or Health in Ontario

Tobacco-attributed cancers and deaths over the past 50 years ... and the next 50

Division of Preventive Oncology
Cancer Care Ontario

Tobacco or Health in Ontario

Eric Holowaty

Sandrene Chin Cheong

Sandro Di Cori

John Garcia

Rita Luk†

Christine Lyons

Marc-Erick Thériault

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Surveillance Unit
and
Prevention Unit
Division of Preventive Oncology
Cancer Care Ontario
and
†Ontario Tobacco Research Unit

Obtaining Further Information

Information similar to that presented in this monograph is available from the Ontario Cancer Registry for other cancer sites.

Requests for data should be made in writing to:

Request Group, Surveillance Unit
Division of Preventive Oncology
Cancer Care Ontario
620 University Avenue
Toronto, Ontario
M5G 2L7

e-mail: gordon.fehringer@cancercare.on.ca

Telephone: 416-217-1237
Fax: 416-971-6888

Summaries of the current burden and determinants of cancer in Ontario are found on our website at: www.cancercare.on.ca

For further information about tobacco and its effects on health, or for additional copies of this monograph, contact:

Research Office, Prevention Unit,
Division of Preventive Oncology
Cancer Care Ontario
620 University Avenue
Toronto, Ontario
M5G 2L7

Telephone: 416-971-9800, ext. 1218
Fax: 416-217-1265
e-mail: christine.lyons@cancercare.on.ca

Disclaimer

Preface

This monograph presents information on incidence, mortality and survival for tobacco-related cancers and on general mortality for Ontario's population back to 1950, and projected forward to 2050. It was developed to provide comprehensive background information for the Prevention Unit, as a resource document for planning, and as a tool for monitoring tobacco control in Ontario.

The basis for the information in this monograph is the Ontario Cancer Registry (OCR). The OCR is operated by Cancer Care Ontario (CCO), through funding from the Ontario Ministry of Health and Long-Term Care. The OCR is situated within the Surveillance Unit of the Division of Preventive Oncology, which is located at the provincial office of CCO in Toronto. The Prevention Unit is also situated within the Division of Preventive Oncology.

The authors would like to thank Mr. Gordon Fehringer and Dr. Michael Finkelstein for providing estimates of tobacco-attributable mortality in Ontario over the period 1994-98. Special thanks to Dr. Jan Barendregt at the University of Antwerpt, in Holland, for permitting us to use the new *Prevent,* version 2.9 (Beta), macro-simulation system to project future smoking scenarios in Ontario and the likely associated outcomes. We are also grateful to Dr. Bill Evans, Vice President System Therapy Program Leader and Director, Cost Evaluation and Quality Improvement; Dr. Terrence Sullivan, Vice President and Head of the Division of Preventive Oncology; Dr. Roberta Ferrance, Director of the Ontario Tobacco Research Unit; and Professor Sue Horton, Chair of the Department of Social Sciences at the University of Toronto, for reviewing a draft of this document.

This monograph could not have been produced without the valuable assistance of Mrs. Virginia Hunter (desktop publishing). The authors would like to acknowledge the contribution of all operations staff within the Surveillance Unit, and the Registry Support Group (within the Information Systems Department), for their ongoing efforts to ensure the generation of timely and high-quality cancer incidence data for Ontario. Finally, the authors would like to acknowledge the contribution of all staff within the Prevention Unit and the Ontario Tobacco Strategy Media Network, as well as the Ontario Tobacco Strategy Steering Committee, for their ongoing efforts in the primary prevention of cancer.

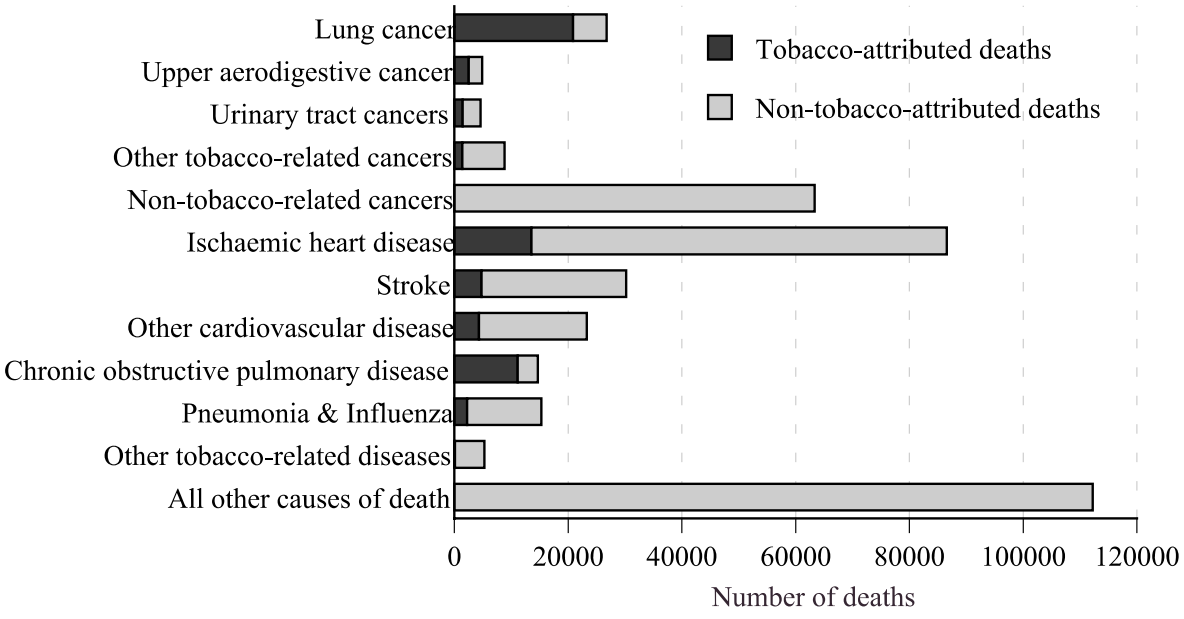
Executive Summary

The serious epidemic of chronic disease in the last century and the early 21st century is the direct result of the production, distribution and marketing of cigarettes and other tobacco products. While tobacco has long historical roots in North America, particularly traditional spiritual ceremonial use by aboriginal peoples, the rise of tobacco use and resulting public health crisis followed the cigarette companies' mass manufacturing, distribution, marketing, and mass addiction of the public to their nicotine delivery devices.

In the late 1920s, the first empirical evidence linking cigarette use to lung cancer began to appear and by 1964 the weight of the evidence led the U.S. Surgeon General to conclude that cigarettes cause lung cancer. Over the decades since then, there has been a steady accumulation of scientific evidence that tobacco company products are responsible for many chronic diseases and are the leading avoidable cause of premature death in North America, including Ontario. Government action on this problem has been slow and not commensurate with its gravity. It has only been since the late 1980s that Canadian governments have actually begun to address tobacco through effective public policy.

For many years now, tobacco use has been considered the single most significant cause of preventable morbidity and mortality in Canada, and in most other developed countries. Within Ontario, over the recent five-year period, 1994-1998, it is estimated that approximately 62,000 Ontarians died from diseases directly attributable to smoking. Approximately 30% (17,000) of all cancer deaths in Ontario men and 17% (8,900) of all cancer deaths in Ontario women are related to cigarette smoking over this period, and 16% (13,500) of all ischaemic heart disease deaths and 76% of chronic obstructive pulmonary disease deaths (11,100) are caused by smoking. While cigarettes are the dominant hazard, other uses of tobacco, including cigars, pipes, smokeless tobacco and environmental tobacco smoke (passive smoking) also are sources of significant morbidity and mortality.

Number of deaths in Ontario, 1994-1998 (Tobacco-attributed compared to Non-tobacco attributed)

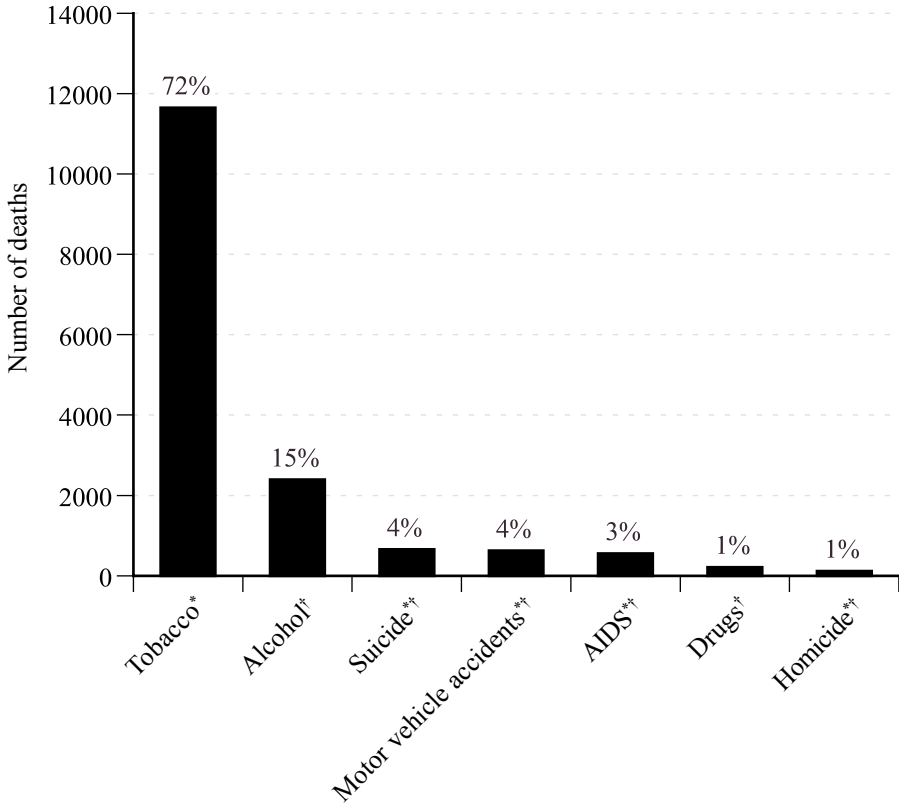


Since 1950, nearly 1/2 million Ontarians have died because of tobacco. This toll is approximately six times greater than the sum of all Ontario deaths attributed to alcohol, drugs, motor vehicle accidents and AIDS over the same period.

Currently, 50 Ontarians die each day because of tobacco. This is equivalent to 2 deaths per hour or 1 death every 30 minutes. To use an analogy, the death toll from tobacco in Ontario is equivalent to one fully loaded jumbo jet crashing every 6th day without any survivors.

At the present time, of every 1,000 Ontarians who smoke, about half will die from smoking, if they continue; approximately one-quarter will die before the age of 65 years. In contrast, of every 1,000 Ontarians, only 9 are expected to die over a lifetime in traffic accidents; and only 1 will be murdered.

Figure 5. Important causes of death in Ontario, 1992



* Source: Single *et al.* (1996)

† Source: OCR SEERStat CD, mortality file (August 2000); see CCO 2000 in References section

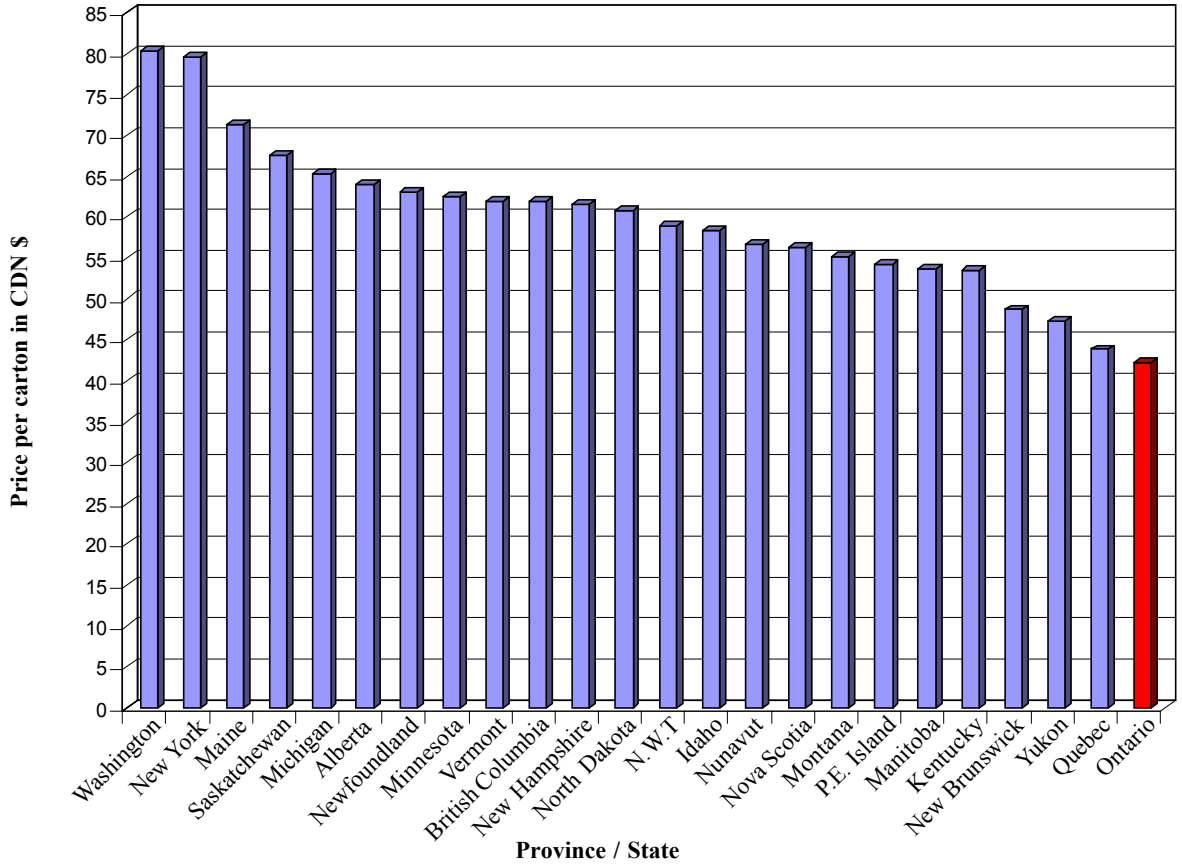
*† Suicide deaths (n=987), excludes those due to Alcohol (n=244) and Drugs (n=78)
Motor Vehicle Accidents deaths (n=1,103), excludes those due to Alcohol (n=468) and Drugs (n=10)
AIDS deaths (n=581), excludes those due to Drugs (n=25)
Homicide deaths (n=185), excludes those due to Alcohol (n=14) and Drugs (n=14)

It is not enough that tobacco use exacts a huge toll on the health of Ontarians. The economic costs of smoking on our society are exorbitant. The direct health care costs associated with smoking in Ontario in 1992 were approximately $1.1 billion (Single *et al.* 1996). It is likely that this represents only a

minority of the real economic toll of smoking because the costs associated with lost productivity and earnings as a result of illness, disability and death have been estimated to be another $2.6 billion (Single *et al.* 1996).

Ever since the combined federal and provincial cuts reduced tax rates in the eastern provinces, Ontario has had the lowest cigarette prices in Canada (and in North America). Currently, even with a $10 per carton tax increase, a carton of cigarettes will be cheaper in Ontario than the US state with the lowest prices, Kentucky ($42.26 +$10= $52.26 in Ontario vs. $53.45 in Kentucky).

Average price of a carton of 200 cigarettes (including taxes) in Canadian dollars[1]
(Exchange rate as of March 28, 2002 - $1 US = $1.5935Cdn)

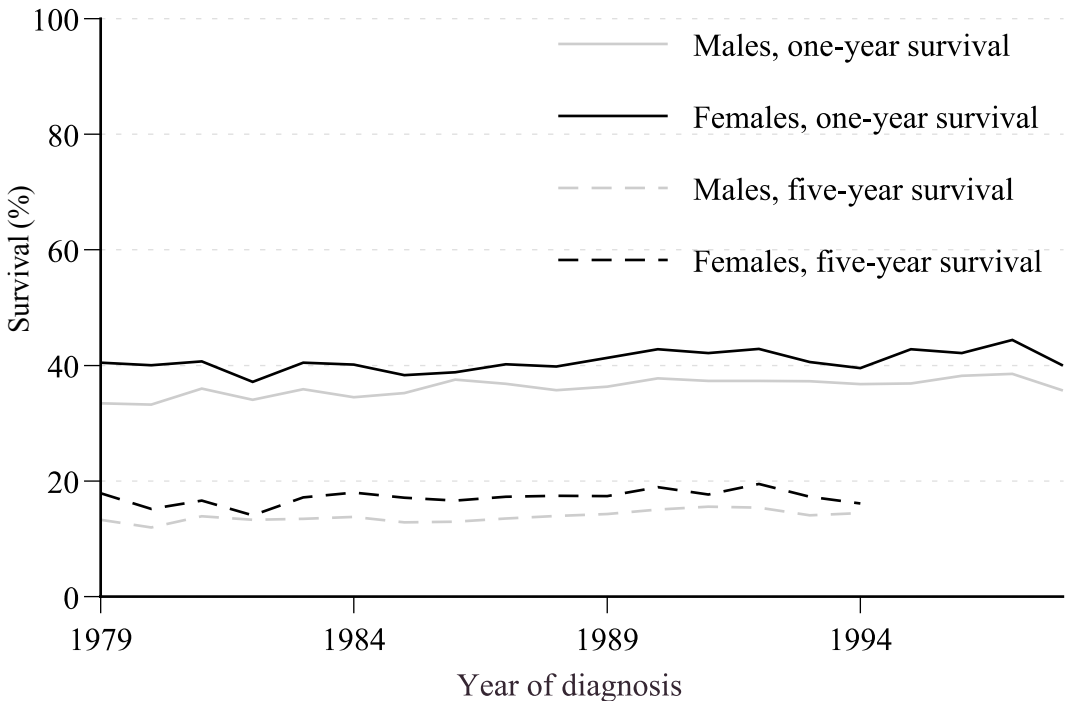


[1] Data courtesy of: Smoking and Health Action Foundation, Ottawa, April 2002

Several studies indicate that increases in cigarette prices lead to reductions in smoking initiation by youth, and reduction in the number of cigarettes smoked by adults (US Dept. of Human and Health Services 2000). According to the World Bank, price increases are the most effective and cost effective deterrent. In fact, a 10% increase in price will reduce adult tobacco consumption by about 3-4% (World Bank 1999). These reductions subsequently reduce the incidence of tobacco-related diseases, which ultimately improve population health and reduce mortality rates.

Based on Health Canada estimates, 18.4 billion cigarettes are sold annually in Ontario. This translates into 736 million packs of 25 cigarettes being sold every year in Ontario. Thus, a tax increase of $10 per carton could raise an estimated $836 million annually in the province, even taking into account the targeted reduction in consumption.

Relative survival for lung cancer in Ontario, by year of diagnosis, 1979-1998



Current chances of surviving lung cancer remain poor. Five-year relative survival for lung cancer of both men and women combined is 15%. Even with recent advances in technology (e.g. CT scanning), survival after diagnosis of lung cancer has remained poor over time. Prevention constitutes the single most effective way to combat lung cancer. State wide experience in the U.S. suggests that comprehensive tobacco control programs can reduce rates of tobacco-related illnesses including cancer rates. One aspect of an effective tobacco control or prevention program is tobacco pricing. As of the time of this monograph, Ontario had the lowest tobacco price per carton cigarette in Canada.

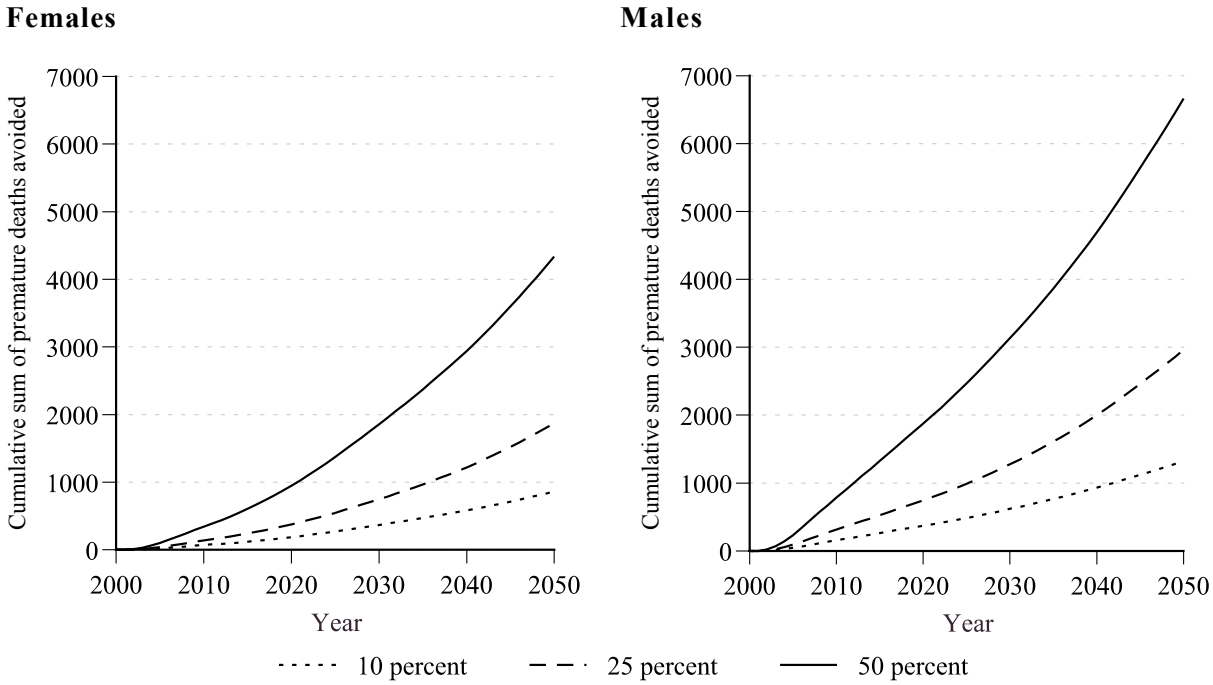
The monograph explores the effects of three price increase scenarios:

1. a 10% increase in the price of a pack of cigarettes
2. a 25% increase, and
3. a 50% increase.

All three scenarios show a clear increase in the percentage of premature deaths avoided over the period stretching from 2002 (the point at which each intervention is assumed to occur) to 2050.

The following figure describes the exponential increase in cumulative number of lives saved as a result of one aspect of tobacco control: effective pricing strategies. The magnitude of the benefit is directly proportional to the relative reduction in smoking prevalence. Further, this benefit continues to increase for at least fifty years following the intervention. Indeed, after 20 years, not even half of the benefit will have been achieved, largely due to the long latency and lag from the time of change in the prevalence of smoking to the full effect.

Cumulative mortality reduction in Ontario adults under 65 years of age after three tobacco control scenarios, by sex, 2002-2050



Most Canadians are aware that smoking causes addiction, lung cancer, emphysema and heart disease. However, there is much less awareness of the wide range and nature of diseases and disorders caused by tobacco, partly because the spectrum is so broad, and these diseases are often managed separately by clinical specialists. The term tobaccosis has been invented to describe, collectively, all those diseases resulting from smoking, chewing and snuffing of tobacco, and the breathing of tobacco smoke (Colditz 2000).

Disorders include:

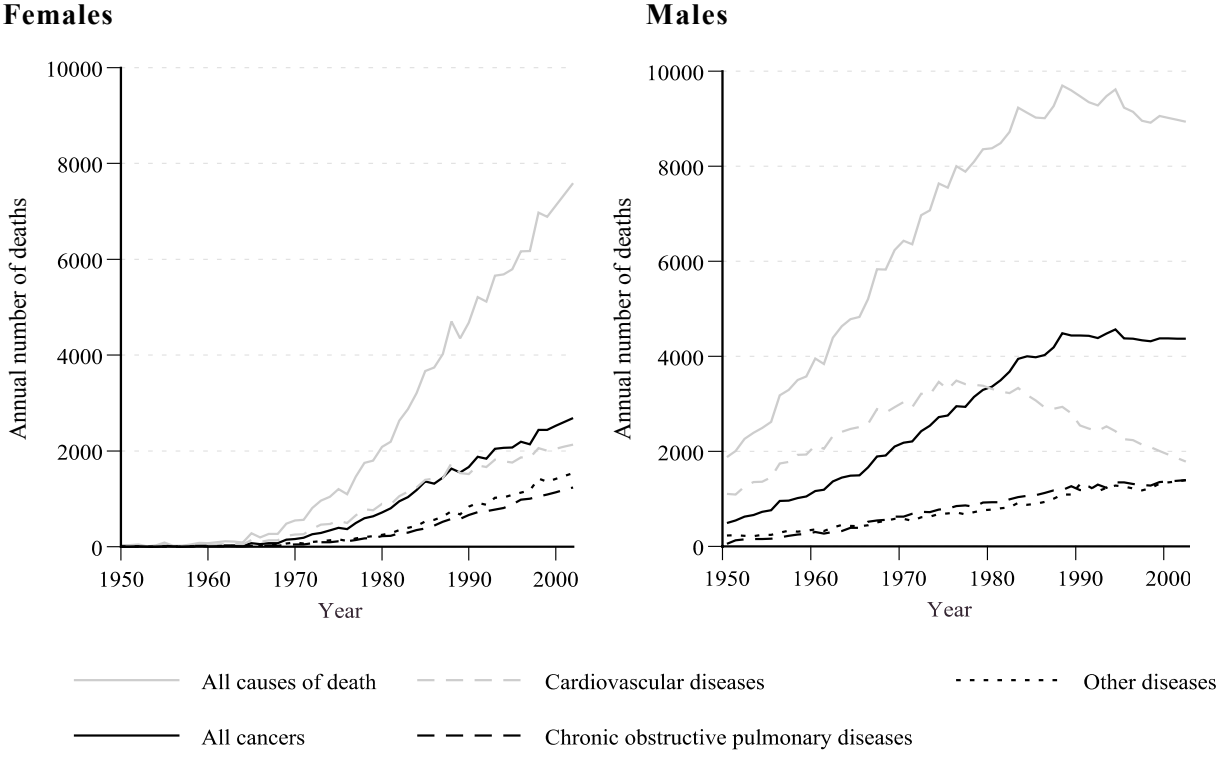
- nicotine addiction
- cancers of the mouth, nasopharynx, larynx, trachea, bronchi, lungs, oesophagus, stomach, liver, pancreas, kidney, bladder, prostate and colorectum
- leukaemias
- atherosclerosis of the cardiovascular system, including ischaemic heart disease, cardiomyopathy, aortic and other aneurysms, cerebral vascular hemorrhages and blockages, renal failure and peripheral vascular disease
- emphysema and other forms of chronic obstructive pulmonary disease
- pneumonia and childhood asthma
- regional ileitis
- cirrhosis of the liver
- immunological deficiencies and failures of endocrine and metabolic functions

- cataracts
- osteoporosis
- optic neuropathy
- infertility
- fetal and neonatal deaths and child disabilities

Over the period 1994-1998, there were approximately 62,350 deaths in attributed to tobacco for men and women combined. This represented 16% of all deaths over this period. Cancer was the commonest cause of tobacco-attributed deaths (TAM) in Ontario, accounting for 42% of all TAM. These cancer deaths were dominated by lung cancer, which accounted for 33.4% of TAM. The second and third largest causes of TAM were ischaemic heart disease and chronic obstructive pulmonary disease, which accounted for 21.7% and 17.8% TAM respectively.

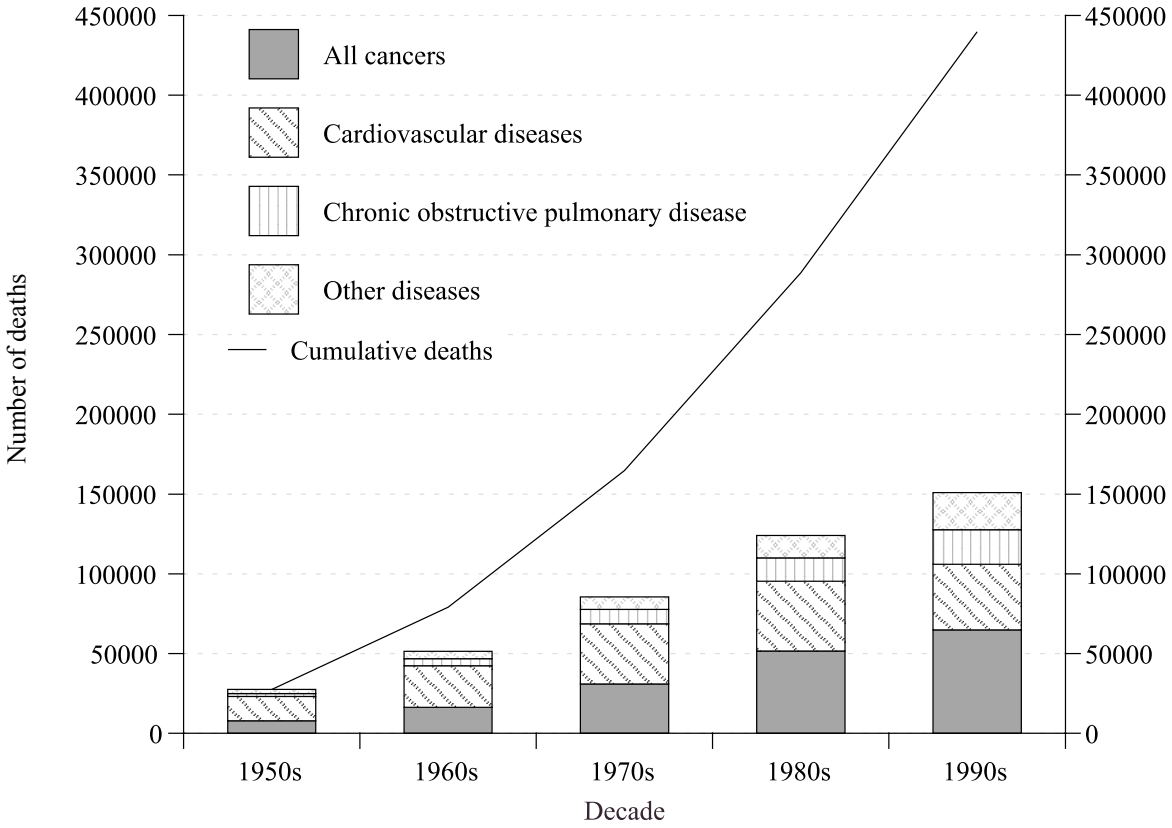
Over the past 50 years, almost 500,000 deaths have occurred among Ontario women and men that can be directly attributed to tobacco (see figure below). Compared to men, the epidemic of TAM in women has lagged by about 25-30 years. Since the late 1980s, there has been a slow but steady fall in the burden of TAM among Ontario men; unfortunately, this trend is not seen among Ontario women.

Annual number of tobacco-attributed deaths in Ontario, by sex, 1950-1999



In the current year, 2002, the annual number of TAM deaths among Ontario women is expected to exceed 7,500; among men, almost 9,000 deaths will be directly attributed to tobacco. These estimates comprise 18% and 21% of all deaths expected among Ontario women and men, respectively, in 2002.

Number of tobacco-attributed deaths in Ontario, 1950-1999, both sexes combined



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Tobacco Use and Health

History of tobacco use and control in Ontario

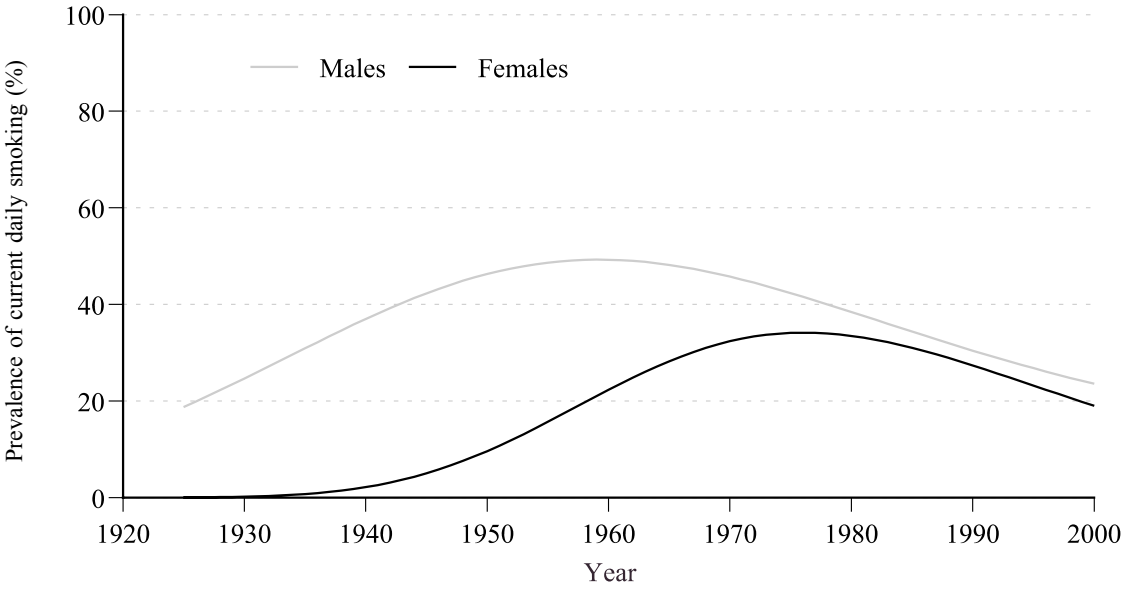
The serious epidemic of chronic disease in the last century and the early 21st century is the direct result of the production, distribution and marketing of cigarettes and other tobacco products. While tobacco has long historical roots in North America, particularly traditional spiritual ceremonial use by aboriginal peoples, the rise of tobacco use and resulting public health crisis followed the cigarette companies' mass manufacturing, distribution, marketing, and mass addiction of the public to their nicotine delivery devices. In the late 1920s, the first empirical evidence linking cigarette use to lung cancer began to appear and by 1964 the weight of the evidence led the U.S. Surgeon General to conclude that cigarettes cause lung cancer. Over the decades since then, there has been a steady accumulation of scientific evidence that tobacco company products are responsible for many chronic diseases and are the leading avoidable cause of premature death in North America, including Ontario. Government action on this problem has been slow and not commensurate with its gravity. It has only been since the late 1980s that Canadian governments have actually begun to address tobacco through effective public policy. In Ontario, the Government demonstrated leadership during the 1990s with the creation of the Ontario Tobacco Strategy and the passage of the omnibus *Tobacco Control Act*, which was, to that time, one of the most comprehensive tobacco control acts in the world. At the turn of the millennium, the Government of Ontario has taken steps to enhance the Ontario Tobacco Strategy, but much more is required to address the crisis.

By 1920, approximately 0.35 kilograms of tobacco were consumed per person 15 years of age or older in Canada (approximately 400 cigarettes per capita). By the early 1970s, per capita consumption had peaked in Canada at approximately 3.0 kilograms (approximately 3,000 cigarettes per capita). The majority of the increase in per capita consumption was attributable to additional consumption by existing cigarette smokers, not a shift from other forms of tobacco use (e.g. cigars, pipes). In 1998, per capita consumption in Canada had fallen to 1.9 kilograms. By 1998, Canadians were consuming approximately 50 billion cigarettes per year, down from the estimated 63.5 billion cigarettes in 1986, when Canada ranked eighth in the world among consumers of manufactured cigarettes (Ferrence 1989).

Filtered cigarettes were introduced in the 1950s in response to the mounting evidence that cigarettes caused a myriad of diseases. In vitro tar yield has also fallen over the years; in 1967, only 2% of cigarettes had less than 15 milligrams of tar; by 1992, this figure had risen to 69%.

Figure 1 shows the trends in prevalence of current daily cigarette smoking among persons 15 years of age or older in Ontario from 1925 to 2000. This figure shows consistent decline in the prevalence of cigarette smoking among men, from 49% in the late 1950s and early 1960s to about half that figure (24%) by the year 2000. In Ontario women, the long-term trend has been one of increasing use in the post-World War II period until about 1970s. Since then, the prevalence of smoking among Canadian women has fallen by almost half, to 19% in the year 2000. Since 1960, the gender gap in smoking has almost disappeared. For example, in 1960, 27% more men than women smoked; by the year 2000, this gap had diminished to 6%.

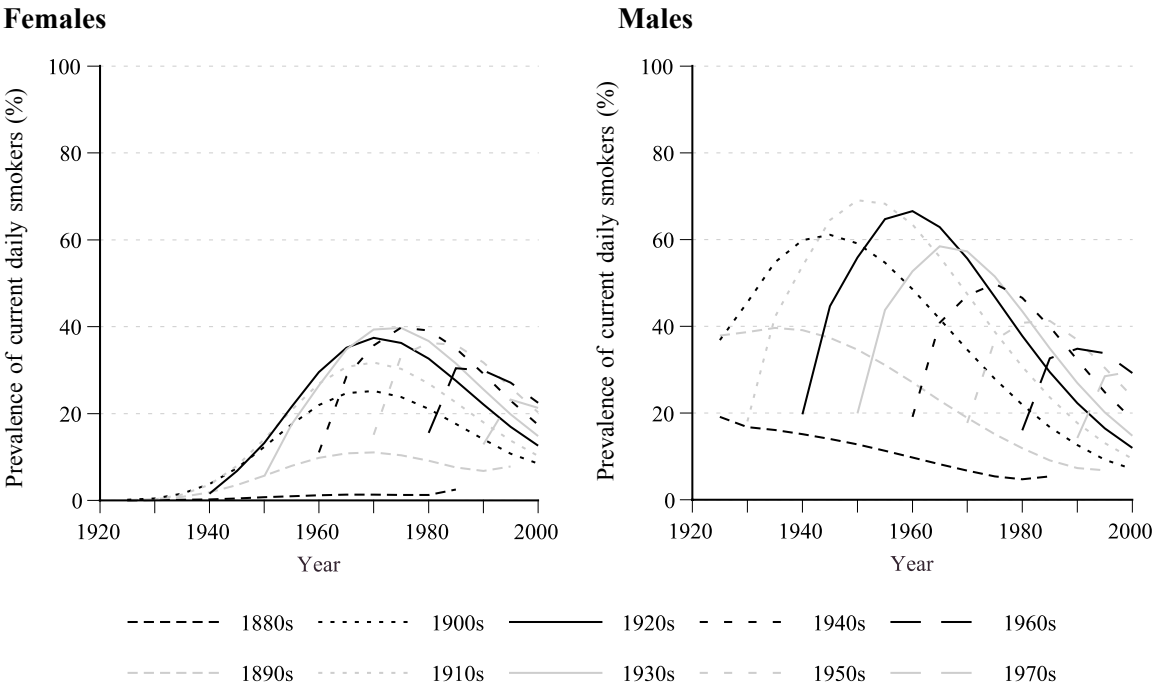
Figure 1. Age-standardized prevalence rate of current daily cigarette smoking in Ontario, 15+ years of age[1], 1925-2000



[1] Standardized to the 1991 Canadian population

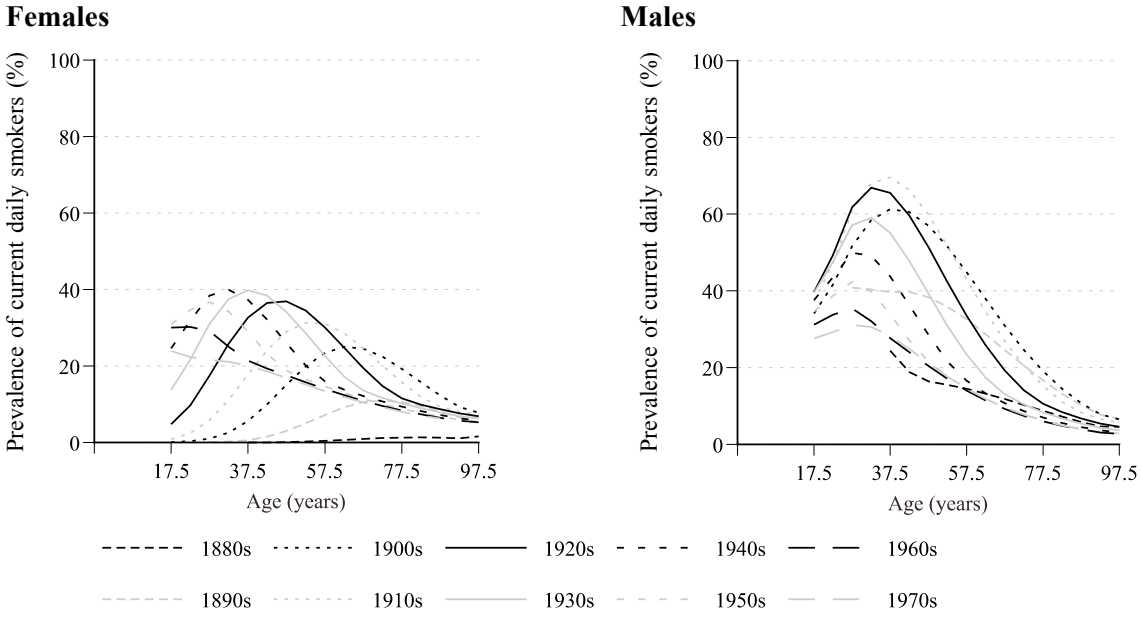
Interesting patterns in the prevalence of cigarette smoking are apparent when temporal trends in different birth cohorts are described. Figure 2 describes the changes in smoking prevalence by decade of birth, beginning with Ontario women and men born in the 1880s.

Figure 2. Prevalence of current daily cigarette smokers, in Ontario, by birth cohort and sex, 1925-2000



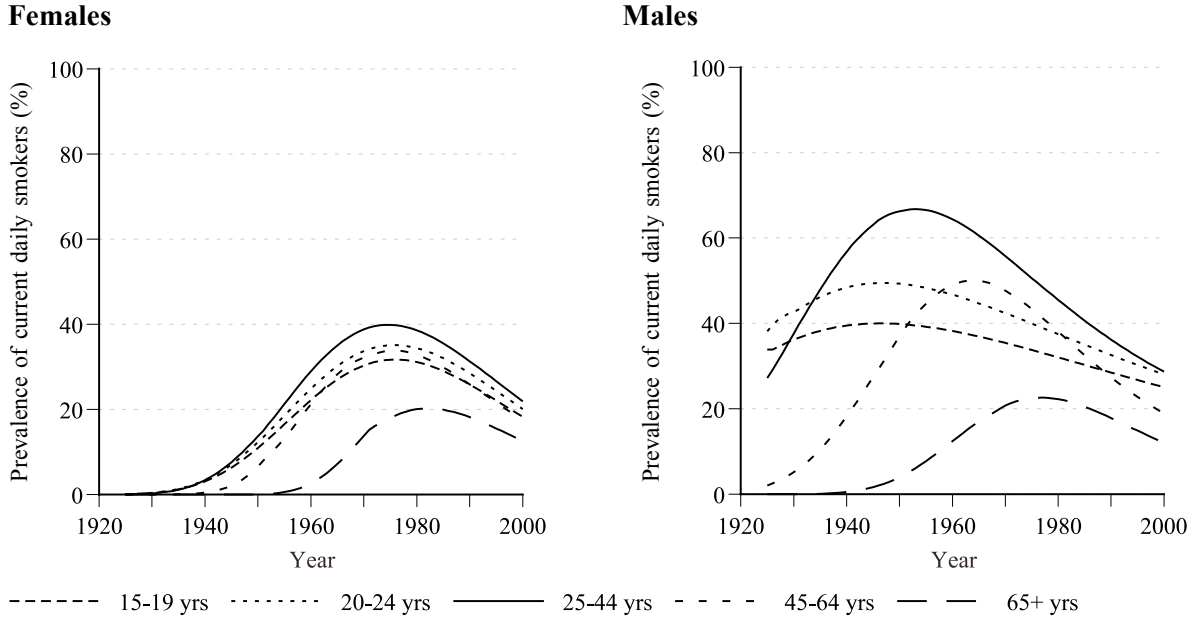
In the cohort of men born between 1910s and 1920s, smoking reached a peak prevalence of 60% when these men were in their 20s and 30s; among Ontario women, the cohort born in the 1930s and 1940s reached a peak prevalence of 38%, also when they were in their 20s and 30s (see Figure 3).

Figure 3. Prevalence of current daily cigarette smokers, in Ontario, by birth cohort and sex, and by age



Finally, Figure 4 describes the prevalence of current daily cigarette smoking, by age group, over time, in Ontario. The prevalence has been highest among both men and women 25 to 44 years of age. However, by year 2000, the gap had closed considerably, largely because of smoking cessation in this age group.

Figure 4. Prevalence of current daily cigarette smokers, in Ontario, by age group[1] and sex, 1925-2000



[1] Standardized to the 1991 Canadian population

Smoking generally begins during adolescence. About 85% of adult smokers who were surveyed in 2000 reported having had their first cigarette by 18 years of age. The age of initiation dropped steadily over most of the 20th century (Adlaf and Paglia 2001).

The Centre for Addictions and Mental Health has tracked cigarette smoking in Ontario among high school students since 1977. In the late 1970s, about 49% of Grade XI students smoked cigarettes on a daily basis. This figure fell to about 30-35% by the 1980s and early 1990s. However, with the drop in cigarette taxes in 1994, there has been a significant increase in the subsequent prevalence of smoking among teenagers (Adlaf and Paglia 2001).

It should be noted that as of March 2002, the average price of a carton of 200 cigarettes was $42.26 in Ontario. As recently proposed, an increase in the price of a carton of cigarettes by $5.00 would translate into a relative increase of 12% in the price of cigarettes in Ontario. In the Forecasting section of this monograph, the impact of this price increase is described on future premature mortality that will be avoided as a result of this intervention.

The health consequences of tobacco use

Tobacco use became popular among European settlers to North America in the 16th Century, but it had likely been used by aboriginal peoples for centuries before that time. Even in the New Millennium, tobacco is still used in much the same fashion as it was in the 16th Century. The adverse health effects have been suspected for almost as long, as witnessed in this quote from nearly 400 years ago:

> *"Tobacco dryeth the brain, dimmeth the sight, vitiateth the smell, hurteth the stomach, destroyeth the concoction, disturbeth the humours and spirits, corrupteth the breath, induceth a trembling of the limbs, exsicateth the windpipe, lungs, and liver, annoyeth the milt, scorcheth the heart, and causeth the blood to be adjusted"* (Tobias Verner 1577-1660).

For many years now, tobacco use has been considered the single most significant cause of preventable morbidity and mortality in Canada, and in most other developed countries. Within Ontario, over the recent five-year period, 1994-1998, it is estimated that approximately 62,000 Ontarians died from diseases directly attributable to smoking. Approximately, 30% (17,000) of all cancer deaths in Ontario men and 17% (8,900) of all cancer deaths in Ontario women are related to cigarette smoking over this period, and 16% (13,500) of all ischaemic heart disease deaths and 76% of chronic obstructive pulmonary disease (COPD) deaths (11,100) are caused by smoking (see below). While cigarettes are the dominant hazard, other uses of tobacco, including cigars, pipes, smokeless tobacco and environmental tobacco smoke (passive smoking) also are sources of significant morbidity and mortality.

It is not enough that tobacco use exacts a huge toll on the health of Ontarians. The economic costs of smoking on our society are exorbitant. The direct health care costs associated with smoking in Ontario in 1992 were approximately $1.1 billion (Single *et al.* 1996). It is likely that this represents

only a minority of the real economic toll of smoking because the costs associated with lost productivity and earnings as a result of illness, disability and death have been estimated to be another $2.6 billion (Single *et al.* 1996).

Most Canadians are aware that smoking causes addiction, lung cancer, emphysema and heart disease. However, there is much less awareness of the wide range and nature of diseases and disorders caused by tobacco, partly because the spectrum is so broad, and these diseases are often managed separately by clinical specialists. The term tobaccosis has been invented to describe, collectively, all those diseases resulting from smoking, chewing and snuffing of tobacco, and the breathing of tobacco smoke (Colditz 2000). These disorders include:

- nicotine addiction
- cancers of the mouth, nasopharynx, larynx, trachea, bronchi, lungs, oesophagus, stomach, liver, pancreas, kidney, bladder, prostate and colorectum
- leukaemias
- atherosclerosis of the cardiovascular system, including ischaemic heart disease, cardiomyopathy, aortic and other aneurysms, cerebral vascular hemorrhages and blockages, renal failure and peripheral vascular disease
- emphysema and other forms of chronic obstructive pulmonary disease
- pneumonia and childhood asthma
- regional ileitis
- cirrhosis of the liver
- immunological deficiencies and failures of endocrine and metabolic functions
- cataracts
- osteoporosis
- optic neuropathy
- infertility
- fetal and neonatal deaths and child disabilities

While tobaccosis may not be the only cause of these disorders, it is either an important contributing factor, or the dominant cause for all of them.

Burning and subsequent inhalation of tobacco smoke is the most hazardous form of exposure, since literally thousands of noxious chemicals, including the addictive drug nicotine, nitriles, aldehydes, hydrogen cyanide, benzopyrenes, phenols, carbon monoxide and the radio-isotope polonium 210 are produced. Many scientists consider tobacco the foremost human poison of the twentieth century (Ravenholt 1990).

Deep inhalation of tobacco smoke results in the deposit of tarry residues on the respiratory mucus that coats the lungs and respiratory passages. Components that are highly soluble are quickly absorbed into the blood stream and transported throughout the body. Less soluble tars may be moved up the respiratory tract and expelled by coughing; however, a significant fraction of these tars will be swallowed and passed to the oesophagus, stomach, intestine and liver. It is clear that the chronic inhalation of tobacco smoke exposes all tissues and organs in the body to powerful mutagens and carcinogens, giving rise to the broad spectrum of cancers, degenerative diseases and other disorders, that constitute tobaccosis. It should be noted that because of the long latent interval from initiation of smoking to serious disease, the important causal role of tobacco for many of these disorders has been obscured. The huge size required of cohort studies, and the careful follow-up over many decades, has been a significant limiting factor in the quantification of tobaccosis. According to Sir Richard Doll:

> *"it should not be thought surprising that smoking should be a cause of cancer in many different organs, for tobacco smoke contains a vast number of chemicals (over 4,000), of which at least 50 have been shown to be carcinogenic in animals; further, inhalation is a most effective way of getting a chemical into the systemic circulation and distributed throughout the body"* (Doll 1996).

Epidemiologic studies which were initiated in the 1940s and 1950s established cigarette smoking as the major cause of lung cancer in North America and Europe. Subsequently, tens of thousands of studies and reports have confirmed this association and have provided additional evidence that tobacco use is a cause of many other cancers, and other diseases and disorders (IARC 1986; MRC 1957; RCP 1962; RCP 1971; Surgeon General 1964; Surgeon General 1979; McLaughlin JK *et al.* 1996; Doll *et al.* 1994).

The sites of cancer that are unequivocally caused by tobacco use are highlighted in bold in Table 1. These cancers are convincingly related to tobacco smoking. For these, a dose-response relationship is clearly seen, and decreasing risks are observed after cessation of tobacco use. The type of tobacco used, the amount smoked per day, the number of years of use, the degree of inhalation and, possibly, individual susceptibility, are important determinants of risk.

Table 1 also summarizes many other important causes of death that have been attributed to tobacco smoking. Collectively, the frequencies of non-cancer tobacco-attributed deaths in Ontario are more

than double the frequencies of tobacco-attributed cancer deaths in Ontario (see Tables 3 and 4 in the Incidence and Mortality section).

Apart from clearly-identified diseases that are attributed to tobacco, there are many well-known pathophysiologic affects attributed to cigarette smoking:

- periodontal disease
- skin wrinkling
- fingernail discolouration; exacerbation of psoriasis
- higher rates of surgical and post-surgical complications; delayed wound healing
- increased metabolic rate; blood sugar abnormalities
- impaired immune responses
- injuries from fires; occupational injuries

It is not apparent that there is any safe level of cigarette smoking. Even after one cigarette, transient physiologic effects of smoking are apparent, particularly on the cardiovascular system. While it is quite possible that smoking a few cigarettes a day might not present much of a health hazard to most people, the reality is that few smokers are able to limit their habit that much. The majority of current Ontario smokers smoke more than 15 cigarettes a day - this is a level that conclusively increases the risk of many serious diseases (NPHS 1998/99).

Concerning the reversibility of the health hazards associated with smoking, there is good evidence that quitting will dramatically reduce the future risk of associated health hazards, but there is also some evidence that prior cumulative exposure can still have permanent consequences. Surveys reveal that many, if not most, current smokers want to quit. Most smokers assume that they will eventually give up the habit. Unfortunately, though, there is evidence that approximately half of all smokers never quit permanently. There is also mounting evidence that the likelihood of quitting has decreased over the years, perhaps because the daily intensity of smoking has increased. Regardless of the claims of the tobacco industry, it has been determined by independent scientists and public authorities that cigarette smoking is strongly addictive. Further, the pharmacologic and behavioural processes that determine tobacco addiction are similar to those that determine addiction to other drugs such as heroin and cocaine. It is sad and ironic that cocaine addicts in treatment find cigarettes harder to give up than cocaine itself (Kozlowski *et al.* 1989).

Table 1. Relative Risks[1] Associated with Tobacco Use, by Cause of Death and Gender

		Relative Risk			
		ex-smokers		current smoking	
	Tobacco-Related Conditions & ICD-9 Codes	M	F	M	F
Cancer	**Lip & Oropharyngeal Cancer, 140-141, 143-146, 148-149, 230.0**	1.76	1.76	4.55	4.55
	Oesophageal Cancer, 150, 230.1	1.79	1.79	4.01	4.01
	Stomach Cancer, 151, 230.2	1.11	1.11	1.41	1.41
	Anal Cancer, 154.2-154.3, 230.5-230.6	1.83	1.83	3.18	3.18
	Pancreatic Cancer, 157, 230.9	1.15	1.15	1.86	1.86
	Laryngeal Cancer, 161, 231.0	2.86	2.86	7.48	7.48
	Lung Cancer, 162, 231.2	6.75	5.07	13.0	11.4
	Vulvar Cancer, 184.4	n.a.	1.37	n.a.	3.42
	Penile Cancer, 187.1-187.4	1.60	n.a.	1.80	n.a.
	Bladder Cancer, 188, 233.7	1.66	1.66	2.72	2.72
	Renal Parenchymal Cancer, 189.0	1.61	1.61	1.64	1.64
	Renal Pelvic & Ureter Cancer, 189.1-189.2	1.95	1.95	3.96	3.96
Cardiovascular	Ischaemic Heart Disease, <65yr., 410-414	1.59	1.59	2.58	2.58
	Ischaemic Heart Disease, 65+yr., 410-414	1.12	1.12	1.54	1.54
	Pulmonary Circulatory Disease, 415.0, 416-417	6.70	6.70	9.80	9.80
	Cardiac Dysrhythmias, <65+yr., 427	1.59	1.59	2.58	2.58
	Cardiac Dysrhythmias, 65+yr., 427	1.12	1.12	1.54	1.54
	Heart Failure, Ill-defined, 428-429	n.a.	n.a.	n.a.	n.a.
	Stroke <65yr., 430-438	1.30	1.30	3.12	3.12
	Stroke 65+yr., 430-438	1.15	1.15	1.65	1.65
	Arterial Disease, 440-448	1.82	1.82	2.54	2.54
Resp	Pneumonia & Influenza, 480-486, 487	1.29	1.29	1.47	1.47
	COPD, 490-492, 496	6.70	6.70	9.80	9.80

Table 1. (continued)

	Tobacco-Related Conditions & ICD-9 Codes	Relative Risk ex-smokers M	ex-smokers F	current smoking M	current smoking F
GI	Ulcers, 531-534*	n.k.	n.k.	6.8	6.8
	Chrohn's Disease, 555	1.92	1.60	1.92	3.27
	Ulcerative Colitis, 556	1.71	1.71	0.63	0.63
Reproductive	Ectopic Pregnancy, 633	n.a.	1.27	n.a.	1.46
	Spontaneous Abortion, 634	n.a.	1.00	n.a.	1.36
	Haemorrhage, 640-641	n.a.	1.00	n.a.	1.62
	Poor Fetal Growth, 656.5	n.a.	1.00	n.a.	2.04
	Premature Rupture Membranes, 658.1-658.2	n.a.	1.00	n.a.	1.93
	Stillbirth, 740-759, 760-779	1.00	1.00	1.27	1.27
	Effect of Premature Rupture of Membranes, 761.1	1.00	1.00	1.93	1.93
	Effect of Ectopic Pregnancy, 761.4	1.00	1.00	1.46	1.46
Infancy	Effect of Spontaneous Abortion, 761.8	1.00	1.00	1.36	1.36
	Effect of Placental Complications, 762.0-762.1	1.00	1.00	1.62	1.62
	Slow Fetal Growth/Low Birthweight, 764-765	1.00	1.00	2.04	2.04
	SID Syndrome, 798.0 (and smoking during pregnancy)	1.00	1.00	2.44	2.44
ETS	Lung Cancer, 162†	n.k.	n.k.	1.32	1.32
	Ischaemic Heart Disease, 410-414†	n.k.	n.k.	1.24	1.24
	Asthma <15yr., 493 (morbidity)‡	n.k.	n.k.	1.4	1.4
	Lower Respiratory Illness <18months, 464, 466, 480-487, 490 (morbidity)‡	n.k.	n.k.	1.6	1.6

[1] Single *et al.* 1996; Ridolfo *et al.* 2001.
ETS Environmental Tobacco Smoke
n.a. not applicable
n.k. not known
* only if H. pylori positive
† never smokers exposed to current spousal smoking
‡ parental smoking

Table 2. Prevalence of smoking in Ontario, by age group and by gender, 1990 and 1996/97

Time Period And Age Group	Smoking Status							
	Current smokers (%)		Ex-smokers (%)		Never smoked (%)		Unknown (%)	
1990*	**M**	**F**	**M**	**F**	**M**	**F**	**M**	**F**
15-19 yrs	28.1	21.6	4.2	6.5	56.3	57.2	11.4	14.8
20-24 yrs	34.3	28.8	7.9	8.0	45.3	50.8	12.5	12.4
25-29 yrs	33.8	28.4	9.9	13.2	46.9	50.6	9.4	7.8
30-34 yrs	35.9	27.7	15.2	16.5	42.0	49.0	7.0	6.8
35-39 yrs	35.5	28.6	17.4	17.6	41.1	46.5	6.0	7.3
40-44 yrs	34.2	25.9	21.7	20.6	36.7	46.8	7.5	6.7
45-49 yrs	28.4	23.4	29.9	21.1	34.5	48.8	7.3	6.7
50-54 yrs	26.9	21.9	33.3	24.4	34.0	48.0	5.9	5.7
55-59 yrs	26.1	21.1	34.7	22.0	34.3	52.2	5.0	4.7
60-64 yrs	18.5	17.2	44.9	22.0	31.0	55.4	5.5	5.5
65-69 yrs	14.8	18.3	48.0	22.8	31.7	53.3	5.5	5.6
70-74 yrs	15.3	12.4	49.0	21.8	30.2	59.6	5.5	6.2
75-79 yrs	10.7	9.2	53.8	21.7	31.0	58.3	4.5	10.8
80-84 yrs	9.1	8.7	50.3	21.0	32.3	59.4	8.2	10.8
85+ yrs	6.6	1.9	41.9	13.3	42.2	75.1	9.3	9.7
Total	28.9	23.1	23.8	17.6	39.7	51.4	7.6	7.9

	Current smokers (%)		Ex-smokers (%)		Never smoked (%)		Unknown (%)	
1996/97†	**M**	**F**	**M**	**F**	**M**	**F**	**M**	**F**
15-19 yrs	27.6	21.3	15.9	21.3	56.3	57.1	0.2	0.2
20-24 yrs	34.2	28.5	19.7	20.2	45.8	51.2	0.2	0.2
25-29 yrs	33.8	27.9	18.9	20.9	46.8	51.0	0.5	0.2
30-34 yrs	36.0	27.5	21.9	23.1	42.0	48.8	0.1	0.6
35-39 yrs	35.7	29.1	23.3	24.3	40.5	46.0	0.5	0.6
40-44 yrs	34.9	25.9	28.2	26.1	36.2	47.1	0.7	0.9
45-49 yrs	28.1	23.7	36.2	26.9	34.7	49.1	1.0	0.2
50-54 yrs	27.3	22.5	38.7	29.3	33.2	47.4	0.7	0.8
55-59 yrs	26.0	20.5	40.1	26.7	33.3	52.5	0.6	0.3
60-64 yrs	18.8	16.5	50.3	28.2	30.2	54.3	0.7	1.1
65-69 yrs	15.6	17.8	51.8	27.5	32.5	53.7	0.2	0.9
70-74 yrs	15.8	12.5	53.4	27.8	29.9	58.9	0.9	0.8
75-79 yrs	10.8	9.0	56.6	30.3	32.0	59.2	0.6	1.5
80+ yrs	8.4	6.0	54.7	25.7	36.3	65.6	0.6	2.8
Total	29.0	23.0	30.9	25.0	39.5	51.4	0.5	0.6

* OHS (Ontario Health Survey) 1990 (n=34,704); estimates are weighted.

† NPHS (National Public Health Survey) 1996/7 (n=37,716); estimates are weighted.

Incidence and Mortality

Tobacco-attributed mortality (TAM) in Ontario females

Over the period 1994-1998, there were approximately 23,400 deaths in females attributed to tobacco. This represented 12.7% of all deaths over this period. Cancer was the commonest cause of tobacco-attributed deaths in Ontario women, accounting for 38.2% of all TAM (Table 3). These cancer deaths were also dominated by lung cancer, which accounted for 31.4% of female tobacco-attributed cancer deaths. Ischaemic heart disease accounted for a slightly larger proportion (20.4%) of female TAM than did chronic obstructive pulmonary disease (18.2%).

Table 3. Tobacco-attributed mortality in Ontario females, 1994-1998[1]

Disease	Tobacco-attributed deaths		Non-tobacco-attributed deaths[2]		Total Deaths	
	#	%	#	%	#	%
Cancers	**8,919**	**38.2**	**41,834**	**24.7**	**50,753**	**26.4**
Lung (ICD9 162)	7,333	31.4	2,820	1.7	10,153	5.3
Upper aerodigestive (ICD9 140-150, 161)	601	2.6	749	0.4	1,350	0.7
Urinary tract (ICD9 188, 189)	376	1.6	1,140	0.7	1,516	0.8
Other tobacco-related cancers	608	2.6	3,516	2.1	4,124	2.1
Non-tobacco-related cancers	0	0.0	33,610	19.9	33,610	17.5
Cardiovascular	**9,146**	**39.1**	**61,117**	**36.1**	**70,263**	**36.5**
Ischaemic heart disease	4,757	20.4	35,008	20.7	39,765	20.7
Stroke	2,443	10.5	15,321	9.1	17,764	9.2
Other	1,946	8.3	10,788	6.4	12,734	6.6
Respiratory	**5,244**	**22.4**	**9,114**	**5.4**	**14,358**	**7.5**
Chronic obstructive pulmonary disease	4,245	18.2	1,733	1.0	5,978	3.1
Pneumonia & Influenza	999	4.3	7,381	4.4	8,380	4.4
Other tobacco-related diseases	**61**	**0.3**	**2,373**	**1.4**	**2,434**	**1.3**
All other causes of death	**0[3]**	**0.0**	**54,629**	**32.3**	**54,629**	**28.4**
Total Deaths	**23,370**	**100.0**	**169,067**	**100.0**	**192,437**	**100.0**

[1] Based on actual underlying causes of death, Ontario, 1994-1998, and 1996 Census population of Ontario.

[2] Non-TAM=Total deaths less TAM deaths

[3] Does not include TAM due to accidents and injuries

Tobacco-attributed mortality (TAM) in Ontario males

Over the period 1994-1998, there were approximately 39,000 deaths in males attributed to tobacco. This represented 19.2% of all deaths over this period. Cancer was the commonest cause of tobacco-attributed deaths in Ontario men, accounting for 44.7% of all TAM (Table 4). These cancer deaths were dominated by lung cancer, which accounted for 34.7% of male tobacco-attributed deaths. The second and third largest causes of male TAM were ischaemic heart disease and chronic obstructive pulmonary disease, which accounted for 22.5% and 17.7% of male TAM respectively.

Table 4. Tobacco-attributed mortality in Ontario males, 1994-1998[1]

Disease	Tobacco-attributed deaths		Non-tobacco-attributed deaths[2]		Total Deaths	
	#	%	#	%	#	%
Cancers	**17,290**	**44.7**	**40,325**	**24.5**	**57,615**	**28.3**
Lung (ICD9 162)	13,515	34.7	3,114	1.9	16,629	8.2
Upper aerodigestive (ICD9 140-150, 161)	1,917	4.9	1,613	1.0	3,530	1.7
Urinary tract (ICD9 188, 189)	1,070	2.7	1,993	1.2	3,063	1.5
Other tobacco-related cancers	788	2.0	3,907	2.4	4,695	2.3
Non-tobacco-related cancers	0	0.0	29,698	18.0	29,698	14.6
Cardiovascular	**13,500**	**34.6**	**56,267**	**34.2**	**69,767**	**34.3**
Ischaemic heart disease	8,770	22.5	38,051	23.1	46,821	23.0
Stroke	2,352	6.0	10,057	6.1	12,409	6.1
Other	2,379	6.1	8,158	5.0	10,537	5.2
Respiratory	**8,138**	**20.9**	**7,484**	**4.6**	**15,622**	**7.7**
Chronic obstructive pulmonary disease	6,885	17.7	1,828	1.1	8,713	4.3
Pneumonia & Influenza	1,253	3.2	5,656	3.4	6,909	3.4
Other tobacco-related diseases	**56**	**0.1**	**2,795**	**1.7**	**2,851**	**1.4**
All other causes of death	**0**[3]	**0.0**	**57,567**	**35.0**	**57,567**	**28.3**
Total Deaths	**38,985**	**100.0**	**164,437**	**100.0**	**203,422**	**100.0**

[1] Based on actual underlying causes of death, Ontario, 1994-1998, and 1996 Census population of Ontario.

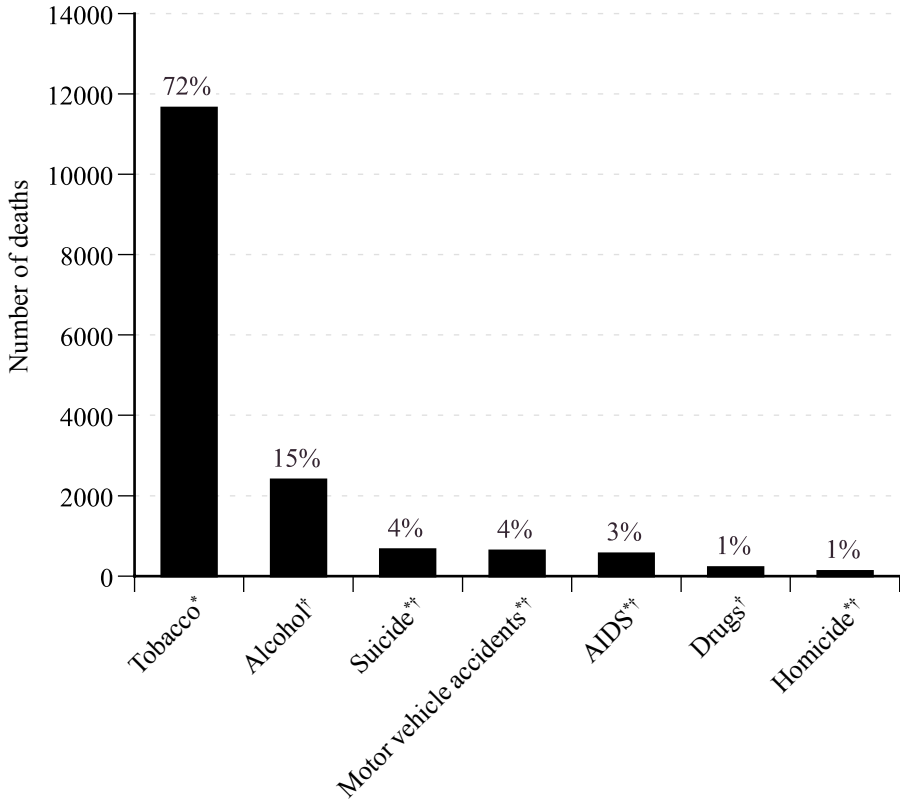
[2] Non-TAM=Total deaths less TAM deaths

[3] Does not include TAM due to accidents and injuries

Causes of death - tobacco compared to other important cancers, 1992

This bar chart (Figure 5) shows the dominance of tobacco as a cause of mortality in Ontario, relative to other notable lifestyle factors. Indeed, the sum total of deaths attributable to alcohol, suicide, motor vehicle accidents, AIDS, drugs and homicides (4,556) only amounts to 39% of all deaths attributable to tobacco in 1992.

Figure 5. Important causes of death in Ontario, 1992



* Source: Single *et al.* (1996)

† Source: OCR SEERStat CD, mortality file (August 2000); see CCO 2000 in References section

*† Suicide deaths (n=987), excludes those due to Alcohol (n=244) and Drugs (n=78)
Motor Vehicle Accidents deaths (n=1,103), excludes those due to Alcohol (n=468) and Drugs (n=10)
AIDS deaths (n=581), excludes those due to Drugs (n=25)
Homicide deaths (n=185), excludes those due to Alcohol (n=14) and Drugs (n=14)

Most common tobacco-attributed cancers in Ontario females

In 1994-1998, tobacco-attributed cancers accounted for 11.7% of all cancers diagnosed in Ontario women (Table 5). Lung cancer was the commonest tobacco-attributed cancer diagnosed, representing 8.8% of all cancers diagnosed. It ranked second or third in frequency for every age group in adult women, except for the age group 20-34 years (data not shown). The remaining tobacco-attributed cancers included a broad variety of types, most common of which were cancers of the bladder, kidney, pancreas, oropharynx, and oesophagus.

Table 5. Most common cancers diagnosed in Ontario females[1], 1994-1998

Cancers		Tobacco-attributed		Non-tobacco-attributed		Total	
Rank	**Site**	**#**	**%**	**#**	**%**	**#**	**%**
1	Breast	0	0.0	31,841	32.4	31,841	28.6
2	Colon/rectum	0	0.0	14,272	14.5	14,272	12.8
3	Lung	9,835	75.2	3,232	3.3	13,067	11.7
4	Corpus uteri	0	0.0	5,990	6.1	5,990	5.4
5	Ovary	0	0.0	4,759	4.8	4,759	4.3
6	Non-Hodgkin's lymphoma	0	0.0	4,453	4.5	4,453	4.0
7	Melanoma	0	0.0	3,238	3.3	3,238	2.9
8	Leukaemia	0	0.0	2,934	3.0	2,934	2.6
9	Cervix uteri	0	0.0	2,838	2.9	2,838	2.5
10	Thyroid	0	0.0	2,779	2.8	2,779	2.5
11	Pancreas	456	3.5	2,211	2.2	2,667	2.4
12	Upper aerodigestive	1,031	7.9	1,484	1.5	2,515	2.3
	Lip and oropharyngeal	506	3.9	960	0.9	1,466	1.3
	Oesophageal	306	2.3	404	0.4	710	0.6
	Laryngeal	218	1.7	121	0.1	339	0.3
13	Kidney	483	3.7	1,830	1.9	2,313	2.1
14	Bladder	673	5.1	1,391	1.4	2,064	1.9
15	Brain & Other CNS	0	0.0	1,907	1.9	1,907	1.7
All other sites		605	4.6	13,142	13.4	13,747	12.3
Total		**13,084**	**100.0**	**98,300**	**100.0**	**111,384**	**100.0**

[1] Non-melanoma skin cancer is not included as it is not recorded in the OCR

Most common tobacco-attributed cancers in Ontario males

In 1994-1998, tobacco-attributed cancers accounted for 20.8% of all cancer diagnosed in Ontario men (Table 6). Lung cancer was the commonest tobacco-attributed cancer diagnosed, representing 13.8% of all cancers diagnosed. It ranked first or second in frequency for every age group in adult men except for the age group 20-34 years (data not shown). The remaining tobacco-attributed cancers include a broad variety of types, most common of which were cancers of the bladder, oropharynx, kidney, larynx, oesophagus and pancreas.

Table 6. Most common cancers diagnosed in Ontario males[1], 1994-1998

Cancers		Tobacco-attributed		Non-tobacco-attributed		Total	
Rank	**Site**	**#**	**%**	**#**	**%**	**#**	**%**
1	Prostate	0	0.0	30,740	32.6	30,740	25.8
2	Lung	16,491	66.7	3,058	3.2	19,549	16.4
3	Colon/rectum	0	0.0	15,839	16.8	15,839	13.3
4	Upper aerodigestive	3,423	13.8	3,156	3.3	6,579	5.5
	Lip and oropharyngeal	1,389	5.6	1,897	2.0	3,286	2.8
	Oesophageal	856	3.5	782	0.8	1,638	1.4
	Laryngeal	1,178	4.8	477	0.5	1,655	1.4
5	Bladder	2,456	9.9	3,476	3.7	5,932	5.0
6	Non-Hodgkin's lymphoma	0	0.0	5,255	5.5	5,225	4.4
7	Kidney	1,182	4.8	2,799	3.0	3,981	3.3
8	Leukaemia	0	0.0	3,718	3.9	3,718	3.1
9	Melanoma	0	0.0	3,563	3.8	3,563	3.0
10	Stomach	425	1.7	2,819	3.0	3,244	2.7
11	Pancreas	542	2.2	1,943	2.1	2,485	2.1
12	Brain & Other CNS	0	0.0	2,262	2.4	2,262	1.9
13	Testis	0	0.0	1,385	1.5	1,385	1.2
14	Liver and intrahepatic bile duct	0	0.0	1,351	1.4	1,351	1.1
15	Thyroid	0	0.0	873	0.9	873	0.7
All other sites		200	3.0	12,189	12.9	12,389	10.4
Total		**24,720**	**100.0**	**94,395**	**100.0**	**119,115**	**100.0**

[1] Non-melanoma skin cancer is not included as it is not recorded in the OCR

Common cancers in Ontario, both sexes combined, 1994-1998

These pie charts show the dominance of tobacco-related cancers as a fraction of all incident cases (Figure 6) and as a fraction of all cancer deaths (Figure 7). It should be noted that the term "tobacco- related" refers to those cancers for which some or all are attributed to smoking.

Figure 6. Most common cancers diagnosed in Ontario, 1994-1998

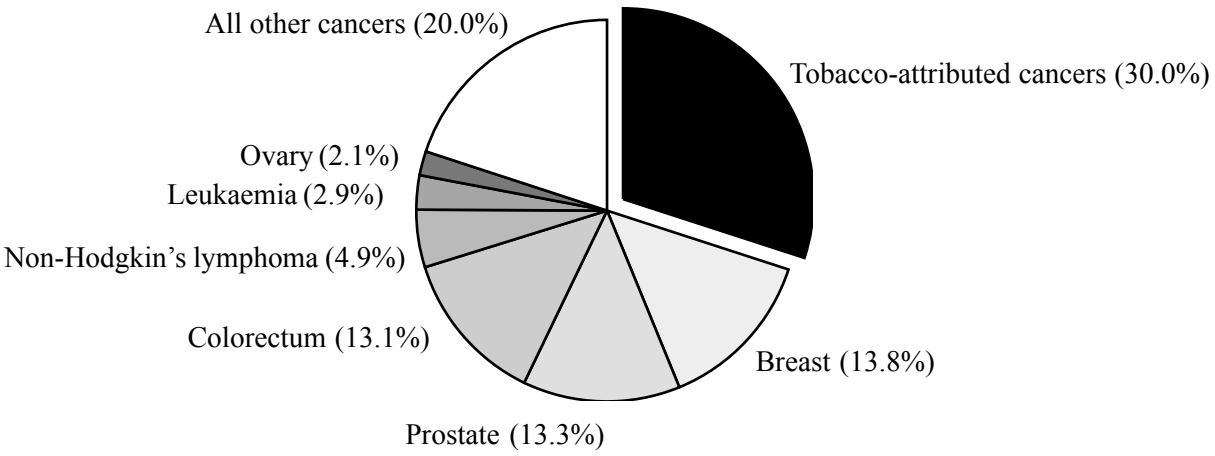
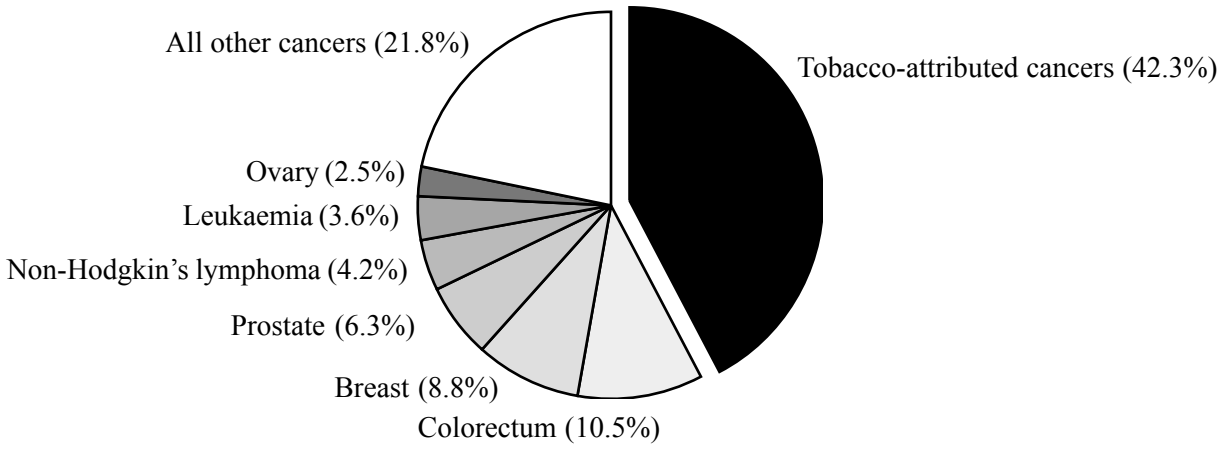


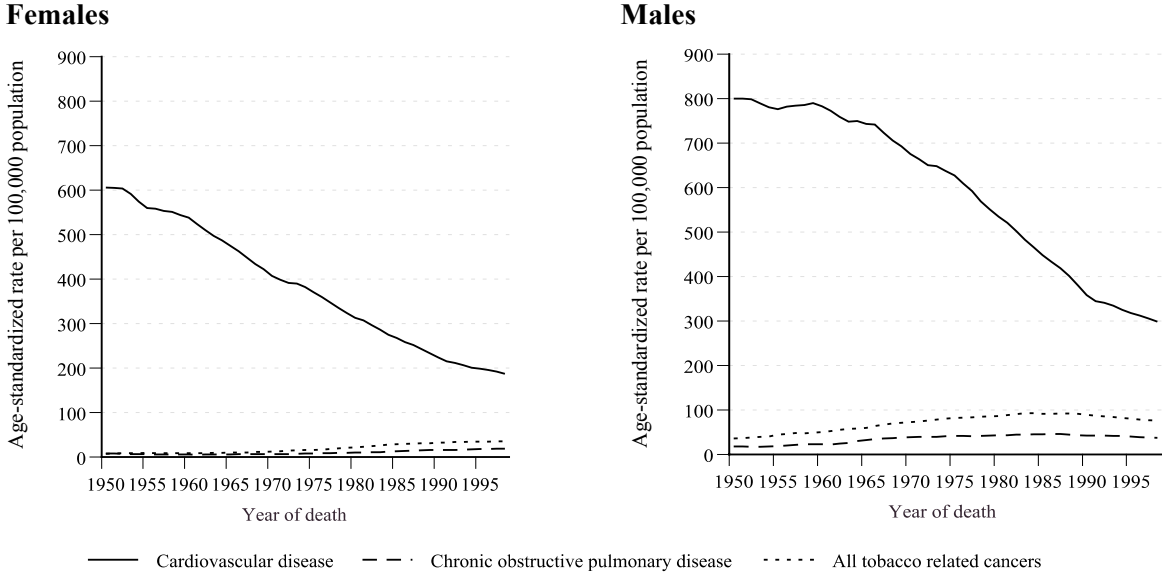
Figure 7. Most common cancer causes of death in Ontario, 1994-1998



Trends in all causes of mortality

Except for lung cancer, which has mostly been caused by smoking over this period, it is difficult to apportion the fractions of the other tobacco-related causes of death (Figure 8) that are attributable to smoking over time, partly because little information exists about the fraction of these various diseases attributable to other causes, as well as the confounding and interactive effects of smoking with these other causal factors. Later in this monograph, employing conservative assumptions about attribution estimates are presented of TAM back to 1950.

Figure 8. Age-standardized mortality rates (3-year moving average) for tobacco-related deaths in Ontario, 1950-1998



[1] Standardized to the 1991 Canadian population

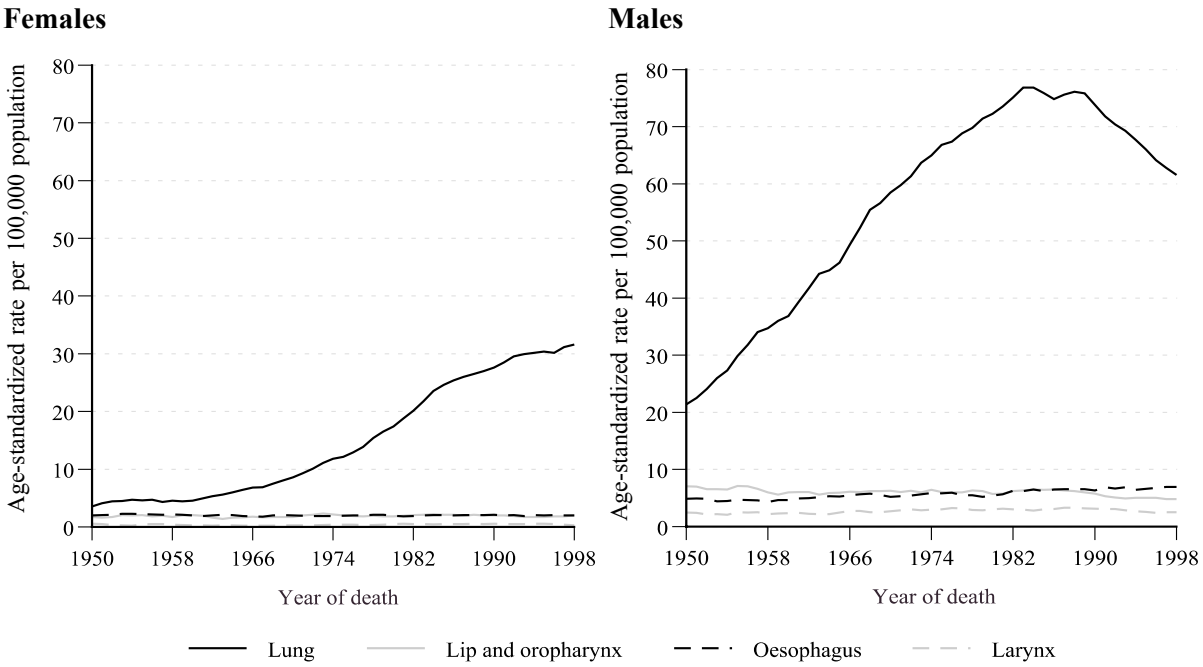
Table 7. Annual percentage change (APC) for tobacco-related mortality in Ontario, 1950-1998

Females	Period	APC (%)	**Males**	Period	APC (%)
Cardiovascular disease	1950-1960	-1.3	Cardiovascular disease	1950-1965	-0.5
	1960-1977	-2.5		1965-1977	-1.8
	1977-1992	-3.3		1977-1991	-3.7
	1992-1998	-2.0		1991-1998	-2.4
Chronic obstructive pulmonary disease	1950-1960	-3.8	Chronic obstructive pulmonary disease	1950-1962	+3.0
	1960-1973	+2.3		1962-1966	+10.9
	1973-1989	+4.8		1966-1986	+1.2
	1989-1998	+2.2		1986-1998	-1.6
All tobacco-related cancers	1950-1969	+1.0	All tobacco-related cancers	1950-1976	-0.4
	1969-1988	+0.6		1976-1985	+0.9
	1988-1998	-1.1		1985-1998	-0.4

Trends in cancer mortality

Lung cancer dwarfs other cancers commonly related to smoking (Figure 9). Temporal trends are similar for lung and laryngeal cancer, but not for oropharyngeal and oesophageal cancers, which are not as strongly associated with smoking and are attributable to other causes as well.

Figure 9. Age-standardized mortality rates (3-year moving average) for tobacco-related cancers in Ontario, 1950-1998



[1] Standardized to the 1991 Canadian population

Table 8. Annual percentage change (APC) for tobacco-related cancer mortality in Ontario, 1950-1998

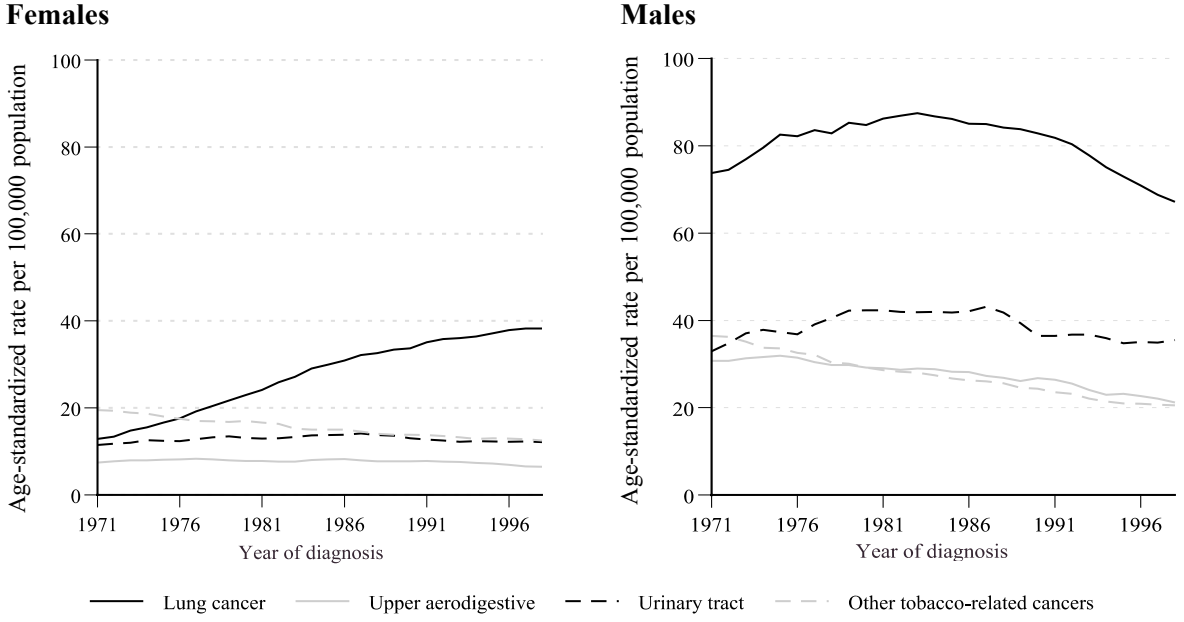
Females	Period	APC (%)	**Males**	Period	APC (%)
Lung cancer	1950-1961	n.s.	Lung cancer	1950-1969	+5.1
	1961-1985	+7.2		1969-1983	+2.0
	1985-1998	+1.9		1983-1989	n.s.
				1989-1998	-2.3
Lip & oropharyngeal cancer	1950-1998	n.s.	Lip & oropharyngeal cancer	1950-1987	-0.1
				1987-1998	-2.5
Oesophageal cancer	1950-1998	n.s.	Oesophageal cancer	1950-1998	+0.9
Laryngeal cancer	1950-1964	n.s.	Laryngeal cancer	1950-1988	+1.1
	1964-1995	+2.6		1988-1998	-3.0
	1995-1998	n.s.			

n.s. not significant

Trends in incidence for tobacco-related cancers

Temporal trends for incidence of lung cancer and upper aerodigestive tract cancers are similar to mortality patterns (Figure 10). The apparent fall in the incidence of urinary tract cancers from the mid 1980s may be largely due to a change in coding practices related to bladder cancer, and should, therefore, be interpreted with caution.

Figure 10. Age-standardized incidence rates (3-yr moving average) for tobacco-related cancers, first primaries only, in Ontario, 1971-1998



[1] Standardized to the 1991 Canadian population

Table 9. Annual percentage change (APC) for tobacco-related cancer incidence in Ontario, 1971-1998

Females	Period	APC (%)	**Males**	Period	APC (%)
Lung cancer	1971-1985	+6.5	Lung cancer	1971-1982	+1.5
	1985-1998	+1.8		1982-1992	+0.9
				1992-1998	-3.3
Upper aerodigestive	1971-1994	n.s.	Upper aerodigestive	1971-1991	-1.0
cancers	1994-1998	-5.4	cancers	1991-1998	-3.3
Urinary tract	1971-1986	+1.1	Urinary tract	1971-1981	+2.7
	1986-1998	-1.3		1981-1998	-1.5
Other tobacco-related	1971-1989	-1.9	Other tobacco-related	1971-1980	-2.6
cancers	1989-1998	-1.2	cancers	1980-1988	-1.7
				1988-1995	-2.7
				1995-1998	n.s.

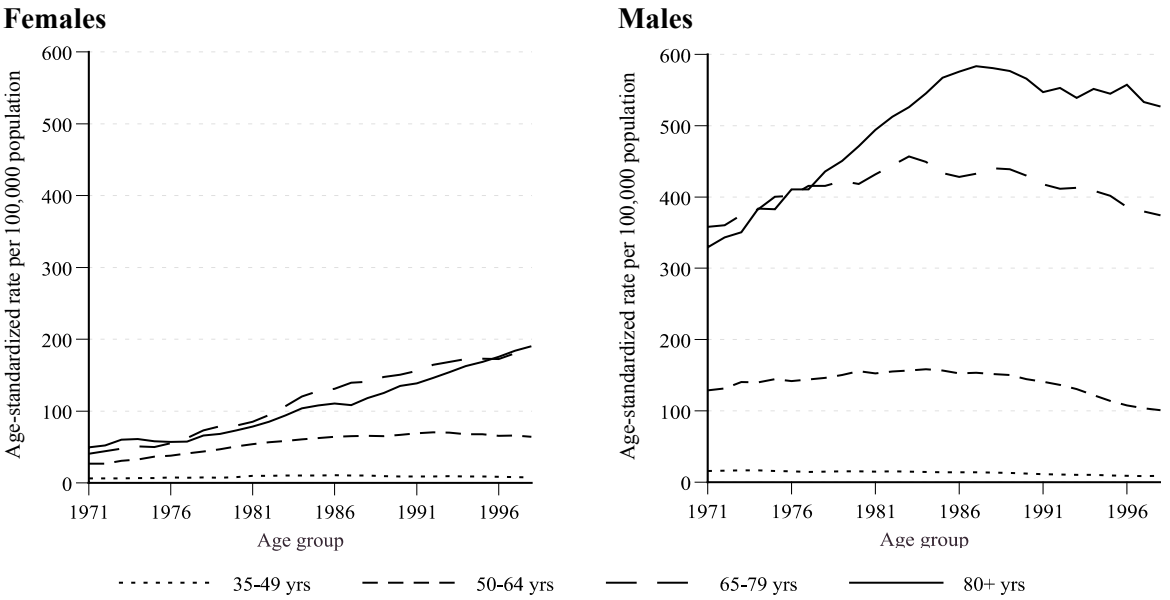
n.s. not significant

Trends in mortality for lung cancer, by age group

The downward trend in lung cancer mortality began in the mid-to-late 1980s in men of all ages in Ontario (Figure 11). The similar change points support the hypothesis that much of the drop is probably due to the period effect of smoking cessation beginning in the mid 1960s, rather than a cohort effect related to the initiation of smoking during the teens and early 20s.

In Ontario women, lung cancer mortality continues to rise, except in younger to middle aged women. These findings are more in keeping with a cohort effect, perhaps reflecting the difficulty many women smokers have in quitting for good.

Figure 11. Age-standardized mortality rates (3-yr moving average) for lung cancer in Ontario, by age group, 1971-1998



[1] Standardized to the 1991 Canadian population

Table 10. Annual percentage change (APC) for lung cancer mortality in Ontario, by age group, 1971-1998

Females	Age group	Period	APC (%)	**Males**	Age group	Period	APC (%)
	35-49	1971-1985	+4.3		35-49	1971-1986	-0.8
		1985-1998	-2.3			1986-1998	-4.4
	50-64	1971-1984	+7.1		50-64	1971-1988	+1.0
		1984-1998	n.s.			1988-1998	-4.7
	65-79	1971-1985	+9.3		65-79	1971-1983	+2.0
		1985-1998	+2.9			1983-1998	-1.2
	80+	1971-1998	+5.3		80+	1971-1986	+3.9
						1986-1998	-0.9

n.s. not significant

Trends in incidence for lung cancer, by age group

Temporal trends in lung cancer incidence (Figure 12) are very similar to lung cancer mortality. Again, similar change points in men of all ages argues in favour of a period effect relating to increased smoking cessation, beginning about 20 years before the changes in incidence. In women, the earlier change points seen in younger women argues for a predominant cohort effect.

Figure 12. Age-standardized incidence rates (3-yr moving average) for lung cancer, first primaries only, in Ontario, by age group, 1971-1998

[1] Standardized to the 1991 Canadian population

Table 11. Annual percentage change (APC) for lung cancer incidence in Ontario, by age group, 1971-1998

Females	Age group	Period	APC (%)	**Males**	Age group	Period	APC (%)
	35-49	1971-1984	+4.2		35-49	1971-1992	-1.7
		1984-1998	-1.6			1992-1998	-5.3
	50-64	1971-1983	+7.0		50-64	1971-1978	+2.4
		1983-1998	+1.2			1978-1990	n.s.
						1990-1998	-4.7
	65-79	1971-1988	+7.2		65-79	1971-1976	+3.3
		1988-1998	+2.0			1976-1990	n.s.
						1990-1998	-2.2
	80+	1971-1998	+4.6		80+	1971-1984	+3.2
						1984-1998	-1.2

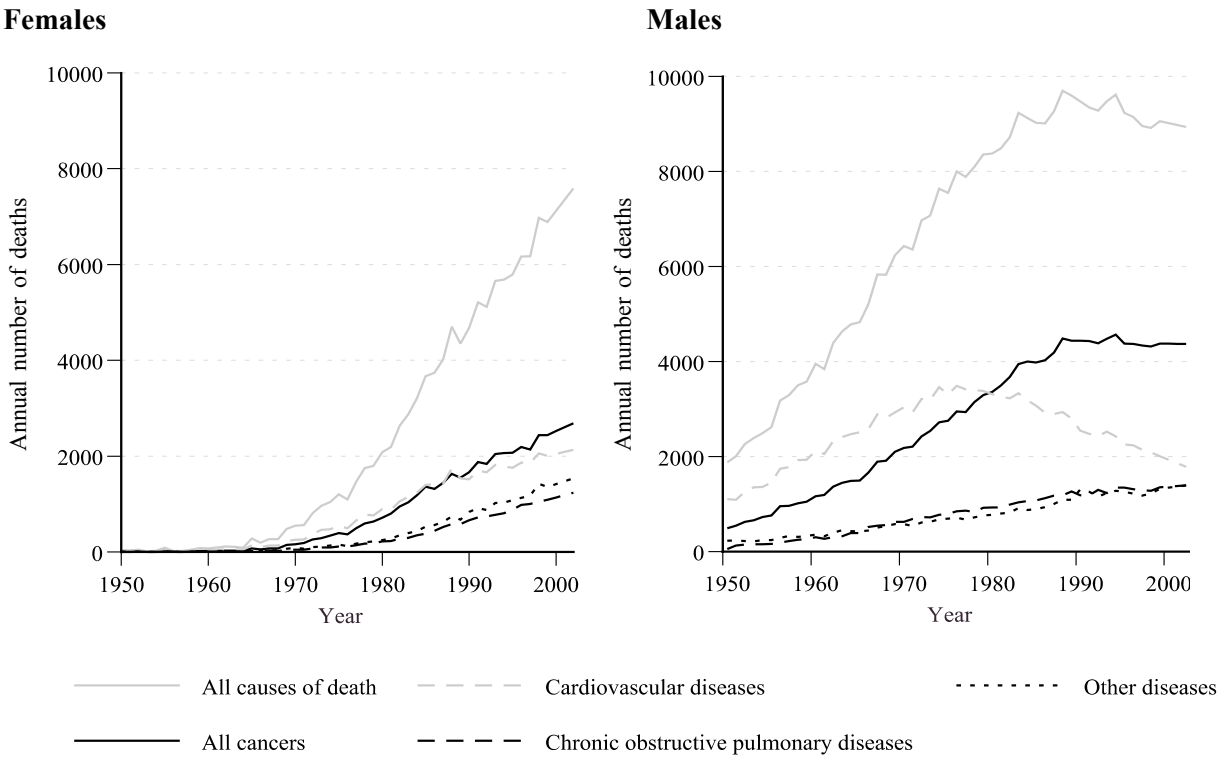
n.s. not significant

Annual burden of tobacco-attributed deaths, since 1950

Over the past 50 years, almost 500,000 deaths have occurred among Ontario women and men that can be directly attributed to tobacco (Figure 13). Compared to men, the epidemic of tobacco-attributed deaths in women has lagged by about 25-30 years. Since the late 1980s, there has been a slow but steady fall in the burden of TAM among Ontario men; unfortunately this trend is not seen among Ontario women.

In the current year, 2002, the annual number of TAM deaths among Ontario women is expected to exceed 7,500; among men, almost 9,000 deaths will be directly attributed to tobacco. These estimates comprise 18% and 21% of all deaths expected among Ontario women and men, respectively, in 2002.

Figure 13. Annual number of tobacco-attributed deaths in Ontario, by sex, 1950-1999



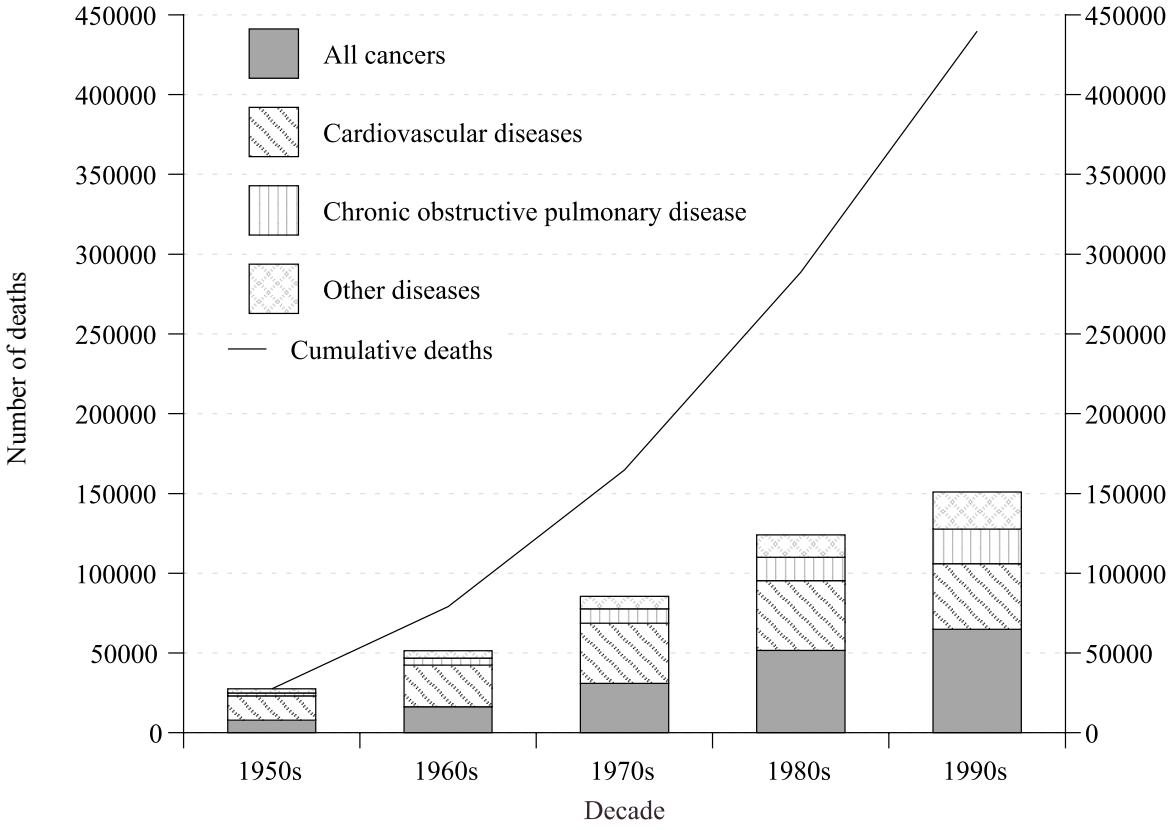
Burden of tobacco-attributed deaths, both sexes combined, 1950-1998

Since 1950, nearly 1/2 million Ontarians have died because of tobacco (Figure 14). This toll is approximately six times greater than the sum of all Ontario deaths attributed to alcohol, drugs, motor vehicle accidents and AIDS over the same period.

At the present time, of every 1,000 Ontarians who smoke, about half will die from smoking, if they continue; approximately one-quarter will die before the age of 65 years. In contrast, of every 1,000 Ontarians, only 9 are expected to die over a lifetime in traffic accidents; and only 1 will be murdered.

Currently, 50 Ontarians die each day because of tobacco. This is equivalent to 2 deaths per hour or 1 death every 30 minutes. To use an analogy, the death toll from tobacco in Ontario is equivalent to one fully loaded jumbo jet crashing every 6th day without any survivors.

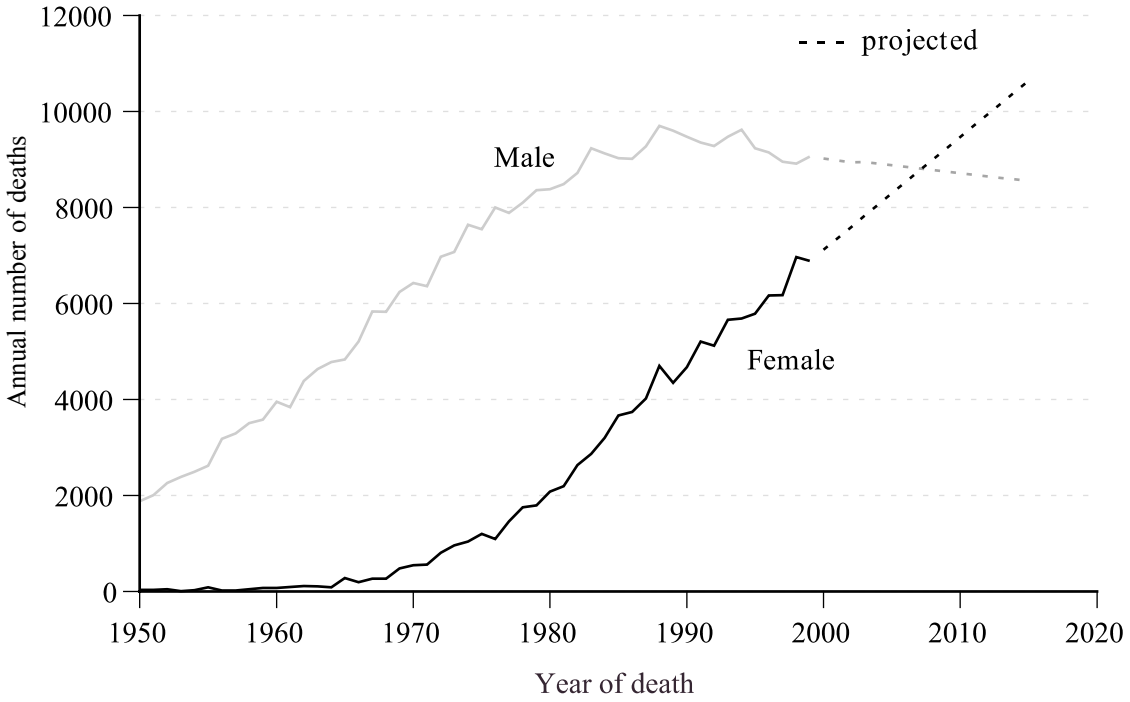
Figure 14. Number of tobacco-attributed deaths in Ontario, 1950-1999, both sexes combined



Tobacco-attributed deaths in Ontario: Equality between the sexes?

While the number of tobacco-attributed deaths (lung cancer, heart disease, stroke, etc.) in men peaked about 10 years ago, the number in women is still increasing (Figure 15). In 2002, it is estimated that more than 7,500 Ontario females will die from tobacco. That will be close to 20% of all deaths in women this year. By 2007, it is projected that female deaths from tobacco will equal the number of male deaths, about 9,000 per year.

Figure 15. Tobacco-attributed deaths in Ontario, 1950-1999, and projected for 2000-2015



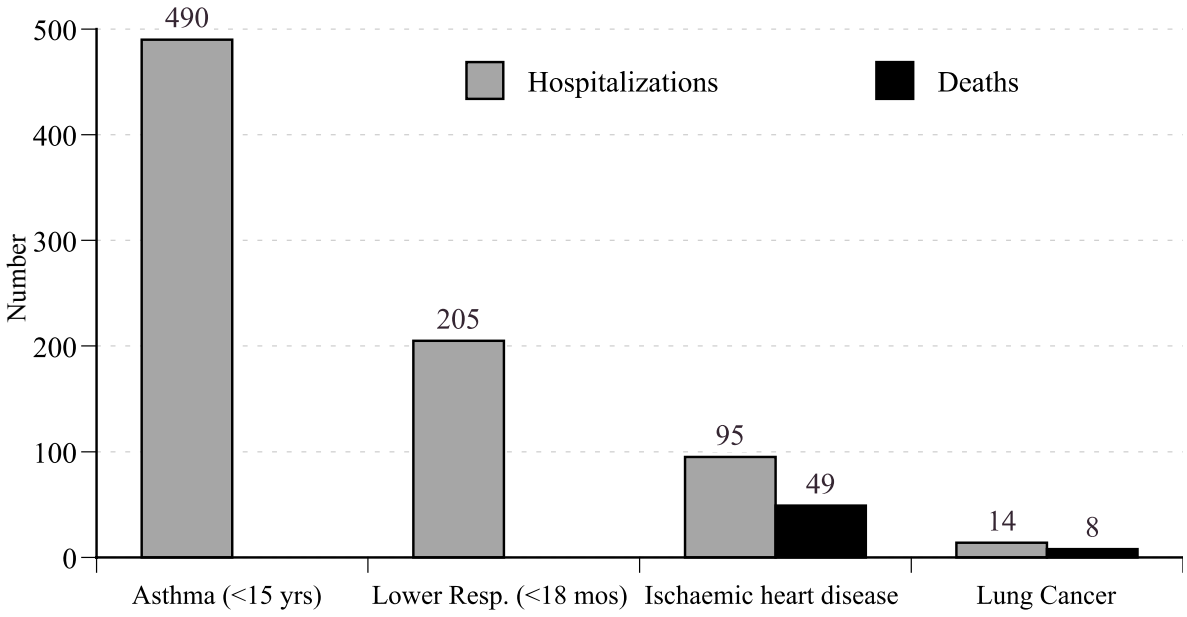
Environmental tobacco smoke in Ontario

Environmental tobacco smoke (ETS) or "second-hand smoke" is a complex mixture formed from the escaping smoke of a cigarette or other tobacco product, as well as the smoke exhaled by the smoker. ETS is an important source of exposure to toxic gases and particulates in indoor air. Despite an increasing number of restrictions on smoking in workplaces, public places and households, exposures continue to be a major public health concern in Ontario, especially for infants and children at home.

At least fifteen major disease groups or conditions are now known or suspected to be caused by ETS (OTRU 2001). The impact of four of these outcomes for which there is sufficient evidence of a causal relationship, is shown in Figure 16. Based on these estimates, it is estimated that ETS exposure caused at least 57 deaths and over 800 hospitalizations in Ontario in 1997.

It should be noted that the method employed to produce these estimates (Luk and Single 2001) focused only on the population attributable risk among people who were never smokers but had spouses or parents who were current smokers in the household. As such, these estimates can only be very conservative, given that ETS exposures in workplaces or public places have been ignored, as well as household exposures due to other than the spouse. Additionally, the hazards among long-time ex-smokers have not been estimated.

Figure 16. Hospitalizations and deaths attributed to environmental tobacco smoke in Ontario, 1997, both sexes combined



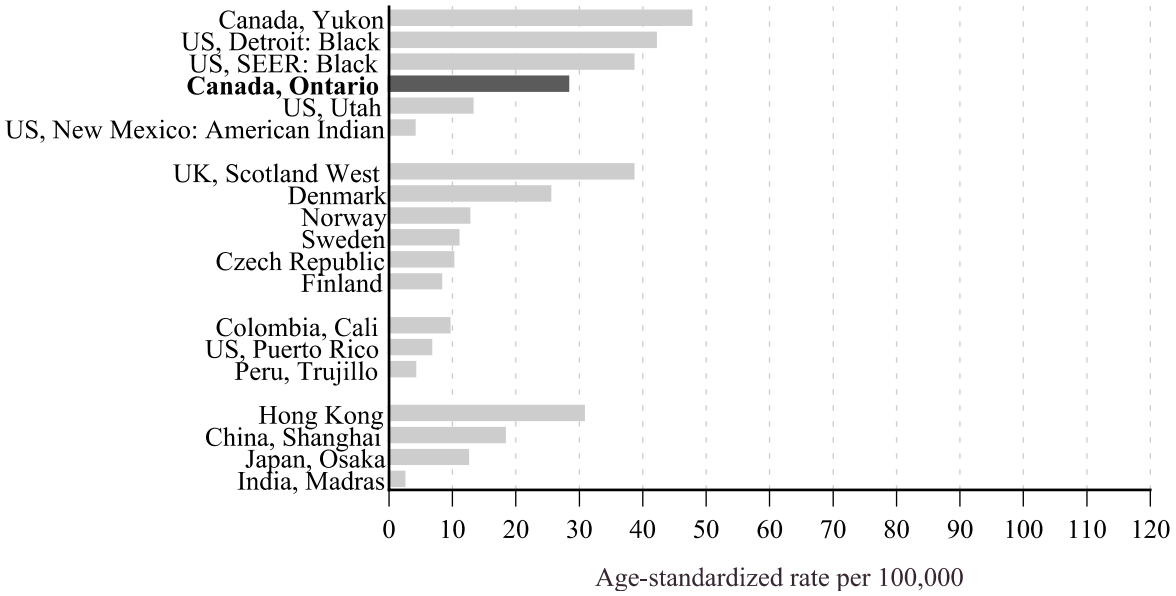
Geographic Patterns

International comparisons of lung cancer incidence

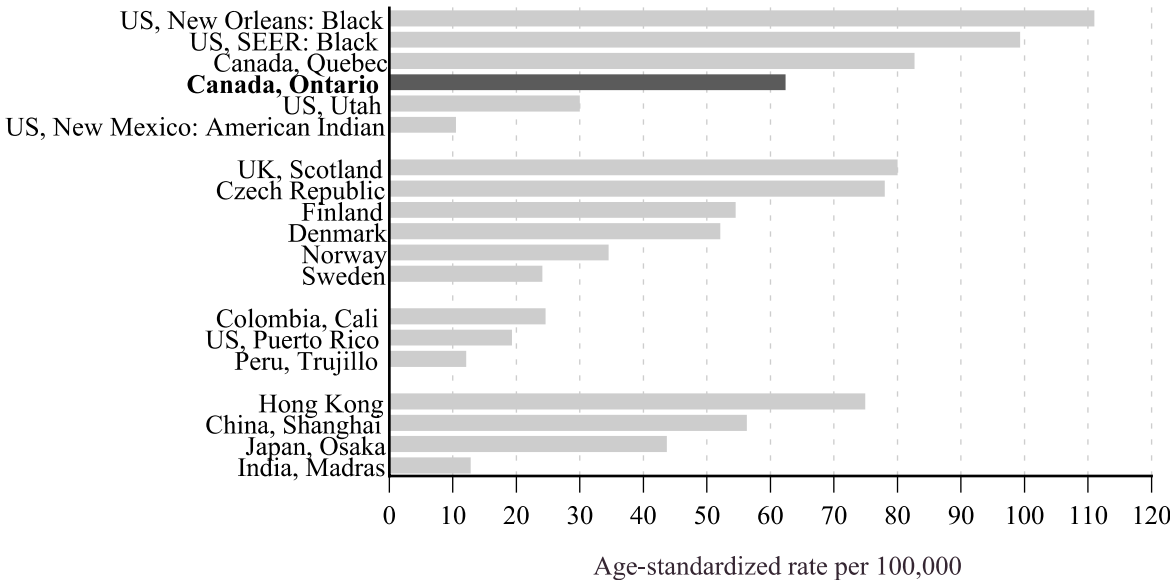
Lung cancer rates vary dramatically around the world. Figure 17 provides examples from a number of cancer registries with registration methods similar to Ontario. Lung cancer is generally much more common in developed countries and less common in developing countries. Lung cancer rates in Ontario (and Canada) have been among the highest in the world for many years now. These international differences are mostly due to differences in tobacco consumption.

Figure 17. Age-standardized[1] incidence rates for lung cancer, by country/region, by sex, 1988-1992

Females



Males



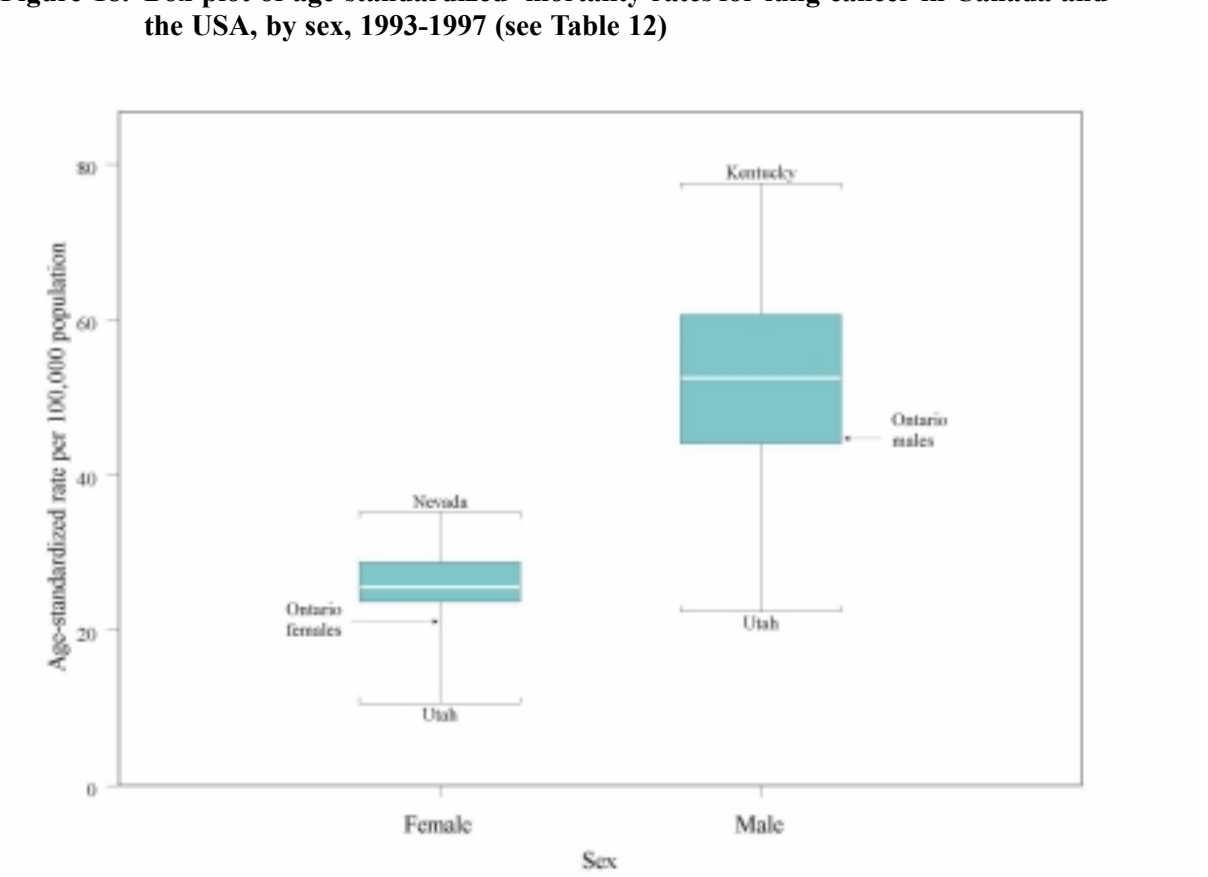
[1] Standardized to the World Standard population

North American comparisons of lung cancer mortality

There is considerable variation in lung cancer mortality across North America (Figure 18). Ontario's rates for the period 1993-1997, while similar to other Canadian rates, were somewhat lower than most American States. The low rates in Utah reflect the low prevalence of tobacco use, mostly because of the influence of the Mormon religion and societal pressure to limit smoking.

There is a noticeable east-west gradient in lung cancer mortality in North America (see Table 12), with higher rates in the eastern half of Canada and the USA and lower rates in the West Coast areas. Again, this gradient is largely attributable to differences in tobacco consumption 20-30 years ago, and not necessarily current patterns of smoking.

Figure 18. Box plot of age-standardized[1] mortality rates for lung cancer in Canada and the USA, by sex, 1993-1997 (see Table 12)



[1] Standardized to the World Standard population

Table 12. Lung cancer mortality in North America by province/state and sex, 1993-1997

	Females	Rate		Males	Rate
1.	Nevada	35.2	1.	Kentucky	77.5
2.	Kentucky	34.9	2.	Arkansas	73.0
3.	Delaware	33.2	3.	Tennessee	72.0
4.	West Virginia	33.0	4.	Mississippi	69.3
5.	Maine	31.9	5.	Louisiana	69.2
6.	New Hampshire	30.9	6.	Quebec	66.8
7.	Oregon	30.7	7.	West Virginia	66.0
8.	Rhode Island	30.0	8.	Alabama	65.1
9.	Prince Edward Island	29.6	9.	Georgia	65.0
10.	Missouri	29.6	10.	North Carolina	65.0
11.	Maryland	29.5	11.	Oklahoma	62.9
12.	Alaska	29.3	12.	South Carolina	62.7
13.	Indiana	29.0	13.	Delaware	62.3
14.	Washington	28.8	14.	Missouri	61.2
15.	Ohio	28.8	15.	Indiana	61.0
16.	Louisiana	28.7	16.	New Brunswick	60.2
17.	Massachusetts	28.5	17.	Nova Scotia	59.0
18.	Arkansas	28.4	18.	Maine	58.6
19.	Vermont	28.3	19.	Ohio	58.6
20.	Oklahoma	28.3	20.	Virginia	58.6
21.	Tennessee	28.3	21.	Rhode Island	56.9
22.	Florida	28.0	22.	Maryland	56.0
23.	Michigan	28.0	23.	Newfoundland	55.8
24.	Nova Scotia	27.3	24.	Texas	55.2
25.	New Jersey	27.0	25.	Illinois	54.6
26.	Illinois	27.0	26.	Michigan	53.9
27.	Virginia	27.0	27.	Prince Edward Island	53.8
28.	Connecticut	26.2	28.	Florida	53.7
29.	California	25.6	29.	Pennsylvania	53.4
30.	Texas	25.6	30.	Nevada	53.0
31.	Mississippi	25.6	31.	New Hampshire	51.9
32.	Manitoba	25.5	32.	Kansas	50.1
33.	Georgia	25.5	33.	Iowa	50.0
34.	Pennsylvania	25.4	34.	Vermont	49.2
35.	Quebec	25.3	35.	Massassachusetts	49.0
36.	Montana	25.1	36.	Alaska	48.9
37.	North Carolina	25.1	37.	New Jersey	48.8
38.	New York	24.9	38.	Oregon	48.0
39.	Alabama	24.8	39.	Nebraska	47.5
40.	British Columbia	24.5	40.	New York	46.4
41.	Wyoming	24.5	41.	Washington	46.0
42.	Kansas	24.5	42.	South Dakota	45.8
43.	South Carolina	24.4	43.	Manitoba	45.4
44.	Arizona	24.3	44.	Arizona	44.9
45.	Iowa	23.9	45.	**Ontario**	**44.6**
46.	New Brunswick	23.6	46.	Connecticut	43.5
47.	Minnesota	23.2	47.	Wisconsin	43.2
48.	Wisconsin	22.8	48.	Montana	41.8
49.	Alberta	22.0	49.	North Dakota	41.4
50.	**Ontario**	**21.9**	50.	California	41.3
51.	Nebraska	21.8	51.	Saskatchewan	40.9
52.	Saskatchewan	21.6	52.	Wyoming	40.8
53.	Idaho	21.4	53.	Minnesota	40.8
54.	North Dakota	20.4	54.	British Columbia	40.0
55.	New Mexico	20.0	55.	Alberta	38.7
56.	Colorado	19.6	56.	Idaho	38.5
57.	South Dakota	19.2	57.	Colorado	35.8
58.	Hawaii	16.9	58.	Hawaii	34.7
59.	Newfoundland	16.8	59.	Mew Mexico	34.4
60.	Utah	10.6	60.	Utah	22.4

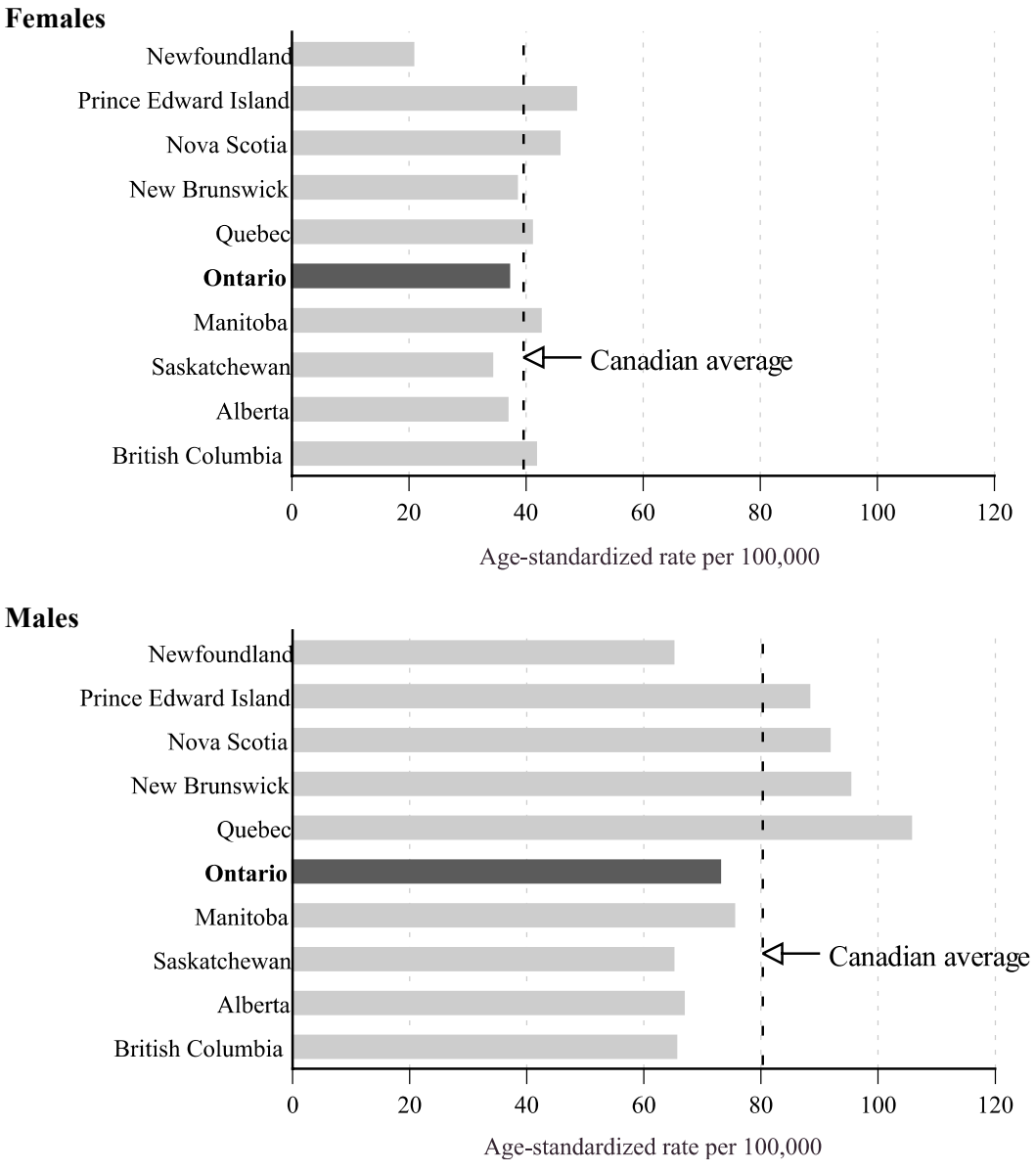
[1] Standardized to the World Standard Population (rate per 100,000 population)

Interprovincial comparisons of lung cancer incidence

There is some variation in lung cancer incidence across Canada (Figure 19), more so for males than females. Ontario's rates for the period 1993-1997 were similar to the Canadian rates, which is not surprising considering Ontario's population comprises about 40% of the national total. These comparisons were restricted to first primaries only in order to remove the effect of variation in the registration of second and later primaries.

There is an east-west gradient in incidence, particularly for males, with highest rates in the Atlantic Region and Quebec and lowest rates in Western Canada. This gradient is largely attributable to differences in tobacco consumption, particularly 20-30 years ago.

Figure 19. Age-standardized[1] incidence rates for lung cancer[2] in Canada, by sex, 1993-1997



[1] Standardized to the 1991 Canadian population

[2] These comparisons are limited to first primaries only

Ontario comparisons of lung cancer incidence

Among females, age-standardized incidence rates for 1994-1998 ranged from a low of 32.3 per 100,000 in the Central East Cancer Care Ontario Region (CCOR), to a high of 48.3 per 100,000 in the Northeastern CCOR, about a 50% difference (Table 13). Among males, age-standardized incidence rates ranged from a low of 63.7 per 100,000 in the Central East CCOR, to a high of 86.8 per 100,000 in the South CCOR, about a 36% difference. Mortality showed a similar pattern (Table 14).

Table 13. Age-standardized[1] incidence rates (per 100,000), rate ratios[2] (RR) and 95% confidence intervals (CI) for lung cancer (first primaries only), by CCOR, 1994-1998

	Females				Males			
CCOR	**#**	**Rate**	**RR**	**95% CI**	**#**	**Rate**	**RR**	**95% CI**
Eastern	1,419	45.6	1.23*	(1.16,1.29)	1,826	74.3	1.07*	(1.02,1.12)
Southeast	1,040	47.8	1.28*	(1.21,1.37)	1,452	79.5	1.14*	(1.08,1.20)
Central East	4,675	32.3	0.87*	(0.84,0.90)	7,355	63.7	0.91*	(0.89,0.94)
Central West	1,653	38.5	1.03	(0.98,1.09)	2,449	69.9	1.00	(0.96,1.04)
Southwest	1,593	34.7	0.93*	(0.89,0.98)	2,581	68.9	0.99	(0.95,1.03)
South	452	41.8	1.30*	(1.22,1.39)	686	86.8	1.25*	(1.18,1.32)
Northeast	863	48.3	1.16*	(1.03,1.31)	1,336	84.9	1.22*	(1.11,1.33)
Northwest	300	43.3	1.12*	(1.02,1.23)	503	80.1	1.15*	(1.06,1.24)
All Ontario[3]	**11,995**	**37.2**	**1.00**		**18,188**	**69.7**	**1.00**	

Table 14. Age-standardized[1] mortality rates (per 100,000), rate ratios[2] (RR) and 95% confidence intervals (CI) for lung cancer, by CCOR, 1994-1998

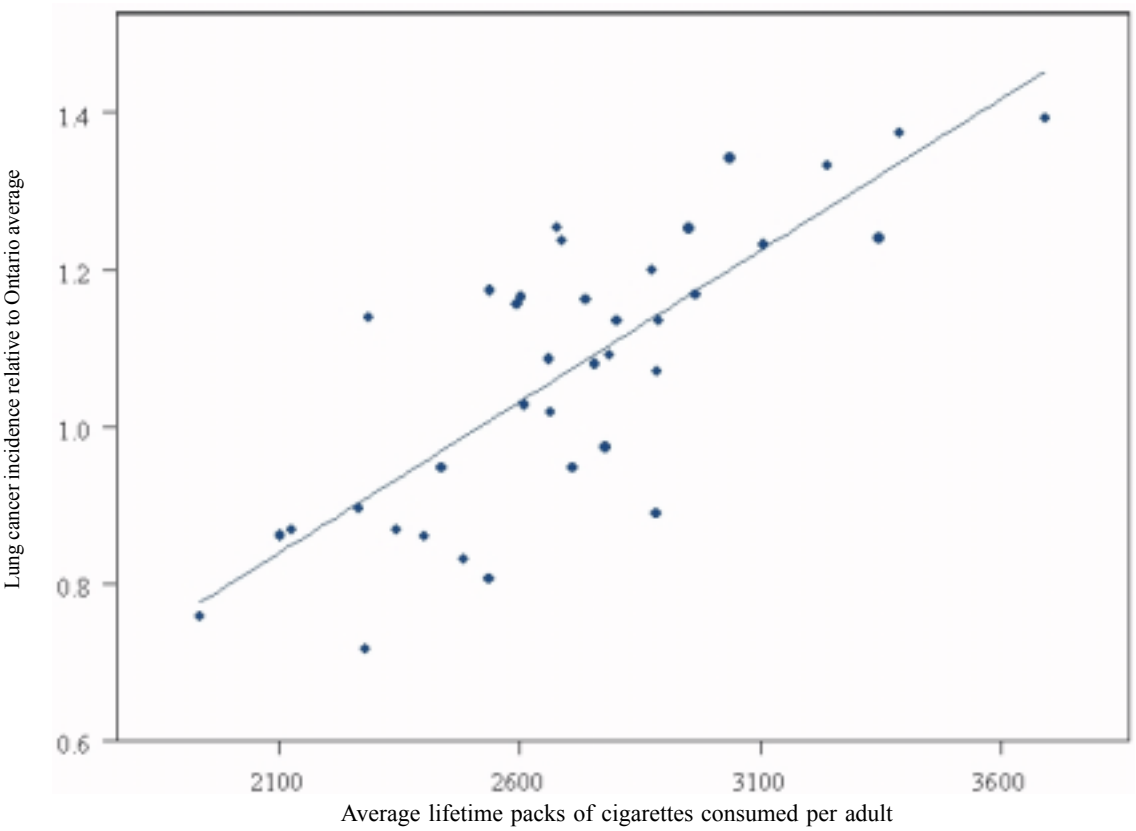
	Females				Males			
CCOR	**#**	**Rate**	**RR**	**95% CI**	**#**	**Rate**	**RR**	**95% CI**
Eastern	944	30.0	1.13*	(1.06,1.20)	1,366	56.3	1.04	(0.98,1.09)
Southeast	762	34.7	1.25*	(1.16,1.34)	1,139	62.6	1.09*	(1.03,1.15)
Central East	3,950	27.1	0.89*	(0.86,0.92)	6,538	57.9	0.90*	(0.88,0.92)
Central West	1,408	32.0	1.03	(0.98,1.09)	2,334	67.4	1.05*	(1.01,1.09)
Southwest	1,352	28.7	0.93*	(0.88,0.98)	2,394	64.5	1.00	(0.96,1.05)
South	404	36.8	1.24*	(1.12,1.37)	669	79.2	1.26*	(1.19,1.34)
Northeast	712	39.4	1.30*	(1.21,1.41)	1,225	81.0	1.16*	(1.05,1.28)
Northwest	251	35.9	1.15*	(1.01,1.31)	439	74.6	1.23*	(1.14,1.33)
All Ontario[4]	**9,783**	**29.9**	**1.00**		**16,104**	**66.1**	**1.00**	

[1] Standardized to the 1991 Canadian population
[2] Ratio of the incidence/mortality rate in a CCOR to that for all Ontario (known residence cases only)
[3] Excludes cases with unknown residence (n=2,433) and second or later primaries (2,063)
[4] Excludes deaths with unknown residence (n=895)
* Significantly different from 1.0 ($p<0.05$)

Correlation of smoking and lung cancer incidence

Figure 20 shows the strong correlation between prior smoking history and risk of lung cancer. Tables 15 and 16 present age-standardized incidence and mortality rates for Ontario's 37 Public Health Units (PHUs) for the time period 1994-1998. Both sexes have been combined in order to increase statistical precision. There is a high correlation between the sex-specific rates across the PHUs (r=0.87). Figure 21 describes the distribution of these rates in the form of a map. Figure 22 repeats this map, but adjusting for average lifetime cigarette consumption (pack-years) as reported in the Ontario Health Survey of 1990. The latter figure demonstrates that the bulk of the inter-PHU variation in rates is explained by the areal adjustment for past cigarette consumption (r=0.77).

Figure 20. Tobacco consumption and lung cancer incidence by Public Health Unit, 1994-1998



Sources: The Ontario Cancer Registry 2001
OHS 1990

Table 15. Age-standardized[1] incidence rates (per 100,000), rate ratios[2] (RR) and 95% confidence intervals (CI) for lung cancer (first primary only), both sexes combined, by Public Health Unit, 1994-1998

	Incidence			
Public Health Unit	**#**	**Rate**	**RR**	**95% CI**
Algoma	502	65.1	1.27*	(1.16,1.39)
Brant	374	54.2	1.06	(0.95,1.17)
Bruce-Grey-Owen Sound	514	49.7	0.97	(0.89,1.06)
Durham	1,092	55.2	1.08*	(1.02,1.14)
Eastern Ontario	733	70.1	1.37*	(1.27,1.47)
Elgin-St. Thomas	258	56.0	1.09	(0.96,1.24)
Haldimand-Norfolk	302	49.3	0.96	(0.86,1.08)
Haliburton-Kawartha-Pine Ridge	708	59.3	1.16*	(1.07,1.25)
Halton	789	45.1	0.88*	(0.82,0.95)
Hamilton-Wentworth	1,559	55.5	1.08*	(1.03,1.14)
Hastings-Prince Edward	625	65.5	1.28*	(1.18,1.38)
Huron	185	45.0	0.88	(0.76,1.03)
Kent-Chatham	390	59.6	1.16*	(1.05,1.29)
Kingston-Frontenac-Lennox-Addington	620	59.9	1.17*	(1.08,1.27)
Lambton	465	57.9	1.13*	(1.03,1.24)
Leeds-Grenville-Lanark	621	61.5	1.20*	(1.11,1.30)
Middlesex-London	1,040	49.6	0.97	(0.91,1.03)
Muskoka-Parry Sound	316	47.0	0.92	(0.82,1.07)
Niagara	1,394	52.1	1.02	(0.96,1.01)
North Bay and District	335	68.0	1.33*	(1.19,1.48)
Northwestern	273	63.8	1.25*	(1.10,1.40)
Ottawa-Carleton	1,905	52.4	1.02	(0.98,1.07)
Oxford	263	45.5	0.89	(0.78,1.01)
Peel	1,356	41.8	0.82*	(0.77,0.86)
Perth	158	36.5	0.71*	(0.61,0.84)
Peterborough	524	60.3	1.18*	(1.08,1.29)
Porcupine	326	71.2	1.39*	(1.24,1.55)
Renfrew	378	63.6	1.24*	(1.12,1.38)
Simcoe	1,118	60.4	1.18*	(1.11,1.25)
Sudbury and District	711	64.5	1.26*	(1.17,1.36)
Thunder Bay	530	60.7	1.19*	(1.09,1.29)
Timiskaming	168	70.5	1.38*	(1.18,1.61)
Toronto	6,148	44.4	0.88*	(0.85,0.89)
Waterloo	901	45.5	0.89*	(0.83,0.95)
Wellington-Dufferin-Guelph	473	43.3	0.85*	(0.77,0.93)
Windsor-Essex	1,138	57.8	1.13*	(1.06,1.20)
York	991	40.4	0.79*	(0.74,0.84)
All Ontario[3]	**30,183**	**51.2**	**1.00**	

[1] Standardized to the 1991 Canadian population

[2] Ratio of the incidence rate in a Public Health Unit to that for all Ontario (known residence cases only)

[3] Excludes cases with unknown residence

* Significantly different from 1.0 ($p<0.05$)

Table 16. Age-standardized[1] mortality rates (per 100,000), rate ratios[2] (RR) and 95% confidence intervals (CI) for lung cancer, both sexes combined, by Public Health Unit, 1994-1998

	Mortality			
Public Health Unit	**#**	**Rate**	**RR**	**95% CI**
Algoma	422	54.9	1.22*	(1.11,1.35)
Brant	355	50.2	1.12*	(1.00,1.24)
Bruce-Grey-Owen Sound	448	42.4	0.94	(0.86,1.04)
Durham	941	48.2	1.07*	(1.01,1.14)
Eastern Ontario	611	58.0	1.29*	(1.19,1.40)
Elgin-St. Thomas	238	50.9	1.13	(0.99,1.29)
Haldimand-Norfolk	281	45.4	1.01	(0.90,1.14)
Haliburton-Kawartha-Pine Ridge	573	47.5	1.06	(0.97,1.15)
Halton	739	42.9	0.96	(0.89,1.03)
Hamilton-Wentworth	1,407	49.7	1.11*	(1.05,1.17)
Hastings-Prince Edward	536	55.3	1.23*	(1.13,1.34)
Huron	187	43.9	0.98	(0.84,1.14)
Kent-Chatham	333	50.0	1.11	(1.00,1.24)
Kingston-Frontenac-Lennox-Addington	548	52.9	1.18*	(1.08,1.28)
Lambton	380	47.0	1.05	(0.94,1.16)
Leeds-Grenville-Lanark	547	53.5	1.19*	(1.09,1.30)
Middlesex-London	977	46.3	1.03	(0.97,1.10)
Muskoka-Parry Sound	293	43.2	0.96	(0.86,1.09)
Niagara	1,271	47.0	1.05	(0.99,1.11)
North Bay and District	308	62.5	1.39*	(1.24,1.56)
Northwestern	226	52.9	1.18*	(1.03,1.34)
Ottawa-Carleton	1,619	44.6	0.99	(0.94,1.04)
Oxford	245	41.9	0.93	(0.82,1.06)
Peel	1,148	36.7	0.82*	(0.77,0.87)
Perth	148	33.3	0.74*	(0.63,0.88)
Peterborough	447	50.2	1.12*	(1.02,1.23)
Porcupine	283	62.5	1.39*	(1.23,1.57)
Renfrew	286	47.6	1.06	(0.94,1.19)
Simcoe	959	51.4	1.14*	(1.07,1.22)
Sudbury and District	632	57.6	1.28*	(1.18,1.39)
Thunder Bay	464	52.8	1.18*	(1.07,1.29)
Timiskaming	159	65.8	1.47*	(1.25,1.73)
Toronto	5,409	38.8	0.86*	(0.84,0.89)
Waterloo	790	40.0	0.89*	(0.83,0.96)
Wellington-Dufferin-Guelph	428	39.1	0.87*	(0.79,0.96)
Windsor-Essex	1,073	54.2	1.21*	(1.14,1.28)
York	814	34.2	0.76*	(0.71,0.82)
All Ontario[3]	**26,525**	**44.9**	**1.00**	

[1] Standardized to the 1991 Canadian population

[2] Ratio of the mortality rate in a Public Health Unit to that for all Ontario (known residence cases only)

[3] Excludes deaths with unknown residence

* Significantly different from 1.0 ($p<0.05$)

Figure 21. Age-standardized incidence rates for lung cancer, by Public Health Unit, 1994-1998, All ages, both sexes combined

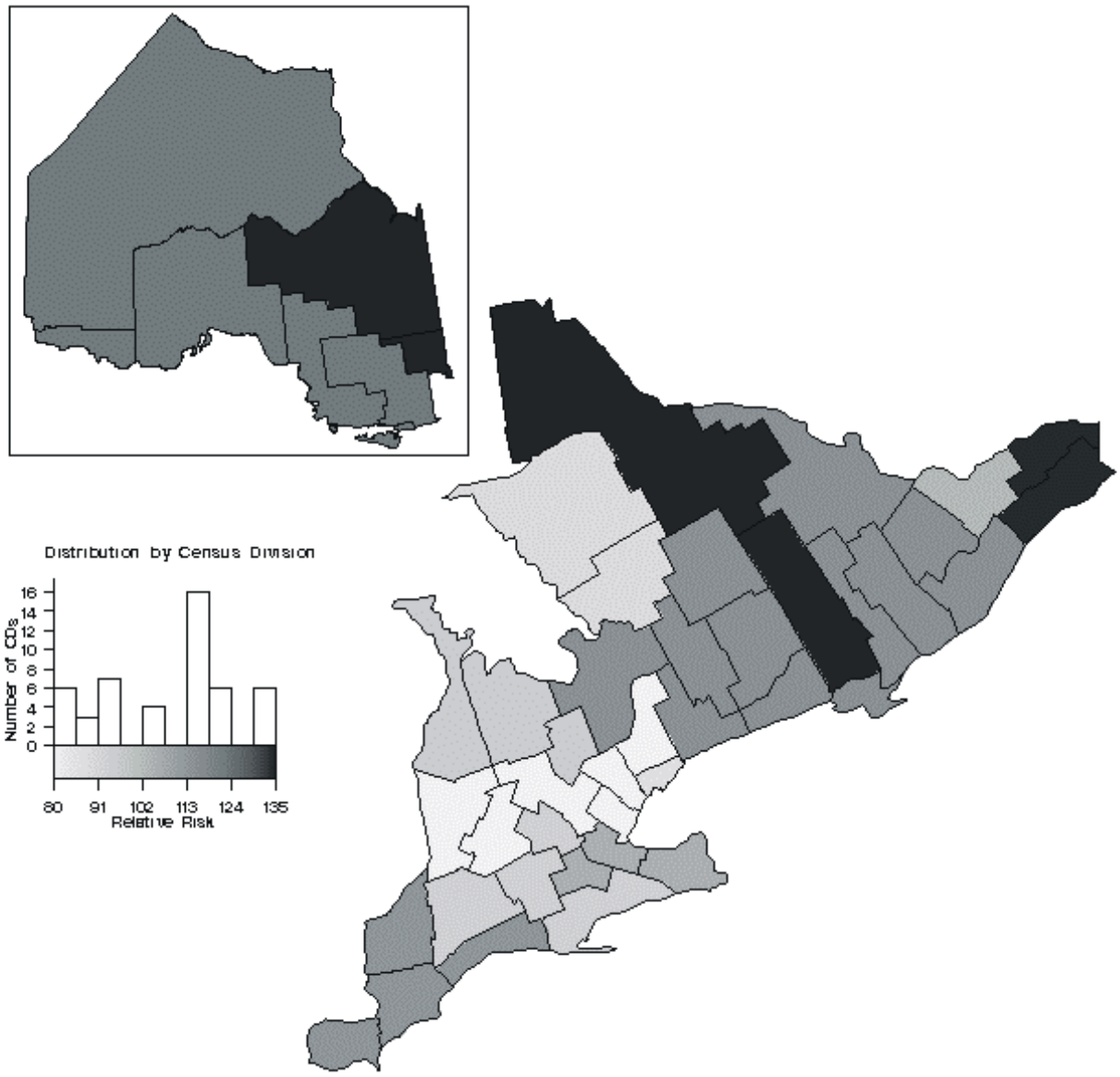
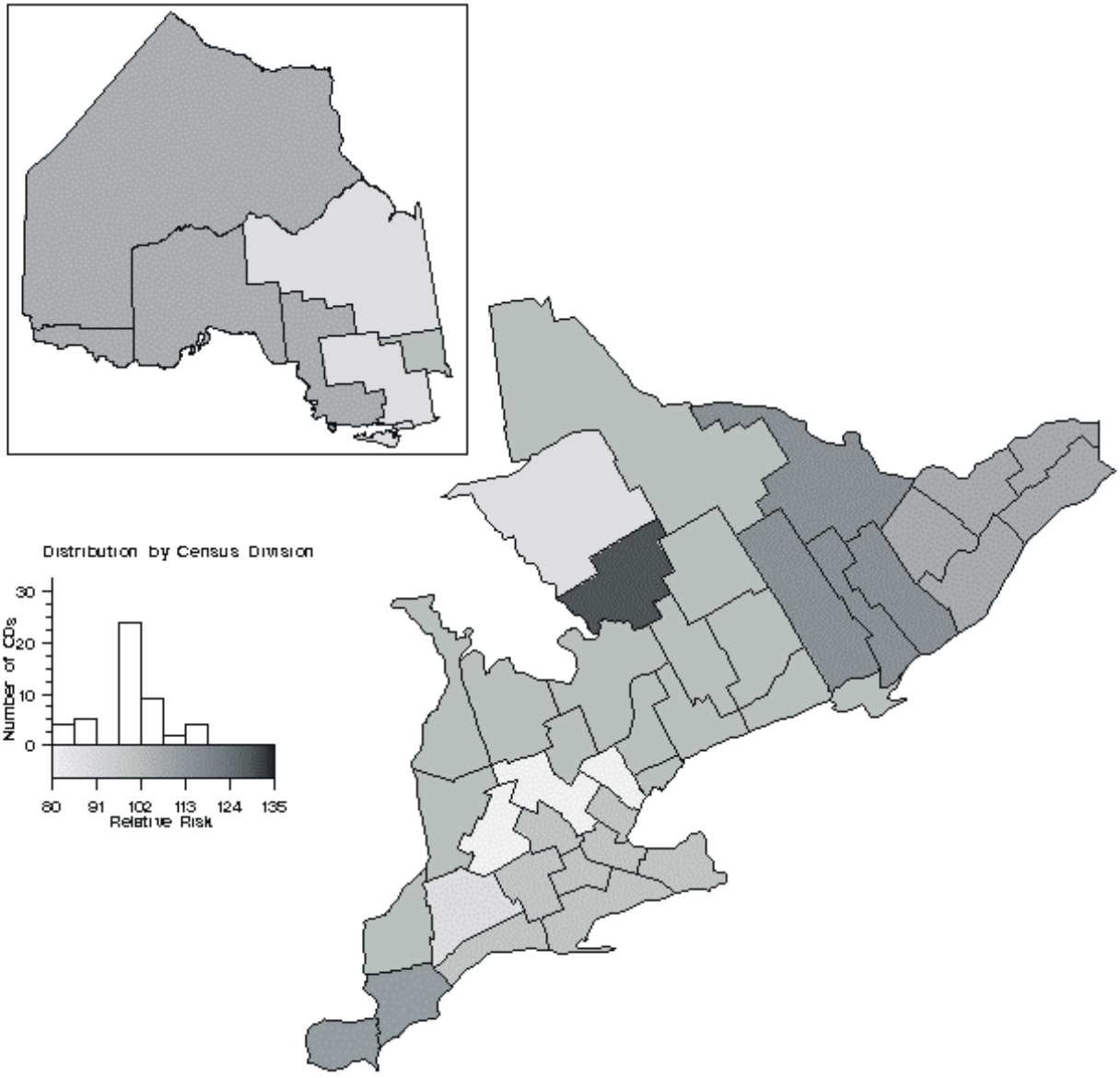


Figure 22. Age-standardized incidence rates for lung cancer, by Public Health Unit, 1994-1998, All ages, both sexes combined, smoking adjusted



Morphology

Morphologic patterns of lung cancer, by age group, in females

The vast majority of lung cancers are neoplasms of the epithelial lining of the respiratory tract. In Ontario females, adenocarcinoma is the predominant morphology in all age groups, although this dominance decreases with age (Table 17). The difference in morphologic patterns between the sexes has also been reported by other cancer registries and may be explained by differences in tobacco products used, as well as some uncertainty in distinguishing primary from secondary adenocarcinomas of the lung, particularly in females. The relatively high proportion of unspecified morphology cases (22.3%) reflects both an inability to do confirmatory biopsies in many cases, as well as some incompleteness in reporting pathologic confirmation to the OCR.

Table 17. Percentage distribution of morphologic groups for lung cancer in Ontario females, by age group, 1994-1998

	All ages[1]		20-34		35-49		50-64		65-79		80+	
	#	%	#	%	#	%	#	%	#	%	#	%
Squamous cell carcinoma	2,042	15.6	5	9.6	103	11.1	559	14.6	1,205	18.7	170	9.5
Adenocarcinoma	4,337	33.2	23	44.2	480	51.8	1,626	42.3	1,921	29.8	287	16.0
Small cell carcinoma	1,754	13.4	1	1.9	89	9.6	620	16.1	925	14.3	119	6.6
Large cell carcinoma	1,027	7.9	2	3.8	99	10.7	348	9.1	493	7.6	85	4.7
Other specified carcinoma	209	1.6	13	25.0	36	3.9	67	1.7	83	1.3	6	0.3
Unspecified carcinoma	1,126	8.6	5	9.6	91	9.8	322	8.4	575	8.9	133	7.4
Sarcomas and other specified morphology	32	0.2	1	1.9	7	0.8	8	0.2	13	0.2	1	0.1
Unspecified morphology	2,919	22.3	3	5.8	67	7.2	421	11.0	1,414	21.9	1,014	56.6
Total	**13,067**	**100.0**	**52**	**100.0**	**927**	**100.0**	**3,840**	**100.0**	**6,449**	**100.0**	**1,793**	**100.0**

[1] Totals across age groups may be less than the total for all ages because there are some cases under age 20 or of unknown age

Morphologic patterns of lung cancer, by age group, in males

Squamous cell carcinoma is the commonest morphology in Ontario males, although up to 64 years of age, adenocarcinomas are commonest (Table 18). The higher relative frequency of sarcomas among young men (20-34 years of age) is due to Kaposi's sarcoma, likely secondary to acquired immune deficiency syndrome.

Table 18. Percentage distribution of morphologic groups for lung cancer in Ontario males, by age group, 1994-1998

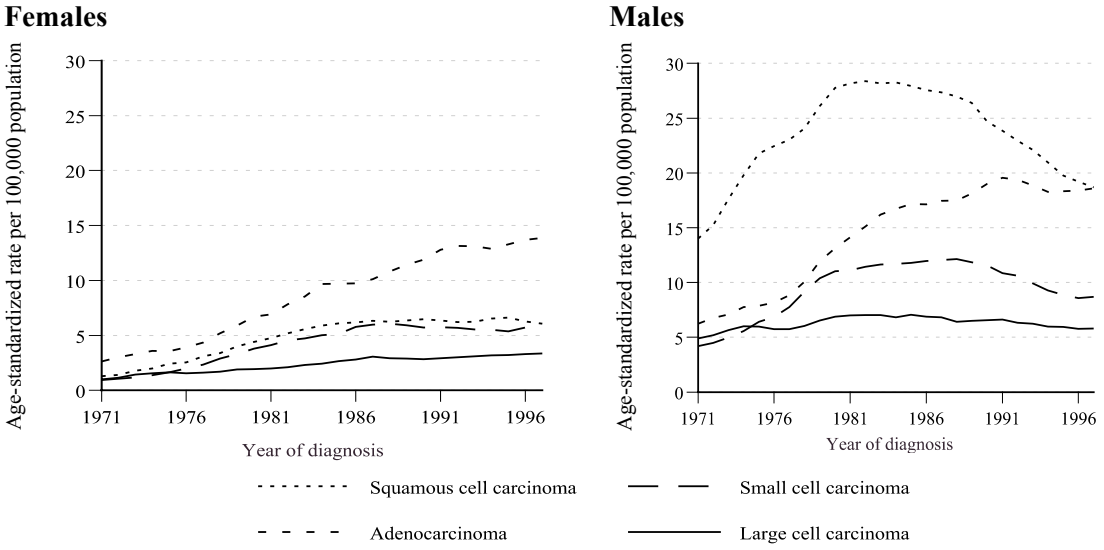
	All ages[1]		20-34		35-49		50-64		65-79		80+	
	#	%	#	%	#	%	#	%	#	%	#	%
Squamous cell carcinoma	4,990	25.5	3	2.9	132	14.2	1,400	25.6	2,967	28.1	488	19.6
Adenocarcinoma	4,934	25.2	19	18.6	363	39.0	1,691	31.0	2,549	24.1	312	12.5
Small cell carcinoma	2,281	11.7	0	0.0	134	14.4	792	14.5	1,193	11.3	162	6.5
Large cell carcinoma	1,508	7.7	11	10.8	89	9.6	447	8.2	811	7.7	150	6.0
Other specified carcinoma	177	0.9	16	15.7	23	2.5	40	0.7	90	0.9	7	0.3
Unspecified	1,743	8.9	11	10.8	77	8.3	506	9.3	942	8.9	207	8.3
Sarcomas and other specified morphology	38	0.2	1	1.0	4	0.4	9	0.2	21	0.2	3	0.1
Unspecified morphology	4,389	22.5	34	33.3	130	14.0	752	13.8	2,268	21.5	1,205	48.4
Total	**19,549**	**100.0**	**102**	**100.0**	**931**	**100.0**	**5,459**	**100.0**	**10,565**	**100.0**	**2,491**	**100.0**

[1] Totals across age groups may be less than the total for all ages because there are some cases under age 20 or of unknown age

Trends in incidence, by morphologic group

These graphs show clearly that among women, the rate of adenocarcinoma of the lung continues to rise, in the face of stabilization in the rates of squamous cell carcinoma and small cell carcinoma (Figure 23). In men, the rate of adenocarcinoma of the lung has now reached equivalence with the rate of squamous cell carcinoma. The rate of squamous cell carcinoma has declined steadily since the early 1980s. While there is no evidence of a decline in adenocarcinoma rates, there is some evidence of a stabilization in the rate over the 1990s.

Figure 23. Age-standardized incidence rates (3-year moving average) for lung cancer in Ontario, by morphologic group, 1971-1998



[1] Standardized to the 1991 Canadian population

Table 19. Annual percentage change (APC) for lung cancer incidence in Ontario, by morphologic group, 1971-1998

Females	Period	APC (%)	**Males**	Period	APC (%)
Squamous cell carcinoma	1971-1983	+13.2	Squamous cell carcinoma	1971-1974	+16.5
	1983-1998	n.s.		1974-1981	+5.0
				1981-1988	n.s.
				1988-1998	-4.3
Adenocarcinoma	1971-1984	+10.9	Adenocarcinoma	1971-1983	+9.0
	1984-1998	+3.2		1983-1991	+2.1
				1991-1998	n.s.
Small cell carcinoma	1971-1979	+20.1	Small cell carcinoma	1971-1979	+13.8
	1979-1987	+7.1		1979-1988	+1.8
	1987-1998	n.s.		1988-1998	-4.5
Large cell carcinoma	1971-1986	+6.0	Large cell carcinoma	1971-1982	+3.3
	1986-1998	+1.6		1982-1998	-1.5

n.s. not significant

Survival

Relative survival of tobacco-related cancers

Table 20. Number of cases, one, three, five and ten-year relative survival (%) for lung cancer (ICD9 162) in Ontario, 1979-1998, by sex and time period

	Time period	Number of cases	1 yr	3 yr	5 yr	10 yr
Females	1979-1988	13,071	39.6	20.7	16.9	13.4
	1989-1998	21,520	42.3	22.6	18.3	14.6
Males	1979-1988	32,079	35.3	16.6	13.3	10.7
	1989-1998	36,448	37.3	18.6	15.1	12.4

Table 21. Number of cases, one, three, five and ten-year relative survival (%) for lip and oropharyngeal cancers (ICD9 140-141, 143-146, 148-149) in Ontario, 1979-1998, by sex and time period

	Time period	Number of cases	1 yr	3 yr	5 yr	10 yr
Females	1979-1988	2,286	79.8	65.3	59.8	50.7
	1989-1998	2,624	82.5	68.7	64.4	53.6
Males	1979-1988	6,114	80.7	62.9	56.7	47.8
	1989-1998	6,219	81.4	63.9	57.6	48.8

Table 22. Number of cases, one, three, five and ten-year relative survival (%) for oesophageal cancer (ICD9 150) in Ontario, 1979-1998, by sex and time period

	Time period	Number of cases	1 yr	3 yr	5 yr	10 yr
Females	1979-1988	959	39.1	18.2	15.6	12.6
	1989-1998	1,158	42.6	20.4	17.4	15.1
Males	1979-1988	2,075	34.2	14.9	11.9	10.0
	1989-1998	2,722	37.5	15.4	12.0	9.3

Table 23. Number of cases, one, three, five and ten-year relative survival (%) for laryngeal cancer (ICD9 161) in Ontario, 1979-1998, by sex and time period

	Time period	Number of cases	1 yr	3 yr	5 yr	10 yr
Females	1979-1988	528	88.5	75.7	67.2	58.4
	1989-1998	617	85.0	72.0	66.0	54.8
Males	1979-1988	2,966	87.6	73.1	67.1	57.7
	1989-1998	3,098	87.9	73.3	67.5	60.0

Table 24. Number of cases, one, three, five and ten-year relative survival (%) bladder cancer (ICD9 188) in Ontario, 1979-1998, by sex and time period

	Time period	Number of cases	1 yr	3 yr	5 yr	10 yr
Females	1979-1988	3,810	84.1	75.9	74.0	72.9
	1989-1998	3,557	82.5	73.3	70.8	69.6
Males	1979-1988	11,223	90.3	81.8	78.4	73.6
	1989-1998	10,435	89.3	80.7	76.9	73.9

Table 25. Number of cases, one, three, five and ten-year relative survival (%) kidney cancer (ICD9 189) in Ontario, 1979-1998, by sex and time period

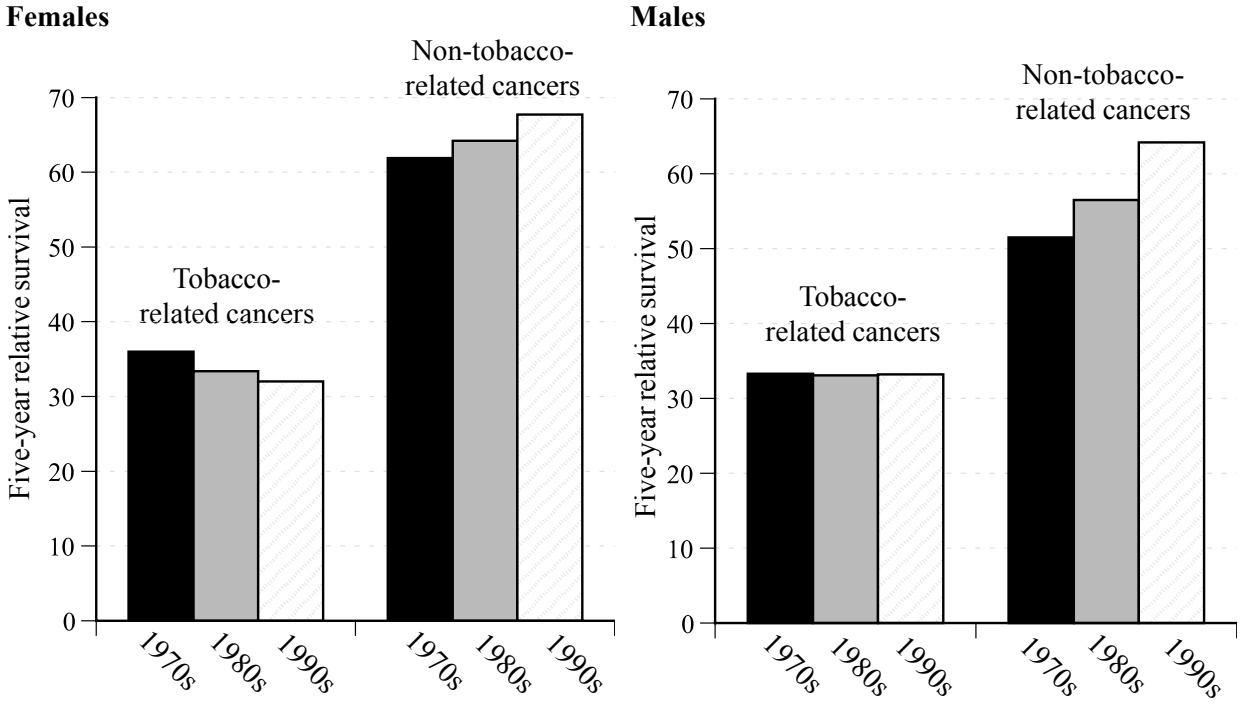
	Time period	Number of cases	1 yr	3 yr	5 yr	10 yr
Females	1979-1988	2,690	72.9	61.3	57.3	53.1
	1989-1998	4,055	79.8	70.0	67.1	66.1
Males	1979-1988	4,582	72.6	60.9	56.8	50.8
	1989-1998	7,022	79.3	70.7	67.5	63.0

Relative survival of tobacco related cancers (continued)

Relative survival after diagnosis of the tobacco-related cancers has not improved significantly over time (Figure 24). If all of the tobacco-related cancers (see Table 1) occurring in both sexes are combined, overall five-year relative survival is quite poor, at 33%. In contrast, the relative survival for non-tobacco related cancers is appreciably higher, 67% for cases diagnosed in the early 1990s, and there has been a steady improvement in survival of these cases over the past three decades.

Research on CT scanning of lung cancer is underway as a potential screening tool; however, the dismal survival rates at this stage do not offer much optimism for an effective screening tool.

Figure 24. Five-year relative survival for tobacco-related cancers and non-tobacco related cancers in Ontario, by decade



Relative survival of lung cancer, by year of diagnosis

Relative survival after diagnosis of lung cancer has only improved minimally over time (Figure 25). In spite of some advances in treatment, such as the introduction of chemotherapy for non-small cell lung cancer in the early to mid-1980s, there appears to have been little population-wide effect on lung cancer survival. No effective screening is yet available, whether for lung cancer or any other of the smoking-related cancers. Indeed, women may have a slightly higher disposition to smoking-related cancers than men.

Figure 25. Relative survival for lung cancer in Ontario, by year of diagnosis, 1979-1998

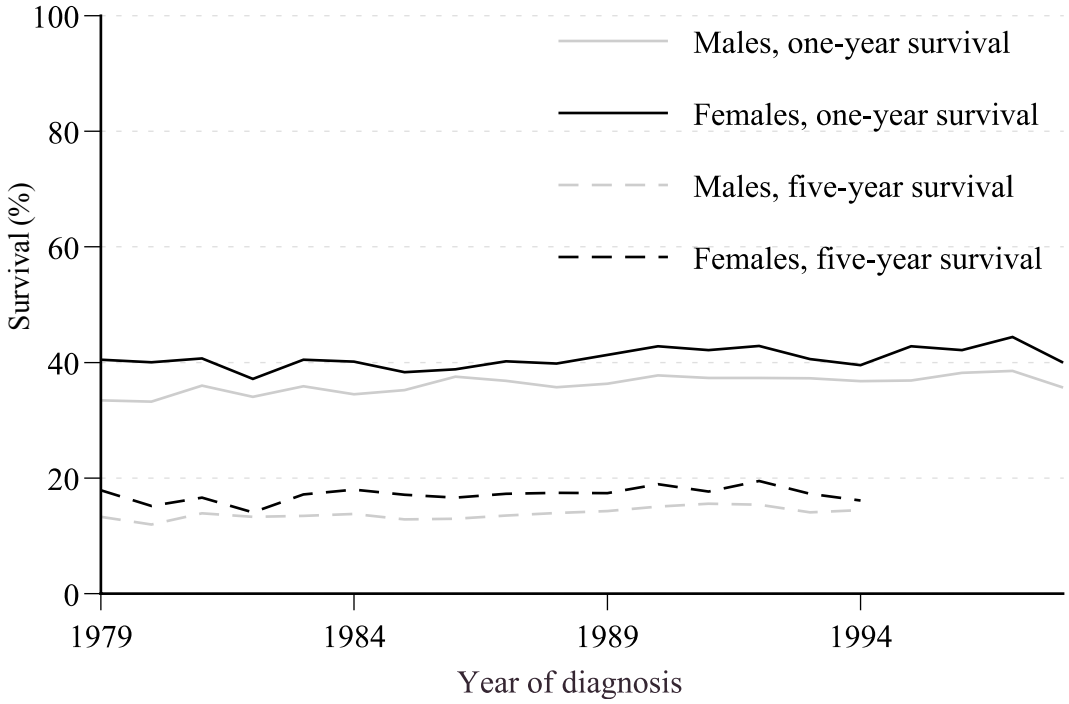


Table 26. Annual percentage change (APC) for lung cancer survival in Ontario, by year of diagnosis, 1979-1998

Females	Period	APC (%)	**Males**	Period	APC (%)
1 yr survival	1979-1998	+0.5	1 yr survival	1979-1998	+0.6
5 yr survival	1979-1998	n.s.	5 yr survival	1979-1998	+1.1

n.s. not significant

Relative survival for lung cancer, by age group

Relative survival shows some variation by age at diagnosis (Figure 26). The highest relative survival is for young men and women diagnosed under age 30, with a five-year relative survival of 61.5% and 50.1% in women and men respectively. However, lung cancer is quite rare in this age group, and it is likely that only a negligible fraction of these can be attributed to tobacco. Women have slightly higher survival rates than men, but this may be partly an artifact of over-registration of lung cancer among women. Notably, the high quality SEER registries fail to demonstrate any survival advantage for women diagnosed with lung cancer (NCI 2001).

Figure 26. Relative survival for lung cancer in Ontario, by age group, 1989-1998

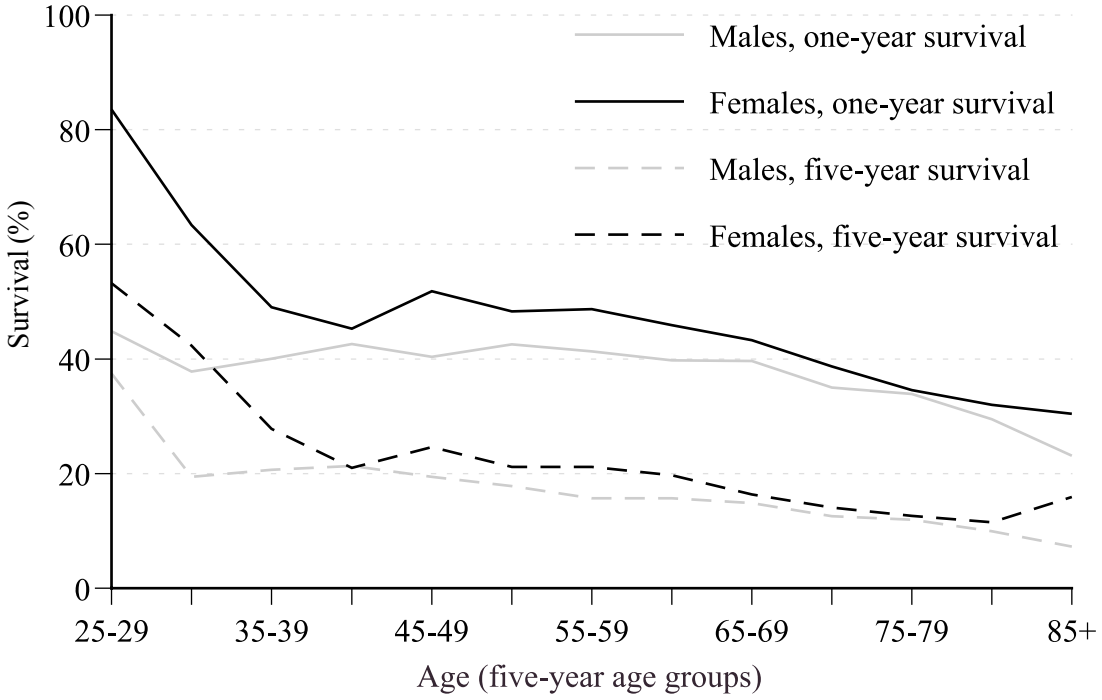


Table 27. Percentage change (PC) with age for lung cancer survival in Ontario, 1989-1998

Females	Age group	PC (%)	**Males**	Age group	PC (%)
1 yr survival	25-29 - 30-34	n.s.	1 yr survival	25-29 - 60-64	n.s.
	30-34 - 50-54	n.s.		60-64 - 85+	-9.8
	50-54 - 85+	-8.1			
5 yr survival	25-29 - 85+	-9.6	5 yr survival	25-29 - 85+	-8.1

n.s. not significant

Relative survival for lung cancer, by morphologic group

While women and men have the same survival experience following diagnosis of squamous cell lung cancer, women have a relative survival advantage following diagnosis of adenocarcinoma, small cell carcinoma and large cell carcinoma (Table 28). The relative dominance of these latter morphologic types among women probably explain this small overall survival advantage for women with lung cancer. It should be noted that these estimates have been age-adjusted, employing a methodology derived by Coleman *et al.* (1999) and Ellison *et al.* (2001).

Table 28. Relative survival (%) and 95% confidence intervals (CI) for lung cancer in Ontario, by morphologic group, 1979-1998

		5-year		10-year	
Females	**#**	**%**	**95% CI**	**%**	**95% CI**
Squamous cell carcinoma	5,742	17.6	(16.5, 18.8)	12.3	(10.7, 14.1)
Adenocarcinoma	10,850	22.4	(21.4, 23.4)	16.1	(14.9, 17.5)
Small cell carcinoma	5,228	7.2	(6.4, 8.1)	4.3	(3.5, 5.3)
Large cell carcinoma	2,738	9.8	(8.5, 11.3)	7.3	(5.5, 9.4)

		5-year		10-year	
Males	**#**	**%**	**95% CI**	**%**	**95% CI**
Squamous cell carcinoma	20,003	17.6	(17.0, 18.3)	13.1	(12.3, 14.0)
Adenocarcinoma	14,908	16.5	(15.7, 17.3)	12.2	(10.9, 13.5)
Small cell carcinoma	8,976	5.8	(5.2, 6.4)	4.0	(3.1, 5.0)
Large cell carcinoma	5,425	8.4	(7.5, 9.3)	6.6	(5.3, 8.2)

Forecasts

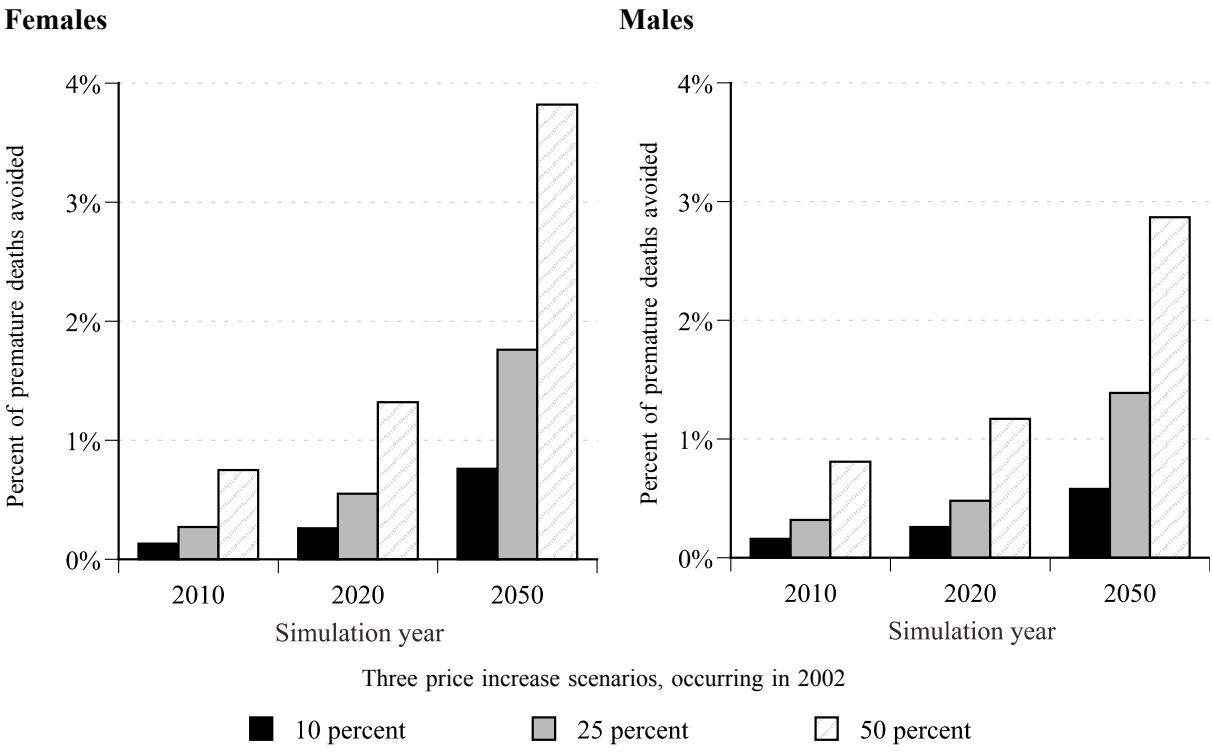
Reduction in premature mortality as a result of three anti-tobacco interventions

Figure 27 describes the results of forecasting the effects of three price increase scenarios:

1. a 10% increase in the price of a pack of cigarettes,
2. a 25% increase, and
3. a 50% increase.

Based on the methodology described below (see Materials and Methods Section), and focusing on premature mortality (under 65 years of age) that will be prevented as a result of reductions in smoking prevalence, these scenarios show a clear improvement in benefits over the period from 2002 (the point at which each intervention is assumed to occur) and 2050.

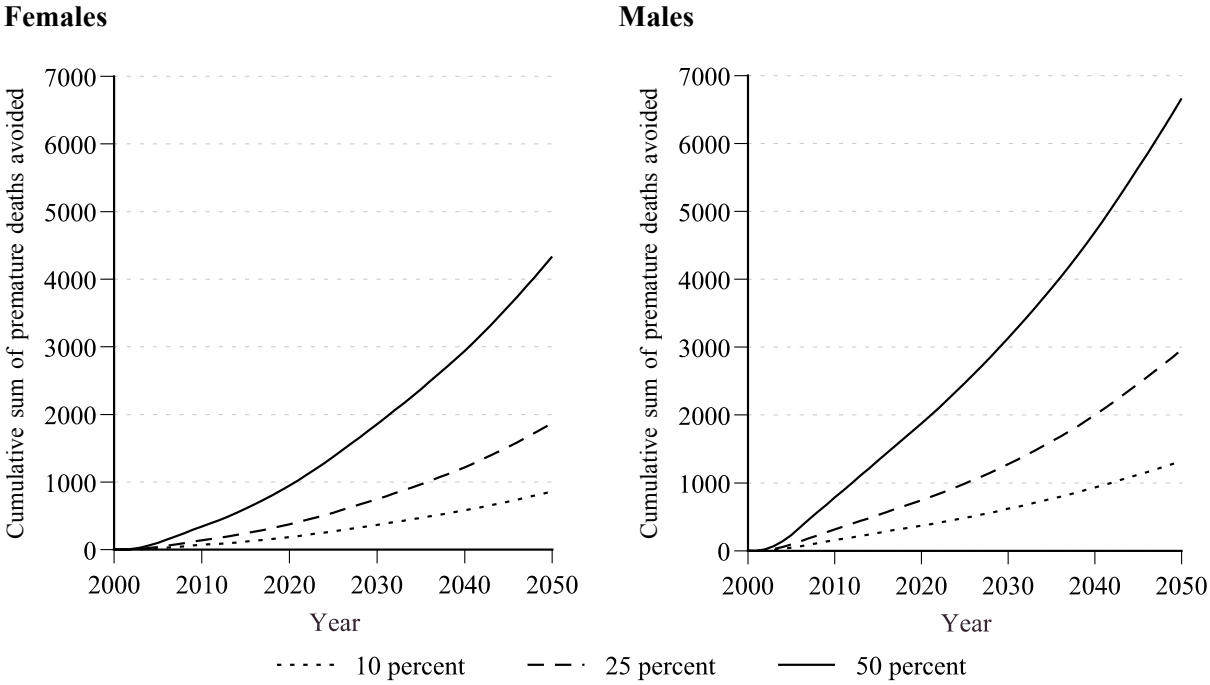
Figure 27. Total annual mortality reduction in Ontario adults under 65 years of age after three anti-tobacco interventions, Ontario 2010, 2020, and 2050, by sex



Cumulative premature deaths avoided as a result of three anti-tobacco scenarios

Figure 28 describes the exponential increase in cumulative number of lives saved under 65 years of age as a result of effective tobacco strategies. The magnitude of the benefit is directly proportional to the relative reduction in smoking prevalence. Further, this benefit continues to increase for at least fifty years following the intervention. Indeed, after 20 years, not even half of the benefit will have been achieved, largely due to the long latency and lag from the time of change in the prevalence of smoking to the full effect.

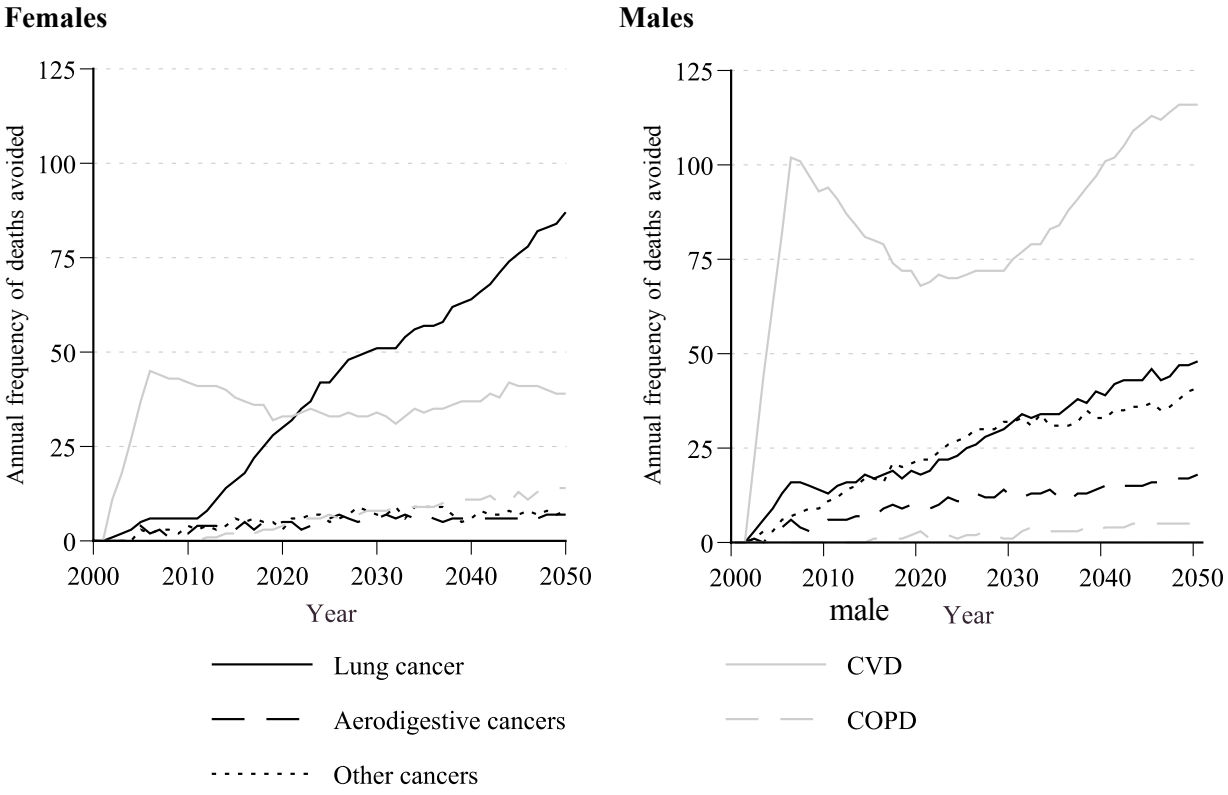
Figure 28. Cumulative mortality reduction in Ontario adults under 65 years of age after three tobacco control scenarios, by sex, 2002-2050



Type of premature deaths avoided in Ontario

Figure 29 describes the fatal diseases that will be avoided as a result of implementing effective tobacco reduction strategies. Based on future forecasting, employing the scenario of a 50% increase in the price of cigarettes in 2002, these graphs display the relative contribution based on the prevention of cancers, cardiovascular diseases and chronic obstructive pulmonary disease. Because of the longer latency, and lag, associated with cancers and chronic obstructive pulmonary disease, it is not surprising that the effects for these conditions take longer to manifest.

Figure 29. Annual premature deaths avoided in Ontario, by sex, various causes, 2000-2050, 50% price rise scenario.



Materials and Methods

Data sources

New cases of cancer

The process of cancer registration in Ontario is passive, relying almost completely on records collected for other purposes. Since 1977, the OCR has relied on the same four major data sources: hospital discharge summaries which include a diagnosis of cancer; pathology reports with any mention of cancer; records of patients referred to CCO's eight Regional Cancer Centres (RCCs) or the Princess Margaret Hospital (PMH) (now part of the University Health Network) - the specialized institutions treating cancer patients in Ontario; and death certificates with cancer as the underlying cause of death.

All records except pathology reports are coded at the source and provided to the OCR in machine-readable form. Paper copies of pathology reports are sent to the OCR by all hospital and private pathology laboratories and are coded and key-entered by OCR staff. Since 1991, the OCR has also received day surgery summaries which include a diagnosis of cancer. The OCR receives about 400,000 records from these multiple sources each year. The OCR is highly automated, relying heavily on automated edit-checking, computerized probabilistic record linkage and automated rule-based systems for summarizing patient and tumour information. Further details about the operation are available in recent monographs (Holowaty *et al*. 1995; Marrett *et al*. 1995; McLaughlin JR *et al*. 1995).

Tobacco-related cancers

This term describes those types of cancer that are partly or wholly caused by smoking. Unlike the term tobacco-attributed cancers, which describes the fraction of tobacco-related cancers that are most likely caused by smoking, the former term describes the full complement of these cancers, regardless of the smoking-attributable fraction. The tobacco-related cancers are summarized in Table 1 (see the Tobacco Use and Health section: The health consequences of tobacco use).

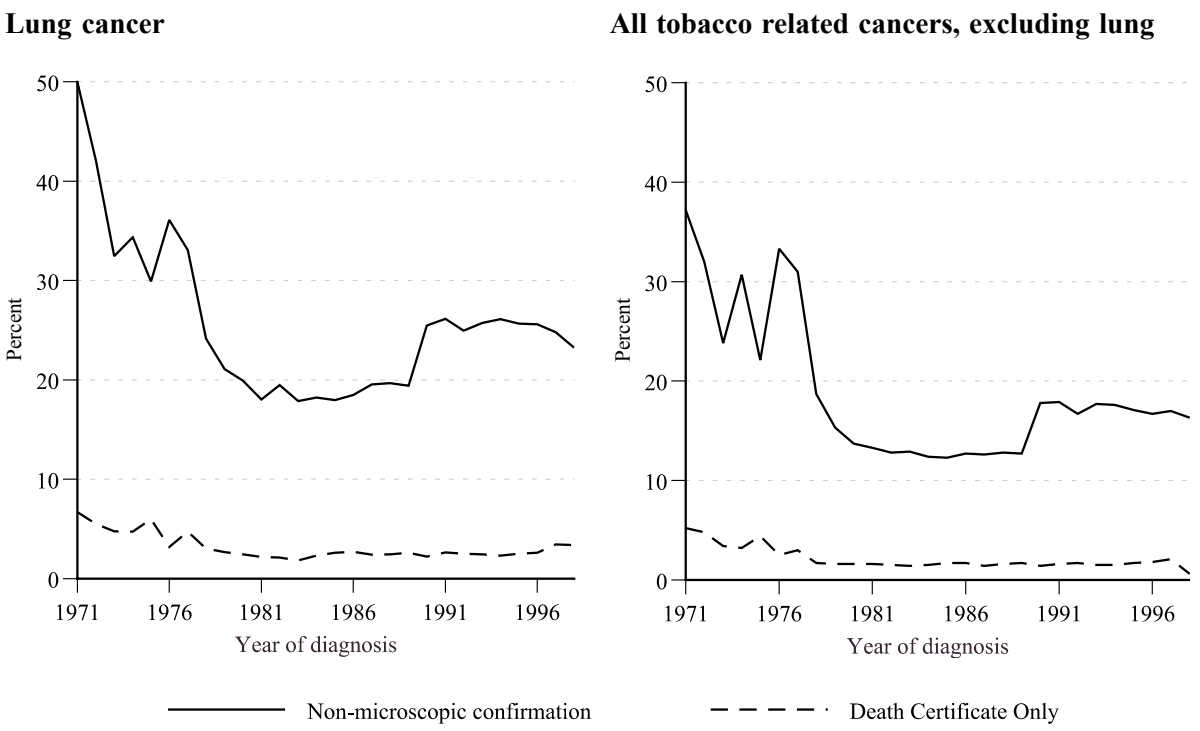
Quality of data for new cases of cancer

Microscopic examination of tissue or cells is the definitive diagnostic test for cancer. During the period 1971-1998, 22.0% of tobacco-related cancers were not microscopically verified in the OCR. The proportion decreased to 19.3% for the more recent interval 1979-1998. This rate of non-microscopic confirmation is still above that reported by established active registries; for instance, the U.S. SEER (Surveillance, Epidemiology and End Results Program) registries report a rate of 8.2% for the tobacco-related cancers.

Another parameter of data quality is the percentage of cases for which a death certificate is the only source of information supporting a diagnosis of cancer. Only 2.5% of tobacco-related cancers registered from 1971-1998 were registered from death certificates only. Figure 30 shows the temporal trends in data quality indicators for these cancers.

Because the management of tobacco-related cancers almost always requires contact with institutions that comprise two or more of the OCR's major reporting sources, it is likely that reporting is quite complete. During the 1971-1998 time period, only 13.5% of tobacco-related cancer cases registered in the OCR were identified by a single source, with 5.0% of cases being identified by pathology reports or hospitalization records only.

Figure 30. Temporal trends in data quality indicators, both sexes combined



Deaths from cancer and other causes

Mortality data in the OCR are largely derived from the Office of the Registrar General of Ontario (within the Ontario Ministry of Community and Business Relations), where death certificate information is collected and coded. Mortality rates are estimated from the reported underlying cause of death, coded according to the International Classification of Diseases - Ninth Revision (ICD-9) (WHO 1977), for persons residing in Ontario at the time of death. The groupings used for this monograph, including the conversions used for 7th, 8th and 9th Revisions, are listed in Appendix D.

Population data

Rates were calculated using annual mid-year estimated resident populations of males and females by 5-year age group. These estimates are based on the Population Census of Canada, conducted every five years, and are corrected for census undercounts. Populations for both Ontario and its census divisions were provided by Statistics Canada (Statistics Canada 1997). Populations for CCO Regions (CCORs) and Public Health Units (PHUs) were determined by adding the populations for all census divisions that comprise each Region or Unit (see Appendices B and C).

The age distribution of the 1991 Canadian population, adjusted for census undercount, was used to calculate most of the age-standardized rates appearing in this monograph (see Appendix A). For international comparisons, the World Standard Population was used to calculate the age-standardized rates (see Appendix A).

National and international data

Health Canada provided incidence rates of tobacco-related cancers by province using data from the Canadian Cancer Registry at Statistics Canada (Cormier 2001). The incidence rates of cancer around the world were recently published by the International Agency for Research on Cancer (IARC) (Parkin 1997). This monograph covers the period 1988-1992 for most reporting registries. For comparison with Ontario incidence data, countries were selected to represent different continents, but also had to meet all of the data quality requirements of the IARC publication. No areas of Africa qualified for this comparison.

Incidence and mortality data

Cancer site

A cancer is generally coded according to the body site at which it first occurs. In the OCR, the primary site of cancer, or cancer cause of death, has been coded according to ICD-9 (WHO 1977). The ICD-9 codes for tobacco-related cancers are listed in Table 1 (see the Tobacco Use and Health section: The health consequences of tobacco use).

Morphology

The microscopic morphology or histopathology of tumours describes the appearance of cancerous cells, tissues and organs under the light microscope. The morphologic type of cancer is an important prognostic factor; additionally, it is important for treatment decisions. Often, it is also important in epidemiologic studies of cancer etiology. Cancers of different morphologic types that occur in the same anatomic site often have different etiologies, incidences and prognoses.

The morphologic data presented in this monograph have been coded according to the First Edition of the International Classification of Diseases for Oncology (ICD-O) (WHO 1976; Percy *et al*. 1990). The morphology code in ICD-O consists of five digits. The first four digits describe the morphologic type, and the fifth digit describes the behaviour. The OCR registers only malignant neoplasms; that is, those with a behaviour code of 3.

As the total number of morphologic types recognized in ICD-O approaches 500, grouping of the various types is necessary. Appendix E shows the grouping scheme employed for this monograph, a variant of common groupings for comparative studies (Parkin *et al*. 1998; Travis *et al*. 1995).

Spread of cancer at diagnosis (stage)

The OCR does not routinely collect information about the stage of cancer at diagnosis, primarily because this information is not captured for most cases by the reporting sources. Stage distribution for a North American population that is reasonably representative of Ontario suggests the patterns displayed in Table 29 for incident cases of lung and other tobacco-related cancers diagnosed as first primaries over the interval 1994 to 1998 (NCI SEER Program 2001).

Table 29. Stage at diagnosis of tobacco-related cancers, first primaries only, 1994-1998, both sexes combined (SEER 2001)

SEER Site groupings for tobacco-related cancers	SEER Summary stage			
	Localised	Regional	Distant	Unstaged
Oral cavity and pharynx	34.9%	46.1%	7.9%	11.1%
Oesophagus	25.2%	25.6%	23.9%	25.3%
Stomach	22.7%	29.1%	31.9%	16.3%
Anus, anal canal and anorectum	47.3%	30.8%	6.4%	15.5%
Pancreas	7.3%	22.8%	48.7%	21.3%
Lung and bronchus	15.9%	23.0%	46.4%	14.8%
Vulva	64.0%	24.3%	3.4%	8.3%
Urinary bladder	73.4%	18.7%	3.2%	4.8%
Kidney and renal pelvis	50.9%	20.7%	20.4%	8.0%

Time periods

Incidence, mortality and survival trends are presented in this monograph for the period 1971-1998. While the OCR actually begins in 1964, population estimates adjusted for census undercount are only available since 1971; 1998 is the most recent year for which there is complete reporting at the time this monograph was compiled.

The most recent 5-year time period, 1994-1998, was used to estimate the recent burden of cancer and to describe selected characteristics of tumours or cases. This 5-year aggregate stabilizes estimates of infrequent events (diagnoses at younger ages, for example) while providing the most current information.

Other periods are used occasionally because of availability; for example, data from other provinces of Canada are for 1993-1997, because that is the most recent period available for all provinces. The most recently published international data are for 1988-1992.

Residence at diagnosis and death

The geographic variation in incidence and mortality in tobacco-associated cancers in Ontario over the period 1994-1998 is described in terms of Cancer Care Ontario Regions (CCORs) and Public Health Units (PHUs), which are aggregations of census divisions (see Appendices B and C). Census division of residence at the time of diagnosis comes primarily from hospital records in the OCR. All but 1.3% of cases of tobacco-related cancer in 1994-1998 have a census division of residence recorded. Because there is a small percentage of cases with missing residence, all incidence rates calculated for the purpose of comparing the CCORs or PHUs to the province exclude cases with missing residence. Census division of residence at time of death comes from death certificates in the OCR. Mortality rates for CCOR comparisons were also calculated with missing residence excluded, since 3.2% of records of tobacco-related cancer deaths for the 1994-1998 time period did not report a valid census division of residence.

The eight CCORs cover the entire province, with populations ranging from 5.3 million in the largest (Central East CCOR) to 367,000 in the smallest (South CCOR) (Statistics Canada 1997). Within each CCOR is situated one of CCO's Regional Cancer Centres (RCCs). In addition, Princess Margaret Hospital (PMH) is located within the Central East CCOR. Maps describing CCOR boundaries and census division components are shown in Appendix B.

Appendix C contains a map showing Ontario PHUs and a table showing corresponding census divisions as used for PHU analysis. It should be noted that actual PHU boundaries are not always equivalent to those for the grouped census divisions as used herein. The discrepancy is greatest in northern Ontario. The following PHUs do not correspond exactly to groups of census divisions: Algoma, Muskoka-Parry Sound, North Bay and District, Northwestern, Porcupine, Renfrew, Sudbury and District, Thunder Bay, and Timiskaming.

Statistical methods

Age-standardized rates

To compare incidence and mortality rates between populations which have different age structures, age-standardized rates were calculated. The age-standardized rate is a weighted average of the age-specific rates, using a standard population age distribution. The standardized rates reflect the incidence and mortality that would be expected if the population of interest had an age structure identical to the standard population. The 1991 Canadian population, adjusted for census undercount (Appendix A), was used as the standard throughout most of this monograph. However, in comparisons between Ontario's rate and those of other parts of the world, the World Standard Population was used (see Appendix A). Age-standardized rates are expressed per 100,000 person-years.

Time trends

To illustrate time trends, 3-year moving averages of age-standardized incidence and mortality rates were plotted, for all ages and by broad age groups. Three-year moving averages were used to smooth out annual fluctuations, which can occur when there are small numbers of cases. Trends by morphologic subgroup were plotted from 1971 to 1998, again using 3-year moving averages.

The average annual percentage change (APC) in age-adjusted rate over time is estimated by fitting a regression line to the logarithm of the rate, using year of diagnosis as the independent variable. Because one single straight line may not fit the log rates over a long time period, a joinpoint analysis was conducted. This method identifies years (if any) where there are significant changes in slope (Ries,Wingo *et al*. 2000; Kim *et al*. 2000) and estimates of the regression coefficients for the distinct time periods are identified. These are then converted into estimates of APC and associated 95% confidence intervals. The APC estimates are reported either for the entire time period or for two or more time periods, depending on the results of the joinpoint analysis.

Rate ratios

The comparison of incidence or mortality rates in two populations (or subgroups), as a ratio of these rates, is termed a rate ratio (RR) or relative rate. In this monograph, this technique was employed for comparison of the rates of lung cancer for each CCOR or PHU to that of the province as a whole.

The rates for each population were age-standardized, using the 1991 Canadian population, adjusted for census undercount (Appendix A), as the standard. A rate ratio in excess of 1.0 for regional comparisons, for example, means that the region has a higher rate than Ontario as a whole.

Confidence intervals

A confidence interval (CI) indicates the range of values for a parameter of interest (e.g. a rate ratio) which has a specified probability of including the true value. Thus, the 95% CI for a RR represents the interval which has a 95% chance of covering the true value of the RR. The 95% CI for each incidence and mortality RR comparing a regional rate to the provincial rate is calculated using the approximate bootstrap confidence interval method (Swift 1995).

When the 95% CI includes 1.0, the RR is considered not to differ significantly from 1.0 (i.e. the rate for the region does not differ significantly from that for the province), and the difference between the RR and 1.0 is ascribed to chance variation. If the interval does not include 1.0, the RR is deemed to be statistically significantly different from 1.0. A RR is declared to be significantly low if the upper limit of the CI is less than 1.0 and significantly high if the lower limit of the CI is greater than 1.0. Due to rounding, a RR may occasionally be declared to differ significantly from 1.0 when one of the end points of the CI is 1.0.

Survival

Cases with a first primary diagnosis of tobacco-related cancer between 1979 and 1998 were used in the survival analyses for all cases by age group. Cases were followed for deaths occurring through December 31, 1999 (as recorded in the OCR). The outcome of interest was all-cause mortality. If no death information was available, cases were assumed to be alive through December 31, 1999.

The relative survival rate is a measure of the influence of the tobacco-related cancers on normal life expectancy, because it is obtained by adjusting the crude survival for the average life expectancy of the general population of the same age and sex as those diagnosed with cancer. (A more detailed description can be found in McLaughlin JR *et al*. 1995.) Since it is generally acknowledged that cause of death information on death certificates is often not accurate enough to determine whether an individual died from his or her original cancer, the relative survival rate is the preferred method for reporting survival from cancer registry data.

Mapping methods

Maps display relative lung cancer incidence in Ontario for the years 1994-1998, for both sexes and all ages combined. The relative incidence for a census division is compared to the provincial average (based on 100). Both maps display lung cancer incidence, but the second is adjusted for smoking (# pack-years).

Time trends and differences in age and sex distributions that may distort regional patterns are controlled for using regression analysis. Cancer incidence data are provided by the Ontario Cancer Registry, population estimates are provided by Statistics Canada, and smoking estimates were obtained from the 1990 Ontario Health Survey (OHS 1990).

Attributable risk

An attributable risk is an indirect estimate of morbidity or mortality due to a specific causal factor. For this monograph, the causal (risk) factor is cigarette smoking. The indirect approach requires estimation of the likelihood of causation by the risk factor, which is then applied to the total number of incident cases, or deaths, partly, or wholly, caused by the risk factor. For example, if there is a probability of 0.85 that a case of lung cancer is caused by smoking, then the product of this probability (also known as the etiologic risk) and the total number of lung cancers diagnosed in the population gives an estimate of the number of lung cancer cases attributable to smoking.

The attributable risks used in this report were developed with the methodology recently outlined by the Australian Institute of Health and Welfare (English *et al*. 1995; Ridolfo *et al*. 2001). The most common source of attributable risk estimates is from studies of the comparative rates of death or illness in cohorts of people exposed and not exposed, or exposed at varying levels, to tobacco. Among those exposed to the risk factor, the attributable risk can be expressed mathematically, as follows:

$$AR = (RR - 1) \div RR.$$

Among the total population, the attributable risk can be expressed as:

$$PAR = p(RR-1) \div [p(RR-1) + 1],$$

where p is the proportion of the total population exposed to the risk factor. This can be extended where a factor has several categories, as follows:

$$PAR = \Sigma\{p(RR-1) \div [p(RR-1) +1]\}.$$

In some cases, pooled relative risk estimates are calculated from a number of studies. A full discussion of pooling multiple study results is beyond the scope of this monograph. Such a discussion can be found in the reports of Ridolfo *et al*. (2001), and English *et al*. (1995).

Tobacco-attributed causes of morbidity and mortality

The conditions included in this monograph are those originally identified by English *et al*. (1995), and later incorporated in a Canadian report (Single *et al*. 1996), (see Table 1). However, based on more recent evidence reviewed by Ridolfo *et al*. (2001), several RR estimates have been revised. For example, it has been concluded that the relationship between tobacco and cervical cancer is probably not a causal one (thus, the attributable risk should be zero). Further, the association between smoking and peptic ulcer disease is mostly due to an interaction between helicobacter pylori and smoking 10 or more cigarettes a day. Mortality associated with peptic ulcer disease is mainly due to bleeding among the elderly, who also have more co-existing morbidity. Indeed, the large majority of these deaths are due to hypertension and ischaemic heart disease, rather than from the bleeding peptic ulcer itself (Ng *et al*. 1994). Overall, it is estimated that less than 1% of peptic ulcer deaths are attributed to tobacco smoking (Ridolfo *et al*. 2001).

Traditionally, estimates of PAR have relied on knowledge of the current prevalence of tobacco smoking. However, many conditions have a long time lag between exposure to tobacco smoke and

their associated ill-effects; in the case of cancers, and chronic obstructive pulmonary disease, it is often many decades. Thus, for these conditions, estimates of the current prevalence of tobacco smoking are not optimal for quantifying the past and current tobacco-attributed disease burden.

For cancers and chronic obstructive pulmonary disease, past smoking prevalence estimates are derived from the Ontario Health Survey of 1990 (OHS 1990). For all other deaths attributed to tobacco, current smoking prevalence estimates are derived from the Ontario component of the National Population Health Survey 1996/97 (NPHS 1998). Both of these series are displayed in Table 2.

Evidence is accumulating that ETS, or passive exposure to tobacco smoke, is a risk factor for a number of diseases in both adults and children (WHO 1999; Collishaw *et al.* 2001; NHMRC 1997). The estimates presented in this monograph are derived from a recent Ontario report (Luk and Single 2001), employing a methodology used by the Australian National Health and Medical Research Council (NHMRC 1997). These estimates are mainly for never smokers who were exposed to ETS from their smoking spouses or mothers. Thus, these estimates do not take into account the impact of ETS from any other source.

Systematic reviews have concluded that the best available evidence is strongest to implicate ETS as a cause of fatal lung cancer and fatal ischemic heart disease, as well as morbidity associated with childhood asthma and lower respiratory tract infections in early life. The relative risks associated with these conditions are summarized in Table 1 (see the Tobacco Use and Health section: The health consequences of tobacco use).

Estimates of the proportion of never smokers, whose spouses are current smokers, by gender and age, were derived from the Ontario portion of the 1996/97 National Population Health Survey (NPHS 1998). Maternal smoking prevalence was derived from the Ontario Component of the National Longitudinal Survey of Children and Youth (1996/7) (Statistics Canada and Human Resources Development Canada 1998).

Historical trends in tobacco-attributed mortality in Ontario

A different methodology, originally proposed by Peto *et al.* (1992), has been used to estimate the past burden of tobacco-attributed mortality, in the absence of long-term historical smoking prevalence data. Employing this methodology, an underlying rate of lung cancer among men and women who never smoked is derived from the huge 1982 American Cancer Society-Cancer Prevention Study (CPS) Cohort (Garfinkel 1985). Assuming this underlying risk applies to men and women in Ontario, it may be used to derive the expected rate of lung cancer in the absence of smoking. Comparing this with the observed lung cancer rate for the population, generates the proportion of lung cancer attributable to smoking. Further, using published rates of lung cancer for CPS smokers and non-smokers, a “synthetic” smoking prevalence rate can be derived, representing the historical prevalence most consistent with the observed lung cancer rate. Combining this synthetic prevalence with the CPS risk ratios described in Peto *et al.* (1992), attributable fractions may be derived for the remaining cancers, for chronic obstructive pulmonary disease, for vascular disease and for other medical disorders.

The Peto methodology subdivides tobacco-attributed mortality into a few broad categories, and then employs conservative assumptions in determining what proportions of tobacco-related deaths to

attribute to tobacco. For example, deaths from accidents and injuries (including fires, suicides and motor vehicle accidents), neonatal deaths (including stillbirths), all other deaths under 35 years of age, and all deaths from cirrhosis of the liver are not attributed to tobacco, even though some of these deaths are probably due to smoking. For diseases other than lung cancer (upper aerodigestive cancers, all other cancers, chronic obstructive pulmonary disease, other respiratory disease, vascular disease and other medical causes), the attributable excesses are estimated by calculating, on an age-specific basis, the excess percentages suggested indirectly by the national lung cancer rates, and then simply halving each excess percentage (among smokers), in the hope of obtaining a conservative estimate of the proportion of such deaths attributable to tobacco. Halving this percentage excess is crude and arbitrary, but it does provide a reasonable degree of "protection" against over-estimating the epidemic, albeit at some risk of under-estimation. The degree of under-estimation is likely not that great. Indeed, for Ontario in 1992, this "conservative" method is found to attribute to tobacco about 10-20 percent more deaths than Single *et al.* (1996) did by combining provincial mortality rates with additional data on the current prevalence of smoking in Ontario.

Again, for other than lung cancer, a more complicated procedure is needed to estimate the fractions attributable to tobacco, since it cannot be assumed that the absolute rates of these diseases among non-smokers will be comparable over time. Using the age-specific, sex-specific lung cancer mortality rates for Ontario, back to 1950, a mixture of CPS smokers and CPS non-smokers ages 35 to 79 years is constructed, with the proportions of smokers at ages 35 to 59, 60 to 64, 65 to 69, 70 to 74 and 75 to 79 chosen to make the lung cancer rates in each of these age and sex groups in the general CPS population equal to those in Ontario over time. The ratio of the CPS non-smoker lung cancer rates to Ontario's rates in these five separate age groupings determines the proportion not attributable to tobacco. Then, using relative risks for other causes of tobacco-attributed mortality as derived from the CPS study (Peto *et al.* 1992), the excess in each age group was determined as a percentage of the non-smoker rates. Finally, for a particular disease category in a particular age and sex group, the method of extrapolation from the synthetic CPS population to Ontario assumes that the proportion of deaths due to smoking is similar in these two populations. But, it cannot be assumed that all excess mortality among smokers is actually caused by tobacco. For example, upper aerodigestive cancers are caused both by tobacco and by alcohol, and smokers may drink more heavily than non-smokers. Thus, part of the excess mortality attributed to smoking in Ontario may be due to factors other than tobacco. To ensure that the hazards of tobacco are not exaggerated, the excess mortality in the synthetic CPS population is halved for estimating the fraction of deaths attributed to tobacco. This simple halving of the excess risk among smokers is obviously quite crude and arbitrary, and likely under-estimates some of the true hazards of tobacco.

Forecasting future tobacco-attributed mortality in Ontario

The future burden of TAM in Ontario is influenced by a number of factors, most notably the future demographic structure of the population, the future prevalence of smoking, the interaction between smoking and other risk factors associated with common diseases, and future interventions that may lower, or increase, the prevalence of smoking. Increasingly, computer-assisted, decision support models have been used to simulate the effects of different scenarios within defined populations, using available data on risk factor prevalence and the related relative risks of mortality (or morbidity), and then projecting the results over future years. Such models are valuable in assessing the relative health gain and costs of alternative prevention strategies, and serve to illustrate the application of epidemiology to decision-making at the population level (Gunning-Schepers 1989).

In this monograph, *Prevent,* (Version 2.9), has been employed to forecast future estimates of tobacco-attributed mortality. Originally developed in the Netherlands in 1988, it is a cell-based macro-simulation model that can estimate the health benefits for a dynamic population (incorporating births and deaths) in the face of changing risk factor prevalence due to baseline trends, or designed interventions, over a maximum period of up to 100 years (Bronnum-Hansen 1999; Baan *et al.* 1999; Barendregt 2001). The outputs of *Prevent* are displayed both in terms of proportional changes in disease-specific mortality (or incidence) and in terms of absolute changes in these parameters.

The underlying methodology used in *Prevent* allows for:

- the possibility that a single risk factor can affect several outcomes, as well as the possibility that a single outcome is affected by several risk factors
- a time dimension to simulate the reduction in excess risk after cessation of exposure to the risk factor (both latency and lag periods are can be set)
- the interaction between the effect of the intervention and the demographic evolution of the population

While earlier versions of *Prevent* included a number of risk factors (cigarette smoking, hypertension, cholesterol, obesity, alcohol) as well as a number of outcomes (ischaemic heart disease, cerebro-vascular accidents, chronic obstructive pulmonary disease, lung cancer, cirrhosis, traffic accidents and accidental falls), for this monograph, only a single risk factor is included (cigarette smoking) and the list of associated outcomes is limited to cardiovascular disease (ICD9 390-549), chronic obstructive pulmonary disease (ICD9 490-496), lung cancer (ICD9 162), upper aerodigestive cancers (ICD9 140-150, 161) and other tobacco-related cancers (ICD9 rest of 140-208) (see Table 1).

Prevent combines principles from epidemiology and demography in a dynamic population model (Barendregt 1999). The dynamic elements describe those characteristics expected to change over time and include:

- the population and its composition, determined both by historical circumstances and as a consequence of interventions
- risk factor prevalence, both as a consequence of historical patterns (resulting from age, period and cohort effects) and as a consequence of interventions
- disease risk, also determined by historical patterns and intervention effects

A formal mathematical description of the models employed in *Prevent* is available in a report recently prepared for the European Commission (Baan *et al.* 1999). A summary is provided below. The input tables for the PREVENT system were populated with Ontario-specific data for this monograph. Specifically, the following information was input:

Cigarette Smoking

1. Look Back Period: This describes the number of years of data about past risk factor exposure that is input. For this monograph, this value is 75 years extending back from the baseline year 2000, to 1925.
2. Past Exposure for the youngest cohort: For each year of the Look Back Period, the prevalence of current regular smoking in Ontario, by sex, for the age group 15 to 19 years.

3. Period Effect for all age groups: For each year of the Look Back Period, by sex and age group. Risk factor prevalence is considered to be a cohort variable, subject to age and period effects. These estimates were provided by the developer, Dr. J. Barendregt, through APC modelling of the original smoking estimates.
4. Age Effect of exposure: by five-year age group, and sex. Values range from 0-1; a value of 1 means no age effect. Again, provided by Dr. Barendregt.
5. Prevalence of Exposure for the baseline year minus the Look Back Period: by five-year age group and sex.
6. Latency: the number of years after a change in risk factor exposure before the mortality outcome begins to change.
7. Lag: the number of years from when the change in mortality begins until it reaches the full effect.
8. Relative Risks: this describes the strength of association between the risk factor, current smoking, and associated outcomes, by five-year age group and sex. These estimates are derived from the large ACS-CPS II cohort study (Peto *et al.* 1992).

Diseases

1. Disease-specific mortality rates, by five-year age group and sex for the baseline year.

Population

1. Total mortality probabilities for the Ontario population in the baseline year (2000), by single year of age and sex.
2. Population counts for Ontario in the baseline year (2000) by single year of age and sex.
3. Birth rates for Ontario women in the baseline year by single year of age for ages 10 to 50 years.

For this monograph, *Prevent* contains two stock variables: smoking prevalence and population frequencies. These are both age dependent, and with a time step of 1 year. Most of the variables are in 5-year age groups (0-4,...90-94,95+), except the population variables (population counts, birth rates, death rates) which are in 1-year ages. It should be noted that each *Prevent* run consists of two scenarios: one reference and one intervention scenario.

Smoking prevalence is considered to be a cohort variable, subject to age and period effects:

$$P_{c,t}^{a} = P_{c,t-1}^{a-1} A^{a} D_{t}$$

Where $P_{c,t}$ is the prevalence of current smoking at time *t, a* is age index, *A* is the age effect, and *D* is the period effect. Prevalence of former smoking is:

$$P_{f,t}^{a} = \begin{cases} P_{f,t-1}^{a-1} + P_{c,t-1}^{a-1}(2 - A^{a} - D_{t}); & A^{a}, D_{t} < 1 \\ P_{f,t-1}^{a-1} + P_{c,t-1}^{a-1}(1 - A^{a}); & A^{a} < 1, D_{t} \geq 1 \\ P_{f,t-1}^{a-1} + P_{c,t-1}^{a-1}(1 - D_{t}); & A^{a} \geq 1, D_{t} < 1 \\ P_{f,t-1}^{a-1}; & A^{a} \geq 1, D_{t} \geq 1 \end{cases}$$

Prevalence of never smoking is:

$$P_{n,t}^{a} = 1 - P_{c,t}^{a} - P_{f,t}^{a}$$

The first three equations apply for all ages from the age considered to be the youngest age of exposure, (a_c *=15-19 yrs)*, to the highest. The prevalence at the youngest age of exposure is governed by the cohort effect *C*:

$$P_{c,t}^{a_c} = C_t$$

An intervention *I* is defined as:

$$P_{c,t}^{a} = P_{c,t-1}^{a-1} I_t^a$$

After the intervention the prevalence of former smoking is:

$$P_{f,t}^{a} = \begin{cases} P_{f,t-1}^{a-1} + P_{c,t-1}^{a-1}(1 - I_t^a); & I_t^a < 1 \\ P_{f,t-1}^{a-1}; & I_t^a \geq 1 \end{cases}$$

Because there are time lags between smoking exposure and its effects on diseases, past smoking prevalence influences the present patterns of risk. A variable *L*, for look back, is defined for each dataset, and determines for how many years in the past data have been specified. For each run with the model the number of years of future projection *T* is specified. The above equations then are executed for *t=-L+1..T*.

The link between changes in smoking prevalence and diseases is calculated using the potential impact fraction, *F*. Barendregt (1999) describes the calculation in two steps, using an intermediate variable *Q*, that summarizes total population risk:

$$Q_t^a = \sum_{\tau=t-CUM}^{t} \sum_{e} P_{e,\tau}^{\alpha} R_e^{\alpha}$$

Where CUM is the variable specifying the number of years of cumulative effect, a constraint is put such that $\tau \geq -L$, *e* is an index for current, former, or never-exposed, and α stands for age such that $\alpha = a - t + \tau$. The above equation is valid only under the constraint that $\alpha \geq 0$. Using this intermediate variable *Q* the *F* is calculated as follows:

$$F_t^{\alpha} = 1 - \prod_{\tau=t}^{T} \left(1 - \beta_{\tau,t} \frac{Q_t^a - Q_{t+1}^a}{Q_t^a} \right)$$

Where:

$$\beta_{\tau,t} = \begin{cases} 0, \tau - t \leq LAT \\ (\tau - t - LAT) / LAG, LAT < \tau - t \leq LAT + LAG \\ 1, \tau - t > LAT + LAG \end{cases}$$

and:

$$\alpha = a + LAT$$

The last two equations implement the time lags, with the former causing the effect of a change in population risk to start to take effect only after LAT years, and have full effect only after LAT+LAG years, with a linear increase in the effect in between. The latter equation assigns the effect to the appropriate age. The first two equations in this section are executed for *t=-L .. T-1*. It should be noted that the potential impact fractions are disease specific, not risk factor specific. When a disease is affected by more than one risk factor, the simultaneous effect is assumed to be multiplicative.

The potential impact fractions are used to adjust disease specific mortality according to the changes in smoking exposure. For fatal diseases, the next equation is executed for *t=0..T,* applying a secular trend (annual percent change) and the adjustment to risk factor change:

$$m_t^a = \left(1 - F_t^a\right) m_0^a \prod_{\tau=0}^{t} S_\tau^a$$

with *m* disease specific mortality rate, m_0 disease specific mortality in the base year, and *S* the secular trend, which is 1 when none is specified.

The disease specific mortality rates are then used to calculate the population changes. First an 'all other' mortality rate is calculated from the total mortality probability and the disease specific mortality rates at *t=0*:

$$M_{other}^a = -\ln(1 - U_0^a) - \sum_d m_{d,0}^a$$

where *U* is total mortality probability and *d* an index for diseases. With the 'all other' mortality rate and the disease specific mortality rates the total mortality probability for each simulation year is calculated:

$$U_t^a = 1 - \exp\left(-\left(M_{other}^a + \sum_d m_{d,t}^a\right)\right)$$

The mortality probabilities then are applied to age the population *V*:

$$V_{t+1}^{a+1} = \begin{cases} V_t^a (1 - U_t^a), a = 1..93 \\ V_t^{a+1} + V_t^a (1 - U_t^a), a = 94 \end{cases}$$

Equations for the highest and lowest ages are:

$$V_{t+1}^{95} = V_t^{95} (1 - U_t^{95})$$

and:

$$V_{s,t}^0 = b_s \sum_{a=10}^{50} V_{f,t}^a B^a$$

with *s* an index for sex, *f* denoting females, *B* birth rates and *b* the distribution of boys and girls.

Ontario-specific parameters and scenarios

Historical trends in cigarette smoking were determined using the Peto methodology (Peto *et al.* 1992) for calculating estimates over the earlier period (1925-1965), and using population surveys over the more recent period (1966-2000). Employing a lead time of 25 years in males, and 20 years in females, the Peto methodology permitted age-specific, sex-specific annual estimates of the current prevalence of smoking, assuming the youngest "start age" of 15-19 years in both sexes. On the whole, these estimates are quite consistent with those published by others (Harris 1983; Garfinkel 1997; Ferrence 1988). Additionally, the more recent estimates derived using the Peto methodology were quite consistent with the survey estimates, at least for the overlapping period of the 1960s and 1970s.

These estimates were then transferred to Dr. Barendregt who "smoothed" them, employing age-period-cohort modelling. These derived, APC estimates were incorporated in the appropriate MS-ACCESS tables in the PREVENT System. These smoothed estimates are also presented graphically in the section on Prevalence of Current Daily Cigarette Smoking in Ontario, 1925-2000.

It is, of course, very difficult to predict the future prevalence of cigarette smoking, based simply on past patterns of use. Without knowledge of changing cohort effects (i.e. initiation of smoking) as well as significant period effects (e.g. the introduction of more effective smoking cessation strategies), there is a weak foundation indeed in forecasting the future prevalence of smoking. Table 30 describes the possible future prevalence of current daily cigarette smoking across Ontario, by sex and age group. It simply assumes persistence of the estimated APC parameters, as described above, based on patterns of the past but, most notably, patterns of the recent past. For the purpose of forecasting the future prevalence of smoking in the face of various tobacco control interventions, these serve as the baseline for future forecasts of the benefits from various smoking prevention/cessation strategies.

Table 30. Future baseline prevalence of current daily cigarette smoking in Ontario

	Year			
	2000	2010	2020	2050
Males				
15-19 yr	25.1%	24.7%	24.7%	24.7%
20-24 yr	28.1%	28.5%	28.5%	28.5%
25-44 yr	25.1%	24.0%	25.3%	26.2%
45-64 yr	15.5%	12.8%	11.6%	12.5%
65+ yr	6.9%	5.9%	4.8%	2.9%
Females				
15-19 yr	18.2%	17.5%	17.5%	17.5%
20-24 yr	20.1%	19.1%	19.1%	19.1%
25-44 yr	20.8%	19.2%	18.4%	18.1%
45-64 yr	15.3%	13.6%	12.8%	11.6%
65+ yr	8.9%	7.5%	7.1%	5.5%

Because *Prevent* permits some future adjustment to background disease rates, estimated annual percent (EAPC) changes in mortality for all causes, lung cancer, aerodigestive cancers, other cancers, cardiovascular disease and chronic obstructive pulmonary disease were input for each *Prevent* run. These EAPC estimates shown in Table 31; they were generated with the Joinpoint methodology, using log-linear modelling of age-specific mortality rates over the period 1971-2000. Of course, it is difficult to know how long these age-specific changes will persist, but for the purpose of this monograph, it is assumed that these changes will persist over the interval 2001 to 2020; from 2020 to 2050, it is further assumed that there will be no additional changes in these estimates. It should be noted that, in the end, the findings based on future projections of various tobacco control scenarios will be presented as relative estimates, or differences, across the scenarios.

Table 31. Estimated annual percentage (APC) change in tobacco-related mortality in Ontario

Cause	Sex	00-19 yrs	20-34 yrs	35-49 yrs	50-64 yrs	65-79 yrs	80+ yrs
All causes	M	-4%	-6%	-7%	-4%	-2%	-1%
	F	-4%	0%	-2%	-2%	-1%	0%
Lung cancer	M	-	0%	-3%	-5%	-2%	-1%
	F	-	0%	-2%	0%	+3%	+5%
Aerodigestive cancers	M	-	-	0%	-4%	-1%	0%
	F	-	-	-2%	-2%	0%	0%
Other cancers	M	-3%	-2%	-1%	-2%	-1%	0%
	F	-3%	-2%	-5%	-3%	-2%	0%
Cardiovascular diseases	M	-4%	0%	-3%	-4%	-3%	-2%
	F	-3%	-2%	0%	-4%	-3%	-1%
Chronic obstructive pulmonary diseases	M	-3%	-4%	-6%	-4%	-3%	0%
	F	-5%	0%	-3%	0%	0%	+5%

As described above, the *Prevent* system recognizes the importance of lag and latency periods in estimating the effect of changing risk factor prevalence on chronic disease outcomes. Based on recent reviews of the epidemiologic evidence (Gunning-Schepers 1999), Table 32 describes the latency and lag estimates that were used in forecasting.

Table 32. Latency and lag estimates for tobacco-related causes of death

Tobacco-related causes of death	Latency period (years)	Lag period (years)
Aerodigestive cancers	0	30
Chronic obstructive pulmonary disease	10	40
Cardiovascular diseases	0	5
Lung cancer	10	50
Other cancers	0	30

Finally, the scenarios assess the relative effect of an increase in the price of cigarettes. Scenarios of 0%, 10%, 25% and 50% price changes, in the year 2002, are modelled. The analysis is restricted to price comparisons because of strong evidence linking cigarette consumption to price elasticity. Based on available literature, a price elasticity of 0.7 (i.e. 70%) for younger smokers (15-24 years of age) and 0.4 (i.e. 40%) for older smokers (25+ years of age) has been assumed. As an example, a price increase of 10% predicts a drop in the prevalence of smoking by 7% among younger smokers, in contrast to a relative drop of 4% among older smokers. As a second example, a 50% increase in price predicts a relative drop in the prevalence of smoking of 35% among younger smokers and 20% among older smokers.

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Appendices

Appendix A. World and Canadian standard populations and Ontario 1996 population

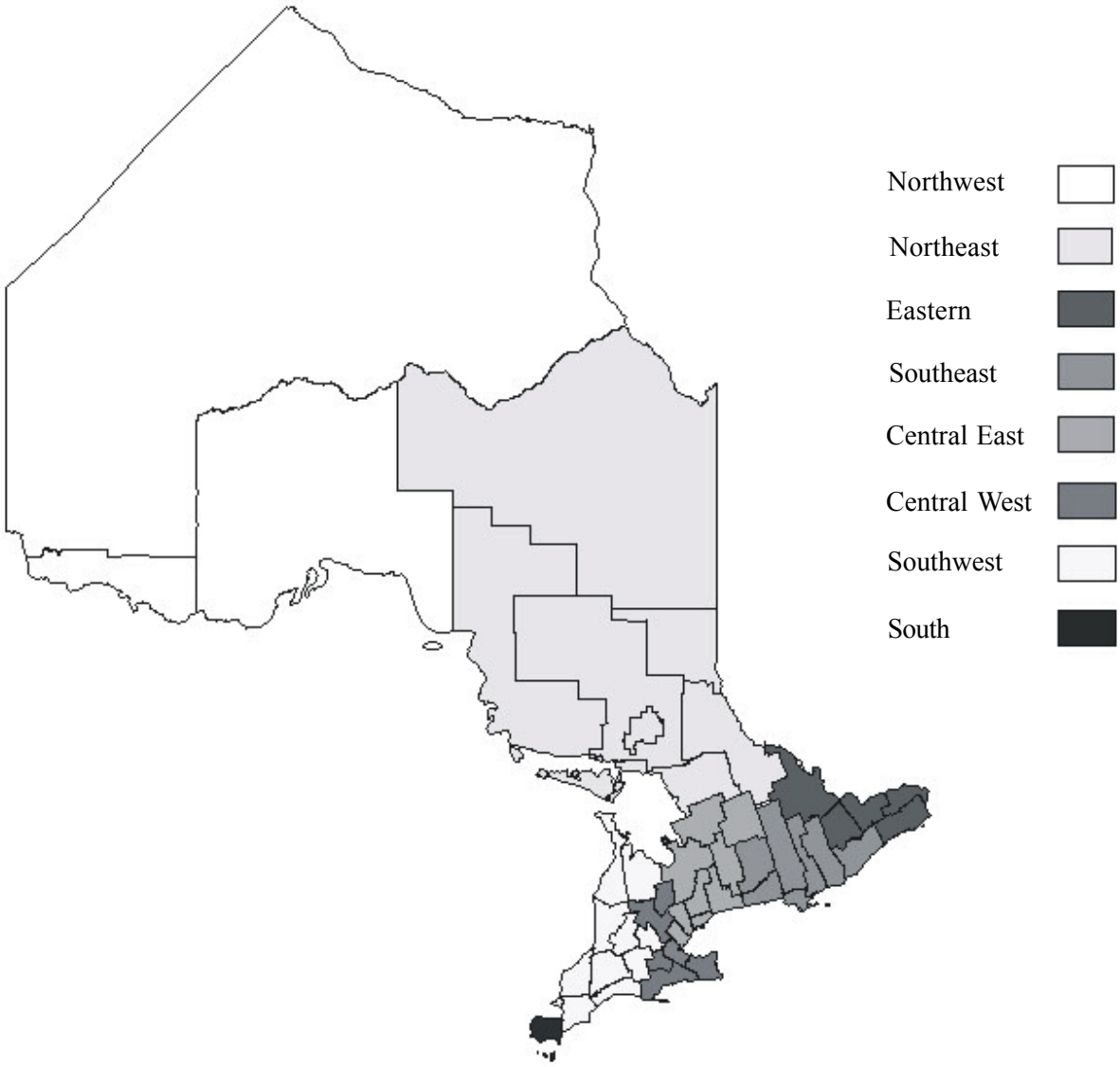
Age	World Standard Population[1]	Canadian 1991 Population[2]	Ontario 1996 Population[3]
0-4	12,000	6946.4	756,053
5-9	10,000	6945.4	760,338
10-14	9,000	6803.4	740,080
15-19	9,000	6849.5	721,396
20-24	8,000	7501.6	747,174
25-29	8,000	8994.4	826,426
30-34	6,000	9240.0	994,836
35-39	6,000	8338.8	973,241
40-44	6,000	7606.3	865,319
45-49	6,000	5953.6	790,268
50-54	5,000	4764.9	608,636
55-59	4,000	4404.1	501,217
60-64	4,000	4232.6	459,364
65-69	3,000	3857.0	431,223
70-74	2,000	2965.9	374,247
75-79	1,000	2212.7	255,754
80-84	500	1359.5	167,495
85+	500	1023.7	127,809
Total	**100,000**	**100,000**	**11,100,876**

[1] Parkin *et al.* 1997

[2] Statistics Canada 1994. Represented for convenience as the proportional distribution across age groups, rather than as actual population counts

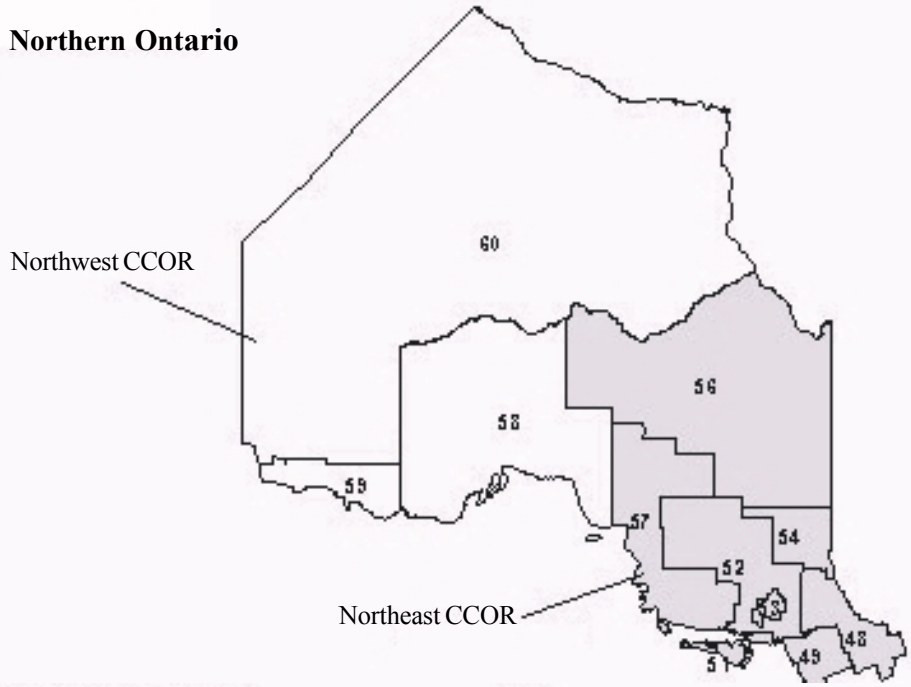
[3] Statistics Canada 1999

Appendix B. Cancer Care Ontario Regions (CCORs) showing census divisions



Appendix B (continued). Cancer Care Ontario Regions (CCORs) showing census divisions

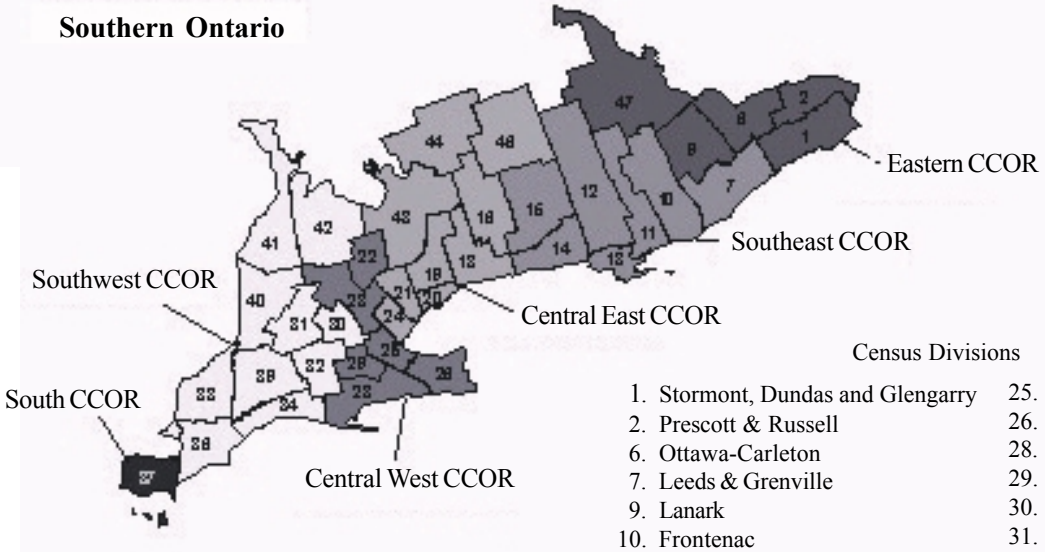
Northern Ontario



Census Divisions

48. Nipissing
49. Parry Sound
51. Manitoulin Island
52. Sudbury District
53. Sudbury R.M.
54. Timiskaming
56. Cochrane
57. Algoma
58. Thunder Bay
59. Rainy River
60. Kenora

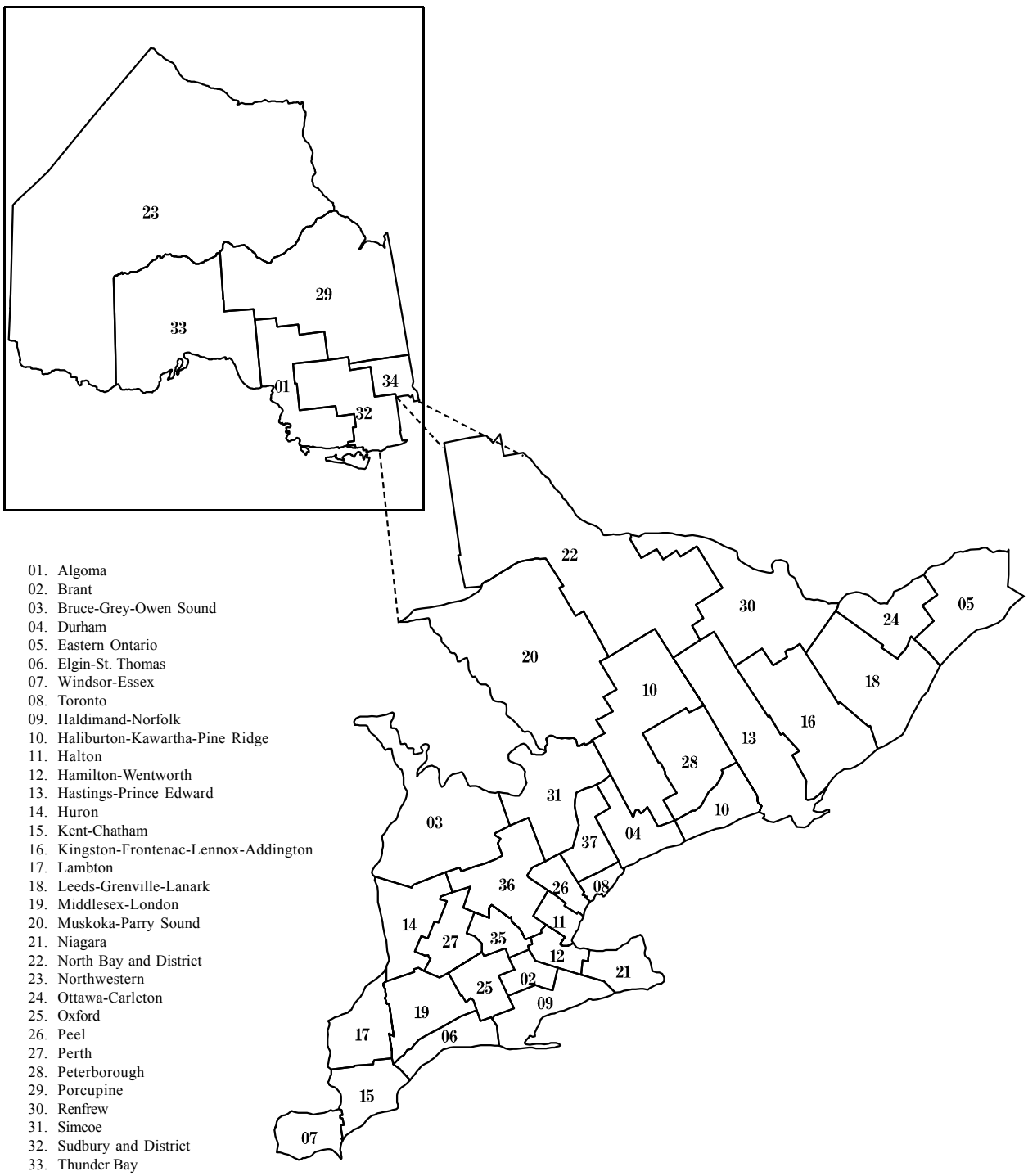
Southern Ontario



Census Divisions

1. Stormont, Dundas and Glengarry
2. Prescott & Russell
6. Ottawa-Carleton
7. Leeds & Grenville
9. Lanark
10. Frontenac
11. Lennox & Addington
12. Hastings
13. Prince Edward
14. Northumberland
15. Peterborough
16. Victoria
18. Durham
19. York
20. Toronto
21. Peel
22. Dufferin
23. Wellington
24. Halton
25. Hamilton-Wentworth
26. Niagara
28. Haldimand Norfolk
29. Brant
30. Waterloo
31. Perth
32. Oxford
34. Elgin
36. Kent
37. Essex
38. Lambton
39. Middlesex
40. Huron
41. Bruce
42. Grey
43. Simcoe
44. Muskoka
46. Haliburton
47. Renfrew

Appendix C. Ontario Public Health Units (PHUs)



01. Algoma
02. Brant
03. Bruce-Grey-Owen Sound
04. Durham
05. Eastern Ontario
06. Elgin-St. Thomas
07. Windsor-Essex
08. Toronto
09. Haldimand-Norfolk
10. Haliburton-Kawartha-Pine Ridge
11. Halton
12. Hamilton-Wentworth
13. Hastings-Prince Edward
14. Huron
15. Kent-Chatham
16. Kingston-Frontenac-Lennox-Addington
17. Lambton
18. Leeds-Grenville-Lanark
19. Middlesex-London
20. Muskoka-Parry Sound
21. Niagara
22. North Bay and District
23. Northwestern
24. Ottawa-Carleton
25. Oxford
26. Peel
27. Perth
28. Peterborough
29. Porcupine
30. Renfrew
31. Simcoe
32. Sudbury and District
33. Thunder Bay
34. Timiskaming
35. Waterloo
36. Wellington-Dufferin-Guelph
37. York

Appendix C (continued). Ontario Public Health Units (PHUs) showing corresponding census divisions

Public Health Unit	Census divisions
Algoma	Algoma
Brant	Brant
Bruce-Grey-Owen Sound	Bruce
	Grey
Durham	Durham
Eastern Ontario	Prescott and Russell
	Stormont, Dundas and Glengarry
Elgin-St. Thomas	Elgin
Haldimand-Norfolk	Haldimand-Norfolk
Haliburton-Kawartha-Pine Ridge	Haliburton
	Northumberland
	Victoria
Halton	Halton
Hamilton-Wentworth	Hamilton-Wentworth
Hastings-Prince Edward	Hastings
	Prince Edward
Huron	Huron
Kent-Chatham	Kent
Kingston-Frontenac-Lennox-Addington	Frontenac
	Lennox and Addington
Lambton	Lambton
Leeds-Grenville-Lanark	Lanark
	Leeds and Grenville
Middlesex-London	Middlesex
Muskoka-Parry Sound	Muskoka
	Parry Sound
Niagara	Niagara
North Bay and District	Nipissing
Northwestern	Kenora
	Rainy River
Ottawa-Carleton	Ottawa-Carleton
Oxford	Oxford
Peel	Peel
Perth	Perth
Peterborough	Peterborough
Porcupine	Cochrane
Renfrew	Renfrew
Simcoe	Simcoe
Sudbury	Manitoulin Island
	Sudbury District
	Sudbury R.M.
Thunder Bay	Thunder Bay
Timiskaming	Timiskaming
Toronto	Toronto
Waterloo	Waterloo
Wellington-Dufferin-Guelph	Dufferin
	Wellington
Windsor-Essex	Essex
York	York

Appendix D. ICD Groupings and conversions for tobacco related causes of death

Cause of death	ICD7 1964-1968	ICD8 1969-1978	ICD9 (1979-present)
Ischaemic Heart Disease	420	410-414	410-414
Pulmonary Cardiac Disease			416-417
Cardiac Dysrhythmias	433-434	427	427
Heart Failure	422-434	428-429	428-429
Stroke/Cerebrovascular accidents	330-334	430-438	430-438
Arterial disease	450-456	440-448	440-448
Pneumonia & Influenza	480-493	470-486	480-486,487
Chronic obstructive pulmonary disease	501, 502	490-492	490-492
Chrohn's disease			555
Ulcerative colitis			556
Ectopic pregnancy	645	631	633
Spontaneous abortion			634
Pregnancy complications	643-644, 648	632	640-641
Stillbirths & perinatal deaths	750-762, 769-771, 773-776	740-779	740-779

Appendix E. Description of morphologic subgroups for lung cancer

Subgroup	ICDO-2 morphology code[1]
Carcinoma	8010-8572
Squamous cell carcinoma	8050-8076
Adenocarcinoma	8140-8143, 8211, 8230-8231, 8250-8260, 8290, 8310 8320, 8323, 8480-8490, 8550-8560, 8570-8572
Small cell carcinoma	8041-8045
Large cell carcinoma	8012-8031
Other specified carcinoma	8200, 8240-8246, 8430
Unspecified carcinoma	8010-8011, 8020-8022, 8032-8034
Sarcoma and Other specified types of cancer	8800-8811, 8830, 8840-8920, 8972, 8980-8981, 8990-8991, 9040-9044, 9120-9133, 9150, 9540-9581
Unspecified morphology	8000-8004

[1] Percy *et al.* 1990