Chronic Diseases in Canada

In this issue

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Announcement

New Associate Scientific Editors

We are pleased to announce that we have expanded the editorial staff of *Chronic Diseases in Canada* after a full year of being indexed by the National Library of Medicine in the *Index Medicus*/MEDLINE database. We welcome two Associate Scientific Editors.

- Dr Gerry B Hill, who is also a member of our Editorial Committee, will be responsible for the review of manuscripts that are epidemiological or biostatistical in nature.

- Dr Stephen B Hotz, who is Assistant Professor, Department of Epidemiology and Community Medicine, and School of Psychology at the University of Ottawa, will manage the review process for manuscripts with a behavioural science approach, including qualitative research.

Dr Christina Mills will continue as principal Scientific Editor.

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Monograph Series on Aging-related Diseases:
X. Prostate Cancer

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Abstract

Prostate cancer is the most commonly diagnosed cancer among Canadian men, excluding non-melanoma skin cancer. Prostate cancer incidence increases almost exponentially with age; most cases are diagnosed in men aged 65 years or older. With the possible exception of animal fat consumption, no known widespread modifiable risk factors have been identified. Although the prognosis is good if appropriate treatment occurs in the early stages of disease, the ability of existing early detection techniques to decrease mortality has not yet been demonstrated. The considerable economic and societal burden of prostate cancer and its treatment, coupled with the projected large increase in the number of new prostate cancer cases as the population ages, make this disease a very important public health issue.

Key words: *Canada; diagnosis; morbidity; mortality; prostatic neoplasms; risk factors; screening; treatment*

Introduction

This monograph on prostate cancer is the 10th in a series of aging-related disease monographs. From 1974 to 1993, over 80% of prostate cancer cases in Canada were diagnosed in men aged 65 and over; 90% of prostate cancer deaths from 1976 to 1995 also occurred in this age group.

The main focus of this paper is to review what is known of the etiology of prostate cancer. The paper also includes a description of the background and natural history of the disease; incidence, mortality and prevalence data for Canada; an examination of screening and diagnosis issues; and a brief section on prostate cancer treatment.

Background and Natural History

The prostate gland is a small, solid organ that lies at the neck of the bladder in males and surrounds the urethra.[1–3] At birth it weighs only a few grams; it increases in size until about age 20, when it reaches its adult weight of approximately 20 grams.[2,4,5] The gland starts to enlarge further at about the sixth decade of life.[1,2] This age-related increase is known as benign prostatic hypertrophy (BPH), which is a common cause of symptoms of urinary outflow obstruction, such as difficulty initiating urination, poor flow and increased frequency. The reader is referred to a previous monograph in this series[1] for more information regarding BPH.

Prostate cancer is often symptomless in its initial stages. When symptoms do develop because of significant localized disease, they are frequently indistinguishable from those caused by BPH. Metastatic disease is a cause of pain, especially bone pain.[6]

Two potential precursors of prostate cancer have been recognized: atypical adenomatous hyperplasia (AAH) and prostatic intraepithelial neoplasia (PIN).[7,8] PIN is an atypical proliferative disorder of the prostate gland[9] that can be either high- or low-grade. There has been some question as to the premalignant potential of AAH;[10] however, high-grade PIN, which may be detected on needle biopsy, has been identified by many as a main precursor.[9,11–15] While its natural history is not known,[16] there have been suggestions that PIN precedes carcinoma by several years.[17–19]

Author References

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Even once carcinoma develops, not all histologic prostate cancers become clinically significant during the life of a patient. Prostate cancer is found incidentally in at least 10% of men undergoing prostatectomy for BPH and in more than 40% undergoing cystoprostatectomy for bladder cancer.[20] A summary of autopsy series shows that the prevalence of latent histological prostate cancer is approximately 30% in men over the age of 50 who had no clinical problems during life.[20]

The intensity of diagnostic efforts in populations and individuals, therefore, is likely closely associated with detection rates, and this may partially explain why the clinical incidence of prostate cancer varies widely across international boundaries.[21] In Canada the observed lifetime incidence rate of prostate cancer has been about one third of the autopsy prevalence.[20] This observation gives rise to the oft-quoted expression that "more men die with prostate cancer than of it" and to the clinical dilemma of separating newly diagnosed cancers destined to behave aggressively from those destined to have a totally latent or relatively benign course.

Survival of the patient with prostate cancer is related primarily to the size and extent of spread of the tumour at the time of diagnosis, which is indicated by the stage. There are two systems generally used to stage prostate cancer. The modified Jewett system[22] describes the size and spread of the tumour from A through D. Substages of each of these further describe details of tumour progression. The American Joint Committee on Cancer uses the tumour, node, metastases (TNM) system[23–25] to stage prostate cancer. The Appendix describes both of these staging systems.

American statistics from the 1980s[26] show that 50–65% of prostate cancer cases were localized at diagnosis (clinical stages A and B), 9–17% had regional spread (stage C) and 20–25% were metastatic (stage D). The more recent use of new early detection techniques (see below) may have shifted these proportions toward earlier stages of disease. Similar data do not exist for Canada.

Besides stage, the prognosis of prostate cancer patients is also affected by the patient's age, existing co-morbid conditions, the histological grade of the tumour and tumour volume.[27–30] The degree of tumour differentiation reported by the pathologist, usually expressed as a Gleason grade,[31,32] has been found to be correlated with likelihood of metastatic spread present at diagnosis as well as with patient survival.[31] In general, the more poorly differentiated the tumour, the poorer the prognosis. Tumour volume correlates with local extent of disease, progression and patient survival, and penetration of the capsule appears to occur only in tumours larger than 1.4 cubic centimetres in volume.[30]

Stage A1 tumours (lesions involving less than 5% of a resected prostate, usually low-grade) have crude survival rates that generally mirror those for the general population.[33] Some of these tumours do progress, though very slowly, such that up to 35% of men will develop clinical problems within 15 years, and up to 12% of untreated patients will die of prostate cancer within a 5–10-year period.[34–36]

Reported crude five-year survival rates for untreated localized cancers range from approximately 80% for stage B1[37] to only 19% for stage B2 cancers.[33] This discrepancy is due, in part, to the fact that many putative stage B2 cancers are found at surgery to be understaged clinically and to have spread beyond the local capsule.[33]

Stage C tumours have penetrated the prostate capsule, usually into the seminal vesicle and neck of the bladder. Lymph node metastases occur in approximately 50% of these cases, and survival of untreated patients is reported to be 42–54% at one year, 22% at three years and 10% at five years. Roughly 75% of untreated patients with Stage D prostate cancer are thought to die within 9–16 months of diagnosis.[33]

Burden of Disease

Incidence

Though the number of small latent tumours seen in autopsy series seems to be rather consistent across countries and ethnic groups,[38,39] considerable international variation exists in the incidence of larger latent or clinically apparent prostate cancer tumours.[40] It is impossible to give accurate worldwide prostate cancer incidence estimates because, where they do exist, the quality of registration systems differs. Nevertheless, American estimates projected 334,500 new cases of prostate cancer in 1997.[41] American men, and African-Americans in particular, are reported to have the highest incidence of prostate cancer in the world (Figure 1). European rates are lower than those in the US,[42] and the lowest rates have been observed in Asia.[42,43] These variations may be partially due to differential use of diagnostic techniques[43] or currently unknown risk factors.

Incidence in Canada

Prostate cancer is the most commonly diagnosed cancer among Canadian men, excluding non-melanoma skin cancer.[44] In 1997 alone, almost 20,000 new cases were expected to be diagnosed,[44] and a recent report[45] estimated that the annual incidence of prostate cancer would reach 35,000 by the year 2016. It is estimated that about half of this projected increase will be due to the increasing incidence of the disease and the other half will be due to the increase in numbers of older men. The rising trend in incidence has been observed for many years; however, a dramatic increase has occurred since 1989 (Figure 2). These sharp increases in incidence have been mostly attributed to earlier detection.[46]
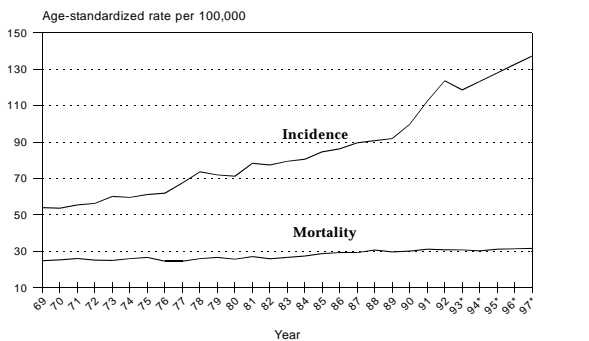
FIGURE 1
World-standardized prostate cancer incidence rates (per 100,000), 1983-1987, by country
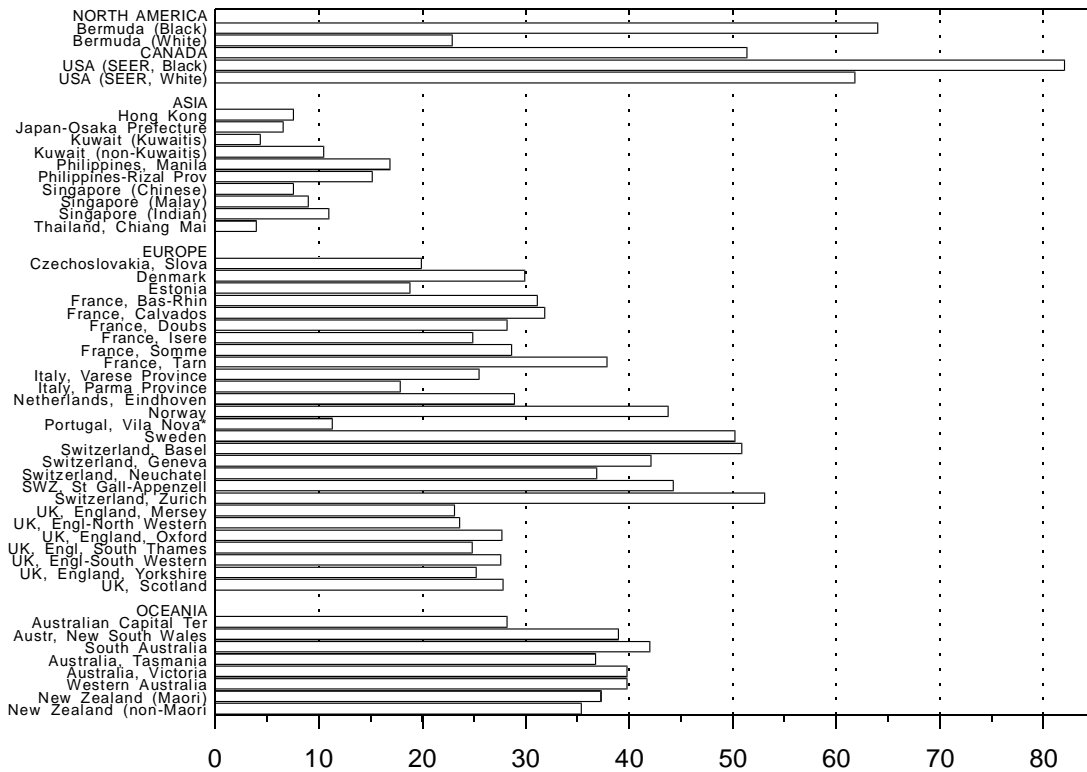
* Vila Nova de Gaia

Source: Laboratory Centre for Disease Control, based on data from Reference 42

FIGURE 2
Age-standardized prostate cancer incidence and mortality rates for Canadian men, 1969-1997

Age-standardized rate per 100,000

Incidence

Mortality

Year

* Estimated rates

Source: Reference 44

Table 1 shows the average annual incidence rates of prostate cancer over five-year intervals from 1974 to 1993. For all four periods, the incidence of prostate cancer increased with age, with at least a fivefold increase from ages 45–64 to 65–69 and more than a doubling of rates from ages 65–69 to 85 and over. While prostate cancer is very rare among Canadian males before age 45, incidence rises faster with age than for any other major cancer.[47] After age 45, incidence rates begin to grow in an almost exponential fashion. Whereas a Canadian male (at birth) has a 4.2% chance of developing prostate cancer by the age of 70, this increases to 9.5% by the age of 80.[44] Unlike lung or female breast cancer, prostate cancer does not reach a peak age of incidence in Canada before the age of 85.[47] The notable increase in new cases from one time period to the next is thought to be largely attributable to increases in the use of various techniques for detecting prostate cancer.[43,48,49]

Table 2 presents provincial variations in average annual incidence rates for prostate cancer in Canada during the same four periods as Table 1. With occasional exceptions, the annual incidence increased over time in every province. During each of the first three periods there appeared to be an east-west trend, with incidence in the Atlantic provinces

generally being relatively low. Some of the authors of this paper have shown previously[48] that this geographical gradient likely reflected differential detection rates related to variations in medical practice rather than differences in the prevalence of risk factors. After the advent of PSA testing (see Screening and Diagnosis section later in article) in approximately 1989, rates rose further throughout the country and those in the east began to approach those in the west.

Mortality

Prostate cancer mortality rates vary from country to country. High rates have been reported in the US, particularly among African-Americans; low rates, in China and Japan.[50,51] Mortality due to prostate cancer among African-American men has been found to be at least double that of Caucasian men,[41,49] and almost 10 times greater than that for men in Hong Kong and Japan.[52]

Mortality in Canada

Prostate cancer is the second leading cause of cancer death in Canadian men aged 65 and over, after lung cancer. It has been estimated that 1 in 27 men will die of prostate cancer.[44] In 1997, 4100 prostate cancer deaths were expected in Canada,[44] and by the year 2016, this number is estimated to reach about 7800.[45] Figure 2 shows the slow increasing trend in prostate cancer mortality since 1969.

The average annual mortality rates for prostate cancer from 1976 to 1995, age-adjusted to the 1991 census population, are displayed in Table 3. The mortality rate rose from 25.5 per 100,000 males in 1976–1980 to 27.2 in 1981–1985 and then to 30.7 in 1991–1995. As expected, mortality due to prostate cancer increased with age for all four time periods. While there was more than a sixfold increase in rates from ages 45–64 to 65–69, there was

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TABLE 1

Average annual incidence rates[a] for prostate cancer (ICD-9 185) by age and period, Canada, 1974–1993

Age (years)	Incidence per 100,000 population			
	1974–1978	1979–1983	1984–1988	1989–1993
ALL AGES	65.1	75.9	86.4	113.8
45–64	45.6	50.8	61.5	95.0
65–69	241.3	288.6	350.1	540.8
70–74	427.4	498.7	577.1	815.7
75–79	620.9	721.2	808.8	1016.6
80–84	827.4	927.0	1007.2	1157.2
85+	898.1	1120.1	1191.9	1174.6

a   Standardized to the 1991 Canadian population
Source: Laboratory Centre for Disease Control, based on data from Statistics Canada

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TABLE 2

Average annual incidence rates[a] for prostate cancer (ICD–9 185) by province and period, Canada, 1974–1993

Province	Incidence per 100,000 population			
	1974–1978	1979–1983	1984–1988	1989–1993
Newfoundland	49.6	47.6	58.4	75.3
Prince Edward Island	59.0	66.0	73.2	124.8
Nova Scotia	60.0	60.5	81.4	108.5
New Brunswick	65.0	66.9	88.3	127.0
Quebec	55.8	74.7	84.4	98.1
Ontario	66.0	71.8	81.0	110.5
Manitoba	71.9	77.3	92.7	142.3
Saskatchewan	81.3	98.6	89.2	119.2
Alberta	67.7	80.7	90.3	112.8
British Columbia	72.1	88.9	106.0	143.8
CANADA	65.1	75.9	86.4	113.8

a   Standardized to the 1991 Canadian population
Source: Laboratory Centre for Disease Control, based on data from Statistics Canada

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TABLE 3

Average annual mortality rates[a] for prostate cancer (ICD-9 185) by age and period, Canada, 1976–1995

Age (years)	Deaths per 100,000 population			
	1976–1980	1981–1985	1986–1990	1991–1995
ALL AGES	25.5	27.2	29.9	30.7
45–64	9.9	10.4	11.8	11.1
65–69	61.1	66.9	75.3	79.1
70–74	135.7	141.4	162.5	152.2
75–79	240.6	264.2	275.0	286.4
80–84	420.5	413.5	462.8	477.1
85+	601.9	678.8	724.8	793.7

a   Standardized to the 1991 Canadian population
Source: Laboratory Centre for Disease Control, based on data from Statistics Canada

roughly a tenfold jump in rates from ages 65–69 to 85 and over.

Prostate cancer mortality varies by province, as seen in Table 4. From 1976–1980 to 1986–1990, Newfoundland had the lowest provincial rate, while the lowest rate for 1991–1995 was in British Columbia. From 1976 to 1995, Prince Edward Island consistently displayed high five-year average mortality rates for prostate cancer compared to the other provinces. All provinces showed growth in mortality rates over the four time periods, except Quebec, Manitoba and British Columbia, where a slight drop was seen from 1986–1990 to 1991–1995.

TABLE 4

Average annual mortality rates[a] for prostate cancer (ICD-9 185) by province and period, Canada, 1976–1995

Province	Deaths per 100,000 population			
	1976–1980	1981–1985	1986–1990	1991–1995
Newfoundland	19.2	19.7	26.0	30.8
Prince Edward Island	28.9	30.4	32.8	40.5
Nova Scotia	28.6	28.4	31.5	34.1
New Brunswick	26.3	27.6	27.6	30.9
Quebec	26.2	28.9	31.2	30.6
Ontario	24.5	26.1	28.9	30.1
Manitoba	27.4	28.2	33.2	32.5
Saskatchewan	26.1	30.3	31.2	33.9
Alberta	26.6	28.0	29.7	32.2
British Columbia	25.4	26.4	29.4	28.9
CANADA	25.5	27.2	29.9	30.7

[a]   Standardized to the 1991 Canadian population
Source: Laboratory Centre for Disease Control, based on data from Statistics Canada

Prevalence in Canada

It is difficult to obtain exact prevalence estimates of prostate cancer because of the uncertain natural history[53,54] and known high prevalence of latent disease.[53,55–57] One paper reported the prevalence of prostate cancer (diagnosed between 1975 and 1989, and patients still alive at the end of 1989) to be 45,500.[58] Another source estimated the number of prostate cancers diagnosed from 1986 to 1990 (five-year prevalence) in patients still living in 1990 to be 34,400 and the ten-year prevalence (diagnosed from 1981 to 1990, and still alive in 1990) to be 48,100.[59] However, these latter figures may be underestimates since they do not include

prostate cancer cases diagnosed prior to 1981. The prevalence of this disease is expected to climb rapidly in the 1990s due to the previously described increase in incidence, assuming the absence of any major increase in mortality.

Risk Factors

We reviewed the literature to summarize current knowledge of potential risk factors for prostate cancer. References were identified through MEDLINE and a review of article bibliographies. Only English-language papers were considered.

Family History

Using genealogical records, Cannon et al.[60] found prostate cancer to have a stronger familial aggregation than either colon or breast cancer. First-degree relatives of prostate cancer cases have been shown to experience statistically significantly increased risks that approach 2.5.[61–64] The risk has been reported as higher in blacks (odds ratio [OR] = 3.2) than in whites (OR = 1.9), though the difference was not statistically significant.[63] The closer genetically a man is to an affected relative[61,62] and the more relatives he has with the disease,[61] the greater his risk. Men with three affected relatives were at an 11-fold risk.[61]

At least two Canadian studies have found evidence for a familial role in prostate cancer development.[65,66] A population-based case-control study conducted in Quebec involving 140 Francophone hospital in-patients detected an OR of almost 9 for men with one to four first-degree relatives with prostate cancer.[65] McLellan and Norman[67] have speculated that this large OR may be due to the investigators' not limiting their calculations to cases with one or two affected relatives, as had been the practice in previous studies. In the other Canadian report, also a population-based case-control study, Fincham et al.[66] used the Alberta Cancer Registry to identify 382 prostate cancer cases. They reported that subjects with an affected first-degree relative were more than three times as likely to develop prostate cancer than those without one.

A segregation analysis by Carter et al.[68] revealed that a form of "hereditary" prostate cancer is the result of an autosomal-dominant inheritance of a rare high-risk gene that predisposes men to the early development of prostate cancer. Another segregation analysis conducted in Sweden confirmed the importance of an autosomal-dominant gene.[69] Subsequent research has identified chromosome 1q24-25 as containing a gene, HPC1, involved in the development of hereditary prostate cancer.[70,71] While hereditary prostate cancer may account for a significant proportion of early onset prostate cancer, the data of Carter et al.[68] suggest that, overall, only about 9% of this disease in the population is due to the effects of the hereditary prostate cancer gene. Although the large majority of prostate cancers, especially among the elderly, appear to result from environmental factors, genetic predisposition is

likely to play a role in the etiology of many prostate cancer cases.

Hormones

Sex hormones, androgens in particular, may play a role in prostate cancer development. Androgens are required for the growth, maintenance and functional activity of the prostate gland.[72] In addition, prostate cancer growth rates can be manipulated through hormonal therapy.[73] Research has suggested that the progression of prostate cancer from histological to clinically significant forms may be partially the result of an altered hormone metabolism.[74]

The principal androgenic hormone in men is testosterone.[72] It has been hypothesized that elevated levels of both testosterone and its active metabolite, dihydrotestosterone, may, over many decades, lead to prostate cancer.[75] Ross et al.[76] found that young African-American men had higher serum testosterone levels than white American men and suggested that the difference could explain the increased prostate cancer risk experienced by the former group. However, prostate cancer risk has not been found to be associated with prediagnostic levels of serum testosterone or serum dihydrotestosterone.[77–80]

The results of Ross et al.[81] raised the possibility that reduced activity of 5-alpha-reductase, the enzyme in the prostate that converts testosterone to dihydrotestosterone, is involved in the low prostate cancer incidence rates observed among Japanese men. Meikle et al.[82] reported that men with prostate cancer had elevated clearance rates of testosterone and an increased conversion ratio of testosterone to 5-alpha-reduced metabolites.

Ethnic Group / Country of Residence

The highest incidence rates for prostate cancer are found among African-American men.[83] Their incidence rates are 1.5 to almost 2 times those for Caucasian-American men, though rates for the latter group are among the highest in the world. High incidence rates are also found in Canada and northern Europe, while very low rates originate from countries in eastern Asia such as Japan and China. Prostate cancer is much more common in developed than developing countries, and the global range of difference in incidence is at least 70-fold.

Several migrant studies have found that prostate cancer rates shift toward those of the host country. Early studies by Haenszel and Kurihara[84] and Locke and King[85] found the rates among Japanese-Americans to be intermediate between the very low rates of Japanese men in Japan and the high rates among white males in the US. More recent studies concur with these results; Yu et al.[86] and Stellman and Wang[71] found white males in the US to have considerably higher prostate cancer rates than Chinese men in China, with Chinese-Americans having intermediate rates. These outcomes suggest that the underlying cause of disease is related, at least in part, to environmental factors.

Socio-economic Status

Whether or not low socio-economic status is a risk factor for prostate cancer has been difficult to test because ethnic minorities are overrepresented in low socio-economic groups in many studies. While both positive and negative results have been found, in general, the data support the concept that socio-economic status is not an important risk factor in the development of prostate cancer.[74]

Occupation

Many industries, occupations, and work-related exposures have been studied in relation to prostate cancer. However, the focus has primarily been on cadmium exposure, work in the rubber industry and farming. Farming was associated with increased risk of prostate cancer in 17 of 24 studies examined in a 1991 review.[87] In 10 of these studies the results were statistically significant. In a retrospective cohort study, Morrison et al.[88] found an association between number of acres sprayed with herbicides and risk of prostate cancer mortality after 17 years of follow-up. The National Academy of Science's committee to review the health effects of exposure to herbicides in Vietnam veterans concluded that there was limited suggestive evidence linking herbicide exposure to prostate cancer.[89]

Analyses based on the rubber industry as a whole have found both positive and negative associations with prostate cancer. The International Agency for Research on Cancer decided that, while there was "limited" evidence for an excess occurrence of prostate cancer in rubber workers, the data were inadequate to establish a causal association.[90]

A review of studies conducted to determine whether exposure to cadmium places a man at greater risk for prostate cancer concluded that cadmium exposure may weakly increase risk.[74] Some research suggests that cadmium interferes with the zinc-hormone relationships in the prostate.[91] Zinc is required by several enzymes involved in the replication and repair of DNA and RNA, and the prostate contains the highest concentration of zinc of any organ in the body.[92] As occupational exposures to zinc and cadmium usually occur together, it is difficult to evaluate their separate or interactive effects.[93] Elghany et al.[93] failed to find an increased risk of prostate cancer among welders or electroplaters, even though people working in such jobs experience high levels of cadmium exposure.

Physical Activity

It has been proposed that physical activity may lower both body fat and testosterone levels and, hence, possibly reduce prostate cancer risk for men who are very active.[94,95] The results to date, however, have been conflicting. Studies have reported that highly physically active men experience decreased,[94–97] increased[2,98–100] or similar[101,102] risks of prostate cancer compared with inactive men.

Research into the relation between occupational exercise and prostate cancer tends toward finding a protective effect for more physically active jobs. Recently conducted studies in China[95] and Turkey[103] indicated that individuals who worked in sedentary jobs were at an increased risk for prostate cancer. The results were independent of whether physical activity was measured by total energy expenditure or percentage of occupational time spent sitting. Two other studies have also reported an inverse association with occupational physical activity.[104,105] However, a study of the lifetime occupational physical activity levels among Hawaiian men concluded that physical activity may be positively associated with the risk of prostate cancer.[106]

Anthropometry

The evidence for an association between high body mass index (BMI) and prostate cancer risk is very limited. In a case-control study of 48–79-year-olds conducted in northern Italy, Talamini et al.[107] observed that the risk of being diagnosed with prostate cancer rose with increasing BMI. The OR for men in the highest group (BMI ≥ 28) was nearly 4.5 times that of the reference group (BMI < 23). Studies of Japanese (relative risk [RR] = 1.33), Dutch (OR = 1.5) and Seventh-Day Adventist men (RR = 1.17) have all reported elevated, though non-significant, risk estimates.[108–110] On the other hand, a cohort study of over 20,000 men of various ethnicities in Hawaii[111] found high BMI to be slightly protective (RR = 0.7; 95% confidence interval [CI] = 0.5–1.2), while several other studies found no difference in mean BMI between cases and controls.[106,112–115]

It has been suggested that previous findings of positive associations between BMI and prostate cancer might be accounted for more by muscle mass than by fat tissue.[108,116] Severson et al.[108] found the muscle, not the fat area, of the upper arm to be significantly related to prostate cancer risk. Increased muscle mass may be a marker for higher levels of androgens.[72]

Diet

A dietary etiology for prostate cancer is consistent with the descriptive epidemiology, including observations on migrants, geographic variations and temporal trends, making it a promising area of research.[117] A high positive correlation has been reported between prostate cancer incidence rates and the corresponding rates of several other cancers thought to be related to diet (e.g. breast and colon cancers).[118] However, epidemiologic studies have not provided consistent evidence concerning the relation between specific dietary factors and prostate cancer risk.[119]

Energy intake

A significant positive association between energy intake and risk of prostate cancer has been reported in at least three case-control studies.[119–121] In one study,[121] the association was stronger for advanced prostate cancer (fourth quartile versus first quartile RR = 1.70; 95% CI = 1.10–2.61) while in another,[120] the effect was restricted to older men (68–74 years old), particularly those with

aggressive tumours. In three other case-control studies,[101,122,123] including one with information on tumour aggression,[101] energy intake was unrelated to prostate cancer risk. This was also the finding in a cohort study conducted by Severson et al., though the result was based on only a 24-hour food recall assessment.[102]

While several possible mechanisms have been proposed,[119,121] including an alteration of the activity of the sympathetic nervous system,[121] the interpretation of a positive association between energy intake and prostate cancer risk is unclear as differences in energy intake between individuals are largely determined by differences in physical activity, body size and metabolic efficiency.[124]

Fat intake

Ecological correlation studies from the 1970s showed strong positive associations between prostate cancer incidence or mortality and fat consumption among a number of countries and across the US.[125–127] Based on a correlation coefficient of 0.74 between national consumption levels of fat and national mortality rates of prostate cancer, Armstrong and Doll[125] hypothesized that dietary fat may be a major cause of prostate cancer.

Many case-control studies have examined the association between fat and prostate cancer,[101,107,113–5,119,120,122,123,128–32] though only five[101,119,120,122,131] adjusted for energy intake. The 14 studies differed in terms of study design (hospital or population controls) and method of dietary assessment (direct or indirect). In some cases, fat intake was inferred from the frequency of consumption of meat, dairy products and other foods known to have a high fat content.[107,128–30,132] Other studies assessed fat intake in a more comprehensive manner using food composition data to approximate actual fat intake.[101,113–5,119,120,122,123,131] Despite these methodological differences, only four studies[119,122,123,130] failed to show a positive association with total fat intake.

The association between fat intake and prostate cancer risk has also been explored in at least eight cohort studies,[102,110,111,133–7] the most methodologically sound of which was conducted by Giovannucci and colleagues.[133] Measuring fat intake as a nutrient and adjusting for energy intake, the only cohort study researchers to do so, they observed a significant positive association between increased fat intake and risk of advanced prostate cancer. A positive association between consumption of foods high in fat and subsequent risk of prostate cancer has been reported in three studies.[110,111,134] While two other studies did not detect an association,[135,136] both had limited food frequency data. Severson et al.[102] detected a weak association with eggs and with margarine, butter and cheese as a group but not with fat as a nutrient, though this was only measured as part of a 24-hour food recall survey.

With respect to specific components of fat, Giovannucci et al.[133] and Gann et al.,[137] who measured plasma fatty

acids, reported similar results. Both found a strong positive association between ∝-linolenic acid, an essential polyunsaturated fatty acid, and prostate cancer risk; no clear linear relation across quartiles of exposure, suggesting a threshold effect; that low levels of linoleic acid, another polyunsaturated fatty acid, may further exaggerate the effect; and an independent association with red meat but no association with dairy foods. The findings with regard to polyunsaturated fat are supported by two case-controls studies.[120,122]

Dietary fat intake has been more consistently linked to prostate cancer than any other modifiable risk factor. Evidence of an association appears to be strongest for ∝-linolenic acid and among advanced stage cases. However, a causal mechanism has yet to be established.

Vitamin A

Vitamin A is a generic term for all substances that possess the biologic properties of retinol.[138] It may be ingested either as a preformed vitamin or as a provitamin.[139] The relation between intake of preformed vitamin A, naturally found only in food from animal sources,[139] and prostate cancer has been specifically examined in at least seven studies. In five of these studies[129,135,140–2] a positive association was reported, although in two of these studies[135,140] the effect was restricted to a certain age range. Slightly decreased risks with increased consumption were found in two related studies;[119,122] however, study response rates were low.

The results of published reports examining the relation between serum vitamin A or serum retinol and prostate cancer have been mixed.[143–147] An increased risk of prostate cancer was associated with lower serum retinol levels in a hospital-based case-control study conducted in the Netherlands.[143] However, a treatment effect or an effect from the disease process itself could not be easily dismissed; low serum retinol levels may be a metabolic consequence of cancer rather than a precursor.[148] The findings from three nested case-control studies differed with regard to serum retinol and prostate cancer incidence. One study suggested an inverse relation,[144] a study of Japanese-Americans in Hawaii reported no association,[149] while a weak positive association was observed in the third,[145] which was based on only 32 prostate cancer cases.

Using data from the National Health and Nutrition Examination Survey, Reichman et al.[146] reported an increased risk of developing prostate cancer for men with a serum vitamin A level in the lowest quartile compared to those with a level in the highest quartile (RR = 2.2; 95% CI = 1.1–4.3). However, in the Nutrition Canada Survey cohort,[147] men with a serum vitamin A level in the highest quartile were found to be at increased risk (RR = 2.0; 95% CI = 1.1–3.5). Reasons for the discrepant results are not readily apparent. The two cohort studies were similar in many respects, including the time period of the study, the length of follow-up, the overrepresentation of elderly and low-income individuals, and the adjustment for the confounding effects of age.

Provitamin A originates from a small percentage of the various carotenoids found in plant sources.[139,150] Because associations related to carotenoids do not necessarily imply a mechanism involving conversion to vitamin A,[117] studies that have only used an index of vitamin A that combines the dietary intake of preformed and provitamin A[114,131,151] are difficult to interpret. Since most carotenoids, including those with provitamin A activity, can also act as singlet oxygen quenchers and as antioxidants under certain conditions,[150] studies relating carotenoids to prostate cancer are reviewed in the next subsection.

Antioxidants

The relation between dietary intake of carotenoids (primarily β-carotene) and risk of prostate cancer has been extensively investigated in both case-control[107,113,119,120,122,123,129,130,132,140,152] and cohort[102,110,134,135,141,142,153,154] studies. Though several of the above-mentioned studies looked at the consumption of fruits and vegetables, both individually and as food groups, the majority were nutrient-based. Nutrient-based studies are preferred because they protect against the potential confounding effects of other nutrients contained in the same food item;[131] such studies have reported positive,[129] negative[113,123,130] and null[119,141,142,153] associations. In two reports the direction of the association was found to differ by the age group studied.[120,135] Serum β-carotene has been shown to be positively associated with prostate cancer risk[145] in one study, but to have no association in two others.[143,144]

Lycopene, a non-provitamin A, is the most efficient scavenger of singlet oxygen among the common carotenoids[155] and is the predominant carotenoid in prostate gland tissue.[156] Tomato-based products or lycopene (the major dietary source of which is tomatoes)[157] have been reported to reduce prostate cancer risk in several studies.[110,132,141,144]

In a recent prospective study of nearly 50,000 health professionals, Giovannucci et al.[141] observed a protective effect for frequent consumption (i.e. more than 10 servings a week versus less than 1.5 servings) of tomatoes, tomato sauce, tomato juice, and/or pizza (RR = 0.65; 95% CI = 0.44–0.95) and an inverse relation between lycopene intake and prostate cancer risk (RR = 0.79; 95% CI = 0.64–0.99). An inverse association (OR = 0.50), particularly among men younger than age 70 (OR = 0.35), was also noted in a nested case-control study that examined prediagnostic plasma lycopene levels.[144] Intake of tomatoes was significantly related to lower risk of prostate cancer in a cohort study of Seventh-Day Adventists[110] and non-significantly related in a case-control study.[132]

Only a sparse body of literature exists concerning relations between prostate cancer and other antioxidants such as selenium and vitamin C. In a recent randomized

controlled trial whose original end-points were incidences of basal and squamous cell carcinomas,[158] selenium (a surrogate for the selenium-containing antioxidant enzyme called glutathione peroxidase)[159] supplementation was found to be associated with a significant reduction in prostate cancer incidence (RR = 0.37; 95% CI = 0.18–0.71). Previously conducted studies using prediagnostic serum selenium levels had not reported a significant association,[160,161] though one study included only 11 prostate cancer cases.[161]

The majority of studies that reported on vitamin C and prostate cancer risk found no effect.[66,115,121,123,131,153,162–4] An exception was the study by Graham et al.[114] that noted a positive association (OR = 2.32, trend $p < 0.01$) that was enhanced among men over age 70 (OR = 3.41, trend $p < 0.05$). Two other studies[115,120] reported elevated, though non-significant, risk estimates of approximately 40–50% among subjects in the highest quartile of vitamin C intake as compared to those in the lowest quartile.

In summary, with the possible exception of lycopene, there is little evidence that prostate cancer risk varies with consumption of dietary antioxidants.

Vitamin D

It has been recently hypothesized[165,166] that vitamin D deficiency may be a risk factor for prostate cancer. Using a nested case-control design, Corder et al.[165] found that lower prediagnostic serum levels of 1,25-dihydroxyvitamin D (1,25-D), a vitamin D metabolite, were significantly associated with an increased risk of clinically detected prostate cancer, particularly in men with low levels of 25-dihydroxyvitamin D (OR = 0.41). In an extension of this study, the observed protective effect was attributed to seasonally lower summer levels of 1,25-D in case subjects.[167] A subsequent nested case-control study,[168] however, failed to support these findings. Higher levels of either 1,25-D or 25-dihydroxyvitamin D were not associated with a reduction in prostate cancer risk, though a non-significant inverse association (OR = 0.67) was observed among men simultaneously in the highest quartiles of both metabolites relative to those simultaneously in the lowest. A smaller study of prediagnostic serum vitamin D metabolite levels,[169] also failed to support the findings of Corder et al.[165]

It has been suggested that the potential protective effects of 1,25-D may be restricted to the biologically active free 1,25-D.[170] Free 1,25-D can be estimated by dividing total 1,25-D concentration by vitamin D-binding protein concentration.[171] A case-control study conducted by Schwartz et al.[172] reported that men with prostate cancer had significantly lower serum levels of free 1,25-D. In contrast, Corder et al.[167] did not find a lower free 1,25-D serum concentration in men with prostate cancer, while Gann et al.[168] reported free 1,25-D to be reduced (though not significantly) among prostate cancer cases older than 61 years (OR = 0.65). Further research into a relation between vitamin D metabolites and prostate cancer is necessary.

Alcohol

A biologically plausible protective role for alcohol in prostate carcinogenesis originated from research reporting that alcohol may increase metabolic clearance of testosterone.[173] However, virtually all studies conducted have demonstrated an absence of any overall relation.[98,110,111,113,128,135,174–81] One exception was a recent case-control study[182] wherein significantly elevated risks were seen for those who had 22–56 drinks per week (OR = 1.4; 95% CI = 1.0–1.8) and 57 or more drinks per week (OR = 1.9; 95% CI, 1.3–2.7) in comparison to never-users.

Smoking

There have been many case-control studies concerning cigarette smoking and prostate cancer,[66,112,113,128,152,174,176,177,180,183–94] only five of which reported a statistically significant association[180,183,185,192] or "marked" disparity in the proportion of smokers between cases and controls.[184] The lack of an association in many of these studies may be partly due to the use of hospital patients as controls; the controls used in all five positive studies cited above were population-based. Despite reporting increased risks for current (OR = 1.5; 95% CI = 1.0–2.4) and former (OR = 1.4; 95% CI = 1.0–1.5) smokers of 40 or more cigarettes per day, the lack of consistent findings in population subgroups and the lack of a clear trend in effect led Hayes et al.[192] to doubt the existence of a causal association.

Early cohort studies of cigarette smoking and prostate cancer mortality[98,135,136,195–7] were relatively small and, with one exception,[135] did not observe an association among former or current cigarette smokers in comparison to never-smokers. Though they reported an 80% increase in risk, Hsing et al.[135] found no evidence of a trend in effect. Three of the studies[98,195,197] also considered the number of cigarettes smoked by current smokers, but still found no association.

Since 1991, results have been published from four large cohort studies that each observed in excess of 500 prostate cancer deaths. An overall statistically significant increase in risk in the range of 20–35% was reported in three of these studies.[198–200] In one case,[199] a trend in effect was noted, the highest risk being experienced by those who smoked 40 or more cigarettes per day (RR = 1.5; 95% CI = 1.2–1.9). A lesser effect was found for former smokers (RR = 1.13; 95% CI = 1.03–1.24). The fourth study,[201] a 40-year follow-up of nearly 35,000 British male doctors, found prostate cancer mortality rates to be virtually identical between current and never-smokers.

Cohort studies of cigarette smoking and prostate cancer incidence have produced mixed results. While three studies,[102,110,202] including a very large Norwegian one,[202] reported no association, two others detected a statistically significant positive relation.[175,181] In the Iowa 65+ Rural Health Study,[181] those who smoked 20 or more cigarettes per day experienced a nearly threefold increase in risk

relative to non-smokers. The positive association reported by Hiatt et al.[175] was limited to those who smoked more than one package of cigarettes per day.

In early 1996, participants of an international consensus conference on smoking and prostate cancer unanimously agreed that there was inadequate evidence that smoking is associated with prostate cancer incidence.[203] The inconsistent results of the incidence studies combined with the findings of the large cohort analyses of mortality have led Rodriguez et al.[200] to suggest that smoking may adversely affect survival in prostate cancer patients.

Sexual Activity

Although extensively studied, the role of sexual activity in the development of prostate cancer is still unclear. Both hormonal factors and infectious agents have been proposed as increasing prostate cancer risk. Key[204] summarized a number of studies and found the relative risks for early first intercourse, large number of sexual partners and a history of any sexually transmitted disease to be elevated. However, it has also been reported that celibate men develop prostate cancer as frequently as the general population.[205]

Vasectomy

Studies examining the relationship between vasectomy and prostate cancer have yet to demonstrate a pattern. Giovannucci and colleagues found significantly elevated relative risks of approximately 1.6 in both a retrospective[206] and a prospective cohort.[207] However, Sidney[208] found no association, a result that was confirmed in a second report based on additional years of follow-up.[209] In a very large multi-ethnic case-control study conducted in the US and Canada, a history of vasectomy was not significantly associated with prostate cancer risk.[210] A similar conclusion had been reached in three previous reports.[211–213]

Though their study included only five prostate cancer cases who reported a history of vasectomy, Ross et al.[214] found vasectomy to be associated with lower risk. On the other hand, a study conducted in China[215] reported a strong positive association using neighbourhood controls, while Rosenberg et al.,[216] as part of a hypothesis-generating exercise, found large risk estimates regardless of whether cases were compared to cancer or non-cancer controls. Other case-control studies have reported increased risks ranging from 40% to 70%.[185,217,218]

A major concern in the study of vasectomy and prostate cancer has been detection bias.[210] Vasectomized men may be more likely to subsequently visit a urologist, resulting in an increased chance of being diagnosed with prostate cancer.[219] In addition, while most studies have used self-reported history of vasectomy, no study to date has validated this against medical records.[210] Many studies have also used self-reported disease status, though it has been suggested that a history of prostate cancer is not always accurately reported.[220] In a review of possible mechanisms, Howards[221] concluded that it seems highly

unlikely that there is a biological mechanism supporting a relationship between vasectomy and prostate cancer.

Screening and Diagnosis

As highlighted in the previous section, modifiable risk factors have not been clearly established, so effective measures to prevent the occurrence of prostate cancer do not exist at this time. As a consequence, much attention has focused on the use of early detection measures to control this disease. Diagnosis of the cancer is usually made by histologic examination of tissue derived from a needle biopsy of the gland. Tests used to aid in the diagnosis include the digital rectal examination (DRE), transrectal ultrasound (TRUS) and serum prostate-specific antigen (PSA). Controversy exists about the appropriateness of using these tests as screening modalities in asymptomatic men, mainly because it is not known if early detection can actually influence the natural history and outcome of the disease.

The DRE is the most commonly used test, though it has not undergone systematic evaluation of its efficacy and it is not known whether routine annual screening by DRE reduces prostate cancer mortality.[222,223] A DRE may not be able to detect small tumours formed in certain sections of the prostate gland, and the quality of the test depends on the skill and experience of the examiner.[26] TRUS is generally thought not to be an appropriate screening test, primarily because of its low sensitivity and specificity, its invasiveness and its cost. It is generally used as a confirmatory diagnostic test and aid to biopsy when DRE or PSA tests indicate the possibility of a tumour.[26]

PSA is a protein found in prostate epithelial cells and secreted into seminal fluid. It can be detected in serum using immunoassays; serum levels are increased in the presence of both BPH and prostate cancer.[224] PSA levels are routinely monitored in patients *after* treatment to assess risk of relapse and treatment success,[225] but, because of the test's simplicity, low cost and independence of examiner's skill, it is currently receiving much attention as a promising test for the early detection of prostate cancer.[226,227] There is evidence that use of PSA increases the detection of early stage prostate cancers.[226,228–231] Catalona et al.[228] found that PSA-detected tumours were organ-confined in 51% of cases versus 34% detected by DRE, focal penetration of the capsule occurred in 15% of PSA-detected cancers versus 23% detected by DRE, extensive capsular penetration occurred in 24% versus 43%, positive seminal vesicles occurred in 6% versus 14% and positive lymph nodes occurred in 4% versus 7%.

The sensitivity of PSA in detecting prostate cancer is thought to be between 70% and 80%,[226,232,233] which means that approximately one man in four with prostate cancer will miss having a diagnosis made when PSA is used to screen an asymptomatic population. The positive predictive value of the PSA test in detecting prostate cancer has been reported to range from 28% to 35%.[226,228,232] This reflects a

low specificity due, in part, to the presence of elevated PSA levels among men with other prostatic conditions such as BPH. As a consequence, approximately three out of four men with an elevated PSA will not have prostate cancer confirmed upon further diagnostic workup.[226] While detecting many cancers early, this widespread use of PSA results in large numbers of unnecessary biopsies and in several missed cancers. Further, there is currently no evidence that screening for prostate cancer with PSA will reduce mortality from the disease. Randomized controlled trials are needed to avoid biases inherent in observational studies (e.g. selection, length and lead-time biases). There are ongoing studies in the US and Europe,[234,235] but these will not yield definitive results until well into the next decade.

At present, there is disagreement as to the appropriateness of the PSA test for routine screening of the general population. Table 5 outlines the screening guidelines for prostate cancer issued by North American organizations. Evidence-based groups such as the Canadian Task Force on the Periodic Health Examination and the US Preventive Services Task Force do not recommend routine use of PSA as a screening tool for prostate cancer. Neither of these groups feel that current evidence warrants its use in the general population to detect prostate cancer, primarily because of its relatively low specificity and the possibility of detecting indolent tumours that would not progress.[236,237]

Treatment

A variety of treatment modalities are used to try to control prostate cancer. Radical prostatectomy (preferably a nerve-sparing procedure, which is thought to have lower rates of associated side effects) or radiation therapy has curative intent in men with localized cancers. Hormonal cytoreductive therapy, using anti-androgen products, is sometimes used as an adjunct in these men too. Local radiotherapy (for regional disease) and partial or total androgen blockade (achieved through chemical or surgical castration) constitute the main treatments for advanced disease. Surgery may be used to assist in staging. The usefulness of hormonal treatments, or anti-androgens, may be enhanced by strategies employing intermittent

TABLE 5

Prostate cancer screening guidelines, Canada and the US

Organization	Guideline/recommendation	Comments
Canadian Task Force on the Periodic Health Examination , 1994	The Task Force does not recommend the routine use of PSA or DRE as part of a periodic health examination.	
US Preventive Services Task Force, 1996	Routine screening for prostate cancer with DRE, serum tumour markers or TRUS is not recommended.	
US National Cancer Institute, 1997	There is insufficient evidence to establish whether a decrease in mortality from prostate cancer occurs with screening by DRE, TRUS or serum markers including PSA.	
American Cancer Society, 1992	Annual PSA for men > 50 years Annual DRE for men > 40 years	Annual PSA if younger than 50 and in high risk group until life expectancy is less than 10 years
Canadian Workshop on Screening for Prostate Cancer, 1994	No PSA for screening unless for a screening trial or patient request after pre-test counselling and informed consent.	
Canadian Urological Society, 1996	The DRE and PSA measurements increase the early detection of clinically significant prostate cancer. Men should be made aware of the potential benefits and risks of early detection so that they can make an informed decision as to whether to have this test performed.	
American Academy of Family Physicians, 1996	Men aged 50–65 should be counselled about the known risks and uncertain benefits of screening for prostate cancer.	
American College of Radiology, 1995	Every man 40 and older should have an annual DRE and at age 50, an annual PSA.	
American Urological Association, 1995	Annual DRE and PSA measurements substantially increase the early detection of prostate cancer. These tests are most appropriate for men 50 and older and for those 40 or older who are at high risk. PSA testing should continue in a healthy male who has a life expectancy of 10 years or more.	

administration. Bone pain may respond specifically to parenteral strontium.

An extensive review of stage-specific treatment approaches is beyond the scope of this Monograph Series. The reader is referred to existing comprehensive reviews.[20,27] Although there is general agreement on some parameters for initial treatments of men with various stages of the cancer, major areas of uncertainty exist. These include whether surgery, radiation or delayed therapy is best in early stage disease, whether androgen blockade is warranted in minimal metastatic disease and how best to manage advanced disease resistant to anti-androgen therapy. All active treatments carry an associated morbidity—for example, impotence (at least 20–40%) and incontinence (5–25%) are common with both radiation and surgery for early stage disease,[238] and erectile dysfunction is certain when hormonal treatments are used for later stage cancers.

Thus men with prostate cancer face several uncertainties regarding treatment options, all of which carry significant attendant risks of negative health effects.

Conclusions and Recommendations

Prostate cancer control is problematic. In part because prostate cancer incidence and mortality vary dramatically internationally and because of the findings of migrant studies, there is widespread belief that behavioural factors play a key role in the etiology of prostate cancer. Unfortunately, with the possible exception of animal fat consumption, no known widespread modifiable risk factors have been identified, notwithstanding numerous epidemiology studies of prostate cancer. Why has epidemiology failed?

One possibility is that epidemiology is a relatively crude tool to examine, what may prove to be, an unusually complex etiology. Most epidemiologic studies of prostate cancer have considerable problems with both exposure and disease characterization. Perhaps an understanding of the interplay between many genetically determined factors (such as 5-alpha-reductase) and environmental factors (such as dietary fat, vitamin A and cigarette smoking) will be necessary before consistency is achieved across epidemiologic studies. Appropriate staging information on prostate cancer cases, absent in most epidemiologic studies, should allow for the control of biases that may occur from the mixing of clinically inconsequential cancers from those that may provide etiologic clues.

Screening remains controversial, as does whether or not treatment extends or improves quality of life. Clearly, further research efforts are warranted. To address the issues concerning prostate cancer, the National Prostate Cancer Forum was held in early 1997 in Toronto.[239] The Forum's recommendations included the development of a comprehensive research program that reflects the importance of the disease. As a first step, it was proposed that a Canadian randomized controlled trial of screening for prostate cancer with PSA be conducted to resolve the debate over the use of PSA for screening. Tools are necessary to monitor the outcomes of changes in practices of screening, increasing earlier diagnosis and changes in treatment. To accomplish this, the Forum recommended the creation of a registry of outcomes data, as well as a serum bank and a tissue bank on prostate cancer.

The implementation of these recommendations would help to increase our understanding of the epidemiology of prostate cancer and to resolve the controversy over screening.

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APPENDIX

Classification of prostate cancer: tumour, node, metastases (TNM) and Jewett systems

TNM	Description	Jewett
T	Primary tumour	
TX	Primary tumour cannot be assessed	
TO	No evidence of primary tumour	
T1	Clinically unapparent tumour, not palpable or visible by imaging	A
T1a	Tumour an incidental histological finding in 5% or less of tissue resected	A$_1$
T1b	Tumour an incidental histological finding in more than 5% of tissue resected	A$_2$
T1c	Tumour identified by needle biopsy (e.g. because of elevated prostate-specific antigen [PSA])	
T2	Tumour confined within the prostate	B
T2a	Tumour involves half a lobe or less	B$_1$
T2b	Tumour involves more than half a lobe but not both lobes	B$_2$
T2c	Tumour involves both lobes	B$_2$
T3	Tumour extends through the prostatic capsule	C
T3a	Unilateral extracapsular extension	C$_1$
T3b	Bilateral extracapsular extension	C$_1$
T3c	Tumour invades seminal vesicle	C$_1$
T4	Tumour is fixed or invades adjacent structures other than seminal vesicles	C$_2$
T4a	Tumour invades bladder neck and/or external sphincter and/or rectum	
T4b	Tumour invades levator muscles and/or is fixed to pelvic wall	
N	Regional lymph nodes	
NX	Regional lymph nodes cannot be assessed	
NO	No regional lymph node metastasis	
N1	Metastasis in a single regional lymph node, <2 cm in greatest dimension	D$_1$
N2	Metastasis in a single regional lymph node, >2 cm <5 cm in greatest dimension, or multiple regional lymph nodes, none >5 cm in greatest dimension	
N3	Metastasis in a regional lymph node >5 cm in greatest dimension	
M	Distant metastasis	
MX	Presence of distant metastasis cannot be assessed	
MO	No distant metastasis	
M1	Distant metastasis	D$_2$
M1a	Nonregional lymph nodes(s)	
M1b	Bone(s)	
M1c	Other site(s)	

Source: Modified from References 24, 25

Pap Smear Utilization in Canada: Estimates after Adjusting the Eligible Population for Hysterectomy Status

Judy A Snider and Janet E Beauvais

Abstract

The 1994 National Population Health Survey (NPHS) confirmed that the trend in Pap smear utilization has not changed in the past 10 years; 15% of Canadian women reported never having had a Pap smear and an additional 15% reported not having had one within 3 years prior to the survey. Most of these underserved women can be characterized as disadvantaged. The overall prevalence of hysterectomy in Canada in 1994 was 16.3%, and prevalence increased sharply to 30% between ages 35 and 55 years. Hysterectomy rates vary according to sociodemographic factors, with women of lower income and education indicating a higher proportion of hysterectomies. We adjusted the female population from the NPHS, by removing the estimated proportion of women reporting hysterectomy, to reveal the true population at risk of developing cervical cancer. Absolute prevalence of Pap smear utilization increased (7–25%); the relative improvement was much higher. Adjusting the eligible population could enable screening programs to better estimate the size of their underserved population.

Key words: *Canada; cervical cancer screening; hysterectomy; Pap smear utilization; sociodemographic*

Introduction

Cervical cancer remains an important disease because, in spite of being largely preventable, it continues to afflict a large number of Canadian women each year. In 1997, it was estimated that 1300 Canadian women would be diagnosed with cervical cancer and approximately 390 would die of the disease.[1] Pap smears were introduced approximately 50 years ago to detect precancerous lesions, but they continue to be an underutilized health procedure by some segments of the population.

Two national workshops[2,3] have addressed cervical cancer screening in general and have also specifically identified the need to better characterize and recruit women who either have never had a Pap smear or have been screened irregularly. A recent study[4] estimated that the proportion of women who had never been screened remained steady at about 15% between 1985 and 1994 and that a further 15% of women over 18 years of age had not had a Pap smear within three years prior to the administration of the last survey.

Considerable effort has focused on characterizing underserved women,[4–6] and results indicate that these women can be generally described as economically and/or socially disadvantaged. Unfortunately these studies have not differentiated between women who require screening but do not access these services from those who are ineligible for screening, such as women who have had a total hysterectomy for benign conditions with their cervix removed or who have never had sexual intercourse.[2] While the largest proportion of ineligible women are those who have had a hysterectomy, a small proportion of ineligible women—those with a previously diagnosed gynecological cancer—are also excluded from many organized screening programs.[7,8]

Hysterectomy is one of the most frequently performed surgical procedures in Canada.[9] Using hospitalization

data,[10] trends in the number of hysterectomies performed in the last 25 years show that incidence peaks in women aged 40–44 years. Notably, the hysterectomy rate has decreased over time from a high of 2419 per 100,000 women in the early 1970s to 1284 per 100,000 women in 1990 (Figure 1).[10]

The purpose of this study is to estimate Pap smear utilization rates in Canada, adjusted for women who are no longer at risk of developing cervical cancer by virtue of having had a hysterectomy, using hysterectomy prevalence data collected as part of a recent Canadian survey. While estimates of the prevalence of hysterectomy are available from many sources,[10–15] data presented here describe the prevalence of hysterectomy by several sociodemographic groups. The newly adjusted Pap smear rates provide a better estimate of the proportion of women who remain underserved and who need to be recruited into cervical cancer screening programs.

Methods

Two data sources were used in the analyses for this study: the public data files from the National Population Health Survey (NPHS) and results from the analysis of proprietary questions in the Canada Health Monitor (CHM) survey. Both were telephone surveys that excluded a number of populations, including residents from the Northwest Territories and Yukon. The NPHS also excluded remote areas of Quebec and Ontario, and members of First Nations living on reserves.

The NPHS is a longitudinal study that will collect information on the same panel of respondents every two years from 1994, for up to two decades. Detailed information on sampling and survey methodology has been published elsewhere.[16] The panel of residents invited to participate consisted of 8848 women aged 18 and older. These respondents were asked several preventive health questions, including whether they had ever had a Pap smear and the recency of their latest Pap smear (Appendix).

The CHM survey (# 11, July–August 1994), which was cross-sectional, interviewed 1382 women aged 15 and older on various health-related issues as well as sociodemographic factors. Included in this survey were questions commissioned by Health Canada on hysterectomy status and/or oophorectomy status (Appendix). The CHM used a stratified two-stage random sampling technique. Quotas were determined for each province proportional to its contribution to the Canadian population. Random digit dialling techniques were used to identify potential households, and one respondent was randomly selected from each of these households. Further details regarding the survey sampling and methodology are available elsewhere.[17]

Prior to adjusting the NPHS Pap smear rates by the CHM hysterectomy rates, we compared the characteristics of the women responding to each of the surveys. Where necessary, we created derived variables to permit the data

FIGURE 1
Trends in the incidence of hysterectomy, 5-year age groups, 1969-1990, Canada

Rate per 100,000 women

Legend: ◆ 25-29 ✳ 30-34 ■ 35-39 — 40-44 ✶ 45-49 ▪ 50-54 + 55-59 ✖ 60-64 ▲ 65-69

Source: Findlater R. Special statistical analysis from Statistics Canada, as published in his thesis (Appendix F) [Reference 10]
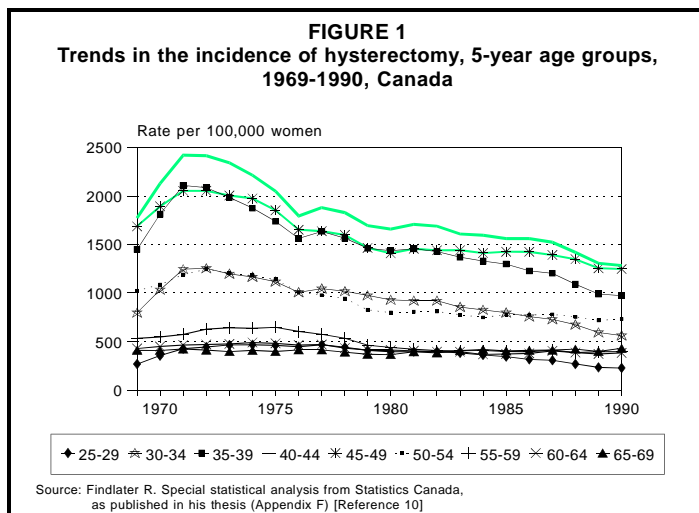
values to be grouped into categories comparable to those used in the CHM results.

The following variables in the NPHS data set were recategorized to match CHM data as closely as possible: provinces were recoded into regions, and education was dichotomized into less than high school and high school education or greater in order to compensate for differences in the classification. Household income in the NPHS is a derived variable and matched CHM cut-points for the three lowest income levels. It was not possible to recode the cut-point between the two highest levels; therefore, the NPHS groupings have a cut-point of $80,000 and the CHM's cut-point is $75,000. Both surveys had a similar distribution of women when categorized by age, region, household income and education (Table 1). Univariate and bivariate analyses were performed on the NPHS data using SAS.[18]

Adjustment of the overall Pap smear rates from the NPHS was performed by removing the proportion of women estimated to have had a hysterectomy from the total population reported in this survey. These calculations were repeated to adjust Pap smears according to the reported recency of the test, either within one year of the survey or within three years of the survey. Pap smear adjustment was also performed for the following sociodemographic indicators: age, region of Canada, household income and education.

Exact binomial 95% confidence intervals were calculated for all proportions using Epi Info 6.[19] The large sample size of the NPHS allowed small confidence intervals for reported Pap smears. The smaller CHM survey had broader confidence intervals, identifying greater variation in the reported point prevalence of hysterectomy. In depicting the Pap smear rates adjusted by hysterectomy (Figure 2), we used the CHM's 95% confidence intervals to calculate the upper and lower limits.

TABLE 1

Proportion of females responding to each question by age, region, household income and education[a]

	NPHS (%)	CHM (%)
AGE (years)		
15–24	16.4	12.4
25–34	20.9	17.7
35–44	20.7	21.3
45–54	14.6	15.3
55–64	11.3	12.7
65+	16.0	21.0
REGION		
Atlantic	8.3	8.7
Quebec	25.3	24.3
Ontario	37.9	36.7
Prairies	15.9	18.4
British Columbia	12.6	12.0
HOUSEHOLD INCOME		
Less than $20,000	24.0	28.0
$20,000–$30,000	13.2	18.8
$30,000–$50,000	27.7	28.1
$50,000–$75,000 (CHM)/ $50,000–$80,000 (NPHS)	23.6	15.1
Over $75,000 (CHM)/ over $80,000 (NPHS)	11.6	9.3
EDUCATION		
Less than high school	33.4	26.4
High school or greater	66.6	73.3

[a] Totals may vary slightly from 100% due to rounding.

Results

Hysterectomy with or without oophorectomy was reported by 16.3% of the women aged 15 and older who responded to the CHM survey. Less than 1% of the women under age 35 had undergone a hysterectomy; however, the proportion of women reporting a hysterectomy increased sharply after this age until 55, by which age almost one third of the women had had a hysterectomy (Table 2). The prevalence of hysterectomy in Canada varied among regions, being highest in the Atlantic region and lowest in Quebec and the Prairies.

Variations in the prevalence of hysterectomy by household income and education are also evident; generally, the highest rates occurred among women with the lowest income and education. Unfortunately, there is some instability in the point estimates due to the CHM

FIGURE 2
Proportion of women reporting a Pap smear, by age group, unadjusted and adjusted for hysterectomy, Canada
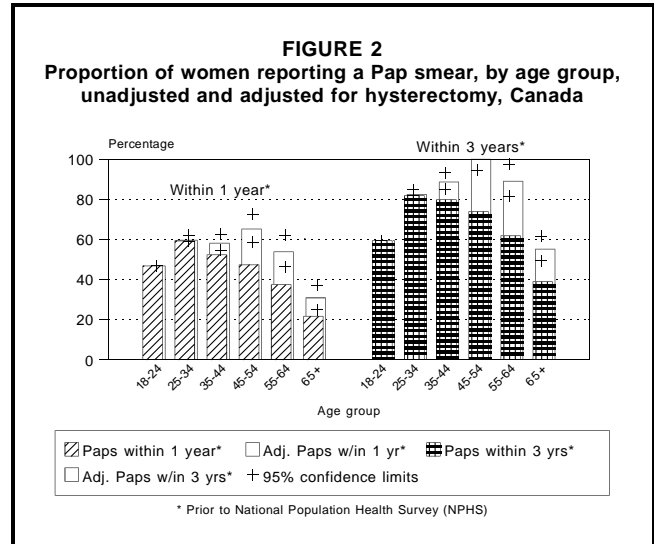
TABLE 2

Prevalence of hysterectomy by age, region, household income and education, from the CHM survey

	Percentage of women reporting hysterectomy	(95% Confidence Interval)
AGE (years)		
TOTAL (crude)	16.3	(14.1–18.6)
15–24	0.0	(0.0–0.0)
25–34	0.6	(0.0–2.9)
35–44	10.0	(6.4–14.4)
45–54	26.8	(20.1–34.0)
55–64	30.6	(23.0–38.8)
65+	29.6	(23.6–35.8)
REGION		
Atlantic	27.5	(18.7–37.5)
Quebec	13.4	(9.6–18.0)
Ontario	16.3	(12.7–20.1)
Prairies	13.8	(9.3–19.2)
British Columbia	18.9	(12.7–26.7)
HOUSEHOLD INCOME		
Less than $20,000	20.2	(16.0–25.4)
$20,000–$30,000	18.9	(13.4–25.1)
$30,000–$50,000	12.4	(8.8–16.8)
$50,000–$75,000	13.3	(8.2–19.6)
Over $75,000	18.6	(11.0–27.7)
EDUCATION		
Less than high school	21.2	(16.6–26.3)
High school or greater	14.9	(12.5–17.6)

survey's small sample size. Specifically, this can be seen in the wide confidence intervals surrounding the high percentage of reported hysterectomy in the Atlantic region.

The proportion of women in the NPHS in each age group who had a Pap smear performed one year prior to the survey is illustrated in Figure 2. Utilization of Pap smears peaks in the 25–34 age group and gradually declines for each subsequent age group. Results by income and education (Table 3) indicate that a lower percentage of women with lower income levels and educational attainment have had a Pap smear.

After removing the estimated proportion of women who had a hysterectomy from the total population of women, we obtained an adjusted target population for Pap smear utilization. For all age groups over age 35, the adjustment yields an increase in the Pap smear rate (Figure 2). For example, the Pap smear rate within the year prior to the survey rose almost 10%, from an unadjusted rate of 45.7% to an adjusted rate of 54.6%. The greatest increase, almost 18%, is seen in the 45–54 age group.

For Pap smears reported within three years of the survey, the overall adjusted estimates increased from 68.1% to 81.3%. After adjusting for hysterectomy, the Pap smear rate for the three-year interval approximates 100% of the eligible Canadian women in the 45–54 age group who are represented in the NPHS population. However, given the potential variation in the CHM survey results, this proportion may be as low as 93.4%.

Pap smear utilization rates were also adjusted separately by region, income and education. Further breakdown of these categories, such as by age, was not possible. Table 3 shows that the greatest increase from the unadjusted to adjusted rates occurred in the Atlantic region, where the Pap smear rates within three years before the survey rose by 25%. The differences between unadjusted and adjusted Pap smear rates by income were fairly constant, ranging from 8% to 13% for tests within one year of the survey and from 10% to 17% for those within three years of the survey.

Discussion

Pap smear utilization rates increased after adjusting the estimates of the actual population at risk for cervical cancer. Results from this analysis suggest that, prior to adjustment, the greatest use of Pap smears occurred among women aged 25–34. However, by adjusting the target population to exclude women with self-reported hysterectomy, the peak rate of Pap smears shifts to the 45–54 age group. The high estimate in this age group may be due to several reasons, including peri-menopausal women visiting their physicians and having a Pap smear taken or the monitoring of women who use hormone replacement therapy and have an intact uterus. This utilization pattern mimics the pattern reported by the established screening programs in Iceland[20] and at the Victorian Cervical Cytology Registry in Australia,[21] where women in the 45–54 age range have the highest Pap smear rates of all reported age groups. These jurisdictions remove ineligible women, such as those with a hysterectomy, from the eligible population when calculating Pap smear utilization rates.

Other findings from the NPHS analysis indicated that 32% of the women had not had a Pap smear within three years of the survey. This rate decreases to 18% once adjusted for prevalence of hysterectomy. However, even with a hysterectomy rate approaching 30% in older women aged 65 and over, the adjusted rate still reveals an unacceptable level of underutilization. Although a larger proportion of disadvantaged women had had a hysterectomy, adjusted Pap smear rates among these groups of women still remain lower than those among women with higher educational attainment and income adequacy. The adjustment in the target population has not narrowed the gap in usage between women of higher and lower sociodemographic status.

TABLE 3

Proportion of women reporting a Pap smear, unadjusted and adjusted for hysterectomy, by region, household income and education

	Unadjusted percentage of women reporting Pap smears in the NPHS		Adjusted percentage of women reporting Pap smears	
	Within 1 year before survey	Within 3 years before survey	Within 1 year before survey	Within 3 years before survey
REGION				
Atlantic	47.0	67.2	64.8	92.6
Quebec	42.9	62.5	49.6	72.2
Ontario	44.3	67.7	52.9	80.9
Prairies	50.3	74.1	58.3	86.0
British Columbia	46.1	68.6	56.9	84.3
HOUSEHOLD INCOME				
Less than $20,000	47.3	56.2	59.3	70.5
$20,000–$30,000	49.4	62.8	60.9	77.5
$30,000–$50,000	55.1	70.8	62.9	80.8
$50,000–$80,000	57.3	75.6	66.1	87.2
Over $80,000	56.9	74.7	69.9	91.8
EDUCATION				
Less than high school	31.1	51.0	39.4	64.7
High school or greater	50.7	73.6	59.5	86.5

When the data are examined by geographic region, there is greater variation after adjustment due to the vast difference in the prevalence of hysterectomies. These regional disparities are not dissimilar to the findings of Gentleman et al.[9] and Miller et al.[11] In the former study, which has the most current data (1988–1990), the age-standardized incidence of hysterectomy is almost 50% greater in the Atlantic provinces than in the rest of the Canada.

There are a number of limitations in our study. Bias may have been introduced either by self-response or by non-response. Population estimates of screening practices may be influenced by the use of self-reported data, as reported by Gordon et al.,[22] who found that self-reported recency of screening was overestimated while screening practices did not differ significantly from screening detected by chart review. There have been few survey data collected on hysterectomy, and the level of self-reporting bias introduced by these questions is unknown. In addition, it was not possible to determine whether women who had undergone this surgical procedure had experienced a total hysterectomy with their cervix removed or a subtotal hysterectomy.

Both surveys excluded women from the First Nations, northern territories and remote regions, and those without phone service. These omissions may lead to an underestimation of the population of women never screened, thereby providing an overestimation of Pap smear utilization. It is also possible that the CHM's small sample size produced highly variable adjusted Pap smear rates for some of the sociodemographic groups and that these small numbers precluded further investigations. Finally, the simplified method of using the CHM's 95% confidence intervals may have underestimated the real confidence intervals.

The strengths of our study are that both surveys used were conducted recently and that the CHM is the first cross-Canada survey to collect hysterectomy data. The advantage of this data set is that it provides some sociodemographic information on women who have had a hysterectomy. This detailed information has never before been collected, nor has it been modelled. The prevalence estimates from this survey generally agree with published results from studies that have modelled the prevalence of hysterectomy using annual hospital procedure data.[10–15]

Conclusion

The results of this study indicate that a high proportion of Canadian women have had a hysterectomy and are ineligible for routine Pap smear testing in cervical cancer screening programs. Removing these women from the eligible population results in higher Pap smear utilization rates than are currently reported. This type of adjustment provides a more accurate estimate of the proportion of women who are eligible for Pap smears but remain underserved.

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APPENDIX

1994 National Population Health Survey Preventive Health Practices

PHP-Q3   Have you ever had a Pap smear test?
☐ Yes
☐ No (Go to next section)
☐ DK (Go to next section)

PHP-Q3a   When was the last time?
(Do not read list. Mark one only.)
☐ Less than 6 months ago
☐ 6 months to less than one year ago
☐ 1 year to less than 3 years ago
☐ 3 years to less than 5 years ago
☐ 5 years or more ago

Canada Health Monitor Survey #11, July–August 1994

Hys1.   Have you ever had a hysterectomy or an operation to remove one or more ovaries?
☐ Yes
☐ No

Hys2.   (if YES...) Was it to remove...
1. Your uterus
2. Uterus and one ovary
3. Uterus and both ovaries
4. One ovary only
5. Both ovaries

VOLUNTEERED
☐ Not sure

Sources: References 16 and 17 ■

Firearms Regulation: Canada in the International Context

Wendy Cukier

Abstract

Gun deaths and injuries in Canada are a serious public health problem, claiming more than 1200 lives each year and resulting in over 1000 hospitalizations. While the issue has been hotly debated in recent years, considerable research in an international context suggests that there is a relationship between access to firearms and deaths and injuries caused by firearms. Interventions to reduce access to firearms include regulation, education and engineering. Legislative reforms aimed at reducing gun deaths and injuries have been introduced recently in Canada and in many other countries. Although domestic controls can affect the supply of guns, efforts are being co-ordinated increasingly on an interjurisdictional basis to decrease the illegal trafficking of firearms. As well, the United Nations Crime Prevention and Criminal Justice Commission recently passed a resolution encouraging all countries who have not done so to strengthen their domestic gun controls since weak controls in one country can affect security in others.

Key words: *Crime; firearms; guns; gunshot; injury; international; prevention; suicide*

Gun Deaths and Injuries in Canada

Countries around the globe have begun to direct their attention to the problems of firearms death and injury, and many have introduced legislative reforms in recent years. Canada has historically had stricter controls on firearms than the United States as well as much lower rates of gun-related death, injury and crime. However, the international context provides a different perspective. Canada has much higher gun death rates than most other industrialized nations, and the new law passed in 1995 is consistent with approaches taken to regulate firearms in most industrialized countries.

From a public health perspective, firearms deaths and injuries, whether intentional or unintentional, are a serious threat to the health of Canadians. An average of more than 1200 Canadians have been killed and over 1000 have been injured with firearms each year during the past 10 years. For example, in 1995, 911 Canadians committed suicide with firearms, 145 were killed with firearms in homicides, 49 died in "accidents," 6 were killed in legal interventions and 14 deaths were undetermined, creating an overall firearms death rate of 3.8 per 100,000.[1] While some have suggested that firearms deaths and injuries are not serious problems compared to other causes of death such as cancer,[2] public health professionals have tended to set priorities based not only on the rate of death but on the extent to which many of the deaths were preventable.[3]

The economic costs of gun deaths and injuries in Canada have been estimated at $6 billion per year.[4] The cost among young people is particularly high: firearms deaths are the third leading cause of death among young people aged 15–24.[5] Canada is fifth among industrialized countries in the firearms death rate among children under the age of 14.[6] The international context provides a useful perspective on the problem of firearms in Canada as well as on the approach to addressing the problem.

Methods

There are many methodological challenges in research on firearms regulation. Cross-cultural comparisons are difficult because of the variability of data and the inconsistencies among different reporting practices and data

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sources.[7,a] Moreover, the complexity of other variables, such as cultural differences, socio-economic conditions or other factors, adds to the difficulty of demonstrating causal links. However, these methodological challenges are not unique to the firearms regulation issue, but affect many other complex issues in crime prevention, public safety and health care. There have been parallels drawn between political influences on research on the effects of guns and on the effects of tobacco.[8]

To ensure a wide range of sources, several different methods were used to collect material for this article. Searches were conducted on MEDLINE, Wilson and related databases for the period 1980–1997 using "firearms" and "gun" as the key words. International sources such as the Victimization Survey and the recent United Nations (UN) *International Study on Firearm Regulation* were consulted along with statistical sources from individual countries, such as Statistics Canada. In addition to the peer-reviewed literature, the records of recent government inquiries and proceedings were examined, including the Canadian House of Commons and Senate Committees and debates on Bill C-68, materials prepared for the Alberta Court of Appeal, the Lord Cullen Inquiry into Dunblane and the Review of Firearms Control in New Zealand. Additional materials were obtained from government and police sources in Great Britain, Japan, Australia and Switzerland.

Link Between Access and Death Rates

A number of researchers maintain that there is sufficient evidence to conclude that rates of firearms death and injury are linked to access to firearms.[9,10] Access to firearms may be defined in a number of ways, including the percentage of households where firearms are present (or various surrogate measures)[11] or the ease with which individuals can obtain firearms and ammunition[b] in a given place at a given time.

The strategies to reduce gun injuries and deaths proposed by the public health community have tended to mirror the approaches taken with infectious diseases, combining education, regulation and engineering.

To prevent an illness or injury, public health experts consider preventative action to control the agent and the vehicle to protect the host. In the case of injury due to gunshot wounds the agent is the force deployed by firing a gun, the vehicle is the gun or

ammunition and the human host is the victim ... access constitutes the universal link—the one against which we can take action—in the chain of events leading to an injury with a firearm.[12]

Access to Guns in the Home

Many research projects examining the accessibility thesis have compared homes where firearms are present with those where they are not.[13] Kellerman and his colleagues, for example, concluded that the homicide of a family member was 2.7 times more likely to occur in a home with a firearm than in homes without guns. After accounting for several independent risk factors, another study concluded that keeping one or more firearms was associated with a 4.8-fold increased risk of suicide in the home.[14] The risks increased, particularly for adolescents, where the guns were kept loaded and unlocked.[15]

Comparisons Between Canada and the United States

Studies have also compared the rates of death from firearms in Canada with those in the United States. One of the most well-known studies was a comparison of Seattle, Washington, and Vancouver, British Columbia.[16] More recently, the costs of firearms death and injury in the two countries were compared and estimated to be $495 (US) per resident in the United States and $195 per resident in Canada.[10]

Canada has always had stronger firearms regulation than the United States, particularly with respect to handguns. Handguns have required licensing and registration in Canada since the 1930s. Ownership of guns has never been regarded as a right, and several court rulings have reaffirmed the right of the government to protect citizens from guns.[17,18] Handgun ownership has been restricted to police, members of gun clubs or collectors. Very few people (about 50 in the country) have been given permits to carry handguns for "self-protection." This is only possible if an applicant can prove that his or her life is in danger and the police cannot protect the person. As a result, Canada has roughly 1 million handguns while the United States has more than 77 million. Although there are other factors affecting rates of murder, suicide and unintentional injury, a comparison of data in Canada with US data suggests that access to handguns may play a role. While the murder rate without guns in the US is slightly higher (1.7 times) than that in Canada, the murder rate with handguns is 15 times the Canadian rate (Table 1).

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a   This report[7] documented variances in data sources among, for example, homicide rates collected from Interpol, the Centers for Disease Control and Prevention, and the UN surveys of Crime Trends and Operations of Criminal Justice Systems. In addition, no data source is complete, although the UN seems to have the most comprehensive one.

b   Accessibility in terms of ease of acquisition may be measured by the rigour of processes in controlling the licensing of gun owners. An analogous situation exists with other forms of licensing where processes are designed to allow only well-qualified individuals to acquire access to potentially dangerous goods, such as automobiles. Licensing regimes may identify risk factors and raise the standards to reduce access by those at risk. For example, in 1991, the age for a Firearms Acquisition Certificate in Canada was raised to 18, although minors' permits were allowed under particular circumstances. The legislation passed in 1995 requires screening of all current owners of firearms for records of criminal behaviour or other risk factors. In cases where individuals wish to acquire guns, screening is more rigorous and includes notification of current and previous spouses to reduce the risks that individuals with a history of domestic violence will have access to firearms.

TABLE 1 US/Canada comparisons related to firearms	Canada[a]	US[b,c]	US/CAN
Population (1995)	29.5 mil	263 mil	8.9 x
Estimated number of all firearms (1993)	7 mil	223 mil	31.9 x
Estimated number of handguns (1993)	1 mil	77 mil	77.0 x
Firearms per capita (1995)	0.24	0.84	3.5 x
1995 RATES OF DEATH AND CRIME (per 100,000 population)			
Accidental deaths with firearms (E-codes)	0.17	0.5	2.9 x
Homicides with firearms (E-codes)	0.5	6.0	12.0 x
Suicides with firearms (E-codes)	3.1	7.0	2.3 x
Total deaths from firearms (E-codes)	3.8	13.7	3.6 x
Murders (UCR)	2.0	7.6	4.1 x
Murders with firearms (UCR)	0.6	5.2	8.7 x
Murders with handguns (UCR)	0.3	4.6	15.0 x
Murders without firearms (UCR)	1.4	2.4	1.7 x

Sources: [a] Reference 1 for Canadian E-codes and UCR codes
[b] Reference 19 for E-codes (US)
[c] Reference 20 for US crime, tables 2.9 and 2.10 for UCR codes

International Comparisons

One study examined the link between rates of gun ownership and firearms death within Canadian provinces, the US, England/Wales and Australia, concluding that 92% of the variance in death rates was explained by access to firearms in those areas.[10]

The international experience with firearms regulation and comparative mortality statistics tends to reinforce the thesis that there is a link between access to firearms and firearms death in industrialized nations, although there are issues concerning uniform reporting and other variables that must be addressed. For example, a review by Killias of 13 countries showed a strong correlation between gun ownership and both homicide with a gun and overall homicide rates (Northern Ireland was excluded from the analysis because of the level of civil unrest). In an analysis of 14 countries, the correlation between gun ownership and gun suicide was also significant as was the correlation of gun ownership with overall suicide rates. Killias found no evidence of a compensation process whereby other means were substituted for firearms.[21]

In another study based on a standardized survey of victimization in 54 countries, gun ownership was significantly related to both the level of robberies and the level of sexual assaults. The relationship between levels of gun ownership and threats/assaults with a gun was strong as well.[22,23] Van Dijk also concluded that high levels of gun ownership, such as those in the USA, the former Yugoslavia, South Africa and several Latin American countries, were strongly related to higher levels of violence generally. While more research could illuminate the interaction between a range of factors that influence firearms violence and suicide, there are strong suggestions of an important relationship between access to firearms and rates of firearms death and crime.

International Firearms Regulation

A review of international approaches to firearms regulation indicates that industrialized countries with lower rates of firearms ownership and lower rates of firearms death than Canada also tend to have higher levels of regulation. Most developed countries have strict laws governing licensing and registration of all firearms and very strict controls on handguns (see Table 2). These measures were included in Canada's 1995 gun control legislation.

The Effect of Legislation

Comparisons of regions with strong regulations to areas with weak regulations within the same country also tend to confirm that gun control works where other factors are more or less the same. For example, Australian states with firearms registration had significantly lower rates of homicide and suicide with firearms than states without registration of firearms.[27]

The accessibility thesis has been supported by studies examining the effects of legislation on death and injury rates in Canada as well.[28] A more recent study suggests that changes to Canada's gun control law have had an effect on accidental firearm death rates, particularly in males.[29]

Others have argued that there is little evidence of a link between access to firearms and rates of death and have disputed the studies proposing that stricter controls on firearms reduce gun death and injury.[30,31] Some have even suggested that increasing access to firearms through arming for self-protection saves lives and reduces injury.[2,32,33] Some of these studies have been criticized for methodological problems.[34,35]

Although much has been written about the failures of gun control, on balance, peer-reviewed scientific literature tends to support the accessibility thesis and the efficacy of restrictions on access.[36,37] The positions held by major public health and safety groups certainly reflect this.

TABLE 2

International firearms regulations, access and deaths

Country	Licensing of owners?	Registration of all firearms?	Other	Households with firearms	Gun homicide (per million)	Gun suicide (per million)
Japan	Yes	Yes	Prohibits handguns with few exceptions	0.6%	0.3	0.36
Netherlands	Yes	Yes		1.9%	2.7	2.8
United Kingdom	Yes	Yes	Prohibits handguns	4.0%	1.3	3.3
Northern Ireland	Yes	Yes		8.4%	35.5	11.8
Germany	Yes	Yes		8.9%	2.1	12.3
Spain	Yes	Yes	Some handguns and rifles are prohibited	13.1%	1.9	5.5
Australia	Yes	All guns in 5 of 8 states until 1997 when national standards began	Banned semi-automatics unless good reason is shown	16.0%	5.6	23.8
Belgium	Yes	Yes	Some rifles are prohibited	16.6%	8.7	24.5
New Zealand	Yes	Handguns only, stopped registering rifles and shotguns in 1983 and have proposed reintroducing it		20.0%	2.2	24.5
France	Yes	Yes, except for selected sporting rifles		22.6%	5.5	49.3
CANADA	Acquisition only, possession starts in 1998	Handguns only, all guns as of 1998	Fully automatic, converted and semi-automatic assault weapons and some handguns are banned	26.0%	6.0	33.5
Switzerland	Acquisition for some	For some firearms		27.2%	4.6	57.4
Norway	Yes	Unknown		32.0%	3.6	38.7
USA	In some states	Handguns in some states	Some weapons in some states	41.0%	62.4	72.3
Finland	Yes	Yes	No prohibitions	50.0%	8.7	57.8

Sources
Rates of households with firearms and firearms deaths for most countries are from the United Nations (UN) *International Study on Firearm Regulation (revised)*[7] [tables 2.7, 6.2 and 7.1]. Rates for the Netherlands, Northern Ireland, Belgium, France, Switzerland and Norway, who did not respond to the UN survey, are from Martin Killias,[21] who cites 1989 figures from the UN interregional study.

Details regarding legislation are from various sources, including the UN study, as well as Joachim J Savelsberg,[24] Wendy Cukier[25] and the Department of Justice Canada.[26]

Although the complexity of factors influencing death rates and crime, particularly over time, makes longitudinal analysis particularly difficult, criminologist Neil Boyd concluded that there is more evidence to support the efficacy of gun control legislation in reducing death and injury than there is for most other legislative interventions. In reviewing the evaluations of the Canadian legislation, he wrote the following.

In three separate forms of statistical analysis— exploratory, time-series and structural—researchers have found evidence to suggest that gun control has had an impact on homicides and firearms homicides. The finding that an amendment to criminal law can change behaviour in the direction desired is unusual. We have had many amendments to Canadian criminal law during the past 40 years: for example changes to the penalty structure for homicide in 1961, 1967, 1973, 1974, 1976 and 1985; changes for the penalty structure affecting illegal drug use and distribution in 1961, 1969 and 1974 .... In none of these circumstances has it been possible to establish that a change in law can impact behaviour in the direction that the law hopes for or anticipates. With gun control legislation, we have some preliminary evidence—some strong suggestions—that the criminal law is working. And it is working, not by manipulating penalty levels for specific forms of crime, but by putting a regulatory system in place that can limit access to firearms, enhance the safety of firearm use, and, in a more general sense, educate the public with respect to the dangers inherent in widespread availability of these potentially lethal commodities.[38]

Approaches to Controlling Access

Most firearms control regimes are based on the assumption that controlling access will reduce death, injury and crime. Measures aimed at controlling access include outright prohibitions for firearms where the risk is considered to outweigh the utility.

In 1979 Canada prohibited fully automatic weapons; in 1991, semi-automatic weapons that could be converted to fully automatic fire; and in 1995, semi-automatic versions of military weapons. In almost all cases, current owners were "grandfathered" or allowed to keep their weapons under certain conditions.

Great Britain banned 90% of handguns in February 1997 and banned the remaining 10% with the change of government in June 1997. Owners were entitled to compensation, but possession of the prohibited guns became illegal. Similarly, Australia banned semi-automatic firearms and shotguns, except for individuals who could demonstrate "good reason" for owning them, and bought back more than 500,000 guns.

In most contexts, it is not possible to ban firearms except those that have little practical purpose. Regulation is a compromise approach to allow products that are inherently dangerous to be used under certain circumstances. Regulations reduce casual gun ownership by increasing the barriers to obtaining firearms. They are also intended to diminish the risks of firearms ownership by improving screening processes.[39] Approaches include criminal record checks, community checks and references, waiting periods, mandatory training programs, etc.

There are various ways of increasing barriers between individuals and firearms to prevent impulsive use and unauthorized access. Increasingly in the US, attention is being focused on technological changes to reduce unauthorized access.[40] Regulations that encourage safe storage practices, such as using locked containers and trigger locks, disabling firearms and separating ammunition from the gun, are standard in most industrialized countries but are the exception, not the rule, in the United States.[41]

Measures have also been taken to reduce demand for firearms by raising awareness of the risks they pose, particularly in the home,[c] and by developing methods such as amnesties and "buy-backs" to encourage individuals to rid themselves of unwanted or unneeded firearms.[43,44] The impact of these methods has been questioned; however, they may have educational effects that have not been measured. Educational programs have focused on promoting awareness of safe firearms practices and compliance with them.[45]

In addition, regulatory restrictions and litigation have been used to encourage suppliers of firearms to control sales and to be more responsible.[46]

Reducing Primary Demand

Some have suggested that efforts to reduce gun death and injury must also consider primary demand. It has been proposed that "gun culture" is largely an American construct[47] that is reinforced by the absence of effective laws and the normalization of violence. Much of the demand for guns, particularly military weapons and handguns that serve little practical purpose, may be fuelled by violent movies and television, which tend to link heroism with guns and violence.

In passing their recent firearms regulation law, the British were explicit: they saw it as a rejection of American style "gun culture."[48] The suggestion that there is a link between values and gun violence is not new.

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[c]   Public programs to discourage keeping guns in the home have been extensive in the US. For example, Project Lifeline is a public service campaign of the HELP Network, Physicians for Social Responsibility and the Center to Prevent Handgun Violence. The advertisements show a handgun pointed out from a picture with the caption "The person most likely to kill you with a handgun already has the keys to your house."[42]

By our readiness to allow arms to be purchased at will and fired at whim; by allowing our movies and television screens to teach our children that the hero is one who masters the art of shooting and the technique of killing ... we have created an atmosphere in which violence and hatred have become popular pastimes.
—*Martin Luther King, November 1963*[49]

Gartner has suggested that the effects of gun control laws are, therefore, both direct and indirect because of the important interaction between laws and values: countries with stricter controls send a signal about the acceptability of violence in the same way that legislation has been observed to have long-term effects on other behaviours such as smoking, driving while drunk and drug abuse.[50,51] Stricter controls on firearms both shape and reflect values.

The irony in this is that countries with strict controls, such as Great Britain, tend to be able to pass additional controls on firearms quickly and with relative ease. Countries without effective controls, such as the United States, have more guns and higher rates of gun death and injury. They also have effective opposition to stricter controls. This principle also operates within countries. For example, the strongest opposition to changes to the law in Canada came from Alberta, the province with the highest rate of gun ownership and one of the highest rates of gun-related death and injury.

Recent Developments in International Regulations

Efforts in the United States to understand the problem of firearms death and injury and measures to reduce it have been well documented.[36] However, relatively little has been published on international efforts to control firearms. The *International Study on Firearm Regulation*[7] prepared for the UN Commission on Crime Prevention and Criminal Justice reported that more than half of the countries responding to the survey indicated being in the process of developing reforms to their firearms regulations. Australia, Canada, Czech Republic, Estonia and the United Kingdom have reforms in progress, and major legislative reform is under discussion in Brazil, Denmark, Finland, India, Jamaica, Poland, South Africa and New Zealand.

Canada

Since the murder of 14 women on December 6, 1989, at *l'École Polytechnique* in Montreal, two pieces of gun control legislation were passed in Canada. Former Justice Minister Kim Campbell's Bill C-17 passed through the Senate on December 5, 1991, and included the following measures.

- A ban on semi-automatic firearms that could be converted to full automatic fire
- Improvements to screening for the Firearms Acquisition Certificate (FAC), including raising the age to 18,

requiring two references, more detailed screening and a mandatory test
- Safe storage regulation requiring all guns to be stored unloaded and secured with a trigger lock, in a secure container or room, or by disabling the firearm
- A ban on large-capacity magazines, with some exemptions

Former Justice Minister Allan Rock's gun control law, which received Royal Assent on December 5, 1995, and is still in the process of being implemented, added these restrictions.
- A ban on semi-automatic military assault weapons
- A ban on short barrelled and small calibre (.25 and .32) handguns
- Licensing of all gun owners by 2001 (previously, the FAC was required to obtain guns, not to possess guns, and only 1/3 of gun owners had valid FACs)
- Registration of all guns by 2003
- Controls on the sale of ammunition

The law was supported by an unusual alliance of 350 groups, including the Canadian Association of Chiefs of Police, the Canadian Public Health Association, the Canadian Association of Emergency Physicians, the Canadian Trauma Association, the YWCA of Canada, CAVEAT and Victims of Violence International.[52]

Great Britain

Great Britain has long had strict controls on firearms. All gun owners are licensed and must provide a reason for owning guns. There are a wide range of grounds for refusal of licensing. In addition, all guns are registered and permits are required to purchase ammunition. The country has one of the lowest rates of gun violence in the world.

The firearms regulation debate was revived in Britain on March 13, 1996, when 16 primary-school children and their teacher were murdered by a member of a local gun club in tiny Dunblane, Scotland. Another 15 children were injured before the gunman killed himself. In response to the outcry, a public inquiry was called that examined many aspects of firearms regulation in an international context. In its submission to the Dunblane Inquiry into the Shootings at Dunblane Primary School, the British Home Office argued that strict licensing helped ensure that only suitable people could have a firearms licence, that there would be fewer guns in circulation and that it would be more difficult for criminals to get hold of guns. It also maintained that stricter controls had had a significant effect and contrasted crime patterns in Britain with those in the US, including the homicide rates as well as the significant use of guns in crime.[53]

Subsequently, a new law was passed that banned 95% of handguns and required that the remainder (.22 calibre) be stored at gun clubs. When the Labour party took power, it

introduced a total ban on handguns. Other regulatory changes are under consideration.[54]

Australia

Gun legislation in Australia is state-controlled rather than federally controlled. Prior to 1996 all states licensed gun owners, but only five of eight Australian states registered firearms. The National Committee on Violence recommended a series of measures related to firearms regulation in its 1990 report, including registration of all firearms,[55] and the former Federal Justice Minister advocated a national system of gun registration as part of the crime prevention strategy announced in May 1995. While advocates of the Australian firearms regulation had been working since 1988 to strengthen Australia's laws, the movement was propelled forward by the murder of 35 people in Port Arthur, Tasmania, on April 28, 1996.

Public outcry was intense and the response was swift. Australian Prime Minister John Howard obtained an agreement from all eight Australian states and territories to pass consistent legislation that included the following.[56]

- Registration of all firearms
- Stronger licensing provisions, including proof of genuine reason to own any firearm; uniform screening that included a five-year prohibition on owning firearms against anyone committing a domestic violence act or subject to a restraining order; a safety course requirement; a minimum age of 18 to purchase firearms; a 28-day waiting period; and strict uniform storage requirements
- A ban on semi-automatic rifles and shotguns, except for those farmers who could prove a genuine need (This was accomplished through a special tax levy to raise $500 million to buy back weapons from their owners.)
- Improved controls on the trading of firearms, including a separate permit for each firearm and a ban on private and mail order sales of firearms

By August 1997, over 500,000 weapons had been surrendered and $259.8 million (AUS) had been paid out.[57]

New Zealand

New Zealand requires possession permits for all gun owners and registers all handguns and military weapons. It discontinued its manual, paper-based firearms registration system for long guns in 1983. In response to police and public concern, a comprehensive review of New Zealand's Firearms Regulations was undertaken and the results were released in the summer of 1997.

Like the Dunblane Inquiry, the Review of Firearms Control in New Zealand considered a broad range of evidence and examined international experiences with gun control. Its principal conclusions were that "the Arms Act 1983 and its subsequent amendments do not provide an effective code for the control of firearms in New Zealand .... There is a need for radical reform of the

firearms laws." Among the reforms proposed were the ones listed below.[58]

- Stricter controls on handguns
- A buy-back of military style semi-automatic weapons
- Amnesty programs
- Stricter licensing and vetting processes
- Training of shooters
- Sanctions for the misuse of firearms
- Controls on the sale of ammunition
- Limits to the size of collections
- A return to the registration of all firearms
- Education

The recommendation related to registration of firearms was of particular significance because the decision of New Zealand to discontinue its paper-based system in 1983 has been used by opponents of registration to demonstrate that firearms registration does not work.[2] Thorp made the following conclusion.

The reasons which led to the abandonment of firearm registration in 1983 no longer present compelling obstacles in 1997. Not only have the technology and methods of administration moved forward since then, but experience has shown that the alternative of total reliance on personal vetting does not meet the reasonable needs of our society.[58]

Japan

Japan has a level of community safety that is unmatched by most of the world and reinforced by strong cultural norms. During all of 1995, fewer gun deaths occurred in Japan than occur in an average day in the United States. There were a total of 168 firearms shootings, in which 34 people were killed and 33 were injured.[59] However, the Japanese are concerned about what they perceive as an escalation in violence.

Gun-related crimes have threatened to undermine the fabric of Japan's peaceful society. A peaceful and safe society is a common desire of the people. In order to stop the spread of firearms and prevent the tragedy of gun-related crimes, it is imperative that each person understands the danger and the anti-social nature of firearms and resolves to eliminate gun-related crimes.[60]

Awareness of the issue of firearms regulation in Japan was influenced by the murder of Japanese citizens travelling in the US. Exchange student Yoshira Hattori was shot and killed on October 31, 1992, in Baton Rouge, Louisiana, when he made the fatal error of knocking on the wrong door. Kei Sunade was killed in 1994 in New York City. Yoshi's father, Masaichi Hattori, presented a petition requesting a ban on guns signed by 1.72 million people, the largest in history, to US President Clinton. Mr Hattori works with Kei's father, Koichi Sunade (of the Association

to End Gun Violence), and has donated the proceeds of the civil case against his son's killer to support community firearms regulation initiatives.[60]

Japanese police are concerned about the increasing proportion of firearms incidents involving individuals not associated with organized crime. In 1991, 93% of guns seized in Japan were from organized crime (Borykudan), but this had decreased to 74% in 1995. Police are also concerned about the problem of gun smuggling. The US was the leading source (32.9%) of smuggled guns, followed by China (20.9%).

Also of significance was the assassination attempt on the life of the Commissioner General of the Japanese Police Agency in March 1995.[59] Takaji Kunimatsu was shot four times with hollow point ammunition. He has since recovered and resumed his duties.

Despite the relatively low level of gun violence in Japan, the Japanese government has taken a leadership role in the United Nations' efforts to stem gun violence internationally.

Switzerland

Opponents of gun control often use Switzerland as evidence that access to guns is not linked to crime or violence. They argue that, since virtually all adult males are members of the army and have military weapons, there is nearly universal access to deadly weapons yet few gun-related problems in Switzerland.[2]

However, Swiss criminologist Martin Killias, of the *Université de Lausanne*, argues that the Swiss rate of households with firearms is actually comparable to that of Canada (27.2%). There is strict screening of army officers, and ammunition is stored in sealed boxes and inspected regularly. Despite these controls, Switzerland has rates of gun suicide second only to the US among the countries Killias surveyed.[61]

While firearms regulations in Switzerland are fragmented and controlled at the regional level, wide-ranging reforms are now under way to establish national standards.[7]

International Resolutions and Agreements

Although the evidence suggests that domestic controls on firearms have a significant impact, the absence of controls in other jurisdictions creates problems worldwide. For example, most of the firearms recovered from crime in Canada are rifles and shotguns, not smuggled handguns.[62] In addition, most of the firearms used to kill in Canada are rifles and shotguns.[1] The rate of handgun use in homicides, suicides and unintentional deaths in Canada is far lower than in the US, but handguns are more commonly used in

murders in large cities whereas rifles and shotguns are more commonly used in smaller, more rural areas.[d,63]

Despite Canada's strict domestic controls on firearms, many of the handguns used in crime and to kill are smuggled in from countries with less rigorous controls, notably the US.[62] Even in Japan, fully 30% of the firearms used in crimes originate in the US.[59] Within the United States, where firearms control is a state responsibility, there is some evidence that guns tend to flow from unregulated areas to more regulated areas.[64]

Concern about the flow of guns from unregulated areas to regulated areas is one of the reasons for the recent resolution passed by the UN Commission on Crime Prevention and Criminal Justice. At the May 1997 meeting of this UN Commission, a resolution sponsored by 33 countries was endorsed, explicitly linking access to firearms with death and injury and identifying the problem of guns flowing from less regulated areas to regulated ones. The resolution included the following points.[65]

4. Requests the Secretary-General to promote, within existing resources, technical co-operation projects that recognize the relevance of firearm regulation in addressing violence against women, in promoting justice for victims of crime, in addressing the problem of children and youth as victims and perpetrators of crime and in re-establishing or strengthening the rule of law in post-conflict peacekeeping projects

5. Encourages Member States to consider, where they have not yet done so, regulatory approaches to the civilian use of firearms that include the following common elements:

   a) Regulations relating to firearm safety and storage

   b) Appropriate penalties and/or administrative sanctions for offences involving the misuse or unlawful possession of firearms

   c) Mitigation of, or exemption from, criminal responsibility, amnesty or similar programs that individual Member States determine to be appropriate to encourage citizens to surrender illegal, unsafe or unwanted firearms

   d) A licensing system, *inter alia*, including the licensing of firearm businesses, to ensure that firearms are not distributed to persons convicted of serious crimes or other persons who are prohibited under the laws of respective Member States from owning or possessing firearms

   e) A record-keeping system for firearms, *inter alia*, including a system for the commercial distribution of firearms and a requirement for appropriate marking of firearms at manufacture and at import, to

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d   Since 1991, handguns were responsible for three quarters of all firearm homicides in Toronto, Montreal and Vancouver—Canada's largest Census Metropolitan Areas (CMAs). Conversely, in smaller, non-CMA areas with population under 100,000, rifles and shotguns were most prevalent in firearm homicides (62%).

assist criminal investigations, discourage theft and ensure that firearms are distributed only to persons who may lawfully own or possess firearms under the laws of the respective Member States

These elements are already components in Canada's new gun control law.

More recently an agreement signed by the Organization of American States (OAS) identified the need to develop additional methods to secure borders in order to fight transnational crime, drug-trafficking and terrorism.[66]

To this end we will combat illegal firearms trafficking, by considering a new international instrument. We will seek to adopt standard systems for firearms identification and a stronger international regime for import and export licensing of firearms.

The OAS convention was signed in November 1997.

Conclusions

Gun deaths and injuries in Canada pose a serious problem that many researchers and practitioners believe can be reduced through effective public health strategies that combine legislation with education and enforcement. While Canada's problem with guns pales compared with that of the United States, many other countries have significantly lower rates of gun death and injury.

Several researchers have identified strong relationships between access to firearms and death rates in a variety of contexts. Although some maintain that there is no evidence of such a link or even maintain that the presence of guns helps reduce crime and violence, the bulk of the scientific literature tends to support the accessibility thesis.

Canada's recently passed legislation, which requires licensing of all firearms owners and registration of all guns, brings the country in line with regulations in most industrialized countries. In fact, many other countries have recently introduced legislative reforms aimed at tightening domestic controls over guns even further.

Although domestic controls can affect the supply of guns, efforts are being co-ordinated increasingly on an interjurisdictional basis to reduce the illegal trafficking of firearms. For example, the UN Crime Prevention and Criminal Justice Commission recently adopted a resolution encouraging all countries who have not done so to strengthen firearms controls, and the Organization of American States adopted a convention and model regulations restricting the import and export of firearms.

Much of the research on firearms controls has originated in and focused on the United States, where the problem is particularly acute. More research on the international context would be helpful both to understand better the shape of the problem and to explore potential solutions.

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Book Review

A Life Course Approach to Chronic Disease Epidemiology

Edited by Diana Kuh and Yoav Ben-Shlomo
Oxford: Oxford University Press, 1997; xviii + 317 pp;
ISBN 0 19 262782 1; $121.50 (CAN)

This book addresses the question of whether and to what extent we may be "programmed" for specific chronic diseases from early life, including gestation, and/or whether and to what extent adult chronic disease reflects cumulative differential lifetime exposure to damaging physical and social environments. The notion that unfavourable circumstances in early life could adversely affect health in adulthood has been held for some time, but the current prevailing belief is that chronic diseases in adulthood are the consequences of adult life style choices and exposures.

This outlook began to change with the discovery that the process of atherosclerosis begins in childhood. For example, over three quarters of young soldiers killed in the Korean war had gross evidence of coronary disease; the arteries of three-year-old children contain fatty streaks; blood pressure and cholesterol levels in individuals "track" from childhood to early adult life; overweight children are at greater risk of becoming overweight adults; and lifelong smoking, dietary and exercise habits are acquired in childhood and adolescence.

David Barker, in England, and Anders Forsdahl, in Norway, are credited with reviving the early life hypothesis in the late 1970s and early 1980s by their work examining the relationships between birthweight and other indicators of fetal nourishment and later chronic disease patterns. Barker has generated a number of hypotheses to explain how undernutrition during different trimesters of pregnancy programs an individual's adult risk of coronary heart disease, stroke, non-insulin-dependent diabetes and chronic bronchitis. Forsdahl's theory links deprivation in adolescence followed by later affluence with coronary heart disease risk.

This book explores a variety of early life events and critically examines the strength of the evidence for a relationship to later chronic disease, specifically cardiovascular disease, cancer, diabetes and insulin action, respiratory and allergic diseases, and blood pressure. It begins with an historical perspective on the "life course" hypothesis, which is both well written and interesting, pointing out that "classic" risk factors for cardiovascular disease, such as smoking, hypertension, raised cholesterol and lack of exercise, are limited in predicting individual risk and only partially explain the striking and well-documented social and geographic inequalities in the distribution of chronic disease. This has stimulated interest in genetic markers, other adult risk factors related to the psychosocial environment, more detailed assessment of adult dietary intake and possible risk factors in childhood.

Four chapters then discuss the specific diseases mentioned above. The treatment is somewhat inconsistent across these chapters, with the section on cancer being the weakest and the one on diabetes the most technical and detailed. Some of the intriguing relationships discussed are the inverse relationship between birthweight and both non-insulin-dependent diabetes and cardiovascular disease, a positive association between infant mortality in the past and subsequent adult mortality from heart disease, and an inverse relationship between mean adult or child height and coronary heart disease.

A third section, containing two chapters, presents an interesting discussion of the complexities of biological and social processes in disease induction. The one chapter deals with the role of nutrition and other factors on fetal growth and development, and the other chapter describes social pathways between childhood and adult health.

A fourth section of three chapters is a good presentation of disease patterns, specifically time trends, geography and migration and socio-economic differentials. This section provides good illustrative material for teaching concepts central to population epidemiology.

A particularly interesting chapter that also provides good teaching material is the second last one, which addresses the question "Should we intervene to improve fetal growth?" This chapter builds on the observed association between low birthweight and coronary heart disease, presenting actual calculations of the reduction in coronary heart disease that could be achieved by interventions to raise birthweights. Fortunately, it also addresses the potential for harm.

The strength of this book is that it provokes thought about the origins of chronic disease, suggests new approaches to identifying particularly susceptible individuals and encourages the identification of optimal points in the life course for possible preventive interventions. In particular, the chapter on diabetes suggests several opportunities for contributions from molecular epidemiology.

Overall rating:	Good
Strengths:	A balanced look at an intriguing approach to chronic disease epidemiology
	Consistency across chapters, with each providing an overview, introduction and conclusions
	Less overlap and greater coherence among chapters than is often the case in books that are a collection of essays written by different authors
	Good referencing
Weaknesses:	Relatively high frequency of typographic errors
	One table that begins on the right page and continues on the overleaf, making it almost impossible to decipher
	Relatively costly
	A relatively weak treatment of cancer
	Some inconsistency in the depth of treatment across diseases

Audience:	Practising epidemiologists interested in exploring novel hypotheses of chronic disease etiology
	Teachers of epidemiology

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New Publications

Atlas of Mortality in Europe: Subnational patterns 1980/1981 and 1990/1991

WHO Regional Publications, European Series, No 75
Copenhagen: WHO Regional Office for Europe, 1997;
245 pages including 150 maps and charts in full colour
(available in English only);
ISBN 92 890 1339 7; $189 (CAN) / $135 (US) / 150 (Sw fr); Order no 1310075

Since its creation, the World Health Organization (WHO) has had the primary goal of securing the best possible level of health for all people. It forms alliances wherever possible to help in the work, and monitors progress towards its goal by gathering and issuing statistics on disease and death. This Atlas thus derives from long-standing WHO goals, tasks and methods. In creating this book, however, the four main partners—the WHO European Centre for Environment and Health, the United Nations Economic Commission for Europe, and the Central Bureau of Statistics and the National Institute of Public Health and the Environment in the Netherlands—have taken important steps forward.

The Atlas does more than give national averages for all the main causes of death in the WHO European Region; it gives data on regions within countries and shows changes in mortality at this level between 1980/1981 and 1990/1991. Further, it literally draws pictures of health in Europe, presenting the data collected in vivid and informative maps and bar charts. By showing differences in mortality from various causes in the European Region, the Atlas also indicates areas in which more study is needed to determine both the reasons for these differences and the most appropriate action to reduce them.

As well as resting on important principles of WHO's work for health for all, this Atlas can contribute to progress towards the goal. It offers substantial food for thought and action by policy makers, professionals and anyone else interested in health and equity.

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NOTICE!
Canadian Cancer Statistics 1998

National Cancer Institute of Canada
Toronto (Ontario), 1998

Canadian Cancer Statistics 1998 is now accessible on the Internet at **http://www.cancer.ca/stats**.

You can download and/or print any sections, graphs, tables, etc. or all of this document from the above Web site.

If you would like to receive a hard copy of this publication, contact
your local office of the Canadian Cancer Society,
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Abstract Reprints

The association between physical activity and prostate cancer was evaluated in the trial-based cohort of the Alpha-Tocopherol, Beta-Carotene (ATBC) Cancer Prevention Study (n = 29,133). During up to nine years of follow-up, 317 men developed incident prostate cancer. The relationship between occupational, leisure, and combined activity and prostate cancer was assessed in multivariate Cox regression models that adjusted for intervention group, benign prostatic hyperplasia, age, smoking, and urban residence. Compared with sedentary workers, relative risks (RR) and 95 percent confidence intervals (CI) for occupational walkers, walker/lifters, and heavy laborers were 0.6 (CI = 0.4–1.0), 0.8 (CI = 0.5–1.3), and 1.2 (CI = 0.7–2.0), respectively. Among working men, leisure activity (active *cf* sedentary) was associated inversely with risk (RR = 0.7, CI = 0.5–0.9). This inverse association for leisure activity was observed, with the exception of heavy laborers, for all occupational activity levels, and was strongest among walkers compared with men sedentary at work and leisure, and to a lesser degree among walker/lifters. These results are consistent with a protective effect of physical activity on prostate cancer.

To examine the relation between fatal prostate cancer and family history of prostate cancer in a first-degree relative, we analyzed data from a prospective mortality study of 481,011 men with no history of cancer at enrollment in 1982. During 9 years of follow-up, 1,922 deaths from prostate cancer occurred. Results from Cox proportional hazard models showed that family history of prostate cancer was related to fatal prostate cancer [rate ratio (RR) = 1.60; 95% confidence interval (CI) = 1.31–1.97]; men with two or more affected relatives had a greater than threefold increase in risk (RR = 3.19; 95% CI = 1.51–6.71). Men whose relatives were diagnosed with prostate cancer before age 65 years (RR = 2.03; 95% CI = 1.33–3.09) had a greater effect of family history than men whose relatives were diagnosed at older ages (RR = 1.50; 95% CI = 1.17–1.91). Rate ratios did not increase with decreasing age of the study participants. The 60% increase in risk for men with at least one affected relative is lower than that reported in previous studies.

A lower risk of prostate cancer among diabetics has been suggested by several but not all studies. However, the studies have not always accounted for time since diagnosis of diabetes mellitus, or have not examined confounding factors such as diet and diagnostic bias. We thus examined this relationship in the Health Professionals Follow-Up Study from 1986 and 1994, in which 1,369 new cases of non-stage Al prostate cancer were documented in 47,781 men. A prior history of a diagnosis of diabetes (mostly adult-onset) was associated with a reduced risk of prostate cancer (multivariate relative risk [RR] = 0.75; 95 percent confidence interval [CI] = 0.59–0.95) controlling for age, body mass index (wt/ht^2) at age 21, and, in 1986, race, vasectomy, and intakes of total energy, total fat, calcium, fructose, and lycopene. After excluding the first year of follow-up after the diagnosis of diabetes, the RR was 0.63 (CI = 0.54–0.89). Prostate cancer was not reduced in the first five years after diagnosis (RR = 1.24, CI = 0.87–1.77), but was lower in the next five years (RR = 0.66, CI = 0.39–1.10) and lowest after 10 years (RR = 0.54, CI = 0.37–0.78); *P*-value for trend across time = 0.004. Similar associations were noted for advanced cases. Detection bias was unlikely to account for our findings. The basis of this relationship is unclear but may reflect hormonal changes related to diabetes, perhaps low testosterone levels.

This paper reports an examination of cognitive processes used by 178 women aged 50 years and older in retrieving information about the frequency with which they received Papanicolaou smears, mammograms, and clinical breast examinations. Women were selected from a health maintenance organization in which they had been enrolled for at least 5 1/2 years. The literature suggested that reporting of regular events such as these kinds of tests is likely to be based on schemas, which is an estimation technique in which events are reported in a format with generic content. Thus, if the procedure is believed to occur annually, the respondent will report receiving five tests in 5 years. The study attempted to evaluate whether use of episodic recall, in which respondents are forced to report individual events, would be more accurate than reports based on estimation using a schema format. The results indicated that most of the errors occurred in Papanicolaou smear reporting, which is consistent with the literature, and that the fewest errors occurred with mammograms. Regardless of the questionnaire format,

respondents persisted in using schemas based on the date of annual physical examination. Most reporting errors occurred because the interval between examinations was estimated incorrectly.

5. Prevalence and predictors of health risk behaviours during early pregnancy: Saskatoon Pregnancy and Health Study

Nazeem Muhajarine, Carl D'Arcy, Lindsay Edouard
Can J Public Health *1997;88(6):375–9*

Canadian data on prenatal exposure to alcohol, tobacco, psychoactive drugs, and caffeine are sparse. This study presents prevalence rates in Saskatoon for these four risk behaviours during the first trimester of pregnancy and their associations with sociodemographic factors. Personal interviews were conducted with 605 pregnant women (83% participation rate). The most commonly used substance was caffeine (87%), followed by alcohol (46%), tobacco (30%), and psychoactive drugs (7%). Overall, 36% of women reported using two substances, 16% three, and 4% all four substances. In general, risk behaviours were more prevalent among women with lower education and income levels, Aboriginal or Métis background, those not living with a partner, those with previous births, and, in some cases, younger women. The findings illuminate the needs of particular groups of pregnant women and the importance of understanding maternal risk behaviour within the structural and cultural realities of women's lives.

6. Estimation of breast cancer risk by women aged 40 and over: a population-based study

N Hébert-Croteau, P Goggin, N Kishchuk
Can J Public Health *1997;88(6):392–6*

Objective: Identify factors associated with knowledge of breast cancer and estimation of risk.

Methods: Telephone survey of 412 women aged 40 and over, living in Montreal and selected by random digit dialing.

Results: The majority of the respondents had recently been exposed to some information on breast cancer, but only a third quoted the average lifetime probability estimate of about 1 in 10. Older individuals systematically considered themselves at low risk (odds ratio (OR) of perceiving risk as lower than average for women aged 50 or over versus under 50: 2.6, 95% confidence interval: (1.5, 4.6)). In addition, both a first-degree family history of breast cancer (OR: 5.3 (1.7, 17.0)) and a recent mammogram (OR: 3.0 (1.4, 6.2)) were strongly associated with a woman's probability of perceiving herself at high risk.

Conclusions: Information campaigns should emphasize the frequency of breast cancer in different age groups and the strength of the established associations with specific risk factors. Better knowledge of risk could promote sustained participation in breast screening programs.

7. Second primary cancers related to smoking and treatment of small-cell lung cancer

Margaret A Tucker, Nevin Murray, Edward G Shaw, David S Ettinger, Mack Mabry, Martin H Huber, Ronald Feld, Frances A Shepherd, David H Johnson, Stefan C Grant, Joseph Aisner, Bruce E Johnson
J Natl Cancer Inst *1997;89(23):1782–8*

Background: An increased risk of second primary cancers has been reported in patients who survive small-cell carcinoma of the lung. The treatment's contribution to the development of second cancers is difficult to assess, in part because the number of long-term survivors seen at any one institution is small. We designed a multi-institution study to investigate the risk among survivors of developing second primary cancers other than small-cell lung carcinoma. *Methods:* Demographic, smoking, and treatment information were obtained from the medical records of 611 patients who had been cancer free for more than 2 years after therapy for histologically proven small-cell lung cancer, and person-years of follow-up were cumulated. Population-based rates of cancer incidence and mortality were used to estimate the expected number of cancers or deaths. The actuarial risk of second cancers was estimated by the Kaplan-Meier method. *Results:* Relative to the general population, the risk of all second cancers among these patients (mostly non-small-cell cancers of the lung) was increased 3.5-fold. Second lung cancer risk was increased 13-fold among those who received chest irradiation in comparison to a sevenfold increase among nonirradiated patients. It was higher in those who continued smoking, with evidence of an interaction between chest irradiation and continued smoking (relative risk = 21). Patients treated with various forms of combination chemotherapy had comparable increases in risk (9.4- to 13-fold, overall), except for a 19-fold risk increase among those treated with alkylating agents who continued smoking. *Implications:* Because of their substantially increased risk, survivors should stop smoking and may consider entering trials of secondary chemoprevention.

8. Mortalité attribuable au tabagisme au Québec

Benoît Lévesque, Louis Rochette, Suzanne Gingras
Can J Public Health *1998;89(1):28–32*

In industrialized countries, tobacco smoking is the main cause of preventable morbidity and premature deaths. Although mortality attributable to smoking has already been estimated for the population of the province of Québec, it has never been studied on a regional basis. We calculated the mortality attributable to smoking by socio-sanitary regions of the province of Québec for 10 fatal diseases positively associated with smoking. The calculations were made for the years 1984 through 1993 taking into account Canadian Census data (demographic variables), the Santé-Québec survey (prevalence of smoking), the death registry of the "Bureau de la statistique du Québec" (mortality data), and the American cohort of the "Cancer Prevention Study II" (relative risks). For the diseases investigated, 24,637 and 62,711 deaths were attributable to smoking for women and men respectively during the period studied, thus representing 29.4% and 51.2% of attributable percentages. There is no statistical difference between the regions, which indicates a general problem for all the province. These data again confirm the incredible impact of smoking on public health. The struggle against smoking should be a primary area for action for the benefit of all Quebecers.

9. L'usage de la cigarette au Québec de 1985 à 1994 : une comparaison avec le Canada

J Aubin, L Caouette

Smoking is responsible for the highest number of avoidable illnesses and deaths in Canada. Cigarette smoking declined considerably in the adult population between 1965 and 1986, but what has happened over the past decade? Quebec and Canadian public surveys were used to compare types of cigarette use in Quebec and Canada between 1985 and 1994, as well as to compare them by sex.

In recent years, the prevalence of smoking has increased among Quebec men only. Differences between Quebec and Canada can be seen in the evolution of the quit rate and the prevalence of smokers. There does not appear to be any indication that differences in cigarette smoking between Quebec and Canada are being eradicated. In Quebec, the evolution of this habit differs according to sex, which indicates that certain factors affect men and women differently. The public survey data make it possible to follow trends in the medium and long term, whereas it is difficult to accurately track the evolution of cigarette smoking from one year to the next, given the small size of the samples in each region and the slow evolution of behaviour.

10. Relations of cigarette smoking and dietary antioxidants with placental calcification

Lisa M Klesges, David M Murray, Judith E Brown, Suzanne P Cliver, Robert L Goldenberg

Associations between maternal cigarette smoking and accelerated placental maturation measured as tissue calcification have been reported. The authors sought to address whether intakes of the dietary antioxidants, vitamin C, alpha-tocopherol, and beta-carotene, were related to placental calcification of the maternal surface and villi in a cohort of smokers and nonsmokers at risk for delivering small-for-gestational age infants. Gross and histologic examination of placentas were used to determine calcification at the surface ($n = 1,213$) and villus sites ($n = 730$), respectively, in a prospective study of black and white women who delivered singleton births between December 1985 and October 1988 at the University of Alabama at Birmingham Hospital in Birmingham, Alabama. Controlling for race and gestational age, likelihood of surface and villus calcification increased as smoking levels increased. Significant reductions in villus calcification were related to alpha-tocopherol intake after controlling for smoking and gestation while intakes of beta-carotene and vitamin C were related to significant reductions in calcification for black but not white women. Surface calcification was not found to be related to antioxidant intake. The authors' findings confirm a pathologic relation between smoking and placental calcification and suggest that dietary antioxidants may reduce villus calcification.

11. The effect of water fluoridation on the bone mineral density of young women

Cathy M Arnold, Donald A Bailey, Robert A Faulkner, Heather A McKay, Robert G McCulloch

Introduction: Osteogenic effects of therapeutic fluoride have been reported; however, the impact of exposure to low level water fluoridation on bone density is not clear. We investigated the effect of long-term exposure to fluoridated water from growth to young adulthood on bone mineral density (BMD).

Methods: BMD was measured in 24 healthy women from Regina (fluoride 0.1 mg/L) and 33 from Saskatoon (fluoride 1.0 mg/L), with no differences between groups for height, weight, lifestyle or dietary factors.

Results: Saskatoon women had significantly higher mean BMD at total anterior-posterior lumbar spine (APS) and estimated volumetric L3 (VLS), with no difference at total body (TB) or proximal femur (PF).

Conclusion: Exposure to water fluoridation during the growing years may have a positive impact on axial spine bone density in young women.

12. Alzheimer's disease as a cause of death in the United States

Donna L Hoyert, Harry M Rosenberg

Objective. To describe the scope of mortality from and trends in Alzheimer's disease, to show how Alzheimer's disease ranks as a leading cause of death, to describe a methodological change regarding ranking, and to discuss issues related to the reporting of Alzheimer's disease on death certificates.

Methods. The authors analyzed mortality data from the National Vital Statistics System.

Results. Alzheimer's disease has increasingly been reported as a cause of death on death certificates in the United States; however, this increase may represent a variety of factors including improved diagnosis and awareness of the disease or changes in the perception of Alzheimer's disease as a cause of death. In 1995, Alzheimer's disease was identified as the underlying cause of 20,606 deaths. Overall, Alzheimer's disease was the 14th leading cause of death in 1995; for people 65 years of age or older, it was the 8th leading cause of death. Both death rates and cause-of-death ranking differed by selected demographic variables.

Conclusions. In recognition of the importance of the condition as a major public health problem, Alzheimer's disease was added to the list of causes eligible to be ranked as leading causes of death in the United States beginning with mortality data for 1994. Several issues need to be kept in mind in interpreting mortality data on Alzheimer's disease, including how diagnoses

realized from this intervention; acute care costs savings exceeded $70,000/year and costs from death decreased by $5 million. An intervention involving enforcement on snowmobile trails can reduce the incidence of injuries from snowmobile-related trauma.

13. Can we monitor socioeconomic inequalities in health? A survey of U.S. health departments' data collection and reporting practices

Nancy Krieger, Jarvis T Chen, Gregory Ebel
Public Health Rep *1997;112(6):481–90*

Objective. To evaluate the potential for and obstacles to routine monitoring of socioeconomic inequalities in health using U.S. vital statistics and disease registry data, the authors surveyed current data collection and reporting practices for specific socioeconomic variables.

Methods. In 1996 the authors mailed a self-administered survey to all of the 55 health department vital statistics offices reporting data to the National Center for Health Statistics (NCHS) to determine what kinds of socioeconomic data they collected on birth and death certificates and in cancer, AIDS, and tuberculosis (TB) registries and what kinds of socioeconomic data were routinely reported in health department publications.

Results. Health departments routinely obtained data on occupation on death certificates and in most cancer registries. They collected data on educational level for both birth and death certificates. None of the databases collected information on income, and few obtained data on employment status, health insurance carrier, or receipt of public assistance. When socioeconomic data were collected, they were usually not included in published reports (except for mothers educational level in birth certificate data). Obstacles cited to collecting and reporting socioeconomic data included lack of resources and concerns about the confidentiality and accuracy of data. All databases, however, included residential addresses, suggesting records could be geocoded and linked to Census-based socioeconomic data.

Conclusions. U.S. state and Federal vital statistics and disease registries should routinely collect and publish socioeconomic data to improve efforts to monitor trends in and reduce social inequalities in health.

14. The effect of a community-based police surveillance program on snowmobile injuries and deaths

Brian H Rowe, Sandra A Therrien, Jennifer A Bretzlaff, Vic S Sahai, K V Nagarajan
Can J Public Health *1998;89(1):57–61*

Serious snowmobile injuries are preventable and associated with late-night travel, alcohol use, and speed. We studied the effectiveness of a community-based policing (STOP) program in the prevention of serious injuries related to snowmobile trauma in Sudbury, Ontario. Volunteers were trained in police protocol and were appointed special constables to increase policing on snowmobile trails from 1993–95. Snowmobile admissions and deaths in Sudbury were examined; the pre- (1990–1992) and post-(1993–1995) STOP seasons were compared.

In the pre-STOP period, 102 injuries, 87 admissions, and 15 deaths occurred compared to 57 injuries (p = 0.0004), 53 admissions (p = 0.00001) and 4 deaths (p = 0.13) in the post-STOP period. All other event and demographic features of the crashes remained similar. Significant economic savings were

15. A descriptive epidemiology of sport and recreation injuries in a population-based sample: results from the Alberta Sport and Recreation Injury Survey (ASRIS)

W Kerry Mummery, John C Spence, Joanne A Vincenten, Donald C Voaklander
Can J Public Health *1998;89(1):53–6*

The 1996 Alberta Sport and Recreation Injury Survey is a retrospective study describing the annual incidence of injuries in the province of Alberta resulting from sport and recreational involvement. Data was collected by means of a telephone survey using random digit dialling techniques to obtain a representative sample of Albertans in the winter of 1995-96. The sample produced a total of 3,790 respondents from 1,478 households evenly split between genders, with an age range of 6 to 93 years. The survey asked information regarding medically attended, non-fatal injuries resulting from sport and recreational activities. Findings reveal an annual incidence of sport or recreational injuries of 11%. Among those reporting a sport or recreational injury, the most common types of injuries were a sprained/torn ligament (31%), strained/pulled muscle (19%), and fracture (13%). The most common bodily locations of injuries were the knees (21%) and the ankle (14%).

16. The mental health of informal caregivers in Ontario: an epidmiological survey

Jeanette J Cochrane, Paula N Goering, Joy M Rogers
Am J Public Health *1997;87(12):2002–7*

Objectives. This study describes the mental health status, disability, physical health, and mental health service utilization of informal caregivers under the age of 65 in the province of Ontario.

Methods. The study analyzed data collected in the 1991 province-wide, population-based mental health supplement to the Ontario Health Survey. Diagnoses from the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition, revised, were generated on the basis of a structured diagnostic interview. Caregivers and noncaregivers are compared here on past-year prevalence of psychiatric disorder, physical illness, disability, and utilization of mental health services. The possible confounding effects of age, sex, employment status, and economic disadvantage are explored.

Results. Informal caregivers (n = 1219) constituted 15.0% of the sample. Caregivers had higher rates of affective (6.3% vs 4.2%) and anxiety (17.5% vs 10.9%) disorders than noncaregivers and used health services for mental health problems at nearly twice the rate.

Conclusions. Documentation of the prevalence of caregiving and the increased prevalence of psychiatric disorders, disability, and service utilization among caregivers is of critical importance as governments continue to move toward community-based care. To accomplish this goal, the needs of caregivers must be acknowledged and met by the establishment of appropriate and readily accessible support services.

Calendar of Events

April 21–23, 1998
Vancouver,
 British Columbia

"The Role of Cancer Registries in Cancer
 Surveillance and Control"
Annual Meeting of the North American
 Association of Central Cancer Registries
Hosted by the British Columbia Cancer Registry

Information
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April 22–24, 1998
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6th International Symposium: Epidemiology and
 Occupational Risks
Organized by the International Research
 Section of the International Social Security
 Association (ISSA)

Information
Symposium Secretariat
Allgemeine Unfallversicherungsanstalt
Kongressbüro
Adalbert-Stifter-Strasse 65
A-1200 Vienna, Austria
Tel: +43-1-33 111 537
Fax: +43-1-33 111 469
E-mail: presse@auva.or.at

April 26–29, 1998
Lucerne, Switzerland

UICC Breast Cancer Meeting
International Meeting on the Psycho-social
 Impacts of Breast Cancer

Information
Jeanne Froidevaux
Swiss Cancer League
Effingerstrasse 40, CH-3001
Berne, Switzerland
Tel: +41 31 389 91 14
Fax: +41 31 389 91 60
E-mail: froidevaux@swisscancer.ch
Web site: http://www.swisscancer.ch

April 27–28, 1998
Ottawa, Ontario

1998 Canadian Pharmacoepidemiology Forum
Canadian Association for Population
 Therapeutics
(April 26: session on "Risk Communication")

Information
Ineke Neutel
Bureau of Drug Surveillance
Therapeutics Program, Health Canada
Ottawa, Ontario
Tel: (613) 954-6788
Fax: (613) 957-0335

April 27–29, 1998
Toronto, Ontario

"Health & Safety '98 Conference and Trade
 Show"

Information
Rabiya Shaikh
OHS CANADA Magazine
Tel: (416) 442-2090
or Surinder Sehdev
Industrial Accident Prevention
 Association (IAPA)
Tel: (416) 506-8888

April 27–30, 1998
Tampa, Florida
USA

"Balance, Support, and Prevention: Act Today
 for a Better Tomorrow"
1998 CDC – Diabetes Translation Conference
Centers for Disease Control and Prevention

Information
Margaret R Hurd
Centers for Disease Control
NCCDPHP, DDT
4770 Buford Hwy NE, Mailstop K-10
Atlanta, Georgia
USA 30341-3724
Tel: (770) 488-5505
Fax: (770) 488-5966
E-mail: mrh0@cdc.gov

May 10–12, 1998 **Toronto, Ontario**	Pulse '98 Conference "The Business of Canada's Health Care Future"	*Information* Institute for International Research 60 Bloor Street West, Suite 1101 Toronto, Ontario M4W 3B8 Tel: (416) 928-1770 *or* 1-800-461-2398 Fax: (416) 928-2994

May 17–20, 1998
Amsterdam,
  The Netherlands
	4th World Conference on Injury Prevention
	  and Control
		Information
		Conference Secretariat
		Van Namen & Westerlaken Congress
		  Organization Services
		PO Box 1558, 6501 BN NIJMEGEN
		The Netherlands
		Tel: (31-24) 3234471
		Fax: (31-24) 3601159
		E-mail: reg.fowoco.nw@prompt.nl

May 26–29, 1998 **Boston, Massachusetts** **USA**	24th Annual Educational Conference National Cancer Registrars Association	*Information* Victoria Bowen NCRA Executive Office PO Box 15945-295 Lenexa, KS 66285-5945 Tel: (913) 438-6272 Fax: (913) 541-0156 E-mail: ncra_usa.org

June 7–10, 1998
Montreal, Quebec
	"Best Practices in Public Health: An Essential
	Contribution, A Promising and Exciting Future"
	  Canadian Public Health Association
	  89th Annual Conference
	Co-sponsored by the *Association pour la santé
	  publique du Québec*
		Information
		CPHA Conference Department
		400—1565 Carling Avenue
		Ottawa, Ontario K1Z 8R1
		Tel: (613) 725-3769
		Fax: (613) 725-9826
		E-mail: conferences@cpha.ca

June 21–26, 1998
San Juan, Puerto Rico
	16th World Conference on Health Promotion
	  and Health Education
	"New Horizons for Health: From Vision
	  to Practice"
	Organized by the School of Public Health
	  (University of Puerto Rico), World Health
	  Organization, UNESCO and UNICEF
		Information
		Conference Secretariat
		Tel: (787) 274-0582
		Fax: (787) 754-6621
		E-mail: HIR_Arroyo@RCMACA.
		        UPR.CLU.EDU

June 24–26, 1998 **Chicago, Illinois** **USA**	Society for Epidemiologic Research (SER) 31st Annual Meeting	*Information* Conferences, University of Utah 1901 E. South Campus Dr., #2174 Salt Lake City, Utah USA 84112 Tel: (801) 581-5809 Fax: (801) 581-3165 E-mail: confer@admin.dce.utah.edu Web site: http://conferences.utah.edu/ser

August 15–19, 1998
Boston, Massachusetts
USA
	10th Conference of the International Society for
	  Environmental Epidemiology and
	8th Conference of the International Society
	  of Exposure Analysis
		Information
		Carol Rougvie, Conference Secretariat
		JSI Research and Training Institute
		44 Farnsworth Street
		Boston, Massachusetts
		USA 02210-1211
		Tel: (617) 482-9485
		Fax: (617) 482-0617
		E-mail: isee&isea98@jsi.com
		Web sites:
		http://www.med.ualberta.ca/PHS/ISEE
		http://www.iit.edu/~butler/isea

August 23–28, 1998 **Rio de Janeiro, Brazil**	17th International UICC Cancer Congress	*Information* Congrex do Brasil Ltda. Av. Presidente Wilson 164/9 andar RJ 20030-020 Rio de Janeiro, Brasil Tel: +55 21 - 509 40 80 Fax: +55 21 - 509 14 92 E-mail: congress@uicc.org

September 9–12, 1998
Lethbridge, Alberta
	"Health in Rural Settings: From the Ground Up"
	International Multi-disciplinary Conference
	  on Rural Health
		Information
		Health in Rural Settings Conference
		c/o The University of Lethbridge
		Box #7, 4401 University Drive
		Lethbridge, Alberta T1K 3M4
		
		Regional Centre for Health Promotion
		  & Community Studies
		Tel: (403) 382-7152
		or School of Health Sciences
		Tel: (403) 329-2699
		Fax: (403) 329-2668
		E-mail: rhc@uleth.ca
		Web site: http://home.uleth.ca/rhc

November 1–4, 1998 **Victoria, BC**	"Itch '98: New Partnerships — Better Care" International Conference on Information Technology Issues in Community Health Web site: http://www.hsd.uvic.ca/HIS/ITCH/ITCH.htm	*Information* ITCH '98 c/o Dr Paul Fisher School of Health Information Science PO Box 3050 University of Victoria Victoria, BC V8W 3P5 Tel: (250) 721-8576 Fax: (250) 721-1457 E-mail: his@hsd.uvic.ca
November 2–4, 1998 **Barrie, Ontario**	"Valuing the Public's Health ... It's Everybody's Business" 49th Annual Ontario Public Health Association Conference Hosted by Simcoe County District Health Unit *Call for abstracts—deadline: **May 15, 1998***	*Information* Heather Edgar Simcoe County District Health Unit Tel: (705) 721-7330 Fax: (705) 721-1495 *or* Tel: (416) 367-3313 (OPHA)

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