

# Chronic Diseases

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## *in Canada*

Volume 27, Number 4, 2007



Public Health  
Agency of Canada

Agence de santé  
publique du Canada

Canada

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## Chronic Diseases in Canada

a publication of the  
Public Health Agency of Canada

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Indexed in Index Medicus/MEDLINE, PAIS (Public  
Affairs Information Service) and Scopus.

This publication is also available online at  
[www.phac-aspc.gc.ca/publicat/  
cdic-mcc/index.html](http://www.phac-aspc.gc.ca/publicat/cdic-mcc/index.html)

# The burden of adult obesity in Canada

Wei Luo, Howard Morrison, Margaret de Groh, Chris Waters, Marie DesMeules, Elaine Jones-McLean, Anne-Marie Ugnat, Sylvie Desjardins, Morgan Lim and Yang Mao

## Abstract

*Obesity is a major public health problem associated with a wide range of health problems. This study estimates the prevalence of obesity, calculates the proportion (or population-attributable fraction [PAF]) of major chronic diseases which is attributable to obesity, estimates the deaths attributable to it and projects its future prevalence trends. In Canada, the overall age-standardized prevalence proportion of obesity has increased from 10% in 1970 to 23% in 2004 (8% to 23% in men and 13% to 22% in women). The increasing prevalence of obesity was observed for all five age groups examined: 20-34, 35-44, 45-54, 55-64 and 65+. On average, the PAF of prevalence of selected major chronic diseases which is attributable to obesity from 1970 to 2004 has increased by 138% for men and by 60% for women. Overall, in 2004, 45% of hypertension, 39% of type II diabetes, 35% of gallbladder disease, 23% of coronary artery diseases (CAD), 19% of osteoarthritis, 11% of stroke, 22% of endometrial cancer, 12% of postmenopausal breast cancer, and 10% of colon cancer could be attributed to obesity. In 2004, 8,414 (95% CI: 6,881-9,927) deaths were attributable to obesity. If current obesity prevalence trends remain unchanged, the prevalence proportion of obesity in Canada is projected to reach 27% in men and 24% in women by the year 2010. These increases will have a profound impact on the treatment needs and prevalence of a wide variety of chronic diseases, and also on the health care system in terms of capacity issues and resource allocation.*

**Key words:** adult obesity, Canada, chronic disease

## Introduction

The total direct health care cost of obesity in 2001 was estimated to be over \$1.6 billion, which corresponded to 2.2% of the total health care expenditures for all diseases in Canada.<sup>2</sup>

Obesity results from the interaction of many factors, including genetic, metabolic, behavioural and environmental influences. The rapidity with which obesity is increasing suggests that behavioural and environmental influences, rather

than biological changes, have fuelled the epidemic.<sup>3,4</sup> Likely a combination of increasing energy consumption and decreasing energy expenditure has led to a positive energy balance and a marked increase in excess weight in our society.<sup>5,6</sup>

According to Statistics Canada, estimated per capita energy consumption in Canada has increased from 2,362 kilocalories per day in 1992 to 2,788 kilocalories per day in 2002.<sup>7</sup> In 2000/01, when asked about their leisure-time activity, more than half (53.5%) of Canadians aged 12 years and

over indicated that they were physically inactive.<sup>8</sup> Although the prevalence of leisure-time physical activity has increased over time,<sup>9,10</sup> possible explanations for the current obesity epidemic are decreased physical activity in the workplace and reduced energy expenditure through improved technology, as well as suburban environments favouring the automobile.

Obesity increases the risk for many chronic diseases, including hypertension, type II diabetes, gallbladder disease, coronary artery diseases (CAD), osteoarthritis and certain types of cancer.<sup>11,12</sup>

## Hypertension

There is compelling evidence indicating that excess body weight is associated with hypertension in men and women,<sup>13-15</sup> with relative risks (RR) ranging from 2.2 to 5.7 for obese persons.<sup>16-19</sup> In other words, obese individuals are 2.2 to 5.7 times more likely than non-obese individuals to become hypertensive. Weight gain promoting a rise in blood pressure may involve a variety of mechanisms, including increased insulin resistance.<sup>20</sup>

## Type II diabetes

Obesity is reported to be one of the strongest lifestyle-related factors for developing type II diabetes.<sup>21</sup> Obesity results in insulin resistance—a state linked to both impaired glucose tolerance and type II diabetes. The published RR of type II diabetes associated with obesity varies

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dramatically across studies, ranging from 1.4 to 47.1.<sup>13,22,23</sup>

### **Gallbladder disease**

Obesity is a well-established risk factor for gallbladder disease.<sup>24-27</sup> A plausible biological mechanism is that obese people have supersaturated gallbladder bile, which appears to account for their predisposition to cholesterol cholelithiasis.<sup>28</sup>

### **Coronary heart disease and stroke**

Obesity can lead to coronary heart disease by enlarging the muscles of the heart in an attempt to improve the blood supply to the larger body mass.<sup>29</sup> It is possible that the apparently independent influence of obesity on the incidence of CVD is related to the distribution and severity of atherosclerotic lesions.<sup>30</sup> Obesity co-exists with a variety of cardiovascular risk factors, but has been independently related to greater cardiovascular risk in a variety of observational studies.<sup>31-33</sup> The RR of obesity and coronary heart disease is reported to range from 1.3 to 3.6.<sup>13,21,34-37</sup> Obesity is also associated with an increased risk of both ischemic and hemorrhagic stroke.<sup>13,16,38,39</sup>

### **Osteoarthritis**

Obesity may increase the risk of osteoarthritis because adiposity is associated with abnormal levels of hormones and growth factors, greater bone mineral density and other metabolic intermediaries. Indeed, the association between obesity and osteoarthritis in non-weight-bearing joints is evidence for a systemic effect of adiposity.<sup>40</sup> The development of osteoarthritis in weight-bearing joints such as knee and hip could potentially be a result of the long-term mechanical efforts of regularly displacing excess weight. Osteoarthritis is a leading cause of chronic pain and mobility limitation, especially in older people.<sup>40-42</sup>

### **Endometrial cancer**

Numerous epidemiological studies have shown a positive association between endometrial cancer and excess weight.<sup>43-46</sup> The increased risk of endometrial cancer with obesity is believed to relate to higher production rates

and increased concentrations of endogenous estrogen which induces endometrial cell proliferation.<sup>47,48</sup>

### **Breast cancer**

The relationship between excess weight and breast cancer risk differs between pre- and post-menopausal women. Excess weight has a strong positive association with post-menopausal breast cancer and an inverse correlation with pre-menopausal breast cancer risk.<sup>49,50</sup> One study suggested that the increase in breast cancer risk with increasing weight among postmenopausal women is largely the result of the associated increase in estrogens, particularly bio-available estradiol.<sup>51</sup> The relative risk of post-menopausal breast cancer related to obesity is generally weak, ranging from 1.1 to 1.9 in major cohort studies.<sup>52</sup>

### **Colon cancer**

There is growing evidence that obesity is positively associated with colon cancer,<sup>53-55</sup> and that the association is stronger in men than in women.<sup>56,57</sup> One proposed biological mechanism is that adiposity increases blood insulin levels,<sup>58</sup> which lower insulin-like growth factor (IGF) binding protein 1 and which may subsequently lead to increased levels of free IGF-1.<sup>59</sup> IGF-1 has been positively associated with the risk of colon cancer in men<sup>60</sup> and women.<sup>61</sup> The International Agency for Research on Cancer has estimated that overweight and obesity cause 11 % of colon cancer cases.<sup>62</sup>

To better understand the impact of obesity on these chronic diseases as well as on premature mortality, we conducted this study to

- 1) describe the prevalence of obesity (BMI  $\geq 30$  kg/m<sup>2</sup>) from 1970 to 2004 using six cross-sectional surveys conducted in Canada;
- 2) estimate the PAF of major chronic diseases attributable to obesity in Canada;
- 3) estimate the number of deaths attributable to obesity in 2004;
- 4) project future prevalence of obesity to 2010.

## **Methods**

### **Data sources**

Trends in the prevalence of obesity were examined using six national population-based health surveys conducted in Canada between 1970 and 2004 that measured respondents' height and weight: the 1970-1972 Nutrition Canada Survey,<sup>63</sup> the 1978-1979 Canada Health Survey,<sup>64</sup> the 1981 Canada Fitness Survey,<sup>65</sup> the 1988 Campbell's Survey on Well-being in Canada,<sup>66</sup> the 1986-1992 Canadian Heart Health Surveys<sup>67</sup> and the 2004 Canadian Community Health Survey (CCHS), Cycle 2.2.<sup>1</sup> Table 1 presents the sample sizes, response rates and national prevalence proportions of obesity for adults over 20 years of age for each survey. National data was restricted to data from the ten provinces as territorial data was not available. A detailed description of the health surveys prior to 1997 is available elsewhere.<sup>68</sup> The CCHS, Cycle 2.2, was a relatively new cross-sectional survey which focused on nutrition. The survey targets respondents from all age groups living in private-occupied dwellings in the ten provinces. Excluded from the sampling frame were residents of the three territories, persons living on Indian reserves or Crown lands, persons living in institutions, full-time members of the Canadian Forces and residents of some remote regions. It is estimated that the sampling strategy employed for the survey covered 98% of the population living in the provinces. All respondents aged two and older were asked their permission to have their height and weight measured by the interviewer. In total, 63 % of respondents had both their height and weight measured by trained interviewers.

Summary relative risk estimates for obesity and eight chronic diseases (hypertension, type II diabetes, gallbladder disease, coronary heart disease, osteoarthritis, stroke, postmenopausal breast cancer and colon cancer) were obtained from a recent meta-analysis undertaken by Katzmarzyk and Janssen<sup>2</sup> (Table 2). This meta-analysis was based on prospective longitudinal studies conducted in the US and a few European countries. These RRs were used



to calculate the PAF, and, in turn, the average increase of the PAF between 1970 and 2004 for those chronic conditions.

The Canadian Mortality Database<sup>2</sup> was used to calculate the total number of deaths for age groups 25-59, 60-69 and 70+ in 2002. Since the most recent data available for mortality was for 2002, we estimated the number of deaths in 2004 by applying the 2002 mortality rate to the 2004 Canadian population.

To estimate the total number of deaths attributable to obesity, we used RRs denoting the risk of death due to obesity by age from a recent US study published by Flegal et al.<sup>69</sup> and the age-gender-specific prevalence proportion of obesity using the CCHS, Cycle 2.2, with measured height and weight.

### Measures

Individuals' height and weight were measured by trained interviewers using standardized procedures for all six health surveys included in the study. The prevalence of obesity is commonly assessed by using the body mass index (BMI), defined as weight in kilograms divided by the square of the height in metres (kg/m<sup>2</sup>). We used the BMI classification system developed by the World Health Organization (WHO), which defines obesity as a BMI of 30 kg/m<sup>2</sup> or greater, a cutoff applied to both genders among adults aged 18 and older.<sup>11</sup>

### Statistical analysis

Calculating the population-attributable fraction of obesity:

A population-attributable fraction (PAF) combines the population prevalence of a risk factor with the relative risk of incidence associated with that risk factor. The PAF was calculated for obesity using the following equation:

$$\text{PAF \%} = [P(RR-1)] / [1 + P(RR-1)]$$

where P is the population prevalence proportion of obesity (gender specific, aged 20 and over) and RR is the summary relative risk of developing a certain disease among obese individuals.

The PAF indicates the proportion of an outcome that can be attributed to a certain risk factor, and thus the proportion that can potentially be prevented by modifying the risk factor, assuming a causal relation between the risk factor and the outcome. It is an important epidemiologic indicator for policy purposes because it illustrates both the impact of a hazardous exposure on a whole population and the potentially preventable proportion of a disease associated with a given risk factor.<sup>70</sup>

Estimating the number of deaths attributable to obesity:

The equation for calculating the total number of deaths attributable to obesity is as follows:

$$Y = \sum D_{i,j} * F_{i,j,k}$$

where Y is the total deaths attributable to obesity, D is the total number of deaths by age (i) and gender (j) and F is the PAF by age, gender and BMI categories (k).

The lower and upper ranges of total deaths attributable to obesity were calculated by applying lower and upper ranges of the PAF, which were derived by applying lower and upper boundaries of 95% CI of age-gender-specific prevalence of obesity in 2004. Standard errors and coefficients of variation for age-gender-specific prevalence of obesity were estimated using the bootstrap technique, which takes into account survey design effects.<sup>1</sup>

Projecting future prevalence of obesity:

The future prevalence of obesity was projected using a log-linear regression model fit to prevalence trends of obesity data extracted from surveys with measured height and weight data, conducted between 1970 and 2004. Age-specific trends were initially investigated using joinpoint analysis, a type which is commonly used to describe changing trends in disease rates. (Please refer to Kim et al.<sup>71</sup> for more information on joinpoint analysis.) These analyses did not detect any variations in trends for the period of our survey data, which supported the decision to use logistic regression to project obesity prevalence. The SAS (version 8.02, SAS Institute, Inc, Cary, North Carolina) LOGISTIC procedure was used to fit a model with a trend term common for all age groups and a model with age-specific trends. The more conservative estimate of future prevalence was selected from the model using a common trend term for all age-specific estimates.

## Results

Overall age-standard prevalence proportions of obesity increased from 10% in 1970 to 23% in 2004 (Figure 1 and Table 1) (8% to 23% in men, 13% to 22% in women). The rise in obesity was observed for all five age groups examined: 20-34, 35-44, 45-54, 55-64 and 65+ (Figure 2). The prevalence proportion of obesity generally increased with age, with people aged 55-64 having the highest proportion of obesity (except in 1970 and 1981), and people in the youngest age group (20-34) having the lowest proportion. If current obesity prevalence trends remain unchanged, by the year 2010, the prevalence proportion of obesity will likely reach 27% in men and 24% in women (Figure 3).

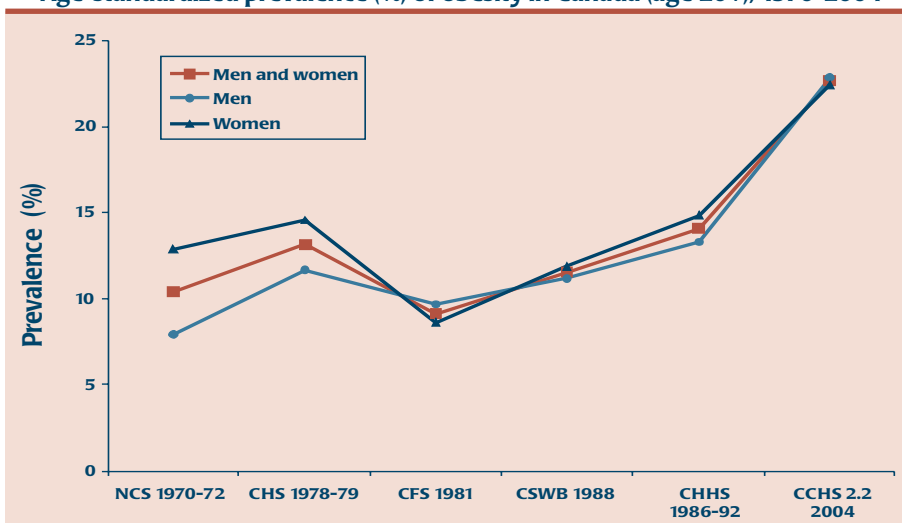
**TABLE 1**  
Age-standardized\* national prevalence proportion of obesity among adults  
(aged 20+ years) in surveys conducted in Canada, 1970-2004

Survey name	Sample size	Response rates (%)**	Height and weight collection method	% Obese BMI kg/m <sup>2</sup> ≥ 30.0		Overall obesity (%)
				Men	Women	
Canadian Community Health Survey, 2.2, 2004	18,668	77	Measured	22.91	22.47	22.69
Canadian Heart Health Surveys, 1986-1992	22,314	78	Measured	12.91	15.48	14.20
Campbell's Survey on Well-being in Canada, 1988	3,445	61	Measured	11.22	11.90	11.56
Canada Fitness Survey, 1981	17,468	76	Measured	9.68	8.66	9.17
Canada Health Survey, 1978-1979	20,351	86	Measured	11.72	14.56	13.14
Nutrition Canada Survey, 1970-1972	10,103	46	Measured	7.94	12.89	10.42

\* Standardized to 1991 Canadian population

\*\*Kendall O, Lipskie T, MacEachern S. Canadian Health Surveys, 1950-1997. Chronic Diseases in Canada. 1997;18(2):70-90.

**FIGURE 1**  
Age-standardized prevalence (%) of obesity in Canada (age 20+), 1970-2004



Source: 6 surveys with measured height and weight (age 20+), Statistics Canada.

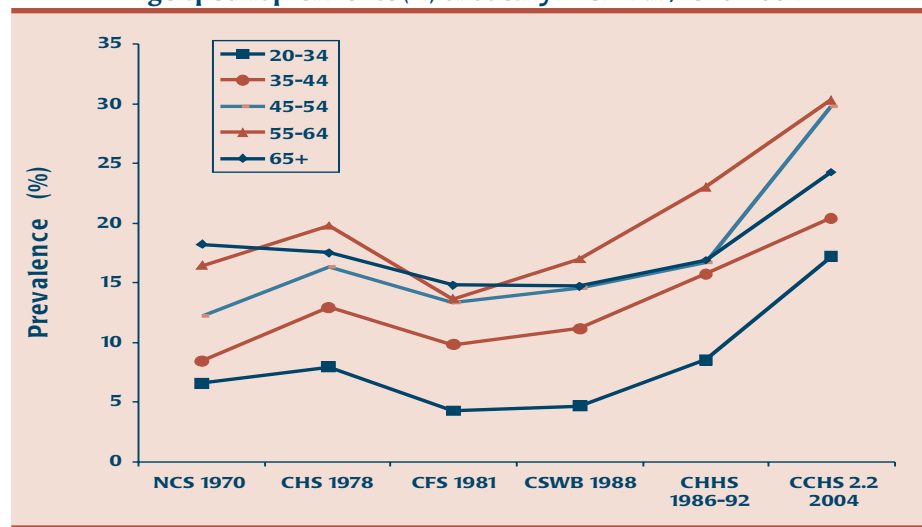
NCS - Nutrition Canada Survey;  
CHS - Canada Health Survey;  
CFS - Canada Fitness Survey;  
CSWB - Campbell's Survey on Well-being in Canada;  
CHHS - Canadian Heart Health Survey;  
CCHS - Canadian Community Health Survey, 2.2

**TABLE 2**  
Population-attributable fraction (PAF\*) (%) by gender and relative risk (RR) of obesity (BMI ≥ 30 kg/m<sup>2</sup>) and major chronic diseases in Canada (1970-2004)

	Hypertension		Type II diabetes disease		Gallbladder disease	
Relative risks (RR) of obesity and chronic diseases	4.5		3.73		3.3	
Year and survey	Men	Women	Men	Women	Men	Women
2004 Canadian Community Health Survey, 2.2 (CCHS)	44.97	45.46	38.93	38.93	35.24	35.68
1986-1992 Canadian Heart Health Survey (CHHS)	31.91	34.83	26.77	29.42	23.78	26.24
1988 Campbell's Survey on Well-being in Canada (CSWB)	26.69	27.25	22.11	22.61	19.51	19.96
1981 Canada Fitness Survey (CFS)	24.76	22.89	20.42	18.80	17.97	16.50
1978-1979 Canada Health Survey (CHS)	28.57	23.78	12.41	28.51	21.03	25.40
1970-1972 Nutrition Canada Survey (NCS)	22.24	31.76	18.24	26.64	15.99	23.66
<b>PAF Increase (%) from 1970 to 2004</b>	<b>102.24</b>	<b>43.10</b>	<b>113.47</b>	<b>47.89</b>	<b>120.33</b>	<b>50.83</b>

\*PAF = [P (RR-1)/(P(RR-1) + 1)] \* 100 (where P = prevalence proportion of obesity; RR = relative risk of obesity and certain diseases [incidence], obtained from a recent meta-analysis,<sup>2</sup> except for endometrial cancer<sup>53</sup>)

**FIGURE 2**  
**Age-specific prevalence (%) of obesity in Canada, 1970-2004**



Source: 6 surveys with measured height and weight (age 20+), Statistics Canada.

NCS - Nutrition Canada Survey;

CHS - Canada Health Survey;

CFS - Canada Fitness Survey;

CSWB - Campbell's Survey on Well-being in Canada;

CHHS - Canadian Heart Health Survey;

CCHS - Canadian Community Health Survey, 2.2

On average, the PAF of obesity and major chronic diseases has increased by 138% from 1970 to 2004 for men and by 60% for women, assuming a causal relation between obesity and the diseases. The PAF % for each chronic disease is shown in Table 2. Overall, in 2004, 45% of hypertension, 39% of type II diabetes, 35% of gallbladder disease, 23% of CAD, 19% of osteoarthritis, 11% of stroke, 22% of endometrial cancer, 12% of postmenopausal breast cancer and 10% of colon cancer were attributable to obesity. In comparison, 27% of hypertension, 22% of type II diabetes, 20% of gallbladder disease, 12% of CAD, 10% of osteoarthritis, 5% of stroke, 14% of endometrial cancer,

9% of postmenopausal breast cancer and 5% of colon cancer were attributable to obesity in 1970.

Table 3 presents the overall number of deaths attributable to obesity in 2004. The result shows that approximately 8,400 (95% CI: 6,900-9,900) deaths in 2004 were attributable to obesity (4% of total deaths in 2004).

## Discussion

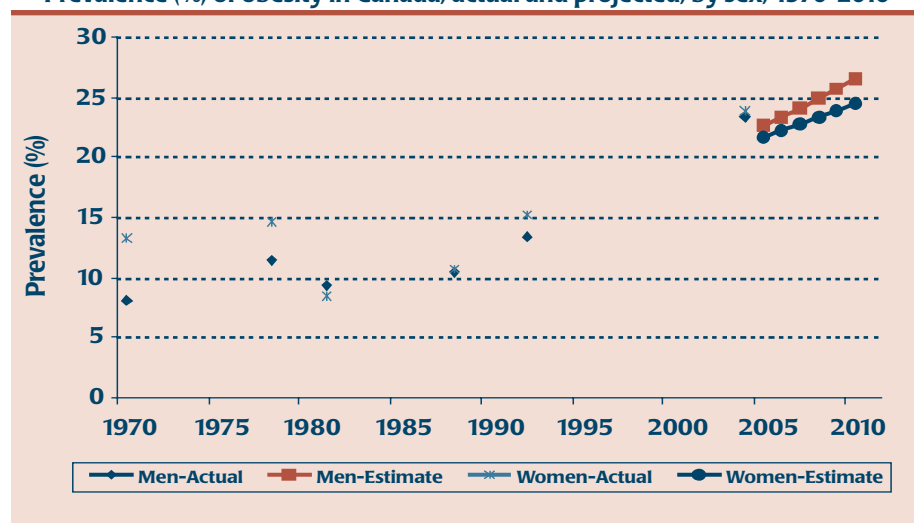
This study highlights the dramatic rise in the prevalence of obesity between 1970 and 2004 in Canadian adults for all age groups. As well, it highlights the impact

of this trend on population mortality. The PAF of obesity related to major chronic diseases more than doubled from 1970 to 2004 for men and increased almost 40% for women. Moreover, 8,414 deaths (4% of total deaths) were attributable to obesity in 2004.

In our study, the prevalence of obesity was estimated using measured height and weight data obtained from six population-based cross-sectional surveys conducted in Canada between 1970 and 2004, including the CCHS, Cycle 2.2. Other national health surveys, such as previous versions of the CCHS, have assessed obesity using self-reported heights and weights; however,

PAF (%) of obesity and chronic diseases											
Coronary artery disease		Osteoarthritis		Stroke		Endometrial cancer		Postmenopausal breast cancer		Colon cancer	
2.24		1.99		1.50		2.52		1.47		1.45	
Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
22.45	22.79	18.78	19.08	10.45	10.64	n/a	22.08	n/a	12.09	9.51	9.68
14.24	15.92	11.70	13.13	6.27	7.09	n/a	15.38	n/a	9.75	5.68	6.43
11.42	11.71	9.33	9.58	4.94	5.08	n/a	11.29	n/a	7.64	4.47	4.59
10.44	9.51	8.51	7.74	4.49	4.07	n/a	9.17	n/a	6.24	4.06	3.68
12.41	15.34	10.17	12.64	5.41	6.81	n/a	14.81	n/a	10.19	4.89	6.17
9.20	14.16	7.48	11.64	3.92	6.24	n/a	13.66	n/a	8.93	3.55	5.65
144.08	61.01	150.92	63.95	166.38	70.62	n/a	61.58	n/a	71.07	168.14	71.38

**FIGURE 3**  
**Prevalence (%) of obesity in Canada, actual and projected, by sex, 1970-2010**



Source: 6 surveys with measured height and weight (age 20+), Statistics Canada.

1970 - Nutrition Canada Survey; 1978 - Canada Health Survey; 1981 - Canada Fitness Survey;  
1988 - Campbell's Survey on Well-being in Canada; 1992 - Canadian Heart Health Survey;  
2004 - Canadian Community Health Survey

that approach results in a significant underestimation in the prevalence of obesity.

Summary relative risks derived from a published meta-analysis were used to calculate the PAF of obesity and related

major chronic diseases. We deem this approach to be more appropriate than relying on RR estimates from a single study.<sup>2</sup>

One limitation of this study may be the tendency for obese individuals to self-select out of any study in which their weight will be measured. This may have resulted in an underestimation of the prevalence of obesity. Evidence of this is suggested by the finding that the prevalence of obesity was substantially lower for two fitness surveys when compared to other general health surveys that measured height and weight.<sup>72</sup> As a result, the future projected prevalence proportion of obesity could also be an underestimate. However, the projected future prevalence trends should be interpreted with caution, as we did not consider the possible impact of any future interventions to reduce obesity. Although there may be a natural ceiling on the proportion of people who will become obese in any given population, it is unlikely that this will be reached in Canada soon, given that our projections indicate that by the year 2010, obesity levels in Canada will

**TABLE 3**  
**Deaths attributable to obesity in Canada in 2004 (95% confidence intervals)**

Age in years	BMI (kg/m <sup>2</sup> ) 30 to < 35		BMI (kg/m <sup>2</sup> ) ≥ 35		BMI (kg/m <sup>2</sup> ) ≥ 30	
25-59						
Prevalence proportion	15.7	(13.9;17.4)	8.9	(7.6;10.2)		
RR*	1.2		1.83			
PAF**	3.0	(2.7;3.4)	6.9	(5.9;7.8)		
# of deaths attributable to obesity	1,027.0	(914;1,139)	2,323.0	(2,008;2,634)	3,350.0	(2,922;3,773)
60-69						
Prevalence proportion	19.7	(16.3;23.0)	8.3	(6.3;10.3)		
RR*	1.13		1.63			
PAF**	2.5	(2.1;2.9)	5.0	(3.8;6.1)		
# of deaths attributable to obesity	782.0	(651;912)	1,557.0	(1,190;1,914)	2,339.0	(1,841;2,826)
70+						
Prevalence proportion	18.2	(15.6;20.7)	6.5	(4.8;8.3)		
RR*	1.03		1.17			
PAF**	0.54	(0.47;0.62)	1.1	(0.8;1.4)		
# of deaths attributable to obesity	900.0	(775;1,026)	1,825.0	(1,343;2,302)	2,725.0	(2,118;3,328)
Total # of deaths attributable to obesity	2,709.0	(2,340;3,077)	5,705.0	(4,541;6,850)	8,414.0	(6,881;9,927)

\* RR = Relative risk obtained from Flegal et al.<sup>69</sup>

\*\*PAF = Population-attributable fraction



be comparable to those currently seen in the United States, and will still be much lower than those currently observed in many countries around the world.

A further limitation of our projections is that they are based on data from only six surveys. Additional analyses were undertaken using data from seven further surveys with self-reported height and weight in the projection model, in which the ratio of the age-sex-specific prevalence of obesity of CCHS (2004) and CCHS (2003) was used to adjust for underreporting. The results were comparable to those based on projections using measured data only (data not shown).

The prevalence trend of obesity observed in this study is similar to previous published Canadian studies.<sup>73,74</sup> Any modest discrepancies in prevalence estimates could be due to our use of “health share files” which are available to Health Canada and the Public Health Agency of Canada for all the surveys, while other researchers have relied on “public use files”. Our estimated PAF for obesity and chronic diseases are comparable with those from other studies.<sup>2,12,74</sup> The average percentage increase of PAF from 1970 to 2004 for each chronic condition was calculated. However, we are not able to adjust for comorbidity of obesity and various chronic conditions due to unavailable data. The results should be interpreted with caution.

We estimated that 8,414 (95% CI: 6,881-9,927) deaths were attributable to obesity in 2004. This is significantly higher than the estimate of 4,321 in 2000 by Katzmarzyk and Ardern.<sup>75</sup> However, their estimate only looked at deaths up to age 65 years, whereas the current estimate was based on all adult mortality. As well, Katzmarzyk and Ardern used self-reported heights and weights to ascertain the BMI, compared to the measured heights and weights used in this analysis.

Estimating the number of deaths attributable to obesity is challenging because of the lack of clarity surrounding the most appropriate relative risks. We used relative risks generated by Flegal and colleagues, which are based on the follow-up of the

National Health and Nutrition Examination Survey (NHANES) I, II and III cohorts. The relative risks were adjusted for all confounding factors (e.g., race, gender, smoking status). Moreover, the NHANES surveys are nationally representative, and the heights and weights of cohort members were measured. These risks were lower than those based only on the follow-up of NHANES-I and other cohorts<sup>69,76</sup>; as a result, Flegal estimated fewer deaths attributable to obesity.<sup>69</sup> Flegal attributed the lower NHANES-II and NHANES-III cohort relative risks to the impact of medical advances in the treatment of obesity-related comorbid conditions and outcomes.<sup>69</sup> In other words, the obese of today are less likely to die of coronary heart disease than the obese of 40 years ago because of advances in the treatment of comorbid conditions such as dyslipidemia and hypertension, and because of improved treatments such as cardiac revascularization.

We are not able to adopt Flegal’s multi-risk approach to estimate the RR and number of excess deaths associated with obesity. However, we estimated the 95% CI of age-gender-specific prevalence of obesity by applying the bootstrap technique, which takes into account the survey design effects. As a result, the ranges of the PAF for obesity and chronic conditions and the ranges of deaths attributable to obesity were calculated. This approach is different from estimating the standard error of the PAFs in its assumption that the prevalence of exposure is measured without error and then by calculating the PAFs using both the lower and upper confidence limits of the RRs. Our results indicated the minimum and maximum number of deaths attributable to obesity, depending on either the lower or higher prevalence of obesity of the study population.

Flegal and colleagues generated their RR using all participants of the NHANES cohorts,<sup>69</sup> whereas most other studies have restricted their analyses to subpopulations such as non-smokers.<sup>76,77</sup> However, for the purposes of estimating the burden of obesity for an entire population, it is most appropriate to use relative risks that apply to the entire population. There is evidence to show that relative risks for obesity are

higher for non-smokers than for smokers.<sup>77</sup> For example, the RR among the obese in the entire population of the Nurses Health Study was 1.1. Whereas when current and former smokers were excluded, the RR increased to 1.8.<sup>26</sup>

There is considerable controversy regarding the relationship between being overweight (but not obese) and the risk of premature mortality. Many studies have observed a modest increased risk of mortality among the overweight,<sup>77-81</sup> whereas the recent follow-up of the NHANES cohorts by Flegal<sup>69</sup> noted a significantly decreased risk. Because of these uncertainties, we have chosen not to attempt to estimate deaths associated with being overweight.

## Conclusion and recommendations

International data indicate that the epidemic of obesity is not restricted to developed nations but is in fact a global health problem.<sup>82</sup> The International Obesity Task Force has concluded that the current obesity pandemic reflects the profound changes in society over the past 20-30 years which have created an environment that promotes a sedentary life style and diets rich in energy-dense foods.<sup>5</sup>

Our study estimated that over 8,000 deaths are attributable to obesity each year in Canada. This is more than the combined number who die annually from motor vehicle traffic accidents, suicide, homicide and HIV infection in Canada, but is significantly less than the estimated 47,000 deaths per year resulting from tobacco use.<sup>83</sup> However, the adverse effects of obesity extend far beyond premature mortality since they also include increased levels of disability and morbidity, and decreased quality of life.

## Acknowledgements

The Canadian Mortality data were provided to PHAC from the Canadian Vital Statistics databases at Statistics Canada. The cooperation of the provincial and territorial vital statistics registries, which supply the data to Statistics Canada, is gratefully acknowledged.

This analysis is based on the Statistics Canada microdata which contains anonymized data collected in the Canadian Community Health Surveys. All computations on these microdata were prepared by PHAC and the responsibility for the use and interpretation of these data is entirely that of the authors. The authors wish to thank Stephanie Jackson, Jane Boswell-Purdy, Paula Stewart and Peter Walsh for their comments on an earlier draft of this manuscript.

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# Prostate cancer risk and diet, recreational physical activity and cigarette smoking

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## Abstract

Associations between prostate cancer and dietary factors, physical activity and smoking were assessed based on data from a population-based case-control study. The study was conducted among residents of northeastern Ontario. Cases were identified from the Ontario Cancer Registry and diagnosed between 1995 and 1998 at ages 50 to 84 years ( $N = 752$ ). Male controls were identified from telephone listings and were frequency matched to cases on age ( $N = 1,613$ ). Logistic regression analyses investigated history of diet, physical activity and smoking as potential risk factors. Tomato intake had a significant positive association with prostate cancer risk for highest versus lowest quartiles ( $OR = 1.6$ ; 95% CI: 1.2-2.0). Associations were observed for tomato or vegetable juices and ketchup ( $OR = 1.5$ ; 95% CI: 1.2-1.9;  $OR = 1.2$ ; 95% CI: 1.0-1.5, respectively). Neither other dietary variables nor smoking were associated with prostate cancer risk. Strenuous physical activity by men in their early 50s was associated with reduced risk ( $OR = 0.8$ ; 95% CI: 0.6-0.9). While the recreational physical activity association was consistent with results from previous studies, the tomato products association was not.

**Key words:** case-control study, diet, physical activity, prostate cancer, smoking

## Introduction

Prostate cancer is the most common cancer in North American males and it is estimated that 20,700 men will be diagnosed with prostate cancer in Canada in 2006.<sup>1,2</sup> Many studies have investigated potential risk factors for prostate cancer, but the only currently established ones are age, family history of prostate cancer and race.<sup>3</sup> There have been inconsistent results in the literature regarding the roles of diet, physical activity and smoking.<sup>4-15</sup> The aim of the current study is to provide further information regarding the potential roles of diet, smoking and physical activity with respect to prostate cancer risk. The results reported here are from a large case-control study of males from northeastern Ontario where the original focus was the investigation of associations among occupational risk

factors and prostate cancer risk. Data about diet, physical activity and smoking were also obtained and results for these variables are reported here.

With respect to diet, fat and meat consumption have been suggested as potential risk factors.<sup>4</sup> Ecological studies show positive correlation between both meat and fat intake and prostate cancer incidence and mortality, but among several case-control and cohort studies the evidence has been equivocal.<sup>4</sup> Suggestion of an association between alcohol consumption and prostate cancer has also not been consistent among studies.<sup>11</sup> Tea consumption is hypothesized to have an inverse association with prostate cancer risk, but few epidemiologic studies in this regard have been performed; some of these indicate an inverse association

between tea consumption and prostate cancer risk while others show no association.<sup>10</sup> Consumption of fish has shown a potential inverse association that is not typically significant.<sup>6</sup> Consumption of fruit and vegetables is hypothesized to have an inverse association. However, in some epidemiologic studies, an inverse association is observed, while there is no association in others.<sup>9</sup> Tomatoes and tomato-based foods are a specific focus since decreased risk has more consistently been observed for high consumption of, primarily, processed tomato products.<sup>9</sup> Given the discrepancies among epidemiological studies of the association between diet and prostate cancer, the results of the large population-based case-control study provided here are important for accumulating evidence both for and against specific nutritional risk factors.

While smoking is an important risk factor for many cancers, associations between smoking and prostate cancer risk have not been consistently demonstrated.<sup>15</sup> In some studies, a lack of differentiation between current and former smokers is suggested to have led to some null results.<sup>15</sup> The data from our study are presented to further investigate the role of cigarette smoking.

A published review of the literature regarding physical activity and prostate cancer risk suggests a potential inverse association, but there are general problems with a lack of control for important confounders.<sup>14</sup> In the results reported here, control for important confounders is considered.

## Author References

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## Materials and methods

Ethics approval for the study was obtained from the Laurentian Hospital Research Ethics Board in Sudbury, Ontario, Canada.

Cases were men aged 50 to 84 years diagnosed between January 1995 and December 1998 with primary histologically confirmed prostate cancer (ICD9 185)<sup>16</sup> and who were identified in the Ontario Cancer Registry (OCR) as residents of northeastern Ontario. The completeness of the OCR is over 95%.<sup>17</sup> Before cases were approached for participation in the study, consent was obtained from the physician listed on the pathology report. Consenting physicians also provided contact information for patients. Cases received a letter describing the study, with telephone contact occurring approximately ten days after the mailing of the letter. Cases were included if they had a residential telephone and were alive at the time of interviewer contact. We excluded men aged 45 to 49 years (N=8) included in original data collection<sup>18</sup> to accommodate age-dependent physical activity variables and to remove men whose early onset prostate cancer may have been predominantly genetic.<sup>19</sup> Information on stage and/or grade of prostate tumours was not available nor was information on prostate-specific antigen (PSA) testing.

Controls were randomly selected from the northeastern Ontario population, based on residential telephone listings, and were 2:1 frequency matched to cases based on five-year age groups. Telephone contact with controls was attempted weekly for five weeks followed by a six-week waiting period, after which contact was again attempted weekly for another five weeks. If contact with a specific control was not successful after this second set of attempts, no further attempt was made.

A mailed questionnaire, sent to consenting cases and controls, was used to collect diet, cigarette smoking and recreational physical activity history. Initially, telephone interviews were used to collect data, but

after eleven months subjects were offered the option of providing questionnaire responses by telephone or by mail. This was due to early high-refusal rates for the telephone questionnaire and a preference expressed by participants to self-complete questionnaires and return them by mail. Prior to this change in the data collection procedure, it was determined by means of a small pilot study that either method of data collection resulted in a similar amount of response information.

Questions about diet and recreational physical activity were derived from the National Enhanced Cancer Surveillance Study in Canada.<sup>19</sup> The questionnaire also included questions regarding socio-demographic variables, physical and health variables, family history of prostate cancer and, as its major focus, occupational history. Telephone follow-up to respondents was conducted if clarification of responses was necessary. Telephone follow-up to non-respondents continued approximately every two weeks for a total of three attempted contacts.

The diet section of the study questionnaire was initially developed for the National Enhanced Cancer Surveillance Study in Canada primarily using two validated instruments: the reduced Block questionnaire and the Nurses Health Study questionnaire.<sup>19</sup> Dietary variables measuring weekly intake of 71 foods two years prior to questionnaire completion were derived based on identified frequency of consumption of specified usual serving sizes. Frequencies of usual serving consumption offered in the questionnaire were as follows: never or less than once per month; 1 to 3 per month; 1 per week; 2 to 4 per week; 5 to 6 per week; 1 per day; 2 to 3 per day; 4 to 5 per day, and; 6 or more per day. Usual serving size specifications were offered in both imperial and metric amounts where appropriate. Caloric intake and fat intake were estimated by adding weekly kilojoules (KJ) and grams of fat, respectively, for each food item in the questionnaire. Quartiles of intake were defined based on the

distribution in the controls. The exception is the variable for tofu: It was defined as "never/ever use" since there was a general lack of intake of this specific food type in the study subjects.

Physical activity indicator variables were based on the frequency and intensity (moderate versus strenuous) of recreational activities for at least twenty minutes based on three times of life (mid-teens, early 30s and early 50s). Specific frequencies of at least twenty minutes of exercise for each of strenuous and moderate activity offered in the questionnaire were as follows: less than once per month; 1 to 3 times per month; 1 to 2 times per week; 3 to 5 times per week; and more than 5 times per week. The questionnaire also offered several examples each of strenuous and moderate activities. For each time of life, a variable was derived that indicated whether or not a subject then participated in relevant activities at least three times per week.

The study questionnaire inquired about cigarette smoking history for individuals who had smoked at least once a day for six months or longer. Details regarding average number of cigarettes per day and number of years of smoking, and information about quitting were requested. From this information, smoking was defined as follows: never; former; or current cigarette smoker. In addition, quartiles of pack-years of cigarette smoking were computed based on the pack-years distribution among controls.

Other variables that were possible confounders were also considered. These "core" potential confounders comprised age (continuous), an indicator of family history of prostate cancer (among first-degree relatives), quartiles of recent (five years ago) body mass index (BMI) and, as surrogates for socio-economic status, level of education (elementary, secondary school, post-secondary school) and type of occupation of longest duration (categorized as blue collar versus white collar, based on standard occupational classification code).

Although race is an established risk factor for prostate cancer, race was not included as one of the core potential confounders since 97% of the study subjects were Caucasian.

Initial descriptive analyses involved examining frequencies and cross-tabulations for variables of interest. Multivariate logistic regression analyses were performed to obtain odds ratio (OR) estimates adjusted for age and other potential confounding variables. These variables included core potential confounders described above as well as variables from each of the diet, physical activity and smoking analyses. If these additional confounders did not change odds ratio estimates for risk factors by more than 15%, they were deleted from the model. All analyses were conducted using version 8.2 of SAS software.<sup>20</sup> Approximate 95% confidence intervals (CI) were computed to provide information on the variability associated with the modeling results. Global *p*-values were used to investigate the significance of categorical variables. Any subjects with missing values for any variables included in a specific model were not included in the estimation of parameters for that particular model.

## Results

Response rates in the original study were 73.6% (760 of 1,033 eligible) for cases and 47.5% (1,632 of 3,433 eligible) for controls.<sup>18</sup> Among the eligible cases and controls who did not respond, 85.4% and 92.4% refused, respectively. The average time between case diagnosis and questionnaire completion was thirteen months; 75% of questionnaires were completed within seventeen months of diagnosis.

Frequencies and age-adjusted odds ratio estimates for study participant characteristics that were included as core potential confounders appear in Table 1. There was a significant positive association between prostate cancer risk and family history of prostate cancer ( $p < 0.0001$ ), a result not surprising, given that family history is an established risk factor. The remaining variables were not significantly

**TABLE 1**  
Frequencies, age-adjusted odds ratio estimates (AOR\*), approximate 95% confidence intervals (CI) and global *p*-values for selected potential confounders for controls (N=1,613) and cases (N=752) from a population sample of northeastern Ontario men aged 50-84 years

Variable	Controls	Cases	AOR	95% CI	p-value
Age group					
50-54	69	25			
55-59	138	50			
60-64	271	134			
65-69	446	222			
70-74	389	181			
75-79	204	109			
80-84	96	31			
Family history of prostate cancer					< 0.0001
No	1,522	643	1.0		
Yes	91	109	2.8	2.1-3.8	
Body mass index (kg/m²)					0.1136
5 years ago					
Q1 (≤ 24)	398	180	1.0		
Q2 (24 to 27)	389	155	0.9	0.7-1.1	
Q3 (27 to 29)	390	211	1.2	0.9-1.5	
Q4 (> 29)	397	184	1.0	0.8-1.3	
Unknown	39	22			
Education					0.0846
Elementary	538	261	1.0		
Secondary	737	364	1.0	0.8-1.2	
Post-secondary	325	122	0.8	0.6-1.0	
Unknown	13	5			
Type of occupation					0.1163
White collar	704	302	1.0		
Blue collar	909	450	1.2	1.0-1.4	

\* All AOR estimates presented were adjusted for age and were calculated from valid responses (excluding missing data).

associated with risk of prostate cancer, although education was of borderline significance at the 5% level ( $p = 0.07$ ) and both BMI and type of occupation had *p*-values that were less than 0.15 (0.12 and 0.11, respectively).

Crude frequencies and adjusted odds ratio estimates for self-reported dietary exposures are presented in Table 2. The results presented in this table are based on multivariate models for each diet variable that included total energy along with core potential confounders. None of the physical activity and smoking variables acted as confounders. Overall, the odds

ratio estimate for the highest versus lowest quartile of weekly combined tomato product intake of 1.6 (95% CI: 1.2-2.0) showed a significant positive association. When specific components of this combined variable were explored, similar associations were observed both for tomato or vegetables juices and for ketchup (OR = 1.5; 95% CI: 1.2-1.9 and OR = 1.2; 95% CI: 1.0-1.5, respectively). In contrast, there was no significant association with prostate cancer risk found for the variable representing raw tomato consumption. All remaining specific dietary variables were not associated with risk of prostate cancer. In addition, it was noted that a positive

**TABLE 2**  
Frequencies, adjusted odds ratio estimates (AOR\*), approximate 95% confidence intervals (CI) and global *p*-values for diet variables based on intake two years prior to questionnaire completion for cases and controls from a population sample of northeastern Ontario men aged 50-84 years, where quartiles are based on the distribution for controls (continued)

Variable	Controls	Cases	AOR	95% CI	<i>p</i> -value
Total fat (g/week)					0.9248
0 - 274	395	171	1.0		
274.1 - 364.1	392	183	1.0	0.8-1.3	
364.2 - 475	394	184	1.0	0.7-1.3	
> 475	393	199	0.9	0.6-1.3	
Unknown	39	15			
Total energy (KJ per week)**					0.1267
0 - 44,707.1	392	161	1.0		
44,707.2 - 54,785.2	392	176	1.1	0.9-1.5	
54,785.3 - 66,331.3	396	187	1.1	0.9-1.5	
> 66,331.3	390	213	1.4	1.1-1.8	
Unknown	43	15			
Tomato or vegetable juices					0.0066
0	576	239	1.0		
0.1 - 0.5	469	197	1.0	0.8-1.3	
0.6 - 1.0	230	107	1.1	0.8-1.5	
> 1.0	302	194	1.5	1.2-1.9	
Unknown	36	15			
Tomatoes					0.7476
< 1.0	352	150	1.0		
1.0 - 2.9	382	168	1.1	0.8-1.4	
3.0	607	283	1.1	0.8-1.4	
> 3.0	248	142	1.2	0.9-1.6	
Unknown	24	9			
Ketchup					0.0124
0	452	206	1.0		
0.1 - 0.5	341	135	0.9	0.7-1.2	
0.6 - 2.9	309	113	0.8	0.6-1.1	
≥ 3.0	458	269	1.2	1.0-1.5	
Unknown	53	29			
All tomato-based foods					0.0007
0 - 2.0	374	145	1.0		
2.1 - 4.0	413	156	1.0	0.7-1.3	
4.1 - 7.5	404	177	1.1	0.8-1.4	
> 7.5	355	240	1.6	1.2-2.0	
Unknown	67	34			
Yellow vegetables					0.8823
< 1.0	234	108	1.0		
1.0	313	137	0.9	0.7-1.3	
1.1 - 3.0	504	245	1.0	0.7-1.3	
> 4.0	507	243	0.9	0.7-1.2	
Unknown	55	19			

association of total energy and prostate cancer risk was observed when comparing the highest versus lowest quartiles (OR = 1.4; 95% CI: 1.1-1.8).

Crude frequencies and adjusted odds ratio estimates for self-reported recreational physical activity appear in Table 3. The results presented in this table are based on separate multivariate models for each physical activity indicator that include core potential confounders. Neither diet nor smoking variables confounded these associations. Separate models were used due to concerns of strong associations among the physical activity indicators, although an overall multivariate model was fitted, including all variables, with no change in conclusions (data not shown). Of note is that strenuous activity in life's early 50s shows significantly reduced risk with an odds ratio estimate of 0.8 (95% CI: 0.6-0.9); the reduced risk associated with strenuous activity in early 30s was of borderline significance (OR = 0.9; 95% CI: 0.7-1.0). Only results for dichotomous strenuous physical activity indicator variables are reported since analyses of three-level categorical variables for strenuous activity versus moderate activity versus low activity resulted in the conclusion that moderate activity—relative to low activity—was not associated with prostate cancer risk in our data.

Crude frequencies and adjusted odds ratio estimates for self-reported cigarette-smoking-related exposures are presented in Table 4. None of the diet and physical activity variables confounded the results of these analyses. Although elevated odds ratio estimates were observed, neither of the smoking variables conferred a significant association, nor did additional separate analyses for filter and for non-filter cigarette smoke exposures (data not shown).

In Table 5, odds ratio estimates from a final logistic regression model are presented, including only the core potential confounding variables and the significant specific diet and recreational physical activity variables. As observed in previous tables, family history and tomato-

**TABLE 2 (continued)**  
**Frequencies, adjusted odds ratio estimates (AOR\*), approximate 95% confidence intervals (CI) and global *p*-values for diet variables based on intake two years prior to questionnaire completion for cases and controls from a population sample of northeastern Ontario men aged 50-84 years, where quartiles are based on the distribution for controls**

Variable	Controls	Cases	AOR	95% CI	<i>p</i> -value
Cruciferous vegetables					0.2692
< 1.0	340	176	1.0		
1.0	367	155	0.8	0.6-1.1	
1.1 - 3.0	441	218	1.0	0.7-1.2	
> 3.0	425	191	0.8	0.6-1.1	
Unknown	40	12			
Green leafy vegetables					0.7841
0	517	248	1.0		
0.1 - 0.5	426	178	0.9	0.7-1.1	
0.6 - 1.0	290	141	1.0	0.8-1.3	
> 1.0	343	168	1.0	0.8-1.3	
Unknown	37	17			
Fruit and fruit juice					0.9560
0 - 11	397	185	1.0		
11.1 - 19.5	396	173	1.0	0.7-1.2	
19.6 - 29.0	366	171	1.0	0.7-1.2	
> 29.0	384	199	1.0	0.8-1.3	
Unknown	70	24			
Rice and noodles					0.7698
0 - 1.0	533	230	1.0		
1.1 - 1.5	268	135	1.1	0.9-1.5	
1.6 - 3.5	390	188	1.1	0.9-1.4	
> 3.5	382	187	1.1	0.8-1.4	
Unknown	40	12			
Grain and cereals					0.5983
0 - 13.5	399	163	1.0		
13.6 - 22.5	379	185	1.1	0.8-1.4	
22.6 - 33	386	198	1.1	0.9-1.5	
> 33	382	179	1.0	0.7-1.3	
Unknown	67	27			
Tofu					0.5475
Never	1,472	685	1.0		
Ever	93	50	1.1	0.8-1.6	
Unknown	48	17			
Baked beans and lentils					0.7030
0	539	232	1.0		
0.1 - 0.5	722	342	1.1	0.9-1.3	
0.6 - 1.0	220	104	1.1	0.8-1.4	
> 1.0	96	61	1.3	0.9-1.8	
Unknown	36	13			

based products were associated with increased risk of prostate cancer, while strenuous recreational physical activity was associated with decreased risk.

## Discussion

Recent review articles present summaries of previous research regarding general risk factors for prostate cancer<sup>3,21,22</sup> and for specific exposures of diet,<sup>4-11,23-26</sup> physical activity<sup>13,14</sup> and smoking.<sup>15</sup> Total dietary fat has not been consistently found to be a risk factor for prostate cancer.<sup>4,5</sup> It is suggested that earlier studies identified dietary fat as a significant risk factor since total energy was not typically controlled for in these analyses.<sup>5</sup> In the current study, there was no significant association observed between dietary fat and prostate cancer, after controlling for total energy.

The only significant dietary exposures identified in this study involve consumption of tomato products, specifically tomato or vegetable juices and ketchup, and these positive associations contrast with some studies that have identified a negative association between tomato intake and prostate cancer risk.<sup>9,23,24,26</sup> The tomato associations in this study were examined for the possibility of confounding by—or interactions with—other variables (e.g., pasta), with no change in conclusion. It is important to note that in our study questionnaire, the three questions requesting information about consumption of tomato products involved servings of juices, ketchup and tomatoes. The question about tomatoes did not distinguish between processed and unprocessed tomatoes.

It has been hypothesized that lycopene is the compound in processed tomato products, such as tomato paste and tomato sauce, that may be negatively associated with prostate cancer risk. However, it has been reported that lycopene intake and lycopene blood levels do not strongly correlate.<sup>23</sup> In addition, while several studies have identified significant and non-significant negative associations for tomato products, some recent ones have

**TABLE 2 (continued)**  
**Frequencies, adjusted odds ratio estimates (AOR\*), approximate 95% confidence intervals (CI) and global *p*-values for diet variables based on intake two years prior to questionnaire completion for cases and controls from a population sample of northeastern Ontario men aged 50-84 years, where quartiles are based on the distribution for controls**

Variable	Controls	Cases	AOR	95% CI	<i>p</i> -value
<b>Fish</b>					
					0.7185
0	199	85	1.0		
0.1 - 0.5	497	222	1.0	0.7-1.4	
0.6 - 1.0	516	243	1.0	0.8-1.4	
> 1	356	187	1.2	0.8-1.6	
Unknown	45	15			
<b>Meat</b>					
					0.2374
0 - 3.0	390	184	1.0		
3.1 - 5.0	400	194	1.0	0.7-1.3	
5.1 - 7.5	380	151	0.8	0.6-1.0	
> 7.5	398	204	1.0	0.7-1.3	
Unknown	45	19			
<b>Processed meat</b>					
					0.1198
0 - 0.5	426	207	1.0		
0.6 - 1.5	422	169	0.8	0.6-1.0	
1.6 - 3.5	331	143	0.9	0.7-1.1	
> 3.5	393	218	1.1	0.8-1.4	
Unknown	41	15			
<b>Tea</b>					
					0.2677
0	438	198	1.0		
0.1 - 7.5	580	307	1.2	0.9-1.5	
7.6 - 18.75	434	180	0.9	0.7-1.2	
> 18.75	131	59	1.0	0.7-1.5	
Unknown	30	8			
<b>Coffee</b>					
					0.9276
0	105	51	1.0		
0.1 - 7.5	449	218	1.0	0.7-1.4	
7.6 - 18.75	730	336	0.9	0.6-1.3	
> 18.75	316	142	0.9	0.6-1.4	
Unknown	13	5			
<b>Alcohol (beer, wine, liquor)</b>					
					0.8821
0	415	198	1.0		
0.1 - 3.5	398	197	1.0	0.8-1.3	
3.6 - 11.0	367	163	0.9	0.7-1.2	
> 11.0	387	177	1.0	0.7-1.2	
Unknown	46	17			

\* Unless otherwise specified, adjusted for total energy, age, family history of prostate cancer, BMI 5 years ago, education, type of occupation

\*\* Adjusted for age, family history of prostate cancer, BMI 5 years ago, education, type of occupation

not identified this effect for either tomato products or lycopene exposure.<sup>27,28</sup> Our result is indicative of the need to further examine the nature and extent of any potential association between tomato product intake, with a focus on processed versus unprocessed tomatoes, and prostate cancer risk. Thus, the exact role of tomatoes could be elucidated by asking subjects of future studies about specific types of tomato products consumed.

An inverse association has been hypothesized between prostate cancer risk and consumption of plant-based foods.<sup>29</sup> The suggested mechanism is based on the protective effect of antioxidants against exposure to carcinogens. A possible weak inverse association between prostate cancer and vegetables has been found in the literature,<sup>9</sup> but our data indicate no association for all vegetables combined or separately for yellow, cruciferous and green leafy vegetables. Grains and cereals, baked beans and lentils, and tofu also were not associated with prostate cancer risk, nor was intake of rice and noodles. It is also suggested that fruit consumption is not likely related to risk of prostate cancer<sup>9</sup> and the results of this study support this.

Meat intake has been positively associated with prostate cancer risk in several studies,<sup>4</sup> with two studies of processed meat reporting no association.<sup>30,31</sup> It is suggested that these significant associations may be due to fat consumption associated with meat consumption or due to chemicals associated with meat preparation. In this study, there was no association observed between meat and prostate cancer risk and between processed meat and prostate cancer risk. As indicated previously, fat consumption was not identified either as a significant risk factor. A possible inverse association is suggested for fish intake due to the possible reduction of testosterone levels resulting from polyunsaturated n-3 fatty acids found in fish. However, reported study results<sup>6</sup> are inconsistent. In this study, no such association was observed.



**TABLE 3**  
Frequencies, adjusted odds ratio estimates (AOR\*), approximate 95% confidence intervals (CI) and global *p*-values for strenuous physical activity variables for cases and controls from a population sample of northeastern Ontario men aged 50-84 years

Variable	Controls	Cases	AOR	95% CI	<i>p</i> -value
Strenuous activity in mid-teens					0.9455
No	461	217	1.0		
Yes	1,111	523	1.0	0.8-1.2	
Unknown	41	12			
Strenuous activity in early 30s					0.1022
No	695	348	1.0		
Yes	875	393	0.9	0.7-1.0	
Unknown	43	11			
Strenuous activity in early 50s					0.0045
No	862	447	1.0		
Yes	709	294	0.8	0.6-0.9	
Unknown	42	11			

\* Adjusted for age, family history of prostate cancer, BMI 5 years ago, education, type of occupation.

**TABLE 4**  
Frequencies, adjusted odds ratio estimates (AOR\*), approximate 95% confidence intervals (CI) and global *p*-values for smoking variables for cases and controls from a population sample of northeastern Ontario men aged 50-84 years

Variable	Controls	Cases	AOR	95% CI	<i>p</i> -value
Cigarette smoking					0.3192
Never	373	158	1.0		
Former smoker	952	454	1.1	0.9-1.4	
Current smoker	270	133	1.2	0.9-1.7	
Unknown	18	7			
Pack-years for cigarettes					0.5265
0	373	158	1.0		
> 0 and ≤ 20	385	189	1.2	0.9-1.5	
> 20 and ≤ 43	391	191	1.2	0.9-1.5	
> 43	387	174	1.1	0.8-1.4	
Unknown	77	40			

\* Adjusted for age, family history of prostate cancer, BMI 5 years ago, education, type of occupation.

While a possible negative association between tea and prostate cancer risk has been identified previously,<sup>10</sup> no such result was observed here. A potential weak positive association between high levels of alcohol consumption and prostate cancer is noted in a review of the literature,<sup>11</sup> but no association was observed in the current study.

With respect to physical activity, this study identified significantly reduced risk

associated with strenuous recreational physical activity later in life (i.e., early 50s) and an effect of borderline significance for strenuous physical activity for those in their early 30s. Moderate physical activity was not associated with prostate cancer risk in this study (data not shown) and there is therefore no dose-response relationship identified between level of physical activity and risk of prostate cancer. A general negative association between physical activity and prostate cancer risk has been

identified in previous studies,<sup>13,14</sup> where one review of the literature identified a median relative risk of 0.8.<sup>13</sup> This median coincides with the odds ratio estimate observed here. The specific identification of strenuous physical activity as negatively associated with prostate cancer risk also was found in a prospective study of middle-aged men as well as in a recent case-control study.<sup>32,33</sup> It is suggested that prostate cancer is a hormone-related disease<sup>14</sup> and, as such, changes in hormone levels, specifically reduction of testosterone levels as a result of strenuous physical activity, may lead to reduced risk of prostate cancer.

The identification of an inverse association between recent physical activity and renal cell carcinoma has been found where past physical activity was not associated.<sup>34</sup> It is suggested that more accurate recall of recent activity could explain why only recent physical activity is a significant risk factor. This also may be the case for this study of prostate cancer where earlier strenuous physical activity may be important but is not identified as significant, possibly because of recall issues.

While cigarette smoking is an established risk factor for many cancers, cigarette smoking in this study was not associated with prostate cancer risk. Similarly, a review of previous studies of incident prostate cancer generally found no association.<sup>15</sup> A suggestion that the effect of smoking (pack-years) on prostate cancer may exist for obese men<sup>35</sup> was investigated in this study by exploring an interaction between an indicator of obesity (highest quartile of BMI versus remaining levels of BMI) and quartiles of pack-years of smoking with a null finding (*p*-value = 0.12; data not shown).

There are several statistical comparisons made in this study and the confidence intervals and *p*-values presented are therefore anti-conservative. Any significant results must be taken as hypothesis confirming or hypothesis generating. The specific results for consumption of tomato-based foods may be spurious due to the issue of multiple testing and this may explain the direction of the observed association. As for strenuous physical activity, the fact that such an association

**TABLE 5**  
**Adjusted odds ratio estimates (AOR\*), approximate 95% confidence intervals (CI) and global *p*-values for diet and recreational physical activity variables in final model along with core potential confounders for cases and controls from a population sample of northeastern Ontario men aged 50-84 years**

Variable	AOR	95% CI	<i>p</i> -value
Family history of prostate cancer			< 0.0001
No	1.0		
Yes	2.9	2.2-4.0	
Body mass index (kg/m <sup>2</sup> ) 5 years ago			0.2397
≤ 24	1.0		
24-27	0.9	0.7-1.2	
27-29	1.2	0.9-1.6	
> 29	1.0	0.8-1.3	
Unknown			
Education			0.0673
Elementary	1.0		
Secondary	1.0	0.8-1.2	
Post-secondary	0.7	0.6-1.0	
Unknown			
Type of occupation			0.0299
White collar	1.0		
Blue collar	1.2	1.0-1.5	
Tomato or vegetable juices			0.0043
0	1.0		
0.1 - 0.5	1.0	0.8-1.3	
0.6 - 1.0	1.1	0.9-1.5	
> 1.0	1.5	1.2-2.0	
Ketchup			0.0071
0	1.0		
0.1 - 0.5	0.9	0.7-1.1	
0.6 - 2.9	0.8	0.6-1.0	
≥ 3.0	1.2	1.0-1.5	
Strenuous activity in early 50s			0.0047
No	1.0		
Yes	0.8	0.6-0.9	

\* Adjusted for age and other variables in table

has been observed in other studies adds credence to this result.

It is recognized that the current study may suffer from potential recall bias, as is true for all case-control studies that involve self-reported exposures. It is not likely that there is any systematic difference between cases and controls with respect to recall since the study was described as a men's

health study and telephone interviews and data coding were performed blind to the disease status of the subject. The low response rate among controls, again a common issue with case-control studies, may contribute to study bias, depending on the representativeness of participating controls. A brief refusal questionnaire of smoking status was conducted as part of this study and it was found that,

while participating cases were similar to non-participating cases with respect to proportion of current smokers, significantly fewer participating controls were current smokers compared to non-participating controls. Thus, any potential bias in our results for the smoking analysis will be bias away from the null.

Information on stage and grade of prostate tumours of cases was not known and information on PSA testing was also not available. If screen-detected prostate cancers were common in our data, this could bias our effect estimates toward the null. However, it must be noted that, in the population from which cases and controls were sampled (northeastern Ontario and Ontario in general), PSA testing in asymptomatic men is not insured, which reduces this potential source of bias.

The results presented here were derived from a large population-based study that had adequate power to investigate the exposures considered. These findings suggest that, while smoking is not associated with prostate cancer risk, there is evidence to suggest a negative association between prostate cancer risk and strenuous physical activity. Further research is necessary to investigate the role of tomato product intake.

## Acknowledgements

This investigation was supported by research grants from the National Health Research and Development Program (Project No. 6606-5574-502), The Northern Cancer Research Foundation, The Natural Sciences and Engineering Research Council and The Canadian Union of the Mine, Mill & Smelter Workers, Local 598. The Ontario Cancer Registry confidentially supplied pathology reports to identify cases in this study. Analysis assistance of Ms. Tara Gomes is greatly appreciated. Also, Mr. Zahid Naseer is thanked for assistance with data quality evaluation and data analysis. The authors are grateful for the cooperation of urologists in northeastern Ontario.

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# Smoking-attributable mortality and expected years of life lost in Canada 2002: Conclusions for prevention and policy

Dolly Baliunas, Jayadeep Patra, Jürgen Rehm, Svetlana Popova, Murray Kaiserman and Benjamin Taylor

## Abstract

Cigarette smoking is one of the most important risk factors for burden of disease. Our objective was to estimate the smoking-attributable deaths and the years of life lost for Canada 2002. For Canada in 2002, 37,209 of all deaths aged 0 to 80+ years were attributable to smoking, 23,766 in men and 13,443 in women. This constituted 16.6% of all deaths in Canada, 21% for men and 12.2% for women. Main causes of smoking-attributable death were malignant neoplasms (17,427), cardiovascular diseases (CVD) (10,275) and respiratory diseases (8,282). Lung cancer (13,401) and chronic obstructive pulmonary disease (COPD) (7,533) were the single largest disease contributors to deaths caused by smoking. 515,608 years of life were lost prematurely in Canada in that year, 316,417 years in men and 199,191 years in women. Cigarette smoking is a major contributor to mortality in Canada and its impact on Canadian society continues to be an unacceptable burden.

**Key words:** Canada, expected years of life lost (EYLL), mortality, relative risks (RR), smoking-attributable fractions (SAF)

## Introduction

Tobacco use is responsible for high levels of mortality and morbidity. Smoking causes substantially increased risk of mortality from lung cancer, upper aerodigestive (i.e., head, neck and oesophageal) and other cancers, heart disease, stroke, chronic respiratory disease and a number of other medical conditions.<sup>1</sup> In the developed world in the year 2000, smoking was reported to be the risk factor with the largest attributable mortality and attributable disability-adjusted life years (DALYS) by the World Health Organization,<sup>2</sup> overall, 12.2% of all DALYS were attributed to this risk factor.

The most recently published Canadian estimate of smoking-attributable mortality (SAM) was produced using 1998 mortality data, 1998/1999 smoking prevalence data<sup>3</sup> and relative risks (RR) from the American Cancer Society's Cancer Prevention Study II (CPS-II). The total SAM was estimated to be 47,581 (21% of all deaths, age  $\geq 35$ ). However, the CPS-II has been criticized for not being generalizable to the entire US population. When compared to the general population, participants in CPS-II tend to overrepresent the middle class and have more education. As well, a disproportionate number of them are white.<sup>4,5</sup> Thus, direct application of a large

US survey to the Canadian population may not be appropriate.

In addition, more recent information may affect current SAM estimates. The prevalence proportion of current smoking has been steadily decreasing in Canada since the mid-1980s<sup>6</sup> and, in 2004, was at 20% for those aged 15 and older.<sup>7</sup> Self-reported consumption has also declined, with the average daily smoker consuming slightly less than 16 cigarettes per day.<sup>7</sup> In 2004, the Surgeon General (SG) added several diseases to the list of those for which evidence is sufficient to conclude a causal relationship between smoking and disease: stomach cancer, renal cell carcinoma, uterine cervical cancer, pancreatic cancer and pneumonia. Some of these had been included in recent SAM estimates, such as those produced by Illing & Kaiserman.<sup>3</sup>

Given the changes in smoking behaviour among Canadians since 1998, the availability of new and possibly more relevant relative risks, as well as continued interest in the issue, there was a need for an updated estimate of smoking-attributable mortality. This paper, using a sex-, age- and disease-specific approach to estimate the number of deaths and years of life lost for Canada in 2002, tries to meet that need.

## Author References

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## Method

The aim of the present study was to estimate the proportion of deaths caused by smoking and the number of premature deaths in Canada for the year 2002. Several elements necessary for this estimate are described below: the method for identification of diseases, the measurement of smoking prevalence, the determination of risk relationships and attributable fractions, and the mortality data source.

### Identification of diseases and meta-analyses

To identify the malignant and non-malignant health conditions for inclusion in the SAM estimate, this analysis was guided by the *Health Consequences of Smoking: A Report of the Surgeon General*,<sup>1</sup> which considers the following criteria in judgments of causality: consistency, strength of association, specificity, temporality, coherence, dose-response and experimental evidence. The 2004 SG report implemented a standardized, hierarchical language to summarize conclusions about causality, the strongest of which is “evidence is sufficient to infer causality”. Our analyses include only health outcomes for which this conclusion was reached.

Once identified, conditions were translated into corresponding codes from the tenth revision of the *International Classification of Diseases (ICD-10)*. Finally, a comprehensive search strategy of current meta-analyses was performed for each disease category and its risk relationship with smoking.

Meta-analyses were identified using the PubMed and OVID (1966–January Week 3, 2005) databases. Search criteria were as follows: smoking or tobacco, meta-analysis, and each malignant and non-malignant disease category described in this paper.

Meta-analyses that included measures of smoking dose were preferred over those that only used the “current”, “former” or “never” categories. However, if relative risks (RR) for dose-responses were not found

from the studies, we used the “current/former/never” or “ever/never” category where available. Similarly, analyses that included age- and sex-stratified estimates of relative risk were preferred over more crude estimates.

In cases where a more current meta-analysis did not exist, the analysis from English et al. was used.<sup>8</sup> When a meta-analysis was published later than 1995, there was usually only one that presented data on smoking dose, so it was used as the source of relative risk. If there was more than one, all were examined and the most comprehensive one was chosen, based on smoking dose and age categories.

### Prevalence of smoking in Canada

Smoking prevalence for different levels of smoking consumption was obtained from the Canadian Community Health Survey (Cycle 2.1).<sup>9</sup> Categories included current, former and never smokers by sex and age group. Current smokers were those who reported occasional smoking or daily smoking (cigarettes per day dose-response: e.g., 1-14, 15-24, 25+ cigs/day; < 20 or ≥ 20 cigs/day, etc.). For each disease for which the identified meta-analysis included dose-response-specific RRs, prevalence estimates were calculated using the same categorizations of smoking consumption. The prevalence of non-smokers whose home is also inhabited by a person who smokes was available as well from the CCHS data set and it was used to calculate 2002 passive smoking deaths.

To ensure comparability between the CCHS sample and the Canadian population, the sample was weighted prior to calculating prevalence, based on sex and age groups. The age groups used were 15-29, 30-44, 45-59, 60-69, 70-79 and 80+.

### Mortality data

Mortality data in Canada for the year 2002 were obtained from Statistics Canada coding according to ICD-10.<sup>10</sup> Table 1 shows the disease conditions that were used, by diagnosis, and the source of measure of association or smoking-attributable fraction (SAF).

### Computing smoking-attributable fractions (SAF) of mortality

“Smoking-attributable fraction” (SAF) is defined as the fraction of the disease in the population that would not have occurred if the effects associated with smoking were absent.<sup>24,25</sup>

SAFs were assessed for specific causes of natural and unnatural deaths by two methods:

- Chronic disease smoking-attributable fractions were calculated by combining smoking prevalence from CCHS and relative risk estimates from meta-analyses.
- Fire injury was calculated using direct estimates of smoking involvement from the Council of Canadian Fire Marshals and Fire Commissioners.<sup>23</sup>

We used the most comprehensive meta-analysis for each condition, as indicated in Table 1. The age- and sex-specific relative risk (where available) for each condition was combined with different levels of smoking for each sex and age group and an attributable fraction was obtained using the following formula.<sup>24,25</sup>

$$SAF = [\sum_{i=1}^k P_i(RR_i - 1)] / [\sum_{i=0}^k P_i(RR_i - 1) + 1]$$

$i$ : smoking category with baseline category or never smoking  $i = 0$ .

RR ( $i$ ): relative risk at smoking level  $i$  compared to never smoking

P ( $i$ ): prevalence of the  $i$ th category of smoking

The SAFs were then applied to the mortality data to estimate the smoking-attributable mortality (SAM) by age and sex.

Passive-smoking-attributable mortality (PSAM), was derived by applying age- and sex-specific relative risk and rates of mortality from lung cancer and ischemic heart disease (IHD) to the population of Canadians who have never smoked but who are exposed to environmental tobacco smoke (ETS) from spouses and other sources. Relative risks estimates were obtained from the most comprehensive meta-analyses applicable to Canada. Our estimates for ETS mortality



**TABLE 1**  
**Smoking-related disease categories and sources of measure of association**

Condition	ICD-10	Source from meta-analysis or SAF
Mental and behavioural disorders due to use of tobacco	F17	100% SAF per definition
<b>Malignant neoplasms</b>		
Oropharyngeal cancer	C00-C14, D00.0	English et al., 1995
Oesophageal cancer	C15, D001	English et al., 1995
Stomach cancer	C16, D002	Tredaniel et al., 1997
Pancreatic cancer	C25, D01.90	English et al., 1995
Laryngeal cancer	C32, D02.0	English et al., 1995
Trachea, bronchus and lung cancers	C32, D02.0	Simonato et al., 2001
Cervical cancer	C53, D06	Plummer et al., 2003
Uninary tract cancer	C64-C68	Zeegers et al., 2000
Renal cell carcinoma	C64	Hunt, 2005
Bladder cancer	C67, D09.0	Brennan et al., 2000; 2001
Acute myeloid leukaemia	C92.0	Brownson et al., 1993
<b>Cardiovascular diseases</b>		
Ischaemic heart disease	I20-I25	Law, 1997 and Law, 2003
Pulmonary circulatory disease	I26-I28	English et al., 1995
Cardiac arrhythmias	I47-I49	Follow IHD
Heart failure; complications and ill-defined descriptions of heart disease	I50-I51	Follow IHD
Cerebrovascular diseases	I60-I69	English et al., 1995
Atherosclerosis	I70-I79	English et al., 1995
<b>Respiratory or intestinal diseases</b>		
Pneumonia and Influenza	J10-J18	English et al., 1995
Chronic obstructive plumonary disease	J40-J44	Single et al., 1996 (cost study)
Ulcers	K25-K28	English et al., 1995
<b>Conditions arising during the perinatal period (maternal use)</b>		
Low birth weight and short gestation	P05-P07	English et al., 1995
Sudden infant death syndrome	R95	English et al., 1995
<b>Unintentional injuries</b>		
Fires	X00-X09	Council of Canadian Fire Marshals and Fire Commissioners. Annual Report 2000, 2003

ICD = *International Classification of Diseases, Version 10*

SAF = smoking-attributable fraction

used an RR estimate of 1.21<sup>26</sup> for lung cancer and RR estimate of 1.24<sup>27,28</sup> for IHD, which are consistent with other results. Zhong et al.<sup>29</sup> cited an RR estimate of 1.2 for lung cancer from a meta-analysis of 35 case-control and five cohort studies. Taylor (2001) estimated an RR estimate of 1.21 for Western industrialized countries. The incidence density ratio (relative risk) associated with exposure to environmental

tobacco smoke for IHD was estimated from two recent meta-analyses. Thun et al.<sup>30</sup> noted relative risks of 1.24 for males and 1.23 for females exposed to passive smoking, while He et al.<sup>31</sup> estimated a relative risk of 1.25 for both sexes.

For reasons of comparability and conservatism, we used an RR estimate of 1.21 for lung cancer and an RR estimate

of 1.24 for IHD. This method had been employed previously, for the year 1998.<sup>3</sup>

Total 2002 SAM was calculated by summing all chronic disease SAM, pediatric disease SAM and total PSAM for each sex and age group.

Two sensitivity analyses were conducted. For each disease, the lower limit of the confidence interval around relative risk and the lower limit around the confidence interval of the accompanying smoking prevalence estimates were used simultaneously to derive the SAF. As before, the SAF was multiplied by number of deaths to produce SAM estimates. This procedure was repeated with the upper limits of the confidence intervals.

### ***Smoking-attributable expected years of life lost (EYLL)***

Expected years of life lost (EYLL) is a measure of the impact of premature mortality on a population. Persons dying due to smoking consumption would have lived longer if they had not smoked. The average extra time such individuals would have lived is known as the residual life expectancy. For example, if a male dies of chronic obstructive pulmonary disease (COPD) at age 50, in Canada he would have a residual life expectancy of 28.4 years.<sup>32</sup> The sum of these extra years for all people dying from smoking in a population is known as EYLL due to smoking. EYLL for each age group category can be estimated from the observed mean age at death in the age interval and the life expectancies tables at the exact ages defining the age interval through interpolation. The life expectancies table for Canada mortality in 2000 is available from the WHO Web site ([www.who.int/evidence](http://www.who.int/evidence)). In calculating the mean ages within the intervals, the rules specified by the Global Burden of Diseases (GBD) study were followed.<sup>33</sup> EYLL due to smoking in Canada has been calculated for each age group (0-14, 15-29, 30-44, 45-59, 60-69, 70-79 and 80+) by multiplying the number of smoking-attributable deaths by the interpolated life expectancy for the observed mean age at death in the interval. Mean ages for 80+ age group for men (84 years) and women (85 years) were calculated from the life

table. EYLL was calculated per 100,000 population.

## Results

Table 2 gives an overview of the estimated degree of smoking prevalence in Canada by sex and age group. As expected, more men than women were current smokers and the prevalence of current consumption decreased as age increased.

Table 3 provides the estimates of SAM by disease and PSAM for lung cancer and IHD. Overall results in Canada show that 37,209 smoking-attributable deaths were estimated, accounting for 23,766 deaths among men and 13,443 among women for the year 2002, including 58 boys and 34 girls under the age of one who died as a result of smoking-related causes. The 37,209 smoking-attributable deaths constituted 16.6% of all Canadian deaths (there were 223,603 deaths in Canada in 2002).

Most of the deaths attributable to smoking may be grouped into three broad categories. The three largest contributors were cancers (malignant neoplasms), cardiovascular diseases (CVD) and respiratory diseases (see Table 3). Cancer accounted for 46.8% of smoking-attributable deaths (17,427 deaths: m: 11,891; f: 5,566), CVD accounted for 27.6% (10,275 deaths: m: 6,373; f: 3,902), and respiratory disease accounted for 22.3% (8,282 deaths: m: 4,788; f: 3,494). Total deaths due to ETS (lung cancer and IHD) accounted for 2.2% (831 deaths: m: 507; f: 324). With respect to single disease

categories within these broad categories, lung cancer (13,401 deaths: m: 9,028; f: 4,373), IHD (5,343 deaths: m: 3,837; f: 1,506) and COPD (7,533 deaths: m: 4,378; f: 3,155) constituted the largest smoking-attributable categories. Together, these three diseases account for more than two thirds (70.6%) of all smoking-attributable deaths in Canada in 2002. Almost two thirds (63.9%) of those who died from smoking-related causes in Canada were men.

In addition, 2.2% of all smoking-attributable deaths (831 deaths: m: 507; f: 324) aged 15 years and over were a result of ETS exposure in 2002. Specifically, 252 Canadians (m: 157; f: 95) died from lung cancer PSAM, while 579 Canadians (m: 350; f: 228) died from IHD PSAM.

For some of the individual causes of death, smoking was responsible for more than 75% of deaths: lung cancer (78.0%), pulmonary circulatory disease (79.1%) and COPD (79.7%). In terms of absolute numbers, more males than females died of smoking-attributable causes. This probably is a reflection of the higher rates of current smoking among males.

Out of 198 fire deaths, smoking caused 28% of mortality (55 deaths).

Canadian residents lost an estimated 515,608 years of EYLL as a result of the premature mortality resulting from cigarette smoking (316,417 years of life lost among men and 199,191 years lost among women). The EYLL rate for deaths due to

smoking was 2,151 per 100,000 for men and 1,302 per 100,000 for women aged 0 to 80+ (Table 4). That is, for every 100,000 population, there was an expected loss of 2,151 years of life among men and 1,302 years of life among women as a result of premature death due to smoking. A high EYLL rate for men was observed, indicating higher levels of premature mortality among men compared to women. Cancer was the leading cause of smoking-attributable EYLL in Canada in 2002, responsible for 262,268 years of expected life lost (162,612 male and 99,656 female). CVD caused a loss of 151,604 years (97,824 male and 53,780 female). Respiratory disease caused 79,330 years to be lost (42,007 male and 37,323 female).

Overall, smoking affected more men than women: In men, 21% of the deaths were smoking attributable, compared to 12.2% of the deaths among women.

The overall average age for smoking-attributable death was 71.2 years for men and 73.4 years for women. There were no such notable sex differences found between disease categories, except fire injury. For this category, the average age for a smoking-attributable death was 46.7 years for men and 58.0 years for women.

The sensitivity analyses produced a low estimate of 31,210 smoking-attributable deaths (20,594 among men and 10,617 among women). The upper estimate was 44,775 smoking-attributable deaths (27,747 among men and 17,028 among women).

**TABLE 2**  
**Estimated smoking prevalence proportions in Canada by sex and age group (years)**

Smoking categories		15-29	30-44	45-59	60-69	70-79	80+	Overall (all ages)
Current	Female	0.263	0.247	0.230	0.151	0.103	0.059	0.218
	Male	0.310	0.307	0.260	0.172	0.098	0.075	0.263
Former	Female	0.262	0.383	0.435	0.452	0.427	0.389	0.378
	Male	0.258	0.389	0.530	0.642	0.704	0.735	0.446
Never	Female	0.475	0.369	0.334	0.398	0.471	0.552	0.404
	Male	0.431	0.304	0.211	0.187	0.197	0.190	0.291
<b>Total per sex</b>		<b>1.000</b>	<b>1.000</b>	<b>1.000</b>	<b>1.000</b>	<b>1.000</b>	<b>1.000</b>	<b>1.000</b>

Source: Canadian Community Health Survey, Cycle 2.1 (2003)

**TABLE 3**  
**Smoking-attributable fractions (SAF), mean age at death and number of deaths due to**  
**smoking by sex, age and disease category in Canada, 2002 (continued)**

Disease condition*	SAF in % (all ages)		Mean age at death		Total		Overall
	M	F	M	F	M	F	
ACTIVE SMOKERS							
Malignant neoplasms							
Oropharyngeal cancer	57.0	47.2	64.7	68.4	430	156	586
Oesophageal cancer	48.4	38.0	67.6	72.9	523	149	672
Stomach cancer	16.1	11.7	70.0	70.6	184	90	273
Pancreatic cancer	17.1	12.7	68.6	70.2	266	209	475
Laryngeal cancer	67.0	59.1	69.9	68.6	271	52	323
Lung cancer	88.6	62.5	70.2	68.1	9,028	4,373	13,401
Cervical cancer	--	34.7	--	58.3	--	126	126
Urinary tract cancer	55.1	36.9	71.1	73.2	1,089	364	1,452
Renal cell carcinoma	26.5	6.8	67.9	69.0	221	35	256
Bladder cancer	67.9	51.3	75.9	77.0	740	223	964
Acute myeloid leukaemia	15.7	13.1	69.9	66.9	70	48	118
Total malignant neoplasms	60.9	43.2	69.9	68.5	11,861	5,566	17,427
Tobacco abuse							
Total tobacco abuse	100.0	100.0	64.2	73.4	37	20	57
Cardiovascular diseases							
Ischaemic heart disease							
Age < 45 yrs	51.9	45.1	36.5	36.4	208	47	254
45-59 yrs	42.2	37.3	52.0	52.0	1,128	232	1,360
60-69 yrs	29.1	23.4	64.5	64.5	1,055	290	1,345
70-79 yrs	10.0	7.3	74.5	74.5	664	300	965
80+ yrs	8.9	5.1	87.0	87.0	782	637	1,419
Pulmonary circulatory disease	83.3	76.5	70.2	72.9	305	446	751
Cardiac arrhythmias							
Age < 45 yrs	50.9	43.8	33.9	32.7	20	6	26
45-59 yrs	42.2	37.3	52.0	52.0	38	12	50
60-69 yrs	29.1	10.5	64.5	64.5	29	10	39
70-79 yrs	10.0	7.3	74.5	74.5	26	13	39
80+ yrs	8.9	5.1	87.0	87.0	40	47	88
Heart failure							
Age < 45 yrs	50.8	44.2	33.7	33.8	19	8	26
45-59 yrs	42.2	37.3	52.0	52.0	38	19	57
60-69 yrs	29.1	23.4	64.5	64.5	60	26	86
70-79 yrs	10.0	7.3	74.5	74.5	54	36	89
80+ yrs	8.9	5.1	87.0	87.0	115	126	241
Cerebrovascular diseases							
Age < 65 yrs	39.2	35.2	53.1	52.0	292	207	499
≥ 65 yrs	14.3	9.6	80.7	82.6	803	814	1,617

\* For condition definition from *International Classification of Diseases, Version 10*, see Table 1.

Note: These results were derived by multiplying SAFs with number of deaths for each category, thereby producing decimal numbers. As a result, there may be rounding errors due to collapsing numbers over different categories.

**TABLE 3 (continued)**  
**Smoking-attributable fractions (SAF), mean age at death and number of deaths**  
**due to smoking by sex, age and disease category in Canada, 2002**

Disease condition*	SAF in % (all ages)		Mean age at death		Total		Overall
	M	F	M	F	M	F	
Atherosclerosis	31.6	31.3	75.4	79.7	697	628	1,325
Total cardiovascular diseases	18.7	11.3	68.5	75.3	6,373	3,902	10,275
<b>Respiratory diseases</b>							
Pneumonia/Influenza	19.9	12.9	79.8	82.1	410	340	750
Chronic obstructive pulmonary disease	83.1	75.4	79.1	79.0	4,378	3,155	7,533
Total respiratory diseases	65.4	51.2	79.1	79.3	4,788	3,494	8,282
<b>Intestinal diseases</b>							
Total ulcers	48.8	36.7	74.6	79.9	107	83	190
<b>Conditions arising during perinatal period (maternal use)</b>							
Low birthweight and short gestation	24.7	20.6	0.0	0.0	37	22	59
Sudden infant death syndrome	31.2	26.5	0.0	0.0	21	12	33
Total paediatric diseases < 1 year of age	26.7	22.4	0.0	0.0	58	33	92
<b>Injury</b>							
Total fire injury	28.0	28.0	46.7	58.0	35	20	55
<b>TOTAL ACTIVE SMOKERS</b>	37.8	24.0	71.2	73.3	23,259	13,119	36,378
<b>PASSIVE SMOKERS</b>							
Lung cancer	1.5	1.4	68.0	67.1	157	95	252
Ischaemic heart disease	1.6	1.2	71.2	79.3	350	228	579
<b>TOTAL PASSIVE SMOKERS</b>	1.6	1.3	70.2	75.7	507	324	831
<b>All smoking-attributable deaths</b>			71.2		23,766	13,443	37,209

\* For condition definition from *International Classification of Diseases, Version 10*, see Table 1.

Note: These results were derived by multiplying SAFs with number of deaths for each category, thereby producing decimal numbers. As a result, there may be rounding errors due to collapsing numbers over different categories.

## Discussion

Tobacco smoking is a major public health concern. It is responsible for significant mortality and years of life lost in Canada. Of the 223,603 deaths in Canada in 2002, almost forty thousand (16.6%) could be attributed to smoking. Among all smoking-attributable deaths, 46.8% were due to cancer, followed by CVD at 27.6% and respiratory diseases at 22.3%.

This study found little change in smoking-attributable mortality from a previous analysis conducted using 1992 data.<sup>21</sup> In the 1992 study, smoking was estimated to account for 17% of all deaths in 1992, compared to our estimate of 16.6%. There is a wide gap between the two time periods, so a more appropriate comparison may be considered. The present study found lower smoking-attributable mortality than was reported in a recent study using 1998 data.<sup>3</sup>

Although the top three causes of death due to smoking were the same between the two studies (lung cancer, ischemic heart disease and chronic obstructive pulmonary disease), their order was different. Among smoking-attributable deaths, this study reported lung cancer (36%) as the top killer, followed by COPD (20%) and IHD (14%). In the Illing article, lung cancer was number one, but the opposite order was reported for IHD (20%) and COPD (14%). It is unclear what the reason is for this difference, but a possible explanation could be the much greater numbers of IHD mortality in 1998 compared to 2002, whereas the numbers for COPD remained fairly similar between these years. This is consistent with an overall trend of increasing COPD and decreasing IHD deaths (IHD only for those under 65) reported for 1989-1998. The decrease in IHD deaths may be due to either a decrease in IHD incidence, improvement in survival, or both. Also

of note is that higher percentages of smoking-attributable mortality due to lung cancer were reported in this study, despite the fact that lung cancer mortality rates in Canada have been slowly decreasing for men and are constant or slightly increasing for women.<sup>34</sup>

From 1989 to 1998, smoking-attributable mortality was shown to have increased in women, but remained fairly constant in men, resulting in the ratio of male-to-female smoking-attributable mortality decreasing from 2.6 to 1.8 over the same time period.<sup>3</sup> If we make a similar comparison between the 1998 ratio and this current analysis, we note that the ratio of male-to-female smoking-attributable mortality, at 1.77, is similar to that obtained for 1998.

There is one important methodological difference between this paper and the study by Illing and Kaiserman<sup>3</sup>: Our study

**TABLE 4**  
**Expected years of life lost (EYLL) attributable to smoking in Canada 2002 by age, sex and disease category**

Sex	Age	Cancer	Deaths			Cancer	EYLL		
			CVD*	RD**	Total		CVD	RD	Total
Male	0-14	n/a	n/a	n/a	58	n/a	n/a	n/a	3,978
	15-29	5	25	2	40	272	1,383	109	2,151
	30-44	173	304	19	522	6,920	12,157	760	20,888
	45-59	1,975	1,462	149	3,708	51,646	38,226	3,896	96,954
	60-69	3,215	1,475	526	5,371	51,279	23,528	8,390	85,672
	70-79	4,144	1,344	1,567	7,260	39,575	12,832	14,965	69,335
	80+	2,349	1,763	2,525	6,807	12,920	9,698	13,888	37,438
						<b>162,612</b>	<b>97,824</b>	<b>42,007</b>	<b>316,417</b>
Rate per 100,000 persons = 2,151 years (men, all ages, all causes EYLL)									
Female	0-14	n/a	n/a	n/a	33	n/a	n/a	n/a	2,459
	15-29	9	16	2	29	536	951	119	1,750
	30-44	156	121	9	293	7,001	5,411	404	13,163
	45-59	1,171	450	115	1,782	35,903	13,798	3,526	54,633
	60-69	1,404	545	400	2,412	27,729	10,758	7,900	47,643
	70-79	1,772	850	1,064	3,807	21,636	10,376	12,991	46,486
	80+	1,054	1,921	1,905	5,085	6,851	12,487	12,383	33,056
						<b>99,656</b>	<b>53,780</b>	<b>37,323</b>	<b>199,191</b>
Rate per 100,000 persons = 1,302 years (females, all ages, all-causes EYLL)									

\* Cardiovascular disease

\*\*Respiratory disease

uses pooled relative risk estimates from existing meta-analyses, not relative risks from only one study. This potentially makes our estimates more generalizable to the Canadian population and more reliable through the use of multiple studies. The relative risks used in this current study tended to be lower, decreasing the smoking-attributable mortality estimates. It must be acknowledged that SAM estimates will vary based on the underlying assumptions implicit in population-attributable fraction methods, an issue that has engendered debate in Canadian estimates.<sup>35</sup> Our own sensitivity analysis resulted in a low estimate of 31,210 and a high estimate of 44,775.

Despite differences in risk estimation, tobacco smoking is responsible for a substantial number of Canadian deaths. The results of this paper indicate that the trend in smoking-attributable mortality may be stable or even declining. The

change in trend may be the result of nearly forty years of tobacco control activities. Since 1965, when the first smoking rates were measured, fewer people have been smoking fewer cigarettes. Indeed, between 1985 and 2002, daily smokers reported smoking four cigarettes fewer per day. In addition, when compared to 1985 data, more smokers in 2002 were distributed in the “light” and “moderate” smoking categories.<sup>36</sup>

Policies and interventions aimed at cessation strategies will be helpful in reducing the short-term mortality burden in Canada. A number of cohort studies and clinical trials have shown that smoking cessation has been shown to reduce all-cause mortality<sup>37,38</sup> and prevent onset or development of cardiovascular<sup>39,40</sup> and respiratory diseases<sup>41,42</sup> in particular. Policies affecting adolescents, such as price, availability of cigarettes, smoking bans and the marketing of cigarettes are important to decreasing

future smoking mortality. Immediate reductions to smoking-attributable fire deaths may be realized by the federal Cigarette Ignition Propensity Regulation that came into effect on October 1, 2005. This regulation requires that all Canadian-manufactured or imported cigarettes must burn their full length no more than 25% of the time when tested using a standard protocol.

While smoking rates and cigarette consumption in Canada decline, smoking-attributable mortality has not yet kept pace due to the latent period between smoking and chronic disease outcomes—it thus remains unacceptably high. There is optimism for the future, however. In Canada, in 2004, there were more former than current smokers and the number of former smokers continues to increase. In fact, between 1999 and 2004, the number of current smokers in Canada declined by nearly one million and those who



continue to smoke are smoking much less than in the past. The benefit of this reduction in smoking prevalence will take some time to be reflected in estimates of smoking-attributable mortality. However, all of this activity, if continued (including government policies and cessation and prevention programs), will result in decreases in smoking-attributable deaths in the near future.

## Acknowledgements

This contribution was, in part, enabled by funding from various sources allocated to the Second Canadian Study on Social Costs of Substance Abuse, under the umbrella of the Canadian Centre on Substance Abuse.

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# Rugby injury in Kingston, Canada: A ten-year study

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## Abstract

Rugby is a high-contact sport with an elevated risk for injury. While many studies have examined the epidemiology of rugby injury, there are no surveillance-based injury studies from North America. The objective of this study was to profile the scope and nature of injuries experienced during the sport of rugby. We analyzed emergency department injury surveillance data over a decade (1993–2003) from the Kingston sites of the Canadian Hospital Injury Reporting and Prevention Program. Rugby injuries were examined by mechanism, nature and anatomical site of injury, with stratification according to sex and age. A total of 1,527 injuries was observed (mean of 153 per year). Results show the tackling phase of play accounted for the highest number of injuries (506/1,527; 33.1%). The most common natures of injury were sprains and strains (426/1,527; 27.9%), while the leading anatomical location of injury was the face (294/1,527; 19.3%). Target patterns of injury were identified as priorities for prevention, based on injury frequency and severity.

**Key words:** emergency department, injury, rugby, sport, surveillance

## Introduction

Rugby is a full-body contact sport that is popular internationally, second only to soccer in its number of participating nations.<sup>1</sup> The sport involves two teams of 15, who are divided for the most part into “forwards” (i.e., the bigger, stronger players) or “backs” (the faster, more agile ones). The aim of rugby is to get the ball across the opposing team’s goal line using a series of plays and maneuvers, many of which involve player-to-player contact. Due to this high level of contact, there has been reported a notable incidence of injury associated with the sport.<sup>2–4</sup>

A number of international studies have examined the incidence of rugby injury, providing insights into the associated health burden. The most common element of rugby play resulting in injury is the tackle phase,<sup>3,5–7</sup> including both receiving a tackle and attempting one. The majority of reported injuries are

sprains or strains.<sup>2–5</sup> Those observed to be at greatest risk for injury are senior male forwards,<sup>3,5,9</sup> and as player age increases, so does the incidence of injury.<sup>3,4,6,10</sup> The majority of rugby injuries occur during the second half of play, when players are more fatigued.<sup>5</sup> Protective gear (e.g., scrum caps, support sleeves—neoprene sleeves that fit over parts of limbs) is effective in preventing minor injuries, but has not been shown to provide significant protection from other forms of injury, such as concussion.<sup>11,12</sup>

Most existing studies of rugby injury have focused on premier levels of play. Injuries associated with women’s rugby have rarely been considered, despite the fact that the number of women involved in the sport has risen.<sup>3</sup> Few studies have examined more general populations of players, including those in North America. Hence, there are significant gaps in this area of sports injury literature.

Our research setting in Kingston, Ontario is a site of the Canadian Hospitals Injury Reporting and Prevention Program (CHIRPP), an emergency-department-based injury surveillance program.<sup>13</sup> We used this opportunity to conduct one of the first North American studies of rugby injuries within a geographically defined population. The objective of this epidemiological study was to describe the injury patterns experienced by male and female sub-elite rugby players. The analysis specifically focused on mechanisms leading to injury, the natures of injuries experienced and common anatomical sites associated with injury. Observed patterns were examined by age and sex. Our hope was that this study would contribute to a better understanding of rugby injuries and their determinants, which in turn would inform prevention efforts.

## Methods

### Injury surveillance

CHIRPP is an ongoing national injury surveillance program that operates in the emergency departments of selected Canadian hospitals.<sup>13</sup> This program was implemented in ten Canadian pediatric hospitals in 1990 and has since been expanded to include four general hospitals. For each visit to the emergency department, the patient or accompanying person completes a one-page self-administered CHIRPP questionnaire which compiles information on mechanisms, circumstances and factors leading to injury. Clinical information is abstracted from the patient’s medical chart by a research nurse and coded to reflect information contained in the hospital discharge summary.

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## Regional injury surveillance in Kingston

Kingston is a Canadian city in eastern Ontario with a population of about 146,000 (2001 Census of Population).<sup>14</sup> Kingston General and Hotel Dieu hospitals have the only two emergency departments in Kingston. Since 1993, injury data from these hospitals have been collected and entered into the national CHIRPP database. The Kingston site is unique among the CHIRPP surveillance sites because of its complete community coverage.

### Rugby in Kingston and area

The Kingston area is home to one local rugby club, known as the Kingston Panthers. It is comprised of three men's teams, and one team each of "old boys" (age > 34), juniors and women. The club itself is a member of the Eastern Ontario Rugby Union, which is a division of the Ontario Rugby Union. In June 2006, there were a total of 120 players registered. Competitive rugby is also played at the high school and university levels. There are nine high schools and two universities in the Kingston district that offer a rugby program. Each high school generally has a team each for juniors, seniors and girls. Together, the universities field seven teams (five male, two female). The total number of area players registered with local clubs and schools is estimated at approximately 1,000, and the population at risk includes local participants and visiting players who might present to the Kingston hospitals for emergency medical care.

### Case identification and data abstraction

Records of injuries to male and female rugby participants were abstracted from the Kingston CHIRPP dataset (from September 1, 1993 to August 31, 2003). Cases were included if 1) sport code = "rugby" and/or 2) the text description of injury event included the word "rugby". All potential cases were reviewed and any cases that were obviously not related to participation in the sport were deleted. These included 1) miscoded cases; 2) spectators injured while watching rugby; or 3) variations

of rugby that were not true to the sport (i.e., rugby basketball). Cases that were younger than 14 years of age ( $N = 2$ ) were excluded from the analyses.

Available CHIRPP descriptors included the nature of injury, its anatomical site and discharge disposition. Text descriptions of circumstances surrounding each injury event were used to classify the mechanism of injury according to the element of play: 1) *tackling phase*; 2) *collision* (general category that included body-to-body contact on the field, excluding intended tackles and contact on the ground); 3) *contact on the ground* (general category that included body-to-body contact on the ground); 4) *falls*; 5) *running or maneuver*; 6) *hit with body part* (unintentional or undirected contact with another player's body lead to the injury); 7) *kicked* (injury inflicted when a player attempts to kick the ball, but another player is unintentionally kicked instead); 8) *foul play* (any *intentionally* inflicted injury to another player); 9) *hit by ball*; or 10) *other* mechanism. To ensure consistency of coding by mechanism, a random sample of 30 cases was examined and coded by three raters, all of whom were blinded to the coding of their fellow raters. There was perfect agreement in 28/30 cases and imperfect agreement in the other two (e.g., only 2/3 raters coded the case identically). The two cases were resolved by consensus. This level of agreement was considered sufficient to indicate a high level of reliability.

### Analysis

Frequencies of various classifications of player injury were reported. Cross-tabulations were used to describe the mechanisms, natures and anatomical sites of injury by age group and sex. The dataset also allows up to three natures and anatomical sites of injury to be specified for each patient encounter. Only the first nature and its corresponding anatomical site were used in the following analyses as they represent the diagnosis most responsible for the visit. Records were divided into four age groups (14-16, 17-19, 20-24,  $\geq 25$  years) defined to correspond to different levels of play in Kingston. Chi-square anal-

yses were used to investigate the statistical significance of any observed differences between groups. "Target injury events" that provided insights for prevention were identified, based upon two criteria: 1) the specific pattern of rugby injury occurred frequently, as indicated by the proportion of the case series that the pattern comprises ( $> 5\%$ ), and 2) the consequences of the specific pattern of injury were generally serious, as indicated by the proportion ( $> 10\%$ ) of the injuries associated with the pattern, requiring hospital admission or treatment with follow-up.

Ethics approvals for CHIRPP and this analysis were granted by the Queen's University Health Sciences Research Ethics Board.

## Results

### General distribution of rugby injuries

A total of 1,527 rugby injuries were observed from September 1, 1993 to August 31, 2003. The number of rugby injuries presenting to the emergency department ranged between 132 and 170 (mean 153) per year. However, we did not observe any pattern or significant temporal trend over the study period. The mean age of injured players was 20.0 years (SD 5.3). The overall sex ratio was 2.4:1 (male:female).

### Mechanisms of injury

Overall, the tackling phase accounted for one third of all rugby injuries in the case series (506/1,527; 33.1%). This phase includes both being tackled (370/1,527; 24.2%) and attempted tackling (136/1,527; 8.9%). Collisions (389/1,527; 25.5%) and contact on the ground (158/1,527; 10.3%) were the other leading mechanisms (Table 1). Leading mechanisms of rugby injury did not differ between males and females, however the proportion of collisions that were experienced by males was significantly higher (27.3% male vs. 21.0% female;  $p < 0.05$ ), while the proportion of injuries resulting from contact on the ground (14.9% vs. 8.5%) and being hit by the ball (4.1% vs. 1.4%) was significantly higher among females ( $p < 0.01$ ). Mechanisms of injury did not vary substantially by age group (data not shown).

**TABLE 1**  
Description of rugby injuries in Kingston, Canada, by age, sex and injury mechanism (1993–2003)

	Male		Female		Total	
	N	%	N	%	N	%
<b>Age groups</b>						
14-16 years	228	21.0	101	22.8	329	21.5
17-19 years	348	32.1	210	47.4	558	36.5
20-24 years	335	30.9	114	25.7	449	29.4
≥ 25	173	16.0	18	4.1	191	12.5
<b>Mechanism of injury</b>						
Tackling (all phases)	351	32.4	155	35.0	506	33.1
Being tackled	254	23.4	116	26.2	370	24.2
Attempted tackle	97	8.9	39	8.8	136	8.9
Collision	296	27.3	93	21.0*	389	25.5
Contact on ground	92	8.5	66	14.9**	158	10.3
Fall	77	7.1	28	6.3	105	6.9
Running/Maneuver	62	5.7	28	6.3	90	5.9
Hit with body part	65	6.0	19	4.3	84	5.5
Kicked	49	4.5	15	3.4	64	4.2
Foul play	31	2.9	4	0.9	35	2.3
Hit by ball	15	1.4	18	4.1**	33	2.2
Mechanism not specified	46	4.2	17	3.8	63	4.1
<b>Total</b>	<b>1,084</b>	<b>100.0</b>	<b>443</b>	<b>100.0</b>	<b>1,527</b>	<b>100.0</b>

\* *p*-value chi-square test < 0.05

\*\* *p*-value chi-square test < 0.01

### Natures of injury

Tables 2 and 3 present the distribution of the natures of injuries most often experienced by rugby participants, by age and sex. Sprain/strain was diagnosed in 27.9% of presentations; superficial injuries (22.6%), fractures (15.4%) and open wounds (14.6%) were also common. The proportions of sprain/strain (154/443; 34.8%) or superficial injuries (123/443; 27.8%) among women were significantly higher than among men ( $p < 0.01$ ). Among males, the proportion of open wounds (202/1,084; 18.6%,  $p < 0.001$ ) was significantly higher than that observed among females (21/443; 4.7%). When examined by age, a test for linear trend in proportions indicated that superficial injuries were significantly higher in younger participants than they were in their more senior counterparts with the opposite trend observed with open wounds ( $p \leq 0.01$ ). Neurotrauma, which includes the diagnoses of concussion ( $N = 71$ ), spinal cord

injury ( $N = 2$ ) and minor closed head injury ( $N = 76$ ) accounted for 9.8% of injuries overall. Higher proportions of neurotrauma, in particular concussion/spinal injury, were seen among younger players.

The CHIRPP coding for nature of injury contains a category for “multiple injuries of more than one nature”, but there were no such instances in our dataset. In our case series, there were 89 cases with a second site of injury specified and six cases with a third. Forty percent of these second or third natures were coded as “superficial” and there were no significant differences in proportions of these cases among sex or age groups (data not shown).

### Anatomical sites of injury

Leading anatomical sites of injury among males were the face (247/1,084; 22.8%), head (148/1,084; 13.7%) and the arm (111/1,084; 10.2%). Anatomical sites of injury among females were more evenly

distributed: Ankle (54/443; 12.2%), arm (50/443; 11.3%), and face and head (both 47/443; 10.6%) were leading sites. Males were significantly more likely to suffer a facial injury (22.8% males vs. 10.6% females;  $p < 0.001$ ) and less likely to suffer an ankle injury (8.8% males vs. 12.2% females;  $p < 0.05$ ) or neck injury (3.3% males vs. 7.4% females;  $p < 0.05$ ). Some variations in anatomical sites of injury were observed by age, with 14-to-16-year-old players experiencing fewer facial injuries and more lower extremity injuries than their older counterparts (Table 4).

### Discussion

This epidemiological study examined acute injuries experienced by rugby participants in Kingston and area in the hopes of providing objective data for prevention. This study is important because it represents a large contemporary analysis performed on a geographically distinct and general pop-



**TABLE 2**  
Frequency and nature of rugby injuries in Kingston, Canada, by sex (1993–2003)

	Male		Female		Total	
	N	%	N	%	N	%
Sprain/Strain	272	25.1	154	34.8**	426	27.9
Superficial	222	20.5	123	27.8**	345	22.6
Fracture	172	15.9	63	14.2	235	15.4
Open wound	202	18.6	21	4.7***	223	14.6
Neurotrauma	112	10.3	37	8.4	149	9.8
Concussion/Spinal injury*	58	5.4	15	3.4	73	4.8
Minor head injury*	54	5.0	22	5.0	76	5.0
Dislocation/Separation	79	7.3	23	5.2	102	6.7
Other	25	2.3	22	5.0	47	3.1
<b>Total</b>	<b>1,084</b>	<b>100.0</b>	<b>443</b>	<b>100.0</b>	<b>1,527</b>	<b>100.0</b>

\*diagnostic subgroup of Neurotrauma

\*\*p-value chi-square test < 0.01

\*\*\*p-value chi-square test < 0.001

ulation. Such fundamental research can assist in the design of prevention methods to reduce the high incidence of injury associated with rugby participation.

In order to put our results into context, comparison with existing biomedical literature is warranted. Leading types of injury reported in our case series included sprains and strains, head and neck injuries, and injuries experienced while tackling or being tackled. This is consistent with much

of the existing literature,<sup>e.g., 1,3,7,8</sup> with the exception that injuries to the lower limb were the leading type of injury observed in Clarke et al., and Sparks. This difference is likely attributable to differences in data collection. Ours was an emergency-department-based case series, while the comparative published studies have been based upon medical records compiled by sports teams during practices and league matches. Differences in injury patterns experienced by males and females possibly

reflect suspected variations in the types and intensity of physical contact experienced by male and female players. Males were significantly more likely to sustain injuries following collisions and larger proportions of their injuries were open wounds, often to the face. Females were significantly more likely to sustain injuries during contact on the ground or from being hit by a falling player. They also reported significantly more sprains/strains (e.g., ankle, neck) and superficial injuries.

**TABLE 3**  
Frequency and nature of rugby injuries in Kingston, Canada, by age (1993–2003)

	Age groups (years)										
	14-16		17-19		20-24		≥ 25		Total		Trend*
	N	%	N	%	N	%	N	%	N	%	
Sprain/Strain	98	29.8	165	29.6	109	24.6	54	28.3	426	27.9	0.21
Superficial	83	25.2	137	24.6	94	20.9	31	16.2	345	22.6	0.01
Fracture	69	21.0	68	12.2	62	13.8	36	18.8	235	15.4	0.36
Open wound	22	6.7	61	10.9	103	22.9	37	19.4	223	14.6	< 0.01
Neurotrauma	41	12.4	62	11.1	32	7.1	14	7.4	149	9.8	0.01
Concussion/Spinal injury**	29	8.8	25	4.5	12	2.7	7	3.7	73	4.8	< 0.01
Minor head injury**	12	3.6	37	6.6	20	4.5	7	3.7	76	5.0	0.61
Dislocation/Separation	13	4.0	43	7.7	30	6.7	16	8.4	102	6.7	0.10
Other/Unspecified	3	0.9	22	3.9	19	4.2	3	1.6	47	3.1	
Total	329	100.0	558	100.0	449	100.0	191	100.0	1,527	100.0	

\*p-value for linear trend in proportions

\*\*diagnostic subgroup of Neurotrauma

**TABLE 4**  
Anatomical site of rugby injuries in Kingston, Canada, by age (1993–2003)

	Age groups (years)										
	14-16		17-19		20-24		≥ 25		Total		
	N	%	N	%	N	%	N	%	N	%	Trend*
Head/Neck	96	29.2	203	36.4	193	43.0	67	35.1	559	36.6	0.01
Face	39	11.9	99	17.7	115	25.6	41	21.5	294	19.3	0.00
Head	45	13.7	74	13.3	56	12.5	20	10.5	195	12.8	0.29
Neck	12	3.6	30	5.4	22	4.9	6	3.1	70	4.6	0.83
Upper extremity	114	34.7	153	27.4	116	25.8	70	36.6	453	29.7	0.68
Arm	50	15.2	55	9.9	31	6.9	25	13.1	161	10.5	0.05
Shoulder	27	8.2	54	9.7	37	8.2	21	11.0	139	9.1	0.56
Finger/Thumb	20	6.1	34	6.1	41	9.1	21	11.0	116	7.6	0.01
Clavicle	17	5.2	10	1.8	7	1.6	3	1.6	37	2.4	0.01
Lower extremity	96	29.2	149	26.7	97	21.6	37	19.4	379	24.8	0.002
Ankle	46	14.0	53	9.5	38	8.5	12	6.3	149	9.8	0.003
Knee	23	7.0	58	10.4	29	6.5	15	7.9	125	8.2	0.62
Leg	27	8.2	38	6.8	30	6.7	10	5.2	105	6.9	0.22
Abdomen/Thorax	21	6.4	38	6.8	35	7.8	14	7.3	108	7.1	0.45
Core	20	6.1	37	6.6	35	7.8	14	7.3	105	6.9	0.39
Spine	1	0.3	1	0.2	0		0		2	0.1	
Other	2	0.6	15	2.7	8	1.4	3	1.6	28	1.8	
Total	329	100.0	558	100.0	449	100.0	191	100.0	1,527	100.0	

\* *p*-value for linear trend in proportions

Our analysis led to the identification of five “target” types of injury that warrant attention as prevention priorities (Table 5). These patterns were identified as priorities, based on objective criteria, and involved consideration of a cross-tabulation of nature of injury and body part, as well as disposition from the emergency department. The identification process admittedly involved some judgment on our part, although we used standard cut-offs pertaining to frequency (> 5% of the case series) and severity (> 10% with “serious” consequences). In the end, we hoped to identify a parsimonious list of target injury patterns that had importance for primary prevention as well as clinical intervention.

Our list includes some common injury types that, although requiring immediate medical procedures and associated follow-up care (e.g., stitching, casting), are likely to have a favourable long-term prognosis. Examples of these are facial wounds (target

pattern 1) and upper extremity fractures (target pattern 2). The list also includes some less common injury patterns (e.g., shoulder dislocations/separations—target pattern 3; neurotrauma—target pattern 5) that have the potential to lead to long-term medical sequelae. Both general categories of target injury events are of importance and this surveillance initiative provides objective data in support of these events as prevention priorities.

#### **Target pattern 1**

Facial wounds were a common form of injury. These types of wounds may not limit on-going participation in rugby activity but often require treatment by stitches. Facial wounds are more commonly observed in the older age groups and among males. This may potentially be attributed to a higher paced and aggressive style of play. As players become more proficient in rugby skills, they begin to perform them quickly and aggressively, leading to high-speed collisions.

#### **Target pattern 2**

Upper extremity fractures were observed predominantly in younger players, a finding consistent with previous reports.<sup>6</sup> This type of injury ranges from a fracture of the clavicle to fractures of digits of the hand. These injuries can be debilitating on a long-term basis, depending upon their severity. The leading mechanism of this injury was the tackling phase (62/142; 43.7%), and this pattern was observed amongst both genders.

#### **Target pattern 3**

Shoulder dislocations/separations are potentially debilitating injuries that can lead to time off from play. The tackling phase resulted in nearly half of all shoulder dislocations/separations (35/76; 46.1%). Generally, this injury occurs when tackled and then landing on the point of the shoulder. Shoulder dislocations/separations were also more common among the senior age groups, likely as a result of high-impact tackles.

**TABLE 5**  
**Target injury events identified for rugby injury in Kingston, Canada (1993–2003)**

Target injury pattern	Frequency		Serious injury*		Example
	N	%	N	%**	
1. Facial wound	232	15.2	195	84.1	A senior male forward is going into a ruck***, hoping to maintain possession of the ball. He then finds himself on the opposition's side of the ruck as they drive over. His face is stepped on during the process. He heads to the emergency room for stitches.
2. Upper extremity fracture	142	9.3	115	81.0	A smaller back is attempting to pass the ball from a ruck, when a large opposing forward tackles him to the ground. The back's arms are pinned to his side by the opposing forward and he falls forward, fracturing his clavicle as he is pressed to the ground. He is taken to hospital for treatment of a clavicle fracture.
3. Shoulder dislocation/separation	76	5.0	29	38.2	A high school male back is running with the ball when he is tackled by a larger player from the side. He falls sideways and lands on the ground with the point of the shoulder. He is unable to move the shoulder and his collar bone can be seen "standing up". He is taken to hospital for treatment of a third degree shoulder separation.
4. Lower extremity sprain/strain	187	12.2	49	26.2	A high school female back is running with the ball in open field and has only the opposing fullback to beat. She attempts to quickly cut to the outside, but her cleat has sunk into the ground and her foot remains planted. She twists her knee, resulting in a sprain and goes to the hospital for further observation.
5. Neurotrauma	149	9.8	23	15.1	A high-school-level back 1 receives a poor pass from back 2, which bounces along the ground. The back 2 is bent over, running forward in an attempt to pick up the ball, when an opposing flanker hits back 2 in the head with his shoulder. The player is immobilized and taken to hospital. Upon arrival, the player is administered the Glasgow Coma Score, the results of which suggest the need for further investigation for a possible concussion.
Concussion/spinal injury	73	4.8	11	15.1	
Minor head injury	76	5.0	12	15.8	

\* Injuries requiring hospital admission or treatment with follow-up required

\*\* Proportion of target injury

\*\*\* A play whereby the two sets of forwards mass together around the ball, struggling to gain possession of it

### Target pattern 4

Sprains/strains of the lower extremity is the only target injury pattern that is more common among females (67/443; 15.1%) than males (120/1,084; 11.1%) and among the youngest age group (46/329; 14.0). Sprains and strains often occur when tendons and ligaments are stretched beyond normal limits. The forces involved may be major or minor, depending upon the level of impact involved in the injury event.<sup>15</sup> The types of rugby played within the above demographic groups are usually a "low impact" version of the game,

and this injury pattern is consistent with what might be expected from that style of play.<sup>3,9</sup> The tackling phase accounted for 36% of these injuries (68/187) with running or making maneuvers on the field leading to another third of these injuries (57/187; 30.5%). Sprains/strains of the ankle and knee represented 12% of all the rugby injuries (187/1,527)

### Target pattern 5

Neurotrauma (operationally defined as all cases of spinal cord injury, concussion or closed head injury) is the final target form. Head and spinal injury have received con-

siderable media attention in recent past.<sup>16</sup> These injuries can be very debilitating and on rare occasions result in paralysis or death.<sup>17,18</sup> According to the International Rugby Board, players reported to have suffered a concussion are put on a mandatory three-week stand down from any rugby activity. Younger players and males experienced the highest proportions of these injuries. The majority of neurotrauma resulted from the tackling phase (59/149; 39.6%) and collisions on the field (58/149; 38.9%). In total, neurotrauma accounted for 9.8% of all rugby injury. Spinal cord injuries (N = 2) were rare.

## Other notable injury patterns

As in other studies, the tackling phase was the leading cause of injury among rugby players.<sup>1,3,8-11</sup> Sprains/strains were also the most common nature of injury in this population.<sup>1-3,7,12</sup> We also found that the face/head was the most frequently injured anatomical location.<sup>5,19</sup> Foul play accounted for only 2.3% of represented injuries, a level below existing reports.<sup>3,5</sup> However, our estimate should be viewed as conservative as this designation was based on a text description of this injury event, collected in a medical setting.

## Prevention

Unlike other community-based sport programs in Canada (e.g., ice hockey, soccer, basketball), rugby is generally introduced at the high school level, when participants are about 14 years of age. This can, speculatively, lead to an increased risk for injury (all target patterns) due to undeveloped levels of skill. Players should practice fundamental skills to some point of proficiency so that they are not a risk to other players or themselves. Many of the injuries observed (e.g., patterns 2, 3, 5) were a result of collisions on the field. The elements of play need to be closely monitored. Concussion and spinal injuries (pattern 5) are specific injuries that have been shown to be reduced through rule enforcement, attention to technique and team skills.<sup>20</sup> There is an inherent role for education and rule enforcement at player, coaching and game official levels in order to minimize playing risks.

A second approach to prevention is the use of protective equipment. In the context of rugby, most existing protective equipment has demonstrated limited efficacy in the prevention of common forms of injury, with some exceptions (e.g., scrum caps—foam helmets no thicker than 1 cm—support sleeves and mouth guards.<sup>11,12</sup>) Though scrum caps have not been shown to reduce concussion (pattern 5), they have led to reductions in lacerations to the head (pattern 1).<sup>11,12</sup> There is the possibility of further optimization of these caps in the prevention of neurotrauma, such as providing more padding near the temporal area, where most concus-

sive blows occur.<sup>21</sup> Support sleeves have been shown to reduce injury, specifically sprain/strain injury (pattern 4).<sup>11</sup> Our identification of sprain/strain injury as a target priority would support programs to test the adoption of support sleeves. Mouth guards are a common piece of equipment that have been shown to prevent orofacial injuries.<sup>9,11,23,24</sup> They are generally worn because of the idea that they help prevent concussion (pattern 5); a perception that remains under debate.<sup>11,22,23</sup>

In terms of tertiary prevention, it is important that rugby injuries be assessed clinically and properly rehabilitated before the player returns to the sport.<sup>22,25</sup> Players who return to the game with a lingering injury from a prior event are significantly more likely to be re-injured (e.g., patterns 2-5).<sup>22,25</sup> Neurotraumas are an important area of clinical concern due to their relatively high frequency. A player who suffers a concussion is required to take a three-week stand down period from any rugby activity, including practices, irrespective of the severity of the concussion.<sup>3,6,26</sup> However, in reality this rule may be disregarded and the player's return dependent upon the coach's or player's own perceptions. This practice requires further discussion as an obvious prevention priority.

## Limitations

Limitations of our analysis warrant recognition. First, this analysis only considers rugby injuries that present to the emergency department for care. As such, these visits represent only a portion of the injuries that require and/or receive medical attention. It was not possible to determine the number of injuries treated in physicians' offices and outpatient clinics, or identify players who did not seek medical care. Second, because exposure data were not available, we were unable to calculate meaningful rates of injury. Third, descriptions of injury circumstances were based upon self-reports collected as part of an established surveillance program. The CHIRPP system was not developed exclusively for the study of rugby injuries, which limits detail at the record level. For example, descriptions of injury mechanisms are based upon close-ended coding items and the exact natures of the playing

circumstances or physical descriptions of injury-producing events are rarely available. This led to some judgments during the coding of specific mechanisms of injury, and the possibility of random coding errors due to missing or sparse information on the CHIRPP record. For a portion of the injuries, the mechanism could not be specified from the CHIRPP records (Table 1). Available CHIRPP descriptions also do not provide an indication of player position or relative time with respect to the match. Fourth, up to 15% of patients that present to emergency are unable or unwilling to complete the CHIRPP surveillance form and injury descriptions are provided by a proxy respondent or abstracted from the medical record. This process may also lead to misclassified or non-specific data reports. Finally, while standard approaches to the triage and initial management of these injuries are in place, emergency physicians vary in their approach to patient management. This may lead to varying and non-specific diagnoses being recorded on the medical chart and hence the CHIRPP surveillance record.

## Conclusion

This novel analysis profiled the scope and nature of injuries experienced during the sport of rugby. This study is unique in that it encompasses participants at all levels of the sport in a defined population and because of its relative size, compared with existing reports. Our hope is that the identified target injury patterns are helpful in indicating priorities for injury prevention at the grassroots level.

## Acknowledgments

We thank Kathy Bowes, Fenni Loye and Sarah Pickett of the Department of Emergency Medicine, Queen's University, for data collection and their coordination efforts.

The Kingston sites of the Canadian Hospitals Injury Reporting and Prevention Program are funded by The Child Injury Section of the Public Health Agency of Canada. This analysis was supported by an Ontario Neurotrauma Foundation Mentor-Student Award held by Dr Pickett.

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