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The burden of adult obesity in Canada

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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.²

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.^{3,4} 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.^{5,6}

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 2002.⁷ 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.⁸ Although the prevalence of leisure-time physical activity has increased over time,^{9,10} 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.^{11,12}

Hypertension

There is compelling evidence indicating that excess body weight is associated with hypertension in men and women,¹³⁻¹⁵ with relative risks (RR) ranging from 2.2 to 5.7 for obese persons.¹⁶⁻¹⁹ 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.²⁰

Type II diabetes

Obesity is reported to be one of the strongest lifestyle-related factors for developing type II diabetes.²¹ 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.^{13,22,23}

Gallbladder disease

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

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.²⁹ 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.³⁰ 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.³¹⁻³³ The RR of obesity and coronary heart disease is reported to range from 1.3 to 3.6.^{13,21,34-37} Obesity is also associated with an increased risk of both ischemic and hemorrhagic stroke.^{13,16,38,39}

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.⁴⁰ 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.⁴⁰⁻⁴²

Endometrial cancer

Numerous epidemiological studies have shown a positive association between endometrial cancer and excess weight.⁴³⁻⁴⁶ 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.^{47,48}

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.^{49,50} 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.⁵¹ 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.⁵²

Colon cancer

There is growing evidence that obesity is positively associated with colon cancer,⁵³⁻⁵⁵ and that the association is stronger in men than in women.^{56,57} One proposed biological mechanism is that adiposity increases blood insulin levels,⁵⁸ which lower insulin-like growth factor (IGF) binding protein 1 and which may subsequently lead to increased levels of free IGF-1.⁵⁹ IGF-1 has been positively associated with the risk of colon cancer in men⁶⁰ and women.⁶¹ The International Agency for Research on Cancer has estimated that overweight and obesity cause 11 % of colon cancer cases.⁶²

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/m²) 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,⁶³ the 1978-1979 Canada Health Survey,⁶⁴ the 1981 Canada Fitness Survey,⁶⁵ the 1988 Campbell's Survey on Well-being in Canada,⁶⁶ the 1986-1992 Canadian Heart Health Surveys⁶⁷ and the 2004 Canadian Community Health Survey (CCHS), Cycle 2.2.¹ 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.⁶⁸ 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 Janssen² (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 Database² 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.⁶⁹ 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/m²). We used the BMI classification system developed by the World Health Organization (WHO), which defines obesity as a BMI of 30 kg/m² or greater, a cutoff applied to both genders among adults aged 18 and older.¹¹

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:

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

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.⁷⁰

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.¹

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.⁷¹ 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 ² ≥ 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.

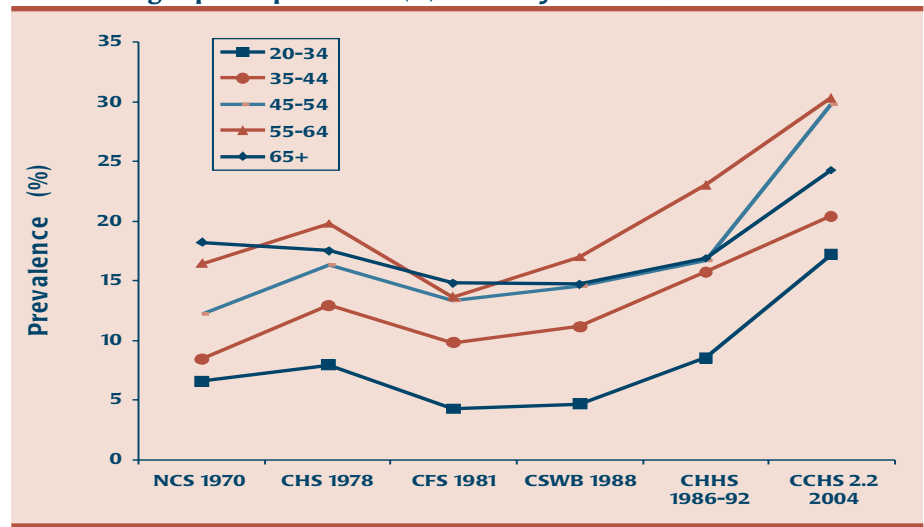
NCS - Nutrition Canada Survey;
CHS - Canada Health Survey;
CFS - Canada Fitness Survey;
CSWB - Campbell's Survey on Well-being in Canada;
CHHS - Canadian Heart Health Survey;
CCHS - Canadian Community Health Survey, 2.2

TABLE 2
Population-attributable fraction (PAF*) (%) by gender and relative risk (RR) of obesity (BMI ≥ 30 kg/m²) and major chronic diseases in Canada (1970-2004)

Relative risks (RR) of obesity and chronic diseases	Hypertension		Type II diabetes disease		Gallbladder disease	
	Men	Women	Men	Women	Men	Women
	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
PAF Increase (%) from 1970 to 2004	102.24	43.10	113.47	47.89	120.33	50.83

*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,² except for endometrial cancer²³)

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

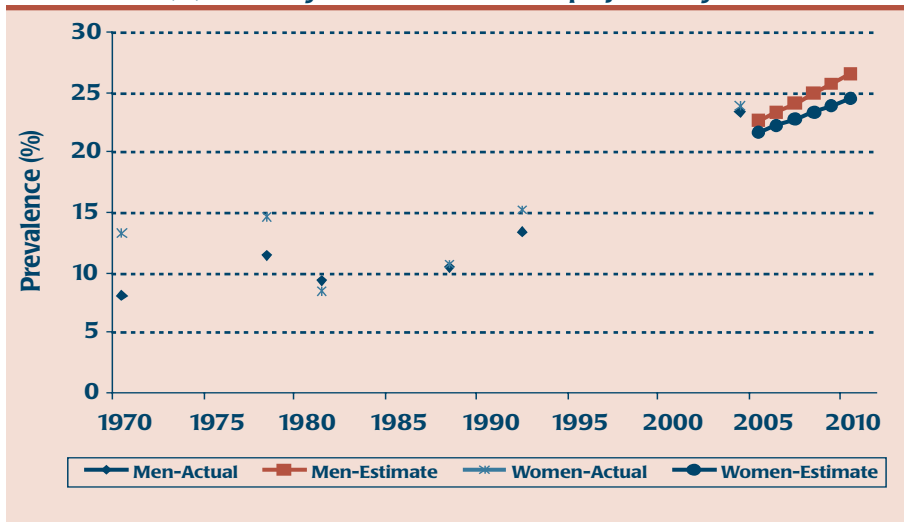
This study highlights the dramatic rise in the prevalence of obesity between 1970 and 2004 in Canadian adults for all age groups. As well, it highlights the impact

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,

Coronary artery disease		Osteoarthritis		Stroke		Endometrial cancer		Postmenopausal breast cancer		Colon cancer	
2.24		1.99		1.50		2.52		1.47		1.45	
PAF (%) of obesity and chronic diseases											
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.²

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.⁷² 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 ²) 30 to < 35		BMI (kg/m ²) ≥ 35		BMI (kg/m ²) ≥ 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.⁶⁹

**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.^{73,74} 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.^{2,12,74} 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.⁷⁵ 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^{69,76}; as a result, Flegal estimated fewer deaths attributable to obesity.⁶⁹ 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.⁶⁹ 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,⁶⁹ whereas most other studies have restricted their analyses to subpopulations such as non-smokers.^{76,77} 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.⁷⁷ 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 1.8.²⁶

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,⁷⁷⁻⁸¹ whereas the recent follow-up of the NHANES cohorts by Flegal⁶⁹ 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.⁸² 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.⁵

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.⁸³ 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.

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