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COST OF OBESITY
IN
ALBERTA

PART I

Prepared for the Alberta Cancer Board

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Errors or misinterpretations and all viewpoints expressed are the sole responsibility of the authors and GPI Atlantic.

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PREFACE: OBESITY COSTS IN THE CONTEXT OF THE GPI

Paradoxically, many health costs associated with obesity, including direct medical costs, are included in the Gross Domestic Product (GDP) and are thus conventionally counted as contributions to economic growth and prosperity. An increase in these costs is therefore mistakenly interpreted as a sign that society is "better off." In the Genuine Progress Index (GPI), by contrast, the costs of illness, accidents, crime, pollution, and other liabilities are counted as costs, not gains, to the economy. Instead, the GPI uses population health and other indicators of wellbeing to measure progress. In other words, an improvement in population health also signifies an overall improvement in societal wellbeing, quality of life, and progress.

This present obesity costing study should be understood in this context—as a wider challenge to our conventional measures of progress and as an effort to provide more accurate signals to policy makers. Because the GDP and economic growth statistics remain our primary measure of progress, increased spending on hospitals, physicians, pharmaceuticals, and other illness- or obesity-related costs is currently counted as a contribution to our wellbeing and prosperity. The same is true for production and spending attributable to cigarette consumption, crime, overwork, toxic pollution, war, accidents, natural disasters, resource depletion, and other liabilities. So long as money is being spent—whether on cigarettes, prisons, weapons, vegetables, or schools—the GDP will continue to grow, regardless of whether that spending signifies an improvement or decline in wellbeing.

Because the GDP makes no distinction between economic activities that create benefit and those that cause harm, it sends misleading signals to policy makers when it is misused as a measure of progress or societal wellbeing, as is generally the case today. As a consequence, disease prevention and health promotion initiatives—including those designed to reduce the current high levels of obesity prevalence—frequently do not receive the same policy attention and funding support accorded to economic stimulus measures.

By contrast, the Genuine Progress Index (GPI) counts the costs of obesity and its consequent adverse health impacts as a liability and loss that should be deducted from rather than added to measures of economic prosperity. The GPI explicitly values health, equity, educational attainment, and peace in society as valuable social assets, and regards higher rates of ill health as signifying a deterioration or depreciation of that human and social capital. Unlike in the GDP, *lower* rates of obesity, cigarette consumption, and ill health make the GPI go up. Consequent reduced health care costs are regarded as savings that can be invested in more productive activities that contribute to wellbeing and social welfare.

In sum, the Genuine Progress Index (GPI), consisting of 20 social, economic, and environmental components, is intended to provide a more comprehensive and accurate assessment of our social wellbeing and quality of life than market statistics are able to do. As such, it is a small step towards a "full cost accounting" system that assigns full value to a society's social, economic, and environmental assets, and also accounts for the full social and environmental costs of

economic activity. The GPI aims to provide regular benchmarks of progress that tell us whether our development strategies and social and economic policies are sustainable and whether they provide net benefits to society when full costs are taken into account. As such, the GPI is both an indicator system that measures progress and an accounting (or economic valuation) system that assesses value.

The latter economic valuation function may be seen as a temporary, but necessary, step in order to overcome the conventional tendency to undervalue the services of unpaid labour, leisure time, natural resources, healthy and safe communities, and other hidden or "free" assets, and in order to make their contribution to prosperity clearly visible. Ideally, it might be argued, cost of illness studies such as this one on obesity costs should not be necessary, and a healthy population should be inherently valued for its own sake. That would be the case if disease prevention and health promotion efforts received strong policy and funding support, and if—as a consequence— obesity trends were declining rather than increasing. In present circumstances where GDP-based measures and economic considerations hold sway in the policy arena, however, economic costing studies such as this one can be vital strategic tools to draw awareness to the true burden of obesity and illness and to the economic value of a healthier populace that are hidden in GDP-based measures, and thus to capture the attention of policy makers and their support for health promotion efforts.

It is often said that a society measures what is important to it. Measuring and understanding obesity trends and costs therefore constitutes one element in a wider effort towards distinguishing between areas of the economy that bring long-lasting societal benefit and those where growth is clearly undesirable. The development of the Genuine Progress Index is dedicated to that broader effort, of which this present study on obesity costs is an illustrative example. In the long term, this work is intended to demonstrate that previously hidden social and natural capital assets and non-material contributions to our quality of life may be extraordinarily valuable, and thus to bring these values and assets more fully into the policy arena for the benefit and wellbeing not only of people with obesity who directly suffer adverse health consequences, but also of the populace as a whole which presently pays the costs of those health outcomes.

EXECUTIVE SUMMARY

In the last three decades obesity has become a global public health challenge on a scale unimaginable in prior generations. The World Health Organization (WHO) reported in 2008 that obesity had reached "epidemic proportions" and that, globally, over 1 billion adults are now overweight, with 300 million of these considered to be clinically obese.[1] WHO notes that "obesity is a complex condition, with serious social and psychological dimensions, affecting virtually all ages and socioeconomic groups."[2] These dimensions include adverse chronic disease consequences, premature mortality, decreased quality of life, social stigma, disability, absenteeism, and productivity losses which together result in substantial economic and social costs to families, governments, businesses, and societies in general.[3]

International and Canadian definitions of obesity define adult obesity for both genders, aged ≥18, in relation to Body Mass Index (BMI), which is calculated as weight in kilograms divided by height in metres squared:[4]

$$\text{Body Mass Index} = \text{weight (kilograms)} / \text{height (metres}^2)$$

The directly measured prevalence of obesity in Canada has more than doubled in the past three decades. Directly measured obesity rates (BMI ≥ 30 kg/m^2) increased from 10.4% of Canadian adults aged ≥ 20 in 1970 to 22.7% in 2004.[5] Among men, the rates nearly tripled—from 7.9% in 1970 to 22.9% in 2004, and for women, the rates increased from 12.9% to 22.5%.

Obesity rates have also risen substantially in all provinces since 1986. Directly measured Canadian and provincial obesity rates for 1986–1992 using data from the Canadian Heart Health Surveys, and for 2004 using data from the Canadian Community Health Survey (CCHS)—both for the population aged ≥18—show that Alberta obesity rates between 1986 and 2004 rose in parallel with those of Canada, although in both time periods the Alberta rates were slightly higher than those of Canada. Thus, obesity rates in Alberta increased by 9 percentage points from 16% to 25% during this time period, while the overall Canadian rate rose by 8 percentage points from 15% to 23%.

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[1] World Health Organization (WHO). *Obesity and Overweight*, 2008; accessed August 2008; available from http://www.who.int/dietphysicalactivity/publications/facts/obesity/en/.

[2] Ibid., accessed.

[3] Patra, Jayadeep, Svetlana Popova, Jurgen Rehm, Susan Bondy, Robynne Flint, and Norman Giesbrecht. *Economic Cost of Chronic Disease in Canada 1995-2003*, Ontario Chronic Disease Prevention Alliance and the Ontario Public Health Association, 2007; accessed July 2008; available from http://www.ocdpa.on.ca/docs/OCDPA_EconomicCosts.pdf.

[4] Statistics Canada. *Health Indicators: Definitions and Data Sources: Body Mass Index (BMI-Canadian Standard)*, Health Canada, Statistics Canada, 2001; accessed June 2008; available from http://www.statcan.ca/english/freepub/82-221-XIE/00401/defin1.htm.

[5] Luo, Wei, Howard Morrison, Margaret de Groh, Chris Waters, Marie DesMeules, Elaine Jones-McLean, Anne-Marie Ugnat, Sylvie Desjardins, Morgan Lim, and Yang Mao. "The Burden of Adult Obesity in Canada," *Chronic Diseases in Canada*, 2007, vol. 27, no. 4: 135-144.

Saskatchewan, Newfoundland and Labrador, and New Brunswick saw the sharpest increases in obesity in this time period (by 15, 12, and 10 percentage points respectively). In 2004, Newfoundland and Labrador had the highest obesity rate in the country (35%), and British Columbia had the lowest (19%).

Among Alberta's 2004 population of 2,345,818 adults aged 18 and over, 37.3% had normal or healthy weights (BMI 18.5–24.9), 35.7% were overweight (BMI 25–29.9), and 25.2% were obese (BMI ≥30). Breaking down the obese adults by class, it is seen that 15.4% of Albertan adults aged ≥18 can be classified as obese class 1 (BMI 30–34.9), 6.7% as obese class 2 (BMI 35–39.9), and 3.2% as obese class 3 (≥40).

Although obesity rates in Alberta appear to be highest for the 45–54 age group (34.9%) and the 55–64 age group (30.5%)—which accords with evidence that the likelihood of obesity increases with age at least into the fifties—it is particularly alarming that 25–34 year-old Albertans also appear to have a very high rate of obesity (26.0%) for such a young age group. This result appears to be considerably higher than the Canadian average for this age group (20.5%), and could have serious implications for future health risks and for Alberta's health care costs as these young adults age.

Obesity is responsible for increased costs to the health care system, employers, and other parties. WHO reports that obesity accounts for between 2% and 7% of total health care costs in high-income countries, but notes that the "true costs are undoubtedly much greater as not all obesity-related conditions are included in the calculations."[6] Very substantial cost savings can potentially be achieved through a reduction in obesity rates and consequent improvements in population health, which in turn can reduce demand on the health care system and improve economic productivity.

This report has estimated the direct and indirect costs of illness that were attributable to obesity among Albertans aged ≥15 years in 2000—with the costs in the summary (Part 2, Chapter 6) then inflated to 2005 Canadian dollars to reflect our use of 2004-05 obesity and disease prevalence rates in the cost calculations. In addition to costs attributable to obesity (Body Mass Index–BMI ≥30 kg/m^2), costs attributable to overweight (BMI 25–29.9), obese class 1 (BMI 30–34.9), and obese classes 2–3 combined (BMI 35–39.9 and BMI ≥40, respectively) were also estimated where data were available. Where possible, the costs were broken down by gender and age group, because these are significant potential confounders of obesity data, produce widely varying results, and enable more effective and targeted interventions among those demographic groups where needs and costs are greatest.

Direct health care costs consist of expenditures for hospitals, drugs, physicians, care in other institutions, and additional direct costs. Indirect costs include the costs of lost economic production by adults due to long- and short-term disability and to premature mortality, defined as death between the ages of 15–74 years.

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[6] World Health Organization (WHO). *Obesity and Overweight*, accessed.

Twenty-two health conditions, for which analysts have found epidemiological evidence that the condition or disease is partially attributable to overweight and obesity, were used to estimate costs attributable to obesity. These health conditions—and the associated excess risks and illness proportions attributable to excess weight—were identified through an extensive literature review, which is reported in Part 1, Chapter 4 of this report.

The specific obesity-related health conditions examined in this study include: type 2 diabetes, hypertension, coronary heart disease, cerebrovascular disease, osteoarthritis, gallbladder disease, asthma, depression, and 14 types of cancer—colorectal cancer, postmenopausal breast cancer, endometrial cancer, kidney cancer, esophageal cancer, ovarian cancer, prostate cancer, pancreatic cancer, non-Hodgkin's lymphoma, multiple myeloma, leukemia, liver cancer, bladder cancer, and stomach cancer. The extensive obesity-related cancer analysis in this study was undertaken to meet the specific interests of the Alberta Cancer Board and is the most detailed breakdown of its kind in Canada to date.

Eric Finkelstein and Phaedra Corso,[7] as well as other analysts,[8] have noted a number of benefits that cost of illness studies, such as this cost of obesity report, can provide to policymakers, health care administrators, and other relevant parties. For example, obesity costing studies can clarify obesity as a societal, public health issue that is larger than an individual, behavioural issue. By focusing attention on the economic burden imposed by obesity, and on the associations of obesity with chronic disease, other health conditions, and productivity losses, costing studies have the potential to mobilize societal interest and resources towards preventing obesity, and can provide motivation for governments to reduce obesity costs and to set priorities for prevention. In addition, costing studies can provide a crucial first step for future economic evaluations of the cost-effectiveness of interventions designed to reduce obesity levels in society.

Since GPI Atlantic's initial work on the cost of obesity in 2000,[9] a number of new developments have occurred that allow far more accurate and methodologically rigorous analysis than was possible at the time. These new developments, which are discussed more fully in subsequent pages, include new obesity definitions, expanded obesity / disease associations based on new and more rigorous epidemiological evidence, new directly measured obesity data from CCHS, new Environmental Burden of Illness in Canada (EBIC) costing data, and substantial methodological improvements that include far-reaching critiques of earlier measurement and costing methodologies.

Most importantly, since 2000, obesity awareness has increased exponentially among both researchers and the general public. This is well illustrated by the substantial increase in new obesity-related literature. A search for obesity-related articles in only one database, Medline, showed that during the 1970s, 10,197 obesity-related articles were indexed in Medline, and

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[7] Finkelstein, Eric, and Phaedra Corso. "Cost-of-Illness Analyses for Policy Making: A Cautionary Tale of Use and Misuse," *Expert Review of Pharmacoeconomics Outcomes Research*, 2003, vol. 3, no. 4: 367-369.

[8] See for example: Colman, Ronald. *The Cost of Chronic Disease in Nova Scotia*, GPI Atlantic and Atlantic Region Population and Public Health Branch, Health Canada, 2002; accessed Nov 2007; available from http://gpiatlantic.org/pdf/health/chronic.pdf.

[9] Colman, Ronald. *Cost of Obesity in Nova Scotia*, GPI Atlantic and Cancer Care Nova Scotia, 2000; accessed June 2008; available from http://www.gpiatlantic.org/pdf/health/obesity/ns-obesity.pdf.

during the 1980s, 11,800 were indexed. During the 1990s, the number rose to 17,754, which then increased by approximately 142% to 42,913 obesity-related articles published between 2000 and 2008 alone. Other databases and grey literature reveal similar increases.

Between 1988 and 2003, Health Canada considered a BMI of 20–24.9 kg/m^2 as "acceptable weight" for adults aged 20 to 64, 25–26.9 kg/m^2 as "some excess weight", and 27 kg/m^2 or higher as "overweight".[10] There was no separate Health Canada classification for "obesity." In 2003, based on new research on the relationship between BMI and the risks of morbidity and mortality, and on emerging international standards, Health Canada updated the guidelines for body weight classifications for (non-pregnant or lactating) adults aged 18 years and over, and for the first time included a separate category for "obesity".[11]

The new guidelines, which describe a body weight classification system that can be used to identify health risks associated with body weight in individuals and populations, are in accord with the World Health Organization (WHO) recommendations that were released in 2000 and have now been widely adopted internationally.[12] The new guidelines identify "underweight" as having a BMI of under 18.5 kg/m^2, "normal weight" as having a BMI of 18.5 to 24.9 kg/m^2, "overweight" as having a BMI of 25.0 to 29.9 kg/m^2, and "obese" as having a BMI of 30 kg/m^2 or greater. The guidelines further divide "obese" into three levels: BMI 30.0 to 34.9 kg/m^2 (obese class 1); 35.0 to 39.9 kg/m^2 (obese class 2); 40 kg/m^2 or greater (obese class 3).[13] In addition, a level of abdominal fat measurement, which is increasingly being used in surveys and studies, was changed from a waist to hip ratio to a waist circumference measure.

Relative health risk levels, as compared with individuals having a "normal" or healthy weight, are associated with the different BMI levels: normal weight is associated with the least health risk; underweight and overweight are associated with increased health risk; obese class 1 is associated with high health risk; obese class 2 is associated with very high health risk; and obese class 3—often called morbid or severe obesity—is associated with extremely high health risk.[14] Peter Katzmarzyk and Caitlin Mason of Queen's University note that obesity class guides treatment options, and that the "use of more aggressive approaches to weight loss (e.g., pharmacotherapy or bariatric surgery) are generally reserved for people with more extreme obesity (class 2 or 3) and those with additional risk factors."[15]

This particular study should be considered as an ongoing work in progress, and all costs should be understood to be estimates rather than precise assessments. In December 2004, the Institute of

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[10] Statistics Canada. *Health Indicators: Definitions and Data Sources: Body Mass Index (BMI-Canadian Standard)*, accessed.

[11] Health Canada. *Canadian Guidelines for Body Weight Classification in Adults*, 2003; accessed June 2008; available from http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/nutrition/weight_book-livres_des_poids-eng.pdf.

[12] Ibid., accessed.

[13] Statistics Canada. *Health Indicators*, Health Statistics Division, Statistics Canada, Catalogue no. 82-221-X, 2008; accessed June 2008; available from http://www.statcan.ca/english/freepub/82-221-XIE/82-221-XIE2008001.pdf. p. 21.

[14] Ibid., accessed. p. 21.

[15] Katzmarzyk, Peter T., and Caitlin Mason. "Prevalence of Class I, II, and III Obesity in Canada," *Canadian Medical Association Journal*, 2006, vol. 174, no. 2: 156-157. p. 156.

Medicine (IOM) of the U.S. National Academy of Sciences held a professional workshop to discuss methodological issues concerning the estimation of the public health burden of lifestyle factors, including obesity, in preventable mortality and morbidity.[16] Speaking about mortality (in remarks that are equally relevant to morbidity), Julie Gerberding, Director of the U.S. Centers for Disease Control and Prevention (CDC), noted during the workshop:

> The biggest challenge is simple: there is not enough research to estimate, with the precision that we would like ultimately to achieve, the contributions of lifestyle factors to mortality, and to reduce their impact. Though much is known that can serve as the basis for public health action, gaps remain concerning how optimally to protect the public's health by measuring the burden of disease, determinants of risky behavior, interventions to change lifestyle, assessing the preventable fraction of deaths from these factors, the cost-effectiveness of interventions, and communications to maximize diffusion of effective interventions.[17]

Gerberding also noted that researchers have been working for four decades to understand the impact of smoking on morbidity and mortality, and that there are still gaps, "particularly regarding multiple risk factors interacting in various populations and at various stages of life."[18] In the beginning stages of this research, lung cancer was the only known co-morbidity of smoking, and it took many more years before heart disease was also found to be associated with tobacco use. However, despite the remaining gaps noted by Gerberding, much more is currently known about the effects of smoking on health than is known about the effects of obesity, diet, or physical activity levels on health, largely because tobacco research has a much longer history, while the bulk of research on the impacts of obesity, diet, and physical inactivity has occurred only within the last decade.

Finkelstein and Corso argue that, although cost of illness studies may "suffer from both data and methodological shortcomings," these studies can still provide valuable information to policy makers and health care providers, provided they are carefully documented:

> [C]arefully documented COI [cost of illness] studies are certainly more valuable than the alternatives of providing no information on the economic burden associated with particular illnesses and injuries. Prevalence-based COI estimates provide valuable information to policy makers, health care providers, and payers and others interested in understanding how their health care dollars are being spent during a particular time period. These estimates can also be used as evidence that more resources should be devoted to prevention efforts for certain conditions…. Before one can assess the possible benefits of an intervention, an understanding of the current environment's burden of disease is necessary…. COI estimates provide that information.[19]

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[16] Institute of Medicine. *Estimating the Contributions of Lifestyle-Related Factors to Preventable Death: A Workshop Summary*, The National Academies Press, 2005; accessed August 2008; available from http://www.iom.edu/?id=27316.

[17] Gerberding, Julie. In Ibid., accessed. p. 3.

[18] Ibid., accessed. p.3.

[19] Finkelstein, and Corso. "Cost-of-Illness Analyses for Policy Making: A Cautionary Tale of Use and Misuse." p. 368.

In Canada there have been very few studies of the cost of obesity, and none of them have included cost breakdowns by age group and gender, or for all BMI classes. In 1999, C. Laird Birmingham of the University of British Columbia et al. produced a frequently cited report on the direct cost of obesity in Canada for 1997, which compared the risk of 10 diseases among Canadian adults aged 20–64 years with a BMI of ≥27—defined at that time as obese—with adults with a BMI <27, who were considered non-obese.[20] Birmingham et al. used self-reported 1994–1995 National Population Health Survey data to estimate the prevalence of obesity in Canada. They estimated the total direct costs attributable to obesity in Canada in 1997 to be over 1.8 billion dollars ($1997)—which amounted to 2.4% of the total health care expenditures for all diseases in Canada in 1997.

Peter Katzmarzyk and Ian Janssen of Queen's University produced a report in 2004 on the direct and indirect economic costs associated with physical inactivity and obesity (BMI ≥30) among Canadian adults aged 20–64 years.[21] They used self-reported BMI data from the 2000/2001 CCHS—which showed the prevalence of obesity to be 14.7% of adults in that age range—to estimate obesity-related costs for 8 diseases. Costs were derived from those reported in EBIC 1998, and inflated to 2001 dollars. Katzmarzyk and Janssen estimated that about 2.2% of total Canadian direct and indirect illness costs, or more than $4.3 billion, were attributable to obesity, with direct costs accounting for about $1.6 billion (1.8% of total health care expenditures), and indirect costs for about $2.7 billion.

This cost of obesity report is the first in Canada to use directly measured BMI data from the 2004 CCHS, as well as 2005 self-reported CCHS data that have been adjusted with a new method developed and tested by Statistics Canada specifically for use with 2005 CCHS data, in order to bring the self-reported data more in line with directly measured data.[22] It is also the first of such studies to include cost breakdowns by obesity classes, gender, and age group, and it estimates obesity-related costs for a wider range of illnesses (particularly cancers) than do the previous studies.

Important caveat:
The 2004 CCHS data could only be used for the illnesses that were enquired about in the survey—diabetes, heart disease, and hypertension. It was deemed important to use these data since height and weight were directly measured in a subsample of the respondents. As discussed in Chapter 3.2 below, directly measured BMI data is vastly more accurate than self-reported BMI data. For example, the directly measured BMI data showed that 25.2% of Albertans were obese in 2004, while the self-reported BMI data showed that 15.8% of Albertans were obese in 2005.

In addition, the data were broken down by gender and age group for the three health conditions because gender and age are considered to be important confounders of the obesity-illness

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[20] Birmingham, C. Laird, Jennifer L. Muller, Anita Palepu, John J. Spinelli, and Aslam H. Anis. "The Cost of Obesity in Canada," *Canadian Medical Association Journal*, 1999, vol. 160, no. 4: 483-488.
[21] Katzmarzyk, Peter T., and Ian Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update," *Canadian Journal of Applied Physiology*, 2004, vol. 29, no. 1: 90-115.
[22] Gorber, Sarah Connor, Margot Shields, Mark S. Tremblay, and Ian McDowell. "The Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity," *Health Reports*, Statistics Canada, 2008, vol. 19, no. 3: 71-82. accessed September 2008; available from http://www.statcan.ca/english/freepub/82-003-XIE/2008003/article/10680-en.htm.

association. Confounding occurs when a third factor (e.g. age) is associated with both the exposure (e.g. obesity) and the outcome of interest (e.g. disease). Concerning age as a confounding factor, Katherine Flegal of the U.S. Centers for Disease Control and Prevention notes:

> The relative risks of obesity among the elderly may well be lower than among young or middle-aged people. Because of the high proportion of health conditions among the elderly and the high health care costs incurred by the elderly, estimates of the attributable fraction are sensitive to relative risks among the elderly.[23]

The importance of using such methods to account for key confounding factors is confirmed by James Robins of the Harvard School of Public Health, who notes that failing to stratify by age when calculating population attributable fractions (PAFs)—the percentage of disease attributable to obesity—for obesity can lead to about a 30 percent error.[24]

Approximately 15,000 Canadians aged ≥14 had their height and weight directly measured in the 2004 CCHS cycle 2.2, compared with about 130,000 Canadians aged ≥12 who reported their own height and weight in the 2005 CCHS cycle 3.1.[25] However, when the data from 2004 were broken down by gender and age groups the sample sizes were significantly reduced and these data had very high coefficients of variation (CV). This is explained more fully in Chapter 4.2.3.6. Basically, the gender breakdowns showed marginal CVs (16.6–≤33.3%), but the age breakdowns showed CVs of more than 33.3%, which is considered to be in the unacceptable range by Statistics Canada.

Therefore, the age and gender breakdowns for diabetes, hypertension, and heart disease are presented here for illustrative purposes, rather than for the purpose of providing statistically significant results. Since gender and age breakdowns are especially relevant to possible interventions designed to reduce obesity in the population, it is important to know which age groups can be targeted most effectively.

Potential costs of obesity that are based on the 2004 CCHS data are also provided by gender and age group for diabetes, hypertension, and heart disease in Part 2 of this report, and these costs are also presented for illustrative purposes because of the high sampling variability of the data. However, summary costs of overweight and obesity in Alberta were not broken down by gender or age group. Therefore, since age and gender costs were not used in the final costing estimates for Alberta, the summary costs can be considered more reliable, although the costs for diabetes, hypertension, and heart disease must still be interpreted with caution.

It must also be noted that the issue of sampling variability for diabetes, hypertension, and heart disease was unfortunately only discovered by the researcher responsible for obtaining the data

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[23] Flegal, Katherine M. "Editorial: Estimating the Impact of Obesity," *Sozial Praventivmedizin / Social and Preventive Medicine*, 2005, vol. 50: 73-74.

[24] Robins, James. In Institute of Medicine. *Estimating the Contributions of Lifestyle-Related Factors to Preventable Death: A Workshop Summary*, accessed.

[25] Approximately 6,000 additional Canadian children and youth aged 2–13 also had their height and weight measured in 2004, but this age group was not included in the costing estimates.

from CCHS after this report was mainly completed. Therefore, although we have added the caveat about the low sample variability for the three diseases in several places throughout the report, it was not taken into consideration during the main writing phase of the research.

Summary of Alberta Cost of Obesity and Overweight Results

This report found that the total direct and indirect costs attributable to overweight and obesity in Alberta for 22 health conditions—for which there exists good epidemiological evidence of partial links to obesity—were **$1,189.1 million** ($C2005), after inflating 2000 costs to 2005 dollars to reflect use of 2004-05 obesity and disease prevalence. This total attributable cost represents approximately 0.6% of Alberta's Gross Domestic Product (GDP) in 2005.[26]

A sample of more specific cost breakdowns follows, with all costs mentioned below also in $2005.

- The total direct health care costs in Alberta attributable to obesity (BMI ≥30) were about $313 million, plus an additional $181 million for private caregiving costs.
- The direct health care costs attributable to obesity (not including caregiving costs) represented about 2.5% of Alberta's total direct health care costs.[27]
- The direct health care costs attributable to overweight (BMI 25–29.9) were about $135 million. This represents about 1.1% of Alberta's total direct health care costs.
- The indirect long-term disability cost attributable to obesity was about $144 million.
- The indirect short-term disability cost attributable to obesity was about $44 million.
- The total premature mortality cost attributable to overweight and obesity combined was about $371 million.
- Coronary heart disease accounted for more than $300 million in total costs attributable to excess weight, diabetes for more than $154 million, and cancers for more than $112 million.

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[26] Statistics Canada. *Gross Domenstic Product (GDP) at Basic Prices. Provincial Gross Domestic Product by Industry. Alberta*, CANSIM Table 379-0025, 2005. According to Statistics Canada, the total Alberta GDP in 2005 was $212,773 million ($2005).

[27] *Economic Burden of Illness in Canada (EBIC), 2000.* Policy Research Unit, Knowledge Information and Data Systems Division, Office of Public Health Practice, Public Health Agency of Canada. Unpublished material supplied by Alan Diener, EBIC Manager to GPI Atlantic, October, 2008. According to EBIC, the total direct health care cost for Alberta in 2000 was $9,589.9 million ($2000), which when inflated to $2005 was $12,345.1 million.

Outline of the report

The current study is divided into two parts, which are separated into two documents. Part 1 (document 1) consists of the Executive Summary and Introduction and an extensive literature review, which determined the main chronic health conditions that can be partially attributed to excess body weight in the population and the prevalence of these conditions in Alberta.

Specifically, Part 1 summarizes several key bodies of evidence that are relevant to (and in some cases essential for) estimates of the burden of obesity, including its economic costs. These sections include:

- A brief summary of results from some of the growing body of evidence on the physical pathways leading from obesity to chronic disease and mortality,
- The methodologies used to estimate the health impacts of obesity,
- The known health impacts of the major obesity-related diseases, and
- The prevalence of these particular diseases in Alberta.

The epidemiological literature connecting obesity with the major chronic diseases is vast, and the examples provided here reference only a small portion of this literature. Where possible we have attempted to reference evidence from meta-analyses that have been conducted for specific health conditions that can be partially attributed to excess weight. Because these meta-analyses in turn assemble, examine, organize, and compare evidence from a very wide range of other studies, adjusting for different variables, it is hoped that this present study ultimately draws on a sufficient body of credible, reliable, and recent epidemiological evidence to provide a reasonably accurate basis for the cost estimates provided.

In Part 1 there are also two sections briefly referencing obesity in Alberta's children and youth and in the province's Aboriginal population. The first section reports the prevalence of obesity in these populations as well as trends over time, and the second section discusses the particular health impacts that have found to be related to obesity in children and youth. However, because the health risks attributable to obesity in these populations have not been quantitatively established in the case of children and youth and because of data limitations in the case of the Aboriginal popoulation, this report has not been able to include children and youth or the Aboriginal population in the cost of obesity estimates, which can therefore be considered be conservative to the degree that they exclude significant portions of the population.

Part 2 of this report, which is in a separate document, estimates the economic costs of obesity in Alberta for the following cost categories—direct health care costs, indirect short-term and long-term disability costs, and costs of premature mortality. These costs are estimated and provided separately for each of the 22 health conditions attributable to overweight and obesity listed above.

Estimates are first provided for Alberta in 2000, since the most recent available cost of illness source for Canada and the provinces is the 2000 *Economic Burden of Illness in Canada*, which was kindly made available to GPI Atlantic by the Public Health Agency of Canada but had not

yet been published at the time of writing. However, because obesity and disease prevalence data used in this study are from the 2004 and 2005 CCHS, the final costs in the summary chapter have been inflated to 2005 dollars.

According to Haslam and James, "obesity is one of the most important known preventable causes of cancer."[28] Because the Alberta Cancer Board, in commissioning this present study, has expressed particular interest in identifying links between obesity and cancer, cancer-related evidence is therefore described in considerably more detail in the pages that follow than evidence related to other illness categories and than in other Canadian obesity cost studies. Fortunately, a major comprehensive new study released in November 2007 by the World Cancer Research Fund and American Institute for Cancer Research and other very recent (2005–08) studies from Germany, the U.K., and Harvard School of Public Health now make this more detailed, site-specific analysis of obesity-cancer links possible.

Basic methodology used in the report

Direct and indirect costs of illness attributable to obesity (BMI ≥30) represent costs for the number of disease cases among obese individuals aged ≥15 years that are in "excess" of the number of disease cases for the same diseases among individuals aged ≥15 years with normal weight (BMI 18.5–24.9).[29] For example, if 125 obese people have colorectal cancer for every 100 people with normal weight who have colorectal cancer, then the costs of colorectal cancer attributable to obesity would be the costs of colorectal cancer among those additional 25 people, which represent the "excess" costs. Costs attributable to obesity are considered to be excess costs that may be partially amenable to interventions taken to reduce obesity in the population.

The association between obesity and health status must be established and quantified before the costs of obesity can be estimated. In order to estimate the economic costs of obesity in any jurisdiction, it is first necessary to assess the total costs of each of the specific health conditions that is related to obesity, then to determine the portion of each of those disease-specific costs that can be partially attributed to obesity, using the previously determined relative risk ratios (RR) and population attributable fractions (PAF), and then to sum the obesity-attributable costs of each of those health conditions.

Thus, cost estimates require information to be calculated or gathered on four basic factors:

1. the total costs of each of the specific health conditions that are related to obesity,
2. the prevalence of the risk factor (in this case obesity) in the population,
3. the relative risk ratio for the outcome (obesity-related diseases) in question, and
4. the proportion of the outcome that can be attributed to the risk.

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[28] Haslam, David W., and W. Philip T. James. "Obesity," *The Lancet*, 2005, vol. 366, no. October 1: 1197-1209. p. 1201.
[29] Hanley, J.A. "A Heuristic Approach to the Formulas for Population Attributable Fraction," *Journal of Epidemiology and Community Health*, 2001, vol. 55: 508-514.

The direct and indirect costs of the specific health conditions were based on cost estimates provided by the Public Health Agency of Canada's *Economic Burden of Illness in Canada, 2000* (EBIC) unpublished report.[30] The EBIC 2000 disease-specific direct and indirect cost estimates for Alberta in 2000 constitute the latest comprehensive cost of illness data available for Canada and the provinces. Therefore all of the costs in this report are first presented in 2000 dollars. However, the final summary costs were then inflated to 2005 dollars to approximate the costs attributable to overweight and obesity for Alberta in the year 2005, which is the base year used, together with 2004, for most of the Alberta disease and obesity prevalence data used in this study.

Where possible, this report relies on directly measured, rather than self-reported, BMI data to determine the prevalence of obesity in the population. The latest directly measured data are only available for 2004 (with a small sub-sample for 2005). Evidence has shown that directly measured BMI data are considerably more accurate than self-reported data, which tend to be biased downward quite sharply.[31] In general, therefore, self-reports tend to underestimate BMI substantially, which results in fewer people being classified as obese than is actually the case. For example, the measured obesity rate for Alberta was 25.2% in 2004, while the self-reported rate in 2005 was 15.8%.

Although 2004 is the most recent year for which directly measured obesity data are available for Canada and the provinces, the 2004 CCHS data did not include results for all of the health conditions needed to estimate the percentages of the obesity-related diseases attributable to excess weight for this study. Therefore, 2005 CCHS data were also used where necessary to supplement the 2004 CCHS data. Although the 2005 data came from self-reported height and weight, which are not as accurate as directly measured rates, the data were adjusted to reflect directly measured rates based on a new method developed by Statistics Canada and tested specifically for use with the 2005 CCHS data.[32] However, this method has not been tested for use with other years of CCHS data. Therefore, although self-reported obesity rates are available through 2007 and are included in this report to show trends in obesity prevalence, unadjusted self-reported rates and their use for costing purposes would give a much more biased and considerably less reliable estimate of costs, as can be seen above by reference to the sharp difference between directly measured and self-reported Alberta obesity prevalence rates.

The relative risk ratio (RR) indicates the degree of risk at the individual level that can be attributed to the causal effects of a risk factor or condition. The relative risk ratio (RR) assessing the link between obesity and a specific illness is determined by comparing the risk among those "exposed" to the risk factor—i.e. the population that is overweight or obese—when compared with the "unexposed"—i.e. the population with "normal" or healthy weights (BMI 18.5–24.9).

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[30] *Economic Burden of Illness in Canada (EBIC), 2000.*

[31] Romero-Corral, A., V.K. Somers, J. Sierra-Johnson, R.J. Thomas, M.L. Collazo-Clavell, J. Korinek, T.G. Allison, J.A. Batsis, F.H. Sert-Kuniyoshi, and F. Lopez-Jimenez. "Accuracy of Body Mass Index in Diagnosing Obesity in the Adult General Population," *International Journal of Obesity*, 2008, vol. 32: 959-966.

[32] Gorber, Shields, Tremblay, and McDowell. "The Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity."

The relative risk ratios were determined to the extent possible, from directly measured BMI data from the 2004 CCHS, cycle 2.2. Where such data were not available from the 2004 CCHS for particular diseases, the relative risks attributable to obesity were estimated on the basis of self-reported BMI data from the 2005 CCHS, cycle 3.1, which were then adjusted to be in line with the directly measured 2004 CCHS data. However, for cancers and gallbladder disease, some of the RRs had to be extrapolated from the epidemiological literature. When RRs were extrapolated they were used with Alberta BMI prevalence data from 2004 CCHS cycle 2.2 to estimate population attributable fractions (PAFs).

Population attributable fractions (PAFs) represent the percentages of specific health conditions or diseases that have been estimated to be attributable to overweight and obesity. PAFs also represent the proportions of the *costs* of each illness attributable to overweight and obesity (also called "excess" costs). The estimation of PAFs in this report are based on a methodology described in detail in Part 1, Chapter 4.2 of this study. PAFs need to be calculated in order to indicate the effect of the risk factor upon the community as a whole—in other words, the proportion of each health outcome at the *population* level that is attributable to the risk factor. That PAF estimation, in turn, is necessary in order to estimate costs attributable to the risk factor.[33]

Due to data limitations, different sources were used to determine the PAFs referenced in this report, which in turn were used to estimate the percentage of the direct and indirect costs of each obesity-related illness that could be attributed to excess weight. Wherever possible, the PAFs have been estimated from 2004 and 2005 CCHS data for Canada. When primary data for Canada from the CCHS were not available for particular illnesses, the PAFs have been extrapolated from the epidemiological literature, or, as noted, estimated using RRs from the literature and the Alberta directly measured BMI prevalence data from the 2004 CCHS cycle 2.2.

Pan-Canadian data were used when the PAFs were estimatated directly from CCHS data because the sample sizes for Alberta were too small to be reliable. Therefore, these pan-Canadian PAF data are assumed to be relevant to Alberta as well—an assumption that can be justified by the similar obesity prevalence rates in Canada and Alberta and by the applicability of the relative risk data to the Canadian population as a whole. In other words, the excess risks attributable to obesity for a particular illness are likely to be relatively similar across the country. To the extent that directly measured Canadian obesity prevalence in 2004 (23%) was somewhat lower than Alberta obesity prevalence (25%), and since the pan-Canadian prevalence was used to estimate PAFs where possible, those PAFs (and the attendant cost results in this study) can be considered conservative.

Using Alberta-specific directly measured 2004 CCHS cycle 2.2 data with RRs found in the epidemiological literature—i.e. for gallbladder disease and some cancer sites—was possible since the estimations in these cases did not rely on the disease distribution found in the CCHS, which was needed to estimate PAFs from the primary data.

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[33] Scott, Keith G., Craig A. Mason, and Derek A. Chapman. "The Use of Epidemiological Methodology as a Means of Influencing Public Policy," *Child Development*, 1999, vol. 70, no. 5: 1263-1272.

The PAFs were then multiplied by the Alberta-specific direct health care costs and indirect disability costs of the disease in order to estimate disease costs attributable to excess weight. Mortality-specific PAFs that were derived from the epidemiological literature were used to estimate premature mortality costs.

Thus, PAFs for type 2 diabetes, hypertension, and coronary heart disease were estimated directly from the 2004 CCHS, cycle 2.2, which directly measured the height and weight of respondents. PAFs for cerebrovascular disease, asthma, osteoarthritis, and depression were estimated directly from 2005 CCHS, cycle 3.1, data. Although 2005 CCHS relied on self-reported height and weight, as previously noted, the 2005 CCHS data were adjusted to reflect directly measured data by the use of a new method developed by Statistics Canada and tested for use with 2005 CCHS.[34]

Because the CCHS did not ask respondents about gallbladder disease or most of the specific types of cancer, the PAFs used to estimate the obesity-attributable costs of 10 of the 14 obesity-related cancer sites examined in this study were derived from two studies by Public Health Agency of Canada researchers.[35] The PAFs for gallbladder disease, esophageal cancer, liver cancer, bladder cancer, and stomach cancer were estimated from RRs found in the international epidemiology literature and BMI prevalence data for Alberta from 2004 CCHS, cycle 2.2. The PAFs used to estimate the premature mortality costs attributable to overweight and obesity combined came from the WHO *Global Burden of Disease* (GBD) report.[36]

Where possible, separate PAFs were estimated for each health condition for total obesity (BMI $\geq$30), obese class 1 (BMI 30–34.9), obese classes 2–3 combined (35–39.9 and $\geq$40, respectively), and overweight (BMI 25–29.9), by gender and age group (ages 15–34, 35–64, and $\geq$65 years). Unfortunately, with the exception of bladder cancer, data were not available to allow the estimation of PAFs for obese class 1 and obese classes 2–3 or for specific age groups for the various cancer sites. The gender, age, and BMI breakdowns provided in this study are more detailed than in any prior obesity cost study in Canada, and may be useful for policy purposes to target interventions towards those demographic groups where the needs and costs are greatest.

Except where indicated otherwise, PAFs by obesity class are reported in this study as proportions of overall disease prevalence or total direct costs within each gender and age group category, rather than as proportions of disease prevalence within that obesity class or as proportions of the total direct cost of the disease. For example, a PAF of 12.8 for females with type 2 diabetes who

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[34] Gorber, Shields, Tremblay, and McDowell. "The Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity."

[35] The PAFs for kidney cancer, colorectal cancer, postmenopausal breast cancer, ovarian cancer, prostate cancer, non-Hdgkin's lymphoma, leukemia, multiple myeloma, and pancreatic cancer were derived from Pan, Sai Yi, Kenneth C. Johnson, Anne-Marie Ugnat, Shi Wu Wen, Yang Mao, and Canadian Cancer Registries Epidemiology Research Group. "Association of Obesity and Cancer Risk in Canada," *American Journal of Epidemiology*, 2004, vol. 159, no. 3: 259-268.
The PAF for endometrial cancer was derived from Luo, Wei, Howard Morrison, Margaret de Groh, Chris Waters, Marie DesMeules, Elaine Jones-McLean, Anne-Marie Ugnat, Sylvie Desjardins, Morgan Lim, and Yang Mao. "The Burden of Adult Obesity in Canada," *Chronic Diseases in Canada*, 2007, vol. 27, no. 4: 135-144.

[36] World Health Organization (WHO). *Global Burden of Disease Summary Tables*, 2008; accessed December 2008; available from http://www.who.int/healthinfo/global_burden_disease/en/index.html.

are aged 35–64 and are in obese class 1 (BMI 30–34.9) indicates that 12.8% of diabetes prevalence among females aged 35–64 can be attributed to obesity (specifically class 1), and 12.8% of the total direct costs of diabetes for females aged 35–64 can be attributed to obesity. Similarly, a PAF of 16.4 for females with type 2 diabetes who are aged ≥15 and in obese class 1 indicates that 16.4% of the total direct costs of type 2 diabetes *among females* aged ≥15 can be attributed to class 1 obesity.

Main results of the costs of obesity study in Alberta

Figure 1 below displays the population attributable fractions (PAFs) for total obesity for both genders combined. These represent the proportions of each health condition that may be attributed to obesity in Alberta. Percentages (PAFs) of the specific health conditions that are attributable to obesity class 1, obesity classes 2–3, and overweight, for both genders combined and for each gender separately, and also for the three age groups were also estimated, as noted, and can be found in Part 2, Chapters 5 and 6, of this report. As noted, these breakdowns are important both because gender, age, and BMI level produce sharply different PAFs and therefore significantly affect obesity-related cost estimates, and because the breakdowns may aid policy makers in targeting interventions cost-effectively where needs and costs are greatest. The PAFs listed below and in Chapters 5 and 6 were used to estimate both the direct and indirect disability-related costs of obesity.

As shown in Figure 1 below, gallbladder disease (45.5%) and type 2 diabetes (36.9%) had the highest proportions of disease attributable to total obesity, followed by esophageal cancer (28.0%), liver cancer (23.3%), hypertension (22.8%), and endometrial cancer (22.0%). Another way of stating these results is that 45% of gallbladder disease prevalence, 37% of type 2 diabetes, 28% of esophageal cancer, and more than a fifth of liver cancer, hypertension, and endometrial cancer could potentially be avoided if all Albertans had healthy weights.

Type 2 diabetes and gallbladder disease also had the highest proportions of obesity attribution in the obesity class 1 and obesity classes 2–3 categories (PAFs not shown in Figure 1 below): Type 2 diabetes had the highest proportion of obesity attribution in the obese class 1 category—with 21.6% of *total* diabetes prevalence attributable to those with a BMI of 30–34.9, and the second highest proportion in the obese classes 2–3 category (15.3% of total diabetes prevalence).

Gallbladder disease had the highest proportion of obesity attribution in the obese classes 2–3 category (27.8%), and the second highest in the obese class 1 category (17.7%). This indicates that, for gallbladder disease in particular, the risk rises sharply with BMI level and degree of obesity. In other words, even though Albertans with a BMI of 35 or more account for about 10% of the adult population, they account for nearly three times as high a proportion of gallbladder disease.

Esophageal cancer (22.9%), and kidney cancer (20.3%) had the highest proportions of illness attributable to overweight—BMI 25–29.9—(PAFs not shown in Figure 1)), followed by gallbladder disease (16.4%), multiple myeloma (13.9%), type 2 diabetes (11.7%), and colorectal

cancer (11.3%). This indicates that susceptibility to these particular illnesses can be significant even at lower BMI levels.

Figure 1. Percentages (PAFs) of specific health conditions attributable to obesity among both genders
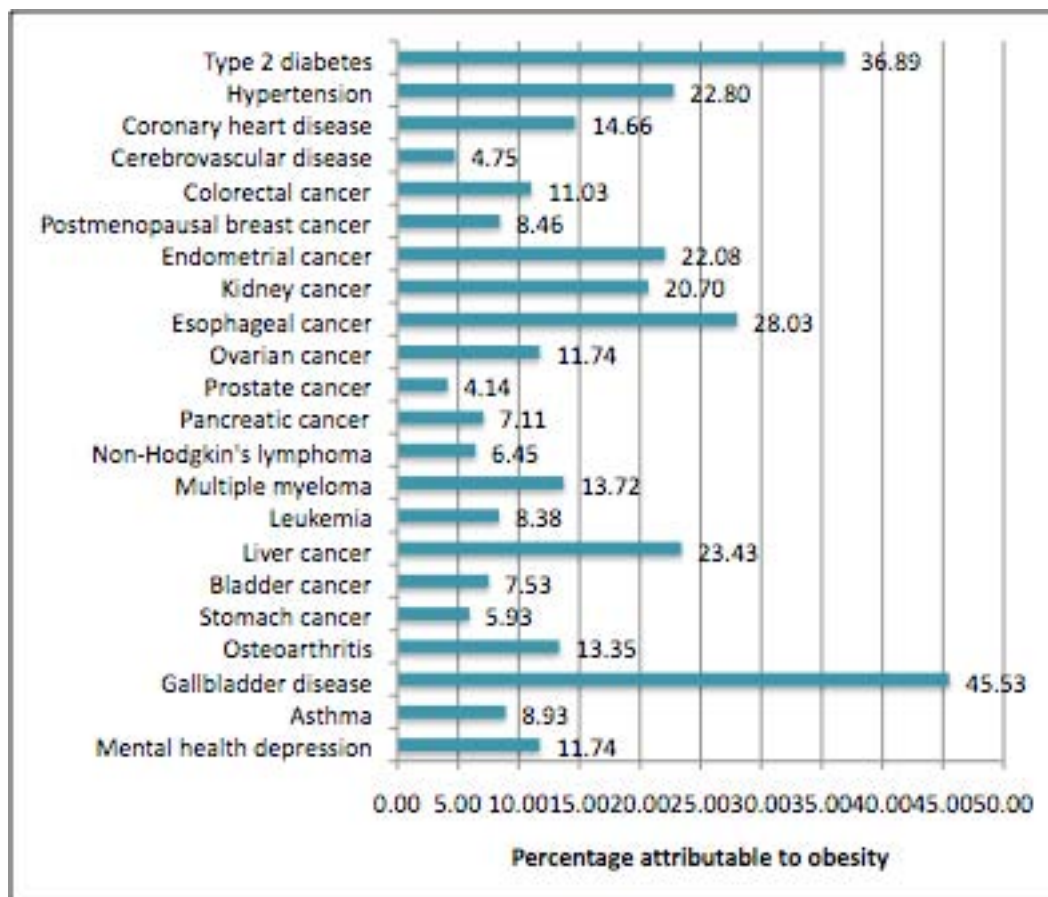
Note: PAF – denotes population attributable fraction.

Figure 2 below shows the percentages (PAFs) of illness attributable to obesity by gender. The discussion below also reports PAFs attributable to overweight and obesity classes 1–3, but only the PAFs attributable to total obesity are shown in Figure 2. Please see Chapter 5 of Part 2 for a full listing of age, gender, and BMI-specific PAFs. In general, males had higher percentages of illness attributable to obesity than females, with the exception of depression, asthma, leukemia, non-Hodgkin's lymphoma, pancreatic cancer, and cerebrovascular disease, to which obese females were more susceptible than males. The PAFs for hypertension were identical for both genders.

Among males, gallbladder disease (67.31%), type 2 diabetes (41.90%), esophageal cancer (30.44%), liver cancer (28.23%), bladder cancer (27.70%), and kidney cancer (25.59%) had the

highest percentages of disease attributable to obesity. For each of these illnesses, the obesity-related PAFs were significantly higher for males than for females.

Among the listed diseases, cerebrovascular disease had the lowest obesity-attributable percentage among males (2% vs 7.1% among females), and only showed an excess percentage for males in the obese class 1 category (1.1%), and in the aged 35–64 category (8.4%).

Among males, the PAFs for type 2 diabetes (27% of total male diabetes prevalence), gallbladder disease (26%), and bladder cancer (17.7%) were also especially elevated in the obese class 1 category. The percentages attributable to obese classes 2–3 among males were also high for gallbladder disease (41.31%) and type 2 diabetes (14.9%). Unfortunately, due to data limitations, it was not possible to estimate BMI-specific PAFs for most of the cancer sites for obese classes 1–3, and these are therefore provided in this study only for total obesity and overweight.

Among the proportions of illness attributable to overweight (BMI 25–29.9) in males, the highest percentages were for bladder cancer (41.1%), kidney cancer (29.2%), esophageal cancer (24.7%), and multiple myeloma (20.4%). This again indicates that susceptibility to these particular illnesses is increased even at lower BMI levels. For cerebrovascular disease, asthma, and depression there were no PAFs attributable to overweight among males, since the epidemiological evidence does not indicate any excess risk for males at lower BMI levels for these particular illnesses.

In general, the highest proportions of illnesses attributable to obesity among males were in the 35–64 age group, indicating that nutrition, exercise, and healthy weight initiatives targeted to middle-aged Albertans might be particularly cost-effective. Gallbladder disease showed an extremely high proportion of illness prevalence attributable to obesity among males under 55 years (98.0% of males in the age group category and 58% of all male gallbladder disease prevalence), indicating that almost the entire prevalence of the disease among Albertan males aged <55 was attributable to obesity, and that the disease might be virtually eliminated among these Albertan males if they all had healthy weights.[37] This is not surprising considering that few people die of gallbladder disease and those that do are mainly over age 55. For males under the age of 55 who die of gallbladder disease, the vast majority of them are obese.

Type 2 diabetes (54.7%) and osteoarthritis (27.1%) showed the highest illness proportions attributable to obesity among males in the 15–34 age group, though these high relative percentages must be tempered by the reality that the prevalence of these and other obesity-related diseases in this younger age group is much less than in older age groups. Cerebrovascular disease showed no obesity-attributable percentages in the youngest and oldest age groups.

Among females, the highest percentages of diseases attributable to obesity were found for gallbladder disease (38.3%), type 2 diabetes (32.0%), hypertension (22.8%), bladder cancer (22.6%), endometrial cancer (22.1%), and esophageal cancer (19.3%). Again, these percentages might be colloquially be thought of as the potential for disease reduction that might exist if all

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[37] Due to data limitations, the age groups for gallbladder disease—<55 and ≥55—were different from the age groups used for the other health conditions.

Albertan females had healthy (normal) weights (BMI 18.5–24.9). For females, stomach cancer showed no association with obesity.

Among all the listed diseases, type 2 diabetes had the highest percentage attributable to obesity in the obese class 1 category among females (16.4% of all female diabetes prevalence), and gallbladder disease had the highest attributable percentage in the obese classes 2–3 category (23.3% of all female gallbladder disease prevalence).

Among the percentages attributable to overweight (BMI 25–29.9) among females, bladder cancer (30.3% of all female bladder cancer prevalence), gallbladder disease (16.8%), esophageal cancer (16.2%), and type 2 diabetes (15.4%) showed the highest PAFs—indicating that these illnesses show strong associations with excess weight at comparatively low levels of BMI.

With the exception of asthma, which had the highest percentage of disease attributable to obesity in the oldest female age group (19.5%), the other diseases showed the highest obesity-attributable percentages among females aged 35–64. Coronary heart disease showed a relatively high obesity-attributable percentage in the female middle age group (23%), but no attributable percentage in the youngest age group (-6.7%) and a low percentage in the oldest age group (4.6%). As noted above, this age-specific evidence again indicates that nutrition, exercise, and other healthy weight initiatives targeted to middle-aged Albertans may be highly cost-effective in reducing the prevalence of a range of obesity-related ailments.

Figure 2. Percentage (PAF) of disease attributable to obesity, by gender, aged ≥15
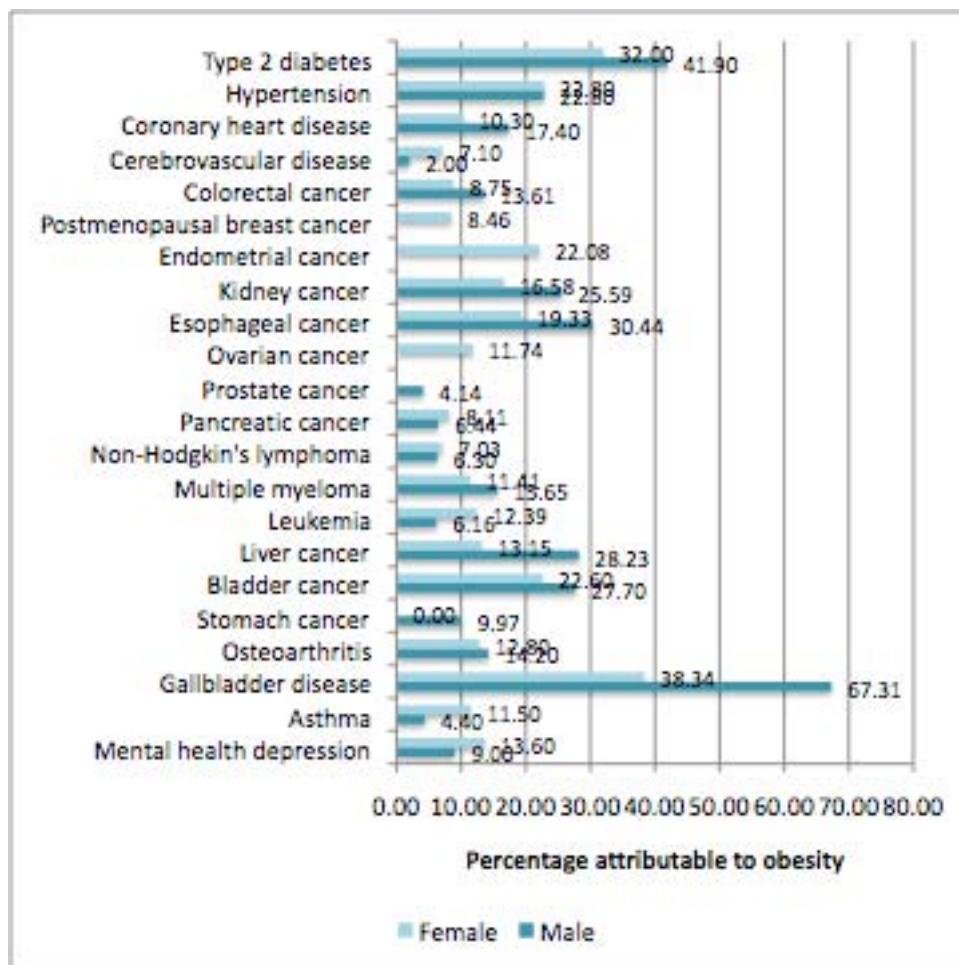
Figure 3 below shows the percentage (PAF) of premature mortality that can be attributed to overweight and obesity combined for selected diseases, as reported in the World Health Organization's (WHO) *Global Burden of Disease* study.[38] Use of this source for obesity-attributable premature mortality PAFs and cost estimates, rather than use of the primary data from the CCHS that were used for the direct and disability cost estimates in this study, was necessary because of major data limitations that are explained more fully in Chapter 5.6.1. Unfortunately PAFs in the WHO study were only available for 7 of the 22 health conditions examined in this report. Premature mortality costs for the other 15 health conditions were based on the PAF for all-cause mortality (10%) as estimated by the WHO study, which likely underestimates the actual obesity-attributable costs.[39]

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[38] World Health Organization (WHO). *Global Burden of Disease Summary Tables*, accessed.

[39] Ezzati, Majid, Stephen Vander Hoorn, Alan D. Lopez, Goodarz Danaei, Anthony Rodgers, Colin D. Mathers, and Christopher J.L. Murray. "Comparative Quantification of Mortality and Burden of Disease Attributable to Selected Risk Factors," in *Global Burden of Disease and Risk Factors*, ed. Lopez, Alan D., Colin D. Mathers, Majid Ezzati, Dean T. Jamison and Christopher J. L. Murray, Chapter 4: 241-396. New York: Oxford University Press; The

For type 2 diabetes, 84.2% of premature mortality cases could be attributed to overweight and obesity combined, as could 55.3% of hypertension, 55.2% of endometrial cancer, 42.0% of coronary heart disease, 26.2% of cerebrovascular disease, 18.7% of colorectal cancer, and 18.2% of postmenopausal breast cancer. These percentages are higher than those for direct health care because in this case the PAF represents both obesity and overweight for both genders combined. PAFs for direct health care include obesity and overweight separately. In addition, for premature mortality the age range is from 15–74 years, while for direct health care, the age range includes ≥75.

Figure 3. Percentage (PAF) of premature mortality from selected diseases attributable to overweight and obesity combined
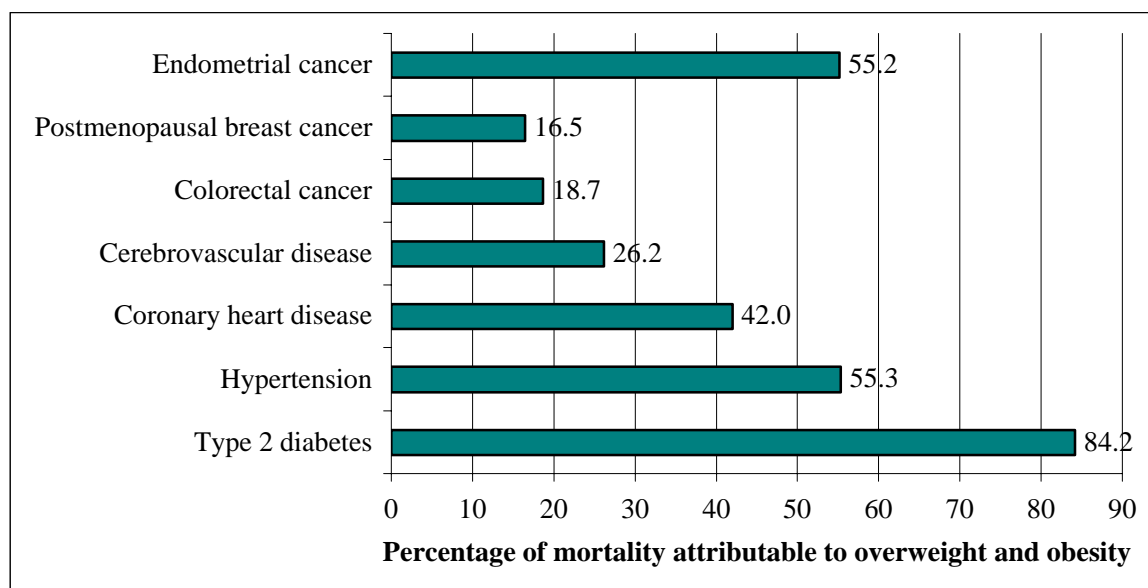
Source: World Health Organization (WHO). *Global Burden of Disease Summary Tables*, 2008; accessed December 2008; available from http://www.who.int/healthinfo/global_burden_disease/en/index.html.

Figure 4 and Table 1 below show the total direct health care costs and indirect costs of illness of the 22 obesity-related health conditions attributable to overweight and obesity for Albertans aged ≥15 years in 2000, with the costs inflated to $2005 to reflect the reality that 2004-05 obesity and disease prevalence rates were used to calculate the PAFs that form the basis of the cost estimates. In Part 2, Chapters 5 and 6, of the full report, the costs are also given by gender and age group.

The results of this study estimate that approximately $1,189 million ($2005) in direct and indirect costs was attributable to overweight and obesity combined (BMI ≥25) in Alberta in 2000 when the costs are inflated to 2005 dollars. Although the latest cost of illness figures for Canada and the provinces are from EBIC 2000, it is reasonable to consider the $1,189 million cost estimate as reflecting 2005 costs in Alberta, as obesity, overweight, and disease prevalence rates for 2004-05 were used in the calculations.

This total cost of obesity and overweight in Alberta ($1,189 million) represents approximately 30% of the $3,788.6 million total direct and indirect costs of the 22 specific health conditions in Alberta that were partially attributable to overweight and obesity. This $1,189 million obesity and overweight cost also represents about 6% of the total direct and indirect costs for all health conditions in Alberta ($18,718 million) in 2000 ($2005).

The $1,189 million in obesity and overweight-related costs in Alberta, which represents direct health care costs and indirect costs, were distributed as follows:

Direct health care costs attributable to overweight and obesity (BMI ≥25) represented approximately 53% of the total direct and indirect costs of illness attributable to overweight and obesity:
- The total direct health care attributable cost was approximately $630 million.
- $312.9 million in direct health care costs plus an additional $181.8 million for private caregiving costs was attributable to obesity (BMI ≥30), and
- $135.4 million in direct health care costs was attributable to overweight (BMI 25–29.9).

Although private caregiving costs could not be estimated by diagnostic category, and are therefore not included in the direct cost estimates for specific diseases, they are included here as an important additional component of direct health care costs that was not included in the EBIC estimates.

Indirect costs attributable to obesity represented 47.2% of the total overweight and obesity attributable costs:
- The total indirect attributable cost was approximately $559.0 million.
- Based on EBIC 2000, the indirect short-term and long-term disability costs attributable to obesity (BMI ≥30) were approximately $187.9 million in productivity losses to the economy—$44.2 million for short-term disability costs, and $143.7 million for long-term costs.
- Indirect disability costs attributable to overweight (BMI ≥25) could not be determined based on the available data. Since overweight-related illness is certainly responsible for a degree of disability and productivity loss, the total cost estimates provided here must therefore be considered conservative to the extent that overweight-related disability costs are excluded from the totals.
- The premature mortality cost attributable to overweight and obesity combined (BMI ≥25) is estimated at approximately $371.1 million.

Coronary heart disease ($299.4 million) had the highest direct and indirect cost of illness attributable to overweight and obesity (BMI ≥25) in Alberta in 2000, with the costs inflated to 2005 dollars to reflect the reality that 2004-05 obesity, overweight, and disease prevalence rates were used in the estimates. If caregiving costs, which could not be attributed by diagnostic category, were included, the total CHD cost attributable to excess weight would certainly be well over $300 million. This BMI-attributable CHD cost was about twice as high as the total cost for type 2 diabetes ($154.1 million)—the disease accounting for the next highest total attributable cost.

Hypertension ($121.8 million), osteoarthritis ($119 million), and the 14 cancer sites combined ($111.8 million) represented the next highest BMI-attributable costs after coronary heart disease and diabetes. Again, all these disease-specific cost estimates would be higher if caregiving costs were included.

Among the cancer sites, colorectal cancer ($29.4 million), postmenopausal breast cancer ($14.3 million), and leukemia ($10.2 million) represented the highest direct and indirect costs attributable to overweight and obesity in Alberta.

Figure 4. Direct and indirect costs of illness attributable to overweight and obesity, Alberta, $2005.

Cost ($2005 Million)

Disease	Cost
Type 2 diabetes	154.0
Hypertension	121.8
Coronary heart disease	299.4
Cerebrovascular disease	45.4
Total cancer	111.8
Colorectal cancer	29.4
Postmenopausal breast cancer	14.3
Endometrial cancer	8.2
Kidney cancer	5.3
Esophageal cancer	5.5
Ovarian cancer	4.4
Prostate cancer	5.9
Pancreatic cancer	6.1
Non-Hodgkin's lymphoma	5.8
Multiple myeloma	3.9
Leukemia	10.2
Liver cancer	4.0
Bladder cancer	4.4
Stomach cancer	4.4
Osteoarthritis	119.4
Gallbladder disease	87.5
Asthma	22.2
Mental health depression	46.0
Caregiving	181.8

Note: Disease-specific cost estimates exclude caregiving costs, which are listed separately.

Table 1. Total direct and indirect costs attributable to overweight and obesity, aged ≥15, Alberta, $2005

| Diagnostic category | Total direct and indirect costs minus caregiving | Direct cost | | | | Indirect cost | | |
		Total obese	Obese class 1	Obese classes 2-3	Over-weight	Long-term Total obese	Short-term Total obese	Premature mortality Total overweight and obese
Type 2 diabetes	154,028,464	58,473,890	34,299,733	24,174,156	18,448,490	21,998,669	1,768,952	53,338,463
Hypertension	121,780,910	72,050,102	38,204,319	33,845,782	29,499,009	6,633,592	2,516,429	11,081,778
Coronary heart disease	299,392,275	50,132,160	32,542,660	17,589,502	34,897,196	8,052,045	3,032,420	203,278,454
Cerebrovascular disease	45,416,554	8,048,704	3,783,126	3,560,678	2,008,587	4,099,779	1,514,074	29,745,410
Colorectal cancer	29,425,235	4,848,951	–	–	4,962,193	1,608,900	521,920	17,483,271
Postmenopausal breast cancer	14,330,807	1,261,430	–	–	608,350	664,475	215,552	11,581,000
Endometrial cancer	8,225,819	766,936	–	–	–	895,703	290,562	6,272,618
Kidney cancer	5,286,336	1,913,440	–	–	1,872,768	250,140	81,140	1,168,848
Esophageal cancer	5,500,881	1,125,091	–	–	919,024	120,965	39,240	3,296,561
Ovarian cancer	4,359,076	606,210	–	–	198,800	243,190	78,890	3,231,986
Prostate cancer	5,895,071	887,828	–	–	1,290,996	425,220	137,940	3,153,087
Pancreatic cancer	6,105,097	581,439	–	–	49,907	61,368	19,907	5,392,476
Non-Hodgkin's lymphoma	5,756,615	1,062,004	–	–	777,157	347,453	112,713	3,457,288
Multiple myeloma	3,910,559	703,827	–	–	714,087	227,985	73,957	2,190,703
Leukemia	9,921,848	1,394,428	–	–	1,544,187	745,044	241,689	5,996,500
Liver cancer	4,030,144	868,077	–	–	222,669	80,891	26,240	2,832,267
Bladder cancer	4,438,334	703,870	399,140	304,730	564,592	435,449	141,258	2,593,165
Stomach cancer	4,366,474	472,633	–	–	0	35,760	11,622	3,846,459
Osteoarthritis	119,446,276	16,758,066	10,215,887	6,542,176	8,599,992	77,853,588	16,235,130	0
Gallbladder disease	87,531,209	56,208,342	21,889,659	34,318,682	20,273,646	3,295,628	7,578,637	174,956
Asthma	22,170,096	7,008,020	3,854,655	3,153,365	1,674,219	7,357,240	5,468,724	661,893
Mental health depression	45,966,095	27,005,317	12,402,023	14,603,295	6,287,141	8,290,577	4,079,261	303,799
Total	1,007,284,175	312,880,765	–	–	135,413,010	143,723,661	44,186,257	371,080,982
Caregiving		181,800,000						
Total direct and indirect costs attributable to overweight and obesity					**$1,189.1 million ($2005)**			

Note: – indicates that the cost is not available because of data limitations.

For several reasons, the costs attributable to overweight and obesity estimated in this report represent conservative estimates, and it is highly likely that the costs may be underestimated. For example, the potentially large costs of pain and suffering that may be attributable to obesity in the population were not included. Also it was not possible to include costs of obesity for children and youth under the age of 15, or for the Aboriginal population living on reserves in Alberta—which has much higher rates of obesity than the population at large.

Because many of the health impacts of obesity are chronic conditions that take time to develop and are generally seen later in life, the links between particular health conditions and obesity in children and youth are less clear than for adults.[40] However, there is clear evidence of increased prevalence in children and youth of various obesity-related health conditions, including type 2 diabetes, some orthopedic complications, and psychosocial problems connected both with social stigma and mental afflictions like depression and anxiety—all of which produce direct and indirect costs excluded from the estimates in this study. Ample evidence now indicates that the average age of so-called "adult-onset" or type 2 diabetics is also getting younger.[41]

Aboriginal peoples have particularly high rates of obesity and obesity-attributable health conditions such as type 2 diabetes. Therefore, this population likely represents considerable direct and indirect costs attributable to excess weight.

Because gender and age are acknowledged as two of the most important confounding factors affecting obesity cost results, this study breaks down cost estimates by gender and age. However, confounding factors beyond gender and age—including risk behaviours like smoking, lack of physical exercise, and an unhealthy diet—were not considered in this report and may have an impact on the costs. For example, physical inactivity and an unhealthy diet are known to contribute to excess weight in the population, and will therefore also contribute to increased obesity costs in the future.

In addition to increasing the direct risks of chronic illness and premature mortality, obesity is also a symptom of deeper, underlying social trends that also have cost implications and that point to potential interventions that may target root rather than proximate causes of obesity-related illnesses. Because of time and resource limitations, this study was not able to review potential interventions that might lower obesity rates in the population, or the many systemic forces driving the rise of obesity, which are areas for potential interventions.[42] These areas have been identified as including modern lifestyles, work environments, urban design and obesogenic (obesity-producing) environments, transportation systems, food production systems,

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[40] Access Economics. *The Economic Costs of Obesity*, Diabetes Australia, 2006; accessed June 2008; available from http://www.accesseconomics.com.au/publicationsreports/showreport.php?id=102.

[41] Gardner, Gary, and Brian Halweil, "Nourishing the Underfed and Overfed," chapter 4 in Worldwatch Institute, *State of the World 2000,* W.W. Norton and Co., New York, 2000, p. 72.

[42] These areas were in fact researched for this study and substantial materials collected. However, the technical, data, and methodological challenges and complications involved in the actual RR, PAF, and cost estimates in this project stretched the resources available for this study so far that intended chapters on causes and interventions were not possible.

technological developments, and economic growth itself.[43] For example, sedentary rather than physically active lifestyles, and western diets that rely on energy-dense and "fast" foods rather than whole foods, are among the elements of modern lifestyles that are known to contribute to obesity.[44] As well, many time-stressed dual-earner families struggling to juggle the combined demands of paid and unpaid work rely on fatty and unhealthy fast foods when they do not have time to shop and cook at home.[45]

However, obesity is only one possible consequence of poor nutrition and a sedentary lifestyle, which are risk factors in their own right for many chronic illnesses. For example, researchers at the World Cancer Research Fund and the American Institute for Cancer Research report that changes in food and nutrition, regular physical activity, and avoidance of obesity could prevent 30%–40% of all cancers world-wide over time—at least as many cases as could be prevented by a cessation of smoking.[46] This indicates that systemic interventions designed to reduce obesity can potentially reduce other links to chronic disease as well. Indeed, interventions directed at underlying systemic factors may altogether be more effective in the long term than those targeting symptoms alone.

In the future, as more data become available, as methodologies become increasingly refined, and as research in these new areas becomes ever more focused, obesity cost estimates will hopefully become more precise over time. Since 2000 alone, as noted, substantial improvements in both data availability and estimation methods, along with significant advances in epidemiological research on obesity-disease associations, now allow far better cost estimates than were possible when GPI Atlantic produced its initial obesity cost studies in 2000. A decade from now, we can expect further significant improvements on what we can offer here.

Although the causes of increasing obesity in the population are complex, and involve lifestyle and social trends that are resistant to change, nevertheless, almost all of the chronic conditions attributable to obesity are reversible and potentially preventable through nutritional education and other weight reduction, health promotion, and social initiatives.

The costs of illness attributable to overweight and obesity in Alberta might be taken to represent the savings that could potentially accrue to Alberta if interventions were able to reduce the total economic burden of illness attributable to overweight and obesity in the population to the burden of illness attributable to the population with healthy (i.e. "normal") weights. Such a use of obesity cost estimates, however, does not take into consideration the potential costs of the interventions needed to help the population reach healthy weight levels. Nevertheless, this cost estimate is a necessary first step for future cost-benefit and cost-effectiveness analyses that also account for the costs and effectiveness of interventions that may help address the increasing rates of obesity in the population. Due to resource limitations, as noted, a review of effective interventions was not possible for this study, and this is strongly recommended as a next step.

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[43] World Health Organization (WHO). *Obesity and Overweight*, accessed.

[44] Ibid., accessed.

[45] Colman. *Cost of Obesity in Nova Scotia*, accessed.

[46] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, 2007; accessed August 2008; available from http://www.dietandcancerreport.org/?p=ER.

The nearly $1.2 billion in estimated costs attributable to overweight and obesity in the Alberta population represent a considerable economic and social cost not only to the Alberta health care system, but also to businesses through lost production, to individuals through out-of-pocket and caregiving costs and through unpaid work losses, and to society as a whole through production losses and through the total costs of a population living with less than optimal health. If even a portion of these very substantial costs of illness attributable to overweight and obesity could be reduced, this would result not only in reduced spending on preventable illness but also in the multiple social, economic, and personal benefits accruing from a healthier population in general.

1. Introduction: Purpose and context of the study

Obesity has become a global public health problem in the last three decades on a scale that would have been unimaginable to prior generations. The World Health Organization (WHO) reported in 2008 that obesity had reached "epidemic proportions" and that, globally, over 1 billion adults can be considered overweight, with 300 million of these considered to be clinically obese.[47] WHO notes that "obesity is a complex condition, with serious social and psychological dimensions, affecting virtually all ages and socioeconomic groups."[48] These dimensions include adverse chronic disease consequences, premature mortality, decreased quality of life, social stigma, disability, absenteeism, and productivity losses which together result in substantial economic and social costs to families, governments, businesses, and societies in general.[49]

W. Phillip T. James et al., writing for the WHO Global Burden of Disease project, found that:

> The proportions of the global burden of disease attributable to increases in BMI [Body Mass Index] were 58% for type II diabetes, 21% for ischaemic heart disease, 39% for hypertensive disease, 23% for ischaemic stroke, 12% for colon cancer, 8% for postmenopausal breast cancer and 32% for endometrial cancer in women, and 13% for osteoarthritis.[50]

WHO also reports that at least 80% of premature heart disease, stroke, and type 2 diabetes, as well as 40% of cancers—all of which are chronic diseases for which obesity is a risk factor—could be prevented through a healthy diet, regular physical activity, and avoidance of tobacco.[51] According to WHO:

> Numerous improvements can be achieved by investing in chronic disease prevention, with greatest improvement in such areas as the health of the general public and healthcare expenditures. As both direct and indirect costs of chronic disease are significantly high, an effective prevention approach can indeed minimize the economic and social burden to the health of society as a whole.[52]

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[47] World Health Organization (WHO). *Obesity and Overweight*, accessed.

[48] Ibid., accessed.

[49] Patra, Popova, Rehm, Bondy, Flint, and Giesbrecht. *Economic Cost of Chronic Disease in Canada 1995-2003*, accessed.

[50] James, W. Phillip T., Rachel Jackson-Leach, Cliona Ni Mhurchu, Eleni Kalamara, Maryam Shayeghi, Neville J. Rigby, Chozuru Nishida, and Anthony Rodgers. "Overweight and Obesity (High Body Mass Index)," in *Comparative Quantification of Health Risks – Global and Regional Burden of Diseases Attributable to Selected Major Risk Factors*, ed. Ezzati, Majid, Alan D. Lopez, Anthony Rodgers and Christopher J.L. Murray, Vol 1, Chapter 8, 497-596, World Health Organization, 2004; accessed September 2008; available from http://www.who.int/publications/cra/chapters/volume1/0497-0596.pdf.

[51] World Health Organization. *The Impact of Chronic Disease in Canada*, 2005; accessed September 2008; available from http://www.who.int/chp/chronic_disease_report/media/canada.pdf.

[52] Patra, Popova, Rehm, Bondy, Flint, and Giesbrecht. *Economic Cost of Chronic Disease in Canada 1995-2003*, accessed.citing World Health Organization (WHO). *Preventing Chronic Diseases: A Vital*

The increase in rates of overweight and obesity among children, who are developing health conditions such as type 2 diabetes, high blood pressure, and heart disease at earlier ages than previously, is especially troubling. According to WHO, worldwide, approximately 17.6 million children under the age of five are overweight.[53]

Figure 5 below illustrates the increasing obesity rates—defined as the percentage of adults aged 15 and older who have a Body Mass Index (BMI) of 30 and over—among the adult population in 17 Organisation for Economic Co-operation and Development (OECD) countries.[54] Though data are available for different years in different countries, it is noteworthy that obesity rates have increased over time in every single one of the reporting countries—in many cases very substantially. Indeed, among 11 of the 17 OECD countries for which trend data are available and reported in Figure 5 below, obesity rates increased by 50% or more.

In the United States, which has the highest obesity rate among all OECD countries, obesity rates more than doubled between 1976 and 2000—from 15% to 31% of the adult population. Between 1994 and 2000 alone, U.S. obesity rates increased by 35%—from 23% to 31% of the adult population. In the U.K., obesity rates more than tripled between 1980 and 2001—from 7% to 22%,—and in Australia they jumped by 160% between 1980 and 1999—from 8% to 21%. In Canada, obesity rates between 1994 and 2001 alone increased by 15%—from 13% to 15% of the population (Figure 5). As indicated below, the Canadian rates are continuing to climb—reaching 18% in 2005 based on self-reported data and 22.7% in 2004 based on directly measured data.

In 2008, the OECD released international obesity rates for 2005.[55] The OECD data are based on either self-reported or directly measured obesity data, depending on which were available from the reporting countries. As shown in Figure 6 below, in 2005 the U.S. still had the highest obesity rate among all OECD countries (based on directly measured BMI rates), with 32.2% of the population considered to be obese, while Japan had the lowest rate, with only 3.0% of the population considered to be obese (based on self-reported rates). Out of 30 OECD countries, Canada had the ninth highest obesity rate at 18.0% (based on self-reported rates). The United Kingdom (30%), Australia (21%), and New Zealand (20%) had higher rates than Canada, while most European countries had considerably lower obesity rates—with Switzerland, Norway, Austria, France, and Italy, for example, registering only about half the Canadian rate.

Investment, 2005; accessed September 2008; available from
http://www.who.int/chp/chronic_disease_report/full_report.pdf. p. 6.
[53] World Health Organization (WHO). _Obesity and Overweight_, accessed.
[54] Organisation for Economic Co-operation and Development (OECD). _Health at a Glance: OECD Indicators 2003: Chart 8. Increasing Obesity Rates among the Adult Population in OECD Countries_, 2003; accessed July 2008; available from
http://www.oecd.org/searchResult/0,3400,en_2649_34631_1_1_1_1_37407,00.html.
[55] Organisation for Economic Co-operation and Development (OECD). _OECD Factbook 2008: Economic, Environmental and Social Statistics: Obesity_, 2008; accessed July 2008; available from
http://puck.sourceoecd.org/vl=1680972/cl=12/nw=1/rpsv/factbook/110103.htm.

However, as discussed in Section 3.2 below, self-reported BMI rates have been shown to be underestimates that consistently yield lower results than directly measured BMI rates.[56] According to Canadian Community Health Survey (CCHS) data, the directly measured Canadian obesity rate in 2004 was actually 22.7%, which is considerably higher than the 18% self-reported 2005 rate indicated in Figure 6 below, and potentially places Canada in either fourth or fifth place among OECD countries based on directly measured data.[57]

In 2004, the directly measured obesity rate for Alberta was 25%. This is higher than the average Canadian rate (22.7%), and higher than the rates in British Columbia (19%), Quebec (22%), and Ontario (23%). Newfoundland and Labrador had the highest directly measured obesity rate in Canada at 34%.[58]

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[56] Shields, Margot, Sarah Connor Gorber, and Mark S. Tremblay. "Estimates of Obesity Based on Self-Report Versus Direct Measures," *Health Reports*, Statistics Canada, Catalogue no. 82-003-X, 2008, vol. 19, no. 2: 1-16. accessed June 2008; available from http://www.statcan.ca/english/freepub/82-003-XIE/2008002/article/10569-en.pdf.

[57] Shields, Margot, and Michael Tjepkema. "Regional Differences in Obesity," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol. 17, no. 3: 61-67.

[58] Ibid.

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Figure 5. Increasing obesity rates (BMI ≥30), among the adult population in OECD countries
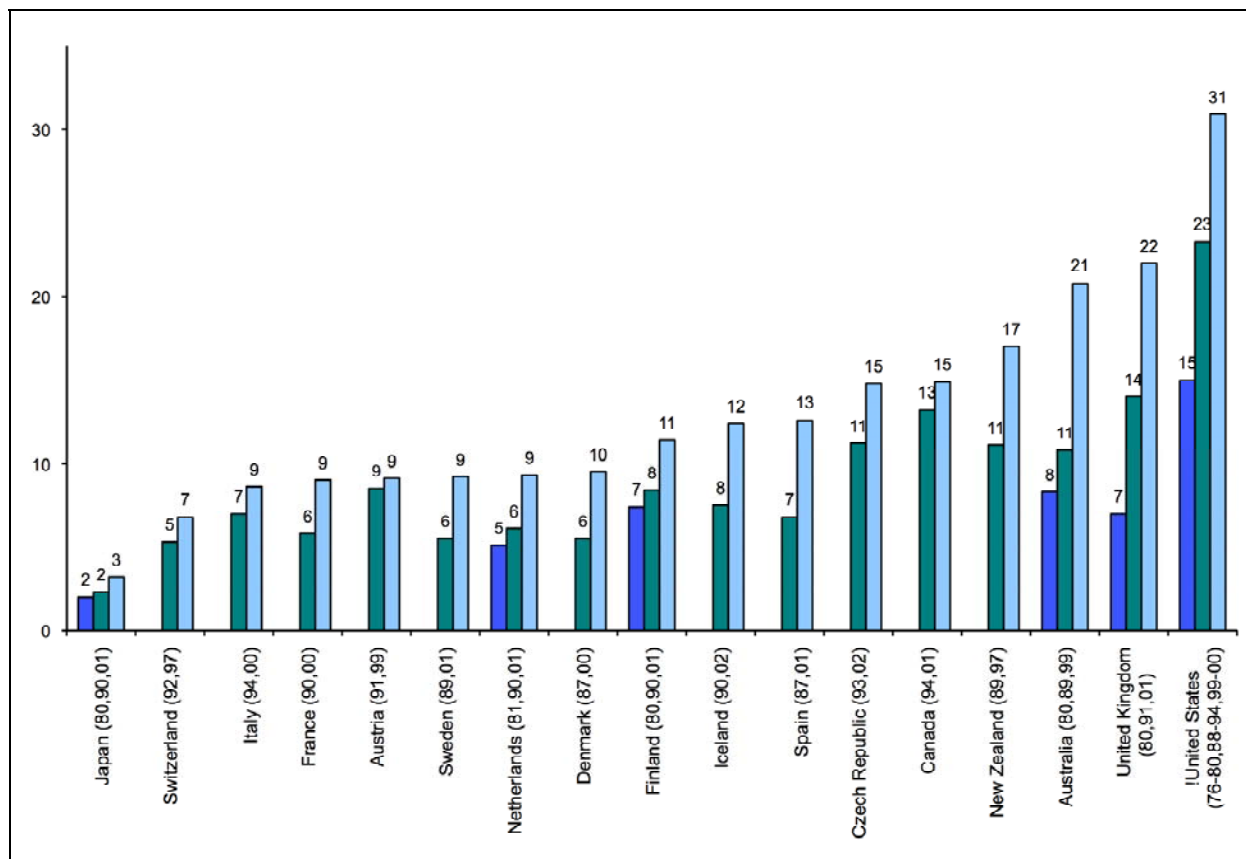
Source: Organisation for Economic Co-operation and Development (OECD). *Health at a Glance: OECD Indicators 2003: Chart 8. Increasing Obesity Rates among the Adult Population in OECD Countries*, 2003; accessed July 2008; available from http://www.oecd.org/searchResult/0,3400,en_2649_34631_1_1_1_1_37407,00.html.

Figure 6. International obesity rates (BMI ≥30), aged ≥15, 2005, percentage.
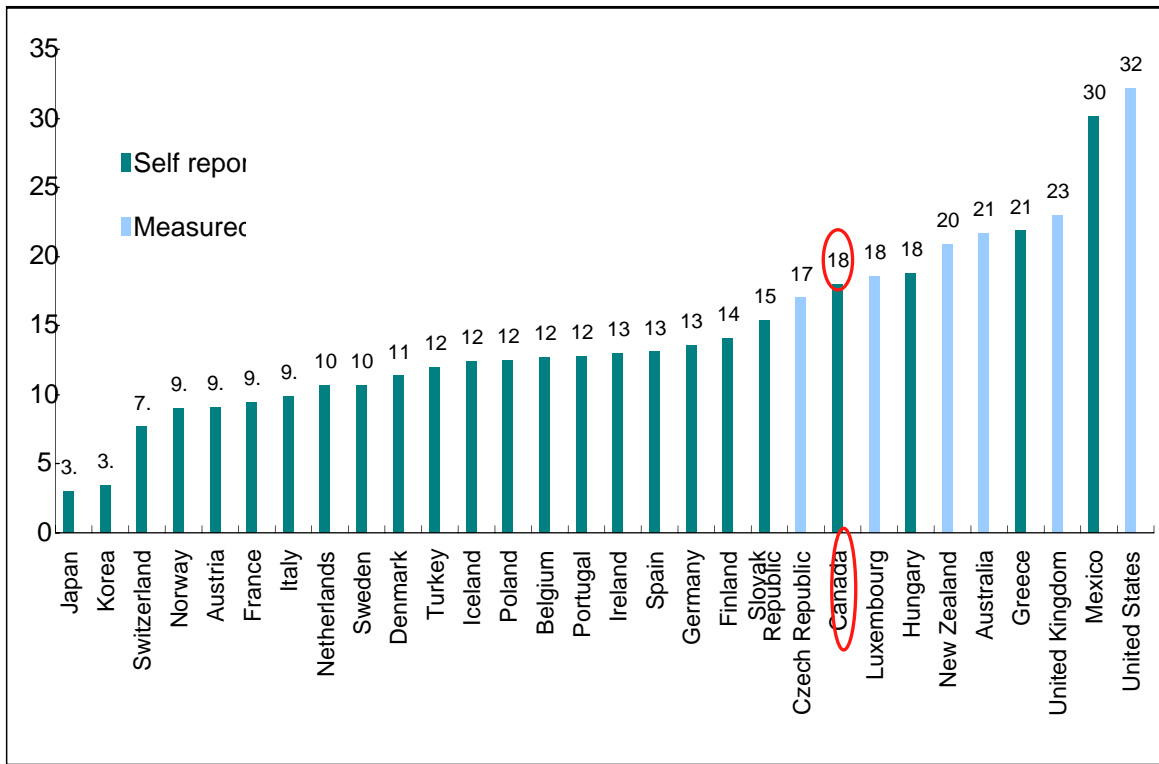
Source: Organisation for Economic Co-operation and Development (OECD). *OECD Factbook 2008: Economic, Environmental and Social Statistics: Obesity*, 2008; accessed July 2008; available from http://puck.sourceoecd.org/vl=1680972/cl=12/nw=1/rpsv/factbook/110103.htm.

The Council of The Obesity Society in the U.S. reports that "obesity causally contributes to ill health, functional impairment, reduced quality of life, serious disease, and greater mortality."[59] The Obesity Society considers obesity to be a disease itself. However, obesity is also widely considered to be a risk factor for a variety of serious chronic diseases, such as type 2 diabetes, cardiovascular diseases, osteoarthritis, and certain cancers, including colon, post-menopausal, and endometrial cancer, and is associated with increased risk of premature death.[60, 61,62]

Obesity is also responsible for increased costs to the health care system, employers, and other parties. Using 1997 data, C. Laird Birmingham of the University of British Columbia, et al. estimated the total direct cost of obesity, defined as BMI ≥27, in Canada to be over $1.8 billion, which amounted to 2.4% of the total health care expenditures for all diseases in Canada. In 2004, Peter Katzmarzyk and Ian Janssen of Queen's University updated these costs using 2001data and adding indirect costs.[63] They estimated the total direct and indirect costs associated with obesity (BMI ≥30) in Canada to be $4.3 billion in 2001 dollars, which included total direct costs of $1.6 billion and indirect costs of $2.7 billion. The total direct costs represented 2.2% of the total health care costs in Canada in 2001. Had Katzmarzyk and Janssen based their estimates on a BMI of ≥27, as Birmingham et al. did, instead of a BMI of ≥30, their estimates would have registered a substantial increase since 1997.

WHO reports that obesity accounts for between 2% and 7% of total health care costs in high-income countries, but notes that the "true costs are undoubtedly much greater as not all obesity-related conditions are included in the calculations."[64] In 2000, the total cost of obesity in the United States was estimated to be US$117 billion—US$61 billion for direct medical costs and US$56 billion for indirect costs.[65] In 2001, the direct and indirect costs of obesity in England were estimated to be over £2.6 billion in 1998 pounds.[66] In

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[59] Allison, David B., Morgan Downey, Richard L. Atkinson, Charles J. Billington, George A. Bray, Robert H. Eckel, Eric A. Finkelstein, Michael D. Jensen, and Angelo Tremblay. "Obesity as a Disease: A White Paper on Evidence and Arguments Commissioned by the Council of the Obesity Society," *Obesity*, 2008, vol. 16: 1161-1177. p. 1161.

[60] Ibid.

[61] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[62] Colman. *Cost of Obesity in Nova Scotia*, accessed.

[63] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[64] World Health Organization (WHO). *Obesity and Overweight*, accessed.

[65] Centers for Disease Control and Prevention (CDC). *Preventing Obesity and Chronic Diseases through Good Nutrition and Physical Activity*, United States Department of Health and Human Services, 2008; accessed September 2008; available from http://cdc.gov/nccdphp/publications/factsheets/Prevention/obesity.htm.

[66] National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, National Audit Office, 2001; accessed July 2008; available from http://www.nao.org.uk/publications/nao_reports/00-01/0001220.pdf.

2004, those costs were updated for 2002 to between £3.3 billion and £3.7 billion in 2002 pounds.[67]

Diabetes Australia estimated the total direct and indirect economic costs of obesity in Australia in 2005 to be AU$3.8 billion (AU$2005).[68] When the non-financial costs of the loss of wellbeing were included, which accounts for years of healthy life lost through disability and /or premature death, the cost increased to AU$21 billion. In 2008, Diabetes Australia used new data to update the total costs to AU$58 billion, including AU$8.3 billion in financial costs and AU$49.9 billion in the value of lost wellbeing (AU$2008). [69]

Although these obesity costing studies use somewhat different methodologies, health conditions, and costing categories in their analyses, and therefore are not strictly comparable, these few examples serve to illustrate the magnitude of the costs of obesity in high-income countries. Conversely, they indicate that very substantial cost savings can potentially be achieved through a reduction in obesity rates and consequent improvements in population health, which in turn will reduce demand on the health care system and improve economic productivity.

The main purpose of this present study is to estimate the direct health care costs and the indirect productivity losses attributable to obesity in Alberta in 2004/2005. This study focuses on the costs of obesity—defined as a BMI of ≥30—but also includes costs of overweight—defined as a BMI of ≥25—when data were available. Although overweight people often continue to gain weight and eventually become obese, the evidence linking overweight to chronic disease and premature mortality is not as strong as that for obesity.[70] A recent meta-analysis of studies linking overweight to mortality found "little evidence of increased risk of mortality in this group."[71]

Eric Finkelstein and Phaedra Corso,[72] as well as other analysts,[73] have noted a number of benefits that cost of illness studies, such as this cost of obesity report, can provide to policymakers, health care administrators, and other relevant parties. For example, obesity costing studies can clarify obesity as a societal, public health issue that is larger than an individual, behavioural issue. By focusing attention on the economic burden imposed by

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[67] House of Commons Health Committee (U.K.). *Obesity: Third Report of Session 2003-04, Annex 1: The Economic Costs of Obesity*, The United Kingdom Parliament, 2004; accessed July 2008; available from http://www.publications.parliament.uk/pa/cm200304/cmselect/cmhealth/23/23.pdf.

[68] Access Economics. *The Economic Costs of Obesity*, accessed.

[69] Access Economics. *The Growing Cost of Obesity in 2008: Three Years On*, Diabetes Australia, 2008; accessed September 2008; available from http://diabetesaustralia.com.au/PageFiles/7832/FULLREPORTGrowingCostOfObesity2008.pdf.

[70] Ogden, Cynthis L., Susan Z. Yanovski, Margaret D. Carroll, and Katherine M. Flegal. "The Epidemiology of Obesity," *Gastroenterology*, 2007, vol. 132: 2087-2102.

[71] McGee, D.L., and Diverse Populations Collaboration. "Body Mass Index and Mortality: A Meta-Analysis Based on Person-Level Data from Twenty-Six Observational Studies," *Annals of Epidemiology*, 2005, vol. 15: 87-97. p. 87.

[72] Finkelstein, and Corso. "Cost-of-Illness Analyses for Policy Making: A Cautionary Tale of Use and Misuse."

[73] Colman. *The Cost of Chronic Disease in Nova Scotia*, accessed.

obesity, and on the associations of obesity with chronic disease, other health conditions, and productivity losses, costing studies have the potential to mobilize societal interest and resources towards preventing obesity, and can provide motivation for governments to reduce obesity costs and to set priorities for prevention. In addition, costing studies can provide a crucial first step for future economic evaluations of the cost-effectiveness of interventions designed to reduce obesity levels in society.

This study mainly uses data from the 2004 and 2005 Canadian Community Health Surveys (CCHS) to estimate disease and obesity prevalence rates, and to determine the percentages of diseases that are attributable to obesity. Although 2004 is the most recent year for which directly measured obesity data are available for Canada and the provinces, 2004 data did not include all of the health conditions needed for the study. Therefore, 2005 data were also used where necessary. Although the 2005 data came from self-reported height and weight, which are not as accurate as directly measured rates, the data were adjusted to reflect directly measured rates based on a new method developed and tested specifically for use with 2005 CCHS data by Statistics Canada.[74] However, this method has not been tested for use with other years of CCHS. Therefore, although self-reported obesity rates are available through 2007 and are included in this report, unadjusted self-reported rates, as noted above, and their use would give a more biased and considerably less reliable estimate of costs.

The economic costs in this report are based on direct health care cost and indirect disability and mortality cost estimates of obesity-attributable diseases in Alberta that were provided by the Public Health Agency of Canada in its, as yet, unpublished *Economic Burden of Illness in Canada 2000* (EBIC 2000) report.[75] These are the latest comprehensive direct and indirect cost estimates available for Canada and the provinces at the present time. The 2000 cost estimates ($2000) were inflated to 2005 dollars in the final cost summaries.

A previous GPI Atlantic report on the cost of obesity in Alberta in 1997, prepared in 2000, estimated that obesity—defined as a BMI of >27—cost the Alberta health care system an estimated $320 million dollars annually ($1997), or nearly 6% of total direct health care costs in the province.[76] When productivity losses due to obesity—including costs attributable to premature death, absenteeism, and disability—were added, the total cost of obesity to the Alberta economy was estimated at between $620 million and $700 million a year, or 0.7%–0.8% of the province's Gross Domestic Product.

Since GPI Atlantic's initial work on the cost of obesity in 2000,[77] a number of new developments have occurred that allow far more accurate and methodologically rigorous analysis than was possible at the time. These new developments, which will be discussed

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[74] Gorber, Shields, Tremblay, and McDowell. "The Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity."

[75] *Economic Burden of Illness in Canada (EBIC), 2000.*

[76] Colman, Ronald. *Cost of Obesity in Alberta*, Halifax, N.S.: Genuine Progress Index Atlantic (GPI Atlantic), 2000.

[77] Colman. *Cost of Obesity in Nova Scotia*, accessed.

more fully in subsequent pages, include new obesity definitions, expanded obesity / disease associations, new directly measured obesity data from CCHS, new Environmental Burden of Illness in Canada (EBIC) costing data, , and substantial methodological improvements that include far-reaching critiques of earlier measurement and costing methodologies.

Most importantly, since 2000, obesity awareness has increased exponentially among both researchers and the general public. This is well illustrated by the substantial increase in new obesity-related literature. A search for obesity-related articles in only one database, Medline, showed that during the 1970s, 10,197 obesity-related articles were indexed in Medline, and during the 1980s, 11,800 were indexed. During the 1990s, the number rose to 17,754, which then increased by approximately 142% to 42,913 obesity-related articles published between 2000 and 2008 alone. Other databases and grey literature reveal similar increases.

This particular study should be considered as an ongoing work in progress, and all costs should be understood to be estimates rather than precise assessments. In December 2004, the Institute of Medicine (IOM) of the U.S. National Academy of Sciences held a professional workshop to discuss methodological issues concerning the estimation of the public health burden of lifestyle factors, including obesity, in preventable mortality and morbidity.[78] Speaking about mortality (in remarks that are equally relevant to morbidity), Julie Gerberding, Director of the U.S. Centers for Disease Control and Prevention (CDC), noted during the workshop:

> The biggest challenge is simple: there is not enough research to estimate, with the precision that we would like ultimately to achieve, the contributions of lifestyle factors to mortality, and to reduce their impact. Though much is known that can serve as the basis for public health action, gaps remain concerning how optimally to protect the public's health by measuring the burden of disease, determinants of risky behavior, interventions to change lifestyle, assessing the preventable fraction of deaths from these factors, the cost-effectiveness of interventions, and communications to maximize diffusion of effective interventions.[79]

Gerberding also noted that researchers have been working for four decades to understand the impact of smoking on morbidity and mortality, and that there are still gaps, "particularly regarding multiple risk factors interacting in various populations and at various stages of life."[80] In the beginning stages of this research, lung cancer was the only known co-morbidity of smoking, and it took many more years before heart disease was also found to be associated with tobacco use. However, despite the remaining gaps noted by Gerberding, much more is currently known about the effects of smoking on health than is known about the effects of obesity, diet, or physical activity levels on health,

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[78] Institute of Medicine. *Estimating the Contributions of Lifestyle-Related Factors to Preventable Death: A Workshop Summary*, accessed.
[79] Gerberding, Julie. In Ibid., accessed. p. 3.
[80] Ibid., accessed. p.3.

largely because tobacco research has a much longer history, while the bulk of research on the impacts of obesity, diet, and physical inactivity has occurred only within the last decade.

In addition to increasing the direct risks of chronic illness and premature mortality, obesity is also a symptom of deeper, underlying social trends that also have cost implications. Because of time and resource limitations, this study was not able to review potentional interventions or the many systemic forces driving the rise of obesity, which are areas for potential interventions. These areas have been identified as modern lifestyles, work environments, urban design and obesogenic (obesity-producing) environments, transportation systems, food production systems, technological developments, and economic growth itself.[81] Thus, sedentary rather than physically active lifestyles, and western diets that rely on energy-dense and "fast" foods rather than whole foods, are among the elements of modern lifestyles that are known to contribute to obesity.[82]

However, obesity is only one possible consequence of poor nutrition and a sedentary lifestyle, which are risk factors in their own right for many chronic illnesses. Therefore, systemic interventions designed to reduce obesity can potentially reduce other links to chronic disease as well. Indeed, interventions directed at underlying systemic factors may altogether be more effective in the long term than those targeting symptoms alone.

In the future, as more data become available, as methodologies become increasingly refined, and as research in these new areas becomes ever more focused, obesity cost estimates will hopefully become more precise over time. Since 2000 alone, as noted, substantial improvements in both data availability and estimation methods, along with significant advances in epidemiological research on obesity-disease associations, now allow far better cost estimates than were possible when GPI Atlantic produced its initial obesity cost studies in 2000. A decade from now, we can expect further significant improvements on what we can offer here.

The current study is divided into two parts, which are separated into two documents. In addition to the Executive Summary and Introduction, Part 1 (document 1) consists of a review of obesity definitions, obesity prevalence and trends in Alberta, and an extensive literature review, which determined the main chronic health conditions that were partially attributable to excess body weight in the population. The literature review found 22 health conditions, including 14 cancer sites, that had evidence of being partially attributable to obesity: type 2 diabetes, hypertension, coronary artery disease, cerebrovascular disease, osteoarthritis, gallbladder disease, asthma, depression, and cancers—colorectal cancer, postmenopausal breast cancer, endometrial (uterine) cancer, kidney cancer, esophageal cancer, ovarian cancer, prostate cancer, pancreatic cancer, non-Hodgkin's lymphoma, multiple myeloma, leukemia, liver cancer, bladder cancer,

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[81] World Health Organization (WHO). *Obesity and Overweight*, accessed.
[82] Ibid., accessed.

and stomach cancer. The costs of each of these health conditions in Alberta was first estimated, and then a portion of the cost was attributed to obesity and overweight.

In addition to the literature review, Chapter 4 in Part 1 also includes information on the physical pathways from obesity to chronic disease and mortality, methodological issues in attributing a portion of the health conditions to obesity, and statistics on the prevalence of the health conditions in Alberta.

In Part 1 there are also two sections concerning Alberta's children and youth and Aboriginal populations. The first section reports the prevalence and trends of obesity in these populations, and the second section discusses the health impacts related to obesity in children, youth, and Aboriginal people. However, because the percentages of the health conditions that are attributable to overweight and obesity in these populations have not been established, this report has not been able to include children and youth or the Aboriginal population in the cost of obesity estimates.

Part 2 of this report, which is in a separate document, estimates the economic costs of obesity in Alberta for direct health care costs, indirect short-term and long-term disability costs, and the costs of mortality of the above 22 health conditions that are attributable to overweight and obesity in Alberta in 2000, with the final costs in the summary chapter inflated to $2005 dollars.

2. Definitions of obesity

2.1 Adult Body Mass Index classification systems

Obesity is defined as an accumulation of excess fat in the body.[83] Cynthia Ogden et al. of the U.S. National Center for Health Statistics note: "The human body contains essential lipids and also nonessential lipids in the form of triglycerides (triacylglycerols) stored in adipose tissue cells known as *adipocytes.*"[84] However, this adiposity, or body fat, is difficult to measure directly, and, therefore, obesity is often defined as excess body weight rather than excess fat. Because of differences in body composition, women generally have a higher percentage of body fat than do men, and older individuals tend to have a higher percentage of body fat than do younger people with the same Body Mass Index (BMI).[85]

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[83] Access Economics. *The Economic Costs of Obesity*, accessed.
[84] Ogden, Yanovski, Carroll, and Flegal. "The Epidemiology of Obesity."
[85] Ibid.

International and Canadian definitions of obesity define adult obesity for both genders, aged ≥18, in relation to Body Mass Index (BMI), which is calculated as weight in kilograms divided by height in metres squared:[86]

$$\text{Body Mass Index} = \text{weight (kilograms)} / \text{height (metres}^2)$$

Between 1988 and 2003, for adults aged 20 to 64, Health Canada considered a BMI of 20–24.9 as "acceptable weight", 25–26.9 as "some excess weight", and 27 or higher as "overweight".[87] There was no separate Health Canada classification for "obesity." In the first comprehensive estimation of obesity costs for Canada published in the *Canadian Medical Association Journal* in 1999, however, Birmingham et al. do use the term "obesity" for a BMI of ≥27. In 2003, based on new research on the relationship between BMI and the risk of morbidity and mortality, and on emerging international standards, Health Canada updated the guidelines for body weight classifications for (non-pregnant or lactating) adults aged 18 years and over, and for the first time included a separate category for "obesity".[88]

The new guidelines, which describe a body weight classification system that can be used to identify health risks associated with body weight in individuals and populations, are in accord with the World Health Organization (WHO) recommendations that were released in 2000 and have been widely adopted internationally.[89] As shown in Table 2 below, the new guidelines identify "underweight" as having a BMI of under 18.5, "normal weight" as having a BMI of 18.5 to 24.9 kg/m^2, "overweight" as having a BMI of 25.0 to 29.9 kg/m^2, and "obese" as having a BMI of 30 kg/m^2 or greater. The guidelines further divide "obese" into three levels: BMI 30.0 to 34.9 kg/m^2 (obese-class 1); 35.0 to 39.9 kg/m^2 (obese-class 2); 40 kg/m^2 or greater (obese - class 3).[90] In addition, a level of abdominal fat measurement, which is increasingly being used in surveys and studies, was changed from a waist to hip ratio to a waist circumference measure.

Relative health risk levels, as compared with individuals having a normal weight, are associated with the different BMI levels: normal weight is associated with the least health risk; underweight and overweight is associated with increased health risk; obese class 1 is associated with high health risk; obese class 2 is associated with very high health risk; and obese class 3 is associated with extremely high health risk.[91] Peter Katzmarzyk and Caitlin Mason of Queen's University note that obesity class guides treatment options, and that the "use of more aggressive approaches to weight loss (e.g., pharmacotherapy or bariatric surgery) are generally reserved for people with more extreme obesity (class 2 or 3) and those with additional risk factors."[92]

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[86] Statistics Canada. *Health Indicators: Definitions and Data Sources: Body Mass Index (BMI-Canadian Standard)*, accessed.
[87] Ibid., accessed.
[88] Health Canada. *Canadian Guidelines for Body Weight Classification in Adults*, accessed.
[89] Ibid., accessed.
[90] Statistics Canada. *Health Indicators*, accessed. p. 21.
[91] Ibid., accessed. p. 21.
[92] Katzmarzyk, and Mason. "Prevalence of Class I, II, and III Obesity in Canada." p. 156.

According to Health Canada, these health risks may underestimate or overestimate health risks in specific groups:

- Young adults (aged 18 and over) who have not reached full growth;
- Adults who have a naturally lean body build;
- Highly muscular adults (— BMI does not distinguish fat from fat-free mass such as muscle and bone[93]);
- Adults over age 65 (— for this group the normal range may begin slightly above a BMI of 18.5 and extend into the overweight range);
- Certain ethnic and racial groups (— BMI classifications are based on Caucasian body types, which are different than Asian and Aboriginal body types). For Asian groups, Inuit people, and other Aboriginal populations, more research is needed to determine whether or not current BMI classifications are accurate.[94] Indeed, such new research on appropriate BMI classifications for different Asian body types led the Government of India in November, 2008, to adopt the following classifications: BMI 23–25 for overweight, and BMI ≥25 for obese.[95]

However, according to Health Canada, for all of these groups, the classification system developed for Canadian adults in general is considered to be appropriate for population measurement purposes within Canada.[96]

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[93] Burkhauser, Richard V., and John Cawley. "Beyond BMI: The Value of More Accurate Measures of Fatness and Obesity in Social Science Research," *Journal of Health Economics*, 2008, vol. 27: 519-529.
[94] Health Canada. *Canadian Guidelines for Body Weight Classification in Adults*, accessed.
[95] Mudur, G.S. *Overnight, Many Overweight Indian Figures Revised to Tone up Flab Fight*, Calcutta, India, *The Telegraph*, 2008; accessed February 2009; available from
http://www.telegraphindia.com/1081126/jsp/frontpage/story_10165735.jsp.
[96] Health Canada. *Canadian Guidelines for Body Weight Classification in Adults*, accessed.

Table 2. Differences in adult BMI classification systems, 1988, 2003

Classification	1988 Guidelines	2003 Guidelines	Relative risk of health problems
Age limits	20 – 64 years	18 years and over with no upper limit	—
Labels for BMI categories	Underweight Acceptable weight Some excess weight Overweight	Underweight Normal weight Overweight Obese	—
Underweight	< 20.0	< 18.5	Increased
Acceptable / Normal weight	20.0 – 24.9	18.5 – 24.9	Least
Some excess weight / Overweight	25.0 – 27.0	25.0 – 29.9	Increased
Overweight / Obese	≥27.0	≥30.0	High
Sub-categories of obese:			
Class 1	—	30.0 – 34.9	High
Class 2	—	35.0 – 39.9	Very high
Class 3	—	≥40.0	Extremely high
Level of abdominal fat	Waist to hip ratio	Waist circumference (WC)	Increased or high risk above cut-off points: Men: WC ≥102 cm (40"); Women: WC ≥88 cm (35")

Source: Adapted from: Health Canada. *Canadian Guidelines for Body Weight Classification in Adults*, 2003; accessed June 2008; available from http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/nutrition/weight_book-livres_des_poids-eng.pdf.

Figure 7 below shows the relationships between adult BMI and weight in pounds and height in feet and inches.[97] Although the BMI does not distinguish between genders, Figure 7 indicates that a 5'5" woman would be considered obese if she weighed at least 180 pounds, and a 5'10" man would be considered obese if he weighed at least 210 pounds.

Figure 7. Adult Body Mass Index, height (feet and inches) and weight (lbs)
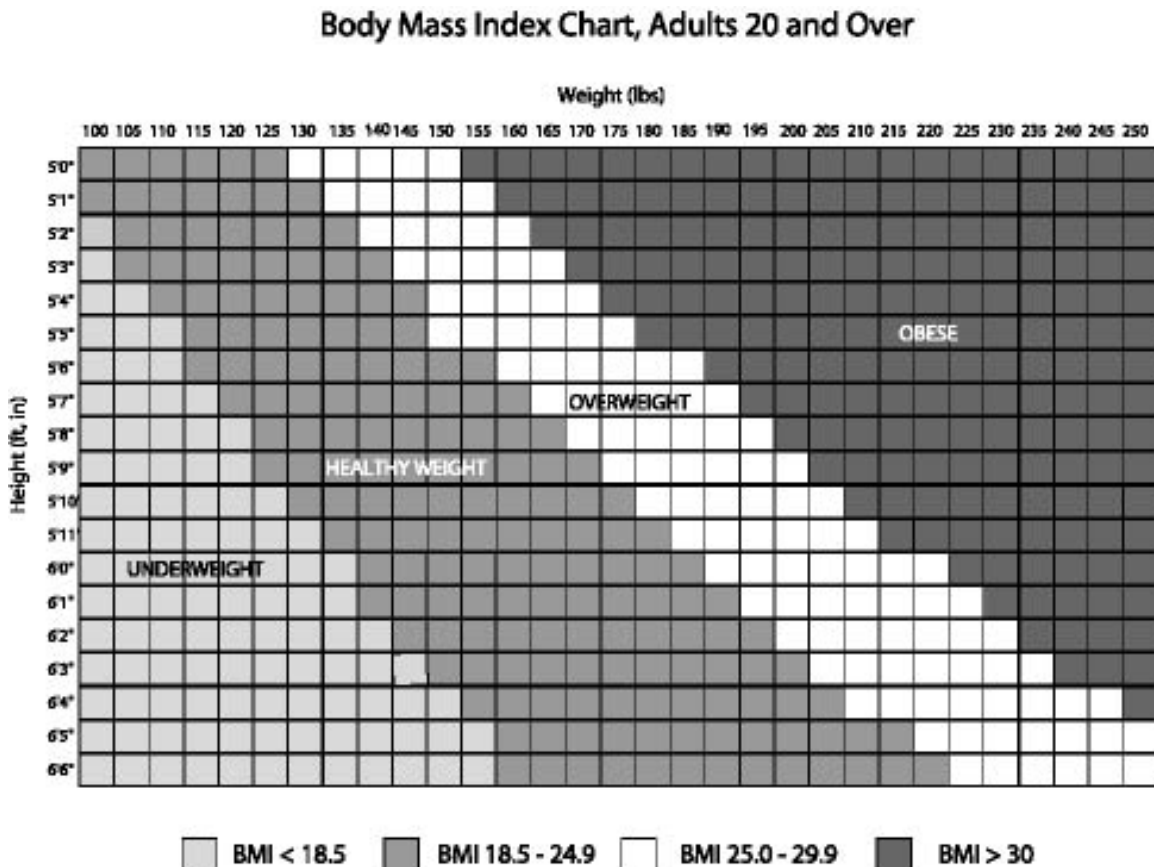
Body Mass Index Chart, Adults 20 and Over

Source: National Cancer Institute (U.S.). *Fact Sheet: Obesity and Cancer: Questions and Answers*, 2004; accessed July 2008; available from http://www.cancer.gov/cancertopics/factsheet/Risk/obesity.

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[97] National Cancer Institute (U.S.). *Fact Sheet: Obesity and Cancer: Questions and Answers*, 2004; accessed July 2008; available from http://www.cancer.gov/cancertopics/factsheet/Risk/obesity.

2.2 Children and youth Body Mass Index

Body Mass Index for children and youth, aged 2 to 17, is calculated in the same way as for adults—weight in kilograms divided by height in metres squared. However, because children and youth are still maturing, the classifications of BMI are different from those used to classify adults. Statistics Canada now uses the international-standard International Obesity Task Force (IOTF) criteria to measure overweight and obesity for children and youth.[98] The criteria were originally developed by T.J. Cole, et al. in 2000, based on data from the U.S., Great Britain, the Netherlands, Brazil, Hong Kong, and Singapore.[99] These criteria for children and youth classify BMI as "neither overweight nor obese", "overweight", or "obese", based on sex- and age-specific BMI thresholds as defined by the IOTF.

The BMI cut-points are lower than those for adults, rise by increments with every 6 months of age, are aligned with the adult obesity threshold of 30 kg/m^2 by age 18, and are not defined by the IOTF in terms of increased health risk, because it is not yet clear which BMI levels are associated with health risks for children and youth.[100] For example, the cut-point for overweight in 7-year old boys is a BMI of 17.92, and for obesity in 13-year old girls it is 27.76. As can be seen in Table 3 below, a 7-year old boy with a BMI of 18.2 (height – 119 cm or 3'11", and weight – 25.8 kg or 56.9 lbs) would be considered to be overweight, and a 13-year old girl with a BMI of 28.5 (height – 160 cm or 5'3", and weight – 73 kg or 161 lbs) would be considered to be obese.[101]

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[98] Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol. 17, no. 3: 27-42.

[99] Cole, T.J., M.C. Bellizzi, K.M. Flegal, and W.H. Dietz. "Establishing a Standard Definition for Child Overweight and Obesity Worldwide: International Survey," *British Medical Journal*, 2000, vol. 320: 1240-1243.

[100] Shields. "Overweight and Obesity among Children and Youth."

[101] Ibid.

Table 3. BMI classification for children and youth

Age (years)	Overweight cut-points BMI greater than or equal to		Obese cut-points BMI greater than or equal to	
	Boys	Girls	Boys	Girls
2	18.41	18.02	20.09	19.81
2.5	18.13	17.76	19.80	19.55
3	17.89	17.56	19.57	19.36
3.5	17.69	17.40	19.39	19.23
4	17.55	17.28	19.29	19.15
4.5	17.47	17.19	19.26	19.12
5	17.42	17.15	19.30	19.17
5.5	17.45	17.20	19.47	19.34
6	17.55	17.34	19.78	19.65
6.5	17.71	17.53	20.23	20.08
7	17.92	17.75	20.63	20.51
7.5	18.16	18.03	21.09	21.01
8	18.44	18.35	21.60	21.57
8.5	18.76	18.69	22.17	22.18
9	19.10	19.07	22.77	22.81
9.5	19.46	19.45	23.39	23.46
10	19.84	19.86	24.00	24.11
10.5	20.20	20.29	24.57	24.77
11	20.55	20.74	25.10	25.42
11.5	20.89	21.20	25.58	26.05
12	21.22	21.68	26.02	26.67
12.5	21.56	22.14	26.43	27.24
13	21.91	22.58	26.84	27.76
13.5	22.27	22.98	27.25	28.20
14	22.62	23.34	27.63	28.57
14.5	22.96	23.66	27.98	28.87
15	23.29	23.94	28.30	29.11
15.5	23.60	24.17	28.60	29.29
16	23.90	23.37	28.88	29.43
16.5	24.19	24.54	29.14	29.56
17	24.46	24.70	29.41	29.69
17.5	24.73	24.85	29.70	29.84
18+	25.00	25.00	30.00	30.00

Source: Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol. 17, no. 3: 27-42.

3. Obesity prevalence and trends in Alberta and Canada

3.1 Data sources for obesity prevalence and trends

3.1.1 1970–1994 surveys that directly measured BMI

Canadian data for directly measured BMI are available from six cross-sectional national health surveys for six reference periods: Nutrition Canada Survey (1970/72), Canada Health Survey (1978/79), Canada Fitness Survey (1981), Campbell's Survey on Well-being (1988), Canadian Heart Health Surveys (1986–95) including the Alberta Heart Health Survey conducted in 1990, and Canadian Community Health Survey, Cycle 2.2 (2004).[102] Although the age groups surveyed in these surveys do not always correspond, Canadian researchers Luo et al. have used these surveys to determine obesity trends in Canada that are based on measured data making appropriate adjustments for age (see section 3.3 below). With the exception of the Nutrition Canada Survey, the territories are not included in this data collection, nor are residents of some remote areas, persons living on Indian reserves or Crown lands, full-time members of the Canadian military forces, and persons living in institutions. However, it is estimated that 98% of the Canadian population is included in the surveys.[103]

The Nutrition Canada Survey was the first comprehensive cross-sectional appraisal of the diets of 13,000 Canadians of all ages and of the prevalence of nutritional diseases.[104] Data, which included measured height and weight, were collected between 1970 and 1972. In addition to collecting information on a representative sample of the Canadian population, the survey also included First Nations people living on reservations and Crown lands, Inuit living in remote settlements, and residents of the Northwest and

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[102] Kendall, Ora, Tammy Lipskie, and Shauna MacEachern. "Canadian Health Surveys, 1950-1997," *Chronic Diseases in Canada*, Public Health Agency of Canada, 1997, vol. 18, no. 2: 1-20. accessed July 2008; available from http://www.phac-aspc.gc.ca/publicat/cdic-mcc/18-2/b_e.html.; and Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."
[103] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."
[104] Nutrition Committee of the Canadian Paediatric Society. "The Nutrition Canada Survey: A Review," *Canadian Medical Association Journal*, 1976, vol. 115: 775-777.

Yukon Territories.[105] Shields and Tjepkema note that this survey had a low response rate, and they did not use it in their analysis of obesity trends in Canada.[106]

Between 1978 and 1979, the comprehensive Canada Health Survey collected data on lifestyle habits and undertook actual physical measures in one third of the interviews. The Canada Fitness Survey was conducted in 1981, and, in 1988, the Campbell's Survey on Well-being surveyed a sub-sample of the earlier 1981 survey. Shields and Tjepkema suggest that these two surveys are not directly comparable with the others because they focused on fitness rather than health per se.[107] Between 1986 and 1992, the Canadian Heart Health Surveys surveyed respondents aged 18 to 74 in all provinces.[108] Measured height and weight were collected for 19,841 respondents nationwide. As part of these surveys, the Alberta Heart Health Survey was conducted in 1990.[109]

3.1.2 Canadian Community Health Survey

Between 1995 and 2003, there were no nationwide health surveys conducted in Canada that directly measured height and weight. In 2004, the Canadian Community Health Survey (CCHS), cycle 2.2, which focused on nutrition, was the next survey to measure directly the height and weight of respondents.[110] The CCHS began in 2000-01 as a series of cross-sectional surveys that consist of two alternating annual cycles—the first ".1" cycle (i.e., 2001, 2003, 2005, and 2007)[111] collects data on the general health of over 130,000 respondents, aged 12 and over, at the health region level,[112] and the second, ".2" cycle (2002, 2004, and 2006), which is conducted the following year, collects information on specific topics from a smaller sub-sample of 30,000 respondents, with that

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[105] Sabry, Z.I., E. Campbell, J.A. Campbell, and D.F. Bray. *Nutrition Canada National Survey; 1970-1972*, (Original publishers: Nutrition Canada and Department of National Health and Welfare, Food and Drug Directorate), Public Archives of Canada, 2005.

[106] Shields, Margot, and Michael Tjepkema. "Trends in Adult Obesity," *Health Reports*, Statistics Canada, Catalogue 82-003, 2006, vol. 17, no. 3: 53-59.

[107] Ibid.

[108] Ibid.

[109] Kendall, Lipskie, and MacEachern. "Canadian Health Surveys, 1950-1997."

[110] Verret, Francois. *Methodological Challenges in Analyzing Nutrition Data from the Canadian Community Health Survey - Nutrition*, Statistics Canada, Proceedings of Statistics Canada's Symposium 2006: Methodological Issues in Measuring Population Health, 2006; accessed July 2008; available from http://www.statcan.ca/english/freepub/11-522-XIE/2006001/article/10394-en.pdf.

[111] The 2001 CCHS data were collected from October 2000 to October 2001, while the other cycle 1 data were collected between January and December of the specific year.

[112] In part, the CCHS cycles include information on breastfeeding, physical activity, self-perceived physical health, disease prevalence including prevalence of heart disease, cancers, diabetes, respiratory diseases (asthma, chronic bronchitis, emphysema, or Chronic Obstructive Pulmonary Disease – COPD), fibromyalgia, chronic fatigue syndrome, and chemical sensitivities, BMI, injuries, health service use, self-perceived mental health (cycle 1.2), and optional modules on fruit and vegetable consumption. Statistics Canada. *Canadian Community Health Survey (CCHS)*, 2007; accessed May 2008; available from http://www.statcan.ca/cgi-bin/imdb/p2SV.pl?Function=getSurvey&SDDS=3226&lang=en&db=IMDB&dbg=f&adm=8&dis=2.

sample size designed to provide valid data at the national and provincial levels only.[113] The cycle 2 surveys are not designed to be comparable with other cycles.[114]

All of the first cycle Canadian Community Health Surveys ask respondents to report their height and weight. However, only Cycle 2.2—Nutrition, which was conducted between January and December 2004, directly measured the height and weight of all respondents aged 2 and older. Prior to taking direct measures in that survey, 10% of respondents aged 18 and older were asked to report their height and weight.[115] In addition, 24-hour dietary recall data were collected from 10,000 respondents, and the survey included a food security module.[116] In Alberta the sample size for this survey was 3,116 respondents.[117] Parents provided information for children under the age of 6 and assisted children aged 6–11 in the interview. Individuals aged 12 and older provided their own information.

Also, in order to further quantify bias resulting from self-reported BMI data, the 2005 CCHS—conducted between January and December of that year—directly measured the height and weight of a small sub-sample of 4,567 respondents (out of a total of 132,947) aged 12 and older, who reported their own height and weight prior to being measured.[118] In a personal communication with the authors of this present report, Margot Shields of Statistics Canada suggested that the 2004 CCHS data should be used to estimate costs of obesity—especially at the provincial level—rather than the 2005 sample, which was much smaller and did not include children younger than 12.[119]

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[113] Thomas, Steven. *Combining Cycles of the Canadian Community Health Survey*, Statistics Canada, Proceedings of Statistics Canada Symposium 2006: Methodological Issues in Measuring Population Health 2006; accessed July 2008; available from http://www.statcan.ca/english/freepub/11-522-XIE/2006001/article/10411-en.pdf.

[114] Ibid., accessed.

[115] Statistics Canada. *Canadian Community Health Survey - Nutrition*, 2006; accessed May 2008; available from http://www.statcan.ca/english/sdds/5049.htm. According to Statistics Canada: "All respondents aged 2 and older were asked for 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 interviewers. The main reasons for non-response include refusal (11%), respondent not available (6%), respondent too tall for the interviewer to measure (5%), equipment problems (5%) and interview conducted over the phone (4%). In order to minimize the potential for non-response bias a special weight was created to be used with the measured height and weight information and the subsequent calculation of measured Body Mass Index."

[116] Health Canada. *Canadian Community Health Survey Cycle 2.2, Nutrition (2004). Income-Related Household Food Security in Canada*, Office of Nutrition Policy and Promotion, Health Products and Food Branch, 2007; accessed March 2008; available from http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/surveill/income_food_sec-sec_alim_e.pdf.

[117] Statistics Canada. *Canadian Community Health Survey - Nutrition*, accessed.

[118] Shields, Gorber, and Tremblay. "Estimates of Obesity Based on Self-Report Versus Direct Measures."

[119] Shields, Margot, personal communication with Karen Hayward, August 21, 2008.

3.1.3 Other health surveys with self-reported BMI

In addition to the CCHS, two other Canadian national health surveys have included self-reported BMI—the National Population Health Survey (NPHS),[120] and the National Longitudinal Survey of Children and Youth (NLSCY).[121] The NPHS began in 1994/95 with both a cross-sectional and longitudinal component. The cross-sectional NPHS component—conducted biennially in 1994/95, 1996/97, and 1998/99—was replaced in 2000/01 by the CCHS, but the longitudinal NPHS component is scheduled to continue until 2014. About 17,000 persons of all ages from all provinces have been surveyed in the longitudinal NPHS every two years since the survey began. The NPHS has both a household component and an institutional component that surveys residents of health institutions such as nursing homes.[122]

The National Longitudinal Survey of Children and Youth (NLSCY) is jointly conducted by Human Resources and Social Development Canada (HRSDC) and Statistics Canada. It is a comprehensive survey that follows the development and wellbeing of children, who were aged 0–11 in 1994, from birth to early adulthood. New children are included in the sample each year, and the children are assessed biannually until age 25. All samples are drawn from the Labour Force Survey's (LFS) sample of respondent households in all provinces. The sample for the latest NLSCY cycle (Cycle 7) was comprised of 37,655 children and youth aged from 0–9 and from 12–23 respectively. Much of the information on children in the NLSCY is collected from parents on behalf of their children by means of a household interview.[123]

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[120] Statistics Canada. *National Population Health Survey (NPHS)*, 2008; accessed July 2008; available from http://www.statcan.ca/cgi-bin/imdb/p2SV.pl?Function=getSurvey&SDDS=3225&lang=en&db=IMDB&dbg=f&adm=8&dis=2.

[121] Statistics Canada. *National Longitudinal Survey of Children and Youth*, 2007; accessed June 2008; available from http://www.statcan.ca/cgi-bin/imdb/p2SV.pl?Function=getSurvey&SDDS=4450&lang=en&db=imdb&dbg=f&adm=8&dis=2.

[122] The NPHS information includes self-perceptions of health, self-reported height and weight, chronic conditions, injuries, repetitive strains, depression, smoking, alcohol consumption, physical activity, consultations with medical professionals, use of medications, and use of alternative medicines. It also includes information on vision trouble, hearing problems, speech trouble, mobility trouble, dexterity trouble, emotional problems, cognition, and activities prevented by pain — with the combination of these eight variables allowing construction of a Health Utility Index. Demographic and economic information collected in the NPHS includes age, gender, education, ethnicity, household income, and labour force status.

[123] Additional information is collected using questionnaires completed by the child's teacher and principal. Children aged 10 and older complete a separate written questionnaire in the home. The survey covers a comprehensive range of topics including family and household composition; relationships; a socio-demographic profile of parents and children; family functioning; neighbourhood; child education, communication, development, behaviour, and custody; child care use; youth education, income, health, height and weight, activities, support, and family situation; family education; ethnic diversity and immigration; family income; labour market activities; and religion.

3.1.4 New Canadian Health Measures Survey

In response to the need for more accurate data, Statistics Canada, Health Canada, and the Public Health Agency of Canada have developed a new survey—the Canadian Health Measures Survey—that directly measures physical health, including BMI, blood pressure, heart rate, lung functioning, and cardiovascular fitness, among other factors.[124] Data collection is currently taking place between 2007 and 2009, and will sample approximately 5,000 people aged 6–79 years.

The survey involves two steps—first, an interviewer administers a household questionnaire in the respondent's home, and second, the respondents are asked to visit a mobile clinic where trained health professionals take physical measurements. Blood and urine specimens collected at the clinic are stored and analysed in laboratories for indicators of general health, chronic diseases, exposure to infectious diseases, and exposure to environmental contaminants. The respondents are also asked to wear a physical activity monitor for a week so that their activity levels can be measured. When the results are released in 2010, they should provide important new data concerning the health of Canadians. However, the sample size is too small to provide reliable data on BMI and disease prevalence for obese class 1–3 and age group categories.

3.2 Self-reported versus directly measured BMI

3.2.1 Collection method bias in adult BMI data

Where possible, this report relies on directly measured, rather than self-reported, BMI data to estimate the costs of obesity, but still reports self-reported data where relevant—particularly to indicate trends over time. It also relies on self-reported BMI data from 2005, but these data have been adjusted to reflect directly measured data by using a new method developed by Statistics Canada, which has only been tested for use with 2005 CCHS data.[125]

Although the latest self-reported data are available for 2007, as discussed above, the latest directly measured data are only available for 2004 (with a sub-sample for 2005). Evidence has shown that directly measured BMI data are considerably more accurate than self-reported data, which tend to be biased downward.[126] This bias is not always

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[124] Tremblay, Mark, and Sarah Connor Gorber. "Canadian Health Measures Survey - Brief Overview," *Canadian Journal of Public Health*, 2007, vol. 98, no. 6: 453-456.
[125] Gorber, Shields, Tremblay, and McDowell. "The Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity."
[126] Romero-Corral, Somers, Sierra-Johnson, Thomas, Collazo-Clavell, Korinek, Allison, Batsis, Sert-Kuniyoshi, and Lopez-Jimenez. "Accuracy of Body Mass Index in Diagnosing Obesity in the Adult General Population."

gender-specific, but in general men tend to overestimate their height, and women tend to underestimate their weight—perhaps, as S. Connor Gorber et al. of Statistics Canada note, because of social desirability and the stigma that can be associated with obesity.[127] As well, overweight and obese individuals tend to misrepresent their height and weight more often than do those with normal weight. In general, therefore, self-reports tend to underestimate BMI, which results in fewer people being classified as obese than is actually the case. In addition, the association found between obesity and morbidity tends to differ depending on the data collection method.[128]

Margot Shields et al. of Statistics Canada note that this bias in BMI reports is a relatively new phenomenon.[129] For those aged 20 to 69 years, directly measured average weight data from the 1981Canadian Fitness Survey compared with self-reported data from the 1985 Health Promotion Survey showed no difference for men, while for women the average weight based on measured data was actually 0.6 kg lower than that based on self-reported data.

According to Canadian researchers Connor Gorber et al., who conducted a systematic review to examine the relationship between self-reported and measured BMI data in international studies, no overall effect size between the two measures can presently be estimated:

> In spite of the trend in the present review of weight and BMI being underestimated and height being overestimated, there were too many gaps in the data to undertake a quantitative analysis or to get a comprehensive understanding of the relationship between self-report and direct measures…. With more complete data, it may be possible to develop correction factors that could be applied to self-reported data when direct measurement is not feasible.[130]

Subsequently, as noted and as described below, Gorber et al. have developed a new method to correct the self-reported data, but this method has only been tested for use with 2005 CCHS data.[131]

Shields, et al. recently compared self-reported and measured data from the 2005 sub-sample of the Canadian Community Health Survey (CCHS) that was conducted to

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[127] Gorber, S. C., M. Tremblay, D. Moher, and B. Gorber. "A Comparison of Direct Vs. Self-Report Measures for Assessing Height, Weight and Body Mass Index: A Systematic Review," *Obesity Reviews*, 2007, vol. 8: 307-326.

[128] Shields, Margot, Sarah Connor Gorber, and Mark S. Tremblay. "Effects of Measurement on Obesity and Morbidity," *Health Reports*, Statistics Canada, Catalogue no. 82-003-X, 2008, vol. 19, no. 2: 1-8. accessed June 2008; available from http://www.statcan.ca/english/freepub/82-003-XIE/2008002/article/10564-en.pdf.

[129] Shields, Gorber, and Tremblay. "Estimates of Obesity Based on Self-Report Versus Direct Measures."

[130] Connor Gorber, S., M. Tremblay, D. Moher, and B. Gorber. "A Comparison of Direct Vs. Self-Report Measures for Assessing Height, Weight and Body Mass Index: A Systematic Review," *Obesity Reviews*, 2007, vol. 8: 307-326. pp. 321, 323.

[131] Gorber, Shields, Tremblay, and McDowell. "The Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity."

facilitate comparison studies.[132] In a sub-sample of 4,535 survey respondents aged 12 and older, CCHS first collected self-reported height and weight measures in face-to-face interviews,[133] and then directly measured the height and weight of the same respondents.[134] Differences in the prevalence of obesity between the self-reported and measured collection methods from the 2005 CCHS are shown in Table 4 below. Shields et al. found that the prevalence of obesity was 7.4 percentage points higher using the measured data than using the self-reported data (22.6% vs. 15.2%) — 8.8 percentage points higher for males (24.2% vs. 15.4%), and 6 percentage points higher for females (21.0% vs. 15.0%).

Table 4. Prevalence of obesity (BMI ≥30), by collection method and gender, aged ≥12, Canada, excluding territories, CCHS, 2005

Prevalence of obesity	Sample size	Collection method		Percentage point difference (Measured minus self-reported)
		Measured (%)	Self-reported (%)	
Both genders	4,535	22.6	15.2	7.4
Males	2,113	24.2	15.4	8.8
Females	2,422	21.0	15.0	6.0

Notes: CCHS – Canadian Community Health Survey
Source: Shields, Margot, Sarah Connor Gorber, and Mark S. Tremblay. "Estimates of Obesity Based on Self-Report Versus Direct Measures," *Health Reports*, Statistics Canada, Catalogue no. 82-003-X, 2008, vol. 19, no. 2: 1-16. Accessed June 2008; available from http://www.statcan.ca/english/freepub/82-003-XIE/2008002/article/10569-en.pdf.

In addition, Figure 8 below shows the differences in obesity rates by gender and age group using directly measured and self-reported collection methods. The directly measured data showed significantly higher obesity rates for all age groups, with the 65 and older age group showing the largest disparity:—The difference was 15 percentage points higher for men aged ≥65 in the measured data compared to the self-reported data (31% vs. 16%), and 13 percentage points higher for women aged ≥65 (28% vs. 15%). It has been speculated that this is because older people often shrink in height but still report their height at a younger age—thus significantly overestimating their current height, which in turn leads to substantial underestimates of BMI and obesity rates.

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[132] Shields, personal communication, with Karen Hayward, August 21, 2008.
[133] Face-to-face interviews generally produce more accurate data than do telephone interviews.
[134] Shields, Gorber, and Tremblay. "Estimates of Obesity Based on Self-Report Versus Direct Measures."

Figure 8. Percentage obese (BMI ≥30) by age and gender, difference by collection method, CCHS, 2005
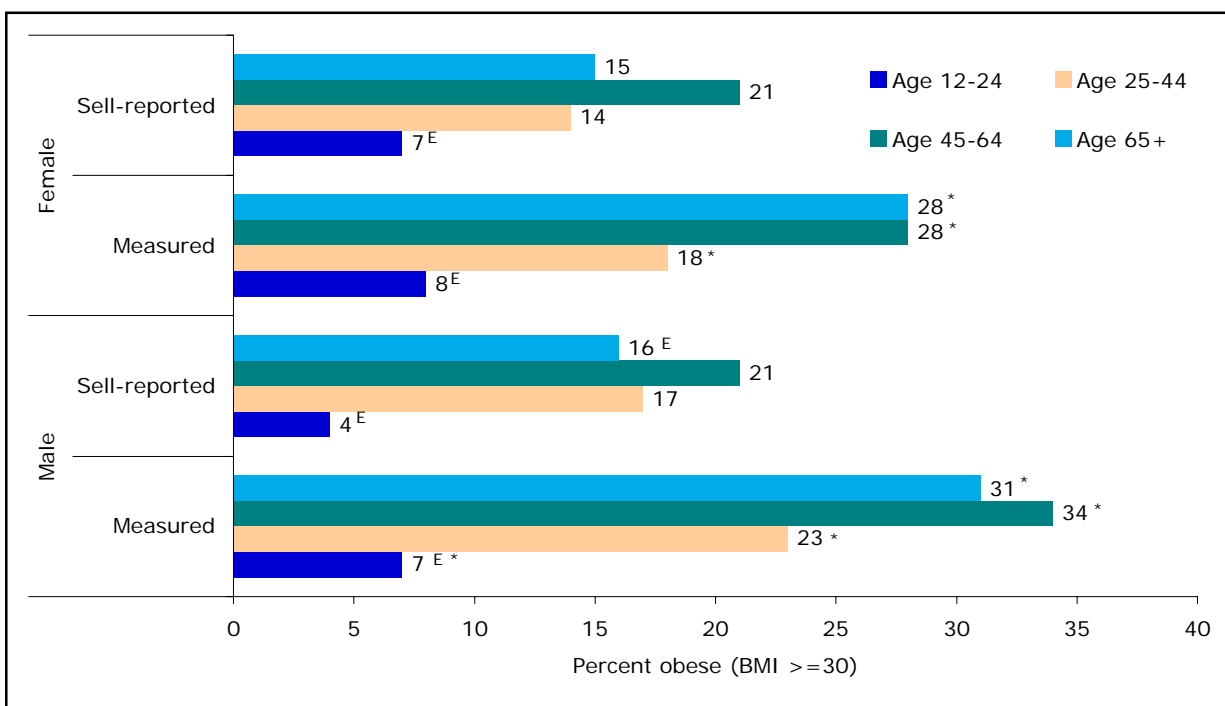
Notes: √ Significantly higher than estimate for same sex based on self-reported values (p < 0.05); [E] − Use with caution (coefficient of variation 16.6% – 33.3%)

Source: Shields, Margot, Sarah Connor Gorber, and Mark S. Tremblay. "Estimates of Obesity Based on Self-Report Versus Direct Measures," *Health Reports*, Statistics Canada, Catalogue no. 82-003-X, 2008, vol. 19, no. 2: 1-16. accessed June 2008; available from http://www.statcan.ca/english/freepub/82-003-XIE/2008002/article/10569-en.pdf.

Since the mid 1990s, the two major Canadian health surveys—the Canadian Community Health Survey (CCHS) and the National Population Health Survey (NPHS)—have generally relied on self-reported data to measure BMI and to assess trends in rates of overweight and obesity. As noted, however, data for directly measured BMI are available for six reference periods: 1970–72, 1978–79, 1981, 1988, 1986–92, and 2004–2005 (although the age groups surveyed do not always correspond, as noted above).[135] According to Shields et al., self-reported data show that BMI rates rose in the 1980s and stabilized between 1994/95 and 2003, but rose again sharply between 2003 and 2004–2005 when the measured collection method was used. Most reports of trends in the prevalence of obesity in Canada have generally been based on self-reported data, and according to Shields et al., "the use of such data means that the accuracy of estimates and

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[135] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

true changes in prevalence over time are unknown."[136] The uncertainty is exacerbated by evidence that the gap between measured and self-reported data is a recent phenomenon and has grown over time.

In addition, Shields et al. note that the accuracy of the association between health conditions and obesity may not be precise when the data used are based on self-reports.[137] The authors compared such associations based on the two collection methods by estimating odds ratios through regression analysis using both self-reported and measured CCHS data for adults aged ≥40 for diabetes, high blood pressure, heart disease, and arthritis or rheumatism. This age group was used because the health conditions considered are more prevalent among older adults. The research model included control variables for age, marital status, education, household income, smoking status, and leisure-time physical activity. Respondents who were underweight were excluded because of the small sample size.

As noted above, a substantial proportion of individuals who were actually obese were categorized as 'overweight' rather than 'obese' when self-reported data were examined, generally due to underestimates of weight and BMI and overestimates of height. The odds of overweight or obese persons having the four health conditions above were therefore considerably higher for models based on self-reported values. This is because many self-reported overweight individuals were actually obese, and those classified as overweight and obese in the self-reported data actually had a higher mean BMI than the self-reported data indicated. This exaggerated the association between the four health conditions and overweight/obesity in the self-reported data and underestimated the number of obese people with the health condition based on self-reported data. When measured data were used and a larger number of individuals were classified as obese, the burden of disease was seen to be higher.

This effect of mis-classification based on the self-reported data was very substantial for some diseases such as diabetes. For example, for diabetes, the odds ratios for overweight (BMI = 25–29.9), obese I (BMI = 30–34.9), and obese 2–3 (BMI ≥35 — the two categories combined because of sample size) using self-reported data were 2.6, 3.2, and 11.8 respectively. When measured data were used, the corresponding odds ratios were 1.4, 2.2, and 7.0. In addition, based on measured CCHS data, 530,000 people, aged ≥40 and classified as obese (BMI ≥30), had diabetes. However, based on self-reported CCHS data, 360,000 people, aged ≥40 and classified as obese (BMI ≥30), had diabetes. Table 5 below shows the differences in the odds ratios for diabetes and in mean BMI by BMI category based on collection method.

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[136] Shields, Gorber, and Tremblay. "Estimates of Obesity Based on Self-Report Versus Direct Measures."
[137] Shields, Gorber, and Tremblay. "Effects of Measurement on Obesity and Morbidity."

Table 5. Odds ratios (OR) for diabetes and mean BMI, by BMI category, based on measured (M) and self-reported (SR) height and weight values, aged ≥40, CCHS, 2005

BMI category	% (based on measured values)	Mean BMI (M-BMI)	Diabetes OR (M-BMI)	% (based on self-reported values)	Mean BMI (SR-BMI)	Diabetes OR (SR-BMI)
Normal weight	30.3	22.6	1.0	39.8	23.6	1.0
Overweight	39.6	27.3	1.4	39.8	28.6	2.6
Obese class I	22.0	31.9	2.2	15.4	33.3	3.2
Obese classes II–III	7.2	39.6	7.0	3.6	42.3	11.8

Notes: OR – Odds ratio; M-BMI – Measured BMI; SR-BMI – Self-reported BMI

Source: Adapted from Shields, Margot, Sarah Connor Gorber, and Mark S. Tremblay. "Effects of Measurement on Obesity and Morbidity," *Health Reports*, Statistics Canada, Catalogue no. 82-003-X, 2008, vol. 19, no. 2: 1-8. Accessed June 2008; available from http://www.statcan.ca/english/freepub/82-003-XIE/2008002/article/10564-en.pdf.

3.2.2 Correction method for 2005 CCHS

As previously noted, Statistics Canada recently conducted a feasibility study to assess the possibility of using prediction equations to correct for the bias in self-reported data by adjusting these self-reported data to bring them in line with measured data results.[138] The results of this study, which tested four models using linear regression statistical analyses, were released in September 2008.[139]

The study concluded that adjusted self-reported data provided more accurate measures of overweight and obesity than did the unadjusted self-reported data, with the results similar to, but slightly lower than, the measured values. The authors of the study, Gorber et al., noted that the corrected and measured data were not statistically different. Based on data from two of the models, the self-reported prevalence of obesity was 16.3% for men and 15.7% for women, the measured prevalence of obesity was 25.6% for men and 22.3% for women, and the corrected obesity rates were approximately 23% for men and 21% for women. In conclusion, the authors of the study noted:

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[138] Shields, Gorber, and Tremblay. "Estimates of Obesity Based on Self-Report Versus Direct Measures."
[139] Gorber, Shields, Tremblay, and McDowell. "The Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity."

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> [T]he improvement in classification for overweight and obese individuals is significant, and thus, we recommend the use of corrected estimates in addition to self-reported values in studies examining overweight and obesity in the adult population of the 2005 CCHS.[140]

Therefore, as previously noted, this report uses the more accurate directly measured data from the 2004 CCHS—the most recent pan-Canadian measured data available with sufficient sample size—to estimate the costs of obesity where possible. However, for some of the health conditions, it was necessary to use 2005 CCHS data. In those cases, which are described in more detail below, the 2005 CCHS data were corrected using the method recommended by Statistics Canada.

Thus, the BMI estimates used to calculate relative risks and population attributable fraction from 2005 CCHS, cycle 3.1 are corrected using the following formulas:[141]

For males: BMI(measured) = 1.08(BMI self-reported) - 1.08   (formula 1)

For females: BMI(measured) = 1.05(BMI self-reported) - 0.12 (formula 2)

Self-reported BMI is the only variable that needs to be added to this formula.
The other numbers, which were determined by Statistics Canada, are fixed statistical factors (i.e. the intercept and slope of the equation of a line) that are needed to make the correction.

3.2.3 Collection method bias in BMI data for children

The collection method bias in BMI data for children aged 2 to 11 is different from that for adults or youth aged 12 and over who report their own BMI. According to Shields et al., parents, who report height and weight for children under the age of 11, tend to underestimate children's height, possibly because children grow quickly.[142] Therefore, when parents report height and weight, BMI, overweight and obesity levels are higher than those calculated from directly measured data—especially for very young children who have the most rapid growth rates.

Shields et al. compared parent-reported data from the 2002/03 National Longitudinal Survey of Children and Youth (NLSCY) with data from the 2004 CCHS, which directly measured the height and weight of the children surveyed. As shown in Table 6 below, the percentage of children, aged 2 to 5, who were either overweight or obese, or obese only was, respectively, 14.6 and 13.8 percentage points higher in parent reports than in measured BMI results. The percentage of children, aged 6 to 11, who were either

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[140] Ibid.
[141] Ibid.
[142] Shields. "Overweight and Obesity among Children and Youth."

overweight or obese or obese only was, respectively, 6.3 and 3.7 percentage points higher in parent reports than in measured results.

Table 6. Difference in BMI for overweight and obesity in children, aged 2–11, by collection method

Age group	2002/ 03 NLSCY (reported by parent)	2004 CCHS (directly measured)	Difference: measured minus parent-reported
Aged 2 to 5			
Average BMI	17.2	16.4	-0.8
% Overweight/obese	36.1%	21.5%	-14.6
% Obese	20.1%	6.3%	-13.8
Aged 6 to 11			
Average BMI	18.2	18.1	-0.1
% Overweight/obese	32.1%	25.8%	-6.3
% Obese	11.7%	8.0%	-3.7

Notes: NLSCY – National Longitudinal Survey of Children and Youth; CCHS – Canadian Community Health Survey

Source: Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol. 17, no. 3: 27-42.

3.3 Adult obesity prevalence and trends in Canada and Alberta

3.3.1 Canadian and provincial obesity trends

The directly measured prevalence of obesity in Canada has more than doubled in the past three decades. As shown in Figure 9 below, directly measured obesity rates (BMI $\geq$ 30) have increased from 10.4% of the adult population aged $\geq$ 20 in 1970 to 22.7% in 2004.[143] For men, the rates nearly tripled—from 7.9% in 1970 to 22.9% in 2004, and for women, the rates increased from 12.9% to 22.5%.

Between 1970 and 1981, as indicated below, no clear trend in obesity is apparent. However, obesity rates began to rise steadily in the 1980s and have increased particularly sharply in the most recent period. Thus, between 1981 and 2004, obesity rates overall increased by 13.5 percentage points or 146.7%, and they have increased by 78% for men in about the last 15 years alone.

Figure 9. Measured prevalence of obesity (BMI $\geq$ 30), aged $\geq$20, Canada, 1970–2004

	NCS 1970-72	CHS 1978-79	CFS 1981	CSWB 1988	CHHS 1986-92	CCHS 2.2 2004
Men	7.9	11.7	9.7	11.2	12.9	22.9
Women	12.9	14.6	8.7	11.9	15.5	22.5
Total	10.4	13.1	9.2	11.6	14.2	22.7

Canadian health surveys - direct measure

Men   Women   Total

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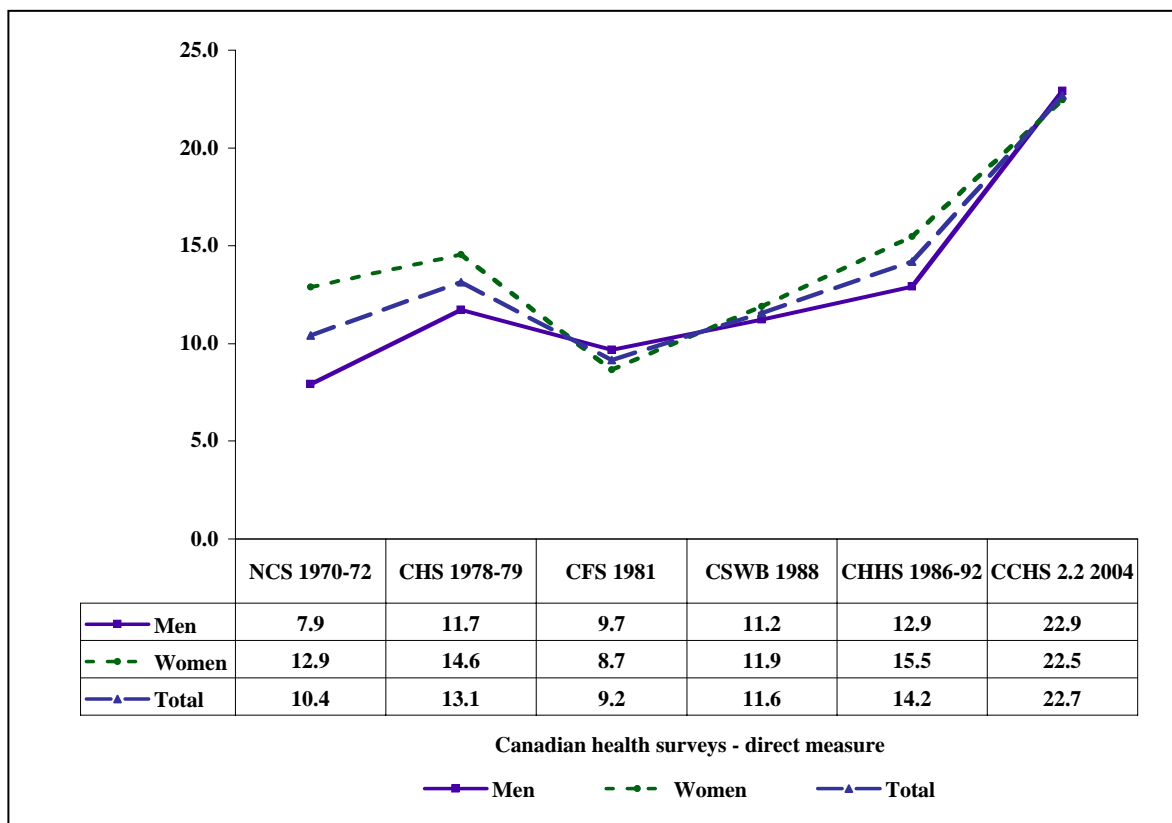
[143] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

Notes: 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, Cycle 2.2

Source: Luo, Wei, Howard Morrison, Margaret de Groh, Chris Waters, Marie DesMeules, Elaine Jones-McLean, Anne-Marie Ugnat, Sylvie Desjardins, Morgan Lim, and Yang Mao. "The Burden of Adult Obesity in Canada," *Chronic Diseases in Canada*, 2007, vol. 27, no. 4: 135-144.

According to Public Health Agency of Canada (PHAC) researchers Luo et al., the prevalence of obesity in Canada could reach 27% for men and 24% for women by 2010 if present obesity trends remain unchanged.[144] The World Health Organization (WHO) has projected that the prevalence of obesity in Canada will increase between 2005 and 2015 by 4.3% for men and by 6.3% for women.[145]

Christel Le Petit and Jean-Marie Berthelot of Statistics Canada examined self-reported longitudinal data from the 1994/95 and 2002/03 National Population Health Surveys (NPHS) to determine the percentage of individuals who moved from normal weight to overweight and from overweight to obesity.[146] They found that almost a third of adults with normal weight in 1994/95 had become overweight by 2002/03, while almost a quarter of those who had been overweight in 1994/95 had become obese by 2002/03. They estimated that by 2003 the observed increase in obesity rates had translated into more than 600,000 new cases of obesity among Canadian men and almost 500,000 new cases among Canadian women. By contrast, only 10% of those who had been overweight in 1994/95 were in the normal range eight years later—indicating very limited success in weight reduction that was overwhelmed by the adverse trends in weight gain.

Heather Orpana et al. of Statistics Canada also analysed longitudinal data from five cycles of the NPHS—from 1996/97 through 2004/05—to examine weight changes over time that cannot be captured using cross-sectional data.[147] They found that adults generally gain weight until the ages of 55 to 60, and then tend to lose weight after that. They also found that individuals who lost weight in one two-year interval tended to gain weight in the next two-year interval. On average, adults gained 0.5 kg to 1 kg (1.1 to 2.2 lbs) in each two-year interval, and over the course of the eight-year time frame, men gained an average of 4.01 kg (8.8 lbs) while women gained an average of 3.44 kg (7.6 lbs). Orpana et al. note:

> While these amounts may appear relatively small, such changes are cumulative, resulting in a further shift of the distribution of an already predominantly

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[144] Ibid.

[145] World Health Organization. *The Impact of Chronic Disease in Canada*, accessed.

[146] Le Petit, Christel, and Jean-Marie Berthelot. *Obesity: A Growing Issue*, Statistics Canada, component of Catalogue no 82-618MWE2005003, 2005; accessed July 2008; available from http://www.statcan.ca/english/research/82-618-MIE/2005003/pdf/82-618-MIE2005003.pdf.

[147] Orpana, Heather M., Mark S. Tremblay, and Philippe Fines. "Trends in Weight Change among Canadian Adults," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2007, vol. 18, no. 2: 9-16.

overweight and obese population toward unhealthy weights. Even a small shift in the population distribution toward excess weight can have important consequences for the incidence of weight-related diseases.[148]

Katzmarzyk and Mason examined seven Canadian health surveys containing self-reported BMI data, which they classified by BMI category and by class of obesity (1, 2, and 3), and found evidence of a dramatic rise in *extreme* obesity.[149] They found that the overall prevalence of self-reported obesity rose by 157% between 1985 and 2003—from 6.1% of Canadians to 15.7%.

Although these self-reported rates are considerably lower than the directly measured 2004 obesity rates indicated in Figure 9 above (22.7%), the overall rate of increase remains sharp, and the evidence reveals particularly dramatic increases of extreme obesity. Thus, the prevalence of class 2 obesity increased by 275% during this time period—from 0.8 percent to 3.0% of Canadians, while class 3 obesity rates increased by 225%—from 0.4% to 1.3% of Canadians. The authors also note that the directly measured results for class 2 and 3 obesity from the 2004 CCHS were 5.1% and 2.7% respectively—considerably higher than the 2003 self-reported CCHS rates of 3.0% and 1.3%.

3.3.1.1 Obesity by place of residence

In keeping with the national statistics outlined above, obesity rates have also risen considerably in all provinces since 1986. Figure 10 below shows directly measured Canadian and provincial obesity rates for 1986–1992 using data from the Canadian Heart Health Surveys, and for 2004 using data from the CCHS—both for the population aged 18-74.

The data show that Alberta obesity rates between 1986 and 2004 rose in parallel with those of Canada, although in both time periods the Alberta rates were slightly higher than those of Canada. Thus, obesity rates in Alberta increased by 9 percentage points from 16% to 25% during this time period, while the overall Canadian rate rose by 8 percentage points from 15% to 23%.

Saskatchewan, Newfoundland and Labrador, and New Brunswick saw the sharpest increases in obesity in this time period (by 15, 12, and 10 percentage points respectively). In 2004, Newfoundland and Labrador had the highest obesity rate in the country (35%), and British Columbia had the lowest (19%).

In 2005, Donald Schopflocher, Senior Biostatistician, Alberta Health and Wellness,

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[148] Ibid. p. 14.
[149] Katzmarzyk, Peter T., and Caitlin Mason. "Prevalence of Class I, II, and III Obesity in Canada," *Canadian Medical Association Journal*, 2006, vol. 174, no. 2: 156-157.

correlated BMI by various characteristics in Alberta such as income, education, health status, prevalence of chronic diseases, and health utilization.[150] Results for these characteristics are reported below. Schopflocher's data were based on self-reported BMI and other variables from the 1996 National Population Health Survey (NPHS), and the 2001 and 2003 Canadian Community Health Surveys (CCHS). He also linked responses to the NPHS with administrative records from the Alberta Health Care Insurance Plan to examine health care utilization by BMI.

Schopflocher found that in Alberta for aged ≥20 years in 1996 that 37.5% of the population was overweight (BMI 25–29.9), 10.3% was obese (BMI 30–39.9), and 1.6% was morbidly obese (BMI ≥40). The rates for obesity combine obese classes 1 and 2, while those for morbidly obese are for obese class 3. By contrast, Statistics Canada used the same NPHS and CCHS data and reported slightly different numbers for the same year in Alberta, but for aged ≥18—35.8% of the Alberta population was overweight (BMI 25–29.9), 10.9% was obese (classes 1 and 2—BMI 30–39.9), and 0.6% was morbidly obese (BMI ≥40).[151]

According to Statistics Canada data for Alberta, by 2005, the percentage of self-reported overweight in the population had declined to 34.7% and the percentage of obesity (classes 1 and 2) had increased to 14.7%. In 2005, the percentage of morbidly obese was 1.1%—which was an increase from 0.6% based on Statistics Canada data, and a decrease from the 1.6% reported by Schopflocher.

[150] Schopflocher, Donald. *Self-Reported Body Mass Index and Its Correlates in Alberta: A Portrait from Survey and Administrative Data Sources*, Alberta Health and Wellness, 2005; accessed July 2008; available from http://www.health.alberta.ca/public/NT_ObesityV6.pdf.

[151] Statistics Canada. *Body Mass Index (BMI), by Sex, Household Population Aged 18 and over Excluding Pregnant Females, Canada, Provinces and Territories, Occasional, 1994 - 2007*, CANSIM Table 105-4009, 2008.

Figure 10. Measured obesity rates, Canada and provinces, aged 18 to 74, 1986–92 and 2004
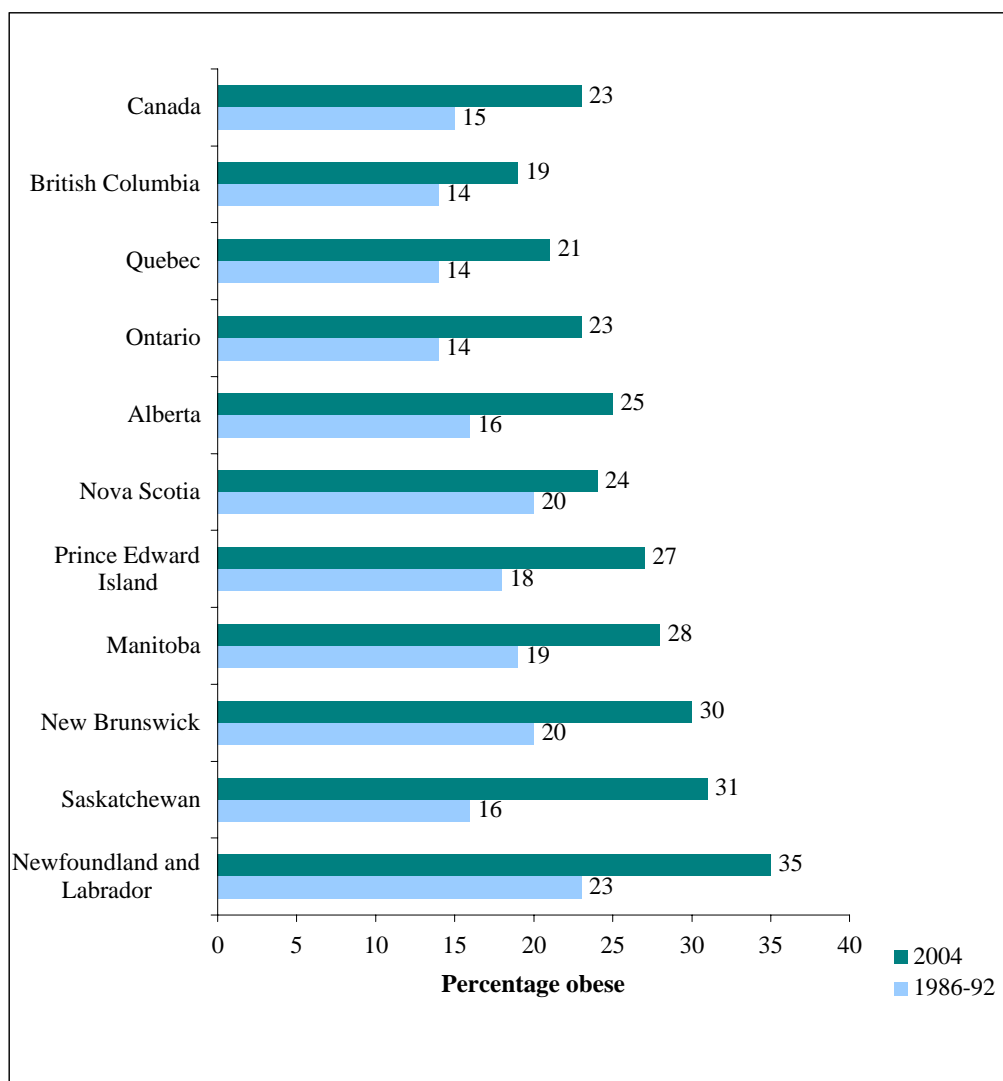
Source: Shields, Margot, and Michael Tjepkema. "Trends in Adult Obesity," *Health Reports*, Statistics Canada, Catalogue 82-003, 2006, vol. 17, no. 3: 53-59.

Shields and Tjepkema of Statistics Canada examined regional differences in obesity between census metropolitan (large urban) areas (CMAs), and non-CMA, or more rural, areas.[152] In Alberta, the prevalence of obesity in 2004 was significantly lower among

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[152] Shields, and Tjepkema. "Regional Differences in Obesity." Statistics Canada defines a Census Metropolitan Area (CMA) as: "Area consisting of one or more neighbouring municipalities situated around a major urban core. A census metropolitan area must have a total population of at least 100,000 of which 50,000 or more live in the urban core." Statistics Canada. *2006 Census Dictionary.* Accessed February 2009. Available from: http://www12.statcan.ca/english/census06/reference/dictionary/geo009.cfm

CMA than non-CMA residents (22.6% vs 32.2%). This pattern of lower obesity rates in large urban areas held true in all provinces except Newfoundland and Labrador, New Brunswick, and Saskatchewan, where obesity rates were not significantly different in CMAs and non-CMAs.

The authors also observed that—among urban areas—obesity rates generally increased in inverse proportion to population size. Thus, obesity rates were lowest in cities with a population of at least 2 million, higher in cities with a population of 100,000 to 2 million, and highest in urban centres with a population of 10,000 to 100,000. They found that in 2004 the obesity rate in Calgary (estimated population 765,000) was 25.7%, and in Edmonton (estimated population 946,000) the rate was 20.1%. By contrast, the rate in Toronto (estimated population 3,772,000) was 15.6%; in Vancouver (estimated population 1,720,000) it was 11.7%; while in Windsor (estimated population 99,000) it was 33.2%.

Schopflocher also found that rural obesity rates in Alberta were higher than urban rates, but the specific data were not given.[153]

3.3.1.2 Obesity by age

As reported by Luo et al., using measured obesity rates, Figure 11 below shows that the pattern of rising adult obesity rates since 1981 is seen across all age groups—20–34, 35–44, 45–54, 55–64, and ≥ 65.[154] Obesity rates have generally increased with age. With the exceptions of 1970 and 1981, the highest proportion of obesity was observed in the 55–64 age group. By 2004, the 45–54 year age group, which had a steep increase in obesity prevalence after 1992, had reached the level of the older group—with about 30% obese in both groups.

The youngest age group—aged 20–34—had the lowest rates of obesity across all time periods, but the sharpest *rate of increase* in obesity prevalence among all age groups. Thus, the 20-34 year group doubled its rate of obesity between 1992 and 2004 alone (from 8.5% to 17.2%) and saw it quadruple since 1981, when it was just 4.3%.

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[153] Schopflocher. *Self-Reported Body Mass Index and Its Correlates in Alberta: A Portrait from Survey and Administrative Data Sources*, accessed.

[154] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

Figure 11. Age-specific prevalence (%) of measured obesity in Canada, aged ≥20, 1970–2004
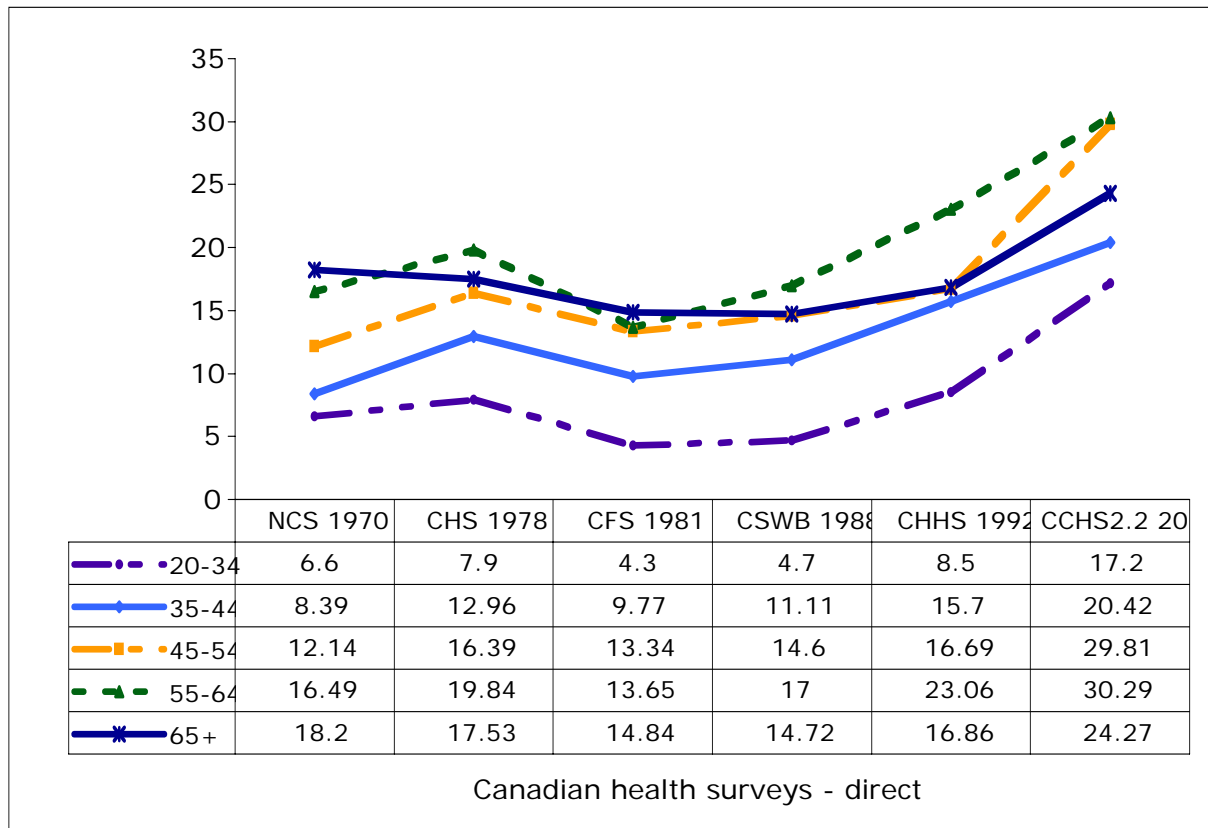
	NCS 1970	CHS 1978	CFS 1981	CSWB 1988	CHHS 1992	CCHS2.2 20
20-34	6.6	7.9	4.3	4.7	8.5	17.2
35-44	8.39	12.96	9.77	11.11	15.7	20.42
45-54	12.14	16.39	13.34	14.6	16.69	29.81
55-64	16.49	19.84	13.65	17	23.06	30.29
65+	18.2	17.53	14.84	14.72	16.86	24.27

Canadian health surveys - direct

Source: Luo, Wei, Howard Morrison, Margaret de Groh, Chris Waters, Marie DesMeules, Elaine Jones-McLean, Anne-Marie Ugnat, Sylvie Desjardins, Morgan Lim, and Yang Mao. "The Burden of Adult Obesity in Canada," *Chronic Diseases in Canada*, 2007, vol. 27, no. 4: 135-144.

Schopflocher also found that in Alberta the rates of obesity in both genders increased with age with the highest rates seen in aged 55–64.[155] After age 65, obesity rates declined, which Schopflocher attributes to the increased tendency for the obese, and especially morbidly obese, to die prematurely.

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[155] Schopflocher. *Self-Reported Body Mass Index and Its Correlates in Alberta: A Portrait from Survey and Administrative Data Sources*, accessed.

3.3.1.3 Obesity by income and smoking status

Matheson et al. combined self-reported data from the 2000/01 and 2003/04 CCHS, Cycles 1.1 and 2.1, with 2001 census tract-level neighbourhood data.[156] They found that, for women, a higher BMI was more associated with living in a neighbourhood with high material deprivation than one with less deprivation. Thus, women living in deprived neighbourhoods had a BMI 1.8 percentage points higher than women living in the most affluent neighbourhoods. However, for men the reverse was true, with men living in the most affluent neighbourhoods recording a higher BMI than men living in deprived neighbourhoods.

Shields and Tjepkema also compared measured obesity rates for Canadians aged 18-74 for the above two time periods—1986–1992 (Canada Heart Health Surveys) and 2004 (CCHS)—by gender, household income quartiles, and smoking status.[157] As seen in Table 7 below, in the 1986–1992 time period, both men and women in the lowest income group had the highest obesity rates. However, by 2004, this pattern had changed dramatically for men, who were more likely to be obese if they were in the highest income category. Obesity rates for men in the lowest income group decreased by one percentage point, but more than doubled for men in the highest income group—rising by 14 percentage points in this time period.

In 2004, when based on income *quartiles*, women in the lowest income category still had the highest obesity rates, and women in the highest income category still had lowest obesity rates. Although obesity rates for women rose in all income categories, the rates for women in the lowest income category rose by only 3 percentage points, compared with increases of 11, 8, and 6 percentage points for women in the lower-middle, upper-middle, and highest income categories, respectively.

However, Tjepkema analyzed 2004 CCHS measured obesity rates by household income *quintiles* for those aged 18 and older, and found different results.[158] As can be seen in Figure 12 below, women in the middle-income category had the highest obesity rate (27.9%), while women in the lowest-income group had the second lowest rate (21.4%) after that of the highest income group (19.6%). Women in the lower-middle (24.8%) and upper-middle (24.7%) income categories had obesity rates higher than women in the lowest income category.

Schopflocher also found similar results to Tjepkema in Alberta when he examined BMI by income quintiles—individuals in the low-middle and middle income quintiles had the highest obesity rates, but the specific data were not provided.

[156] Matheson, Flora I., Rahim Moineddin, and Richard H. Glazier. "The Weight of Place: A Multilevel Analysis of Gender, Neighborhood Material Deprivation, and Body Mass Index among Canadian Adults," *Social Science & Medicine*, 2008, vol. 66: 675-690.
[157] Shields, and Tjepkema. "Trends in Adult Obesity."
[158] Tjepkema, Michael. "Adult Obesity," *Health Reports*, Statistic Canada, catalogue no. 82-003, 2006, vol. 17, no. 3: 9-25.

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The reasons for these apparently disparate income effects by gender—and the different rates of change over time—are not well understood and require further analysis. In particular, the very sharp increase in obesity among rich men since the mid-1980s is noteworthy, with higher-income men now considerably more likely to be obese than higher-income women, while lower-income women are more likely to be obese than lower-income men.

Obesity rates rose for both men and women regardless of smoking status. For both men and women, the highest obesity rates in both time periods are seen in former smokers, which might suggest that quitting smoking could be a risk factor for obesity. According to Shields and Tjepkema, however, this phenomenon may also be explained by age, since former smokers tend to be older than current smokers, and obesity rates tend to be higher in older individuals. They note:

> In fact, when associations between smoking status and obesity were examined in multivariate models controlling for age, among women, being a former smoker was no longer associated with a higher likelihood of obesity in either 1986-92 or 2004. On the other hand, among men, the finding that former smokers were more likely to be obese persisted for both periods.[159]

Table 7. Percentage obese, by gender, household income, and smoking status, aged 18 – 74, Canada, 1986–92 and 2004

	1986 – 92	2004		1986 – 92	2004
TOTAL	14.6	23.1			
Men	13.4	23.2			
Women	15.8	22.9			
HOUSEHOLD INCOME			**SMOKING STATUS**		
Men			**Men**		
Lowest	21.9	20.7[E]	Current smoker	14.4	19.7
Lower-middle	14.6	24.7	Former smoker	16.8	29.7
Upper middle	13.8	23.8	Never smoked	8.2	21.0
Highest	11.8	25.9			
Women			**Women**		
Lowest	25.5	28.3	Current smoker	14.0	23.9
Lower-middle	14.5	25.8	Former smoker	19.1	27.0
Upper middle	16.2	24.1	Never smoked	15.3	20.7
Highest	13.1	19.4			

Note: [E] – Coefficient of variation between 16.6% and 33.3% (interpret with caution).

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[159] Shields, and Tjepkema. "Trends in Adult Obesity."

Source: Adapted from: Shields, Margot, and Michael Tjepkema. "Trends in Adult Obesity," *Health Reports*, Statistics Canada, Catalogue 82-003, 2006, vol. 17, no. 3: 53-59.

Figure 12. Obesity rates by household income and gender, percentage, aged 18 and older, Canada, 2004
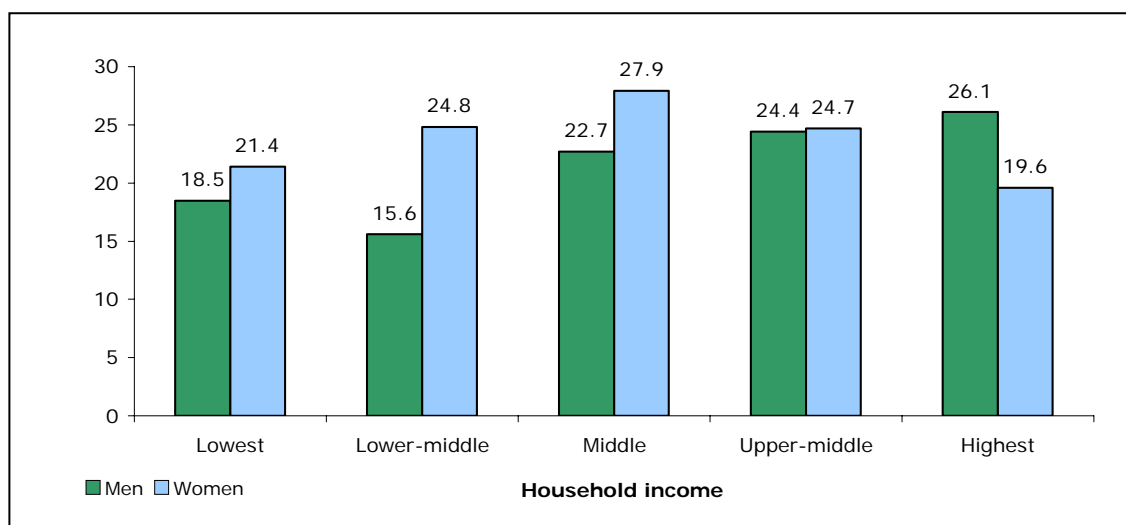
Source: Tjepkema, Michael. "Adult Obesity," *Health Reports*, Statistic Canada, catalogue no. 82-003, 2006, vol. 17, no. 3: 9-25.

3.3.1.4 Obesity by physical activity, diet, and education

Tjepkema also analyzed measured obesity data from 2004 CCHS for specific characteristics, including physical activity and fruit and vegetable consumption for those aged 18 and older, and formal educational attainment for those aged 25 to 64.[160] Results can be seen in Figures 13 – 15 below. As expected, obesity was significantly related to diet and physical exercise, and it was generally inversely correlated with formal educational attainment.

Thus, both men and women whose leisure time was largely sedentary were more likely to be obese than those who were more physically active in their leisure time. The obesity rate among men who were sedentary was 27%, compared with 19.6% for physically active men and 16.7% for moderately active men—indicating that even moderate activity apparently conferred as much protection against obesity for men as higher levels of physical activity.

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[160] Tjepkema, Michael. *Measured Obesity - Adult Obesity in Canada: Measured Height and Weight*, Analytical Studies and Reports, Statistics Canada, Catalogue no. 82-620-MWE2005001, 2005; accessed June 2008; available from http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/aobesity.pdf.

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Nearly twice as many sedentary women (26.8%) were obese as those who were physically active (13.5%). However, by contrast to men, moderately active women also had relatively high obesity rates (20.8%), indicating that moderate activity apparently conferred less protection against obesity for women than for men and that women appear to require higher levels of physical activity than men to guard against obesity. At the same time, the results appear to indicate that high levels of physical activity confer even more protection against obesity for women than for men. These apparent gender differences are also not well understood and require further investigation.

Clearly diet plays a major role in determining the propensity for overweight and obesity. Thus, men and women who consumed fruit and vegetables less than three times a day were more likely to be obese (25.8% and 27.4%, respectively) than those who ate these foods five or more times a day (19.1% and 20.3%, respectively). While it is possible to correlate BMI with fruit and vegetable consumption using CCHS data—since these are specific questions in that survey—further study is needed of a wide range of other dietary correlations with obesity.

Obesity is generally inversely correlated with formal educational attainment. Thus, for those aged 25 to 64 with less than a secondary education, obesity rates were extremely high for both men (34.5%) and women (36.9%). Obesity rates were lowest for both men (22.0%) and women (20.5%) with a postsecondary education. Confounding the pattern somewhat, however, women with some postsecondary education were found to have higher obesity rates (32.3%) than women who had only completed a secondary education (24.9%).

Schopflocher also found that individuals in Alberta without a secondary education had higher levels of overweight and obesity that those with secondary educations, some post-secondary education, and college or university degrees.[161] Although he did not provide specific data, Schopflocher noted that the differences between the other educational groups did not have statistical significance.

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[161] Schopflocher. *Self-Reported Body Mass Index and Its Correlates in Alberta: A Portrait from Survey and Administrative Data Sources*, accessed.

Figure 13. Obesity rates, by leisure-time physical activity level and sex, household population aged 18 or older, Canada excluding territories, 2004
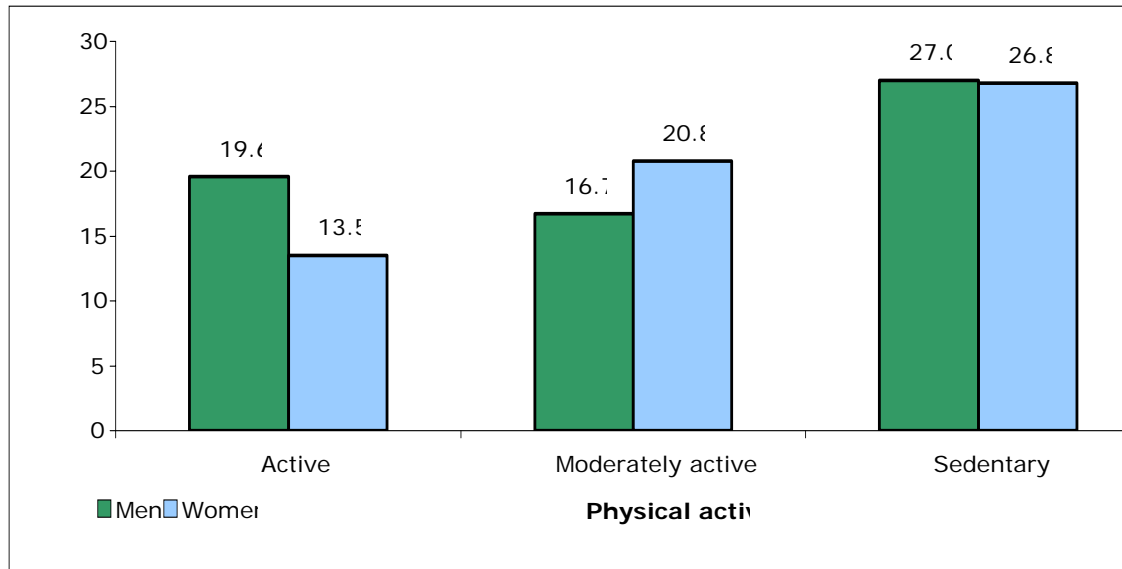
Source: Tjepkema, Michael. *Measured Obesity - Adult Obesity in Canada: Measured Height and Weight*, Analytical Studies and Reports, Statistics Canada, Catalogue no. 82-620-MWE2005001, 2005; accessed June 2008; available from http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/aobesity.pdf.

Figure 14. Obesity rates, by fruit and vegetable consumption and sex, household population aged 18 or older, Canada excluding territories, 2004
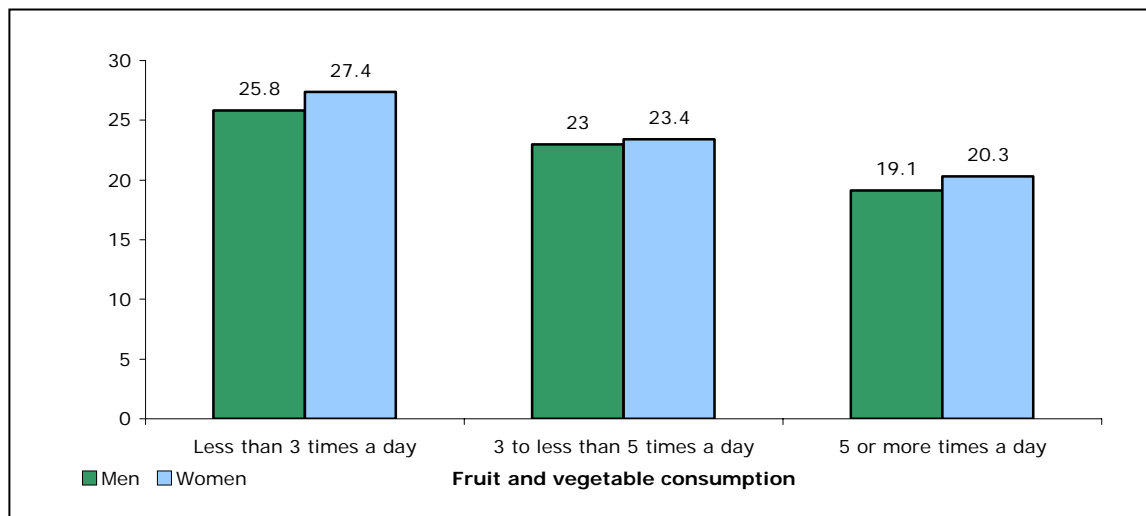
Source: Tjepkema, Michael. *Measured Obesity - Adult Obesity in Canada: Measured Height and Weight*, Analytical Studies and Reports, Statistics Canada, Catalogue no. 82-620-MWE2005001, 2005; accessed June 2008; available from http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/aobesity.pdf.

Figure 15. Obesity rates, by educational attainment and sex, household population aged 25 to 64, Canada excluding territories, 2004
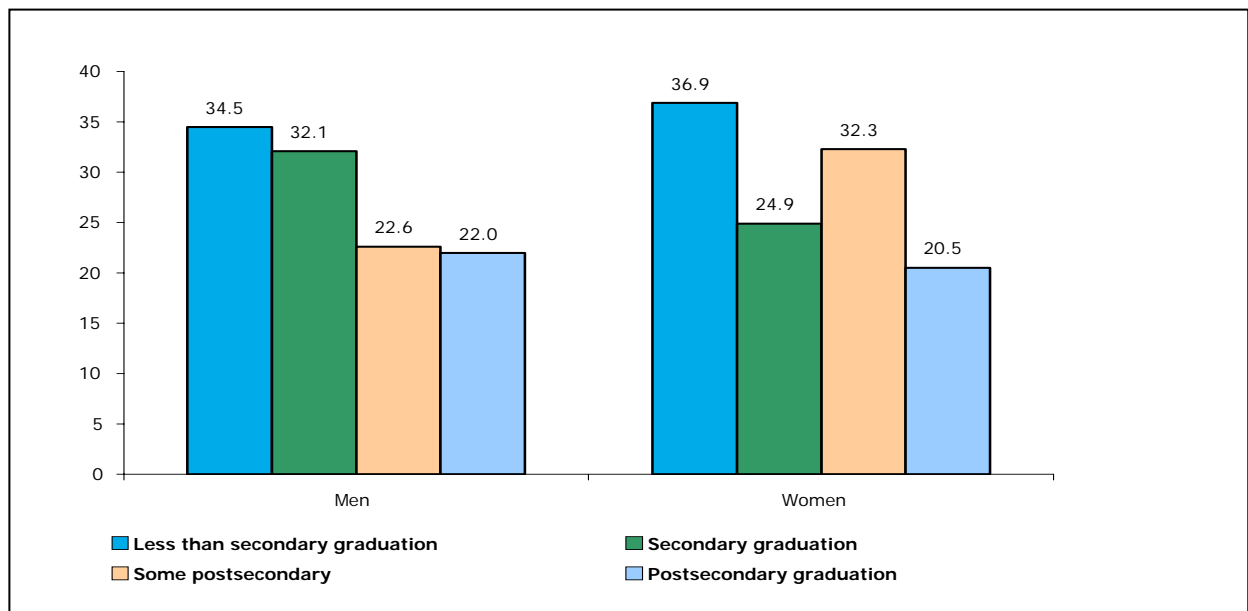
Source: Tjepkema, Michael. *Measured Obesity - Adult Obesity in Canada: Measured Height and Weight*, Analytical Studies and Reports, Statistics Canada, Catalogue no. 82-620-MWE2005001, 2005; accessed June 2008; available from http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/aobesity.pdf.

As previously noted, Schopflocher also correlated BMI in Alberta by health status, prevalence of chronic diseases, and health utilization using data from 1996 NPHS, and 2001 and 2003 CCHS.[162] He found that 12% of obese classes 1 and 2, and 9% of morbidly obese reported that they had fair or poor health, compared with 8% of the normal weight population. The proportion of persons reporting having been diagnosed with one or more chronic condition also generally increased with increasing BMI—16% for obese classes 1 and 2, and 22% of morbidly obese reported having two or more chronic diseases, compared with 14% with normal weight.

In terms of health care utilization, Schopflocher linked NPHS data with Alberta administrative data. Although specific data were not provided, he found that the gradation

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[162] Ibid., accessed.

in health utilization was "as expected: individuals with morbid obesity requiring greater levels of utilization than those with obesity and with the obese having higher levels than overweight or those with acceptable weights."[163]

3.3.2 Alberta self-reported and measured obesity prevalence and trends

Both self-reported and measured obesity rates for Alberta are presented in this section. Table 8 below shows Alberta self-reported BMI rates for normal weight (BMI 18.5–24.9), overweight (BMI 25–29.9), obese (BMI ≥30.0), and obese classes 2–3 (BMI 30–34.9, 35–39.9, and ≥40.0, respectively), by gender, for 1994 – 2007. Statistics Canada did not consistently release rates for those who are underweight (BMI <18.5) because the data were based on small sample sizes and, therefore, were considered to be unreliable.

As discussed in Section 3.3 above, self-reported BMI rates are considerably lower than those obtained through directly measured collection methods. For example, in 2004, as shown in Table 9 below, the measured obesity rate for Alberta was 25.2%, while the self-reported rate in 2005 was 15.8%. The 2007 self-reported obesity rate in Alberta was 18.3%—a 2.5 percentage point increase from 2005—but still considerably lower than the 2004 measured rate. Therefore only measured rates and adjusted self-reported rates, which are much more reliable and accurate than self-reported rates, are used to estimate the costs of obesity.

Nevertheless, self-reported obesity rates are provided in this section because they are available more consistently and for more years than are measured rates, and are therefore necessary to indicate trends over time. For the data reliability reasons stated, the following self-reported data should therefore be used only to assess *relative* changes over time and not to estimate actual or absolute rates of obesity (for which only the directly measured data can accurately be used)

Self-reported weight and height data show an increase of more than 50% in Alberta obesity rates in just 13 years—from 12% in 1994/95 to 18.3% in 2007. The proportion of males with a self-reported BMI in the obese category nearly doubled during this time period—from 10.1% to 20.1%, while the proportion of obese females increased from 13.9% to 16.4%.

Again, the high degree of bias in these data is revealed by the fact that 2004 directly measured obesity rates in Alberta were 27.7% for men and 22.6% for women compared to 2005 self-reported rates of 17.6% for men and 13.9% for women in the 2005 self-reported data. This indicates that Albertan men and women may under-report BMI data by more than 36% and 38% respectively.

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[163] Ibid., accessed. p. 25.

Self-reported rates for obesity classes 1, 2, and 3 in Alberta show increases from 9.3% to 13%, from 1.7% to 3.8%, and from 0.9% to 1.4%, respectively between 1994/95 and 2007. Directly measured Alberta rates for these three classes of obesity in 2004 were 15.4%, 6.7%, and 3.2%, respectively, compared to 11.6%, 3.1%, and 1.1% in the self-reported Alberta data for 2005. This indicates even stronger under-reporting bias among the most obese Albertans—with class 2 obesity Albertans (BMI 35–39.9) under-reporting by 54%, and class 3 obesity Albertans (BMI ≥40.0) under-reporting by 66%).

Self-reported overweight rates (BMI 25–29.9), which indicate an increased risk of health problems according to the definitions of overweight and obesity discussed in Chapter 2 above, have remained much more stable over time, as the number of Albertans moving from normal weight (BMI 18.5–24.9) to overweight (BMI 25–29.9), has been more than offset by the number moving from the overweight category to the obese category (BMI ≥30.0). In fact, the proportion of Albertans in the overweight category decreased by 3 percentage points from 35.5% in 1994/95 to 32.5% in 2007. The rates for men fell from 45.3% in 1994/95 to 39.2% in 2007, and remained fairly stable for women (25.4% in 1994/95 and 25.5% in 2007).

In line with the phenomenon that under-reporting increases with BMI, it is noteworthy that under-reporting is much less apparent in the overweight category than in the obese categories noted above. Thus. directly measured overweight rates in 2004 were 35.7%—41.1% for men and 30.3% for women, compared to 2005 self-reported overweight rates of 34.7%—42% for men and 27.1% for women.

Self-reported normal, or healthy, weights (BMI 18.5–24.9) in Alberta, fell from 49.1% in 1994/95 to 42.9% in 2007. Rates of healthy weight for men fell from 43.9% to 37.4% in this time period, and for women from 54.4% to 48.7%.

Significantly, normal or healthy weights are over-reported in roughly the same degree to which obesity is under-reported. Thus, 2004 directly measured normal weight rates in Alberta were only 37.3%—29.5% for men and 45.3% for women, compared to 2005 self-reported rates of 45%—39.2% for men and 51% for women. The difference is significant and has serious implications for the health of Albertans, since the directly measured data indicate that fewer than 3 in 10 Albertan men and less than half of Albertan women have healthy weights.

Table 8. Alberta adult self-reported BMI rates (%), aged ≥18, by gender, 1994/95 – 2007

Year/ gender	Normal (BMI 18.5-24.9)	Overweight (BMI 25-29.9)	Obese (BMI ≥30)	Obese Class 1 (BMI 30-34.9)	Obese Class 2 (BMI 35-39.9)	Obese Class 3 (BMI ≥40)
1994/ 95	49.1	35.5	12.0	9.3	1.7[E]	0.9[E]
Male	43.9	45.3	10.1	8.3	F	F
Female	54.4	25.4	13.9	10.3	2.5[E]	F
1996/ 97	47.0	35.8	11.4	9.0	1.9	0.6
Male	40.0	45.4	12.0	9.8	1.7	0.5[E]
Female	54.2	26.1	10.8	8.2	2.0	0.6
1998/ 99	45.8	34.1	16.0	12.3	2.1[E]	1.7[E]
Male	38.3	44.9	14.1	12.4	F	F
Female	53.5	23.1	17.9	12.1	3.6[E]	2.2[E]
2001	47.2	32.7	15.5	11.8	2.7	1.1
Male	41.8	39.6	16.8	13.4	2.8	0.6[E]
Female	52.7	25.5	14.3	10.1	2.7	1.5
2003	45.3	34.1	15.5	11.5	2.9	1.2
Male	40.0	42.0	16.6	13.0	2.4	1.2[E]
Female	50.9	26.0	14.3	9.9	3.3	1.1
2005	45.0	34.7	15.8	11.6	3.1	1.1
Male	39.2	42.0	17.6	13.8	2.9	0.9[E]
Female	51.0	27.1	13.9	9.4	3.2	1.3
2007	42.9	32.5	18.3	13.0	3.8	1.4
Male	37.4	39.2	20.1	15.3	3.7[E]	1.1[E]
Female	48.7	25.5	16.4	10.6	4.0	1.7[E]

Notes: [E] — use with caution (coefficient of variation between 16.6% and 33.3%); F — too unreliable to be published.

Sources: 1994 – 1999: National Population Health Surveys; 2001 – 2007: Canadian Community Health Surveys; Statistics Canada. Body Mass Index (BMI), by Sex, Household Population Aged 18 and over Excluding Pregnant Females, Canada, Provinces and Territories, Occasional, 1994 – 2007, CANSIM Table 105-4009, 2008.

Table 9 below shows the 2004 directly measured *number* of Alberta adults, aged ≥18, in each BMI category by gender and seven age groups. This level of detail is needed in order to calculate population attributable fractions (PAF) by gender and age, which in turn is necessary to estimate obesity costs according to these breakdowns. This methodology is discussed in Chapter 4 below. Table 9 corresponds with Table 10 below, which lists the 2004 directly measured *percentages* of Albertans in each category by the same age groups.

Tables 8 and 9 below show that, among Alberta's 2004 population of 2,345,818 adults aged 18 and over, 37.3% had normal or healthy weights (BMI 18.5–24.9), 35.7% were overweight (BMI 25–29.9), and 25.2% were obese (BMI ≥30). Breaking down the obese adults by class, it is seen that 15.4% of Albertan adults aged ≥18 can be classified as obese class 1 (BMI 30–34.9), 6.7% as obese class 2 (BMI 35–39.9), and 3.2% as obese class 3 (≥40), although this last result for obese class 3 is subject to a wide confidence interval.

Although obesity rates appear to be highest for the 45-54 age group (34.9%) and the 55–64 age group (30.5%)—which accords with evidence presented earlier that the likelihood of obesity increases with age at least into the fifties—it is particularly alarming that 25–34 year-old Albertans also appear to have a very high rate of obesity (26.0%) for such a young age group. This result appears to be considerably higher than the Canadian average for this age group (20.5%), and could have serious implications for future health risks and for Alberta's health care costs as these young adults age.

It should be noted that the statements about age group comparisons above are qualified by phrases like "*appear* to have" and "subject to a wide confidence interval." This is because most of the results in the tables below are subject to the further serious qualification that many of the results have very large confidence intervals. Therefore Statistics Canada has added the caveat that most of the data in Tables 8 and 9 below should be "used with caution." These data, which have a coefficient of variation from 16.6% to 33.3%, are indicated in the tables by the symbol "[E]". Thus, for example, we noted above that 25–34 year-old Albertans "appear to have" a very high rate of obesity (26.0%). However, the confidence interval for this result shown in Table 10 below indicates that this rate could potentially be as low as 17% and as high as 35%.

That said, some major caveats must be added concerning these comparisons by age group, and indeed to interpretation of most of the age-specific results for Alberta listed in Tables 9 and 10 below. This is due to a number of serious data limitations in the tables, which come directly from Statistics Canada's CANSIM database. Because of extreme sampling variability, Statistics Canada has suppressed most of the Alberta data for obese classes 1, 2, and 3 for most age groups, as well as for the total male data for obesity class 3 (BMI ≥40). Some data by gender have also been suppressed in some of the age groupings, whenever the sample size was too small to yield statistically significant results—such as obesity rates for males and females in the 18–24 age group, females in the 35–44 age group, and males in the 65 and over age groups. Suppressed data are indicated by the letter "F" in the tables.

Therefore, it was only possible to estimate obesity costs by gender and age group within each obesity class (1, 2, and 3) by extrapolating some results for Alberta from the national CCHS data, including age-specific population attributable fractions for illnesses linked to obesity, and then by applying those data to direct and indirect costs estimated specifically for Alberta. This methodology is described in more detail in Chapter 4.2 below.

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Table 9. Alberta, number of adults, measured BMI rates, aged ≥18, by gender, CCHS, 2004

Age	Gender	Number of persons						
		TOTAL	Normal	Overweight	Obese	Obese Class 1	Obese Class 2	Obese Class 3
18+	TOTAL	2,345,818	876,107	837,430	591,343	360,697	156,449	74,197[E]
	Male	1,181,555	348,537	485,091	327,812	208,677	88,365	30,771[E]
	Female	1,164,262	527,570	352,339	263,531	152,020	68,085	43426[E]
18 - 24	Total	332,241	169,910	106,165[E]	42,969[E]	F	F	F
	Male	176,398	59,855[E]	77,059[E]	F	F	F	F
	Female	155,843	110,055	29,106	F	F	F	F
25 - 34	Total	419,804	209,513[E]	95,626[E]	109,035	66,333	F	F
	Male	194,523	73,216[E]	61,384[E]	58,122[E]	F	F	F
	Female	225,281	136,296[E]	F	50,913[E]	F	F	F
35 - 44	Total	496,879	170,611[E]	216,134[E]	95,491[E]	61,701[E]	F	F
	Male	264,515	60,228[E]	128,141[E]	70,294[E]	F	F	F
	Female	232,364	110,383[E]	87,994[E]	F	F	F	F
45 - 54	Total	496,961	138,108[E]	180,222	173,332[E]	102,396[E]	44,848[E]	F
	Male	253,481	72,488[E]	97,578[E]	83,415[E]	F	F	F
	Female	243,480	65,621[E]	82,644[E]	89,917[E]	F	F	F
55 - 64	Total	294,337	98,109[E]	106,338	89,891[E]	51,467[E]	F	F
	Male	148,088	F	57,954[E]	49,443[E]	F	F	F
	Female	146,249	57,417[E]	48384[E]	40,448[E]	F	F	F
65 - 74	Total	177,995	43,044[E]	83,905[E]	48,879[E]	F	F	F
	Male	89,887[E]	F	42,072	F	F	F	F
	Female	88,108	27,169[E]	41,833	18,506[E]	F	F	F
75+	Total	127,600	46,813[E]	49,040[E]	31,747[E]	29,534[E]	F	F
	Male	54,663	26,183[E]	29,904[E]	F	F	F	F
	Female	72,937	20,630[E]	28,136[E]	F	F	F	F

Notes: F – Too unreliable to be published. Data with a coefficient of variation greater than 33.3% were suppressed by Statistics Canada due to extreme sampling variability. For that reason, underweight rates were also too unreliable to be published, and are therefore not shown here. E – use with caution (— data with a coefficient of variation from16.6% to 33.3%).

Source: Statistics Canada. Measured Adult Body Mass Index (BMI), by Age Group and Sex, Household Population Aged 18 and over Excluding Pregnant Females, Canada and Provinces, CANSIM Table 105-2001 (Alberta), 2004 Canadian Community Health Survey Cycle 2.2, 2008.

Table 10. Alberta adult measured BMI rates (%), aged ≥18, by gender, age group, and (confidence interval – CI), CCHS, 2004

Age	Gender	TOTAL Number	Percentage					
			Normal	Overweight	Obese	Obese Class 1	Obese Class 2	Obese Class 3
18+	TOTAL	2,345,818	37.3	35.7	25.2	15.4	6.7	3.2[E]
CI			(32.5 - 42.2)	(30.7 - 30.7)	(21.4 - 29.0)	(11.8 - 18.9)	(4.7 - 8.6)	(1.8E - 4.6E)
	Male	1,181,555	29.5	41.1	27.7	17.7[E]	7.5[E]	F
CI			(22.9 - 36.1)	(33.2 - 48.9)	(21.9 - 33.6)	(11.8E-23.5E)	(4.3E - 10.7E)	F
	Female	1,164,262	45.3	30.3	22.6	13.1	5.8[E]	3.7[E]
CI			(38.9 - 51.7)	(24.2 - 36.4)	(18.1 - 27.2)	(9.4 - 16.8)	(3.2E - 8.5E)	(1.4E - 6.1E)
18 - 24	Total	332,241	51.1	32	12.9[E]	F	F	F
CI			(40.8 - 61.5)	(21.1 - 42.8)	(5.3 - 20.6)	F	F	F
	Male	176,398	33.9[E]	43.7[E]	F	F	F	F
CI			(20.6E-47.3E)	(27.2E-60.2E)	F	F	F	F
	Female	155,843	70.6	18.7[E]	12.1	F	F	F
CI			(58.4 - 82.8)	(7.5E - 29.8E)	F	F	F	F
25 - 34	Total	419,804	49.9	22.8[E]	26.0[E]	15.8[E]	F	F
CI			(38.4 - 61.4)	(14.0E-31.6E)	(17.0E-35.0E)	(8.1E - 23.5E)	F	F
	Male	194,523	37.6[E]	31.6[E]	29.9[E]	F	F	F
CI			(20.7E-54.6E)	(16.6E-46.5E)	(14.5E-45.3E)	F	F	F
	Female	225,281	60.5	F	22.6[E]	F	F	F
CI			(45.1 - 75.9)	F	(11.8E-33.4E)	F	F	F
35 - 44	Total	496,879	34.3[E]	43.5[E]	19.2[E]	12.4[E]	F	F
CI			(22.3E-46.3E)	(29.2E-57.8E)	(9.5E - 28.9E)	(4.5E - 20.3E)	F	F
	Male	264,515	22.8[E]	48.4[E]	26.6	F	F	F
CI			(9.1E-36.4E)	(28.1 - 68.8E)	(9.3E - 43.8E)	F	F	F
	Female	232,364	47.5[E]	37.9[E]	F	F	F	F
CI			(28.7E-66.3E)	(19.9E-55.9E)	F	F	F	F
45 - 54	Total	496,961	27.8[E]	36.3	34.9[E]	20.6[E]	9.0[E]	F
CI			(17.8E-37.8E)	26.0 - 46.5	(24.8E-44.9E)	(11.6E-29.6E)	(3.7E - 14.4E)	F
	Male	253,481	28.6[E]	38.5[E]	32.9[E]	F	F	F
CI			(13.3E-43.8E)	(23.2E-53.8E)	(17.1E-48.8E)	F	F	F
	Female	243,480	27.0[E]	33.9[E]	36.9[E]	18.6[E]	F	F
CI			(15.1E-38.8E)	(19.2E-48.7E)	(23.8E-50.0E)	(7.6E - 29.5E)	F	F
55 - 64	Total	294,337	33.3[E]	36.1[E]	30.5[E]	17.5[E]	F	F
CI			(18.7E-47.9E)	23.5E- 48.8E)	18.9E-42.1E)	(8.5E - 26.5E)	F	F
	Male	148,088	F	39.1[E]	33.4[E]	F	F	F
CI			F	(20.2E-58.1E)	(16.6E-50.2E)	F	F	F
	Female	146,249	39.3[E]	33.1[E]	27.7[E]	F	F	F
CI			(24.2E-54.4E)	(18.2E-48.0E)	(14.1E-41.2E)	F	F	F
65 - 74	Total	177,995	24.2[E]	47.1	27.5[E]	F	F	F
CI			(13.3 - 35.0E)	(33.4 - 60.9)	14.8E-40.1E)	F	F	F
	Male	89,887[E]	F	46.8[E]	F	F	F	F
CI			F	(22.5E-71.1E)	F	F	F	F

Age	Gender	TOTAL Number	Percentage					
			Normal	Overweight	Obese	Obese Class 1	Obese Class 2	Obese Class 3
	Female	88,108	30.8^E	47.5	21.0^E	F	F	F
CI			(16.1^E-45.6^E)	(32.6 - 62.4)	(9.4^E - 32.6^E)	F	F	F
75+	**Total**	127,600	36.7^E	38.4^E	24.9^E	23.1^E	F	F
CI			(22.7^E-50.7^E)	(24.1^E-52.8^E)	(12.5^E-37.2^E)	(10.7^E-35.6^E)	F	F
	Male	54,663	47.9^E	38.2^E	F	F	F	F
CI			(26.3^E-69.5^E)	(15.7^E - 60.8^E)	F	F	F	F
	Female	72,937	28.3^E	38.6^E	33.1^E	32.0^E	F	F
CI			(11.1^E-45.4^E)	(20.5^E-56.7^E)	(13.8^E-52.5^E)	(12.2^E-51.8^E)	F	F

Notes: F – Too unreliable to be published. Data with a coefficient of variation greater than 33.3% were suppressed by Statistics Canada due to extreme sampling variability. For that reason, underweight rates were also too unreliable to be published, and are therefore not shown here. E – use with caution (— data with a coefficient of variation from16.6% to 33.3%). CI – 95% confidence interval (low – high).

Source: Statistics Canada. Measured Adult Body Mass Index (BMI), by Age Group and Sex, Household Population Aged 18 and over Excluding Pregnant Females, Canada and Provinces, CANSIM Table 105-2001 (Alberta), 2004 Canadian Community Health Survey Cycle 2.2, 2008

3.4 Children and youth obesity prevalence and trends

3.4.1 Canadian prevalence and trends

It was not possible to estimate the costs of obesity for children and youth because the health risk for obesity in children has not been established. Therefore, the percentages of costs attributable to obesity in children could not be estimated with any degree of accuracy. Because obesity rates for children and youth have been increasing, however, it is important to include some information on the effects of obesity on children's health, and to flag this area as important for future considerations.

As discussed in Chapter 2.2 above, BMI for children and youth, aged 2 to 17, is calculated in the same way as for adults—weight in kilograms divided by height in metres squared. However, because children and youth are still maturing and therefore have different body types at different ages, the classification of BMI to assess overweight and obesity in children is different from that used to classify adults. Since 2004, Statistics Canada has used the standard International Obesity Task Force (IOTF) criteria, which is based on sex- and age-specific BMI thresholds, to measure overweight and obesity in

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children and youth.[164] Based on their BMI at different ages, these criteria classify children and youth as "neither overweight nor obese," "overweight," or "obese".

Margot Shields of Statistics Canada has produced two comprehensive reports on overweight and obesity among Canadian children and youth using 2004 measured CCHS data. Because these are the most accurate available results to date and are based on the same data source used in this study to assess adult obesity costs in Alberta, her results are reported in some detail below.

Shields found that more than one-quarter of all Canadian children and youth aged 2-17 were overweight or obese in 2004—which amounted to more than 1.1 million overweight and more than half a million obese.

In order to assess trends over time using the more accurate directly measured data, Shields compared the measured overweight and obesity rates for Canadian children and youth, aged 2 to 17, using data from the 1978/79 Canada Health Survey and the 2004 CCHS.[165] As shown in Figure 16 below, the prevalence of overweight among Canadian children and youth has increased from 12% to 18% over the past 25 years,, and the prevalence of obesity among children and youth has nearly tripled—from 3% to 8%. Combined, the overweight/obesity rates increased by 73% in this time period—from 15% to 26% of Canadian children and youth. The increase was similar for both boys and girls.

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[164] Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol. 17, no. 3: 27-42.
[165] Ibid.

Figure 16. Percentage overweight or obese, by sex, aged 2 to 17, Canada, 1978/79 and 2004
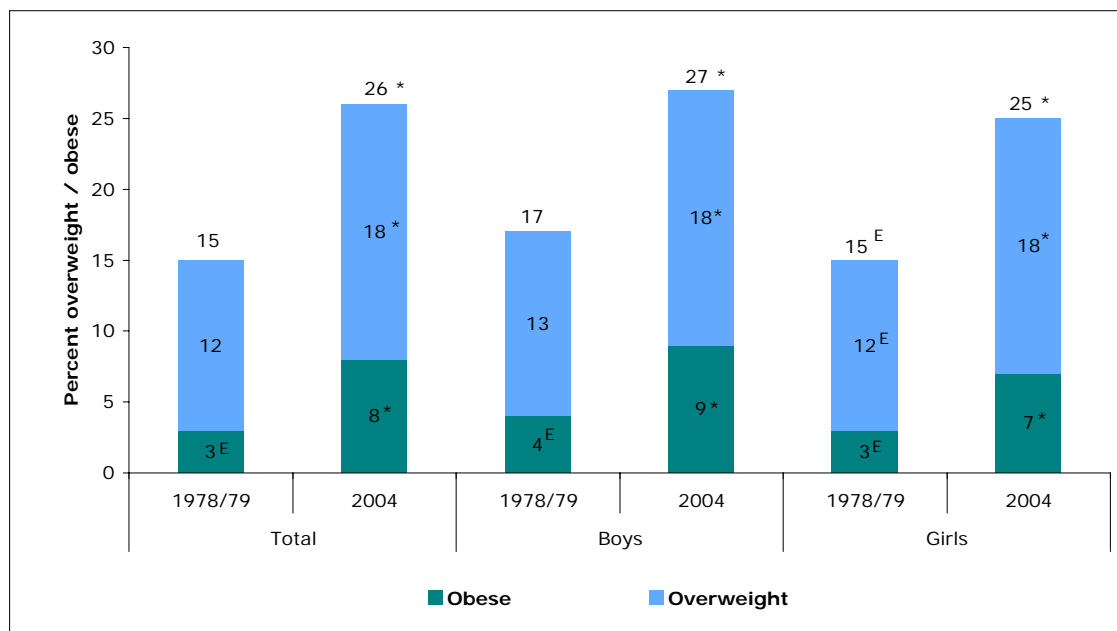
E = Coefficient of variation 16.6% to 33.3% (interpret with caution); * Significantly different from estimate for 1978/79. Data sources: 1978/79 Canada Health Survey; 2004 Canadian CommunityHealth Survey, cycle 2.2.

Source: Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol. 17, no. 3: 27-42.

Figure 17 below illustrates the overweight and obesity rates by age group for the same time periods. The percentage of combined overweight and obese children aged 2 to 5 remained unchanged at 21%, but doubled for older children—rising from 13% in 1978/79 to 26% in 2004 among those aged 6-11 and from 14% to 29% among 12- to 17-year-olds. However, the most dramatic changes in all age groups, including the youngest, appear to have occurred in obesity rates, which tripled among 12-17 year-olds—rising from 3% in 1978/79 to 9% in 2004, and also likely rose sharply in the younger age groups. While 1978/79 obesity results for 2-11 year-olds were suppressed because their coefficient of variation was greater than 33.3%, it is noteworthy that 6% of Canadian infants and toddlers aged 2-5 and 8% of 6-11 year-olds were classified as obese in 2004.

While the small sample size of obese children aged 2-11 in 1978/79 does not allow a statistically significant trend assessment using measured data for this age group, it is highly likely that a significant proportion of Canadian children and youth of all ages moved from the overweight to the obese category during this 25-year time period. Thus, for example, the unchanged 21% proportion of 2-5 year-olds who were either overweight

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or obese both in 1978/79 and 2004 likely conceals the reality that average BMI within this age group may have increased sharply in this time period, and that a higher proportion of Canadian infants, toddlers, and children, as well as adolescents, were likely at heightened risk in 2004 of serious health problems both in childhood and subsequent adulthood than faced such risks 25 years earlier.

Shields notes that the "most pronounced increases were in the proportions of 12- to 17-year-olds whose BMI exceeded 25 or 30, the overweight and obese thresholds for adults. This is particularly important, given that adolescence is a critical period for the development of adult obesity."[166]

Figure 17. Measured overweight and obesity rates for children and youth, by age group, aged 2–17, Canada 1978/79 and 2004
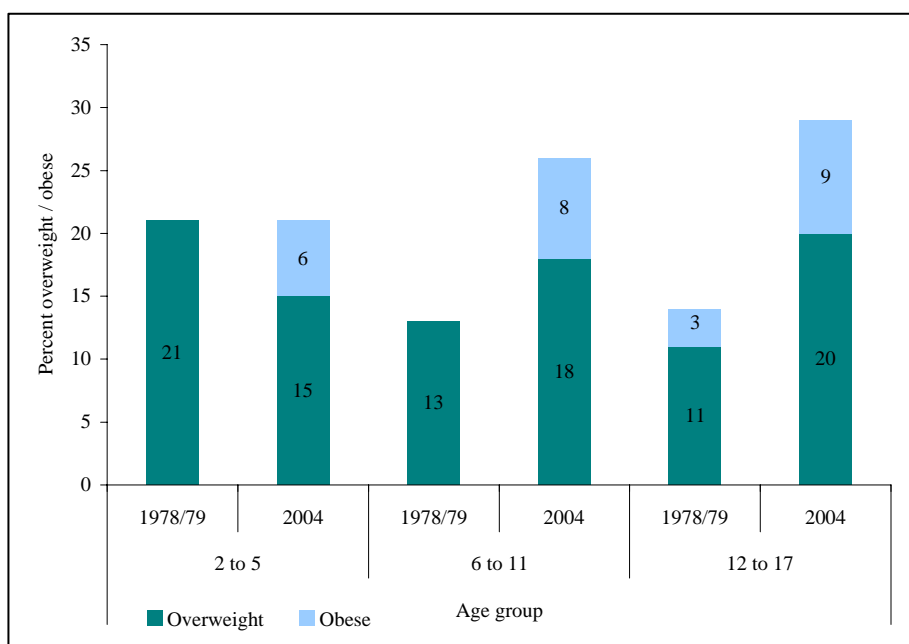
Notes: The obesity and overweight rates have been combined in the 1978/79 results for 2 to 11 year olds because of small sample sizes. 1978/79 directly measured data are from the Canada Health Survey, and 2004 directly measured data are from CCHS Cycle 2.2.

Source: Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol.17, no. 3: 27-42.

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[166] Shields. "Overweight and Obesity among Children and Youth." p. 31.

In a separate report, Shields also compared the 2004 measured overweight and obesity rates for children and youth, aged 2–17, by province.[167] As seen in Figure 18 below, 26.3% of Canadian children and youth were either overweight or obese, and 8.2% were obese. Alberta had the lowest combined rate of childhood and youth overweight and obesity in the country (21.8%), with 14.3% of Albertan children and youth overweight and another 7.5% obese.[168] The highest rates of childhood and youth overweight and obesity (combined) were in Newfoundland and Labrador (35.6%), New Brunswick (34.2%), and Nova Scotia (32.0%), and the highest rates of childhood and youth obesity were in Newfoundland and Labrador (16.6%) and New Brunswick (13.1%).

Figure 18. Child measured overweight and obesity rates by province, aged 2–17, 2004
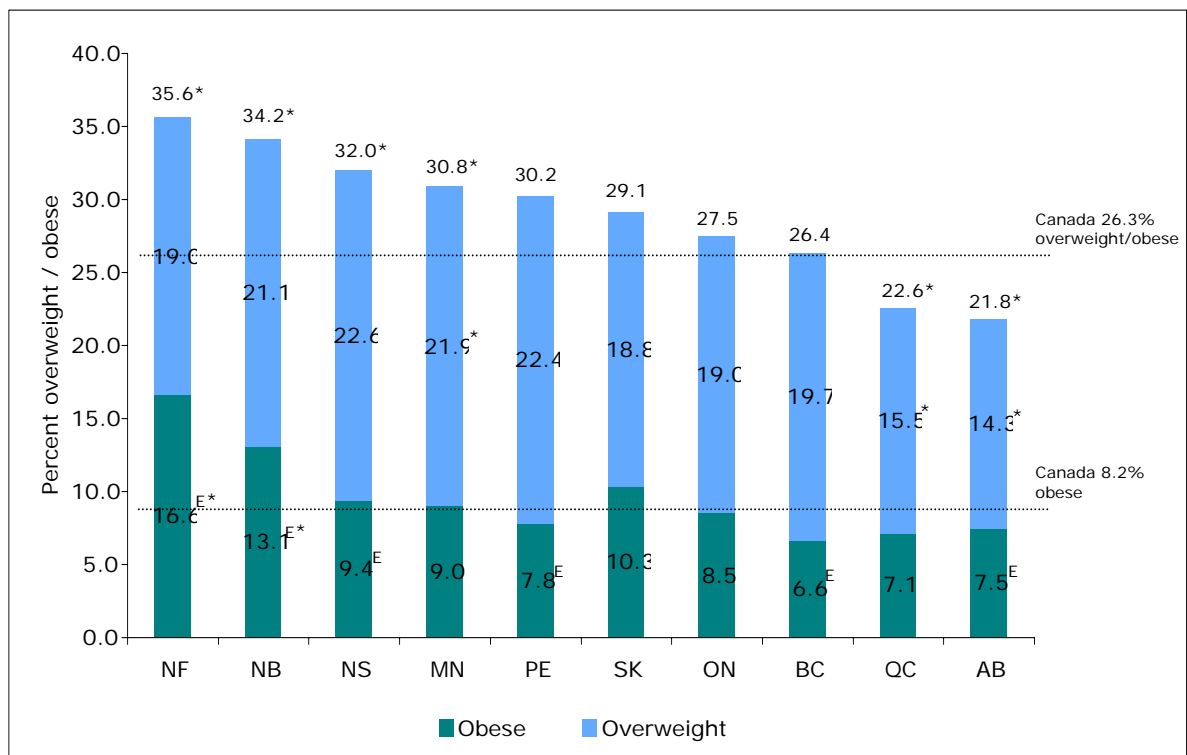
Note: E = Coefficient of variation 16.6% to 33.3% (interpret with caution). * Significantly different from estimate for Canada.

Source: Shields, Margot. *Nutrition: Findings from the Canadian Community Health Survey: Measured Obesity. Overweight Canadian Children and Adolescents*, Statistics Canada, Catalogue

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[167] Shields, Margot. *Nutrition: Findings from the Canadian Community Health Survey: Measured Obesity. Overweight Canadian Children and Adolescents*, Statistics Canada, Catalogue no. 82-620-MWE2005001, 2005; accessed June 2008; available from http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/cobesity.pdf.

[168] Percentages may not sum to 100% due to rounding or statistical aggregation methods.

Parallel to their study of regional patterns of obesity among adults, Shields and Tjepkema also attempted to correlate overweight and obesity rates among children and youth with region of residence.[169] Unlike adult patterns, however, which showed an inverse correlation with size of town or city—with larger centres generally having lower rates of overweight and obesity than smaller towns and rural areas (see Section 3.3.2.2 above), Shields and Tjepkema did not find that measured overweight and obesity rates among children and youth were significantly associated with urban or rural residence.

Thus, Shields and Tjepkema found that the average Canadian rates for childhood and youth overweight and obesity were 25.8% in urban or census metropolitan areas (CMAs), and 27.0% in non-CMAs. However, they note that Alberta is the only province that did have significant differences—with children and youth significantly more likely to be overweight or obese in non-CMAs (26.9%), than in CMAs (18.6%). The rates for Calgary and Edmonton were 16.0% and 21.4% respectively.

Shields also analysed the 2004 CCHS data to correlate rates of childhood and youth overweight and obesity with selected characteristics that have a known relationship with BMI—daily fruit and vegetable consumption, physical activity, daily and weekly hours of screen time, household income, highest level of education in the household, and self-perceived very good or excellent health. The results are reproduced in Figures 19–24 below. Shields notes that the results persisted when socioeconomic status was taken into account.

Not surprisingly, Shields found a very robust association between diet and weight among children and youth—with those consuming fewer fruits and vegetables more than twice as likely to be overweight and more than three times as likely to be obese than those who ate more fruits and vegetables. Thus, of the 59% of children and youth, aged 2 to 17, who consumed fruits and vegetables less than 5 times a day, 38% were overweight and 19% were obese. Among the 41% who ate fruits and vegetables 5 or more times per day, 17% were overweight, and 6% were obese.

More surprisingly, Shields did not find a statistically significant association between physical activity levels and rates of overweight or obesity among boys and girls aged 6 – 11 and among girls aged 12–17. Among the 26% of 12-17 year-old boys who were sedentary, however, 13% were overweight and 16% were obese—compared to 24% overweight and 9% obese among the 74% of 12-17 year-old boys who were active or moderately active. While higher rates of obesity among the sedentary adolescent boys were expected, Shields notes that their lower rates of overweight were "unexpected." Indeed, when the overweight and obesity categories are combined, it is seen that more

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[169] Shields, Margot, and Michael Tjepkema. "Regional Differences in Obesity," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol. 17, no. 3: 61-67.

active and moderately active adolescent boys were either overweight or obese (33%) than sedentary boys (29%) in that age group.[170]

Shields also analysed the relationship between overweight, obesity, and "screen time,"— or the amount of time spent watching television, playing video games, and using the computer. She reports that it is difficult to establish trends in screen time because of the recent proliferation of computers and video games, while earlier data are confined to television viewing alone. For example, both 1978/79 and 2004 data showed average weekly television hours to be about 10 for children and youth aged 12-17, but the screen time doubled to 20 hours in 2004 when using a computer and playing video games were included. [171]

Shields found a strong correlation between amount of screen time and rates of overweight and obesity. Thus, the 36% of children, aged 6–11, who had more than two hours of daily screen time in 2004 were nearly twice as likely to be overweight or obese (35%) than the 21% who had one or less hours of daily screen time (18%).

Screen time for youth, aged 12–17, was measured on a weekly, rather than daily, basis. Results for this age group also showed overweight/obesity rates rising with the amount of time spent in front of the screen—from 23% in the group that clocked less than 10 hours per week of screen time to 35% in the group having 30 or more hours of screen time per week.

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[170] Shields. "Overweight and Obesity among Children and Youth."

[171] Shields did not specify whether "using a computer" included ALL computer uses including schoolwork or if computer use was confined to pleasure uses (e.g. internet chatting, facebook, play). Therefore, it is assumed that "using a computer" implies all uses.

Figure 19. Percentage overweight or obese, by daily fruit and vegetable consumption, aged 2 to 17, Canada, 2004
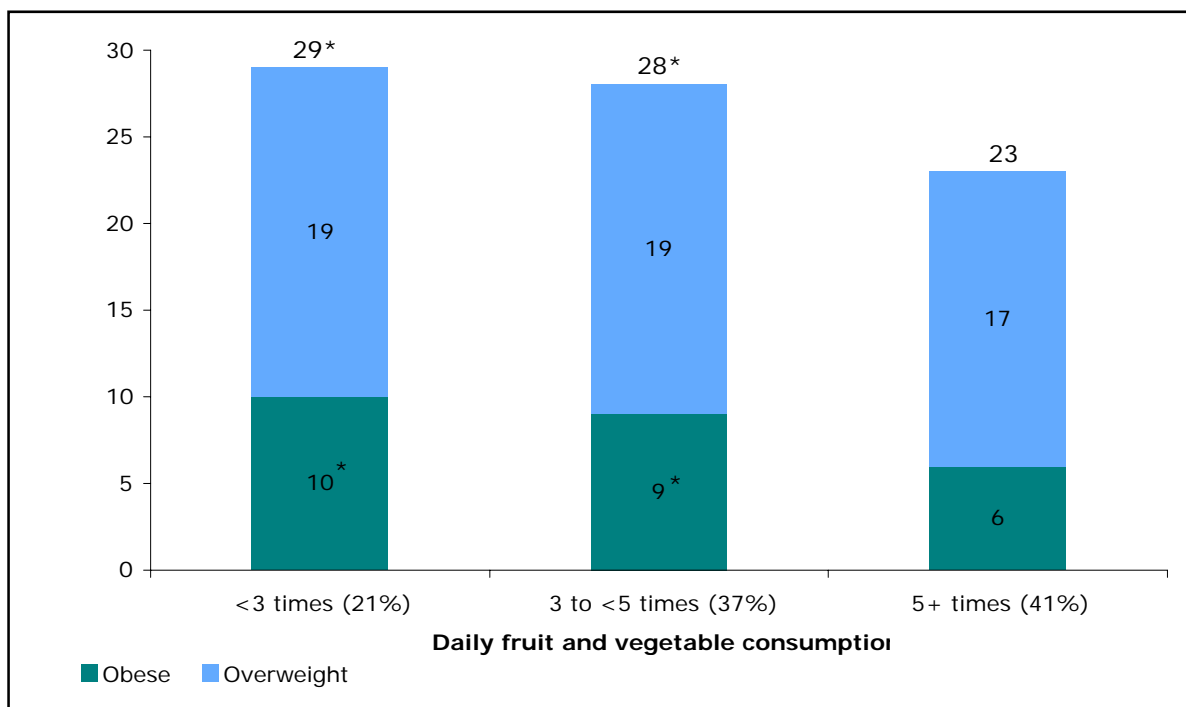
Notes: Data are from 2004 Canadian Community Health Survey (CCHS), cycle 2.2, Nutrition; * Significantly different from estimate for 5 or more times.

Source: Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol.17, no. 3: 27-42.

Figure 20. Percentage overweight or obese, by weekly hours of physical activity, aged 6 to 11, Canada, 2004
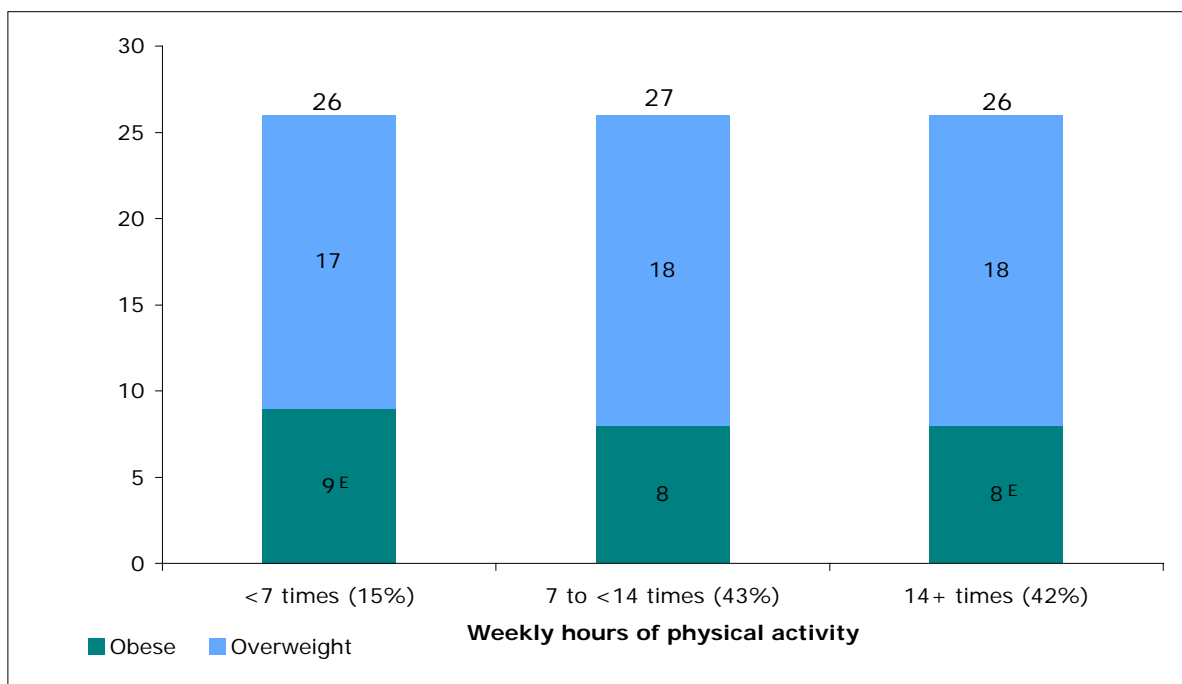
Notes: Data are from 2004 Canadian Community Health Survey (CCHS), cycle 2.2, Nutrition; E = Coefficient of variation 16.6% to 33.3% (interpret with caution).

Source: Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol.17, no. 3: 27-42.

Figure 21. Percentage overweight or obese, by gender and leisure-time physical activity level, household population aged 12 to 17, Canada, 2004
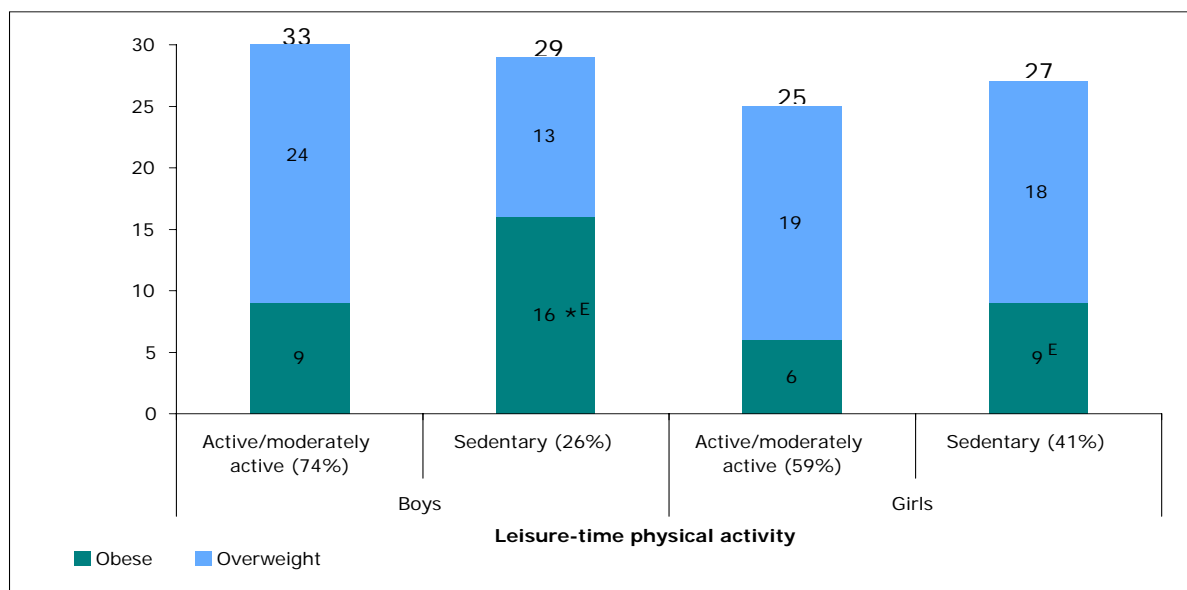
Notes: Data are from 2004 Canadian Community Health Survey (CCHS), cycle 2.2, Nutrition; * Significantly different from estimate for active/moderately active; E = Coefficient of variation 16.6% to 33.3% (interpret with caution).

Source: Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol.17, no. 3: 27-42.

Figure 22. Percentage overweight or obese, by daily hours of screen time, aged 6 to 11, Canada, 2004
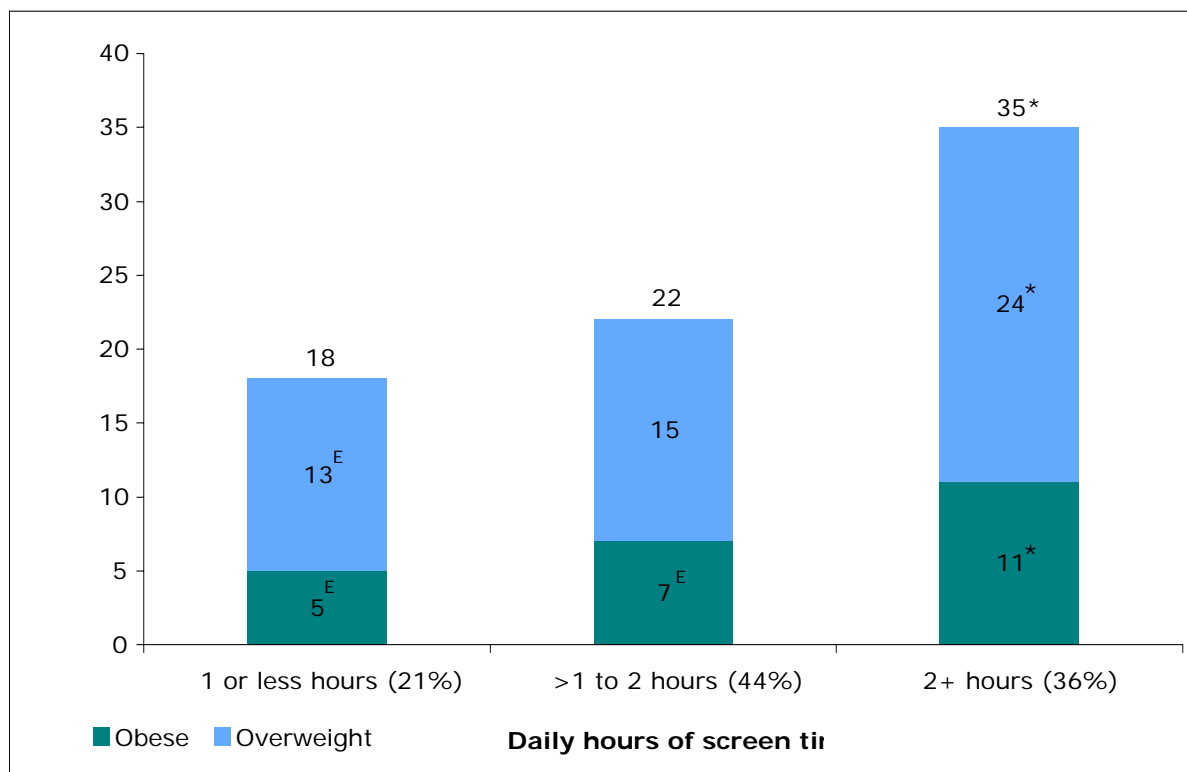
Notes: Data are from 2004 Canadian Community Health Survey (CCHS), cycle 2.2, Nutrition; * Significantly different from estimate for 1 or less category; E = Coefficient of variation 16.6% to 33.3% (interpret with caution).

Source: Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol.17, no. 3: 27-42.

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Figure 23. Percentage overweight or obese, by weekly hours of screen time, aged 12 to 17, Canada, 2004
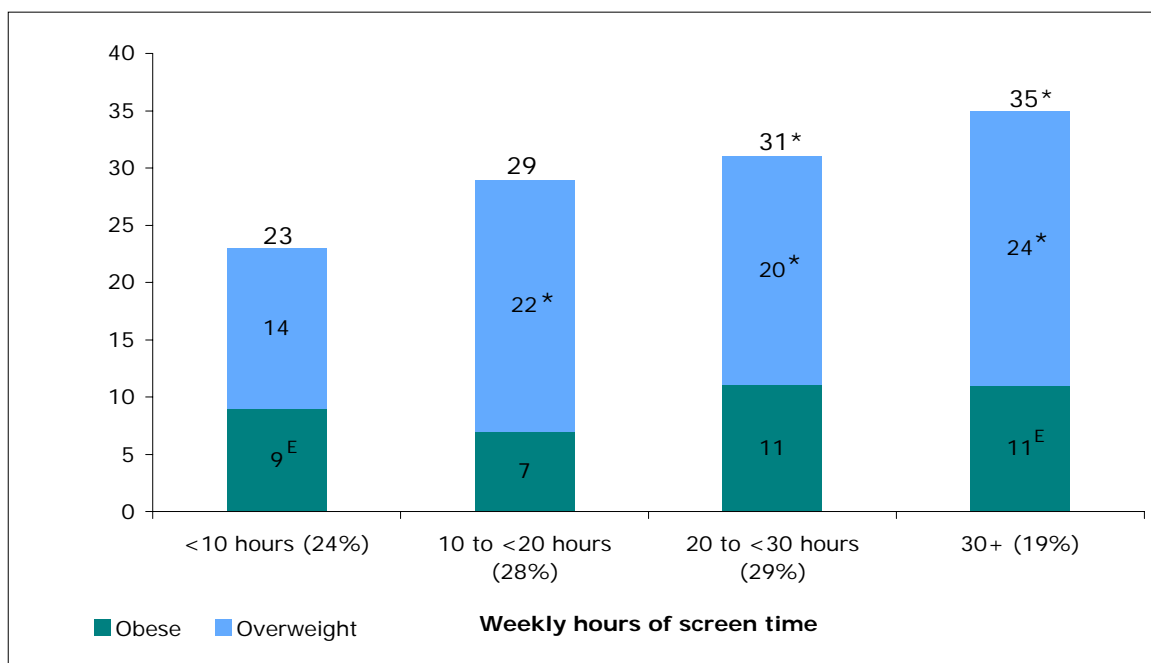
Notes: Data are from 2004 Canadian Community Health Survey (CCHS), cycle 2.2, Nutrition; * Significantly different from estimate for less than 10 hours; E = Coefficient of variation 16.6% to 33.3% (interpret with caution).

Source: Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol.17, no. 3: 27-42.

According to Shields, research results do not show a consistent association between childhood and youth obesity and socioeconomic status. She found that, in 2004, children and youth aged 2-17 in middle-income households were more likely to be obese (10%) than those in low- and high-income households, which both had 6% obesity rates among children and youth.

However, Shields found a much clearer inverse association between the formal educational attainment of household members and the propensity of children and youth to be overweight or obese. Thus, children and youth living in households where no members had graduated from secondary or high school were significantly more likely to be overweight or obese (31%) than those living in households where the highest level of education was postsecondary graduation (25%).

Figure 24. Percentage overweight or obese, aged 2-17, by household income and highest level of education in household, Canada, 2004
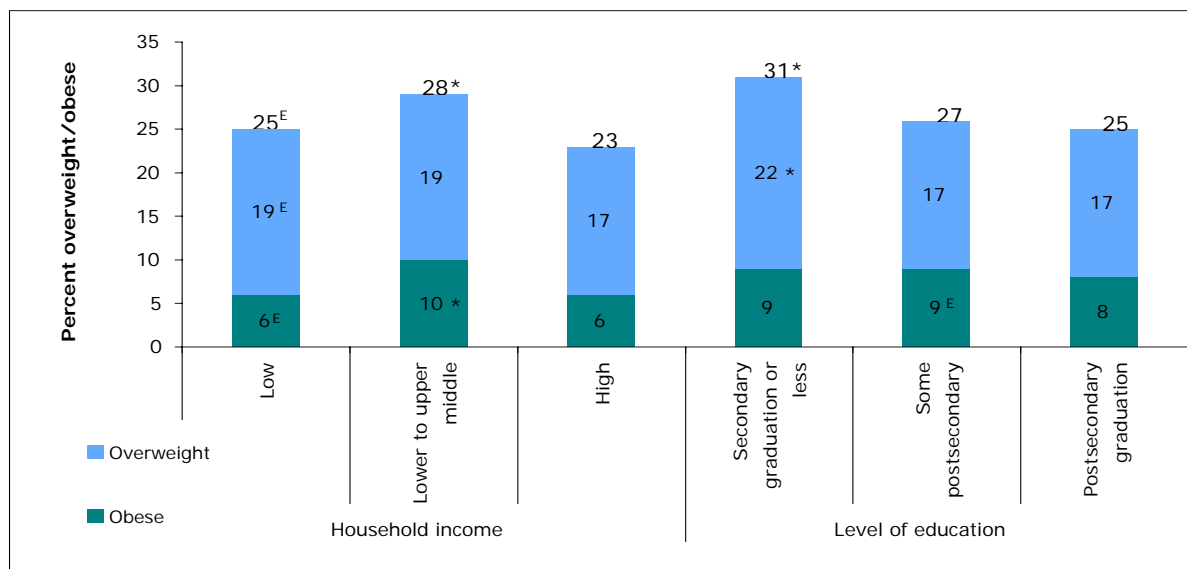
Notes: Data are from 2004 Canadian Community Health Survey (CCHS), cycle 2.2, Nutrition; * Significantly different from estimate for high household income/postsecondary graduation; E = Coefficient of variation 16.6% to 33.3% (interpret with caution).

Source: Shields, Margot. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol.17, no. 3: 27-42.

Finally, 2004 data for youth aged 12–17 did not show a significant association between BMI and a self-reported, diagnosed chronic condition, although 18% of the youth reported that they had at least one diagnosed chronic condition. This lack of association in childhood and youth is likely due to the reality that the chronic illnesses associated with obesity (including diabetes, heart disease, stroke, colon cancer, hypertension, and gallbladder disease) mostly take time to develop and manifest later in life.

However, BMI did affect young people's perceptions of their health. Thus, girls aged 12 to 17 who were overweight or obese were significantly less likely to report that their health was very good or excellent (61% of overweight and 45% of obese) than girls with normal weight (74%). For boys aged 12 to 17 only those in the obese category were significantly less likely to report very good or excellent health (52%) than boys of normal weight (77%) and those who were overweight (70%).

Concluding her analysis of childhood and youth obesity, Shields notes:

> The burden that childhood obesity places on the health care system is difficult to

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quantify because the related physical health problems are usually not evident until later in life. Nonetheless, the upturn in the prevalence of overweight/obesity among young people is important because excess weight in adolescence often persists into adulthood. Longitudinal data indicate that once an adult is overweight, further weight gain is likely and very few lose enough weight to return to the normal weight range.[172]

3.4.2 Alberta obesity prevalence and trends for children and youth

Data on trends in overweight and obesity among Alberta's children and youth are not readily available. The 2007 Alberta Health and Wellness report, *Health Trends in Alberta,* notes that these data have only recently been collected at the national and provincial levels, and that most of the available data are for self-reported measures.[173] The Alberta report uses parent-reported data from the National Longitudinal Survey of Children and Youth (NLSCY) to illustrate trends in overweight among Alberta children aged 10–11 only. Results are reproduced in Figure 25 below, which appears to show that overweight among Alberta children aged 10–11 rose from 17.7% in 1994/95 to 29.0% in 2000/01. However, the report qualifies these results with a major caveat:

> Despite the appearance of a rising trend, the confidence intervals reveal that no time trend exists during this time period. Because this data represents only a short time frame, and is based on self-report, trends cannot be inferred…. Due to sampling variability and small sample sizes of provincial data, regional health authority comparisons cannot be made for childhood overweight or obesity.[174]

An earlier report from Alberta Health and Wellness, *Alberta Child Health Surveillance Report 2005*, also concludes that trends in overweight among Albertan children, aged 2–3 and 10–11, could not be established. Like the later 2007 Alberta Health and Wellness study, the 2005 report confirms that data reported for a limited time period (i.e., 1994–2001) "can make trends difficult to discern," and that "recent increasing trends may apply to directly measured BMI rather than self-reported (or parent-reported) data."[175]

The 2005 Alberta Health and Wellness report also provides data from the NLSCY for average BMI for children aged 10–11. As seen in Figure 25 below, the average BMI of Albertan 10-11 year-olds ranged from 18.2 in 1994/95 to 18.8 in 2000/01. Neither the 2005 nor the 2007 report provided overweight rates or trends among Albertan children in

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[172] Shields. "Overweight and Obesity among Children and Youth." p. 38.
[173] Alberta Health and Wellness Project Team. *Health Trends in Alberta*, Surveillance and Environmental Health Branch, Alberta Health and Wellness, 2007; accessed July 2008; available from http://www.health.alberta.ca/resources/publications/trends_2007.pdf.
[174] Ibid., accessed. p. B-14.
[175] Child Health Surveillance Project Data Group. *Alberta Child Health Surveillance Report 2005*, Alberta Health and Wellness, 2005; accessed August 2008; available from http://www.health.alberta.ca/resources/publications/ACH_Surveillance.pdf. p. 57.

age groups other than 10- to 11-year-olds.

Figure 25. Overweight children aged 10–11, Alberta, 1994/95–2000/01, based on parent-reported data from NLSCY
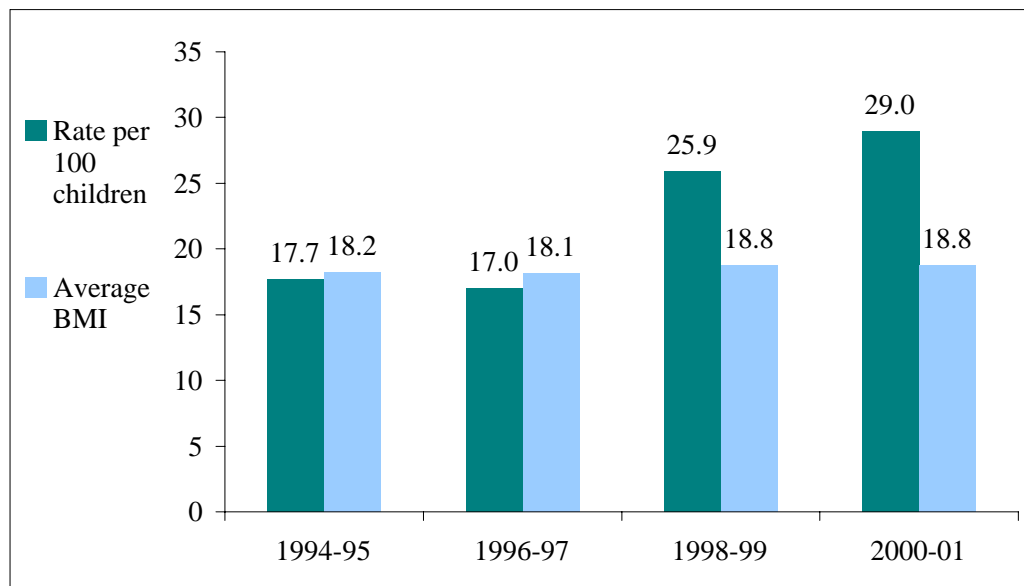
Note: NLSCY – National Longitudinal Survey of Children and Youth, Cycles 1 – 4, Statistics Canada, 2005; According to Alberta Health and Wellness (see first source below): "Despite the appearance of a rising trend, the confidence intervals reveal that no time trend exists during this time period. Because this data represents only a short time frame, and is based on self-report, trends cannot be inferred."

Sources: Adapted from: Alberta Health and Wellness Project Team. *Health Trends in Alberta*, Surveillance and Environmental Health Branch, Alberta Health and Wellness, 2007; accessed July 2008; available from http://www.health.alberta.ca/resources/publications/trends_2007.pdf. Average BMI data retrieved from: Child Health Surveillance Project Data Group. *Alberta Child Health Surveillance Report 2005*, Alberta Health and Wellness, 2005; accessed August 2008; available from http://www.health.alberta.ca/resources/publications/ACH_Surveillance.pdf.

Figure 26 below, which is based on data shown in Table 11 below (which also includes data by gender), shows Canadian and Albertan rates for overweight, obesity, and 'neither overweight nor obese' for children aged 2-11 and youth aged 12-17. using measured BMI data from the 2004 CCHS, Cycle 2.2. It is seen that considerably fewer Albertan children, aged 2–11, are overweight or obese than Canadian children. While the differences in overweight rates, and also in the overweight and obesity rates combined, is significant, the difference in obesity rates alone is not statistically significant given the wide confidence interval for the Albertan data (see Table 11 below).

Figure 26 seems to indicate that Albertan youth aged 12-17 have slightly higher rates of overweight and obesity than the Canadian average. However the high confidence interval for

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these Albertan data (see Table 11 below) renders the difference for youth statistically insignificant. Thus, the 2004 measured CCHS data show that 83.9% of Albertan children aged 2–11 were neither overweight nor obese, compared to just 75.8% of children in Canada as a whole. In Alberta, 10.3% of children in this age group were overweight (10.7% of males and 9.8% of females), compared to 16.9% (15.5% of males and 18.2% of females) in Canada. Bearing in mind the caveat on lack of statistical significance noted above, the data seem to indicate that 5.8% of Albertan 2-11 year-olds were obese (male and female rates were suppressed due to the small sample size and high coefficient of variation) compared to 7.4% (7.7% of males and 7.1% of females) of Canadian 2-11 year-olds.

In 2004, 20.2% of Alberta youth, aged 12–17, were overweight (17.8% of males and 23.3% of females), 10.1% were obese (gender rates suppressed), and 69.7% were neither overweight nor obese (74.3% of males and 63.9% of females). In Canada, 19.8% of youth aged 12–17 were overweight (21.1% of males and 18.3% of females), 9.4% were obese (11.1% of males and 7.4% of females, and 70.8% were neither overweight nor obese (67.7% of males and 74.2% of females). As noted above, the small sample size and high coefficient of variation for the Albertan data render the very small difference with Canadian results statistically insignificant for this age group.

Figure 26. Measured child and youth BMI, by age group, aged 2 to 17, Canada and Alberta, CCHS, 2004
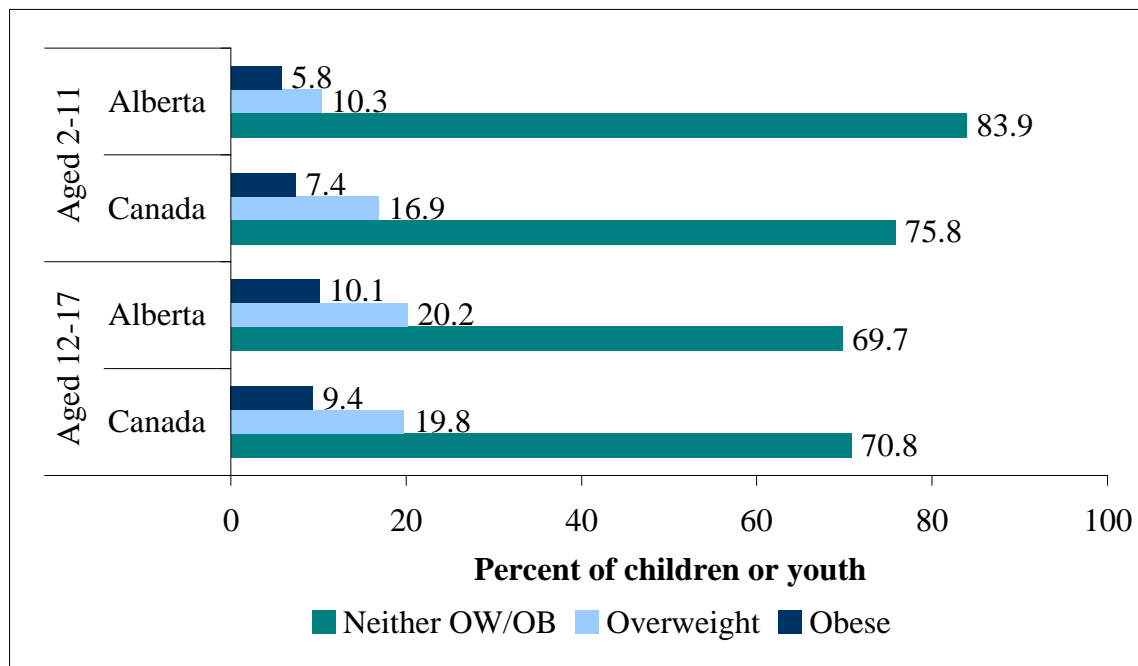
Note: OW – Overweight; OB – Obese
Source: Statistics Canada. Measured Child Body Mass Index (BMI), by Age Group and Sex, Household Population Aged 2 to 17 Excluding Pregnant Females, Canadian Community Health Survey Cycle 2.2, Canada and Provinces, Occasional, CANSIM Table 105-2002, 2007.

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Table 11. Measured child and youth BMI, by age group and gender, aged 2 to 17, Canada and Alberta, CCHS, 2004

Area and age	Gender	TOTAL Number of persons	Neither number	Neither %	Overwt number	Overwt %	Obese number	Obese %
Canada								
2 TO 17	BOTH	6,184,425	4,561,372	73.8	1,116,840	18.1	506,213	8.2
CI				72.3 - 75.2		16.8 - 19.3		7.3 - 9.1
	Male	3,177,843	2,320,807	73.0	567,963	17.9	289,073	9.1
CI				70.7 - 75.4		16.0 - 19.8		7.7 - 10.5
	Female	3,006,582	2,240,565	74.5	548,877	18.3	217,139	7.2
CI				72.4- 76.6		16.4 - 20.1		6.0 - 8.4
2 to 11	BOTH	3,669,733	2,780,278	75.8	619,039	16.9	270,416	7.4
CI				73.9 - 77.6		15.2 - 18.5		6.2 - 8.6
	Male	1,857,760	1,426,706	76.8	288,868	15.5	142,186	7.7
CI				73.9 - 79.7		13.2 - 17.9		5.8 - 9.5
	Female	1,811,974	1,353,572	74.7	330,172	18.2	128,230	7.1
CI				72.0 - 77.4		15.8 - 20.7		5.4 - 8.7
12 TO 17	BOTH	2,514,691	1,781,094	70.8	497,800	19.8	235,796	9.4
CI				68.5 - 73.1		17.8 - 21.8		7.9 - 10.9
	Male	1,320,083	894,101	67.7	279,095	21.1	146,887	11.1
CI				64.4 - 71.1		18.3 - 24.0		8.8 - 13.4
	Female	1,194,608	886,993	74.2	218,705	18.3	88,910	7.4
CI				71.1 - 77.4		15.6 - 21.0		5.6 - 9.3
Alberta								
2 TO 17	BOTH	669,385	523,224	78.2	95,785	14.3	50,376[E]	7.5[E]
CI				74.5 - 81.8	–	11.3 - 17.3	–	4.7[E]-10.3[E]
	Male	349,427	276,289	79.1	48,110	13.8	25,028[E]	7.2[E]
CI				73.4 - 84.7	–	9.5 - 18.0	–	3.3[E]-11.0[E]
	Female	319,958	246,935	77.2	47,676	14.9	25,347[E]	7.9[E]
CI				72.0 - 82.3	–	10.6 - 19.2	–	3.9[E]-11.9[E]
2 to 11	BOTH	398,396	334,447	83.9	40,959[E]	10.3[E]	22,990	5.8[E]
CI				79.6 - 88.3	–	6.7[E]-13.8[E]	–	2.4[E]- 9.2[E]
	Male	199,378	164,798	82.7	21,425[E]	10.7[E]	F	F
CI				75.7 - 89.6	–	5.4[E] - 16.1[E]	–	F
	Female	199,018	169,649	85.2	19,534[E]	9.8[E]	F	F
CI				79.5 - 91.0	–	5.4[E] - 14.2[E]	–	F
12 TO 17	BOTH	270,989	188,778	69.7	54,826	20.2	27,385[E]	10.1[E]
CI				62.9 - 76.4	–	14.5 - 25.9	–	5.5E-14.7[E]
	Male	150,049	111,492	74.3	26,685[E]	17.8[E]	11,873[E]	F
CI				65.8 - 82.8	–	10.6[E]-25.0[E]	–	F
	Female	120,940	77,286	63.9	28,142[E]	23.3[E]	15,512[E]	12.8[E]
CI				53.2 - 74.6	–	14.5[E]-32.0[E]	–	4.5[E]-21.2[E]

Notes: F – Too unreliable to be published: Data with a coefficient of variation greater than 33.3% were suppressed by Statistics Canada due to extreme sampling variability. E – use with caution (— data with a coefficient of variation from16.6% to 33.3%). CI – 95% confidence interval (low – high); Neither – Neither overweight nor obese.

Source: Statistics Canada. Measured Child Body Mass Index (BMI), by Age Group and Sex, Household Population Aged 2 to 17 Excluding Pregnant Females, Canadian Community Health Survey Cycle 2.2, Canada and Provinces, Occasional, CANSIM Table 105-2002, 2007.

3.5 Aboriginal peoples

Data on Aboriginal peoples are extremely limited, although ongoing work in Canada will hopefully remedy this situation in the future. For example, Statistics Canada's general population health surveys such as the CCHS do not include Aboriginal peoples living in the territories or on reserves. This means that more than half of Canada's Aboriginal population is not represented by these data. Given these large data gaps, there is currently limited ability to ascertain the extent, type, and magnitude of the obesity-related health conditions affecting Canada's Aboriginal population as a whole.

The 2004 CCHS is presently the best available source to assess the prevalence of measured obesity at least in the *off-reserve* population. Therefore, the costs of obesity for the off-reserve population are included in the total costs of obesity for Alberta. However, because of data limitations, it was not possible to estimate the costs of obesity specifically for Alberta's Aboriginal population. Information on the Aboriginal population is included here to flag the importance of increasing obesity in this population and to point to the need for future assessments of Aboriginal people in Alberta in relation to the costs of obesity.

3.5.1 Aboriginal population in Alberta

According to the 2006 Canada Census, the total Aboriginal population in Alberta in that year was 188,365 persons—91,740 male and 96,625 female—which is about 5.8% of the total Alberta population of 3,256,360 (1,630,870 male and 1,625,485 female), and 14% of the total Canadian Aboriginal population.[176, 177] Of the total Aboriginal population in Alberta, 41,275 live on reserves.[178]

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[176] Statistics Canada. *Aboriginal Population Profile, 2006 Census: Alberta*, 2008; accessed August 2008; available from http://www12.statcan.ca/english/census06/data/profiles/aboriginal/Details/Page.cfm?Lang=E&Geo1=PR&Code1=48&Geo2=PR&Code2=01&Data=Count&SearchText=Alberta&SearchType=Begins&SearchPR=01&B1=All&GeoLevel=&GeoCode=48. These data include the Aboriginal population living both on and off-reserve. However, Statistics Canada notes that three reserves in Alberta were incompletely enumerated: Little Buffalo, Saddle Lake 125, and Tsuu T'ina Nation 145 (Sarcee 145).

Table 12 below shows the Alberta Aboriginal population numbers by age group, and Figure 27 below illustrates these age group data graphically. Table 12 shows that over 40% of Alberta's Aboriginal population is below the age of 20.

Table 12. Alberta Aboriginal population, by age group, Census, 2006

Age	Population	Age	Population
0 to 4	18,330	45 to 49	11,295
5 to 9	19,325	50 to 54	9,140
10 to 14	20,965	55 to 59	6,485
15 to 19	19,655	60 to 64	4,210
20 to 24	16,545	65 to 69	2,795
25 to 29	14,905	70 to 74	2,165
30 to 34	13,835	75 to 79	1,250
35 to 39	13,140	80 to 84	600
40 to 44	13,390	85 +	315
TOTAL Alberta Aboriginal population: 188,365			

Source: Statistics Canada. *Aboriginal Population Profile, 2006 Census: Alberta*, 2008; accessed August 2008; available from http://www12.statcan.ca/english/census06/data/profiles/aboriginal/Details/Page.cfm?Lang=E&Geo1=PR&Code1=48&Geo2=PR&Code2=01&Data=Count&SearchText=Alberta&SearchType=Begins&SearchPR=01&B1=All&GeoLevel=&GeoCode=48.

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[177] Statistics Canada. "Aboriginal Peoples in Canada in 2006: Inuit, Metis and First Nations, 2006 Census." *The Daily*, January 15, 2008, accessed August 2008; available from http://www.statcan.ca/Daily/English/080115/d080115a.htm.
[178] Statistics Canada. *Aboriginal Identity (8), Area of Residence (6), Age Groups (12) and Sex (3) for the Population of Canada, Provinces and Territories, 2006 Census of Population*, Catalogue no. 97-558-XCB2006006, 2008; accessed August 2008; available from http://www12.statcan.ca/english/census06/data/topics/RetrieveProductTable.cfm?Temporal=2006&PID=89121&METH=1&APATH=3&PTYPE=88971&THEME=73&AID=&FREE=0&FOCUS=&VID=0&GC=99&GK=NA&RL=0&d1=0&d2=0&GID=614144.

Figure 27. Alberta Aboriginal population by age group, Census, 2006
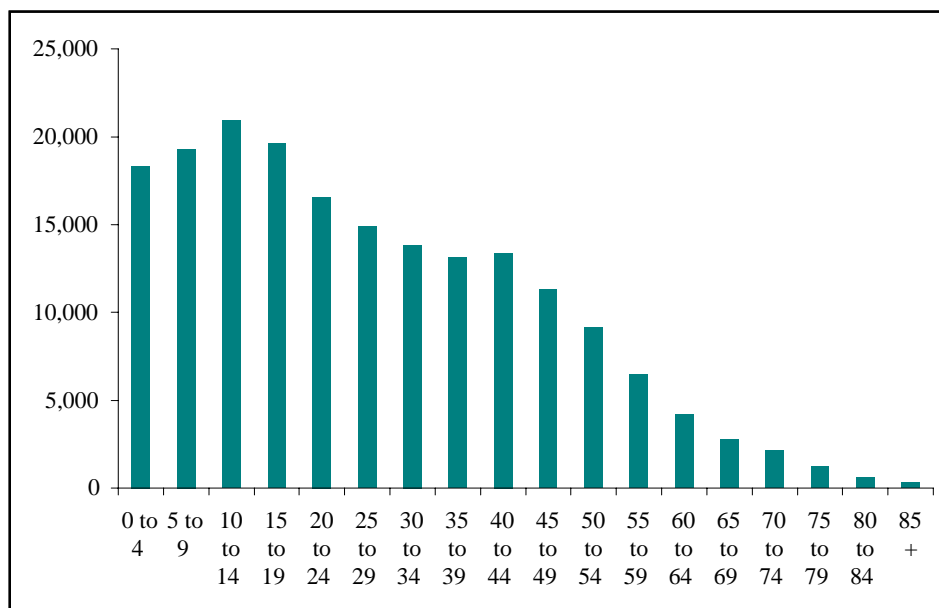
Source: Statistics Canada. *Aboriginal Population Profile, 2006 Census: Alberta*, 2008; accessed August 2008; available from http://www12.statcan.ca/english/census06/data/profiles/aboriginal/Details/Page.cfm?Lang=E&Geo1=PR&Code1=48&Geo2=PR&Code2=01&Data=Count&SearchText=Alberta&SearchType=Begins&SearchPR=01&B1=All&GeoLevel=&GeoCode=48.

3.5.2 Aboriginal adult overweight and obesity prevalence

The most inclusive health data on Aboriginal peoples are available from the First Nations Regional Longitudinal Health Survey (RHS), which surveys First Nations and Inuit peoples living both on- and off-reserve, and is the only national health survey in Canada that is governed by First Nations peoples.[179] The First Nations people control, coordinate, administer, and own the survey. The first RHS took place in 1997 and involved First Nations and Inuit from across Canada. Data collection for the second iteration of the RHS—called RHS Phase 1 (2002/03)—began in the Fall of 2002 and was completed in mid-2003, though Inuit communities did not participate in this round. The RHS is scheduled to be repeated every four years.

Despite the importance and inclusivity of the RHS in providing health information on Canada's entire Aboriginal population—on- and off-reserve—Health Canada notes that

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[179] Assembly of First Nations / First Nations Information Governance Committee. *First Nations Regional Longitudinal Health Survey (RHS) 2002/03: Results for Adults, Youth and Children Living in First Nations Communities*, RHS National Team, 2007; accessed August 2008; available from http://www.rhs-ers.ca/english/pdf/rhs2002-03reports/rhs2002-03-technicalreport-afn.pdf.

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RHS data cannot be compared with data on non-Aboriginal peoples because of important differences between population groups and data sources in the Aboriginal and non-Aboriginal surveys.[180]

The Heart and Stroke Foundation of Canada's 2006 report, *Tipping the Scales of Progress* uses BMI data from the 2002/03 RHS data to show that 37% of Aboriginal people living on-reserve are overweight, 35% are obese, and only 27% have normal weight.[181] Also using 2002/03 RHS data, Human Resources and Social Development Canada (HRSDC) reports that just over a quarter of First Nations people living on-reserve have normal weight—25% of men and 27% of women—thus roughly confirming the Heart and Stroke Foundation results.[182] According to HRSDC, more on-reserve Aboriginal men (42%) than women (32%) are overweight, but more women (40%) than men (32%) are obese. The results point to extraordinarily high rates of obesity and overweight among on-reserve Aboriginals.

In a recent article in *Obesity,* Peter Katmarzyk of Queen's University uses directly measured BMI data for non-reserve Aboriginal people aged 2–64 years from the 2004 CCHS, Cycle 2.2.[183] The prevalence of obesity among non-reserve Aboriginal adults (37.8%) is seen to be very much higher than that of the general adult population (22.6%), with the disparity particularly large among women.

Figure 28 below compares overweight and obesity prevalence rates among non-Aboriginal and off-reserve Aboriginal adults, aged 18–64, by gender. Aboriginal men have considerably higher obesity rates (31.2%) than the non-Aboriginal male population (22.8%), but marginally lower overweight rates (39.3% compared with 40.8%, respectively). The pattern is the same, but even more marked, for women—with non-reserve Aboriginal women have very much higher obesity rates than the non-Aboriginal female population (41.6% vs. 22.4%, respectively), but somewhat lower overweight rates (26.2% vs. 28.9%, respectively).

These results also indicate that non-reserve Aboriginal women are considerably more likely to be obese than non-reserve Aboriginal men (41.6% vs 31.2%), while there is no notable gender difference in obesity rates in the non-Aboriginal population. However, for

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[180] First Nations Inuit Health Branch (FNIHB) of Health Canada is the primary funder of the RHS. Health Canada used RHS data for the section on Aboriginal health in its report titled *Healthy Canadians–A Federal Report on Comparable Health Indicators 2006*. In that report, an overview of some of the challenges of data collection in Aboriginal populations—in particular in First Nations populations living on reserves—is available in Chapter 5: Health information, challenges and next steps, and in Annex 3: Data source exclusions and limitations.

[181] Heart and Stroke Foundation of Canada. *Tipping the Scales of Progress: Heart Disease and Stroke in Canada 2006*, 2006; accessed June 2008; available from http://ww2.heartandstroke.ca/images/english/Tipping_the_Scales.pdf.

[182] Human Resources and Social Development Canada (HRSDC). *Indicators of Well-Being in Canada: Obesity*, 2008; accessed August 2008; available from http://www4.hrsdc.gc.ca/indicator.jsp?lang=en&indicatorid=6.

[183] Katzmarzyk, Peter T. "Obesity and Physical Activity among Aboriginal Canadians," *Obesity*, 2008, vol. 16, no. 1: 184-190.

both Aboriginal and non-Aboriginal populations, men are more likely to be overweight than are women.

Figure 28. Prevalence of measured overweight and obesity among non-Aboriginal and off-reserve Aboriginal adults, aged 18–64, CCHS, 2004
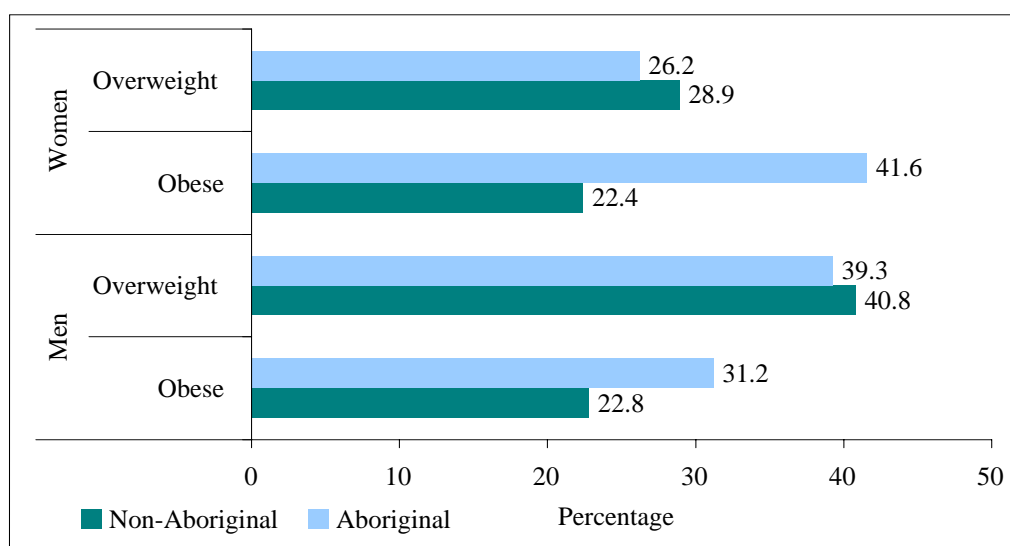
Source: Data from Canadian Community Health Survey (CCHS), Cycle 2.2, 2004. Katzmarzyk, Peter T. "Obesity and Physical Activity among Aboriginal Canadians," *Obesity*, 2008, vol. 16, no. 1: 184-190.

3.5.3 Aboriginal children and youth overweight and obesity prevalence

Using measured data from the 2004 CCHS, Shields found that 41.3% of off-reserve Aboriginal children and youth, aged 2 to 17, were overweight or obese, which is approximately 58% higher than the Canadian average (26.1%).[184] The percentage of off-reserve Aboriginal children and youth who were obese (20%) was 2.5 times the national average (8.0%).

The difference was particularly dramatic for girls—with off-reserve Aboriginal girls nearly 2.7 times as likely to be obese (18.3%) as non-Aboriginal girls (6.9%). Thus, Figure 29 below shows the 2004 CCHS prevalence of overweight and obesity for boys and girls aged 2-17 of Aboriginal and non-Aboriginal origin. For girls, both overweight and obesity rates were higher in the Aboriginal population than in the non-Aboriginal population—with nearly 40% of Aboriginal girls either overweight or obese (39.7%) compared to a quarter of non-Aboriginal girls (25.2%).

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[184] Shields. "Overweight and Obesity among Children and Youth."

Aboriginal boys were nearly 50% more likely to be obese than non-Aboriginal boys (13.4% vs 9.1%), and had the same rates of overweight (17.8%). It is seen that both obesity and overweight rates were higher among Aboriginal girls than among Aboriginal boys (18.3% vs 13.4% for obesity; 21.4% vs 17.8% for overweight).

Figure 29. Prevalence of measured overweight and obesity among non-Aboriginal and Aboriginal children and youth, aged 2–17, CCHS, 2004
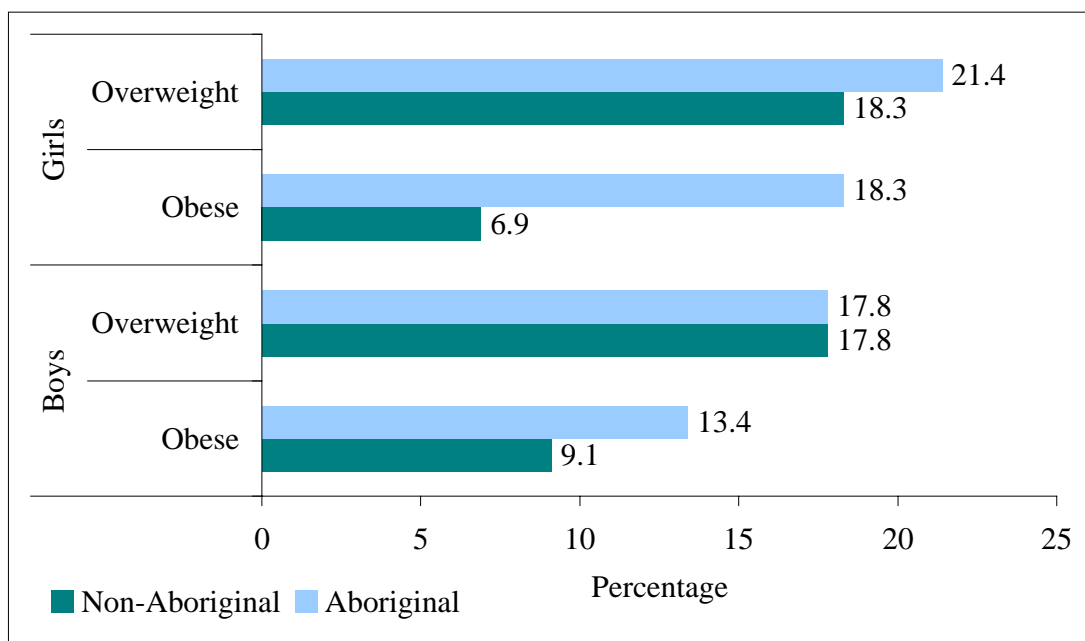
Source: Data from Canadian Community Health Survey (CCHS), Cycle 2.2, 2004; Katzmarzyk, Peter T. "Obesity and Physical Activity among Aboriginal Canadians," *Obesity*, 2008, vol. 16, no. 1: 184-190.

4. Health impacts of obesity

Obesity is associated with a wide range of chronic health conditions and premature mortality in adults, accounting for a significant impact on the health of the population, as well as increased costs to the health care system, business, and society in general.

A search of the epidemiology literature has identified the particular chronic diseases for which the strongest evidence exists that overweight or obesity is a key risk factor. These include:
- type 2 diabetes,
- cardiovascular diseases (i.e., hypertension, coronary artery disease, and cerebrovascular disease),
- 14 types of cancer,
- osteoarthritis,
- gallbladder disease,
- asthma, and
- mental illnesses like depression and anxiety.

Specifically, the types of cancer that have been found to be partially attributable to excess body weight include cancers of the
- colon,
- breast (postmenopausal),
- endometrium or uterus,
- kidney,
- esophagus,
- ovaries,
- prostate,
- pancreas,
- leukemia,
- Non-Hodgkin's lymphoma,
- multiple myeloma,
- liver,
- bladder, and
- stomach.

The above health conditions have been included in the cost estimates found in Chapter 5 of this report, and the evidence found for obesity as a risk factor in their development is discussed below. Several other health conditions, such as obstructive sleep apnea, back pain, non-alcoholic fatty liver disease, gout, certain reproductive disorders, gallbladder cancer, and more (see Table 13 below), have also been strongly associated with obesity in the literature, but these conditions were not included in the obesity cost estimates in this study because of a lack of sufficient data availability and consequent difficulties calculating reliable relative risk ratios.

Figure 30 below shows a wide range of medical complications of obesity identified in the literature. As noted, other health conditions—such as sleep apnea, back pain, dermatitis, gout, menstrual disorders, infertility, and many others[185]— are also associated with obesity, but these conditions cannot be included in the obesity cost estimates at this time because of the lack of relevant data required for cost exercises—such as data on prevalence of the condition in Canada and Alberta, unknown risk ratios linking obesity to the illness, and unknown medical costs for the specific diagnostic categories (in cases where the *Economic Burden of Illness in Canada* database subsumes those particular diagnostic categories under larger categories, for example).

According to one report from Diabetes Australia, these additional obesity-related health conditions, listed in Table 13 below but not currently included in obesity cost estimates, are nevertheless likely to result in increased personal and economic costs—a substantial portion of which are in fact attributable to overweight and obesity.[186] Indeed, it is seen in Table 13 below that only about one-third of the health conditions associated with obesity are included in the cost estimates in this study. Therefore, the overall estimate of the cost of obesity to Alberta presented in this report is likely to be conservative to the extent that these conditions are excluded, and will therefore underestimate the total cost burden of obesity.

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[185] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."
[186] Access Economics. *The Economic Costs of Obesity*, accessed.

Figure 30. Medical complications of obesity
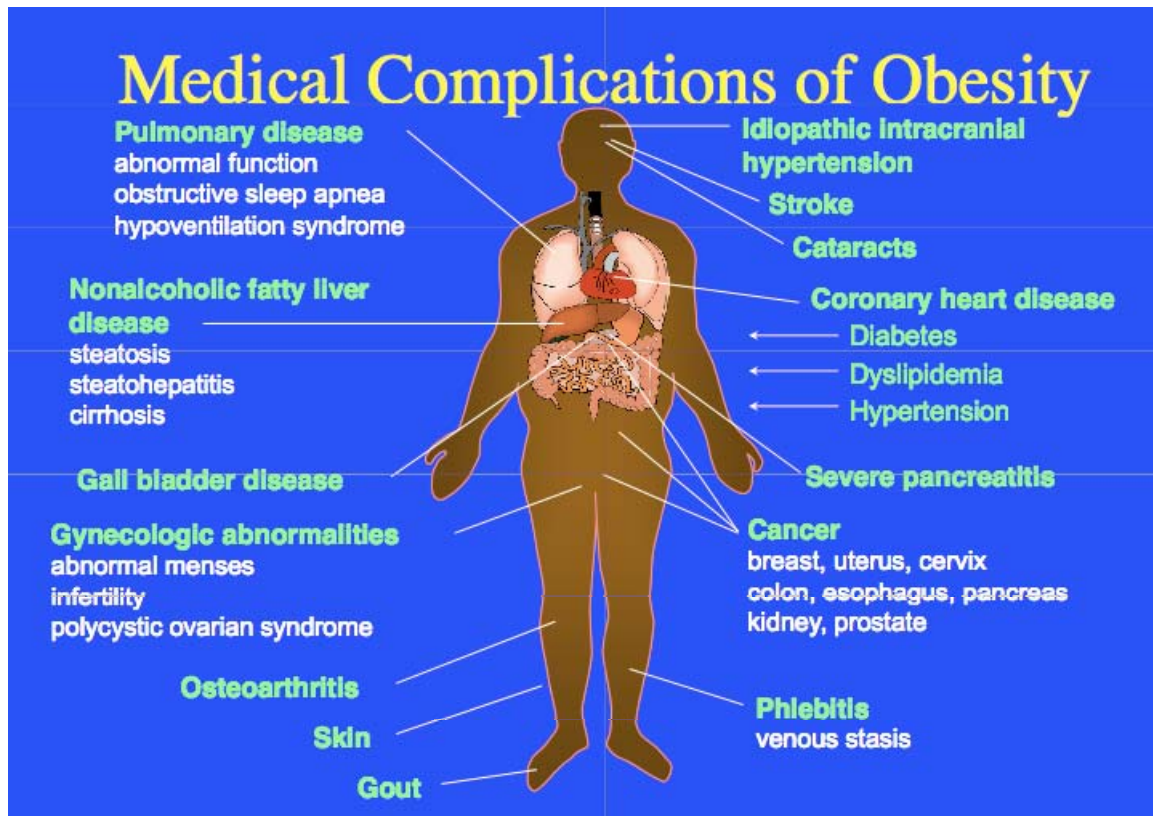
Source: Foster, Gary D. *Obesity: Causes, Consequences and Treatment*, Center for Obesity Research and Education, Temple University School of Medicine, 2008; accessed June 2008; available from http://www.globalfit.com/hrsummit/2008/pdf/Foster.pdf.

Table 13 below shows the expanded list of diseases associated with adult obesity by category of risk ratio (RR) (i.e. risk greatly increased – RR >3; risk moderately increased – RR 2–3; and risk slightly increased – RR 1–2). Thus, for example, RR 2–3 means that an obese person is 2–3 times as likely to have this condition as one with normal weight. This expanded list, which was compiled by the National Health and Research Council in Australia, is composed of the obesity-related health conditions that are included, as well as not included, in the cost estimates in this report.[187] As noted, only about one-third of the conditions listed in Table 13 below are included in the cost estimates in this study due to the data limitations cited above.

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[187] National Health and Medical Research Council (Australia). *Clinical Practice Guidelines for the Management of Overweight and Obesity in Adults*, 2004; accessed July 2008; available from http://www.health.gov.au/internet/main/publishing.nsf/Content/obesityguidelines-guidelines-adults.htm.

Table 13. Diseases associated with adult obesity, by category of relative risk ratio (RR)

Relative risk ratio (RR)	Diseases associated with obesity through metabolic consequences[188]	Other diseases associated with obesity
Greatly increased (RR >3)	Diabetes* Hypertension* Gallbladder disease* Dyslipidaemia Insulin resistence Non-alcoholic fatty liver disease	Sleep apnea Breathlessness Asthma* Social isolation and depression* Daytime sleepiness and fatigue
Moderately increased (RR 2-3)	Cardiovascular diseases* Cerebrovascular disease* Gout / hyperuricamia	Osteoarthritis* Respiratory disease Hernia Psychological problems
Slightly increased (RR 1-2)	Cancers (esp. postmenopausal breast, endometrial, colon)* Reproductive abnormalities / impaired fertility Polycystic ovaries Skin complications Cataract	Varicose veins Musculoskeletal problems Back pain Stress incontinence Oedema / cellulitis

Note: * included in this costing study
Source: Access Economics. *The Economic Costs of Obesity*, Diabetes Australia, 2006; accessed June 2008; available from
http://www.accesseconomics.com.au/publicationsreports/showreport.php?id=102.

Table 14 below shows examples of the percentages of particular diseases that can be attributed to adult obesity as assessed in two separate studies—one from Wei Luo et al. of the Public Health Agency of Canada (PHAC)[189] and one from the WHO Global Burden

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[188] See section 4.1 below where metabolic syndrome is defined as a "constellation of risk factors including central adiposity, low high-density lipoprotein-cholesterol levels, high serum triglyceride levels, increased blood pressure, and impaired fasting glucose" that in combination lead to many of the diseases associated with obesity such as diabetes and cardiovascular disease, and increase the risk for both morbidity and mortality. Ogden, Yanovski, Carroll, and Flegal. "The Epidemiology of Obesity."

[189] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

of Disease project.[190] Although the two studies are not strictly comparable because of differences in the population samples and methodologies used, the table gives a broad indication of the magnitude of the association between obesity and each of the main chronic diseases for which the obese population is at greater risk relative to the population with healthy weights.

Another way of thinking about these results is simply that these are the proportions of each illness that could potentially be avoided if all the population had healthy weights. The word 'potentially' is inserted deliberately here, as Steven Goodman of Johns Hopkins School of Medicine points out that removal of obesity does not necessarily eliminate the fraction of disease cases attributable to obesity, "one reason being that complex causal connections, such as that between obesity and mortality, are not fully understood."[191]

Despite that major caveat, it is also essential to communicate the underlying meaning of the association between obesity and particular disease outcomes to a wider public, so we use of such colloquial expressions with the insertion of the qualifier 'potentially' in order to make the case for interventions designed to promote healthy weights. As well, as noted below, I.U. Eneli et al. note that evidence of *reversibility* is also an important epidemiological criterion of causality, and that such reversibility has been demonstrated to be the case with the chronic conditions associated with obesity—namely that weight loss reduces the incidence of these conditions.[192]

Tu further describes the proportion of an illness outcome attributable to a risk factor as "the degree to which the total number of cases in a community would be reduced if the frequency of the outcome among those exposed to the risk factor was the same as those not exposed to the outcome."[193] And Mathers et al., writing for the World Health Organization, describe the proportion of disease attributable to a risk factor like obesity as "the percentage reduction in disease or death that would occur if exposure to the risk factor was reduced to zero."[194] From the perspectives of Eneli, Tu, Mathers, and others, therefore, it is indeed justifiable to interpret the results that follow as the proportion of illness cases that could potentially be avoided if the population had healthy weights.

According to Canadian health researchers Luo et al., who recently estimated the proportion of major chronic diseases attributable to obesity in Canada, the overall proportion of chronic diseases attributable to obesity increased sharply between 1970 and

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[190] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

[191] Goodman, Steven. In Institute of Medicine. *Estimating the Contributions of Lifestyle-Related Factors to Preventable Death: A Workshop Summary*, accessed. p. 8.

[192] Eneli, I .U., T. Skybo, and C.A. Camargo Jr. " Weight Loss and Asthma: A Systematic Review," *Thorax*, 2008, vol. 63: 671-676.

[193] Tu, Shihfen. "Developmental Epidemiology: A Review of Three Key Measures of Effect," *Journal of Clinical Child and Adolescent Psychology*, 2003, vol. 32, no. 2: 187-192. p. 190.

[194] Mathers, Colin, Theo Vos, Alan Lopez, Josh Salomon, and Majid Ezzati. *National Burden of Disease Studies: A Practical Guide*, Edition 2.0. Global Program on Evidence for Health Policy. Geneva: World Health Organization, 2001; accessed June 2008; available from http://www.who.int/healthinfo/nationalburdenofdiseasemanual.pdf.

2004—by 138% for men and by 60% for women, due to rising rates of obesity among Canadians.[195] Using directly measured data from the 2004 CCHS for adults aged ≥20 and stratified by gender, they found that the percentages of disease prevalence in Canada that could be attributed to obesity—called the 'population attributable fractions' or PAF—were:

- type 2 diabetes – 38.93% for both males and females;
- hypertension – 44.97% male and 45.46% female;
- coronary artery disease (CAD) – 22.45% male and 22.79% female;
- stroke – 10.45% male and 10.64% female;
- endometrial cancer – 22.08% female;
- postmenopausal breast cancer – 12.09% female;
- colon cancer – 9.51% male and 9.68% female;
- osteoarthritis – 18.78% male and 19.08% female; and
- gallbladder disease – 35.24% male and 35.68% female.

They also found that, in 2004, 8,414 premature deaths in Canada, or almost 14% of all Canadian deaths in that year, could be attributed to obesity.

Again, as noted above and with the Goodman's qualifier in mind, another way of expressing these results colloquially is simply that 45% of hypertension in this country, as well as 39% of diabetes, 22% of coronary artery disease and endometrial cancer, etc. could potentially be avoided if all Canadians had healthy weights.

In 2004 the World Health Organization (WHO) reported markedly higher obesity-attributable fractions for the U.S., Canada, and Cuba than the Canadian estimates above for all categories of illness with the exception of postmenopausal breast cancer. This is in part due to the considerably higher rates of obesity in the U.S. compared to Canada, which in turn results in higher overall proportions of these obesity-related illnesses being attributable to obesity, and in part to the fact that the WHO study was for adults aged ≥30, while the Canadian study was for adults aged ≥20.

Thus, the WHO found the percentage of diseases that could be attributed to obesity for adults aged ≥30 in the AMA-A subregion (which includes Canada, United States, and Cuba) was:

- type 2 diabetes – 83% male and 88% female;
- hypertension – 63% male and 58% female;
- coronary artery disease – 37% male and 32% female;
- stroke 40% male and 37% female;
- colon cancer – 17% male and 18% female;
- postmenopausal breast cancer – 12% female;

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[195] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

- endometrial cancer – 52% female; and
- osteoarthritis – 22% male and 24% female.[196]

A particularly striking outcome of the WHO estimates is the qualified conclusion that about 85% of type 2 diabetes cases in this region could potentially be avoided if Americans, Canadians, and Cubans had healthy weights—a finding especially noteworthy in light of the epidemic five-fold global increase in type 2 diabetes in the last two decades (from 30 million to nearly 150 million), and the fact that diabetes is a causal factor in a wide range of other serious conditions, including heart disease, kidney failure, and blindness. Indeed, at present rates of increase, the global incidence of the disease is expected to double to 300 million by the year 2025.[197]

It is also notable that—based on these WHO estimates—60% of hypertension (which is also a major causal factor in heart disease), nearly 40% of strokes, and more than a third of coronary artery disease could potentially be avoided if Americans, Canadians, and Cubans had healthy weights. With Goodman's qualifier in mind, these dramatic findings still very clearly point to the huge potential burden of obesity in this region—in premature death, human suffering, and economic costs—and to the urgency of instituting effective interventions designed to improve diet and promote healthy weights.

As noted, the sharp apparent contrast between many of the Canadian and WHO estimates may be accounted for in large part by the considerably higher U.S. obesity prevalence rate and the different age groups used in the calculations (≥ 20 in the Canadian study and ≥ 30 in the WHO study). As noted in Chapter 1, OECD figures show the United States with the highest rate of obesity in the world—with 32.2% of the population aged 15 and older with a directly measured BMI of 30 or more in 2005, compared to the 2004 directly measured rate of 22.7% for Canada. That considerable disparity as well as the much greater relative risk of developing all the listed chronic illnesses at older ages clearly explain a major part of the large differences between the two sets of study results.

However, the magnitude of some of the differences—e.g. type 2 diabetes (39% vs. 88% female and 83% male), stroke (10.5% vs. 40% male and 37% female), and others—also points to the fact that country-specific data based on directly measured height and weight are likely considerably more accurate, particularly for use in costing studies, than more general data that include several countries and reflect different collection methodologies. While the former are used in this study to estimate obesity costs in Alberta, the latter are nevertheless very useful in pointing towards the very serious potential consequences of not stemming the present obesity epidemic in a timely way.

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[196] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

[197] Gardner, Gary, and Brian Halweil, "Nourishing the Underfed and Overfed," Chapter 4 in Worldwatch Institute, *State of the World 2000,* W.W. Norton and Co., New York, 2000, p. 72; LeBlanc, John, "The Prevalence of Diabetes Mellitus in Nova Scotia: What Can We Learn from Routinely Collected Health Data," in *Diabetes Care in Nova Scotia,* October.1998, 8. (October) 4, Diabetes Care Program of Nova Scotia, Halifax.

Table 14. Proportions of particular disease prevalence attributable to adult obesity (population attributable fraction – PAF), BMI ≥30 kg/m^2, percentage, Canada and WHO–AMA-A subregion

Disease	Canada-PHAC PAF (%), aged ≥20		WHO-AMA-A PAF (%), aged ≥30	
	male	female	male	female
Type 2 diabetes	38.93	38.93	83	88
Hypertension	44.97	45.46	63	58
Coronary artery disease	22.45	22.79	37	32
Stroke	10.45	10.64	40	37
Colon cancer	9.51	9.68	17	18
Postmenopausal breast cancer	na	12.09	na	12
Endometrial cancer	na	22.08	na	52
Gallbladder disease	35.24	35.68	–	–
Osteoarthritis	18.78	19.08	22	24

Note: Obesity = BMI – Body Mass Index ≥30 kg/m^2, na – not applicable; – indicates the disease was not used in the study.

Sources: Canada – PHAC–Population Health Agency of Canada: Luo, Wei, et al. "The Burden of Adult Obesity in Canada," *Chronic Diseases in Canada*, 2007, vol. 27, no. 4: 135-144; and WHO-AMA-A – World Health Organization, Subregion Americas-A (Canada, Cuba, and United States): James, W. Phillip T., et al. "Overweight and Obesity (High Body Mass Index)," in *Comparative Quantification of Health Risks – Global and Regional Burden of Diseases Attributable to Selected Major Risk Factors*, ed. Ezzati, Majid, et al., Vol 1, Chapter 8, 497-596, World Health Organization, 2004; accessed September 2008; available from http://www.who.int/publications/cra/chapters/volume1/0497-0596.pdf.

Because many of the health impacts of obesity are chronic conditions that take time to develop and are generally seen later in life, the link between health conditions and obesity in children and youth is less clear than for adults.[198] However, there is clear evidence of increased prevalence in children and youth of type 2 diabetes, some orthopaedic complications, and psychosocial problems connected both with social stigma and the mental afflications (like depression and anxiety) that have been linked with obesity. Ample evidence now indicates that the average age of so-called "adult-onset" or type 2

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[198] Access Economics. *The Economic Costs of Obesity*, accessed.

diabetics is getting younger.[199] The health impacts related to obesity in children and youth are discussed separately in Chapter 4.13 below.

Following in this chapter are summaries of several key bodies of evidence that are relevant to (and in some cases essential for) estimates of the burden of obesity, including its economic costs. These sections include:

- a brief summary of results from some of the growing body of evidence on the physical pathways leading from obesity to chronic disease and mortality,
- the methodologies used to estimate the health impacts of obesity,
- the known health impacts of the major obesity-related diseases, and
- the prevalence of these particular diseases in Canada and Alberta.

The epidemiological literature connecting obesity with the major chronic diseases described below is vast, and the examples provided here reference only a small portion of this literature. Where possible we have attempted to reference evidence from meta-analyses that have been conducted for specific health conditions that can be partially attributable to excess weight. Because these meta-analyses in turn assemble, examine, organise, and compare evidence from a very wide range of other studies, adjusting for different variables, it is hoped that this present study ultimately draws on a sufficient body of credible, reliable, and recent epidemiological evidence to provide a reasonably accurate basis for the cost estimates provided.

4.1 Physical pathways leading from obesity to chronic disease and mortality

According to U.S. researchers Cynthia Ogden et al. of the National Center for Health Statistics, the "metabolic syndrome" is a unifying principle that somewhat explains the relationship between obesity and risk factors for chronic disease.[200] They define metabolic syndrome as a "constellation of risk factors including central adiposity, low high-density lipoprotein-cholesterol levels, high serum triglyceride levels, increased blood pressure, and impaired fasting glucose."[201] To be diagnosed as having metabolic syndrome, individuals must have three of the five features. The authors note that considering metabolic syndrome as a distinct diagnostic entity is controversial, but that the metabolic risk factors lead to many of the diseases associated with obesity such as diabetes and cardiovascular disease, and increase the risk for both morbidity and mortality.

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[199] Gardner, Gary, and Brian Halweil, "Nourishing the Underfed and Overfed," chapter 4 in Worldwatch Institute, *State of the World 2000,* W.W. Norton and Co., New York, 2000, p. 72.
[200] Ogden, Yanovski, Carroll, and Flegal. "The Epidemiology of Obesity."
[201] Ibid. p. 2096.

Abdominal fat, an active metabolic tissue, is considered to be more strongly associated with metabolic syndrome than subcutaneous fat.[202] Abdominal fat, or adiposity, releases fatty acids, which accumulate in the liver and other tissues, and are utilized by the muscles, thereby causing glucose to be elevated in the blood, which in turn results in increased insulin output by the pancreas. When individuals do not produce the amount of insulin needed to metabolize the elevated glucose levels in the blood or do not properly utilize the insulin they do produce, they are at risk of developing type 2 diabetes, coronary heart disease, hypertension, and other chronic diseases.[203]

Thus, a meta-analysis conducted by Andrea Galassi et al. of Tulane University found that individuals with the metabolic syndrome, compared with those without the syndrome, had an elevated overall relative risk (RR) of cardiovascular disease of RR 1.61, an increased mortality rate from all causes (RR 1.74) and from cardiovascular disease (RR 1.74), and an increased incidence of cardiovascular disease (RR 1.53), coronary heart disease (RR 1.52), and stroke (RR 1.76).[204]

Figure 31 below illustrates the physical pathways from obesity to the outcomes of morbidity, disability, or mortality, as documented by medical and clinical researchers. Three conditions caused by obesity—sex hormone imbalance, increased free fatty acids, and mechanical stress—lead to different conditions and outcomes:

- Sex hormone imbalance leads to the development of hormone dependent tumours, such as those found in cancers, which in turn lead either to mortality or disability.
- Increased free fatty acids lead to insulin resistance, hypertension, and dyslipidemia,[205] or other aspects of the metabolic syndrome, which then in turn lead to type 2 diabetes or cardiovascular diseases, and to consequent disability or mortality.
- And mechanical stress caused by the extra burden that obesity places on the physical structure of the body can lead to shortness of breath, sleep apnea, osteoarthritis, low back pain, and other musculoskeletal disorders, which can then frequently lead to disability or (much more rarely than the conditions above) to mortality.

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[202] Caterson, I.D. "Obesity, Part of the Metabolic Syndrome," *Clinical Biochemist Reviews*, 1997, vol. 18, no. 1: 11-21.

[203] Ibid.

[204] Galassi, Andrea, Kristi Reynolds, and Jiang He. "Metabolic Syndrome and Risk of Cardiovascular Disease: A Meta-Analysis," *The American Journal of Medicine*, 2006, vol. 19: 812-819.

[205] Dyslipidemia: "A disorder of lipoprotein metabolism, including lipoprotein overproduction or deficiency. Dyslipidemias may be manifested by elevation of the total cholesterol, the 'bad' low-density lipoprotein (LDL) cholesterol and the triglyceride concentrations, and a decrease in the 'good' high-density lipoprotein (HDL) cholesterol concentration in the blood." MedicineNet.com. *Definition of Dyslipidemia*, 2008; accessed November 2008; available from http://www.medterms.com/script/main/art.asp?articlekey=33979.

The pathogeneses (physical pathways) related to obesity and specific disease conditions and outcomes are provided in the disease descriptions below, to the extent that these pathways are known or hypothesized.

Figure 31. Physical pathways from obesity to morbidity, disability, and mortality
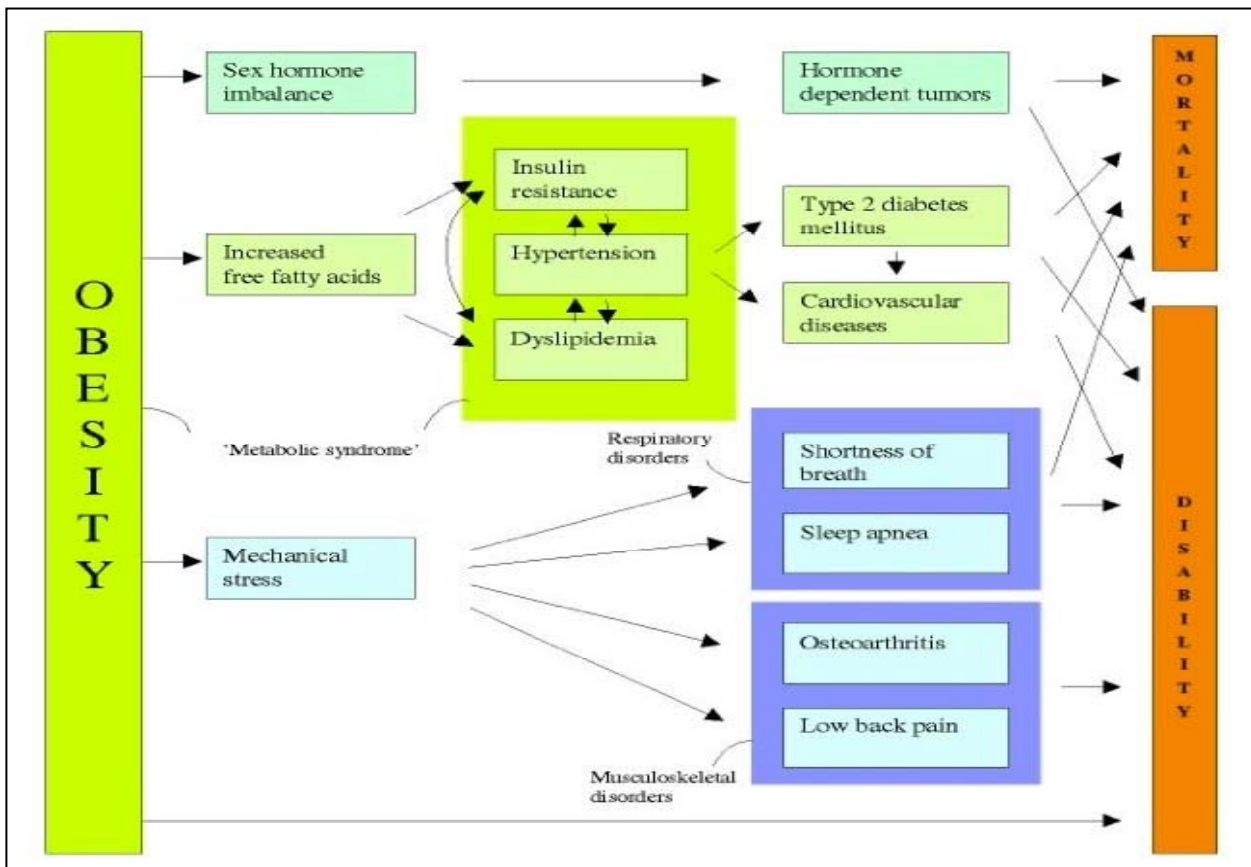
Source**:** Visscher, Tommy L.S., and Jacob C. Seidell. "The Public Health Impact of Obesity," *Annual Review of Public Health*, 2001, vol. 22: 355-375. Fig. 1.

4.2 Methodological issues in attributing a proportion of health conditions to obesity

4.2.1 Obesity as a risk factor or a disease

In this report, obesity is considered to be a risk factor for chronic disease, rather than as a disease itself, although it is noteworthy that Statistics Canada—in its reporting of BMI—lists overweight and obesity as 'health conditions' rather than as 'determinants' of health and illness. New Zealand researchers Tony Blakely et al. define risk factors as including "behaviours and conditions or states of individuals that are causally associated with the incidence of disease."[206] As examples, they include "malnutrition, indoor air pollution, unsafe water and sanitation, unsafe sex, tobacco and alcohol consumption, exercise, diet, blood pressure, weight, and cholesterol."[207]

Researchers Stanley Heshka and David B. Allison, of Columbia University's Institute of Human Nutrition, note that there is disagreement about whether obesity should be considered a disease, as can be seen in divergent descriptions in both popular and scholarly articles.[208] The argument often revolves around whether or not insurance companies will pay for drugs or other treatments if the disease label is not applied rather than around scholarly and analytical considerations. Some analysts argue that treating obesity as a disease ignores the systemic social forces that drive the condition and that demand a societal response.[209]

The Council of The Obesity Society in the U.S. notes that there is no "clear, specific, widely acceptable, and scientifically applicable definition of 'disease' that allows one to objectively and empirically determine whether specific conditions are diseases."[210] Therefore, the Council decided to take a "utilitarian approach," and recently released its official position that obesity should be *considered* a disease, rather than that obesity *is* a disease:

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[206] Blakely, Tony, Simon Hales, Charlotte Kieft, Nick Wilson, and Alistair Woodward. "Distribution of Risk Factors by Poverty," in *Comparative Quantification of Health Risks: Global and Regional Burden of Disease Attribution to Selected Major Risk Factors*, ed. Ezzati, Majid, Alan D. Lopez, Anthony Rodgers and Christopher J.L. Murray, Volume 1, Chapter 24: 1941-2127. Geneva: World Health Organization, 2004; accessed June 2008; available from http://www.who.int/publications/cra/chapters/volume2/1941-2128.pdf. p. 9.

[207] Ibid. p. 9.

[208] Heshka, Stanley, and David B. Allison. "Is Obesity a Disease?" *International Journal of Obesity*, 2001, vol. 25: 1401-1404.

[209] Elliott, Victoria Stagg. "Is Obesity a Disease? Clinicians Disagree." *AMNews*, Feb. 6, 2006.

[210] Allison, Downey, Atkinson, Billington, Bray, Eckel, Finkelstein, Jensen, and Tremblay. "Obesity as a Disease: A White Paper on Evidence and Arguments Commissioned by the Council of the Obesity Society." p. 1161

We believe that considering obesity a disease will: (1) benefit our citizenry by soliciting more resources for prevention and treatment of, and research on, obesity; (2) encourage health-care professionals to view treating obesity as a vocation worthy of effort and respect; and (3) reduce the stigma and discrimination experienced by many persons with obesity. After extensive dialogue and careful consideration, the Council concludes that the official position of The Obesity Society is that obesity should be declared a disease.[211]

Heshka and Allison agree that obesity is a major health problem, and that signs of impairment, which might meet most definitions of disease, usually accompany *severe* obesity (BMI ≥40).[212] However, this is not the case for milder obesity (BMI 30–40). They argue:

In sum, to call obesity defined solely on the basis of a BMI or percentage body fat in excess of some threshold a disease leads immediately to the following problems:

- the only sign or symptom may be the excess fat which is also the only defining feature of the condition—there are no other inevitable clinical or subclinical signs;
- many obese persons suffer no impairment as a consequence of their obesity;
- it ignores the probabilistic nature of the relation between obesity and consequent adverse events which is accurately conveyed by the term risk factor;
- it poses conceptual problems, eg is the obesity which is a sign of a disease, itself a disease?[213]

Determining whether or not obesity should technically be considered a disease or a risk factor for disease is beyond the scope of this report, which is specifically concerned with the costs of illnesses and other impairments that can be attributed to obesity. The methodology needed to estimate costs, which is described below, employs relative risk ratios and population attributable risk proportions that estimate the proportion of various adverse health conditions and costs that can be attributed to obesity. Because of these methodological considerations and data limitations that do not allow direct costing of obesity but only of specified obesity-related illnesses, this report, as noted, treats obesity as a risk factor for disease.

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[211] Obesity Society Council. "Obesity as a Disease: The Obesity Society Council Resolution," *Obesity*, 2008, vol. 16: 1151.
[212] Heshka, and Allison. "Is Obesity a Disease?"
[213] Ibid. p. 1403.

4.2.2 Causality and confounding

WHO defines risk as the probability of an adverse outcome, or a factor that raises this probability.[214] Blakely et al. observe that "an underlying assumption in all calculations of the disease burden is that the associations between risk factors and disease are causal."[215] Writing for WHO's Global Burden of Disease project, W. Phillip James et al. note simply that the associations found in the epidemiological literature "between BMI and many disease outcomes satisfy the widely-accepted criteria for causal relationships: they are strong, consistent, have a dose-response relationship and are biologically plausible."[216]

I. U. Eneli et al. note that evidence of *reversibility* is also an important epidemiological criterion of causality.[217] Therefore, if high BMI is the risk factor, then reducing BMI would decrease the prevalence of the associated health condition, or reduce related symptoms. This has been found to be the case with the chronic conditions associated with obesity—a weight loss reduces the incidence of these conditions.[218] Evidence of reversibility has important implications for interventions designed to promote weight loss.

Blakely et al. describe two major problems with determining causality for risk factor relationships: endogeneity and confounding.[219] Endogeneity refers to the inseparability of the risk factor (e.g. obesity) and health owing to "dynamic, synergistic and bi-directional causal associations,"[220] while confounding refers to the fact that a risk factor like obesity may be correlated with other determinants of health such as gender, age, diet and physical activity levels.

For example, in the first instance (endogeneity), it may not be clear whether obesity leads to poor health or whether poor health leads to obesity. However, the research consensus is that obesity most often occurs before poor health. The reverse causation theory—that poor health leads to obesity—is not seriously considered in the literature as the main direction of causality.[221] And in the second instance, it may not be clear whether health

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[214] World Health Organization. *Reducing Risks, Promoting Healthy Life*, 2002; accessed Dec 2007; available from http://www.who.int/whr/2002/Overview_E.pdf.

[215] Blakely, Tony, Simon Hales, and Alistair Woodward. *Poverty: Assessing the Distribution of Health Risks by Socioeconomic Position at National and Local Levels*, Environmental Burden of Disease Series No. 10, World Health Organization, 2004; accessed June 2008; available from http://www.who.int/quantifying_ehimpacts/publications/en/ebd10.pdf. p. 8.

[216] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)." p. 535.

[217] Eneli, Skybo, and Jr. " Weight Loss and Asthma: A Systematic Review."

[218] Ibid.

[219] Blakely, Hales, and Woodward. *Poverty: Assessing the Distribution of Health Risks by Socioeconomic Position at National and Local Levels*, accessed.

[220] Blakely, Hales, Kieft, Wilson, and Woodward. "Distribution of Risk Factors by Poverty."

[221] Allison, Downey, Atkinson, Billington, Bray, Eckel, Finkelstein, Jensen, and Tremblay. "Obesity as a Disease: A White Paper on Evidence and Arguments Commissioned by the Council of the Obesity Society."

outcomes are the result of obesity, or of other factors, including some that are closely correlated with obesity.

Isolating specific causes of chronic disease is difficult, considering the interdependent relationships that exist between various risk factors.[222] Multicausality, which implies that diseases have several causes acting together, is an established concept.[223] Most researchers agree that many factors work together in producing health or ill health, with the presence of any number of variables influencing the effect of the other. However, empirical studies have also found that obesity has an independent effect beyond its collective influences.[224] Therefore, it has been argued that direct associations between obesity and health conditions can be reliably determined even after due consideration is given to possible confounding factors.

Because of the multidimensionality of obesity, researchers often use statistical analysis when attempting to determine causality, in order to control for various confounding variables such as income, education, physical activity levels, smoking, diet, age, gender, and so on that may influence determination of the causes of disease and of the relative risks associated with a particular causal factor like obesity.

Referring to multicausality, Goodman remarks that population attributable fractions (PAFs) are not additive—i.e. the proportions of a particular illness that may be attributable to different risk factors (like obesity, physical inactivity, and smoking, for example) do not sum to 100 percent. He continues:

> In fact, PAFs are not additive when multiplicative (e.g. logistic) models of data analysis are used to generate the relative risk inputs, models which are standard in epidemiologic analyses. PAFs are also non-additive when causes are multifactorial, when individual lifestyle factors require each other to exert their effect, or when one factor is in the causal pathway of the other (cholesterol elevation and obesity, for example). The main implication of non-additivity is that it is incorrect to say that if 30 percent of deaths are attributable to one lifestyle factor (e.g., poor diet), then 70 percent are due to the other factors (e.g., tobacco, alcohol, firearms, sexual behavior). As with PKU disease, it can be completely correct to say that a case of disease is 100 percent attributable to an environmental factor (phenylalanine exposure) and is also 100 percent genetic (the

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[222] Haydon, Emma, Michael Roerecke, Norman Giesbrecht, Jurgen Rehm, and Marianne Kobus-Matthews. *Chronic Disease in Ontario and Canada: Determinants, Risk Factors and Prevention Priorities*, Ontario Chronic Disease Prevention Alliance and the Ontario Public Health Association, 2006; accessed June 2008; available from http://www.ocdpa.on.ca/docs/CDP-FullReport-Mar06.pdf.

[223] Hallqvist, J., A. Ahlbom, F. Diderichsen, and C. Reuterwall. "How to Evaluate Interaction between Causes: A Review of Practices in Cardiovascular Epidemiology," *Journal of Internal Medicine*, 1996, vol. 239: 377-382.

[224] Hubert, H.B., M. Feinleib, P.M. McNamara, and W.P. Castelli. "Obesity as an Independent Risk Factor for Cardiovascular Disease: A 26-Year Follow-up of Participants in the Framingham Heart Study," *Circulation*, 1983, vol. 67, no. 5: 968-977.

phenylketonuria [PKU] gene).[225]

Blakely et al., who prefer not to control for confounding variables in certain situations that show independent effects, argue that, in practice, controlling for variables is difficult because there is little information on many potentially confounding factors, and—even when the information is known—it is often not clear how best to control for the confounding factors.[226] In addition, when controlling for confounding variables, risk ratios cannot be accurately determined because a proportion of the population (e.g. those who smoke) may be left out of the calculation.

Dennis Raphael of York University notes that causality is probabilistic rather than absolute—i.e. a cause leads to an increase in the *probability* of an outcome, rather than always definitively leading to the outcome per se:

> Many philosophers and scientists use the idea of *efficient cause* based upon Aristotle's notion of what puts an event in motion. For a situation such as low income to be an efficient cause of an outcome such as cardiovascular disease it must: a) occur prior in time to the outcome; b) represent a process that produces the changes that lead to the outcome; and c) be part of a causal network that includes direct and indirect effects on the outcome of interest.[227]

Using this definition, therefore, obesity can generally be considered to be an *efficient cause* of various health conditions that have been identified in the research literature.

Health Canada suggests that, in order to take action on health problems and identified determinants of health and illness, public health needs "sufficient evidence," but "it does not need absolute evidence."[228] It offers the following quote from McKeown on the degree of evidence necessary for public action:

> [A]ction is often needed to protect and promote health in circumstances where the evidence is less than complete. Moreover, in many cases it is questionable whether within the foreseeable future the evidence will be complete. To assess precisely the respective roles of diet, exercise and smoking in the causation of coronary artery disease, a massive human experiment would be needed, with division of a population into multiple experimental and control groups. Such an

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[225] Goodman, Steven. In Institute of Medicine. *Estimating the Contributions of Lifestyle-Related Factors to Preventable Death: A Workshop Summary*, accessed. p. 9.
[226] Blakely, Hales, and Woodward. *Poverty: Assessing the Distribution of Health Risks by Socioeconomic Position at National and Local Levels*, accessed.
[227] Raphael, Dennis. *Inequality Is Bad for Our Hearts: Why Low Income and Social Exclusion Are Major Causes of Heart Disease in Canada*, Toronto: North York Heart Health Network, 2001; accessed Nov 2007; available from http://action.web.ca/home/narcc/attach/Inequality%20is%20Bad%20for%20Our%20Hearts%20-%202001%20Report%5B1%5D.pdf. p. 5.
[228] Health Canada. *Canadian Handbook on Health Impact Assessment. Vol 3: Roles for the Health Practitioner*, 1999; accessed Dec 2007; available from http://dsp-psd.communication.gc.ca/Collection/H46-2-99-235-3E-9.pdf. p. F8.

investigation would present formidable ethical, technical and administrative difficulties. Does this mean that no action can be taken in this and similar cases because the grounds, however suggestive, are not conclusive?

In the light of such difficulties … it will often be desirable to act on the basis of high, or even moderate probabilities, on what has been called 'a burden of prudence' rather than 'a burden of proof.' [.… I]t should be recognized that conclusive evidence of harm or benefit to health is often an unrealistic requirement.[229]

In sum, although confounding variables are often controlled for in epidemiological studies, it is not always possible to reach definitive conclusions on the degree of causality attributable to obesity and on the precise impact of potentially confounding factors like those indicated above. Therefore, causality is often assumed based on the best available epidemiological and other evidence, and 'conclusions' (on relative risks, PAFs, costs, intervention impacts, and other factors) can only be considered to be estimates or approximations that incorporate recognition of uncertainties.

For the reasons Health Canada states above, the challenges associated with determining causality and accounting for uncertainties and confounding factors by no means negate the importance of undertaking the kind of analysis of obesity costs in Alberta attempted here. But this discussion on causality and confounding provides a necessary context for consideration, understanding, and proper use of the results that will presented below.

4.2.3 Basic methodology steps

The association between obesity and health status must first be established and quantified before the costs of obesity can be estimated. Cost estimates require information to be gathered or calculated on three basic factors:

- the prevalence of the risk (in this case obesity) in the population,
- the relative risk ratio for the outcome (obesity-related diseases) in question, and
- the proportion of the outcome that can be attributed to the risk.

The most commonly used epidemiological measures of relative risk are the risk ratio (RR) and the odds ratio (OR),[230] and the measure most commonly used to determine the risk-related outcome proportion is the population attributable fraction (PAF).

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[229] McKeown, T. *The Role of Medicine - Dream, Mirage or Nemesis?*, Princeton, New Jersey: Princeton University Press, 1979. Cited in Health Canada. *Canadian Handbook on Health Impact Assessment. Vol 3: Roles for the Health Practitioner*, accessed. p. F8.
[230] Tu. "Developmental Epidemiology: A Review of Three Key Measures of Effect."

Risk ratios, which compare the number of cases **with** the outcome to the **total** number of cases—with and without the outcome—are to be distinguished from odds ratios, which compare the number of cases **with** the outcome to the number of cases **without** the outcome.[231] Please see the formula used—and its accompanying explanation in Section 4.2.3.4 below—to illustrate the calculation of relative risks in relation to the total number of cases with and without the outcome in question.

Logistic regression, which is a commonly used method of statistical analysis, yields an odds ratio rather than a risk ratio, and therefore odds ratios rather than risk ratios are commonly seen in clinical research reports. Except in certain cases such as for rare diseases, analysts generally do not recommend using odds ratios to calculate PAFs, because the odds ratio tends to magnify an effect compared with a risk ratio.[232, 233] Risk ratios can be estimated from odds ratios if the frequency of the outcome among those lacking the risk factor is known, but this information is often missing in research and medical journal articles that report odds ratios.[234]

Population attributable risk proportions are actually referred to by various terms in the literature, including Population Attributable Fraction (PAF), Population Attributable Risk (PAR), Impact Fraction (IF), and Attributable Fraction (AF). Beverly Rockhill et al. of the University of North Carolina argue that using the word "risk" in this context as attributable risk is technically incorrect, because it is more correct to speak of a proportion or fraction of risk. They suggest that the terms "population attributable risk proportion" or "population attributable fraction" are more accurate than "population attributable risk."[235] Steven Goodman of Johns Hopkins School of Medicine also notes that PAF is not a measure of risk:

> Unlike attributable risk, population attributable fraction applies to a population rather than to an individual, and it is not a measure of 'risk.' PAF is the fraction of disease cases in a population associated with an exposure. 'Attributable' is somewhat misleading because it implies causality, i.e. that removal of that exposure would in fact eliminate that fraction of cases. We will see that is typically not true, one reason being that complex causal connections, such as that between obesity and mortality, are not fully understood…. Perhaps the best term would be population *associated* fraction (which would maintain the same acronym), but for the purposes of consistency with current terminology, I will

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[231] Holcomb, Jr., William L. , Tinnakorn Chaiworapongsa, Douglas A. Luke, and Kevin D. Burgdorf. "An Odd Measure of Risk: Use and Misuse of the Odds Ratio," *Obstetrics and Gynecology*, 2001, vol. 98, no. 4: 685-688.

[232] Ibid.

[233] See also: Schmidt, Carsten Oliver, and Thomas Kohlmann. "When to Use the Odds Ratio or the Relative Risk?" *International Journal of Public Health*, 2008, vol. 53: 165-167.

[234] For more information on the difference between risk ratios and odds ratios, and the conversion of odds ratios to risk ratios, see: Zhang, Jun, and Kai F. Yu. "What's the Relative Risk? A Method of Correcting the Odds Ratio in Cohort Studies of Common Outcomes," *JAMA (Journal of the American Medical Association)*, 1998, vol. 280, no. 19: 1690-1691.

[235] Rockhill, Beverly, Beth Newman, and Clarice Weinberg. "Use and Misuse of Population Attributable Fractions," *American Journal of Public Health*, 1998, vol. 88, no. 1: 15-19.

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retain the term 'attributable'.[236]

Goodman continues that PAF is "the probability of the disease in the overall population (the average risk in both unexposed and exposed people) minus the probability of disease in the unexposed population."[237]

Epidemiological studies use regression-based measures to find associations—or relative risk ratios (RR)—between variables that can model causal relationships or correlations between variables. These associations—in this case between obesity and specific health conditions—can be found in the epidemiological literature, or they can be directly calculated from raw data in sources like the Canadian Community Health Survey (CCHS). Once this association and the distribution of the risk factor and health outcome levels in the populations living with obesity are known, then the actual costs of obesity can be estimated.

Thus, prior to determining costs, a number of steps are required to assess the health impacts of obesity.[238] These steps, which are used in most burden of disease and cost of illness studies, generally include:

1. Through a review of the epidemiological literature, determine which diseases have a "causal" association with obesity, based on the modified notions of causality as discussed above in Section 4.2.2.
2. Through the use of survey data, determine the prevalence and distribution of obesity within the population,
3. Through the use of administrative or survey data, determine the proportion of cases of each obesity-related disease (from #1 above) that have been exposed to the risk factor of interest (i.e. obesity, in this case),
4. Determine the relative risk ratio (RR) for the association between obesity and specific health outcomes, and adjust RRs if it is necessary to control for confounding.
5. Calculate the population attributable fractions (PAF)—i.e. the estimated proportion of each health outcome attributable to obesity, and
6. Estimate uncertainties.

Population attributable fractions (PAF) can then be used in a 7th step to estimate the proportion of the costs of each obesity-related illness that can be attributed to obesity, and the total of such costs across a range of illnesses.

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[236] Goodman, Steven. In Institute of Medicine. *Estimating the Contributions of Lifestyle-Related Factors to Preventable Death: A Workshop Summary*, accessed. p. 8.

[237] Goodman, Steven. In Ibid., accessed. p. 8.

[238] Colman, Ronald. *Cost of Obesity in Nova Scotia*, accessed.; and Colman, Ronald. *The Cost of Chronic Disease in Nova Scotia*, accessed.

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4.2.3.1 Determination of obesity-attributable diseases

The first step in estimating the cost of obesity in Alberta, therefore, was to determine which diseases are partially 'caused' by or attributable to obesity. As noted above, Blakely et al. observe that "an underlying assumption in all calculations of the disease burden is that the associations between risk factors and disease are causal."[239] Writing for WHO's Global Burden of Disease project, W. Phillip James et al. note simply that the associations found in the epidemiological literature "between BMI and many disease outcomes satisfy the widely-accepted criteria for causal relationships: they are strong, consistent, have a dose-response relationship and are biologically plausible."[240] The diseases for which enhanced risks attributable to obesity have been demonstrated in the epidemiological literature are described below in Chapters 4.3–4.10.

4.2.3.2 Estimation of Body Mass Index (BMI)

The Public Use Microdata Files of the 2004 and 2005 Canadian Community Health Survey (CCHS), cycles 2.2 and 3.1 were used to determine the proportion of the Canadian and Albertan populations aged 15 years and older in each of six body mass index (BMI) categories: underweight (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2), obese class 1 (30.0–34.9 kg/m^2), obese class 2 (35.0–39.9 kg/m^2), and obese class 3 (40.0 kg/m^2 and over).

Because of its larger sample size and the relatively close approximation between the Albertan and overall Canadian health and disease profiles, the larger Canadian population was used to determine prevalence rates of health conditions that were included in 2004 and 2005 CCHS. This ensures a narrower sampling variability and coefficient of variation when determining PAFs directly from the raw CCHS data than would use of CCHS Albertan disease prevalence rates, which in most cases do not produce statistically significant results when correlated with obesity rates due to small sample sizes.

However, the Alberta directly measured BMI rates derived from the 2004 CCHS and provided by Statistics Canada[241] were used in the calculation of some PAFs for health conditions not included in the 2004 and 2005 CCHS, and for which RRs therefore had to be extrapolated from the epidemiological literature (e.g. gallbladder disease and five

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[239] Blakely, Hales, and Woodward. *Poverty: Assessing the Distribution of Health Risks by Socioeconomic Position at National and Local Levels*, accessed. p. 8.

[240] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)." p. 535.

[241] Statistics Canada. Measured Adult Body Mass Index (BMI), by Age Group and Sex, Household Population Aged 18 and over Excluding Pregnant Females, Canada and Provinces, CANSIM Table 105-2001 (Canada and Alberta), 2004 Canadian Community Health Survey Cycle 2.2, 2008.

cancer sites).[242] In the case of gallbladder disease and bladder cancer, the CCHS sampling variability is also an issue for BMI prevalence rates for gender-specific obese classes 1–3 and age groups, but not for total obesity. These methodologies are discussed more fully in relation to the specific health conditions in Part 2, Chapter 5, of this report.

As discussed above, the 2004 CCHS cycle 2.2 directly measured the height and weight of respondents, which is used to calculate BMI and is considered far more accurate than self-reported data. Self-reports can result in serious underestimates of the prevalence of overweight and obesity, since respondents have a tendency to overestimate their height and underestimate their weight. Therefore, the CCHS cycle 2.2 was used whenever possible to assess BMI levels and related PAF estimates—namely in cases where the 2004 CCHS cycle 2.2 provided information on obesity-related health conditions (i.e., diabetes, hypertension, and heart disease).

When calculating the prevalence of obesity among individuals with chronic conditions that were not captured in 2004 CCHS cycle 2.2 but were included in the 2005 CCHS (i.e., cerebrovascular disease (stroke), osteoarthritis, asthma, and depression), the 2005 CCHS cycle 3.1 was used. This was possible because a correction factor for self-reported BMI has recently been developed by Statistics Canada and tested for CCHS cycle 3.1.[243] While use of direct height and weight measurements (as provided in CCHS cycle 2.2) would be ideal, Statistics Canada's correction formulae were developed based on rigorous tests of self-reported 2005 BMI results against the directly measured data and found to improve significantly the classification of overweight and obesity compared to use of the unadjusted self-reported results. Thus, the developers of the correction factor recommend its use for obesity studies based on the adult population in CCHS cycle 3.1.

Thus, the 2005 BMI estimates—which in turn have been used to estimate obesity-related population attributable fractions based on CCHS cycle 3.1 data—have been corrected using the following formulae:[244]

For males: BMI (measured) $= 1.08$(self-reported BMI)$- 1.08$ (formula 1)

For females: BMI (measured) $= 1.05$(self-reported BMI)$- 0.12$ (formula 2)

Self-reported BMI from the 2005 CCHS is the only variable that needs to be added to these formulae in order to obtain reasonable equivalence to directly measured data. The other numbers used by Statistics Canada to produce these formulae are fixed statistical factors (i.e. the intercept and slope, respectively, of the equation of a line) that were determined to be necessary to make the correction. A full explanation of these statistical methods is available in the Statistics Canada report by Sarah Gorber et al. titled "The

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[242] The cancer sites for which RRs from the epidemiological literature and 2004 CCHS cycle 2.2 BMI prevalence data for Alberta were used to estimate PAFs were endometrial cancer, esophageal cancer, liver cancer, bladder cancer, and stomach cancer.
[243] Gorber, Shields, Tremblay, and McDowell. "The Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity."
[244] Ibid.

Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity"[245]

4.2.3.3 Prevalence of chronic health conditions

As noted above, the prevalence of several health conditions identified in the epidemiological literature as being partially attributable to obesity was estimated using responses to the CCHS cycles 2.2 (2004) and 3.1 (2005). These prevalence estimates were used in the estimation of the obesity-related population attributable fractions described below. The relevant questions that the respondents were asked in the 2004 and 2005 CCHS are presented in Table 15 below.

The CCHS cycle 2.2 (2004) provided information on the prevalence of high blood pressure, diabetes, and heart disease, and the CCHS cycle 3.1 (2005) provided information on asthma, osteoarthritis, total cancer, stroke, and mood disorders (depression).

Table 15. Questions used in the Canadian Community Health Survey (CCHS) 2004, cycle 2.2, and 2005, cycle 3.1, to determine prevalence of chronic health conditions

Preamble to questions on chronic conditions: I'd like to ask about certain chronic health conditions which you may have. We are interested in "long-term conditions" which are expected to last or have already lasted 6 months or more and that have been diagnosed by a health professional.		
Chronic Condition	**CCHS Cycle**	**Questions**
Asthma	3.1	Do you have asthma?
Osteoarthritis	3.1	Do you have arthritis or rheumatism, excluding fibromyalgia? If yes, do you have rheumatoid arthritis, osteoarthritis, rheumatism, or other?
High blood pressure (hypertension)	2.2	Do you have high blood pressure?
Diabetes	2.2	Do you have diabetes?
Heart Disease	2.2	Do you have heart disease?
Cancer	3.1	Do you have cancer? Have you ever been diagnosed with cancer?
Stroke (cerebrovascular disease)	3.1	Do you suffer from the effects of a stroke?
Mood Disorder	3.1	Do you suffer from a mood disorder such as depression,

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[245] Ibid.

| | | bipolar disorder, mania or dysthymia? |

Source: Statistics Canada, Canadian Community Health Survey, 2004, cycle 2.2 and 2005, cycle 3.1.

For those obesity-related conditions (specific cancer sites and gallbladder disease) where prevalence could not be determined using the CCHS, and where PAFs could not therefore be directly estimated by the authors, RRs and/or PAFs were derived from estimates in the relevant epidemiological literature and did not depend on disease prevalence rates.

4.2.3.4 Relative risk ratios for the association between obesity and health outcomes

As noted above, relative risk ratios (RRs) determine the *individual* risk in the population of developing specified health outcomes that can be associated with a particular risk factor such as obesity. For example, RR can determine the extent of risk of developing colon cancer for an obese individual compared to the degree of risk of colon cancer in someone with "normal" or healthy weight.

RR is a relative measure of effect that is determined through statistical analysis. It compares the number of cases with a particular outcome to the total number of cases (with and without the outcome), and has been defined as "the increase in the probability of an outcome given one situation, relative to the probability of an outcome given some other situation."[246] Please see the formula below and its accompanying explanation to illustrate the meaning, application, and use of the "total number of cases (with and without the outcome)."

The relative risk ratio (RR) is a comparison between the risk of one group that is experiencing the risk factor (e.g. obesity) and that has the probability of developing a particular health outcome, compared with the risk of developing that health outcome in a reference group (e.g. usually the group which has the best health outcome—those with normal weight).

According to Majid Ezzati and other members of the WHO Comparative Risk Assessment Collaborating Group, the obese groups that are at risk of chronic diseases are compared with the group with the lowest risk:

> In the Comparative Risk Assessment project, the estimates of burden of disease and injuries due to risk factors are based on a counterfactual exposure distribution that would result in the lowest population risk, irrespective of whether currently attainable in practice, referred to as the theoretical minimum exposure distribution. Use of theoretical minimum exposure distribution as the counterfactual has the advantage of providing a vision of potential gains in

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[246] Tu, Shihfen. "Developmental Epidemiology: A Review of Three Key Measures of Effect," *Journal of Clinical Child and Adolescent Psychology*, 2003, vol. 32, no. 2: 187-192.

population health by risk reduction from all degrees of suboptimum exposure in a consistent way across risk factors.[247]

In most cases, the increased risk of experiencing a negative, rather than positive, outcome is compared between the groups, since the outcome is ill health. An RR value of 1.0 indicates no particular effect for the at-risk (obese) group, with outcomes among obese individuals identical to outcomes for those with normal weight—indicating that both groups being compared are equally at risk. If the RR is greater than 1.0, it indicates that the group experiencing the risk factor is at greater risk relative to the reference group, and if the value is less than one, the group with the risk factor is less at risk than the reference group.

RRs can be calculated when there is a representative sample, such as when the entire population is included in the sample, or when there is a cohort designed study, consisting of two subgroups with one exposed to the risk and the other not exposed, where valid probabilities can be calculated.

As noted above, RR is usually determined through statistical analysis, which can adjust the data to control for confounding variables and thus specifically determine probable 'causal' associations to the extent possible. However, a simple formula, using both an exposed and a non-exposed group, can illustrate the basic premise. The following formula was used to estimate unadjusted relative risks in this report:

$$RR = [a/(a + b)] / [c/(c + d)]^{248}$$

Where (as illustrated in Table 16 below):
a = those who have been exposed to the risk factor of interest (e.g. the obese group) and who have developed the health condition,
b = those who have been exposed to the risk factor (e.g. the obese group) and who have not developed the health condition,
c = those who have not been exposed to the risk factor (e.g. the non-obese group being compared, or the reference group, such as those with normal weight) and who have developed the health condition, and
d = those who have not been exposed to the risk factor (e.g. the non-obese group being compared, or the reference group, such as those with normal weight) and who have not developed the health condition.

a + b = the total number of individuals exposed to the risk factor of interest
c + d = the total number of individuals not exposed to the risk factor of interest

Note that both a and c groups (the obese and non-obese groups with the health condition) are divided by the total number in each group (those with and without the condition), rather than only by the number without the outcome. The latter would produce an odds ratio rather than a

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[247] Ezzati, Majid, Alan D. Lopez, Anthony Rodgers, Stephen Vander Hoorn, Christopher J. L. Murray, and the Comparative Risk Assessment Collaborating Group. "Selected Major Risk Factors and Global and Regional Burden of Disease," *Lancet*, 2002, vol. 360, no. November 2: 1347-1360.
[248] Tu. "Developmental Epidemiology: A Review of Three Key Measures of Effect."

relative risk ratio.

Therefore:
* a/(a + b) = risk of developing the health condition in the exposed group (e.g. the risk of developing diabetes for an obese person);
* c/(c + d) = risk of developing the health condition in the non-exposed reference group (e.g. the risk of developing diabetes for a person who is not obese)
* RR = risk of the health condition in the exposed group (e.g. the risk of having diabetes if you are obese) / the risk in the non-exposed reference group (e.g. risk of having diabetes if you are not obese, or of normal weight).

The RR formula is illustrated in Table 16 below, which shows how the probability of two groups either developing or not developing a health outcome is estimated.

Table 16. Relative risk groups

	Develop health outcome (e.g. develop diabetes)	Not develop health outcome (e.g. not develop diabetes)	Probability	Relative risk
Experience risk factor (obese)	a	b	a / (a + b)	a / (a + b) / c / (c + d)
Not experience risk factor (reference group – not obese, or normal weight)	c	d	c / (c + d)	1.0 (reference group)

Source: Adapted from Tu, Shihfen. "Developmental Epidemiology: A Review of Three Key Measures of Effect," *Journal of Clinical Child and Adolescent Psychology*, 2003, vol. 32, no. 2: 187-192.

Tu gives the following example, shown in Table 17 below, of the relative risk of elementary school youth who were Severely Emotionally Disturbed (SED) in 1994 being involved in a juvenile court case five years later. In the example provided, the RR is 8.02, which indicates that the youth identified as being SED in elementary school were 8.02 times more likely to be involved in a juvenile court case than were youth who were not SED.[249]

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[249] Ibid.

Table 17. Relative risk study

	Develop outcome: court case	Not develop outcome: no court case	Total	Probability	Relative Risk
Experience risk factor – SED	a – 230	b – 1,564	1,794	a /(a + b) = 230 / (230 + 1,564) = 0.128	0.128 / 0.016 = 8.02
Not experience risk factor factor – no SED (reference group)	c – 2,271	d – 139,802	142,073	c / (c + d) = 2,271 / (2,271 + 139,802) = 0.016	1.0 (reference group)

Source: Adapted from: Tu, Shihfen. "Developmental Epidemiology: A Review of Three Key Measures of Effect," *Journal of Clinical Child and Adolescent Psychology*, 2003, vol. 32, no. 2: 187-192.

For this present analysis of obesity costs in Alberta, relative risks for each gender *adjusted for age* (age groups 15-34, 35-64, and 65+) were estimated, as age and gender are known to be major confounders of risk for chronic conditions.[250] This is because age, in particular, is associated in this case both with the risk factor (obesity) and with the outcome of interest (prevalence of chronic health conditions), while risks and outcomes have also been shown to vary considerably by gender in many cases.

Ideally—since obesity costs are here being estimated for Alberta in particular—relative risks for this study would be calculated from data specific to Alberta. However, the larger Canadian sample in the Canadian Community Health Survey was used here for assessment of relative risk ratios because—for this particular purpose of determining relative risks—the Canadian data represent a reasonable approximation of Albertan data, and have the considerable advantage of reflecting a much greater sample size, which in turn produces more stable and statistically significant estimates. This is especially true for the 2005 CCHS data that reflect a considerably greater sample size than the 2004 CCHS. (See Chapter 4.2.3.6 below for a discussion of uncertainties in the 2004 CCHS estimates).

A recent study by Luo et al. noted that using relative risk ratios that are summarized from a number of previously published sources is more appropriate than relying on estimates from a single study.[251] Upon further inspection however, there are several drawbacks to using published summary relative risks as they:

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[250] Gerstman, B. B. *Stratified Analysis: Introduction to Confounding and Interaction*, 2000; accessed November 2008; available from http://www.sjsu.edu/faculty/gerstman/StatPrimer/stratified.PDF.
[251] Luo, Wei, Howard Morrison, Margaret de Groh, Chris Waters, Marie DesMeules, Elaine Jones-McLean, Anne-Marie Ugnat, Sylvie Desjardins, Morgan Lim, and Yang Mao. "The Burden of Adult Obesity in Canada," *Chronic Diseases in Canada*, 2007, vol. 27, no. 4: 135-144.

a) often are calculated for disparate populations that may not be generalisable to Canada or Alberta;
b) have been adjusted for unknown potential confounders; and
c) compare the risk of chronic diseases for inconsistent categories of BMI and age groups.

Due to these concerns, where possible we have chosen here to use estimates of relative risk from the CCHS— which is geographically and temporally generalisable to our population of interest—for the health conditions addressed in the survey. Using this data source has also allowed us to be certain that the relative risk ratios used in our calculation of population attributable fractions (PAFs) were adjusted appropriately for our analysis, as this has been identified as an important criterion for calculating accurate PAFs.[252]

4.2.3.5 Population attributable fractions (PAF)

As noted, RR indicates the degree of risk at the individual level that can be attributed to the causal effects of a risk factor or condition. However, population attributable fractions (PAF) need to be calculated in order to indicate the effect of the risk factor upon the community as a whole— in other words, the proportion of the health outcome at the *population* level that is attributable to the risk factor—and hence to estimate societal costs attributable to the risk factor.[253]

According to U.S. researchers Keith Scott et al., epidemiological measures such as PAF have direct relevance to public policy and action, since these measures focus on differences in proportions in the population, and therefore have the ability to separate risk to the population from risk to the individual.[254]

Once the RR is determined, the PAF is calculated by a fairly simple statistical method that partially attributes the prevalence of an illness (e.g. asthma) at the population level to another factor such as the prevalence of a health determinant or risk factor (e.g. obesity) in the population. Basically, PAF compares the current situation of ill health in the obese population with a hypothetical reference situation in which everyone in the obese group has the same health status as the healthiest (reference) group—usually those who have normal weight. The difference between the current and hypothetical situation represents the potential health disparity between the obese population and those with normal weight and thus allows an estimate of the degree of illness burden that could potentially be avoided if everyone had healthy weights.

Thus, the basic methodology used in this report for estimating the *proportion* of a specific illness that can be attributed to excess weight compares the number of cases of the

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[252] Flegal, Katherine M., David F. Williamson, and Barry I. Graubard. "Using Adjusted Relative Risks to Calculate Attributable Fractions," *American Journal of Public Health*, Letter to the editor, 2006, vol. 96, no. 3: 398.
[253] Scott, Mason, and Chapman. "The Use of Epidemiological Methodology as a Means of Influencing Public Policy."
[254] Ibid.

specific illness among the overweight or obese population with the number of cases of the specific illness in the population that has a normal or healthy weight.[255] The illness cases in the overweight or obese population are considered to be in "excess" of what can be "expected" in the normal weight population if the number of illness cases for the same illness in the overweight or obese population is higher than that in the normal weight population. The excess cases of illness in the overweight or obese group are referred to as being "attributable to overweight or obesity."[256]

Thus, the population attributable fraction (PAF) for the BMI-illness association estimates the percentage of excess cases of illness in the overweight or obesity group. In other words, hypothetically, if there are 100 cases of colorectal cancer in the normal weight population, and 150 cases of colorectal cancer in an obese population of the same size, then 50 cases of colorectal cancer—the "excess" cases—are considered to be attributable to obesity (BMI ≥30). If there are also 120 cases of colorectal cancer in an overweight population of the same size, the 20 "excess" cases are considered to be attributable to overweight (BMI 25-29.9) in the population.

PAFs are used to estimate the potential changes in population health from altering or eliminating the risk (e.g. obesity). Mathers et al., writing for WHO, note that the PAF can be defined as "the percentage reduction in disease or death that would occur if exposure to the risk factor was reduced to zero."[257]

One clear policy goal of estimating the burden of disease or risk associated with obesity or any other risk factor is, therefore, to estimate the percentage of disease that is potentially *avoidable* in the future, which in turn is based on time lags between exposure to the risk factor and the onset of disease. However, Blakely et al. argue that there is no clear understanding of the time lag between exposure to risk factors and disease outcomes, and that it is therefore only possible to estimate the attributable, rather than avoidable, burden of risk factors.[258] Referring to poverty as a risk factor, in an observation that is also relevant to obesity as a risk factor, Blakely et al. note:

> It is critical to realize that any such estimated population attributable risks are not necessarily accurate predictors of the avoidable burden of the risk factors. Changing only poverty [/obesity] within a population, for example, would not necessarily immediately reduce the risk-factor burden by a commensurate amount. This is because it is likely that the population distribution of relative risks by socioeconomic factor [/obesity] are confounded by other factors, and because time lags are uncertain. Nevertheless, it is possible to state that: 'If people with socioeconomic [/obesity] level X had the same risk-factor prevalence as people

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[255] Hanley. "A Heuristic Approach to the Formulas for Population Attributable Fraction."

[256] Flegal, Katherine M., Barry I. Graubard, and David F. Williamson. "Methods of Calculating Deaths Attributable to Obesity," *American Journal of Epidemiology*, 2004, vol. 160, no. 4: 331-338.

[257] Mathers, Vos, Lopez, Salomon, and Ezzati. *National Burden of Disease Studies: A Practical Guide*, accessed.

[258] Blakely, Hales, and Woodward. *Poverty: Assessing the Distribution of Health Risks by Socioeconomic Position at National and Local Levels*, accessed. p. 6.

with socioeconomic [/obesity] level Y, then the overall risk-factor prevalence would be decreased/ increased by Z.'[259]

The authors also note that this scenario *assumes* "that changing the poverty [obesity] level will change the levels of risk factors in the population," but the extent of this change can only be estimated.[260] (Note that [obesity] is added to the above citations for illustrative purposes). Despite Blakely's major caveat here on the difference between attribution and avoidable burden and Goodman's similar qualification cited earlier, we have also noted that Eneli et al. cite evidence of the reversibility of illness burden in response to weight reduction, and that Tu, Mathers and others do define population attributable fractions in terms of potential disease reduction.

Because the policy benefits of indicating potential disease avoidance through risk factor reduction are considerable, we do in this report sometimes use PAFs to refer to potential disease avoidance, but generally with the qualifier "potential", in order to acknowledge Blakely's argument that PAFs "are not necessarily accurate predictors of the avoidable burden of the risk factors."

As well, we have accounted to the extent possible for the two reasons (namely confounding and time lags) that Blakely et al. give for their argument above—first by presenting results by age and gender, which are two key confounding factors in estimating the disease burden of obesity, and second by making no claims that disease avoidance through obesity reduction would be "immediate." Just as disease development attributable to obesity is gradual, so it is clear that disease avoidance through obesity reduction is also gradual.

That, however, does not negate the longer-term reality that interventions designed to reduce obesity prevalence will likely reduce the burden of chronic disease, other factors being equal, which—in the view of the authors of this report—justifies use of PAFs to point to potential disease avoidance. In the end we balance Blakely's argument that PAFs "are not necessarily accurate predictors" of potential disease avoidance with Health Canada's argument, cited earlier, that "it will often be desirable to act on the basis of high, or even moderate probabilities, on what has been called 'a burden of prudence' rather than 'a burden of proof.' [.… I]t should be recognized that conclusive evidence of harm or benefit to health is often an unrealistic requirement."[261]

PAFs are especially useful to estimate costs when there are co-morbidity or lifestyle risk factors involved.[262] Risk factors, such as obesity or tobacco use, have few directly attributable costs because the costs are usually estimated for the diseases that are caused by, or correlated with, the risk factor. For example, obesity has well-established, quantifiable co-morbidity with more than twenty illnesses such as type 2 diabetes,

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[259] Ibid., accessed. pp. 11–12.
[260] Ibid., accessed. p. 11.
[261] McKeown. *The Role of Medicine - Dream, Mirage or Nemesis?* Cited in Health Canada. *Canadian Handbook on Health Impact Assessment. Vol 3: Roles for the Health Practitioner*, accessed. p. F8.
[262] Rockhill, Newman, and Weinberg. "Use and Misuse of Population Attributable Fractions."

hypertension, colorectal cancer, and coronary artery disease for which cost estimates are possible, and demonstrated links to several more diseases for which reliable cost estimates are not yet possible due to data limitations.[263] Therefore, costs attributable to obesity, for example, can be derived by multiplying the total direct health care cost and indirect productivity-loss cost for each obesity-related disease (as provided by PHAC's *Economic Burden of Illness in Canada*—EBIC[264]) by the PAF, or the proportion of the particular disease prevalence that is attributable to obesity.[265]

In sum, risk factor costs cannot be assessed directly as the direct costs incurred by smoking or by being physical inactivity or obese per se, but—by means of PAFs—can be assessed indirectly through the costs associated with the diseases that they engender. For example, the costs of smoking may be limited to the cost of cigarettes, the immediate discomfort caused to others by being exposed to smoky air, and other factors. The true (or long-term) cost of smoking can only be assessed indirectly by assessing the proportion of the burden of lung cancer, heart disease, respiratory disorders, low birthweight babies, and other health conditions that can be attributed to smoking. Thus, it is seen that PAFs are the essential means to establish the link between risk factors like obesity and established co-morbidities that in turn can be used as the basis of obesity cost estimates.

Methodological issues in estimation of PAFs

Calculations of PAF, then, can estimate the health outcome / disease prevalence that would potentially be the case in the population as a whole if all overweight or obese people had healthy weights and had the same prevalence of disease as those who have healthy weights.

According to Steenland and Armstrong, "[I]deally, the attributable fraction should be estimated from a lifetime follow-up of exposed and non-exposed cohorts in the population of interest. In practice, the AF is usually based on one or more epidemiologic studies of specific exposed and unexposed populations with incomplete follow-up."[266] Indeed, for the purposes of this study, a long-term cohort was not available, so estimates from the 2004 and 2005 Canadian Community Health Surveys (CCHS)—a large cross-sectional population-based survey—were used to estimate PAFs for the health conditions included in the CCHS.

According to Hanley, a PAF can be estimated using two equivalent basic formulae:—one is based on the prevalence of exposure (e.g. obesity) in the population, and the other is

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[263] Colman. *Cost of Obesity in Nova Scotia*, accessed.
[264] Health Canada. *Economic Burden of Illness in Canada, 1998*, 2002; accessed Nov 2007; available from http://www.phac-aspc.gc.ca/publicat/ebic-femc98/pdf/ebic1998.pdf. PHAC is in the process of updating EBIC from 1998 to 2000 costs. Unpublished data from the new update are used to estimate costs in this report.
[265] Colman. *Cost of Obesity in Nova Scotia*, accessed.
[266]Steenland, Kyle, and Ben Armstrong. "An Overview of Methods for Calculating the Burden of Disease Due to Specific Risk Factors," *Epidemiology*, 2006, vol. 17, no. 5: 512-519.

based on the distribution of cases of illness in the population.[267] The latter case-based formula was used in this report to estimate PAFs for diabetes, hypertension, heart disease, stroke, osteoarthritis, asthma, and depression. Unfortunately because of a lack of data, the obesity-prevalence formula, which as described below can create biases when used with adjusted relative risk ratios, was used in this report to estimate PAFs for gallbladder disease and four cancer sites (i.e., esophageal cancer, liver cancer, bladder cancer, and stomach cancer). (The methodologies for these diseases are explained more fully in Part 2, Chapters 5.4.3 and 5.4.5 of this report.) Both the obesity-prevalence based and the case-based formulae used to estimate PAFs are shown below.

Hanley recommends using the latter case-based formula—which was used to estimate PAFs in this report for the health conditions reported in the CCHS—when more than two levels or strata of an exposure of interest are present, and/or when RRs have been adjusted for confounding by such factors as gender and age. Multiple exposure levels occur when there is more than one level of exposure to a specific risk factor (e.g. obesity classes 1–3). Confounding occurs when a third factor (e.g. age) is associated with both the exposure and the outcome of interest. Concerning age as a confounding factor, Flegal notes:

> The relative risks of obesity among the elderly may well be lower than among young or middle-aged people. Because of the high proportion of health conditions among the elderly and the high health care costs incurred by the elderly, estimates of the attributable fraction are sensitive to relative risks among the elderly.[268]

The importance of using such methods to account for key confounding factors is confirmed by James Robins of the Harvard School of Public Health, who notes that failing to stratify by age when calculating PAFs for obesity can lead to about a 30 percent error.[269]

In this report the case-based formula was used to estimate PAFs where possible, both because the RRs, which were estimated separately by gender, were adjusted for age, and because five category-specific multiple levels or strata of exposure were used—i.e. normal weight (18.5–24.9), overweight (BMI 25–29.9), total obese (BMI ≥30), obese class 1 (BMI 30–34.9), and obese classes 2 (BMI 35–39.9) and 3 (BMI ≥40) combined —rather than a two-level exposure comparing only total obesity with normal weight. Rockhill et al. note: "A category-specific attributable fraction is the fraction of the total disease risk in the population that would be eliminated if persons in only that specific exposure category were to be shifted to the unexposed group."[270]

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[267] Hanley. "A Heuristic Approach to the Formulas for Population Attributable Fraction."
[268] Flegal. "Editorial: Estimating the Impact of Obesity."
[269] Robins, James. In Institute of Medicine. *Estimating the Contributions of Lifestyle-Related Factors to Preventable Death: A Workshop Summary*, accessed.
[270] Rockhill, Newman, and Weinberg. "Use and Misuse of Population Attributable Fractions."

The formula noted by Hanley that is based on the prevalence of exposure (e.g. obesity) in the population is the most well-known formula for calculating PAFs.[271] Often referred to as the "Levin formula" after M.L. Levin who first proposed the formula in 1953, this formula is based on only two levels of exposure to a given risk factor in the population, and requires the use of unadjusted RRs in order to avoid bias in the PAF estimate:[272]

$$PAF = [P(RR–1)] / [P(RR–1)] + 1$$

where:
P = the proportion or prevalence of the risk factor in the population, and
RR = the relative risk of the disease of interest.
The number 1 signifies the RR of the reference (e.g. normal weight) group.

The above formula is widely used incorrectly with adjusted RRs in the epidemiological literature. For example, some previous attempts to calculate the burden of obesity in Canada, such as the studies by Birmingham et al. and Luo et al., have used Levin's formula with adjusted RRs found in the epidemiological literature.[273] However, according to Rockhill et al., Flegal et al., and others, the Levin formula is only appropriate for use with relative risks that are *unadjusted* for confounding—which is not the case for almost all RRs found in the epidemiological literature—and when only two exposure categories, rather than multiple-exposure categories are used.[274] In fact, Rockhill et al. note: "Probably the most common error [in calculating PAFs] is the use of adjusted relative risks in formula 3 [the Levin formula]."[275]

Steenland and Armstrong also state: "[The Levin] formula is strictly valid only when there is no confounding or effect modification affecting the RR. In the presence of confounding, this estimate is biased, sometimes appreciably so, even if the RR in this formula has been adjusted for confounding."[276] Flegal concurs, noting: "It is important to adjust relative risk estimates for confounding factors such as age and gender that are associated with both [disease] and obesity. However, when relative risks are adjusted for confounding factors, it is also necessary to use properly adjusted estimators of attributable fraction to avoid bias." [277]

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[271] Hanley. "A Heuristic Approach to the Formulas for Population Attributable Fraction."
[272] Levin, M.L. "The Occurence of Lung Cancer in Man," *Acta Unio Internationalis Contra Cancrum*, 1953, vol. 9: 531-535.
[273] Birmingham, Muller, Palepu, Spinelli, and Anis. "The Cost of Obesity in Canada." Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."
[274] Flegal, Williamson, and Graubard. "Using Adjusted Relative Risks to Calculate Attributable Fractions." Rockhill, Beverly, Clarice Weinberg, Robert A. Hahn, Nancy D. Barker, Steven M. Teutsch, Elaine Eaker, Waldemar Sosniak, and Nancy Krieger. "Error in Population Attributable Risk Calculation," *Epidemiology*, 1996, vol. 7, no. 4: 453-454.
[275] Rockhill, Newman, and Weinberg. "Use and Misuse of Population Attributable Fractions." p. 16.
[276] Steenland, and Armstrong. "An Overview of Methods for Calculating the Burden of Disease Due to Specific Risk Factors." p. 513.
[277] Flegal, Williamson, and Graubard. "Using Adjusted Relative Risks to Calculate Attributable Fractions."

According to Hanley, it is also incorrect to calculate PAFs for specific exposure categories (e.g. overweight, obese class 1, etc.) by using the Levin formula separately for each level of exposure and then summing the results to get a single PAF. [278] Hanley discusses the bias that results from incorrectly applying the Levin formula in this way, and he recommends the use of the case-based method to correctly calculate PAFs for individual exposure levels.

Thus, the Levin formula is not applicable in complex situations where there are multiple exposure levels (e.g. overweight, obesity classes 1–3), in the presence of confounding by factors such as gender or age, or when the RRs have been adjusted. In this case, since age is associated with both the exposure (obesity) and the outcome (disease), its confounding effects must be controlled for in the analysis, and using Levin's formula is not sufficient for this purpose. Thus, as noted, where possible the case-based formula was used to calculate PAFs in this report. Caution should be used, therefore, when comparing the estimates of PAF calculated in this report with previous estimates that are based on the Levin formula.

According to Rockhill et al., Hanley, and others, PAF is correctly calculated in situations where there are multiple levels of exposure or where there is confounding, by using the "case-based" formula originally developed by O. S. Miettinen.[279] Therefore, to calculate PAFs in this report for the health conditions identified in the CCHS, the following formula (shown for only one level of exposure, for explanatory purposes) was used:

$$pd\ (RR-1/RR)$$

Where:
pd = the proportion of illness *cases* (e.g. of diabetes) in the population exposed to the risk factor (e.g. obesity), and
RR = the adjusted relative risk (age-adjusted in the case of this study).

According to Rockhill et al., this formula produces internally valid estimates when confounding exists and when, as a result, adjusted relative risks must be used.[280] This formula was used by Katzmarzyk and Janssen in their report on the economic costs of obesity and physical activity in Canada, along with the above rationale from Rockhill et al.[281] However, they used the formula incorrectly:—Instead of pd representing the proportion of illness cases exposed to the risk factor (e.g. proportion of illness cases in the obese population), as the formula was intended to do, the authors substituted the prevalence of obesity in the population. According to Rockhill et al., this is not a correct use of the formula.

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[278] Hanley. "A Heuristic Approach to the Formulas for Population Attributable Fraction."
[279] Miettinen, O.S. . "Proportion of Disease Caused or Prevented by a Given Exposure, Trait or Intervention," *American Journal of Epidemiology*, 1974, vol. 99: 325-332. Rockhill, Newman, and Weinberg. "Use and Misuse of Population Attributable Fractions."
[280] Rockhill, Newman, and Weinberg. "Use and Misuse of Population Attributable Fractions."
[281] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

The more complex formula used to calculate PAFs in this report, which is based on the above basic formula, includes the multiple exposure categories (e.g., overweight, obese class 1, obese classes 2–3):

$$\sum_i \text{pd}_i (\text{RR}_i - 1) / \text{RR}_i$$

Where:
i is the exposure category (overweight, obese class 1, etc.)
$\sum_i$ – indicates sum
RR_i is the (adjusted) relative risk for the ith exposure category (relative to the unexposed stratum), and
pd_i is the proportion of total cases in the population exposed to the ith exposure category[282]

In sum, the case-based method involves the calculation of stratum-specific PAFs for each exposure category, which are then summed.[283]

The population attributable fractions (PAF) were then used in a next step to estimate the proportion of the *costs* of each illness that could be attributed to excess weight. This was done by multiplying the PAF for each disease by the cost of that disease to the health care system ("direct costs" like hospital, drug, and physician costs), and to the economy (indirect productivity losses due to short- and long-term disability). In Canada, these total disease costs by diagnostic category are found in the Public Health Agency of Canada's *Economic Burden of Illness in Canada.*[284] (Premature death costs had to be calculated separately as explained in detail in Part 2, Chapter 5.6.1 of this report).

The key point here is that Levin's formula ideally should not be used with the adjusted relative risks generally available in the epidemiological literature, even though this has, unfortunately, been the most common methodology used to date in cost of obesity studies. Indeed, as noted, because of the lack of data we have had to employ this partially adjusted method in order to estimate costs of gallbladder disease and several cancer sites attributable to excess weight. However, in order to avoid the methodological difficulties outlined above and the resulting biases engendered by use of the Levin formula with adjusted RRs, the Miettinen case-based formula was used in this report to calculate PAFs for diabetes, hypertension, heart disease, stroke, osteoarthritis, asthma, and depression for each of the three age groups (15-34 years, 35-64 years, and ≥65 years) by gender.

The biggest challenge in applying this method is the need for samples of sufficient size to provide statistically valid results for each of these subgroups. Unfortunately, many of the obese classes 2 and 3, and age group results based on the available directly measured BMI data from 2004 CCHS cycle 2.2 had higher sampling variability and coefficients of

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[282] Rockhill, Newman, and Weinberg. "Use and Misuse of Population Attributable Fractions."
[283] Hanley. "A Heuristic Approach to the Formulas for Population Attributable Fraction."
[284] *Economic Burden of Illness in Canada (EBIC), 2000*.

variation than are desirable, and we have had to add cautionary notes in several places concerning interpretation of results. The 2004 CCHS, however, was used for diabetes, hypertension, and heart diease because directly measured BMI is considerably more reliable in general than self-reported BMI. Despite these data challenges, however, we hope that our determined use of the more analytically correct methods where possible— using directly measured BMI data and adjusted self-reported data from the 2004 and 2005 CCHS and the Miettinen case-based formula rather than the Levin formula— will spur collection of needed data from larger sample sizes in the future.

As well, the CCHS survey data used in this report were limited by the type of diagnosed diseases that CCHS respondents were asked about, and the CCHS was therefore not useful in estimating the fraction of cases attributable to obesity of site-specific obesity-related cancers or of gallbladder disease, which has a particularly strong association with obesity. Therefore despite our best efforts at methodological rigour, it was necessary to use published figures that rely on the Levin formula to estimate costs for most cancer sites and for gallbladder disease.

In addition, population health surveys like the CCHS can obviously only provide data on living patients and cannot therefore be used directly to estimate mortality-related relative risks and PAFs attributable to obesity. For estimates of premature death costs, we have also therefore relied on published estimates from the World Health Organization that have generally used the Levin formula to estimate PAFs.

For all these reasons, the cost estimates presented in this report cannot pretend to a precision that is simply not possible at present given existing data availability and CCHS sample sizes, particularly for directly measured BMI data. As well, the data on specific cancer sites, gallbladder disease, and premature mortality are particularly subject to bias, due to widespread use of the Levin formula with adjusted RR estimates, and must therefore be treated with particular caution. It is hoped that data collection for specific obesity-related types of cancers can be expanded in Canada, and that in the future the cost estimates can be updated with greater accuracy as better data become available. Detailed explanations of the methodologies used to estimate RRs, PAFs, and costs for specific cancer sites, gallbladder disease, and premature death attributable to obesity are located in Part 2, Chapters 5.4.3, 5.4.5, and 5.6.1.

4.2.3.6 Uncertainties

Uncertainties are generally unknown factors that may have an effect on the outcome. For example, if obesity were eliminated in the population, other factors such as lack of physical activity or poor diet might prevent some of the benefits of obesity reduction from being realized. Blakely et al. note that uncertainties can be caused by limited data, confounders that cannot be assessed independently, unknown time lags such as the length

of time it takes for an improvement in risk factors to manifest as a change in outcomes, and other contextual factors.[285]

In epidemiology studies, uncertainty is sometime expressed as a range of estimates indicated by a confidence interval (CI) showing high and low estimates.[286] Confidence intervals (usually at the 95% confidence level) are sometimes presented as part of RR estimates, and hence require separate PAF calculations for both the high and low estimates in addition to the PAF that is based on the reported RR. According to Flegal et al., PAF confidence intervals are often particularly wide, because a small variation in RR can produce a large variation in PAF.[287]

Another method for estimating uncertainties, used less often, is to perform a sensitivity analysis, as used by Birmingham et al. in the first systematic assessment of obesity costs in Canada published in 1999. Birmingham et al. varied each PAF (and hence each disease-specific health care cost) by ±20% of the mean estimate in order to assess the potential impact of uncertainties on their total obesity cost estimates.[288]

Time, space, and resources do not allow a full explication here of the complexities involved in estimating uncertainties, calculating confidence intervals, and qualifying the presentation of results based on RRs and PAFs to account for such uncertainties. For a detailed explanation and exploration of the mathematical and statistical issues involved in calculating confidence intervals and estimating and incorporating uncertainties, please see "Standard Errors for Attributable Risk for Simple and Complex Sample Designs" by U.S. National Cancer Institute researchers Barry Graubard and Thomas Fears,[289] and "A Review of Adjusted Estimators of Attributable Risk" by Jacques Benichou of the University of Rouen in France.[290]

Here we confine ourselves of necessity to a simple acknowledgement of key uncertainties and of data limitations based on limited sample sizes (particularly in the 2004 CCHS that has the most recent directly measured BMI data) and other factors noted above, and we provide a brief overview of the policy relevance of dealing with uncertainties, as summarized in the following citations from Health Canada and the World Health Organization. As well—in the caveat below—we summarize some of the key issues involved in dealing with uncertainties arising from sampling variabilities and errors based on our use of Statistics Canada's CCHS data.

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[285] Blakely, Hales, and Woodward. *Poverty: Assessing the Distribution of Health Risks by Socioeconomic Position at National and Local Levels*, accessed.

[286] Briggs, Andrew H., Bernie J. O'Brien, and Gordon Blackhouse. "Thinking Outside the Box: Recent Advances in the Analysis and Presentation of Uncertainty in Cost-Effectiveness Studies," *Annual Review of Public Health*, 2002, vol. 23, no. 377-401.

[287] Flegal, K.M., D.F. Williamson, E.R. Pamuk, and H.M. Rosenberg. "Estimating Deaths Attributable to Obesity in the United States," *American Journal of Public Health*, 2004, vol. 94, no. 9: 1486-1489.

[288] Birmingham, Muller, Palepu, Spinelli, and Anis. "The Cost of Obesity in Canada."

[289] Graubard, Barry I., and Thomas R. Fears. "Standard Errors for Attributable Risk for Simple and Complex Sample Designs," *Biometrics*, International Biometric Society, 2005, vol. 61, no. 3: 847-855.

[290] Benichou. "A Review of Adjusted Estimators of Attributable Risk."

The "precautionary principle," which is used by Health Canada, is an example of the Canadian government's commitment to incorporate uncertainty into decision making. Health Canada's argument, cited earlier, is that "it will often be desirable to act on the basis of high, or even moderate probabilities, on what has been called 'a burden of prudence' rather than 'a burden of proof.' [.... I]t should be recognized that conclusive evidence of harm or benefit to health is often an unrealistic requirement."[291]

UNESCO defines the precautionary principle as "an anticipatory model to protect humans and the environment against uncertain risks of human action."[292] Ian Shugart notes: "In public health, the precautionary principle is a well-established tenet and a core value."[293] It is founded in the Rio Declaration of 1992 and, in Canada, is legislated under the Canadian Environmental Protection Act.[294] Health Canada remarks:

> A key feature of health risk management is that decisions are often made against a backdrop of considerable scientific uncertainty. A precautionary approach to decision-making emphasizes the need to take timely and appropriately preventive action, even in the absence of a full scientific demonstration of cause and effect. This emphasis in decision-making is reflected in the final report of the Krever Commission of Inquiry, which concludes that a lack of full scientific certainty should not be used as a reason not to take preventive measures when reasonable evidence indicates that a situation could cause some significant adverse health effect.[295]

Colin Mathers et al., writing for WHO, suggest that, since mechanisms of causality are only partially known, "[i]t is therefore important to make judgements based on the best available science and data and document all assumptions and sources of uncertainty."[296] To the extent possible, this principle and practice have been followed throughout this report.

Important caveat

As noted, the bias in self-reported BMI data—hitherto used in all Canadian obesity cost

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[291] McKeown. *The Role of Medicine - Dream, Mirage or Nemesis?* Cited in Health Canada. *Canadian Handbook on Health Impact Assessment. Vol 3: Roles for the Health Practitioner*, accessed. p. F8.

[292] World Commission on the Ethics of Scientific Knowledge and Technology (COMEST) Expert Group. *The Precautionary Principle*, Paris: UNESCO, 2005; accessed December 2005; available from http://unesdoc.unesco.org/images/0013/001395/139578e.pdf.

[293] Shugart, Ian. *Summary of Discussion, Science Advisory Board Meeting, June 6-7, 2000 - The Precautionary Approach in Health Canada*, Health Canada, 2000; accessed February 2008; available from http://www.hc-sc.gc.ca/sr-sr/pubs/advice-avis/sab-css/meetings-reunions/june-juin00_e.html#12.

[294] Ibid., accessed.

[295] Health Canada. *Canadian Handbook on Health Impact Assessment. Vol 3: Roles for the Health Practitioner*, accessed.

[296] Mathers, Vos, Lopez, Salomon, and Ezzati. *National Burden of Disease Studies: A Practical Guide*, accessed. p. 129.

studies to date—is so great, and so significantly underestimates obesity prevalence, that a decision was made to use the most recently available directly measured Canadian BMI data, where possible, for the estimates of obesity costs in Alberta in this study. It was seen that Shields et al. found that the prevalence of obesity in Canada was a full 7.4 percentage points higher using the measured data than using the self-reported data (22.6% vs. 15.2%) — 8.8 percentage points higher for males (24.2% vs. 15.4%), and 6 percentage points higher for females (21.0% vs. 15.0%).[297] The difference was particularly large for seniors—15 percentage points higher for men aged ≥65 in the measured data compared to the self-reported data (31% vs. 16%), and 13 percentage points higher for women aged ≥65 (28% vs. 15%).

In Alberta, the gap between directly measured and self-reported data was even wider than for Canada as a whole. Thus, we saw earlier that the measured obesity rate for Alberta from the 2004 CCHS was 25.2%, while the self-reported rate in the 2005 CCHS was 15.8%—an enormous gap that would very significantly bias and underestimate obesity cost estimates for the province. However, it must be acknowledged that the substantial gain in precision and accuracy achieved by using the directly measured CCHS data— which was only possible for diabetes, hypertension, and heart disease—is compromised by the smaller sample size of the 2004 CCHS, which particularly affects breakdowns by obesity class, gender, and age. After a careful investigation of this trade-off in precision, it was decided to use the 2004 directly measured data to the extent possible, particularly to adhere to the most analytically correct methods, procedures, and data sources, and thus, hopefully, to make a case for the expansion of sample sizes in collecting directly measured data in the future. However, this choice does require explicit qualification and statement of the resulting uncertainties here.

Thus, estimates based on directly measured BMI data from Statistics Canada's 2004 CCHS, cycle 2.2 are particularly subject to uncertainties through sampling variabilities and errors. As noted earlier, confidence intervals calculated using bootstrapping methods are one way to estimate this sampling error. According to Graubard and Fears, however, common replication methods to calculate confidence interval variance such as bootstrap methods "rely on large samples to be consistent [and] could be biased for sparse exposures or in the case of small sample size."[298]

For this report, researchers obtained CCHS data from the Public Use Microdata Files, which do not include the necessary bootstrapping files. According to Statistics Canada: "The computation of coefficients of variation (or any other measure of precision) with the use of the bootstrap method requires access to information that is considered confidential and not available on the public use microdata file."[299] Therefore the magnitude of the sampling error in this report could not be estimated through this method.

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[297] Shields, Gorber, and Tremblay. "Estimates of Obesity Based on Self-Report Versus Direct Measures."
[298] Graubard, and Fears. "Standard Errors for Attributable Risk for Simple and Complex Sample Designs."
[299] Statistics Canada. *Canadian Community Health Survey 2004 User Guide for the Public Use Microdata File*, Catalogue no. 82M0024GPE, 2005; accessed February 2009; available from http://www.statcan.gc.ca/imdb-bmdi/document/5049_D8_T9_V1-eng.pdf. p. 63.

Instead, this report estimated sampling error by using tables of coefficients of variation (CVs), which Statistics Canada acknowledges are "considered crude."[300] The CCHS documentation on sampling variability includes tables for Canada and all of the provinces. For the 2004 CCHS, two different sets of CV tables were provided by Statistics Canada—one set for the total sample and a separate set for the sub-sample with direct measures of body mass index.[301]

The basis for the CVs is the standard deviation in the estimates derived from survey results. Due to the large range of estimates that can be produced from a survey, the standard deviation is expressed relative to the particular estimate to which it pertains. The CV of an estimate is obtained by dividing the standard deviation of the estimate provided in the CV tables by the estimate itself and is expressed as a percentage of the estimate.

Statistics Canada provides the following example in its documentation:

> For example, suppose hypothetically that one estimates that 25% of Canadians aged 12 and over are regular smokers and that this estimate is found to have a standard deviation of 0.003. Then the CV of the estimate is calculated as: (0.003/0.25) x 100% = 1.20%.[302]

Statistics Canada commonly uses CV results when analyzing data, and urges users producing estimates from CCHS data files also to do so. The sample size for the 2005 CCHS cycle 3.1 data was approximately 130,000 Canadians, and data used from the survey in this report to estimate RRs and PAFs for asthma, mood disorders, osteoarthritis, and stroke were all well within the acceptable CV limits ($0.0 - \leq 16.6$) for all categories and breakdowns examined in this study—gender, age, and BMI class (overweight, obesity class 1, and obesity classes 2 and 3)."[303]

However, the smaller sub-sample size from the 2004 CCHS cycle 2.2 that reports directly measured BMI data—and which was used in this report to assess obesity-related diabetes, hypertension, and heart disease RRs, PAFs, and costs—was further substantially reduced when the data were broken down by gender, age group, and obesity class. As a result, these 2004 CCHS-based data are therefore subject to high sampling variability in many of the sub-categories.

As noted, CCHS cycle 2.2 data were used in this report to estimate RRs and PAFs for diabetes, hypertension, and heart disease. The sample size for the 2004 CCHS cycle 2.2

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[300] Ibid., accessed.

[301] Ibid., accessed.

[302] Ibid., accessed. p. 49.

[303] It should be recalled that different cycles of the CCHS asked about different disease categories, so that data on diabetes, hypertension, and heart disease prevalence from the 2004 CCHS can be correlated with directly measured BMI data from the same 2004 survey, whereas RRs and PAFs for asthma, mood disorders, osteoarthritis, and stroke had to be calculated from the 2005 CCHS that asked questions about these illnesses.

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was much smaller than that for CCHS cycle 3.1, when 130,000 Canadians were surveyed. By contrast, approximately 35,000 Canadians were surveyed in 2004, but only about 15,000 Canadians aged ≥14 had their height and weight directly measured.[304] When this smaller sub-sample was further divided into gender, age, and obesity class categories, the sample sizes were further considerably reduced and produced data with high CVs for the three health conditions. Basically, the gender breakdowns showed marginal sampling variability (16.6%–≤33.3%), but the age breakdowns showed a sample variability of more than 33.3%, which is considered to be in the unacceptable range by Statistics Canada.

Despite these very serious data challenges, it was deemed important to use the 2004 CCHS data to the extent possible, since height and weight were directly measured in a sub-sample of the respondents. As discussed in Chapter 3.2 above, directly measured BMI data are vastly more accurate than self-reported BMI data. There we noted that the directly measured BMI data showed that 25.2% of Albertans were obese in 2004, while the self-reported BMI data showed that 15.8% of Albertans were obese in 2005.

In addition, the data were broken down by gender and age group for the three health conditions because gender and age are considered to be among the most important confounders of the obesity-illness association, and because such reporting by subgroup is highly recommended to account for key confounding factors. Confounding occurs when a third factor (e.g. age) is associated with both the exposure (e.g. obesity) and the outcome of interest (e.g. disease). As cited earlier, concerning age as a confounding factor, Katherine Flegal of the U.S. Centers for Disease Control and Prevention notes:

> The relative risks of obesity among the elderly may well be lower than among young or middle-aged people. Because of the high proportion of health conditions among the elderly and the high health care costs incurred by the elderly, estimates of the attributable fraction are sensitive to relative risks among the elderly.[305]

Also as cited earlier, the importance of using such methods to account for key confounding factors is confirmed by James Robins of the Harvard School of Public Health, who notes that failing to stratify by age when calculating PAFs for obesity can lead to an error of about 30 percent.[306]

Thus, the age and gender breakdowns for diabetes, hypertension, and heart disease are presented in this study for illustrative purposes, rather than for the purpose of providing statistically significant results, which is generally not possible given the data challenges described above. Since gender and age breakdowns are especially relevant to possible interventions designed to reduce obesity in the population, it is important to know which age groups can be targeted most effectively. As well, the breakdowns provided in this

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[304] Approximately 6,000 additional Canadian children and youth aged 2–13 also had their height and weight measured in 2004, but this age group was not included in the costing estimates.
[305] Flegal. "Editorial: Estimating the Impact of Obesity."
[306] Robins, James. In Institute of Medicine. *Estimating the Contributions of Lifestyle-Related Factors to Preventable Death: A Workshop Summary*, accessed.

report at least provide a potential framework for more statistically significant analysis as more accurate data based on larger directly measured BMI sample sizes hopefully become available in the future.

However, it is important to note these major caveats should not seriously affect the final cost estimates in this study, as the summary costs of overweight and obesity in Alberta were not broken down by gender or age group. Therefore, since age and gender costs were not used in the final costing estimates for Alberta, the summary costs can be considered more reliable, although the costs for diabetes, hypertension, and heart disease (based on the 2004 CCHS data) must still be interpreted with caution. However, as shown in Table 18 below, the PAFs estimated for these three health conditions are generally lower than or close to those used for these particular health conditions in other studies— indicating the conservative nature of the results presented. Based on these comparisons and analysis, the authors recommend that the overall estimated obesity cost results for Alberta presented in this report can be confidently used as the basis for policy interventions designed to reduce obesity and promote healthy weights in Alberta.

It must also be noted that this caveat on data reliability—and particularly on the high sampling variability for diabetes, hypertension, and heart disease data—was added to the report only at the review stage. While the caveat is briefly noted in several other relevant places in the study, it is not extensively repeated, so this extensive explanation should be kept in mind in the interpretation of all results.

4.2.4 Examples of relative risk ratios and population attributable fractions for obesity from the epidemiological literature

Having reviewed key methodological steps, definitions, and issues involved in assessing the links between obesity and particular health outcomes—which in turn is the essential process required to assess obesity costs—we now turn to the evidence itself, and to the available data on the health impacts of obesity.

Prior to a detailed illness-by-illness review of key evidence on obesity-related health outcomes—with particular emphasis on cancers (since this report is prepared for the Alberta Cancer Board)—we first look at a few key examples of summary evidence on relative risks and population attributable fractions from the literature—primarily in order to indicate the range of estimates currently in use. This initial summary is also needed in order to place the RRs and PAFs used in this particular Alberta study in the context of the comparable RR estimates most widely and currently in use, so that readers can easily and quickly see the degree to which our own RR estimates correspond with or differ from those in other studies.

We then turn to a disease-by-disease analysis of some of the available evidence on the health impacts of obesity in order to assess the disease-specific evidence more closely. Because PAFs can only be assessed on a disease-by-disease basis—since relative risks

differ by illness—such separate analysis of known obesity-related impacts by disease category is necessary to evaluate the differential strength of the association between obesity and illness for different diseases and also the differential robustness or variation in the evidence based on various data sources.

Table 18 below shows the relative risk ratios (RRs) for health conditions that are consistently, robustly, and quantifiably recognized as being associated with obesity, and population attributable fractions (PAF) where available, as provided in four major studies on obesity costs—two from Canada, one from England, and one from Australia. In addition, for comparative purposes, the RRs and PAFs used in this report for Alberta are included—but only for those obesity-related health conditions referenced and costed in these previous four reports. In fact, the table below includes only 11 of the 22 health conditions examined in this report, omitting most site-specific cancers that have been partially linked to obesity and that are a particular focus of this present report based on the interests and concerns of the Alberta Cancer Board. Although the studies used different methodologies and are therefore not strictly comparable, Table 18 serves to show the range of RRs and PAFs commonly found in the epidemiological literature.

1. ***In 1999, C. Laird Birmingham et al***. estimated the 1997 Canadian costs of obesity— the first such national study to be undertaken in Canada—using a definition of obesity as BMI ≥27. It should be recalled that the study by Birmingham et al. was published prior to Health Canada's and Statistics Canada's switch to the WHO and international standards that define obesity as a BMI of ≥30. Because some health effects are attributable to excess weight or overweight (at BMI levels below 30), the sample demonstrating links between obesity and illness outcomes in Birmingham et al. includes a larger number and proportion of individuals (producing correspondingly higher RRs and PAFs) than the later studies that use an obesity definition of BMI ≥30.[307]

   Birmingham et al. identified 10 comorbidities of obesity and their RRs from the medical and epidemiological literature: hypertension, coronary artery disease, stroke, type 2 diabetes, colon cancer, postmenopausal breast cancer, endometrial cancer, gallbladder disease, pulmonary embolism (sudden blockage in a lung artery), and hyperlipidemia (high cholesterol or triglyceride levels).

   Other studies, including this one, have not included the latter two health conditions in their reports for the reasons summarized by Katzmarzyk and Janssen: "Although we agree that obesity may have an impact on the additional illnesses included in the analysis of Birmingham et al., a lack of prospective studies documenting a relationship with obesity caused us to choose a more conservative approach for the current analysis."[308]

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[307] Birmingham, Muller, Palepu, Spinelli, and Anis. "The Cost of Obesity in Canada."
[308] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

Birmingham et al. selected RRs for each disease for use in their cost estimates based on the "most valid study" that they found in the literature. If more than one valid study was identified, they chose the study with the largest sample size and the longest follow-up.

The selected RRs in the Birmingham et al. study ranged from 1.14 for stroke to 4.37 for type 2 diabetes, and their PAFs estimated that over 50% of type 2 diabetes, over 30% of hypertension, and over 20% of gallbladder disease, endometrial cancer, and pulmonary embolism in Canada were attributable to obesity.

Obesity prevalence data were taken from self-reported results in the 1994–1995 National Population Health Survey (NPHS) for non-pregnant adults aged 20–64. The reference group used to compare obese with non-obese individuals was the group with a BMI below 27. Equal weighting was given for both men and women, except for postmenopausal breast and endometrial cancers, which clearly used only data for women.

Birmingham et al. estimated that the direct cost of obesity in Canada was $1.8 billion in 1997 ($1997).

2. ***In 2004, Katzmarzyk and Janssen*** estimated the 2001 economic costs of obesity in Canada, based on an obesity definition of BMI ≥30.[309] They identified 8 obesity co-morbidities: hypertension, coronary artery disease, stroke, type 2 diabetes, colon cancer, postmenopausal breast cancer, gallbladder disease, and osteoarthritis. Thus, they added osteoarthritis to the list of obesity-related conditions included by Birmingham et al., but omitted endometrial cancer, pulmonary embolism, and hyperlipidemia for the reasons stated above.

The RRs used by Katzmarzyk and Janssen for the eight health conditions considered, represented a summary of adjusted RRs taken from a meta-analysis of the international epidemiological literature. The adjustments made to estimate the summary RRs were not identified.

The summary RRs ranged from 1.45 for colon cancer to 4.50 for hypertension. PAFs (referred to by Katzmarzyk and Janssen as PARs—population attributable risks) estimated that over 34% of hypertension, and over 20% of type 2 diabetes and gallbladder disease in Canada could be attributed to obesity.

Obesity prevalence data for non-pregnant adults aged 20–64 were obtained from self-reported results in the 2000/2001 CCHS. Katzmarzyk and Janssen's data were not stratified by age or gender, and the reference group was not identified in their report.

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[309] Ibid.

Katzmarzyk and Janssen estimated the cost of obesity in Canada to be $4.3 billion dollars in 2001—$1.6 billion for direct costs and $2.7 billion for indirect costs (C$2001).

3. ***In 2001, the National Audit Office in the U.K.*** completed a report on the cost of obesity, defined as BMI ≥30, in England in 1998.[310] It identified RRs, stratified by gender, for 9 diseases: type 2 diabetes, hypertension, coronary artery disease (myocardial infarction), stroke, angina, colon cancer, ovarian cancer, osteoarthritis, and gallbladder disease.

   The RRs ranged from a low of 1.30 for stroke to a gender-specific high of 12.70 for type 2 diabetes among women. The RRs were taken from a review of the epidemiological literature, and mainly came from U.S. studies. The PAFs provided in the study estimated that 47% of type 2 diabetes, 36% of hypertension, and almost 30% of colon cancer in England could be attributable to obesity.

   Obesity prevalence data were for those aged 16 to 74, based on self-reported results, and the reference group was non-obese individuals (i.e. with a BMI of less than 30). The report particularly addressed obesity-prevention initiatives targeting children and youth, and noted that the only nationally representative British study of the height and weight of primary school children was undertaken in 1994.

   The U.K. report found that £2.6 billion was attributable to obesity in England in 1998, including £.5 billion for direct costs and £2.1 billion for indirect costs (£1998). This was the equivalent of about $3.8 billion—$.7 billion for direct costs and $3.1 billion for indirect costs in 1998 Canadian dollars.

4. ***Diabetes Australia*** sponsored a report on the economic costs of obesity, defined as BMI ≥30, in Australia ***in 2005***, which it later ***updated for 2008***.[311, 312] RRs were stratified by gender and two age groups—< 65 and ≥ 65— and provided for type 2 diabetes, cardiovascular diseases (hypertension, stroke, and coronary artery disease), osteoarthritis, and four types of cancer (breast, colon, uterine, and kidney). The RRs were taken from different studies found in the 1990–1999 literature, as reported by C. Mathers et al. in 1999.[313] The RRs ranged from a low 1.15 for stroke (men) to a high of 3.20 for type 2 diabetes (both men and women).

   New directly measured 2008 prevalence data and updated RRs resulted in substantially higher PAFs (referred to as AFs in the Diabetes Australia reports) compared to the 2005 PAFs, which were also based on directly measured data. As a result, PAFs for cardiovascular diseases in Australia rose from 13.5% in 2005 to

[310] National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, accessed.

[311] Access Economics. *The Economic Costs of Obesity*, accessed.

[312] Access Economics. *The Growing Cost of Obesity in 2008: Three Years On*, accessed.

[313] Mathers, C., T. Vos, and C. Stevenson. *The Burden of Disease and Injury in Australia*, Australian Institute of Health and Welfare, Catalogue no PHE17, 1999; accessed July 2008; available from http://www.aihw.gov.au/publications/index.cfm/title/5180.

21.3% in 2008. In 2005, the PAF was 14% for hypertension and 12% for coronary artery disease and stroke. This breakdown among different categories of cardiovascular disease was not given for 2008.

Similarly, the PAF for four types of cancer in Australia rose from 14.9% in 2005 to 20.5% in 2008. In 2005, the PAF for colon and kidney cancer combined was 13%, and for postmenopausal breast and endometrial cancer combined was 16%. The PAF for osteoarthritis rose from 14% in 2005 to 24.5% in 2008, and for type 2 diabetes the PAF more than doubled in this time period—from 10.8% to 23.8%.

Prevalence data were stratified by all age groups and by gender (but RRs were only stratified by aged under 65 and 65 and older). The reference group was individuals with normal weight (BMI <25).

The Australian studies found that the cost of obesity was $3.8 billion—$1.7 billion for direct costs and $2.1 billion for indirect costs—in 2005 (AU$2005), and $8.3 billion—$3.9 for direct costs and $4.4 billion for indirect costs—in 2008 (AU$2008)

A comparison of results from these five studies plus this present one, as seen in Table 18 below, shows the very wide range of possible RRs and PAFs that exist for some diseases, despite the careful review undertaken by the authors of all these studies of the available epidemiological literature on obesity-related relative risk ratios. Indeed, the comparison shows that there is not even agreement among highly reputable peer-reviewed studies on a complete list of illnesses for which obesity-related RRs and PAFs can be reliably quantified. Such differences and uncertainties will directly affect obesity cost estimates.

Among other things, the comparison quite dramatically demonstrates the importance and value of gender stratification in assessing relative risks (RRs)—which was not undertaken in the Birmingham et al. and the Katzmarzyk and Janssen Canadian studies cited above. Needless to say, such stratification and the consequent estimate of RRs will also in turn affect estimates of PAFs and obesity costs. For example, as seen in Table 18 below, the U.K. National Audit Office used obesity-related RRs for type 2 diabetes of 12.7 for women and 5.2 for men, compared to non-gender-specific RRs of 4.37 and 3.73, respectively, in the two Canadian studies cited above. Even gender and age stratification, while highly desirable, clearly does not eliminate substantial uncertainties and a very wide range of possible RR estimates, as evidenced by the Diabetes Australia use of an RR of 3.2 for both men and women under 65 and 65 and over—very much lower than the U.K. estimates (Table 18 below).

The last three columns of Table 18 also show the RRs and PAFs used in this study for those health conditions reported in the other studies. As noted, this comparison omits half of the health conditions examined in this report—particularly most site-specific cancers that have been partially linked to obesity. It is seen that many of the PAFs used in this study—with the notable exception of gallbladder disease—are lower than or fairly close to those used in the other studies—especially those studies from Canada and England.

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This indicates that the cost estimates in this present study can be regarded as reasonably conservative.

It is particularly interesting to compare the PAFs used in this study for the three diseases—hypertension, coronary heart disease, and diabetes—where estimates are based on the directly measured 2004 CCHS cycle 2.2 data, with estimates for those diseases in the other studies. In light of the detailed discussion on methodological and data issues above, including the major caveat on sampling variability, these are the illnesses where the greatest variance from other studies might be expected. Interestingly, the PAFs for those diseases used in this study are not as different from those in the other studies as might be anticipated, and are generally on the conservative side despite our use of directly measured BMI data that show considerably higher actual rates of obesity than self-reported data.

Thus, the PAF for both genders combined used in this study was lower for hypertension (22.8) than those used in the two Canadian studies (31.6 for Birmingham et al. and 34.0 for Katzmarzyk and Janssen) and the England study (36.0), but higher than that used in Australia in 2005 (14.0). The Australian PAF for 2008 combines hypertension, coronary heart disease, and stroke (21.3), and so is not comparable with the other studies that separate these diseases.

The PAF for both genders combined for coronary heart disease used in this report (14.7) was also lower than those used in the Katzmarzyk and Janssen report (15.4), Birmingham et al. report (17.9) and in England (18.0), and higher than that used in Australia in 2005 (12.0).

For diabetes, the PAF for both genders combined used in this study (36.9) was lower than those used in the Birmingham et al. (50.7) and England studies (47.0), but higher than those used in the Katzmarzyk and Janssen (28.6) and the Australia studies (2005–10.8, and 2008–23.8).

While time and resources did not permit a full analysis of the reasons underlying these variations, it is possible that our use of directly measured BMI data in this study—which yields higher obesity rates than self-reported data—was balanced by the more conservative results yielded by the method used here that accounted for confounders by aggregating separate data sets specific to gender, age, and obesity class. Based on a preliminary comparative analysis, at least, it does not appear that the high sampling variability in the 2004 CCHS results has compromised the comparability of the overall RRs, PAFs, and cost estimates in this study with those in the major studies referenced above and in Table 18 below. If anything, the results in this study are seen to be conservative.

PAFs for stroke and osteoarthritis in this study were estimated from 2005 CCHS cycle 3.1 data, which, as seen earlier, had a significantly larger sample size (130,000) than the 2004 CCHS (35,000 overall, and 15,000 for the directly measured BMI sub-sample). The PAF for both genders combined for stroke used in this study (4.7) was higher than the

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PAF used in the Birmingham et al. study (4.0), but lower than the PAFs used in the Katzmarzyk and Janssen (6.8), England (6.0) studies, and Australian study in 2005 (12.0).

For osteoarthritis, the PAF for both genders combined used in this study (13.3) was higher than that used by Katzmarzyk and Janssen (12.7) and England (12.0), and lower than those used in Australia (14.0 in 2005 and 24.5 in 2008).

For gallbladder disease, the PAF for both genders combined used in this report (45.5) was based on directly measured obesity prevalence data from the 2004 CCHS cycle 2.2 for Alberta, and RRs found in the epidemiological literature that were calculated by U.S. researchers using data from the Third National Health and Nutrition Examination Survey (NHANES III).[314] RRs for this study are not included in Table 18 because the RRs were for obese classes 1, 2, and 3 for each age group (aged <55 and ≥55) by gender, and a RR for total obesity was not provided. Therefore, it was not actually possible to calculate a PAF for total obesity for this study. The PAF for total obesity shown in the table was estimated *for comparison purposes only* by dividing the total gallbladder disease costs attributed to total obesity by the total direct costs of gallbladder disease in Alberta. The total cost of gallbladder disease attributed to total obesity was estimated by summing the gender-specific costs of gallbladder disease attributed to obese classes 1, 2, and 3 in each age group. (The separate RRs by obese class, gender, and age group used in this study are given in the section on gallbladder disease–Part 2, Chapter 5.4.5 of this report).

As seen in Table 18 below, the PAF for both genders combined for gallbladder disease reported for this study (45.5) is much higher than those used by Birmingham et al. (20.6), Katzmarzyk and Janssen (25.5), and in England (15.0). (Gallbladder disease was not included in the Australian reports.)

There are several reasons why the PAF in this report may be higher than those used in the other studies. One reason for the higher PAF is that gallbladder disease is more prevalent in age groups aged ≥60, and all adult age groups were included in this report. Birmingham et al. and Katzmarzyk and Janssen did not include adults over the age of 64, and the England study did not include adults over the age of 74. Another reason that the PAF in this study is so much higher might lie in our use of directly measured BMI data which yield obesity prevalence rates significantly higher than those based on self-reported data.

A third reason that the PAF in this study might be higher than the others is because the RRs used in this study for aged <55—which as noted above are not included in Table 18—for obese classes 1–2 for males (4.1 and 6.8) and obese classes 2–3 for females (4.3 and 5.2) are considerably higher than the RRs used in the other studies (1.8 in England, 1.85 by Birmingham et al., and 3.33 by Katzmarzyk and Janssen). In addition, the RR

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[314] Must, A., J. Spadano, and E.H. Coakley. "The Disease Burden Associated with Overweight and Obesity," *Journal of the American Medical Association*, 1999, vol. 282: 1523–1529.

provided for males aged <55 for obese class 3 is extremely high (21.1). The RRs used in this study for the obese classes 1–3 for aged ≥55 are within the range of the other studies.

For this report, 14 specific types of cancer with demonstrated links to obesity were examined. Of these 14 cancer types, five are referenced in one or more of the five other studies listed in Table 18 below for the purpose of comparing PAFs. The PAFs used in this study for colon cancer, kidney cancer, postmenopausal cancer, and ovarian cancer were drawn from a Canadian study in the epidemiological literature.[315] The PAF for endometrial cancer was estimated using a RR from the epidemiological literature and directly measured BMI prevalence data from the 2004 CCHS cycle 2.2 for Alberta.[316]

Some PAFs are not directly comparable. For example, Birmingham et al. and the Australia (2005) study provide PAFs for colorectal cancer (both colon cancer and rectal cancer combined), while the Katzmarzyk and Janssen and England studies provide PAFs for colon cancer specifically. This study used separate PAFs for both colon cancer and rectal cancer, but only the PAF for colon cancer is shown in the table. The PAF for colon cancer (12.2) used in this study (which as noted used a separate PAFs for rectal cancer— 8.9) was lower that that used in England (29.0) and Australia in 2005 (13.0 for colorectal cancer) but higher that those used by Birmingham et al. (4.7 for colorectal cancer) and Katzmarzyk and Janssen (6.2 for colon cancer). The 2008 Australian study PAF combined colorectal, kidney, postmenopausal breast, and endometrial cancers (20.5) and, therefore, is not comparable with the other studies.

For kidney cancer, the PAF used in this study (20.7) was higher than that used in Australia (13.0) in 2005, which was the only other study that examined kidney cancer separately.

The postmenopausal breast cancer PAF used in this study (8.5) was lower than that used by Birmingham et al. (9.1), and Australia in 2005 (16.0), but higher than the PAF used by Katzmarzyk and Janssen (6.5). Postmenopausal breast cancer was not included in the England study.

The endometrial cancer PAF used in this study (22.1) was lower than that used by Birmingham et al. (26.6), but higher than that used in England (14.0) and Australia in 2005 (16.0). Katzmarzyk and Janssen did not include endometrial cancer in their study of the costs of obesity in Canada.

The PAF for ovarian cancer used in this study (11.7) was lower than that used in England (13.0), which was the only other study to include ovarian cancer.

[315] Pan, Sai Yi, Kenneth C. Johnson, Anne-Marie Ugnat, Shi Wu Wen, Yang Mao, and Canadian Cancer Registries Epidemiology Research Group. "Association of Obesity and Cancer Risk in Canada," *American Journal of Epidemiology*, 2004, vol. 159, no. 3: 259-268.

[316] Luo, Wei, Howard Morrison, Margaret de Groh, Chris Waters, Marie DesMeules, Elaine Jones-McLean, Anne-Marie Ugnat, Sylvie Desjardins, Morgan Lim, and Yang Mao. "The Burden of Adult Obesity in Canada," *Chronic Diseases in Canada*, 2007, vol. 27, no. 4: 135-144.

Table 18. Relative risk ratios (RRs) and population attributable fractions (PAF) from five example studies on obesity costs

	Canada		Canada		England			Australia						Canada		
	Birming-ham, et al., C$1997		Katzmar-zyk & Janssen, C$2001		National Audit Office (used by WHO), £1998			Diabetes Australia (used by WHO), AU$2005 & $AU2008						Cost of Obesity in Alberta (this report), C$2005		
	RR	PAF %	RR	PAF %	RR		PAF %	RR				PAF %		RR		PAF %
Total cost of obesity	$1.8 billion – Total direct cost (C$1997) – for obesity				£2.6 billion – £.5 billion-direct; £2.1 billion-indirect) (£1998) – for obesity			2005 - $3.8 billion – $1.7 billion-direct; $2.1 billion-indirect (AU$2005) 2008 -– $8.3 billion $3.9 billion-direct; $4.4 billion-indirect (AU$2008) – for obesity						For Alberta: $1.2 billion –$443 million direct + 182 million-caregiving; $559 million indirect (C$2005) – for overweight and obesity		
Gender	Both combined		Both combined		M	F	Both	M		F		Both (2005)	Both (2008)	M	F	Both
Age group	20–64		20–64		16–74			<65	≥65	<65	≥65	All ages		≥15		
Comorbidity:																
Hypertension	2.51	31.6	4.50	34.0	2.60	4.20	36.0	2.35	2.35	2.35	2.35	14.0	21.3 (CVD com-bined)	2.41	2.25	22.8
Coronary heart disease	1.72	17.9	2.24	15.4	1.50	3.20	18.0	1.80	1.20	1.20	1.25	12.0		2.20	1.43	14.7
Stroke	1.14	4.0	1.50	6.8	1.30	1.30	6.0	1.50	1.15	1.60	1.20	12.0		1.07	1.41	4.7
Type 2 diabetes	4.37	50.7	3.73	28.6	5.20	12.70	47.0	3.20	3.20	3.20	3.20	10.8	23.8	4.36	3.21	36.9
Colon cancer	1.16	4.7*	1.45	6.2	3.00	2.70	29.0	1.40	1.40	1.40	1.40	13.0*	20.5	–	–	12.2
Renal (kidney) cancer	–	–	–	–	–	–	–	1.00	1.00	1.50	1.50	13.0	(4 cancer com-bined)	–	–	20.7
Postmenopau-sal breast cancer	1.31	9.1	1.47	6.5	na	–	–	na	na	1.30	1.30	16.0		–	–	8.5
Endometrial cancer	2.19	26.6	–	–	na	2.52	14.0	na	na	1.75	1.75	16.0		–	–	22.1

	Canada				England			Australia						Canada		
	Birming-ham, et al., C$1997		Katzmar-zyk & Janssen, C$2001		National Audit Office (used by WHO), £1998			Diabetes Australia (used by WHO), AU$2005 & $AU2008						Cost of Obesity in Alberta (this report), C$2005		
	RR	PAF %	RR	PAF %	RR		PAF %	RR				PAF %		RR		PAF %
Ovarian cancer	–	–	–	–	na	1.70	13.0	na	na	–	–	–	–	–	–	11.7
Gallbladder disease*	1.85	20.6	3.33	25.5	1.80	1.80	15.0	–	–	–	–	–	–	–	–	45.5
Osteoarthritis	–	–	1.99	12.7	1.90	1.40	12.0	2.40	2.40	2.40	2.40	14.0	24.5	1.75	1.73	13.3
Angina	–	–	–	–	1.80	1.80	15.0	–	–	–	–	–	–	–	–	–
Pulmonary embolism	2.39	29.8	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Hyperlipide-mia	1.41	11.2	–	–	–	–	–	–	–	–	–	–	–	–	–	–

Notes: * Note that Birmingham et al. and the Australia (2005) study provide PAFs for colorectal cancer (both colon cancer and rectal cancer), while this study as well as the the Katzmarzyk and Janssen and England studies provide PAFs for colon cancer specifically. M – male, F – female; na – not applicable: – i.e. not included in the study; Birmingham et al. – obesity BMI = ≥27, for the other reports – obesity BMI = ≥30; For this report all of the co-morbidities examined are not shown here. For the other reports all of the co-morbidities are shown.

Sources: Birmingham, C. Laird, Jennifer L. Muller, Anita Palepu, John J. Spinelli, and Aslam H. Anis. "The Cost of Obesity in Canada," *Canadian Medical Association Journal*, 1999, vol. 160, no. 4: 483-488.
Katzmarzyk, Peter T., and Ian Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update," *Canadian Journal of Applied Physiology*, 2004, vol. 29, no. 1: 90-115.
National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, National Audit Office, 2001; accessed July 2008; available from www.nao.org.uk/publications/nao_reports/00-01/0001220.pdf. Access Economics. *The Economic Costs of Obesity*, Diabetes Australia, 2006; accessed June 2008; available from http://www.accesseconomics.com.au/publicationsreports/showreport.php?id=102; Access Economics. *The Growing Cost of Obesity in 2008: Three Years On*, Diabetes Australia, 2008; accessed September 2008; available from http://diabetesaustralia.com.au/PageFiles/7832/FULLREPORTGrowingCostOfObesity2008.pdf.

4.3 Type 2 diabetes

Diabetes mellitus, or type 2 diabetes, is characterized by high blood glucose (sugar) levels that result from an inadequate production of insulin—a hormone vital to metabolism—or an inadequate response of target cells to insulin, or both.[317] About 40% of adults who have type 2 diabetes develop secondary health conditions, such as stroke, coronary heart disease, kidney damage, peripheral nerve problems that may necessitate limb amputations, and eye problems that can result in blindness.[318] According to Alberta Health and Wellness: "People with diabetes are 2.5 times more likely to have heart disease, 11 times more likely to have kidney failure, 17 times more likely to have an amputation, and 8 times more likely to undergo bypass surgery" than those without diabetes.[319]

Conventional estimates of mortality, disability, and health expenditures attributable to diabetes are almost always under-estimated. This is because of the convention of classifying illnesses and causes of death by principal diagnosis. Since diabetes so often leads to other serious illnesses that may cause death, diabetes is almost certainly under-reported on death certificates, in medical reports, and in administrative data on health care utilization. According to Health Canada:

- There were 5,447 deaths in 1996 for which diabetes was certified as the underlying cause. This ranks diabetes as the seventh leading cause of death in Canada. However, the actual number of deaths for which diabetes was a contributing factor is probably five times this number.[320,321]

The nature and progression of diabetes result in very substantial social and economic costs. Staving off the most serious disease consequences of diabetes depends on managing the disease effectively. But self-management involves a complex array of education, planning skills, and access to a broad range of medications, medical devices, and supplies to regulate glucose and cholesterol levels in the blood.[322]

Of the three forms of diabetes—type 1, type 2, and gestational diabetes—type 2 diabetes is responsible for approximately 90% to 95% of diabetes prevalence.[323] Type 1 diabetes and gestational diabetes are not statistically related to obesity. Type 1 diabetes is insulin-dependent, usually begins in childhood, and is responsible for about 5% to 10% of all diabetes. It develops

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[317] Access Economics. *The Economic Costs of Obesity*, accessed.

[318] Public Health Agency of Canada (PHAC). *Diabetes. Facts and Figures*, n.d.; accessed July 2008; available from http://www.phac-aspc.gc.ca/ccdpc-cpcmc/diabetes-diabete/english/index.html.

[319] Alberta Health and Wellness. *Keeping Albertans Healthy*, 2004; accessed September 2008; available from http://www.health.alberta.ca/public/in_healthy.pdf.

[320] Health Canada, *Diabetes in Canada*, available at: http://www.hc-sc.gc.ca/hpb/lcdc/publicat/diabet99/d02_e.html.

[321] Public Health Agency of Canada (PHAC). *Diabetes. Facts and Figures*, accessed.

[322] Canadian Diabetes Association. "Canadian Diabetes Association 2008 Clinical Practice Guidelines for the Prevention and Management of Diabetes in Canada," *Canadian Journal of Diabetes*, 2008, vol. 32, no. Supplement 1: S1-S201.

[323] Alberta Health and Wellness. *Alberta Diabetes Strategy 2003-2013*, 2003; accessed September 2008; available from http://www.health.alberta.ca/public/dis_diabetesstrategy.pdf.

when the cells that produce insulin are destroyed by the body's immune system.[324] Gestational diabetes occurs in approximately 3% to 5% of pregnant women not previously diagnosed with diabetes. It is generally is resolved after pregnancy, but women who have had the condition are at increased risk for developing type 2 diabetes.[325]

Edward Ng of Statistics Canada et al. recently created an algorithm to differentiate between the three types of diabetes in the CCHS, which asks respondents whether they have been diagnosed with diabetes in general.[326] The algorithm is based in part on subsequent questions concerning medication use and age at the time of diagnosis. The authors found that 95% of respondents to the 2000/01 CCHS cycle 1.1 who said they had diagnosed diabetes, in fact had type 2 diabetes, about 5% had type 1, and about 1% had gestational diabetes. The authors note that application of the algorithm to CCHS cycle 3.1 yielded type 1 and 2 diabetes prevalence estimates similar to those derived from cycle 1.1.

Of all chronic diseases, type 2 diabetes has the strongest association with obesity.[327] Obesity often results in insulin resistance and impaired glucose tolerance, and is considered to be the principal factor in the development of type 2 diabetes.[328] In addition to obesity, other leading risk factors for type 2 diabetes are physical inactivity, diet, age, and genetics. However, James et al. note that "body-weight gain *per se* enhances insulin resistance, and thus physical inactivity is not the sole explanation. The development of insulin resistance is a powerful predictor of excess levels of triglycerides in the blood and of the propensity to develop type II diabetes."[329] James et al. describe the basic physical mechanisms through which obesity leads to type 2 diabetes:

> Type II diabetes develops when the pancreatic capacity to generate insulin cannot maintain the markedly increased demand induced by insulin resistance. Insulin resistance itself is affected not only by increases in weight, particularly if the extra energy is stored in abdominal, i.e. visceral, fat, but also by dietary composition. Dietary fat induces insulin resistance and there is increasing interest in the possibility that rapidly absorbed carbohydrates, which cause sudden increases in concentrations of blood glucose, place extra demands on the pancreas. Adipose tissue itself, particularly visceral adipose tissue, secretes cytokines such as interleukin-6 (IL-6) and tumour necrosis factor (TNFa) which are recognized to be important inducers of insulin resistance. Circulating adiponectin, an adipocyte-derived hormone which markedly improves insulin sensitivity is reduced as the fat cells expand with body-weight gain.[330]

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[324] Public Health Agency of Canada (PHAC). *Diabetes in Canada: Highlights from the National Diabetes Surveillance System 2004 - 2005*, 2008; accessed October 2008; available from http://www.phac-aspc.gc.ca/publicat/2008/dicndss-dacsnsd-04-05/pdf/dicndss-04-05-eng.pdf.

[325] Alberta Health and Wellness. *Alberta Diabetes Strategy 2003-2013*, accessed.

[326] Ng, Edward, Kaberi Dasgupta, and Jeffrey A. Johnson. "An Algorithm to Differentiate Diabetic Respondents in the Canadian Community Health Survey," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2008, vol. 19, no. 1: 1-9.

[327] National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, accessed.

[328] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[329] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)." p. 540.

[330] Ibid. p. 550.

Estimates of the proportion of type 2 diabetes prevalence that can be attributed to obesity have a large variance across different studies. As previously noted, the WHO *Comparative Quantification of Health Risks* study found that in the America-A subregion (consisting of Canada, United States, and Cuba), 83% of type 2 diabetes in males, and 88% in females, aged ≥30 years, could be attributed to obesity (BMI ≥30).[331] WHO has also estimated that approximately 64% of type 2 diabetes in U.S. men and 74% in U.S. women could be avoided if everyone had a BMI below 25 kg/m^2.[332] U.K. researcher Peter Kopelman writes that 90% of type 2 diabetes can be found in individuals who have a BMI of >23, which he describes as at the cut point for overweight classification.[333]

As seen in Table 18 above, other studies have produced more modest estimates of the proportion of type 2 diabetes prevalence attributable to obesity—23.8% in Diabetes Australia's estimate for 2008, 28.6% in Katzmarzyk and Janssen's estimate for Canada for 2001, 47% in the U.K. National Audit Office's estimate for England in 1998, and 50.7% in Birmingham et al.'s estimate for Canada in 1997. The first three of these studies define obesity as BMI ≥30, while the last defines obesity as BMI ≥27.

With the higher prevalence of obesity in the U.S., it stands to reason that U.S. studies will attribute higher proportions of diabetes prevalence to obesity than those in other countries. As well, Statistics Canada found that Canadians have a significantly higher risk of diabetes even at a lower BMI between 25 and 30.[334] Therefore, it also stands to reason that studies like Kopelman's and that of Birmingham et al. will find higher overall proportions of diabetes prevalence attributable to overweight and obesity to the degree that the cut-off for analysis is lower than BMI ≥30.

Researchers Edward W. Gregg of the Centers for Disease Control and Prevention et al. examined five cross-national surveys that were conducted in the U.S. between 1960 and 2000, to determine trends in the prevalence of diagnosed and undiagnosed diabetes according to obesity levels among adults aged 20–74 years.[335] All of the surveys directly measured BMI, and undiagnosed diabetes was identified through a fasting glucose test that was administered beginning in 1976. Gregg et al. found that the prevalence of diagnosed diabetes increased in the population at large (i.e. in all BMI groups) from 1.8% in 1960 to 5.8% in 2000.[336]

They also found that the diagnosed diabetes rate among obese individuals in class 1 (BMI 30.0–

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[331] Ibid.

[332] Cited in Hubert, H.B., M. Feinleib, P.M. McNamara, and W.P Castelli. "Obesity as an Independent Risk Factor for Cardiovascular Disease: A 26-Year Follow-up of Participants in the Framingham Heart Study," *Circulation. Journal of the American Heart Association*, 1983, vol. 67: 968-977.

[333] Kopelman, Peter G. "Health Risks Associated with Overweight and Obesity," *Obesity Reviews*, 2007, vol. 8, no. Suppl. 1: 13-17.

[334] Gilmore, Jason, "Body Mass Index and Health," *Health Reports,* Statistics Canada, Catalogue no. 82-003, volume 11, no. 1, Summer, 1999, 31-43.

[335] Gregg, Edward W., Betsy L. Cadwell, Yiling J. Cheng, Catherine C. Cowie, Desmond E. Williams, Linda Geiss, Michael M. Engelgau, and Frank Vinicor. "Trends in the Prevalence and Ratio of Diagnosed to Undiagnosed Diabetes According to Obesity Levels in the U.S.," *Diabetes Care*, 2004, vol. 17, no. 12: 2806-2812.

[336] Ibid.

34.9) increased from 2.3% in 1960 to 6.7% in 2000, but the most significant increase was seen among severely obese individuals (BMI ≥35) whose rate of diabetes increased from 4.9% in 1960 to 15.1% in 2000. In the 2000 survey sample, individuals with BMI ≥35 accounted for 13.5% of the overall population, but for 36% of the population with diabetes.

Gregg et al. also estimated that approximately 30% of the overall diabetic population was not diagnosed as diabetic in 2000.[337] However, in the most severely obese group (BMI ≥35), there was a sharp increase over time in the proportion diagnosed as diabetic—with the percentage of undiagnosed diabetic cases falling from 59.2% in 1976 to 17.5% in 2000. The authors suggest that this may be the result of increased awareness among patients and heath care providers of the historical association between extreme obesity and high risk of undiagnosed diabetes, making screening (and hence diagnosis) more likely in this group.

One study in the U.S.—using data from the Third National Health and Nutrition Examination Survey (NHANES III)—estimated the prevalence ratios of type 2 diabetes in relation to level of BMI, and found that type 2 diabetes prevalence generally increases in direct proportion to increases in BMI, with the exception of men in obese class 2.[338] Thus, among males aged <55 years, 0.2% of individuals with normal weight, 3.27% of those who were overweight (BMI 25-29.9), 10.14% of those in obese class 1 (BMI 30.0–34.9), 7.95% of those in obese class 2 (BMI 35.0–39.0), and 18.08% of those in obese class 3 (BMI ≥40.0) had type 2 diabetes. For females <55 years, 0.4% of those with normal weight, 3.82% of those who were overweight (BMI 25-29.9), 2.49% of those in obese class 1, 10.67% of those in obese class 2, and 12.87% of those in obese class 3 had type 2 diabetes.

According to Katzmarzyk and Janssen, the risk of type 2 diabetes associated with obesity has an extremely large variance across studies—between 1.36 found in a study of U.S. women and 47.10 found in the study of U.S. female nurses.[339] U.K. researchers N. Freemantle et al. recently conducted a systematic review of longitudinal studies to assess the relationship between measures of abdominal obesity and type 2 diabetes in middle-aged adults.[340] They found odds ratios (ORs) ranging from 0.95 to 5.10, which they summarized as 2.14.

In order to determine the self-reported prevalence of diabetes and other health conditions by obesity level, Ali H. Mokdad et al. of the Centers for Disease Control and Prevention in the U.S. examined data from the 2001 Behavioral Risk Factor Surveillance System survey of 195,005 adults aged 18 years or older.[341] They found that all BMI levels were significantly associated with diabetes, but diabetes risk rose with each BMI category and those with the most severe

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[337] Ibid.

[338] Must, Spadano, and Coakley. "The Disease Burden Associated with Overweight and Obesity."

[339] Katzmarzyk, Peter T., and Ian Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update," *Canadian Journal of Applied Physiology*, 2004, vol. 29, no. 1: 90-115.

[340] Freemantle, N., J. Holmes, A. Hockey, and S. Kumar. "How Strong Is the Association between Abdominal Obesity and the Incidence of Type 2 Diabetes?" *International Journal of Clinical Practice*, 2008, vol. 62, no. 9: 1391-1396.

[341] Mokdad, Ali H., Earl S. Ford, Barbara A. Bowman, William H. Dietz, Frank Vinicor, Virginia S. Bales, and James S. Marks." Health Risk Factors, 2001 Prevalence of Obesity, Diabetes, and Obesity-Related Health Risk Factors, 2001," *Journal of the American Medical Association (JAMA)*, 2003, vol. 289, no. 1: 76-79.

obesity (BMI ≥40) had the highest risk (OR 7.37) of having diagnosed diabetes. Thus, of those in the normal weight range (BMI 18.5-24.9) 4.1% reported having diabetes, compared to 7.3% of those who were overweight (BMI 25–29.9), 14.9% of those who were obese (30.0–39.9), and 25.6% of those who were severely obese (BMI ≥40).

A 2007 U.K. study conducted by C.L. Hart et al. analysed data from two surveys that took place between 1970–1973 and 1972–1976 among adults aged 45–64 years.[342] None of the respondents had type 2 diabetes at the time of the survey. The researchers used acute hospital discharge data and death certificates to identify adults who developed type 2 diabetes between the initial surveys in the early 1970s and 2004. They found that, compared with individuals with normal weight, the odds of developing type 2 diabetes among respondents to one of the two surveys (Renfrew/Paisley study) were OR 2.73 for overweight men, OR 2.54 for overweight women, OR 7.26 for obese men, and OR 5.82 for obese women. In the other survey (Collaborative Occupational Study), which had too few women for inclusion in the Hart et al. analysis, the odds of developing type 2 diabetes were OR 3.28 for overweight men and OR 9.96 for obese men.

Alison Field of the Harvard Medical School and colleagues estimated ORs for a number of chronic diseases associated with BMI, including type 2 diabetes.[343] They examined 10-year follow-up data (1986–1996) from the Nurses' Health Study, which began in 1976 and re-surveyed the 121,701 female respondents, who were aged 30 to 55 years at the beginning of the survey, every two years.[344] In addition, for men they used data from the Health Professionals Follow-up Study, which is a prospective study of 515,529 men, aged 40 to 75 years at the beginning of the study in 1986, who have also been followed-up and re-surveyed every two years.

Confirming the findings of the studies cited above, Field et al. found that the risk of developing diabetes increased with severity of excess weight. Compared with respondents with normal weight, the odds of developing diabetes were 4.6, 10.0, and 17.0 for women who were overweight, obese (class 1– BMI 30.0–34.9), and severely obese (classes 2 and 3 – BMI ≥35), respectively. For men who were overweight, obese (class 1) and severely obese (classes 2 and 3), the odds of developing diabetes were 3.5, 11.2, and 23.4, respectively.[345]

In their cost of obesity study for Canada, as noted, Katzmarzyk and Janssen found the range of obesity-related RRs for type 2 diabetes in the epidemiological literature to vary enormously— from 1.36 to 47.10. The next highest RR (after 47.10) was 17.63. Based on the range of studies examined, Katzmarzyk and Janssen used a summary RR for type 2 diabetes of 3.73, which

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[342] Hart, C. L., D. J. Hole, D. A. Lawlor, and G. Davey Smith. "How Many Cases of Type 2 Diabetes Mellitus Are Due to Being Overweight in Middle Age? Evidence from the Midspan Prospective Cohort Studies Using Mention of Diabetes Mellitus on Hospital Discharge or Death Records," *Diabetic Medicine*, 2007, vol. 24, no. 1: 73-80.
[343] Field, Alison E., Eugenie H. Coakley, Aviva Must, Jennifer L. Spadano, Nan Laird, William H. Dietz, Eric Rimm, and Graham A. Colditz. "Impact of Overweight on the Risk of Developing Common Chronic Diseases During a 10-Year Period," *Archives of Internal Medicine*, 2001, vol. 161: 1581-1586.
[344] Ibid.
[345] Ibid.

indicates that people with obesity are 3.73 times more likely to have type 2 diabetes than people with normal weight.[346]

Despite considerable variations in the actual quantified odds ratios and relative risks linking obesity and type 2 diabetes, this sampling of studies suffices to demonstrate a reasonably clear 'dose-response' pattern—that the more overweight or obese someone is, the greater his or her chances of developing type 2 diabetes, with severely obese individuals at very increased risk. The fact that these findings are repeatedly confirmed by a wide range of different types of studies—from cross-sectional analyses, to longitudinal studies, to use of administrative data—many based on very large sample sizes, leaves no doubt about the direct, observed, and quantifiable link between obesity and type 2 diabetes.

4.3.1 Diabetes statistics for Alberta

In 2008, using data from the National Diabetes Surveillance System (NDSS), PHAC reported that among individuals aged 1 year and older, the 2004/05 prevalence of diabetes in Canada was 5.5% (5.8% of males and 5.2% of females).[347] When the prevalence rates were age-standardized, the Canadian diabetes rate was 4.7% (5.2% of males and 4.2% of females).

When only the adult population is considered, diagnosed diabetes rates are considerably higher—more than 50% higher than the age-standardized rate for all Canadians aged 1 and older, and nearly 30% higher than the unadjusted rate. Thus, the Canadian Diabetes Association and PHAC estimate that 1.8 million Canadians, aged 20 years and older, or 7.1% of the adult population, had diagnosed diabetes in 2004/05—6.6% of females and 7.6% of males—representing an increase of 70% since 1998.[348]

While part of this remarkably sharp increase in a very short period of time may well be due to higher nationwide rates of obesity and diabetes in 2004/05 than in 1998, part of the increase may also be due to higher rates of diagnosis and reporting. Thus, as has also been found in the U.S. and Australia, the Canadian Diabetes Association and PHAC report that about a third of adults who have diabetes are not aware that they have the condition.[349, 350, 351] Therefore, the diabetes prevalence rates may be considerably higher than the reported rates, with individuals who are not aware they have the condition also not being treated for the disease and therefore at increased

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[346] Katzmarzyk, Peter T., and Ian Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update," *Canadian Journal of Applied Physiology*, 2004, vol. 29, no. 1: 90-115.
[347] Public Health Agency of Canada (PHAC). *Diabetes in Canada: Highlights from the National Diabetes Surveillance System 2004 - 2005*, 2008; accessed October 2008; available from http://www.phac-aspc.gc.ca/publicat/2008/dicndss-dacsnsd-04-05/pdf/dicndss-04-05-eng.pdf.
[348] Canadian Diabetes Association. "Canadian Diabetes Association 2008 Clinical Practice Guidelines for the Prevention and Management of Diabetes in Canada."
[349] Ibid.
[350] Gregg, Cadwell, Cheng, Cowie, Williams, Geiss, Engelgau, and Vinicor. "Trends in the Prevalence and Ratio of Diagnosed to Undiagnosed Diabetes According to Obesity Levels in the U.S."
[351] Access Economics. *The Economic Costs of Obesity*, accessed.

risk of developing the potentially serious consequences that may ensue when diabetes is not effectively managed.

The risk of diabetes increases with age. Thus, in 2004/05, among Canadian children aged 1–19 years, 0.3% were diagnosed with diabetes—with no marked gender differences.[352] By comparison, 3% of Canadians aged 35-64 years had diagnosed diabetes, as did about 10% of seniors 65 and older, and 21% of seniors between the ages of 75 to 79. Aboriginal people had diabetes rates from three to five times higher than those in the general population.[353]

Diagnosed adult diabetes cases in Canada are expected to increase to 2.4 million by 2016, partly in response to the substantial nationwide increase in obesity rates.[354] The Canadian Diabetes Association also reports:

> Eleven percent of Canadians living with diabetes also have 3 or more chronic health conditions, and compared to the general population, they are 4 times more likely to be admitted to a hospital or a nursing home, 7 times more likely to need home care and 3 to 5 times more likely to see a health care provider.[355]

According to PHAC, the 2004/05 age-standardized prevalence of diabetes in Alberta among individuals aged 1 year and older was 4.2%—(4.8% of males and 3.9% of females)—somewhat lower than the Canadian age-standardized rate of 4.7%.[356]

The Alberta Diabetes Surveillance System (ADSS) notes that, although diabetes in children is increasing, the majority of people with diabetes are still adults and that the NDSS case definition has only been validated for adults, aged 20 years and over.[357] Indeed, type 2 diabetes—which is estimated to account for 95% of all diabetes cases—is commonly referred to as "adult onset diabetes." Therefore, ADSS has only reported diabetes rates for adults in this age group (20 and older). However, ADSS also reports that future versions of the Alberta Diabetes Atlas will include the full population.

The Alberta Diabetes Surveillance System (ADSS), which utilizes provincial administrative health care data but is not yet able to distinguish between the types of diabetes, reports that between 2000 and 2005, ADSS prevalence rates for adult diabetes rose steadily from 90,644 cases in 2000 to 129,184 cases in 2005—an increase of 42.5%.[358] The average yearly rate of increase was 7.34%.[359] Again, as noted above

[352] Canadian Diabetes Association. "Canadian Diabetes Association 2008 Clinical Practice Guidelines for the Prevention and Management of Diabetes in Canada."

[353] Public Health Agency of Canada (PHAC). *Diabetes in Canada: Highlights from the National Diabetes Surveillance System 2004 - 2005*, accessed.

[354] Canadian Diabetes Association. "Canadian Diabetes Association 2008 Clinical Practice Guidelines for the Prevention and Management of Diabetes in Canada."

[355] Ibid.

[356] Public Health Agency of Canada (PHAC). *Diabetes in Canada: Highlights from the National Diabetes Surveillance System 2004 - 2005*, accessed.

[357] Alberta Diabetes Surveillance System. *Alberta Diabetes Atlas 2007*, Institute of Health Economics, 2007; accessed September 2008; available from http://www.albertadiabetes.ca/pdf/atlas/AlbertaDiabetesAtlas2007_001.pdf.

[358] Ibid., accessed.

for Canada as a whole, part of this very sharp increase in a remarkably short period of time, is undoubtedly due to higher rates of obesity and diabetes, while part may be due to higher rates of diagnosis and reporting.

In 2004, according to ADSS, 120,465 Albertan adults aged ≥20 years had diagnosed diabetes, of whom 11,927 were newly diagnosed cases.[360] Edward Ng et al. of Statistics Canada report that in Canada about 5% of diabetes cases are type 1 and 95% are type 2.[361] Therefore, it can be estimated that 114,442 adults aged ≥20 years in Alberta had diagnosed type 2 diabetes in 2004.

In 2004/05 the crude prevalence rate for diagnosed diabetes in Alberta, according to ADSS, was 5.5% of the population aged ≥20 years.[362] This is 1.6 percentage points lower than the 2004/05 NDSS Canadian diabetes prevalence rate of 7.1% (6.6% of females and 7.6% of males) for adults aged ≥20 years.[363]

ADSS reports that the highest total diabetes prevalence rate was in the 55–59 year age group, and—although diabetes rates remained high in older age groups—they began to decline after age 60 mainly because of deaths associated with diabetes at older ages.[364] Between the ages of 50 and 80 years, males had higher diabetes rates than females.

Since relative risk ratios linking obesity to diabetes have been estimated for this study from CCHS data, self-reported diabetes prevalence for Alberta, as reported in Statistics Canada's CANSIM database for 2005 CCHS, is shown in Table 19 below for illustration purposes.[365] The self-reported diagnosed diabetes data that follow are the closest in time to the 2004 CCHS directly measured obesity data and self-reported diabetes data that were used to estimate relative risk ratios and population attributable fractions. CANSIM did not report these data for 2004. More recent (2007) CCHS data on self-reported diabetes are available but would be less appropriate to establish linkages to the 2004 CCHS obesity data, which remain the most recently available directly measured obesity data in Canada.

The self-reported 2005 CCHS diabetes rate for Albertan adults aged ≥20 years (4.5%) was one percentage point lower than the 2004/05 ADSS reported rate of 5.5% of the population which, as noted above, was based on provincial administrative health care data. Thus, the CCHS 2005 self-reported rates show that 104,296 or 4.5% of Albertan adults aged ≥20 years had diagnosed diabetes—4.8% of males and 4.2% of females.

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[359] The prevalence of diabetes cases in Alberta for adults aged ≥20 were 90,644 cases in 2000, increasing by 7.67% to 97,600 in 2001, increasing by 7.76% to 105,176 cases in 2002, increasing by 6.76% to 112,287 in 2003, increasing by 7.28% to 120,465 cases in 2004, and increasing by 7.24% to 129,184 cases in 2005.

[360] Alberta Diabetes Surveillance System. *Alberta Diabetes Atlas 2007*, accessed.

[361] Ng, Dasgupta, and Johnson. "An Algorithm to Differentiate Diabetic Respondents in the Canadian Community Health Survey."

[362] Alberta Diabetes Surveillance System. *Alberta Diabetes Atlas 2007*, accessed.

[363] Public Health Agency of Canada (PHAC). *Diabetes in Canada: Highlights from the National Diabetes Surveillance System 2004 - 2005*, accessed.

[364] Alberta Diabetes Surveillance System. *Alberta Diabetes Atlas 2007*, accessed.

[365] Statistics Canada. *Diabetes, by Age Group and Sex, Household Population Aged 12 and over, Canada, Provinces, Territories, Health Regions (June 2005 Boundaries) and Peer Groups, Every 2 Years*, Canadian Community Health Survey, CANSIM Table 105-0411, 2005.

For the Albertan population 12 and older—which is the full CCHS sample for the province—3.9% reported that they had been diagnosed with diabetes—4.2% of males and 3.7% of females.[366] The rate was about 1.2% for those aged 20-44, 6.7% for those aged 45-64 (7.4% of males and 6% of females), and 11.7% for those 65 and older (13.2% of males and 10.5% of females).

For the population aged 12 years and older, the self-reported rate of diagnosed diabetes was 3.9% of the population, or 105,242 people. The rate for the 56,488 males who reported the prevalence of diabetes (4.2%) was 0.5 percentage points higher than the rate for the 48,754 females (3.7%) who reported the same.

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[366] Ibid.

Table 19. Alberta diabetes prevalence rates by age group and gender, CCHS, 2005

Age Group	Population	No. and %	Both	Males	Females
12 and over	Total pop.	# persons	2,686,120	1,351,451	1,334,669
		percent	100.0	100.0	100.0
	with diabetes	# persons	105,242	56,488	48,754
		percent	3.9	4.2	3.7
	without diabetes	# persons	2,579,419	1,294,550	1,284,869
		percent	96.0	95.8	96.3
15-19	Total pop.	# persons	209694	105,205	104,489
		percent	100.0	100.0	100.0
	with diabetes	# persons	F	F	F
		percent	F	F	F
	without diabetes	# persons	209341.0	104,963	104,378
		percent	99.8	99.8	99.9
20-34	Total pop.	# persons	710,849	363,093	347,756
		percent	100.0	100.0	100.0
	with diabetes	# persons	7,782^E	F	F
		percent	1.1^E	F	F
	without diabetes	# persons	702,757	358,944	343,813
		percent	98.9	98.9	98.9
35-44	Total pop.	# persons	513,940	263,331	250,609
		percent	100.0	100.0	100.0
	with diabetes	# persons	6,903^E	F	F
		percent	1.3^E	F	F
	without diabetes	# persons	507,037	260,167	246,870
		percent	98.7	98.8	98.5
45-64	Total pop.	# persons	785,752	396,708	389,045
		percent	100.0	100.0	100.0
	with diabetes	# persons	52,681	29,463^E	23,218^E
		percent	6.7	7.4^E	6.0^E
	without diabetes	# persons	732,053	366,831	365,222
		percent	93.2	92.5	93.9
≥65	Total pop.	# persons	314,812	144,013	170,800
		percent	100.0	100.0	100.0
	with diabetes	# persons	36,930	19,012^E	17,918
		percent	11.7	13.2	10.5
	without diabetes	# persons	277,794	125,001	152,793
		percent	88.2	86.8	89.5

Note: E – marginal sample variability (16.6–≤33.3); F – too unreliable to be published, due to coefficient of variation >33.3%. Thus, data with a coefficient of variation greater than 33.3% were suppressed by Statistics Canada due to extreme sampling variability.

Source: Statistics Canada. Diabetes, by Age Group and Sex, Household Population Aged 12 and over, Canada, Provinces, Territories, Health Regions (June 2005 Boundaries) and Peer Groups, Every 2 Years, Canadian Community Health Survey, CANSIM Table 105-0411, 2005.

The prevalence of diabetes (2004 CCHS cycle 2.2) was higher among obese males and lower among obese females compared to the general population overall in Canada in 2004. According to the 2004 CCHS cycle 2.2, the overall prevalence of diabetes among aged ≥35 was 12.3%, which compares with 14.7% among obese males (BMI ≥30) and 10.3% among obese females (BMI ≥30) aged ≥35 in Canada.

Comparing the obese population (i.e. the exposed population) (BMI ≥30) aged ≥15 to the normal weight population (i.e. the unexposed) (BMI 18.5–24.9) aged ≥15 using data from 2004 CCHS cycle 2.2, the age-adjusted prevalence-based risk ratio for diabetes among the obese population was 4.4 for males and 3.2 for females. This indicates that the risk of diabetes among the aged ≥15 population was approximately 4.4 times higher among obese males and approximately 3.2 times higher among obese females than the risk of diabetes among males and females with normal weight.

The portion (population attributable fraction–PAF) of diabetes that was found to be attributable to obesity (BMI ≥30) among aged ≥15 was 32.0% for females and 41.9% for males.

Diabetes data by gender for several age groups were suppressed by Statistics Canada, so it is not possible to determine the actual number of cases of diabetes that were attributable to obesity in the Alberta population aged ≥15 in 2004. However, the number of diabetes cases attributable to obesity in Albertans aged ≥45 was 19,148 diabetes cases among males and 13,161 diabetes cases among females in 2004, based on population attributable fractions in aged 45–64 of 52.6% for males and 32.3% for females, and in aged ≥65, of 19.2% for males and 31.6% for females.

As noted in Chapter 4.2.3.6, gender breakdowns for diabetes as reported in CCHS cycle 2.2 showed marginal sample variability (16.6–≤33.3). Age breakdowns showed a sample variability of more than 33.3, which is considered to be in the unacceptable range by Statistics Canada. Thus, the age and gender breakdowns for diabetes are presented in this report for illustrative purposes, rather than for the purpose of providing statistically significant results.

4.4 Cardiovascular disease (CVD)

Cardiovascular disease (CVD) is a generic name given to a number of diseases that impair the functioning of the heart and other organs of the cardiovascular system. The major CVD diseases include:

- hypertensive heart disease—which includes hypertension or high blood pressure),
- coronary heart disease—which includes coronary artery or ischaemic heart disease and also heart attack or myocardial infarction, and
- cerebrovascular disease (or stroke).[367]

Obesity has been independently and strongly linked to increased cardiovascular risk in both men and women, and is specifically associated with hypertension, coronary heart disease, cerebrovascular disease, and increased mortality risk from CVD.[368]

One of the earliest studies on this association, which re-examined data from the Framingham Heart Study, showed that in 26 years of follow-up of approximately 5,200 men and women, aged 28–62 years, obesity was an independent risk factor for CVD.[369] High relative weights were associated with coronary heart disease, stroke, hypertension, and CVD-related mortality, independent of age, cholesterol, smoking, and glucose intolerance.[370] H.B. Hubert et al. estimated that if everyone in the Framingham study had normal weight, 25% of coronary heart disease and 35% of strokes would be eliminated, and that a 20% weight loss among the obese could result in a 40% lower risk of a coronary event.[371]

According to James et al., the mechanisms by which obesity leads to CVD and excess mortality are not always clear, although high blood pressure and increased cholesterol and triglyceride concentrations, which are associated with obesity, are known risk factors for CVD.[372] Triglycerides, which make up most of the vegetable oil and animal fats digested by humans, are formed from a single molecule of glycerol combined with three fatty acids, and play an important role in metabolism as energy sources and transporters of dietary fat.[373] In addition, excess adipose tissue puts an extra strain on the heart because greater output and workload from the heart are needed to increase the flow of the additional blood required in people with excess weight. Diet has also been linked with an increased risk of CVD: dietary sodium, trans-fats, and saturated fats are associated with an increased risk, while polyunsaturated fats are associated with decreased risk.[374]

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[367] Alberta Health and Wellness. *Report on the Health of Albertans*, Edmonton: Alberta Health and Wellness, 2006; accessed July 2008; available from http://www.health.alberta.ca/resources/publications/Health-of-Albertans-2006.pdf.

[368] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[369] Hubert, Feinleib, McNamara, and Castelli. "Obesity as an Independent Risk Factor for Cardiovascular Disease: A 26-Year Follow-up of Participants in the Framingham Heart Study."

[370] Ibid.

[371] Ibid.

[372] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

[373] Ibid.

[374] Reddy, K. Srinath, and Martijn B. Katan. "Diet, Nutrition and the Prevention of Hypertension and Cardiovascular Diseases," *Public Health Nutrition*, 2004, vol. 7, no. 1A: 167–186.

Using a nationally representative sample from the 1995 U.S. National Health Interview Survey, Guijing Wang et al. of the Centers for Chronic Disease Control and Prevention in the U.S. found that among adults, aged ≥25 years, 38.55% of those who were obese (36.05% of men and 40.64% of women) had CVD, compared with 19.63% of adults with normal weight (18.80% of men and 20.20% of women).[375]

Wang et al. also found that the prevalence of CVD in obese adults increased with age. Obese men aged 25–44, 45–64, and ≥65 had CVD prevalence rates of 17.33%, 43.89%, and 67.21%, respectively, and obese women in the same age groups had rates of 14.42%, 45.28%, and 69.11%, respectively. For those under 65, these rates were well over double the rates of adults with normal weight. Thus, normal weight men aged 25–44, 45–64, and ≥65 had CVD prevalence rates of 5.0%, 18.46%, and 50.93% respectively, while normal weight women had rates of 3.94%, 19.63%, and 51.79%, respectively.

U.S. and Danish researchers Majken Jensen et al. recently followed up 54,783 adults for a median of 7.7 years from the Danish Diet, Cancer and Health Study. The subjects were aged 50–64 years at the beginning of the study (1993 to 1997).[376] Jensen et al. found that obese adults had a 5% to 7% higher risk of acute coronary events, per one unit increase of BMI, than those with normal weight, who had the lowest risk. This was the case whether the individuals were healthy or not at the beginning of the study — though those with coronary artery disease and cancer at the start of the study were excluded from the study —, physically active or inactive, having a heart-healthy diet or not, or were smokers or nonsmokers.

4.4.1 Hypertension

Hypertension, or high blood pressure, is a risk factor for CVD, but it is also considered to be a disease in its own right, and in this report it is treated as a separate disease. Criteria for hypertension refer to an elevated systolic or diastolic pressure, or both.[377] Systolic pressure occurs when the heart is pumping blood into the arteries, and diastolic pressure occurs between heartbeats when the heart is at rest. Blood pressure is considered to be high if the systolic pressure is consistently 140 mm Hg or greater and/or diastolic pressure is consistently 90 mm Hg or greater.[378]

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[375] Wang, Guijing, Zhi-Jie Zheng, Gregory Heath, Carol Macera, Mike Pratt, and David Buchner. "Economic Burden of Cardiovascular Disease Associated with Excess Body Weight in U.S. Adults," *American Journal of Preventive Medicine*, 2002, vol. 23, no. 1: 1-6.

[376] Jensen, Majken K., Stephanie E. Chiuve, Eric B. Rimm, Claus Dethlefsen, Anne Tjonneland, Albert M. Joensen, and Kim Overvad. "Obesity, Behavioral Lifestyle Factors, and Risk of Acute Coronary Events," *Circulation. Journal of the American Heart Association*, 2008, vol. 117, no. 24: 3062-3069.

[377] Canadian Hypertension Education Program (CHEP). *CHEP Recommendations for the Management of Hypertension*, 2007; accessed September 2008; available from http://www.hypertension.ca/chep/wp-content/uploads/2007/10/chep-2007-spiral-mar16.pdf.

[378] Ibid., accessed.

One of the difficulties in estimating hypertension prevalence in the population, especially when self-reported data are used, is that hypertension is often undiagnosed because there may be no obvious symptoms in the early stages of the disease.[379] Therefore, prevalence estimates based on self-reported surveys such as CCHS may underestimate the true prevalence of hypertension. Karen Tu et al. of the University of Toronto recently found that administrative data for physician-diagnosed hypertension show prevalence rates that are 3–4% higher than those found in national self-report surveys.[380]

James et al. note that obesity is consistently associated with hypertension, and give the following explanation of the physical mechanisms through which obesity can produce the disease:

> The mechanisms by which weight gain promotes a rise in blood pressure may involve the accentuation of insulin resistance, increases in the tone of the sympathetic nervous system control of the arterioles and the production by the adipose tissue itself of a variety of vasoactive cytokines and hormones, such as angiotensinogen, which increase blood pressure. These vasoactive compounds act in part by reducing sodium excretion by the kidney, thereby increasing the blood volume and therefore blood pressure.[381]

R. Wolk and V.K. Somers of the Mayo Clinic note that "adiposity [is] the best single predictor of hypertension, and changes in body fat over 8 years were related to changes in both systolic and diastolic blood pressure."[382] However, they also note that obesity is more prevalent than hypertension, and "the higher prevalence of obesity, compared with hypertension, suggests that a significant proportion of obese individuals do not develop hypertension at an earlier age. Why some obese individuals are more resistant to the development of hypertension is not known."[383]

Evidence has found that obese individuals are from 2.2 to 5.7 times more likely than non-obese individuals to develop hypertension or increased blood pressure, and one report suggests that over 75% of hypertension can be directly attributed to obesity.[384, 385] Haslam and James note that the risk of hypertension among obese individuals is up to five times greater than among those with normal weights, and that two-thirds of hypertension cases are associated with excess

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[379] Haslam, and James. "Obesity."

[380] Tu, Karen, Zhongliang Chen, and Lorraine L. Lipscombe. "Prevalence and Incidence of Hypertension from 1995 to 2005: A Population-Based Study," *Canadian Medical Association Journal*, Canadian Hypertension Education Program Outcomes Research Taskforce, 2008, vol. 178, no. 11: 1429-1435.

[381] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)." p. 540.

[382] Wolk, R., and V. K. Somers. "Obesity-Related Cardiovascular Disease: Implications of Obstructive Sleep Apnea," *Diabetes, Obesity and Metabolism*, 2006, vol. 8: 250-260. p. 251.

[383] Ibid.

[384] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[385] Krauss, Ronald M., Mary Winston, Barbara J. Fletcher, and Scott M. Grundy. "Obesity: Impact on Cardiovascular Disease," *Circulation. Journal of the American Heart Association*, 1998, vol. 98: 1472-1476.

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weight.[386] U.S. researchers Deborah King and Marion Wofford report that approximately one-third of hypertensive disease is related to obesity.[387]

The WHO *Comparative Quantification of Health Risks* study found that in the America-A subregion (consisting of Canada, United States, and Cuba), 63% of hypertension in males and 58% in females aged ≥30 years, could be attributed to a BMI of ≥30.[388] U.K. researcher Peter Kopelman reports that the risk of hypertension is five times higher in the obese than in those with normal weight, that 66% of hypertension can be linked to excess weight, and that 85% of hypertension is associated with a BMI >25, which includes both overweight and obesity.[389]

U.S. researchers Alviva Must et al. used data from the Third National Health and Nutrition Examination Survey (NHANES III) to estimate the disease burden associated with obesity.[390] They found that 47.95% of females in obesity class 1, 54.51% in obesity class 2, and 63.16% in class 3 had high blood pressure, compared with 23.26% of those with normal weight.[391] For males, 48.95% in obesity class 1, 65.48% in obesity class 2, and 64.53% in class 3 had high blood pressure, compared with 23.47% with normal weight.[392]

A recent Swedish study used data from military physical examinations that took place between 1969 and 1994 that measured the blood pressure and BMI of 1,145,758 young men, aged 16–25 years at the time of the first examination, in order to assess the differential hazards (according to BMI category) of high blood pressure later leading to CVD, stroke, and heart attack[393] The men were followed through 2006 for CVD events, including myocardial infarctions and strokes. The authors, Karri Silventoinen et al., analysed the modifying effect of BMI on the association between blood pressure and CVD, including stroke and heart attack, by estimating hazard ratios (HRs) per increases in systolic and diastolic blood pressures for standard BMI categories (i.e., underweight, normal weight, overweight, and obese).

Silventoinen et al. found that the strongest associations of high systolic blood pressure with CVD (HR 1.16) and stroke (HR 1.29) were seen among men in the obese category, while the strongest association with myocardial infarctions (HR 1.19) was seen among men in the overweight category—where 1.0 is the risk of these events among normal weight men with the same level of high blood pressure.[394] The strongest associations with high diastolic pressure with CVD (HR 1.18), stroke (HR 1.13), and myocardial infarctions (HR 1.22) were seen among men

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[386] Haslam, and James. "Obesity."

[387] King, Deborah S., and Marion R. Wofford. " Obesity and Hypertension," *Drug Topics*, 2000, vol. 144, no. 1: 59-66.

[388] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

[389] Kopelman. "Health Risks Associated with Overweight and Obesity."

[390] Must, Spadano, and Coakley. "The Disease Burden Associated with Overweight and Obesity."

[391] Ibid.

[392] Ibid.

[393] Silventoinen, Karri, Patrik K.E. Magnusson, Martin Neovius, Johan Sundstrom, G. David Batty, Per Tynelius, and Finn Rasmussen. "Does Obesity Modify the Effect of Blood Pressure on the Risk of Cardiovascular Disease?: A Population-Based Cohort Study of More Than One Million Swedish Men," *Circulation. Journal of the American Heart Association*, 2008, vol. 118, no. 16: 1637-1642.

[394] Ibid.

in the obese category.[395] In other words, the authors found that obesity and overweight substantially increased the risk of CVD, stroke, and heart attack.

Field et al. estimated the risk of hypertension by BMI for middle-aged U.S. adults in a 10-year follow-up study of the Nurses Health Study and in the Health Professionals Follow-up Study in the U.S.—which was described in Chapter 4.3 above on type 2 diabetes.[396] Approximately 19% of men and 16% of women in the total sample had high blood pressure. Compared with respondents with normal weight, the odds of developing hypertension were 1.7, 2.1, and 2.3 for women who were overweight, obese class 1 (BMI 30.0–34.9), and severely obese (BMI ≥35), respectively. For men who were overweight, obese class 1 (BMI 30.0–34.9), and severely obese (BMI ≥35), the odds of developing hypertension were 1.7, 2.7, and 3.0, respectively.[397]

In their cost of obesity study in Canada, Katzmarzyk and Janssen found a range of RRs linking obesity and hypertension in the epidemiological literature—ranging from 2.23 to 5.70 in the epidemiology literature.[398] They used a summary RR of 4.5 for the association between obesity and hypertension.[399]

4.4.1.1 Hypertension statistics for Alberta

Using administrative data, Tu et al., recently examined trends in hypertension prevalence in Ontario between 1995 and 2005, and found that the number of adults with the condition significantly increased during this time period.[400] Part of this increase was attributable to the aging of the population. Adjusted for age and sex, therefore, the authors found that hypertension prevalence actually increased by 60% in this time period—from 153.1 per 1,000 adults in 1995 to 244.8 per 1,000 adults in 2005.[401] Between 2000 and 2005, the prevalence increased by 20.9%, and the average annual increase in hypertension prevalence was 4.4%.

According to Statistics Canada's Canadian Community Health Surveys, the prevalence of high blood pressure in the Alberta population, aged 12 and over, increased by 7.9% between 2003 and 2005—from 317,939 cases to 343,015 cases, and by another 17.3% between 2005 and 2007–from 343,015 cases to 402,424 cases.[402] Alberta Health and Wellness reports that the treated

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[395] Ibid.

[396] Field, Coakley, Must, Spadano, Laird, Dietz, Rimm, and Colditz. "Impact of Overweight on the Risk of Developing Common Chronic Diseases During a 10-Year Period."

[397] Ibid.

[398] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[399] Katzmarzyk and Janssen note, "Summary relative risk (RR) estimates were calculated using a general variance-based method of meta-analysis," but they did not explain this methodology. p. 100.

[400] Tu, Chen, and Lipscombe. "Prevalence and Incidence of Hypertension from 1995 to 2005: A Population-Based Study."

[401] Ibid.

[402] Statistics Canada. *Health Indicators*, 2006; accessed July 2008; available from http://www.statcan.gc.ca/pub/82-221-x/2006001/4198117-eng.pdf.

prevalence of hypertension increases with age, and is most commonly present in adults aged 50 and older.[403]

Table 20 below shows the self-reported prevalence of high blood pressure in the Alberta population, aged 12 years and older, by age group and sex, based on self-reported data in the 2005 CCHS.[404] In 2005, 343,015 Albertans, or 12.8% of the Alberta population aged 12 and over, reported diagnosed high blood pressure, which was significantly lower than the Canadian average of 14.9%.[405] The difference was particularly marked for females. Thus, when stratified by gender, 12.9% of Alberta males and 12.7% of females reported high blood pressure, compared to the Canadian average of 14.1% of males and 15.7% of females.

Among older age groups, however, the proportion of Albertans with high blood pressure is much higher than the averages for the aged ≥12 population indicate. Thus, 18.6% of Albertans aged 45-64 (20.9% of males and 16.2% of females) reported high blood pressure in the 2005 CCHS, while 46.5% of seniors 65 and older (43.6% of males and 48.9% of females) had high blood pressure. As noted above, hypertension may not manifest noticeable symptoms in its early stages, so part of the sharp age-related increase indicated in Table 20 below may be due to greater awareness and higher levels of diagnosis and management of the condition at older ages.

Table 20. High blood pressure (HBP) prevalence, by age group and sex, Alberta, CCHS, 2005

Age Group	Population	No. & %	Both	Males	Females
12 and over	Total pop.	# persons	2,686,120	1,351,451	1,334,669
		percent	100.0	100.0	100.0
	with HBP	# persons	343,015	174,137	168,879
		percent	12.8	12.9	12.7
	without HBP	# persons	2,338,998	1,175,016	1,163,983
		percent	87.1	86.9	87.2
15-19	Total pop.	# persons	209,694	105,205	104,489
		percent	100.0	100.0	100.0
	with HBP	# persons	F	F	F
		percent	F	F	F
	without HBP	# persons	207,350	104,449	102,901
		percent	98.9	99.3	98.5
20-34	Total pop.	# persons	710,849	363,093	347,756
		percent	100.0	100.0	100.0
	with HBP	# persons	17,019[E]	10,177[E]	6,842[E]

[403] Alberta Health and Wellness. *Report on the Health of Albertans*, accessed.

[404] Statistics Canada. *High Blood Pressure, by Age Group and Sex, Household Population Aged 12 and over, Canada, Provinces, Territories, Health Regions (June 2005 Boundaries) and Peer Groups, Every 2 Years*, Canadian Community Health Survey, CANSIM Table 105-0410, 2005.

[405] Statistics Canada. *Health Indicators*, accessed.

		percent	2.4^E	2.8^E	2.0^E
	without HBP	# persons	693,120	352,206	340,914
		percent	97.5	97.0	98.0
35-44	Total pop.	# persons	513,940	263,331	250,609
		percent	100.0	100.0	100.0
	with HBP	# persons	32,021	17,786	14,235^E
		percent	6.2	6.8	5.7^E
	without HBP	# persons	480,768	244,626	236,142
		percent	93.5	92.9	94.2
45-64	Total pop.	# persons	785,752	396,708	389,045
		percent	100.0	100.0	100.0
	with HBP	# persons	145,958	82,771	63,187
		percent	18.6	20.9	16.2
	without HBP	# persons	639,469	313,655	325,814
		percent	81.4	79.1	83.7
65+	Total pop.	# persons	314,812	144,013	170,800
		percent	100.0	100.0	100.0
	with HBP	# persons	146,260	62,771	83,489
		percent	46.5	43.6	48.9
	without HBP	# persons	168,172	81242	86,931
		percent	53.4	56.4	50.9

Notes: E - use with caution (— coefficient of variation between 16.6% and 33.3%); F - too unreliable to be published: Data with a coefficient of variation greater than 33.3% were suppressed by Statistics Canada due to extreme sampling variability.

Source: Statistics Canada. *High Blood Pressure, by Age Group and Sex, Household Population Aged 12 and over, Canada, Provinces, Territories, Health Regions (June 2005 Boundaries) and Peer Groups, Every 2 Years*, Canadian Community Health Survey, CANSIM Table 105-0410, 2005.

According to the 2004 CCHS cycle 2.2, the prevalence of hypertension among obese males 15 years of age and over in Canada was 23.1% and the prevalence among obese females was 25.6%.

Comparing the obese population (i.e. the exposed population) (BMI ≥30) to the normal weight population (i.e. the unexposed) (BMI 18.5–24.9) for Canadians aged ≥15 using data from 2004 CCHS cycle 2.2, the age-adjusted prevalence-based risk ratio for high blood pressure was 2.4 for males and 2.2 for females aged 15 years and over.

The portion (population attributable fraction–PAF) of hypertension that can be attributed to obesity was 22.8% for both males and females.

Applying the PAF to the number of cases of high blood pressure in Alberta in 2005 shows, for adults 20 years of age and over, that 39,559 cases in males and 38,247cases in females could be

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attributed to obesity. (High blood pressure prevalence data were not given by Statistics Canada for aged 15–19.)

As discussed in Chapter 4.2.3.6, gender breakdowns for hypertension showed marginal sample variability (16.6–≤33.3). Age breakdowns showed a sample variability of more than 33.3, which is considered to be in the unacceptable range by Statistics Canada. Thus, the age and gender breakdowns for hypertension are presented here for illustrative purposes, rather than for the purpose of providing statistically significant results.

4.4.2 Coronary heart disease (CHD)

Coronary heart disease (CHD) includes both coronary artery disease or ischemic heart disease, and also heart attack or myocardial infarction. The former condition—coronary artery disease— is often a precursor to the latter—namely heart attack—which is the most common cause of sudden death in Canada and other high-income countries.[406]

CHD prevalence is generally low until age 40 when it begins to rise significantly, especially among males. Since the mid 1970s, both hospital separation and mortality rates attributable to CHD have been declining, which has been linked both to the improved treatment of the disease (including effective medical interventions like coronary bypass surgery) and to reduced cigarette smoking.[407, 408] However, Alberta Health and Wellness expects the number of Albertans with CHD, aged ≥40, to rise dramatically over the next 30 years, mainly due to the aging of the population.[409]

Alberta Health and Wellness explains the biological mechanisms of CHD:

> Ischaemic heart disease refers to a condition where there is a lack of blood and oxygen being delivered to the heart muscles. This condition is caused by cholesterol deposits which block arteries, and is one of the main causes of death in Canada. Clogged arteries cannot deliver enough blood to the muscles of the heart, resulting in the death of heart-muscle cells, and the loss of elasticity of the heart muscle. Typically, this causes angina pectoris, or chest pain. Should a blood clot form and the artery become completely blocked, a heart attack, and possibly sudden death, will result. Although the number of deaths due to coronary heart disease has dropped in the past decade among men and women, it remains a leading cause of death among Albertans.[410]

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[406] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."
[407] Alberta Health and Wellness. *Report on the Health of Albertans*, accessed.
[408] Mann, J.I. "Diet and Risk of Coronary Heart Disease and Type 2 Diabetes," *The Lancet*, 2002, vol. 360, no. Sept 7: 783-789.
[409] Malo, Shaun. *Chronic Disease Projections 2006 to 2035. Ischemic Heart Disease*, Alberta Health and Wellness, 2008; accessed July 2008; available from http://www.health.alberta.ca/resources/publications/CDP_Ischemic-HD-2006-35.pdf.
[410] Alberta Health and Wellness. *Report on the Health of Albertans*, accessed. p. 70.

Evidence shows that, in North America, about 14% of heart failure in women and 11% in men is attributable to obesity.[411] Obesity tends to enlarge the muscles of the heart because of the need to increase the blood supply to the larger body mass, and may therefore result in coronary heart disease.[412] Haslam and James report that the "effect of obesity on heart function is probably due to a combination of factors including hypertension, dyslipidaemia, diabetes mellitus, increased fat mass and left-ventricular mass, endothelial dysfunction, and atherosclerosis."[413]

Gary Whitlock et al. systematically reviewed 14 large North American and European cohort studies, including one from Manitoba,[414] and found that all but one showed a positive relationship between BMI and the risk of both fatal and non-fatal ischaemic heart disease.[415] Systematic reviews completed in Australia and the United Kingdom have found similar associations.[416, 417] The WHO *Comparative Quantification of Health Risks* study found that in the America-A subregion (consisting of Canada, United States, and Cuba), 37% of coronary heart disease in males, and 32% in females, aged ≥30 years, could be attributed to a BMI of ≥30.[418]

James et al. report that the Asia-Pacific Cohort Collaboration Study, which followed more than 300,000 adults for 7 years, found a 9% difference in ischaemic heart disease events for each unit (1 kg/m^2) increase in BMI above 21 kg/m^2.[419] U.K. researcher Peter Kopelman notes that for each unit (1 kg/m^2) increase in BMI above 21 kg/m^2, the risk of developing coronary artery disease is magnified 3.6 times, and that obesity contributes to cardiac failure in more than 10% of CHD patients.[420]

U.S. researchers Alviva Must et al., who used data from the Third National Health and Nutrition Examination Survey (NHANES III) to estimate the disease burden associated with obesity, found that 12.56% of females in obesity class 1, 12.31% in obesity class 2, and 19.22% in class 3 had coronary heart disease, compared with 6.87% with normal weight.[421] For males, 16.01% in obesity class 1, 10.21% in obesity class 2, and 13.97% in class 3 had coronary heart disease, compared with 8.84% with normal weight.[422]

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[411] Kenchaiah, S., J.M. Gaziano, and R.S. Vasan. "Impact of Obesity on the Risk of Heart Failure and Survival after the Onset of Heart Failure," *Medical Clinics of North America*, 2004, vol. 88: 1273–1294.

[412] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[413] Haslam, and James. "Obesity." p. 1200.

[414] Tate, R.B., J. Manfreda, and T.E. Cuddy. "The Effect of Age on Risk Factors for Ischemic Heart Disease: The Manitoba Follow-up Study, 1948–1993," *Annals of Epidemiology*, 1998, vol. 8: 415–421.

[415] Whitlock, Gary, Sarah Lewington, and Cliona Ni Mhurchu. "Coronary Heart Disease and Body Mass Index: A Systematic Review of the Evidence from Larger Prospective Cohort Studies," *Seminars in Vascular Medicine*, 2002, vol. 2: 369–381.

[416] Mathers, Vos, and Stevenson. *The Burden of Disease and Injury in Australia*, accessed.

[417] National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, accessed.

[418] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

[419] Ibid.

[420] Kopelman. "Health Risks Associated with Overweight and Obesity."

[421] Must, Spadano, and Coakley. "The Disease Burden Associated with Overweight and Obesity."

[422] Ibid.

Fields et al. estimated the risk of heart disease by BMI for middle-aged U.S. adults in a 10-year follow-up study of the Nurses Health Study and in the Health Professionals Follow-up Study in the U.S.—which was described in Chapter 4.3 above on type 2 diabetes.[423] Compared with respondents with normal weight, the odds of developing heart disease were 1.4, 1.5, and 1.5 for women who were overweight, obese class 1 (BMI 30.0–34.9), and severely obese (classes 2 and 3 – BMI ≥35), respectively. For men who were overweight, obese, and severely obese, the odds of developing heart disease were 1.5, 2.0, and 2.2, respectively.[424]

In the INTERHEART study, Salim Yusuf of McMaster University et al. conducted a case-control study with 27,098 participants in 52 countries for the purpose of assessing markers of obesity in connection with myocardial infarction.[425] The study found that the risk of myocardial infarction increased with increasing BMI values, and that those in the obese category had an OR of 1.44 for risk of the disease. When only European countries were considered, the OR was 1.32.

In their cost of obesity study in Canada, Katzmarzyk and Janssen found RRs for the association between obesity and coronary heart disease in the epidemiological literature that ranged from 1.31 to 3.56.[426] They used a summary RR of 2.24 in their study.[427]

4.4.2.1 Coronary heart disease statistics for Alberta

Alberta Health and Wellness reported that the diagnosed and treated prevalence of CHD in Alberta in 2003 for all ages was 2.1% of the population—2.5% of males and 1.5% of females.[428] As noted, the rates remain low until about age 40 and then begin increasing with age. Malo notes that about 9.7% of Albertan adults 40 and older, or almost 138,000 Albertans in this age group, had CHD in 2005.[429] The rates were not estimated for people below the age of 40, since the numbers of people with the disease younger than 40 are low.

Because of the aging of the population, Malo estimates that the number of cases of CHD in Alberta will rise to about 363,000 by 2035.[430] The prevalence of CHD in Albertan males aged 40 and above is projected to increase from 11.6%, or 80,472 cases, in 2005 to almost 17.5%, or 208,315 cases, by 2035. Female CHD prevalence is expected to increase from 7.9%, or 57,359

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[423] Field, Coakley, Must, Spadano, Laird, Dietz, Rimm, and Colditz. "Impact of Overweight on the Risk of Developing Common Chronic Diseases During a 10-Year Period."

[424] Ibid.

[425] Yusuf, Salim, Steven Hawken, Stephanie Ounpuu, Leonelo Bautista, Maria Grazia Franzosi, Patrick Commerford, Chim C. Lang, Zvonko Rumboldt, Churchill L. Onen, Liu Lisheng, Supachai Tanomsup, Paul Wangai Jr., Fahad Razak, Arya M. Sharma, and Sonia S. Anand. "Obesity and the Risk of Myocardial Infarction in 27,000 Participants from 52 Countries: A Case-Control Study," *Lancet*, 2005, vol. 366: 1640-1649.

[426] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[427] Katzmarzyk and Janssen note, "Summary relative risk (RR) estimates were calculated using a general variance-based method of meta-analysis," but they did not explain this methodology. p. 100.

[428] Alberta Health and Wellness. *Report on the Health of Albertans*, accessed.

[429] Malo. *Chronic Disease Projections 2006 to 2035. Ischemic Heart Disease*, accessed.

[430] Ibid., accessed.

cases, in 2005 to 12.35%, or 153,804 cases, by 2035.

CHD prevalence rates by gender and age group for Albertans aged ≥40 years, based on 2005 administrative data (i.e., Alberta Health Care Insurance Claims data), are shown in Table 21 below. It is seen that CHD prevalence rises sharply with each age group—with Albertans over 70 more than twice as likely to have CHD as those as those under 60. By the time they are 75, more than a third of Albertan males will have CHD.

Table 21. Coronary heart disease prevalence rates in Alberta, by gender and age group, population aged ≥40 years, percent, 2005

AGE	Males	Females	Both
40-44	1.7	0.8	01.3
45-49	3.4	1.5	2.5
50-54	6.2	2.9	4.6
55-59	10.9	5.5	8.2
60-64	16.1	8.7	12.4
65-69	22.2	12.9	17.5
70-74	28.7	18.2	23.2
75-79	34.4	23.1	28.2
80-84	39.7	28.7	33.1
85-89	41.8	32.8	35.8
90+	41.8	34.4	36.4
TOTAL ≥40 years	11.6	7.9	9.7

Source: Malo, Shaun. *Chronic Disease Projections 2006 to 2035. Ischemic Heart Disease*, Alberta Health and Wellness, 2008; accessed July 2008; available from http://www.health.alberta.ca/resources/publications/CDP_Ischemic-HD-2006-35.pdf.

The Heart and Stroke Foundation of Canada (HSFC) notes that mortality data for cardiovascular disease are "well accounted for," but "the data on the burden of cardiovascular diseases in Canada are limited… There are few data on morbidity and nonfatal events."[431] It notes that "some sense of the prevalence of cardiovascular diseases" can be obtained from the CCHS.[432] According to the Heart and Stroke Foundation of Canada website:

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[431] Heart and Stroke Foundation of Canada. *Tipping the Scales of Progress: Heart Disease and Stroke in Canada 2006*, accessed. p. 13.
[432] Heart and Stroke Foundation of Canada. *The Growing Burden of Heart Disease and Stroke in Canada*, Health Canada, Canadian Cardiovascular Society, and Heart and Stroke Foundation of Canada, 2003; accessed September 2008; available from http://www.cvdinfobase.ca/cvdbook/CVD_En03.pdf.

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- 54% of all cardiovascular deaths in Canada are due to coronary artery disease and heart attack;
- 21% to stroke;
- 16% to other forms of heart disease such as problems with the electrical system of the heart, viral heart infarctions, and heart muscle disease; and
- 9% to vascular problems such as high blood pressure and hardening of the arteries.[433]

The latest available Alberta-specific data on CVD mortality by specific cause from the PHAC Cardiovascular Disease Surveillance On-Line database are for 1999.[434] According to this source, the proportions of cardiovascular deaths in Alberta in 1999 attributable to specific categories of CVD were as follows:

- 28% were due to coronary artery disease;
- 21% to heart attack;
- 19% to stroke; and
- 32% to other CVD (e.g., aortic aneurysm –3%, heart failure–5%, and other–24%).[435]

The PHAC Chronic Disease Infobase also reports the proportions of cardiovascular deaths by specific cause in Alberta, and does so by gender, but the latest data are for 2000, and the CVD breakdowns do not always match those in the PHAC Cardiovascular Disease Surveillance On-Line database cited above.[436] For Albertan males, the proportions of cardiovascular deaths by category in 2000, as provided by this source were as follows:

- 63.6% were due to coronary artery disease, including heart attack;
- 16.1% to stroke;
- 1.8% to hypertensive heart disease; and
- 18.5% to other CVD.[437]

For females, the CVD mortality proportions were as follows:

- 50.7% were due to coronary artery disease, including heart attack;
- 23.7% to stroke;
- 2.8% to hypertensive heart disease; and
- 22.9% to other CVD.[438]

The PHAC Chronic Disease Infobase also reports the proportions of cardiovascular disease

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[433] Heart and Stroke Foundation of Canada. *Incidence of Cardiovascular Disease*, 2002; accessed November 2008; available from http://ww2.heartandstroke.ca/Page.asp?PageID=33&ArticleID=1077.
[434] Public Health Agency of Canada (PHAC). *Cardiovascular Disease Surveillance on-Line*, 2008; accessed November 2008; available from http://dsol-smed.phac-aspc.gc.ca/dsol-smed/cvd/c_quik_e.html.
[435] Ibid., accessed.
[436] Public Health Agency of Canada (PHAC). *Chronic Disease Infobase*, 2008; accessed November 2008; available from http://cvdinfobase.ca/ncdasp/Profile_e.aspx.
[437] Ibid., accessed.
[438] Ibid., accessed.

hospital separations by province for 1999, and by age-standardized discharge rate per 100,000 population by province for 2005.[439] However, according to the Heart and Stroke Foundation of Canada, cardiovascular events, including nonfatal heart attacks and strokes, are counted without identifying whether they are initial or recurrent events for individuals.[440] Therefore, it is not possible to use these hospital data to estimate disease prevalence, and it is therefore also not possible to estimate the proportion of cardiovascular events attributable to obesity using these data.

According to 2004 CCHS, cycle 2.2, which was the database used to estimate relative risk ratios and population attributable fractions for coronary heart disease, the prevalence of heart disease among obese males aged ≥15 years was 7.5% and the prevalence among obese females aged ≥15 years was 6.2%.

Comparing the obese population (i.e. the exposed population) (BMI ≥30) to the normal weight population (i.e. the unexposed) (BMI 18.5–24.9) for Canadians aged ≥15 using data from 2004 CCHS cycle 2.2, the age-adjusted prevalence-based risk ratio for heart disease was 2.2 for males and 1.4 for females. The indicates that obese males and females were 2.2 times and 1.4 times, respectively, more likely to have heart disease than males and females with normal weight.

The portions (population attributable fractions–PAFs) of heart disease found in this report that can be attributed to obesity were 17.4% for males and 10.3% for females.

As discussed in Chapter 4.2.3.6, gender breakdowns for heart disease showed marginal sample variability (16.6–≤33.3). Age breakdowns showed a sample variability of more than 33.3, which is considered to be in the unacceptable range by Statistics Canada. Thus, the age and gender breakdowns for heart disease are presented here for illustrative purposes, rather than for the purpose of providing statistically significant results.

4.4.3 Cerebrovascular disease (stroke)

According to Alberta Health and Wellness, cerebrovascular disease, or stroke, is responsible for the second largest number of deaths in Canada, after coronary heart disease, but mortality rates are showing a decreasing trend over time.[441] Approximately one-third of stroke victims die within 12 months after a stroke, and half of the survivors are disabled over the long-term.[442] Recurrence of stroke is frequent, and approximately 25% of people who recover from the first stroke will have another stroke within the next five years. The prevalence of cerebrovascular

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[439] Public Health Agency of Canada (PHAC). *Cardiovascular Disease Surveillance on-Line*, accessed.
[440] Heart and Stroke Foundation of Canada. *Tipping the Scales of Progress: Heart Disease and Stroke in Canada 2006*, accessed.
[441] Alberta Health and Wellness. *Report on the Health of Albertans*, accessed.
[442] Access Economics. *The Economic Costs of Obesity*, accessed.

disease increases sharply later in life, and is fairly stable until age 65, after which it rises rapidly.[443]

Alberta Health and Wellness reports that stroke is also a leading cause of long-term disability in both male and female adults.[444] It occurs when a blood vessel to the brain is suddenly blocked so that oxygen and nutrients do not reach the brain. If it is not fatal, such a blockage can result in mainly permanent damage to the brain that can impair functions such as movement, vision, and speech. In addition, Alberta Health and Wellness notes:

> Strokes often lead to paralysis, limb weakness, mental problems, pain in the hands and feet, and death. Stroke survivors may never recover from the brain damage inflicted by the stroke, and rehabilitation and lifestyle management associated with having a stroke lasts a lifetime.[445]

Obesity is one of the main modifiable risk factors for stroke.[446] The WHO *Comparative Quantification of Health Risks* study found that in the America-A subregion (consisting of Canada, United States, and Cuba), 40% of stroke in males, and 37% in females, aged ≥30 years, could be attributed to a BMI of ≥30.[447]

Field et al. estimated the risk of stroke by BMI category for middle-aged U.S. adults in a 10-year follow-up study of the Nurses Health Study and in the Health Professionals Follow-up Study in the U.S.[448] Interestingly, this study found very significant gender differences, with obese men more than twice as susceptible to stroke as obese women. Thus, compared with women of normal weight, the odds of having a stroke were found to be 1.2, 1.0, and 1.1 for women who were overweight, obese class 1 (BMI 30.0–34.9), and severely obese (classes 2 and 3 – BMI ≥35), respectively. For men who were overweight, obese, and severely obese, the odds of having a stroke were 1.2, 2.0, and 2.3, respectively.[449]

Katzmarzyk and Janssen found that the RRs associating obesity and stroke in the literature ranged from 1.02 to 1.70, and they used a summary RR for stroke of 1.50 in their study on the cost of obesity in Canada.[450, 451]

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[443] Alberta Health and Wellness. *Report on the Health of Albertans*, accessed.

[444] Ibid., accessed.

[445] Ibid., accessed.

[446] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

[447] Ibid.

[448] Field, Coakley, Must, Spadano, Laird, Dietz, Rimm, and Colditz. "Impact of Overweight on the Risk of Developing Common Chronic Diseases During a 10-Year Period."

[449] Ibid.

[450] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[451] Katzmarzyk and Janssen note, "Summary relative risk (RR) estimates were calculated using a general variance-based method of meta-analysis," but they did not explain this methodology. p. 100.

4.4.3.1 Cerebrovascular disease (stroke) statistics in Alberta

Based on administrative data, Alberta Health and Wellness reports that the age-standardized treated prevalence of stroke in Alberta in 2003—or the percentage of Albertans receiving care that year as the result of a stroke—was 0.63 per 100 people in the population.[452] As elsewhere, stroke is relatively rare in Alberta before the age of 65, but then increases sharply and is highest in adults over age 75.[453]

Although the age-standardized rates for stroke have remained stable, the actual number of cases has increased, mainly because the population is aging. Thus, the age-adjusted rate for stroke in Alberta remained fairly stable at around 7.7 per 1,000 persons between 1995 and 2005. However, 18,531 Albertan adults aged 20 and over suffered a stroke in Alberta between in 2004—an increase of over 35% from 1985 when 13,700 adults suffered a stroke.[454]

Please see Chapter 4.4.2.1 above for information on the proportion of cardiovascular disease mortality attributable to stroke.

According to 2005 CCHS, cycle 3.1, the percentage of the Canadian population 15 years of age and over who reported suffering from the effects of a stroke was 1.4% among obese males and 1.6% among obese females.

Comparing the obese population (i.e. the exposed population) (BMI $\geq$30) to the normal weight population (i.e. the unexposed) (BMI 18.5–24.9) for Canadians aged $\geq$15 using data from 2005 CCHS cycle 3.1, the age-adjusted prevalence-based risk ratio for stroke was 1.1 for males and 1.4 for females 15 years of age and over. This indicates that obese males and females were 1.1 and 1.4 times, respectively, more likely to have a stroke than the normal weight population.

The portion (population attributable fraction–PAF) of obesity that can be attributed to stroke that was found in this report was 2.0% for males and 7.1% for females.

4.5 Cancer

Although the link between obesity and cancer is less well defined and quantified than the links between obesity and CVD and diabetes, obesity has consistently been associated with a high risk of developing four types of cancer in particular—colon cancer, postmenopausal breast cancer, endometrial (uterine) cancer, and kidney cancer.[455] In addition, other cancers—linked with obesity less often than those main four types but which a growing body of epidemiological evidence is associating with obesity—include cancer of the ovary and gallbladder in women; cancer of the rectum and prostate in men; and cancer of the esophagus (adenocarcinomas) and

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[452] Alberta Health and Wellness. *Report on the Health of Albertans*, accessed.
[453] Alberta Diabetes Surveillance System. *Alberta Diabetes Atlas 2007*, accessed.
[454] Ibid., accessed.
[455] National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, accessed.

pancreas, and non-Hodgkin's lymphoma, leukemia, and multiple myeloma in both men and women.[456] Evidence for a link between obesity and gallbladder cancer has been considered to be 'probable' rather than 'convincing'.[457]

Because the Alberta Cancer Board, in commissioning this present study, has expressed particular interest in identifying links between obesity and cancer, cancer-related evidence is therefore described in considerably more detail in the pages that follow than for other illness categories and than in other Canadian obesity cost studies. Fortunately, a major comprehensive new study released in November 2007 by the World Cancer Research Fund and American Institute for Cancer Research, and other very recent (2005–08) studies from Germany, the U.K., and Harvard School of Public Health now make this more detailed, site-specific analysis of obesity-cancer links possible.

According to Haslam and James, "obesity is one of the most important known preventable causes of cancer."[458] However, they note that the underlying mechanisms that connect obesity and cancer are difficult to define:

> [C]olon cancer has been linked to hyperinsulinism. Breast cancer seems to be related to the abnormally high concentrations of free oestrogen in postmenopausal obese women caused by peripheral conversion of sex hormones in adipose tissue by aromatase, together with a fall in the concentrations of plasma sex-steroid-binding globulin. These changes probably also explain the propensity to endometrial cancer and could be relevant to the suggested link between overweight and prostate cancer.[459]

In general, physiological mechanisms through which excess body weight might influence the risk of cancer have been identified as an elevated inflammatory response, increases in circulating hormones, and decreases in insulin sensitivity.[460]

The portion of cancer mortality and morbidity that can be attributed to obesity has been estimated for various populations using different methodologies, and is, therefore, not uniform across studies. Indeed, estimates vary widely. Thus, WHO estimates that globally between one-fourth and one-third of cancer cases can be attributed to excess weight.[461] In Australia, it is estimated that 20.5% of the four major types of cancer are caused by obesity.[462] In Canada, Pan et al. estimate that, in 2001, 4.85% of all cancers (including 19 types) in adults aged 20–76— 4.43% for men and 5.43% for women—could be attributed to obesity.[463] Pan et al. found that risk for cancer was 34% higher in obese adults than in normal weight adults.

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[456] Ibid., accessed.
[457] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.
[458] Haslam, and James. "Obesity." p. 1201.
[459] Ibid. p. 1201.
[460] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.
[461] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."
[462] Access Economics. *The Growing Cost of Obesity in 2008: Three Years On*, accessed.
[463] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

In November 2007, the U.K.-based World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR) released a 537-page report on cancer and body weight, diet, and physical activity titled *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*.[464] The report, which it called "the most comprehensive report on cancer prevention ever published," is the result of a five-year process involving 9 teams of scientists and 21 experts from around the world who between them analysed over 7,000 studies.[465]

The 2007 WCRF/AICR report is a ten-year update of an earlier report, *Food, Nutrition and the Prevention of Cancer: A global perspective,* which was published in 1997. The emphasis of the new report is on biological factors that modify the risk of cancer. A separate 200-page report that identifies deeper social, cultural, economic, and ecological factors that affect cancer rates, and the implications of these underlying links for policy, was released in February 2009.[466] Time and resources did not permit GPI Atlantic to undertake further research to incorporate the findings of this new report—which appeared between the time our own research was undertaken and publication of this present report—but it is strongly recommended that the findings of this 2009 report be considered by the Alberta Cancer Board in conjunction with the findings of this present report.

The 2007 WCRF/AICR report concludes: "Maintenance of a healthy weight throughout life may be one of the most important ways to protect against cancer."[467]

> The scientific community is convinced that inherited high susceptibility to cancer accounts for only a small proportion of cases. Although we are all more or less susceptible to various diseases, most adult cancers are caused mainly by environmental factors. This means that most cancers are at least in theory preventable.[468]

The 2007 WCRF/AICR report does not use the traditional BMI classification *cut-points* for its analysis (e.g. "overweight–BMI 25–29.9, "obese"–BMI ≥30, etc.) so it is difficult to compare its results with other reports, and the WCRF/AICR report did not attempt to do so. Instead, the WCRF/AICR report uses the term "body fatness" rather than "overweight" or "obesity," and estimates the risks associated with body fatness as continuous across the range of BMI. In other words it uses a measurement of change in weight that, it notes, "tends to be more precise than static measures such as weight or BMI [categories]."[469] Therefore, it reports increased risk as a per unit increase in kg/m^2 (e.g. increased risk per 1 kg/m^2 or per 5 kg/m^2, etc.).

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[464] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[465] Ibid., accessed.

[466] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Policy and Action for Cancer Prevention. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, Washington, D.C.: AICR, 2009; accessed February 2009; available from http://www.dietandcancerreport.org/downloads/Policy_Report.pdf?JServSessionIdr008=kts074vm65.app46a.

[467] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed. p. xvii.

[468] Ibid., accessed. p. 244.

[469] Ibid., accessed. p. 214.

In another report, however, Bergstrom et al. explain how results reported in terms of unit of BMI increases could potentially be compared to measures based on standard BMI categories. Thus, they note that one unit of increase in BMI (1 kg/m^2) corresponds to 3.1 kg for a man of average height (1.77 m or approximately 5'9"), and to 2.7 kg for a women of average height (1.64 m or approximately 5'4").[470] Thus, an increase in RR of 1.07 per unit of BMI (1 kg/m^2) corresponds roughly to a RR of 1.35 for overweight adults, 1.70 for obese class 1, 2.05 for obese class 2, and 2.40 for obese class 3, when compared to those with healthy ("normal") weight.[471]

WCRF/AICR estimates the following summary relative risk ratios for specific cancer sites. (When one RR is listed, it is summarized from cohort studies. When two RRs are listed, the first one is from cohort studies and the second is from case control studies):

> RR per 1 kg/m^2 –
> - Colorectal – 1.03
> - Esophageal – 1.11
>
> RR per 2 kg/m^2 –
> - Postmenopausal breast – 1.03, 1.05
>
> RR per 5 kg/m^2 –
> - Kidney – 1.31, 2.05
> - Pancreatic – 1.14, 1.00
> - Endometrial – 1.52, 1.56
> - Gallbladder – 1.23, 1.19

WCRF/AICR also summarizes the evidence found in 20 specially commissioned systematic literature reviews related to 17 cancer sites. It found that about 14 cancer sites are specifically related to "body fatness", among which 8 cancer sites are strongly related to "body fatness". The authors of the WCRF/AICR report remark that the 17 cancer sites profiled amount to roughly 80% of the incidence of, and deaths from, all cancers worldwide. Some of this evidence, based on which the authors conclude that "body fatness" is a "cause" of the 8 specific cancers for which clear links were found, is described in the following sections concerning specific cancers.

In a study released in 2008, researchers from the University of Manchester conducted a systematic review and meta-analysis of reports produced between 1966 and November 2007, which included 221 data sets and more than 282,000 incident cases, to estimate the impact of excess weight on 20 cancer types.[472] The analysis was designed to determine the risk of cancer associated with a 5 kg/m^2 increase in BMI. The authors, Andrew Renehan et al., note that the obesity-cancer associations found in studies from North America, Europe, Australia, and the Asia-Pacific region are similar to their results.

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[470] Bergstrom, A., C-C. Hsieh, P. Lindblad, C-M Lu, N.R. Cook, and A. Wolk. "Obesity and Renal Cell Cancer - a Quantitative Review," *British Journal of Cancer*, 2001, vol. 85, no. 7: 984-990.

[471] Ibid.

[472] Renehan, Andrew G, Margaret Tyson, Matthias Egger, Richard F Heller, and Marcel Zwahlen. "Body-Mass Index and Incidence of Cancer: A Systematic Review and Meta-Analysis of Prospective Observational Studies," *Lancet*, 2008, vol. 371: 569-578.

Renehan et al. found that a 5 kg/m^2 increase in BMI was strongly associated with esophageal and kidney cancer in both sexes; colon cancer in men; and with endometrial and gallbladder cancer in women.[473] They also noted weaker positive associations between increased BMI and leukemia, multiple myeloma, and non-Hodgkin's lymphoma in both sexes; rectal cancer and malignant melanoma in men; and postmenopausal breast, pancreatic, and colon cancers in women.

Goodarz Danaei et al. of the Harvard School of Public Health estimated mortality rates from 12 types of cancer that could be attributed to 9 risk factors, including high BMI, in seven World Bank regions.[474] An expert working group conducted comprehensive and systematic reviews and meta-analyses of epidemiological studies and other sources such as government reports and international databases to obtain data on the specific risk factors and associated risk ratios associated with these twelve cancer types.

In the report published in 2005, Danaei et al. found that in high-income countries, the most important causes of cancer were smoking, alcohol use, and overweight and obesity—in that order. They also found that the cancer sites most frequently affected by obesity were cancers of the uterus (endometrial), colon, breast (postmenopausal), gallbladder, and kidney. In high-income countries, they attributed 3% of all cancer deaths to overweight and obesity—with 14% of colorectal, 13% of breast, and 43% of uterine cancer deaths attributable to overweight and obesity.

Very recently—in 2008—and based on a review by the WHO International Agency for Research on Cancer and other relevant studies, German researchers Tobias Pischon et al. provided another major overview of the demonstrated associations between cancer risk and excess body weight, mainly in Europe in 2006.[475] The main cancers found to be related to excess weight included colon, postmenopausal breast, endometrial, kidney, esophageal, pancreatic, and advanced prostate cancer. The relative risk ratios they found were generally higher than those reported by WCRF/AICR. Some of these site-specific relative risk ratios from Pischon et al. are referenced in the sections on specific cancer sites below.

In addition, Pischon et al. note that other cancers have been linked to obesity, but "overall the amount of data available is limited and does not allow definite conclusions."[476] Cancers they place in this category include cancers of the gallbladder, cervix and ovaries, and non-Hodgkin's lymphoma, multiple myeloma, and leukemia. According to Pischon et al., most of the evidence concerning the relationship between BMI and these malignancies has come mainly from limited case-control studies and from studies that have included only a small number of cases.

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[473] Ibid.

[474] Danaei, Goodarz, Stephen Vander Hoorn, Alan D. Lopez, Christopher J.L. Murray, Majid Ezzati, and the Comparative Risk Assessment collaborating group (Cancers). "Causes of Cancer in the World: Comparative Risk Assessment of Nine Behavioural and Environmental Risk Factors " *Lancet*, 2005, vol. 366: 1784-1793.

[475] Pischon, Tobias, Ute Nothlings, and Heiner Boeing. "Obesity and Cancer," *Proceedings of the Nutrition Society*, 2008, vol. 67: 128-145.

[476] Ibid. p. 137

It should be noted that the lack of present capacity to quantify links between these other cancers and obesity does not mean that strong associations do not in fact exist. Rather, because obesity-cancer research is so new, more definitive, quantifiable determination of the relative risks must await the availability of more robust data sources based on larger sample sizes. Just as it took many years for the association between smoking and lung cancer to be proved definitively and many additional years for the link with heart disease to be demonstrated, so we are presently only beginning to investigate and understand the links between obesity and cancer. As the very recent dates of the studies cited above indicate—mostly from 2005-2008—it would not have been possible to include this present section in this report even a few years ago.

The following sections (4.5.1 – 4.5.14) review some of the available site-specific evidence that does exist on the specific cancer types that have been found to be partially attributable to obesity, and Chapter 4.5.15 then provides cancer statistics that are specific to Alberta for the purpose of estimating relevant obesity-related costs for these cancers.

The relative risk ratios and population attributable fractions used for the specific cancer sites, as well as the methodology employed, are reported in Part 2 of this document in Chapters 5.4.3 and 5.6.

4.5.1 Colorectal (colon and rectal) cancer

According to WCRF/AICR, the evidence that body fatness, in part, causes colorectal cancer (colon and rectal cancers) is convincing, although the evidence is stronger for colon cancer than for rectal cancer.[477] WCRF/AICR reports that colorectal cancers are the 3rd most common types of cancer worldwide, accounting for 9% of cancer cases overall.[478] Colorectal cancer is also fatal in half of all cases, is the 4th most common cause of cancer death, and accounts for 8% of all cancer deaths. It is mainly a disease of high-income countries, and risk increases with age until it levels off in old age. Pischon et al. report that in Europe, colorectal cancer incidence accounts for 12.8% and 13.1% of total cancer incidence in men and women respectively, and for 11.3% and 13.3% of cancer deaths in men and women respectively.[479]

Edward Giovannucci and Dominique Michaud of the Harvard School of Public Health note that obesity is consistently associated with colon cancer in men (RR 2.0), but this has not been observed in women.[480] However, when the waist circumference or waist-to-hip ratio is used as the measure of obesity rather than BMI, the association with colon cancer among obese women becomes clear. For example, Pischon et al. note that when waist-hip ratios were used as measures of excess body fat, both men and women in the highest quintile of waist-hip ratios had a 50%

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[477] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.
[478] Ibid., accessed.
[479] Pischon, Nothlings, and Boeing. "Obesity and Cancer."
[480] Giovannucci, Edward, and Dominique Michaud. "The Role of Obesity and Related Metabolic Disturbances in Cancers of the Colon, Prostate, and Pancreas," *Gastroenterology*, 2007, vol. 132: 2208-2225.

higher risk of developing colon cancer than those in the lowest quintile over a mean follow-up period of 6 years.[481]

James et al. describe the potential mechanisms by which weight gain might potentially increase the risk of developing colon cancer, though they acknowledge that the physio-biological pathways are not yet properly understood:

> The mechanisms by which weight gain might accentuate the risk of developing large adenomas and colon cancer are unclear, but the stronger association of high BMIs with large rather than small adenomas suggests that excess weight operates at a relatively late stage in the promotion of tumour formation. Excess weight is associated with a wide range of hormonal and metabolic effects that may be involved in the promotion of cancer. Dietary factors could, in theory, be confounders with high meat intake, especially processed meat, and a low intake of fibre-rich vegetables and fruit being particularly linked to colon cancer and also being part of a weight-gain-inducing, energy-dense diet. However, several of the studies also assessed diet, and the impact of higher BMIs seemed to be independent of the direct dietary effects.[482]

The WHO *Comparative Quantification of Health Risks* study found that in the America-A subregion (consisting of Canada, United States, and Cuba), 17% of colon cancer in males and 18% in females, aged ≥30 years, could be attributed to a BMI of ≥30.[483] The WCRF/AICR meta-analysis of 60 cohort studies that examined the risk association between excess weight and colorectal cancer showed a 15% increased risk per 5 kg/m^2, with a summary RR estimate of 1.03 per 1 kg/m^2.[484, 485]

As noted, in their study of cancer risk and obesity in Canada for adults aged 20–76, Pan et al. estimated that 12.24% of colon cancer—15.65% among men and 9.73% among women—could be attributed to obesity in 2001.[486] For overweight men and women, they estimated that 11.66% of colon cancer—17.76% among men and 5.21% among women—could be attributed to overweight in 2001.

Fields et al. estimated the risk of colon cancer by BMI for middle-aged U.S. adults in a 10-year follow-up study of the Nurses Health Study and in the Health Professionals Follow-up Study in the U.S.—which was described in Section 4.3 above on type 2 diabetes.[487] Compared with respondents with normal weight, the odds of developing colon cancer were 1.2, 1.3, and 1.8 for women who were overweight, obese class 1 (BMI 30.0–34.9), and severely obese (BMI ≥35),

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[481] Pischon, Nothlings, and Boeing. "Obesity and Cancer."

[482] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)." p. 562.

[483] Ibid.

[484] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[485] James et al. do not describe the method for summarizing the RRs beyond noting: "the relative risk is calculated from each study's provision of the distribution of BMIs within the studied population." p. 561.

[486] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[487] Field, Coakley, Must, Spadano, Laird, Dietz, Rimm, and Colditz. "Impact of Overweight on the Risk of Developing Common Chronic Diseases During a 10-Year Period."

respectively. For men who were overweight, obese, and severely obese, the odds of developing colon cancer were found to be 1.2, 1.7, and 1.3, respectively.[488]

Susanna Larsson and Alicja Wolk of Karolinska Institutet in Sweden recently conducted a meta-analysis of studies published between 1966 and April 2007, in order to summarize the available evidence from prospective studies on the associations between obesity and the risk of colon and rectal cancers, which they analysed separately.[489] They found that a 5-unit (5 kg/m^2) increase in BMI was a stronger risk for colon cancer in men (RR 1.30) than in women (RR 1.12). They also found an increased risk per 5-unit (5 kg/m^2) increase in BMI for rectal cancer in men (RR 1.12), but not in women (RR 1.03) where the apparently increased risk was not significant.

Australian researchers Alireza Moghaddam et al. also conducted a recent meta-analysis of studies assessing the association between obesity and the risk of colorectal cancer.[490] Their pooled estimate of risk ratios from the studies indicated that obese adults generally had a 40% greater risk of colorectal cancer (RR 1.40) than adults with normal weight. The pooled estimate for the risk of colorectal cancer among obese men (RR 1.46) was significantly higher than that for obese women (RR 1.15). When looked at separately, the pooled estimates of colon and rectal cancer risks indicated a higher association with obesity for colon cancer than for rectal cancer. However, the researchers noted that, when corrected for the presence of publication bias—a statistical method sometimes used to correct for small sample size—the difference between colon cancer and rectal cancer risks among obese adults was not significant.

The majority of the 31studies examined by Moghaddam et al. were from the U.S. or Europe, but two were from Canada. The first was a 2002 Canadian cohort study by P.D. Terry et al. that looked at colon cancer risk in women, and the other was the case-control study conducted by Sai Yi Pan et al., which was previously discussed (see this section above).[491, 492] Terry et al. used data from the Canadian National Breast Screening Study for 89,835 women aged 40–59 years at recruitment who were followed for an average of 10.6 years. During that time, 527 of the women were diagnosed with colorectal cancer. Terry et al. found only a weak positive association between obesity and colorectal cancer overall (RR 1.08), no association among postmenopausal women (RR 0.73), and an increased association among women who were premenopausal at the time of recruitment (RR 1.88).[493]

Yang Mao et al. of the Canadian Cancer Registries Epidemiology Research Group used 1994–1997 data from the Canadian National Enhanced Cancer Surveillance System (NECSS) to

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[488] Ibid.

[489] Larsson, Susanna C., and Alicja Wolk. "Obesity and Colon and Rectal Cancer Risk: A Meta-Analysis of Prospective Studies," *American Journal of Clinical Nutrition*, 2007, vol. 86: 556-565.

[490] Moghaddam, Alireza Ansary, Mark Woodward, and Rachel Huxley. "Obesity and Risk of Colorectal Cancer: A Meta-Analysis of 31 Studies with 70,000 Events," *Cancer Epidemiology Biomarkers & Prevention*, 2007, vol. 16, no. 12: 2533-2547.

[491] Terry, P. D., A. B. Miller, and T. E. Rohan. "Obesity and Colorectal Cancer Risk in Women," *Gut*, 2002, vol. 51, no. 2: 191-194.

[492] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[493] Terry, Miller, and Rohan. "Obesity and Colorectal Cancer Risk in Women."

examine the relationship between obesity and rectal cancer.[494] They found a statistically significant increased risk of rectal cancer among obese women (OR 1.44), and among overweight (OR 1.40) and obese men (OR 1.78). The authors note that although studies of rectal cancer that are separate from studies of colon cancer are relatively rare, their findings are consistent with those from previous studies.

4.5.1.1 Colorectal cancer statistics for Alberta

Katzmarzyk and Janssen found RRs for colon cancer in the literature ranging from 1.41 to 3.30, and used a summary RR of 1.45 in their 2004 study of the economic costs of obesity in Canada in 2001.[495, 496] They did not include rectal cancer in their analysis or cost estimates.

In their study of obesity and cancer risk in Canada for adults aged 20–76, Pan et al. estimated the total OR for colon cancer among obese adults (BMI ≥30) to be 1.93—OR 2.16 for men and OR 1.77 for women, compared to those with normal weight.[497, 498] They also estimated the total OR for colon cancer among overweight adults (BMI 25–29.9) to be 1.40—OR 1.54 for men and OR 1.22 for women.[499]

For rectal cancer, Pan et al. estimated the total OR to be 1.65 among obese adults—OR 1.75 for men and OR 1.50 for women.[500, 501] They also estimated the total OR for rectal cancer among overweight adults to be 1.36—OR 1.41 for men and OR 1.28 for women.[502]

According to the Alberta Cancer Registry, colorectal cancer accounted for 12.4% of all new cancers among Albertan adults aged ≥15 in 2005—6.8% of new cases were among males and 5.5% were among females.[503] There were 1,569 new cases of colorectal cancer in the province in 2005—867 among males and 702 among females.

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[494] Mao, Yang, Saiyi Pan, Shi Wu Wen, Kenneth C. Johnson, and The Canadian Cancer Registries Epidemiology Research Group. "Physical Inactivity, Energy Intake, Obesity and the Risk of Rectal Cancer in Canada," *International Journal of Cancer*, 2003, vol. 105: 831-837.

[495] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[496] Katzmarzyk and Janssen note, "Summary relative risk (RR) estimates were calculated using a general variance-based method of meta-analysis," but they did not explain this methodology. p. 100.

[497] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[498] Confidence intervals (CI) for colon cancer / obesity associations are: OR 1.93 (CI 1.61–2.31); OR 2.16 (1.68–2.78); OR 1.77 (1.35–2.32).

[499] Confidence intervals (CI) for colon cancer / overweight associations are: OR 1.40 (1.21–1.61); OR 1.54 (1.27–1.86); OR 1.22 (0.98–1.52).

[500] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[501] Confidence intervals (CI) for rectum cancer / obesity associations are: OR 1.65 (1.36–2.00); OR 1.75 (1.35–2.28); OR 1.50 (1.11–2.02).

[502] Confidence intervals (CI) for rectum cancer / overweight associations are: OR 1.36 (1.17–1.57); OR 1.41 (1.15–1.71); OR 1.28 (1.02–1.61).

[503] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* Alberta Health Services, Alberta Cancer Board, 2008; accessed July 2008; available from

Among individuals below the age of 25 there were 2 new cases (and none among children aged 0–14 years), and between ages 25 and 40 there were 27 new cases. After the age of 40 the incidence of new cases rose progressively by age group until the number of new cases peaked at age 80, after which the number of cases began to decline—presumably because of fewer people in the 80+ age group. This age-related pattern was the same for both males and females.

As of January 1, 2005 there were 9,483 Albertans living with colorectal cancer in the province.[504] Therefore, in 2005, when the incidence of 1,569 new cases is added, it can be estimated that 11,052 Albertans were living with colorectal cancer for all or part of 2005. This includes the people who died from colorectal cancer during that year, but who also incurred costs associated with the disease prior to their deaths.[505] These data, however, were not used in the cost estimates.

The PAFs from Pan et al. were used in this report to estimate colorectal costs that were attributable to obesity. For colon cancer, the PAFs were 12.24 for both genders, 15.65 for males and 9.73 for females, and for rectal cancer, the PAFs were 8.88 for both genders, 10.71 for males, and 6.54 for females.

4.5.2 Postmenopausal breast cancer

According to WCRF/AICR, breast cancer is the most common cancer in women worldwide, accounting for 23% of all cancer incidence in women and 11% of adult cancer cases overall.[506] Breast cancer incidence rates in high-income countries are three times those found in low- or middle-income countries. Globally, breast cancer is also the leading cause of cancer death in women, and—as the 5th most common cause of cancer death overall—accounts for 14% of total cancer deaths worldwide. However, 5-year survival rates for breast cancer in high-income countries are around 73%.

Pischon et al. note that breast cancer is the most frequent type of cancer among European women, and accounts for a significantly higher percentage of overall cancer incidence and mortality in Europe than the WCRF/AICR global average rates—28.9% of all new female cancers and 17.6% of female cancer deaths in Europe.[507]

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http://www.cancerboard.ab.ca/NR/rdonlyres/2BADD738-9C4D-487F-B8C1-68D5D962CD81/0/AlbertaCancerRegistry200531Oct08.pdf.

[504] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

[505] These data, however, were not used to estimate the mortality costs of colorectal cancer.

[506] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[507] Pischon, Nothlings, and Boeing. "Obesity and Cancer."

The link between excess weight and breast cancer in postmenopausal women—generally those over the age of 50 years—is clear and robust.[508] As reported in *The Lancet*, University of Oxford researchers Timothy Key et al. found that the risk for breast cancer in postmenopausal women is 50% higher in obese women than in women of normal weight. By contrast Key et al. observed no association between excess weight and breast cancer in *pre*menopausal women.[509]

James et al. explain the mechanisms through which obesity can affect postmenopausal breast cancer:

> Mechanistically, it seems clear that the risk of developing postmenopausal breast cancer is increased in women with raised plasma and tissue concentrations of estrogens. The activity of these hormones is greater when there are lower circulating concentrations of the sex hormone-binding globulin (SHBG). Obesity, with its associated insulin resistance, lowers SHBG levels; overweight women are also found to have higher circulating concentrations of total and bioavailable androgens and estrogens. Confirmation of the importance of these hormonal changes comes from the observation that women exposed to combined estrogens and progesterones as part of postmenopausal hormone replacement therapy subsequently have increased rates of breast cancer, the risk being greater in those on combined compared with estrogen-alone treatment.[510]

The WCRF/AICR meta-analysis that examined the association between breast cancer and excess weight found an 8% increased risk per 5 kg/m^2 for postmenopausal breast cancer in cohort studies, and a 13% increased risk per 5 kg/m^2 for case-control data, although WCRF/AICR notes that other studies have found even higher risks.[511]

Eighteen researchers from four countries who were working with the Pooling Project of Diet and Cancer, pooled data from 7 cohort studies that followed a total of more than 337,000 women for an average of 5 years, during which more than 4,300 breast cancer cases were reported.[512] The studies included different age ranges—28–90, 34–59, 40–59, 40–65, 40–76, and 55–69 for two studies. One of the seven studies examined was the Canadian National Breast Screening (CNBS) study, which showed an 11% increased risk of postmenopausal breast cancer per 4 kg/m^2 above BMI <21. In general, however, the study found a 26% risk of postmenopausal breast cancer among women with a BMI of ≥28, which only rose to 27% with a BMI of ≥33.

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[508] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

[509] Key, Timothy J., Pia K. Verkasalo, and Emily Banks. "Epidemiology of Breast Cancer," *The Lancet*, 2001, vol. 2, no. March: 133-140.

[510] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)." p. 561.

[511] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[512] van den Brandt, Piet A. , Donna Spiegelman, Shiaw-Shyuan Yaun, Hans-Olov Adami, Lawrence Beeson, Aaron R. Folsom, Gary Fraser, R. Alexandra Goldbohm, Saxon Graham, Larry Kushi, James R. Marshall, Anthony B. Miller, Tom Rohan, Stephanie A. Smith-Warner, Frank E. Speizer, Walter C. Willett, Alicja Wolk, and David J. Hunter. "Pooled Analysis of Prospective Cohort Studies on Height, Weight, and Breast Cancer Risk," *American Journal of Epidemiology*, 2000, vol. 152: 514-527.

This finding appears to indicate that—unlike most obesity-related illnesses where risk increases in direct proportion to increases in BMI, with the severely obese (BMI ≥35) and obese (BMI ≥30) at proportionately and substantially greater risks than those who are overweight (BMI 25–29.9)—in this case the bulk of the risk of postmenopausal breast cancer appears to occur once women become somewhat overweight and does not increase substantially after that. If confirmed in other studies, this would have important implications for the effective targeting of prevention programs among women—with possible messaging aimed at pre-obese women who might otherwise comfortably accept a degree of excess weight without concern. As seen below, however, other studies, including that of Pan el al. in Canada, have found a much greater difference in risk between the overweight and obese groups than did the Pooling Project of Diet and Cancer study.

The WHO *Comparative Quantification of Health Risks* study found that 12% of postmenopausal breast cancer among obese women aged ≥30 years in the America-A subregion (consisting of Canada, United States, and Cuba) could be attributed to a BMI of ≥30.[513]

As noted, in their study of obesity and cancer risk in Canada, Pan et al. estimated that over 8.46% of postmenopausal breast cancer in Canadian women could be attributed to obesity (BMI ≥30) in 2001, and 4.08% could be attributed to overweight (BMI 25-29.9). The odds ratios were 1.17 for overweight and 1.66 for obesity. Although not strictly comparable, this difference appears to point to a potentially larger difference between rates of risk by BMI category than indicated by the Canadian National Breast Screening (CNBS) study results cited above.[514]

Pischon et al. report that postmenopausal European women who gain more that 20 kg between the ages of 20–60 years have a 52% increased risk of developing postmenopausal breast cancer compared with women whose weight has remained stable during adulthood.[515]

Other researchers have reached similar conclusions. For example, Swedish and American researchers Cecilia Magnusson et al. studied whether obesity during childhood, weight gain during adulthood, or adult obesity per se increased the risk of postmenopausal breast cancer. To investigate this question, they conducted a population-based Sweden-wide case-control study, which included 2,818 women aged 50–74 years who had been diagnosed with invasive breast cancer, and 3,111 women of similar age with no previous diagnosis of cancer, who served as controls.[516]

The results were both expected and surprising. Overall, Magnusson et al. found that the risk of postmenopausal breast cancer for obese women was 50% higher than for women of normal weight. They also found that adult weight gain increases the risk of postmenopausal breast cancer, but that this effect does not emerge until from 10–20 years after menopause (OR 1.52 ten

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[513] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

[514] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[515] Pischon, Nothlings, and Boeing. "Obesity and Cancer."

[516] Magnusson, Cecilia, John Baron, Ingemar Persson, Alicja Wolk, Reinhold Bergstro, Dimitrios Trichopoulos, and Hans-Olov Adami. "Body Size in Different Periods of Life and Breast Cancer Risk in Post-Menopausal Women," *International Journal of Cancer*, 1998, vol. 76: 29-34.

years after menopause). Women who by 20 years after menopause had gained 30 or more kg since age 18 had double the odds (OR 2.04) of breast cancer compared with those who had not gained weight.

Surprisingly, Magnusson et al. also found that women who were in the leanest body weight category at age 7 had three times the risk of postmenopausal breast cancer compared to women who were obese at the same age. Although the authors note that this inverse association has been previously reported, the reason why obesity in childhood might protect against postmenopausal breast cancer is unclear and the "issue awaits further documentation and biological interpretation."[517] However, they speculate that obesity in childhood may be associated with a diminished production of progesterone, which appears to increase the breast epithelial cell proliferation that is a risk factor for malignancy, and that consequent reduced serum levels might lower breast cancer risk.

4.5.2.1 Breast cancer statistics in Alberta

In their study of the costs of obesity in Canada, Katzmarzyk and Janssen used a RR for postmenopausal breast cancer of 1.47, which was summarized from RRs in the epidemiological literature that ranged from 0.98 to 1.60.[518, 519]

Pan et al., in their study of obesity and cancer risk in Canada, estimated the total OR for breast cancer—both pre- and postmenopausal—among obese Canadian women aged 20–76 to be 1.51.[520] The OR of obesity for premenopausal breast cancer was 1.13, and for postmenopausal breast cancer the OR was 1.66.[521]

Pan et al. also estimated the total OR for breast cancer—both pre- and postmenopausal—among overweight Canadian women aged 20–76 to be 1.08.[522] The OR of overweight for premenopausal breast cancer was 0.89, and for postmenopausal breast cancer the OR was 1.17.[523] As noted, the very substantial difference in risk for postmenopausal breast cancer between overweight and obese women (OR 1.17 vs OR 1.66) found by Pan et al. appears to contradict the rather counter-intuitive finding of the Pooling Project of Diet and Cancer study cited above, which did not find a substantial difference in risk between women with a BMI of ≥28 and those with a BMI of ≥33.

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[517] Ibid. p. 32.

[518] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[519] Katzmarzyk and Janssen note, "Summary relative risk (RR) estimates were calculated using a general variance-based method of meta-analysis," but they did not explain this methodology. p. 100.

[520] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[521] Confidence intervals (CI) of obesity for breast cancer are: OR 1.51 (CI 1.26–1.80); 1.13 (0.82–1.58); OR 1.66 (1.33–2.06).

[522] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[523] Confidence intervals (CI) of overweight for breast cancer are: OR 1.08 (0.94–1.24); OR 0.89 (0.70–1.14); OR 1.17 (1.00–1.39).

According to the Alberta Cancer Registry, breast cancer accounted for 14.2% of the 12,669 new adult cancer cases among Albertans of all ages in 2005.[524] There were a total of 1,788 new breast cancer cases among Albertan women aged ≥20 (the earliest age in which breast cancer occurred) in 2005, which amounted to about 30% of new female cancer cases (excluding nonmelanoma skin cancer, as does the Alberta Cancer Registry).

Because excess body weight is mainly associated with postmenopausal breast cancer, which therefore rarely appears before age 50, it is important to break down the available Alberta breast cancer data by age group in order to assess the proportion of cases that are likely to be postmenopausal.[525] Table 22 below shows the estimated number of females in Alberta in 2005 by age group, age-standardized breast cancer incidence rates per 100,000 females by age group, and the actual number of new breast cancer cases per age group in 2005.

Of the total number of new female breast cancer cases in Alberta in 2005 (1,788), 458 cases can be estimated to be premenopausal and 1,330 can be estimated to be postmenopausal, based on an age cut-off of 50 years for the occurrence of menopause. Based on this breakdown, premenopausal new breast cancer cases are seen to represent about 25.6% of all new female breast cancer cases in 2005, while postmenopausal cases represent about 74.4%.

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[524] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.
[525] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

Table 22. Breast cancer incidence by age group, age-standardized incidence rates per 100,000 females, and actual number of new cases, Alberta, 2005

Age group	Population estimate: Number of females	Breast cancer incidence rate	Actual number of new cases
0 – 19	417,712	0.00	0
20 – 24	121,839	0.8	1
25 – 29	120,970	6.9	8
30 – 34	116,379	20.8	24
35 – 39	116,308	46.6	55
40 – 44	136,155	109.2	150
45 – 49	131,871	164.7	220
Total premeno-pausal	1,161,234	—	458
Postmenopausal:			
50 – 54	109,626	210.4	232
55 – 59	88,375	216.1	192
60 – 64	63,425	297.4	189
65 – 69	50,534	350.5	177
70 – 74	44,264	382.0	169
75 – 79	37,680	371.5	140
80 – 84	29,066	490.4	142
85 – 89	17,109	383.1	66
90+	9,802	230.3	23
Total Postmeno-	449,881	—	1,330

pausal			
TOTAL	1,611,115	—	1,788

Sources: Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics* Alberta Health Services, Alberta Cancer Board, 2008; accessed July 2008; available from http://www.cancerboard.ab.ca/NR/rdonlyres/2BADD738-9C4D-487F-B8C1-68D5D962CD81/0/AlbertaCancerRegistry200531Oct08.pdf; Statistics Canada. *Annual Demographic Statistics 2005*, Catalogue no. 91-213-XIB, 2006; accessed November 2008; available from http://www.statcan.ca/english/freepub/91-213-XIB/0000591-213-XIB.pdf.

As of January 1, 2005, there were 18,530 Albertan women living with breast cancer.[526] If postmenopausal breast cancer accounted for an estimated 74.4% of female breast cancers in 2005, it may be estimated that 13,786 Albertan women were living with postmenopausal breast cancer at the beginning of 2005. When the incidence of the 1,330 new postmenopausal cases is added to this total, it can be estimated that 15,116 Albertan women were living with postmenopausal cancer for all or part of 2005. This, of course, assumes that the mortality rates from premenopausal and postmenopausal breast cancer were the same. The 15,116 estimate includes those women who died from breast cancer during the year. These data, however, were not used in the cost estimates.

The PAF for postmenopausal breast cancer used in this report (8.46) was found in the study by Pan et al.[527]

4.5.3 Endometrial (uterine) cancer

According to WCRF/AICR, endometrial cancer in the lining of the uterus is the 17th most common cancer overall, and the 8th most common in women.[528] Globally, it accounts for 4% of all new cancer cases in women and 2% of new cancer cases overall. It is also the 21st most common cause of cancer death overall and the 13th most common cause of death in women, accounting for 1% of all cancer deaths overall, and 2% of cancer deaths in women worldwide.[529]

The highest incidence rates of endometrial cancer are in Europe and North America.[530] Pichon et al. report that in Europe endometrial cancer accounted for 10% of female cancer incidence and

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[526] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

[527] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[528] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[529] Ibid., accessed.

[530] Bjorge, Tone, Anders Engeland, Steinar Tretliand, and Elisabete Weiderpass. "Body Size in Relation to Cancer of the Uterine Corpus in 1 Million Norwegian Women," *International Journal of Cancer*, 2006, vol. 120: 378-383.

6.2% of cancer deaths in women in 2006.[531] In the U.S., endometrial cancer is the fourth most common incident cancer among women.[532]

U.S. researchers Marjorie McCullough et al. note that endometrial cancer was among the first cancers to be identified as being related to obesity.[533] WCRF/AICR finds that the evidence is convincing that body fatness is a cause of cancer of the endometrium, especially in postmenopausal women in high-income countries, who have an approximate 82% 5-year survival rate after diagnosis.[534] WCRF/AICR reports that its meta-analysis showed an overall 52% increased risk of endometrial cancer per 5 kg/m^2 in the cohort data,, and an overall 56% increased risk per 5 kg/m^2 in case-control data.[535]

James et al. explains the biological mechanisms through which obesity affects the development of endometrial cancer:

> The dominant mechanistic theory relates to the unopposed estrogen hypothesis, according to which estrogenic contraceptives or hormone replacement therapy enhance the risk of endometrial cancer, whereas progesterone-containing preparations confer protection. Estrogens are known to induce endometrial proliferation via local production of insulin growth factor (IGF-1), whereas progesterone induces the production of an endometrial IGF-1-binding protein. Women with low levels of plasma SHBG [Sex hormone-binding globulin], high levels of androgens and, after the menopause, elevated levels of total and bioavailable estrogens have an increased risk of endometrial cancer, as have younger women with the polycystic ovarian disease, which is associated with chronic anovulation and therefore low rates of production of progesterone. All these findings, therefore, fit the concept of excess available bioactive estrogen, which induces endometrial cell proliferation. Insulin resistance and higher concentrations of circulating IGF-1 induced by the lower concentrations of IGF-binding proteins in women who gain weight may also be involved.[536]

Rudolf Kaaks of the International Agency for Research on Cancer et al. note that endometrial cancer has strong environmental risk factors that are related to the western lifestyle, and that epidemiological studies have shown that over 40% of its incidence can be attributed to excess

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[531] Pischon, Nothlings, and Boeing. "Obesity and Cancer."

[532] Patel, Alpa V., Heather Spencer Feigelson, Jeffrey T. Talbot, Marjorie L. McCullough, Carmen Rodriguez, Roshni C. Patel, Michael J. Thun, and Eugenia E. Calle. "The Role of Body Weight in the Relationship between Physical Activity and Endometrial Cancer: Results from a Large Cohort of Us Women," *International Journal of Cancer*, 2008, vol. 123: 1877-1882.

[533] McCullough, Marjorie L., Alpa V. Patel, Roshni Patel, Carmen Rodriguez, Heather Spencer Feigelson, Elisa V. Bandera, Ted Gansler, Michael J. Thun, and Eugenia E. Calle. "Body Mass and Endometrial Cancer Risk by Hormone Replacement Therapy and Cancer Subtype," *Cancer Epidemiology Biomarkers and Prevention*, 2008, vol. 17, no. 1: 73-79.

[534] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[535] Ibid., accessed. p. 301.

[536] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

body weight.[537] The WHO *Comparative Quantification of Health Risks* study found that in the America-A subregion (consisting of Canada, United States, and Cuba), 52% of endometrial cancer in females, aged ≥30 years, could be attributed to a BMI of ≥30.[538]

Pichon et al. note that in Europe obese women have a two to three times increased risk of endometrial cancer, when compared to women with normal weight, and that about 40% of endometrial cancer cases can be attributed to obesity.[539] They also note that one European study—the European Prospective Investigation into Cancer and Nutrition (EPIC) that included 223,088 women with a mean follow-up of 6.4 years—found that obese women (BMI ≥30) and severely obese women (BMI ≥40) had significantly increased risks of developing endometrial cancer (RRs 1.78 and 3.02, respectively) when compared with women of normal weight,, but overweight women (BMI 25–29.9) did not show an increased risk.[540]

In Norway, between 1963 and 2001, the height and weight of over one million women aged 20–74 years were measured. Tone Bjorge of the University of Bergen et al. used these data to examine the relation between body size and endometrial cancer in the 9,227 patients who subsequently developed the cancer.[541] The mean age at diagnosis was 64.5 years. Compared with women of normal weight, overweight and obese women had RRs of 1.36 and 2.51, respectively. For severely obese women with a BMI ≥40 the RR rose dramatically to 4.96.

McCullough et al. examined the association between BMI and endometrial cancer in postmenopausal U.S. women aged 50–74 years using data from the Cancer Prevention Study II.[542] Out of a final analytical cohort of 33,436 women, 318 developed endometrial cancer between the date of enrolment in the study in 1992/93 and the June 2003 follow-up 11 years later. McCullough et al. found BMI to be strongly related to endometrial cancer incidence. Women who were obese class I (BMI 30–34.9) when the study started had a risk almost 345% higher (RR 3.45) than women of normal weight, and women with a BMI of ≥35 had an almost 500% increased risk (RR 4.99). The authors also report that women who had gained 30.0+ kg during the study period had an increased risk of endometrial cancer compared to women whose weight remained stable during the time of the study, but the association was no longer significant when the data were adjusted for BMI at the start of the study.

In Canada, Meera Jain of the University of Toronto et al. studied the relationship between BMI, nutritional factors, and endometrial cancer in Toronto and three other regions of Ontario in

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[537] Kaaks, Rudolf, Annekatrin Lukanova, and Mindy S. Kurzer. "Obesity, Endogenous Hormones, and Endometrial Cancer Risk: A Synthetic Review," *Cancer Epidemiology, Biomarkers & Prevention*, 2002, vol. 11, no. 12: 1531-1543.

[538] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

[539] Pischon, Nothlings, and Boeing. "Obesity and Cancer."

[540] Ibid.

[541] Bjorge, Engeland, Tretliand, and Weiderpass. "Body Size in Relation to Cancer of the Uterine Corpus in 1 Million Norwegian Women."

[542] McCullough, Patel, Patel, Rodriguez, Feigelson, Bandera, Gansler, Thun, and Calle. "Body Mass and Endometrial Cancer Risk by Hormone Replacement Therapy and Cancer Subtype."

2000.[543] Patients aged 30–79 years were identified through the Ontario Cancer Registry and controls were chosen randomly. Results, which were based on 552 cases (out of a possible 1,113 patients who had endometrial cancer) and 562 controls, showed that excess body weight had a significant association with the risk of endometrial cancer. The risk of endometrial cancer increased 1.36 times per 10 kg increase in body weight, and was more than twice as high in overweight and obese women with a BMI ≥25 (RR 2.15) as in women with a BMI of <25. Increased risk was also associated with high intakes of total nutritional energy and animal fat, and reduced risk was associated with a high consumption of vegetables.

4.5.3.1 Endometrial cancer statistics in Alberta

Even though numerous studies like those cited above have shown a strong positive association between obesity and endometrial cancer, Katzmarzyk and Janssen did not include endometrial cancer in their 2004 study of 2001 obesity costs in Canada.[544] Pan et al. also did not include endometrial cancer in their study of the association between obesity and the risk of 19 types of cancer in Canada.[545]

However, Birmingham et al. did include endometrial cancer in their 1999 study of the 1997 costs of obesity in Canada, and used a RR of 2.19 for the association of endometrial cancer with obesity.[546] Luo et al., who mainly used RRs from Katzmarzyk and Janssen, included endometrial cancer in their study on the burden of adult obesity in Canada.[547] Although they did not present a range of RRs for endometrial cancer, Luo et al. used a RR of 2.52, which was taken from the U.K. *Tackling Obesity in England* study.[548]

According the Alberta Cancer Registry, there were 377 new cases of endometrial cancer in Alberta in 2005, which amounted to 3.0% of all new adult cancers.[549] There were no reported cases below the age of 30, four new cases in women aged 30–39, and 15 new cases in the 40–44 age group. The number then progressively rose until it peaked at 77 cases in the 55–59 age group, and then progressively declined in each older age group. Between ages 30–69, there were 156 new cases, and between the ages 60–90+, there were 221 new cases.

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[543] Jain, Meera G., Geoffrey R. Howe, and Thomas E. Rohan. "Nutritional Factors and Endometrial Cancer in Ontario, Canada," *Cancer Control*, 2000, vol. 7, no. 3: 288-296.

[544] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[545] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[546] Birmingham, Muller, Palepu, Spinelli, and Anis. "The Cost of Obesity in Canada."

[547] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[548] National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, accessed.

[549] Public Health Agency of Canada (PHAC). *Cancer Surveillance on-Line*, 2008; accessed November 2008; available from http://dsol-smed.phac-aspc.gc.ca/dsol-smed/cancer/index_e.html.

As of January 1, 2005, there were 4,006 women living with endometrial cancer in Alberta.[550] Therefore, in 2005, when the incidence of the 377 new cases is added, it can be estimated that 4,383 Albertan women were living with endometrial cancer for all or part of 2005. This 4,383 estimate includes those women who died from endometrial cancer during the year. These data, however, were not used in the cost estimates.

The PAF used for endometrial cancer in this report was taken from Luo et al., who estimated it using obesity prevalence data from 2004 CCHS cycle 2.2.[551]

4.5.4 Kidney cancer

WCRF/AICR reports that kidney cancer is the 15th most common type of cancer worldwide, and accounts for about 2% of new cancer cases.[552] Incidence rates in high-income countries, which have been increasing, are almost five times higher overall than in low- and middle-income countries. Risk increases with age and most cases occur in adults between the ages of 60 and 80 years.

Kidney cancer is the 16th most common cause of cancer death, and accounts for about 1% of all cancer deaths. The 5-year survival rate is about 95% for early stage kidney cancers, which account for more than half of the cases, and about 20% for advanced stage cancers. Overall, in high-income countries the 5-year survival rate after diagnosis is about 50%.[553]

Pischon et al. report that in Europe, kidney cancer accounted for 3.1% of total cancer incidence in men and 2.3% of total cancer incidence in women in 2006, as well as 2.5% of male cancer deaths and 2.0% of female cancer deaths.[554] Because between 25–60% of patients do not have symptoms, approximately 25–30% of patients have metastatic disease by the time they are diagnosed, which lowers the overall survival rate for this cancer type.

According to Pichon et al., renal cell carcinoma is the major type of kidney cancer, accounting for about 80–90% of kidney cancers.[555] Bergstrom et al. note that it is often difficult to disentangle renal cell cancer from general kidney cancer in epidemiological studies because both are often examined together as one disease.[556]

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[550] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.
[551] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."
[552] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.
[553] Ibid., accessed.
[554] Pischon, Nothlings, and Boeing. "Obesity and Cancer."
[555] Ibid.
[556] Bergstrom, Hsieh, Lindblad, Lu, Cook, and Wolk. "Obesity and Renal Cell Cancer - a Quantitative Review."

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WCRF/AICR reports that there is convincing evidence that "greater body fatness is a cause of kidney cancer."[557] Y. Wang of Johns Hopkins University et al. note that the current understanding of the biological mechanisms by which obesity may help induce kidney cancer is limited:

> Obesity may promote kidney damage directly through hemodynamic and hormonal effects or indirectly by favoring the development of diabetes and hypertension. … [H]ypothesized reasons for the association between obesity and RCC [renal cell carconoma] include increased levels of estrogens and insulin associated with obesity, a higher concentration of growth factors in the excess adipose tissue, an abnormality in the metabolism of cholesterol, as well as alterations in the immune system.[558]

U.S. researchers Kristin Nicodemus et al. remark that almost all of the studies that have examined associations between obesity and kidney cancer have found an excess risk associated with high BMI, with the odds ratios ranging from 1.4 to 5.2.[559] In their study involving 34,651 U.S. postmenopausal women aged 55–69 years, the RR of kidney cancer among obese women was 2.49 compared with women of normal weight. The RR for severely obese women (BMI ≥40) was 4.75. The WCRF/AICR meta-analysis showed a 31% (unadjusted) increased risk of kidney cancer per 5 kg/m^2 for cohort studies, and a 42% (unadjusted) increased risk per 5 kg/m^2 for case-control studies.

Pischon et al. note that many studies have found that women are at greater risk for kidney cancer than men.[560] For example, the European Prospective Investigation into Cancer and Nutrition Study (EPIC) found that obese women had a RR of 1.68 for kidney cancer and obese men had a RR of 1.06 compared to those of normal weight.[561] However, other studies have found that men and women have similar risks. For example, in an earlier meta-analysis, A. Bergstrom of Karolinska Institutet in Sweden et al. found that 27% of renal cell cancer cases among men and 29% among women could be related to overweight and obesity together (BMI >25).[562] Wang et al. note that further studies are needed to clarify the gender differences in incidence of kidney cancer, since they seem to vary by study population.[563]

Bergstrom et al. identified all studies examining the association between kidney cancer and body weight published between 1966 and 1998.[564] Their quantitative summary pooled all risk ratios and transformed the BMI category measures into continuous exposure variables based on per

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[557] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[558] Wang, Y., X. Chen, Y. Song, B. Caballeroand, and L.J. Cheskin. "Association between Obesity and Kidney Disease: A Systematic Review and Meta-Analysis," *Kidney International*, 2008, vol. 73: 19-33. p. 27.

[559] Nicodemus, Kristin K., Carol Sweeney, and Aaron R. Folsom. "Evaluation of Dietary, Medical and Lifestyle Risk Factors for Incident Kidney Cancer in Postmenopausal Women," *International Journal of Cancer*, 2004, vol. 108: 115-121.

[560] Pischon, Nothlings, and Boeing. "Obesity and Cancer."

[561] Ibid.

[562] Bergstrom, Hsieh, Lindblad, Lu, Cook, and Wolk. "Obesity and Renal Cell Cancer - a Quantitative Review."

[563] Wang, Chen, Song, Caballeroand, and Cheskin. "Association between Obesity and Kidney Disease: A Systematic Review and Meta-Analysis."

[564] Bergstrom, Hsieh, Lindblad, Lu, Cook, and Wolk. "Obesity and Renal Cell Cancer - a Quantitative Review."

unit (1 kg/m^2) increases in BMI.[565, 566] The results showed that the summary RR was 1.07 per unit of increase in BMI for total risk and for both men and women, which they remark corresponds to a 3 kg body weight increase for a subject of average height. According to Bergstrom et al., a unit increase in BMI of 1.07 per 1 kg/m^2 is the equivalent of a RR of roughly 1.35 for overweight adults, 1.70 for adults with class 1 obesity, 2.05 for adults with class 2 obesity, and 2.40 for adults with class 3 obesity, compared to adults with normal weight.[567]

Wang et al. recently assessed the epidemiological evidence relating obesity and kidney disease.[568] They identified 16 studies published between 1980 and 2006, which were mainly from the U.S., had numbers of respondents ranging from 2,585 to 2,001,719, and a mean follow-up period of 15 years—with follow-up periods actually ranging from 3 to 35 years. In the U.S., they found that 18.6% of all kidney cancers, including 14.2% of male cases and 22.4% of cases in women, were attributable to obesity. In industrialized countries in general, they found that 10.5% of all kidney cancers, including 6.4% in cases in men and 14.9% of cases in women, were attributable to obesity.

Pischon et al. examined the association between BMI and renal cell cancer risk using data from the EPIC study referenced above.[569] Among 348,550 European adults from 8 countries, who were free of cancer at the start of the study and followed up for 6 years, 287 cases of renal cell cancer were identified. During the study, the height and weight of respondents were directly measured.

Women in the fifth weight quintile category (BMI ≥29.1) were found to have an increased renal cell cancer risk of (unadjusted) RR 2.26 compared with women in the first weight quintile category (BMI <21.8). Men in the fifth weight quintile category (BMI ≥29.4) had an increased renal cell cancer risk of (unadjusted) RR 1.12 compared with men in the first weight quintile (BMI <23.6). Pischon et al. also assessed the relative risk of renal cell cancer in extreme categories of BMI by comparing adults in the highest 10% of BMI in the study sample (BMI ≥31.2 in men and ≥31.8 in women) with those in the lowest 25% of BMI (BMI <24.1 in men and <22.4 in women). They found the adjusted RR to be 1.08 for men and 2.63 for women. Although these results showed significantly higher obesity-related risks for renal cell cancer among European women than men, the authors did not find significant differences between countries.[570]

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[565] Translating BMI categories and corresponding RRs from epidemiological studies into continuous variable estimates of per unit increases in BMI involves an advanced statistical calculation using a mixed effects weighted regression model. A more detailed explanation of this methodology can be found on page 985 of the Bergstrom et al. article.

[566] Bergstrom et al. noted that to produce the summary measure they "used a mixed effects weighted regression model to combine estimates from BMI categories from the individual studies." p. 985.

[567] Bergstrom, Hsieh, Lindblad, Lu, Cook, and Wolk. "Obesity and Renal Cell Cancer - a Quantitative Review."

[568] Wang, Chen, Song, Caballeroand, and Cheskin. "Association between Obesity and Kidney Disease: A Systematic Review and Meta-Analysis."

[569] Pischon, Nothlings, and Boeing. "Obesity and Cancer."

[570] The countries included in the study were Sweden, Denmark, United Kingdom, Netherlands, Germany, Italy, Spain, and Greece.

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4.5.4.1 Kidney cancer statistics in Alberta

In 2003, Jinfu Hu and members of the Canadian Cancer Registries Epidemiology Research Group used data from the National Enhanced Cancer Surveillance System (NECSS) to assess the role of BMI in the risk of renal cell carcinoma in Canada.[571] Between 1994 and 1997, NECSS identified from cancer registries a large population-based sample with 18 types of cancer in 8 provinces including Alberta. These respondents were surveyed along with over 5,000 population controls. A total of 2,199 incident cases of kidney cancer—995 females and 1,204 males—were identified, and 1,279 of these—691 male and 588 female—were included in the Hu et al. study. For weight, the survey collected information on how much the adult weighed "about two years ago" and the most the person had ever weighed, which avoided measuring current weight that might be affected by weight loss that had occurred due to the disease.

Hu et al. found that a high BMI, especially in obese class 3 (BMI ≥40), was strongly associated with an increased risk of developing renal cell cancer in both men and women. When compared with normal weight adults, the odds ratios for men were: overweight–OR 2.2, obese class 1–OR 2.8, obese class 2–OR 1.9, and obese class 3–OR 3.7. For women, the odds ratios were: overweight–OR 1.5, obese class 1–OR 2.5, obese class 2–OR 2.7, and obese class 3–OR 3.8. Why men in obese class 2 had lower ORs than men in the other BMI classes was not addressed in the study, and appears anomalous given the otherwise clear correspondence between increased BMI and increased risk of renal cell cancer in both men and women.

As previously noted, in 2004, Pan et al. estimated that 20.70% of kidney cancer—25.59% among men and 16.58% among women—could be attributed to obesity in Canada in 1997.[572] They estimated the total OR (BMI ≥30) for kidney cancer to be 2.74—OR 3.15 for obese men and OR 2.42 for obese women. Risks for overweight men were OR 2.03, and for overweight women were OR 1.49. It is unusual to find higher kidney cancer risk ratios for men than for women, as were found by Pan et al. in Canada. However, since the data used to estimate these odds ratios come from provincial cancer registries, they are likely to be more accurate than data that come from self-reports.

In 2006, Pan et al. repeated the 2004 study for kidney cancer using the same 1994–1997 data from the National Enhanced Cancer Surveillance System (NECSS).[573] However, in the new study they divided kidney cancer risks into renal cell cancer—which they estimated is responsible for more than 80% of kidney cancers—and renal pelvis cancer, which they note is responsible for the remainder of kidney cancers. In this study, risks for renal cell kidney cancer in obese men and women were almost identical (OR 2.57 for men and OR 2.56 for women), but risks for non-renal cell cancer were higher for obese men (OR 3.22) than for obese women (OR 2.23). Risks for renal cell cancer in overweight men (OR 2.05) were higher than risks for

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[571] Hu, Jinfu, Yang Mao, Kathy White, and The Canadian Cancer Registries Epidemiology Research Group. "Overweight and Obesity in Adults and Risk of Renal Cell Carcinoma in Canada," *Sozial- und Präventivmedizin / Social and Preventive Medicine*, 2003, vol. 48: 178-185.

[572] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[573] Pan, Sai Yi, Marie DesMeules, Howard Morrison, Shi Wu Wen, and the Canadian Cancer Registries Epidemiology Research Group. "Obesity, High Energy Intake, Lack of Physical Activity, and the Risk of Kidney Cancer," *Cancer Epidemiology Biomarkers & Prevention*, 2006, vol. 15, no. 12: 2453-2460.

overweight women (OR 1.68), but for renal pelvis cancer were lower for overweight men (OR 1.71) than for overweight women (OR 1.81).

Neither Katzmarzyk and Janssen nor Luo et al. included kidney cancer in their studies on obesity in Canada.[574, 575] However, in the cost of obesity study in Australia, Diabetes Australia used a RR of 1.50 for obese Australian women and 1.00 (no risk) for obese men and a PAF for both genders of 13%.[576, 577]

According to the Alberta Cancer Registry, there were 325 new adult cases of renal cell kidney cancer in Alberta in 2005, and 2 new cases among children—for a total of 219 cases among males, including one child, and 108 cases among females, including one child.[578] The adult cases accounted for 2.6% of the total new adult cancer cases in Alberta. Male kidney cancer accounted for 1.7% of all new adult cancer cases and female kidney cancer accounted for 0.8%. One girl in the 10–14 year age group and one boy in the 0–4 age group developed kidney cancer in 2005. Among both men and women aged ≥15 in Alberta, there were 10 new cases of renal cell kidney cancer occurring in individuals below the age of 40 years, after which the incidence rose progressively, peaked between the ages of 70–74 at 49 cases, and then began to decline progressively probably due to lower population numbers in the higher age groups.

As of January 1, 2005, there were 2,250 people living with kidney cancer in Alberta.[579] Therefore, in 2005, when the incidence of the 325 new cases is added, it can be estimated that 2,575 Albertan adults were living with kidney cancer for all or part of 2005. This 2,575 estimate includes those Albertans who died from kidney cancer during the year. These data, however, were not used in the cost estimates.

The PAFs for kidney cancer used in this report were found in the report by Pan et al. who estimated that 25.59% of kidney cancer among males and 16.58% among females could be attributed to obesity.[580]

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[574] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[575] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[576] National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, accessed.

[577] Access Economics. *The Economic Costs of Obesity*, accessed.

[578] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

[579] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

[580] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

4.5.5 Esophageal cancer (oesophageal adenocarcinoma)

WCRF/AICR reports that cancer of the esophagus, which is also known as oesophageal adenocarcinoma, is the 8th most common type of cancer, accounts for 4% of all cancer incidence, and is twice as common in men as in women.[581] It is the 6th most common cause of death and is usually fatal. Very few cases are diagnosed in people under the age of 40, and, in the U.S., rates are higher among African-Americans than among Caucasians.[582]

The incidence of esophageal cancer is increasing in high-income countries, although it is mainly found in low-income countries.[583] In the past three decades the incidence of esophageal cancer has increased by more than 400% in the U.S., which is the fastest rate of increase of any cancer in that country.[584]

Pischon et al. report that in Europe in 2006, 2.0% of total cancer incidence in men and 0.7% in women, as well as 3.1% of cancer deaths in men and 1.2% in women were caused by esophageal cancer.[585] Jesper Lagergren, of the Unit for Oesophageal and Gastric Research, Karolinska University Hospital in Sweden, notes that oesophageal adenocarcinoma is seven times more prevalent in men than in women in almost all developed countries, and that the incidence in U.S. men has increased by about 10% a year since the 1970s.[586]

According to Lagergren, obesity is an established risk factor for oesophageal adenocarcinoma, and apart from gastro-oesophageal reflux, old age, and male sex, it is the only well-established risk factor.[587] He observes: "An increase in body mass has been shown to be positively and probably causally associated with risk of developing oesophageal adenocarcinoma."[588] According to WCRF/AICR, of two types of esophageal cancer, only adenocarcinoma is caused, in part, by excess weight. The WCRF/AICR meta-analysis of eight case-control studies of esophageal cancer showed a summary effect of RR 1.11 increased risk per 1 kg/m^2, which translates to a 55% increased risk for each 5 kg/m^2.[589]

Lagergren explains that, although several biological mechanisms for the association between excess weight and esophageal cancer have been proposed, none has been established:

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[581] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.
[582] Ibid., accessed.
[583] The second type of esophageal cancer is squamous cell carcinoma.
[584] Kubo, Ai, and Douglas A. Corley. "Body Mass Index and Adenocarcinomas of the Esophagus or Gastric Cardia: A Systematic Review and Meta-Analysis," *Cancer Epidemiology Biomarkers & Prevention*, 2006, vol. 15, no. 5: 872-878.
[585] Pischon, Nothlings, and Boeing. "Obesity and Cancer."
[586] Lagergren, Jesper. "Controversies Surrounding Body Mass, Reflux, and Risk of Oesophageal Adenocarcinoma," *The Lancet*, 2006, vol. 7, no. April: 347-349.
[587] Ibid.
[588] Ibid. p. 348.
[589] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

> Some data suggest that the combination of reflux and increased body mass has a multiplicative effect in the development of oesophageal adenocarcinoma, adding further evidence that being overweight is an independent risk factor. Several biological mechanisms have been postulated, but no convincing evidence has been reported for this association…. Although reflux and obesity are risk factors for oesophageal adenocarcinoma, the interplay between these two factors and other factors with regard to oesophageal adenocarcinoma is uncertain and several inconsistencies remain. The contribution of these risk factors to the increasing incidence of oesophageal adenocarcinoma remains unclear, and none of these factors explains the strong predominance of oesophageal adenocarcinoma in men.[590]

Ai Kubo of Columbia University and Douglas Corley of the University of California, San Francisco, recently conducted a systematic review and meta-analysis of studies relating BMI and esophageal cancer.[591] After searching for articles published between 1966 and July 2005, they found 14 studies that met their criteria. They defined body mass categories somewhat differently than the currently accepted categories because these groupings represented the divisions most frequently reported in the articles they found. However, the different categories make it somewhat difficult to compare the findings of the Kubo and Corley report with others. The categories used include: normal–BMI 18.5–24.9, overweight–BMI 25–28, and obese–BMI ≥28.

Kubo and Corley did not find large gender differences in the risk of esophageal cancer. Their pooled results showed ORs for overweight men to be 1.8, for obese men to be 2.4, for overweight women to be 1.5, and for obese women to be 2.1, compared with adults with normal weight.[592] However, for various methodological reasons, all studies were not included in these results. For example, one study by Lagergren et al. was so dominant that it influenced the initial results, and its exclusion provided more homogeneous results for both genders.[593] Lagergren et al. found a much stronger association between increased BMI and esophageal cancer—OR 7.6—than was found in the other studies. Kubo and Corley note that the Lagergren et al. study used different outcome measures than did the other studies.

One of the largest studies of the association between BMI and esophageal cancer was conducted in Norway, and the results were close to those of Kubo and Corley.[594] Norwegian researchers Ander Engeland et al. used data from multiple linked files that was made possible by the identification numbers assigned to all individuals living in Norway after 1960. The databases included surveys conducted between 1963 and 2001 that directly measured the height and weight

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[590] Lagergren. "Controversies Surrounding Body Mass, Reflux, and Risk of Oesophageal Adenocarcinoma." pp. 348, 349.
[591] Kubo, and Corley. "Body Mass Index and Adenocarcinomas of the Esophagus or Gastric Cardia: A Systematic Review and Meta-Analysis."
[592] Ibid.
[593] Lagergren, J., R. Bergstrom, and O. Nyren. "Association between Body Mass and Adenocarcinoma of the Esophagus and Gastric Cardia," *Annals of Internal Medicine*, 1999, vol. 130: 883-890. Cited in Kubo, and Corley. "Body Mass Index and Adenocarcinomas of the Esophagus or Gastric Cardia: A Systematic Review and Meta-Analysis."
[594] Engeland, Anders, Steinar Tretli, and Tone Bjorge. "Height and Body Mass Index in Relation to Esophageal Cancer; 23-Year Follow-up of Two Million Norwegian Men and Women," *Cancer Causes and Control*, 2004, vol. 15, no. 8: 837-843.

of more than two million Norwegians, who were then followed up for an average of 23 years after measurement until 2001, as well as the Death Registry at Statistics Norway, and the Cancer Registry of Norway.

The Engeland et al. study included 2,001,719 persons (963,709 males and 1,038,010 females). After accounting for deaths and emigration, only about 80 people out of the two million study sample were lost in the follow-up. During the follow-up period, 2,245 verified esophageal cancer cases were diagnosed and registered—1,597 among men and 648 among women. By the end of the 23-year average follow-up period, 62% of the more than two million Norwegians in the original study sample were alive and without a diagnosis of esophageal cancer, 38% were deceased, and 0.1% had a diagnosis of esophageal cancer.

Engeland et al. found that only 28% of the cases of esophageal cancer in men (447 cases in all) and 20% in women (130 cases in all) were adenocarcinomas. They also found that the mean age at diagnosis for adenocarcinoma was 70 years for men and 76 years for women and they observed only 14 male cases and 4 female cases of oesophageal adenocarcinoma below the age of 50 years. [595]

In order to estimate relative risks, Engeland et al. only included persons who were measured for height and weight when they were aged 20–74 years. The study used the commonly accepted international BMI categories, which were compared with adults with normal weight. RRs for oesophageal adenocarcinoma for overweight (BMI 25–29.9) and obese (BMI ≥30) men were 1.80 and 2.58, respectively. For overweight and obese women, the RRs were 1.64 and 2.06, respectively. When reported using BMI as a continuous variable, the RR of oesophageal adenocarcinoma for one unit increase in BMI was 1.12, which was comparable to the WCRF/AICR reported rate of 1.11 per unit increase in BMI.

4.5.5.1 Esophageal cancer statistics in Alberta

Neither Katzmarzyk and Janssen, nor Luo et al., nor Pan et al. included esophageal cancer in their studies on obesity and disease in Canada.[596, 597, 598]

According to the Alberta Cancer Registry, there were 109 new cases of esophageal cancer in Alberta in 2005—89 among men and 20 among women.[599] Overall, these cases accounted for 0.9% of all new cancer cases in 2005. Male esophageal cancer accounted for 0.7% of all new cancer cases, and female esophageal cancer accounted for 0.2%.

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[595] Ibid.
[596] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."
[597] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."
[598] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."
[599] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

Seven of the cases were diagnosed in individuals below the age of 50 (and none among children aged 0–14 years), after which the incidence began to rise in older age groups, peaked at ages 75–79 years with 27 new cases, and then declined in the remaining older age groups. This pattern was dictated by male cases. In females, out of the 20 new cases, there were only four cases below the age of 65. Between the ages of 65–69 there were 5 new cases, and in the older age groups there were 3 new cases in the 75–79 age group, and 2 new cases each among the 70-74, 80-84, 85-89, and 90+ groups.

As of January 1, 2005, there were 182 people living with esophageal cancer in Alberta.[600] Therefore, in 2005, when the incidence of the 109 new cases is added, it can be estimated that 291 Albertans were living with esophageal cancer for all or part of 2005. This 291 estimate includes those Albertans who died from esophageal cancer during the year. These data, however, were not used in the cost estimates.

The PAFs used in this report for esophageal cancer were based on the RRs in the Engeland et al. report as noted above and obesity prevalence in Alberta from 2004 CCHS cycle 2.2 data.[601]

4.5.6 Ovarian cancer

According to WCRF/AICR, ovarian cancer is the 7th most common cancer in women globally, and the 16th most common cancer overall, accounting for 4% of new cancer cases in women and 2% overall.[602] It is also the 7th most common cause of cancer death in women globally and the 15th most common cause of cancer death overall, and accounts for about 4% of cancer deaths in women. The five-year survival rate is between 30–50 percent.

Ovarian cancer is almost three times higher in high-income countries than in middle- to low-income countries, and in Europe and North America the incidence rates are more than 10 cases per 100,000 women. In the U.S., rates are higher among white women than among women in other ethnic groups. The risk increases with age, and is generally highest in postmenopausal women, with only 10–15 percent of cases occurring in premenopausal women.[603] There are different subtypes of ovarian cancer, but most studies combine these subtypes, although they may have independent risk factors.[604]

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[600] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.
[601] Engeland, Anders, Steinar Tretli, and Tone Bjorge. "Height and Body Mass Index in Relation to Esophageal Cancer; 23-Year Follow-up of Two Million Norwegian Men and Women,"
[602] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.
[603] Ibid., accessed.
[604] Olsen, Catherine M., Christina M. Nagle, David C. Whiteman, David M. Purdie, Adele C. Green, Penelope M. Webb, and Australian Cancer Study (Ovarian Cancer) and Australian Ovarian Cancer Study Group. "Body Size and Risk of Epithelial Ovarian and Related Cancers: A Population-Based Case-Control Study," *International Journal of Cancer*, 2008, vol. 123: 450-456.

The biological mechanisms that lead to ovarian cancer are not well known, but because ovarian cancer is hormone related, the possible mechanisms have been compared with those for breast and endometrial cancer. WCRF/AICR notes: "Lifetime exposure to oestrogen—increased by early menarche, late menopause, not bearing children, and late (over 30) first pregnancy—raises the risk of, and may be seen as a cause of, breast, ovarian, and endometrial cancers in women."[605] Olsen et al. offer similar explanations that indicate potential links with obesity:

> Adiposity influences the synthesis and bioavailability of endogenous sex steroids (oestrogens, androgens and progesterone). Endogenous hormones are believed to be involved in the aetiology of ovarian cancer, and obesity is a well-established risk factor for two other hormone-related cancers in women, postmenopausal breast cancer and endometrial cancer. …. Other plausible hormonal candidates include insulin and androgen. High BMI has been associated with increased serum testosterone concentrations among women, especially postmenopausal women. High androgen and high insulin levels characterise the condition of polycystic ovary syndrome (PCOS), which has been found to be a risk factor for ovarian cancer in one study. Obesity is associated with increased insulin levels, which lead to increases in the insulin-like growth factor-1 (IGF-I), and high levels of IGF-I have been associated with other hormone-dependent cancers: breast and prostate.[606]

WCRF/AICR reports that there is an increased risk of ovarian cancer with greater adult attained height (8% increased risk per 5 cm of height), which may be connected with hormonal or other factors that promote linear growth in childhood.[607] However, for other exposures, including "body fatness," WCRF/AICR notes that "the data were either of too low quality, too inconsistent, or the number of studies too few to allow conclusions to be reached."[608] However, the two systematic reviews and meta-analyses described below have found modest but statistically significant associations.

The first such review was conducted in 2001 by Australian researchers David Purdie et al. as part of a large case-control study of 775 ovarian cancer cases and 846 controls in Australia.[609] Purdie et al. included studies of associations between ovarian cancer and obesity published between 1966 and 2000. They concluded that there was a small or moderate positive relation between obesity and the risk of ovarian cancer. Out of 34 identified studies, 29 studies fit their review criteria, which included sufficient information to enable a calculation of relative risk for obesity. The population-based studies showed a summary 40% increased risk (RR 1.4) of ovarian cancer in obese adults, with RRs ranging from 1.1–2.0.

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[605] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed. p. 39.

[606] Olsen, Catherine M., Adele C. Green, David C. Whiteman, Shahram Sadeghi, Fariba Kolahdooz, and Penelope M. Webb. "Obesity and the Risk of Epithelial Ovarian Cancer: A Systematic Review and Meta-Analysis," *European Journal of Cancer*, 2007, vol. 43: 690-709. p. 698.

[607] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[608] Ibid., accessed. p. 298.

[609] Purdie, David M., Christopher J. Bain, Penelope M. Webb, David C. Whiteman, Sandi Pirozzo, and Adele C. Green. "Body Size and Ovarian Cancer: Case-Control Study and Systematic Review (Australia)," *Cancer Causes and Control*, 2001, vol. 12: 855-863.

Purdie et al. also used data from the Australian case-control study of ovarian cancer to examine the association between ovarian cancer and BMI.[610] Results of the case-control study showed unadjusted ORs for the risk of ovarian cancer to be 1.4 for overweight women and 1.9 for obese women—meaning a 40% and 90% increased risk, respectively. The adjusted ORs were almost the same—1.5 for overweight women and 1.9 for obese women.[611] When BMI was treated as a continuous variable in a logistic model, an overall increased risk of 3% per unit increase in BMI was estimated. The authors also separately estimated the risk of various subtypes of ovarian cancer by BMI category and found very small differences.

In 2007, the second systematic review and meta-analysis was conducted by Australian researchers Catherine Olsen et al.[612] It updates the Purdie et al. studies described above, and includes all population-based studies that assessed the relation between BMI and ovarian cancer that were published up to April 2006. The analysis found a modest but statistically significant risk for ovarian cancer among obese adults, and "consistent epidemiological evidence that the risk of ovarian cancer increases with increasing BMI."[613] Olsen et al. note that out of 28 identified studies, 24 reported a positive association between obesity and ovarian cancer. They concluded: "Ovarian cancer should be added to the list of cancers likely to be related to obesity."[614]

Results of the Olsen et al. meta-analysis showed a summary effect estimate of RR 1.16 for overweight women and RR 1.30 for obese women, when compared with women of normal weight. When a sensitivity analysis revealed that one cohort study had significantly affected the summary statistics, the authors recalculated the estimate without that particular study and found the risk for obese women to be 1.35, but they did not include overweight women in those recalculated results. Olsen et al. note that case-control studies showed higher relative risk ratios than cohort studies for both overweight (RR 1.19 versus 1.07, respectively) and obese (RR 1.49 versus 1.12, respectively) women, with the difference particularly marked for obesity.

Olsen et al. also estimated the magnitude of risk in young women, aged 17–20 years, to be 1.22 for overweight and obese young women combined. However, out of the nine studies that included young women and that were included in the summary analysis, only two found a significantly increased obesity-related risk of ovarian cancer in this young age group.

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[610] Ibid.

[611] ORs were adjusted for age in years, age squared, geographic location, education, duration of oral contraceptive use, smoking history, ever use of talc in the perineal region, tubal sterilization, hysterectomy, and history of breast or ovarian cancer in a first-degree relative.

[612] Olsen, Green, Whiteman, Sadeghi, Kolahdooz, and Webb. "Obesity and the Risk of Epithelial Ovarian Cancer: A Systematic Review and Meta-Analysis."

[613] Ibid. p. 697.

[614] Ibid. p. 690.

4.5.6.1 Ovarian cancer statistics in Alberta

Neither Katzmarzyk and Janssen nor Luo et al. included ovarian cancer in their studies on obesity and chronic disease in Canada.[615, 616] However, Pan et al. did include ovarian cancer in their study of obesity and cancer risk in Canada in 2001.[617] They found that 11.74% of ovarian cancer in Canada could be attributed to obesity, and 3.85% could be attributed to overweight. Their adjusted OR for obese women, compared with women of normal weight, was 1.95, and for overweight women it was 1.16. This RR for obese women in Canada is considerably higher than the summary RR of 1.30 estimated by Olsen et al. and reported above, but is closer to the RR of 1.70 used by the National Audit Office in the U.K. for ovarian cancer.[618]

Olsen et al. used the Pan et al. study in their meta-analysis and remarked that it showed a stronger association than found in other studies, but they were unsure of the reason:

> We cannot explain why this study showed a stronger association as it did not differ significantly from the other case–control studies included in the meta-analysis in terms of the population profile, study design or implementation.[619]

However, analysis indicates that risk ratios are often country-specific and may be conditioned by the prevailing socio-demographic characteristics in particular cultures and regions.

According to the Alberta Cancer Registry, there were 185 new cases of adult ovarian cancer in Alberta in 2005, and one new case in a child in the 10–14 year age group.[620] Ovarian cancer accounted for 1.5% of all new cancer cases in Alberta. Eleven of the new cases occurred in women under the age of 40. After the age of 40, the rates began to rise, but not progressively, with the highest number of cases (29) in the 50–54 age group.

As of January 1, 2005, there were 1,624 women living with ovarian cancer in Alberta.[621] Therefore, in 2005, when the incidence of the 185 new cases is added, it can be estimated that 1,809 Albertan women were living with ovarian cancer for all or part of 2005. This 1,809 estimate includes those Albertan women who died from ovarian cancer during the year. These data, however, were not used in the cost estimates.

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[615] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[616] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[617] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[618] National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, accessed.

[619] Olsen, Green, Whiteman, Sadeghi, Kolahdooz, and Webb. "Obesity and the Risk of Epithelial Ovarian Cancer: A Systematic Review and Meta-Analysis." p. 695.

[620] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

[621] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

This study used PAFs for ovarian cancer as estimated by Pan et al., who as noted above found that 11.74% of ovarian cancer in Canada could be attributed to obesity, and 3.85% could be attributed to overweight.[622]

4.5.7 Prostate cancer

WCRF/AICR reports that, globally, prostate cancer is the 2nd most common cancer in men and the 6th most common cancer overall.[623] New cases account for 6% of all cancer cases, and 12% of cancer cases in men. The risk rises with age and is highest after 40 years. Prostate cancer is the 6th leading cause of death in men, and is responsible for 6% of male cancer deaths and 3% of all cancer deaths, although the 5-year survival rate in high-income countries is high at 76%. Pischon et al. report that in Europe prostate cancer is the most common cancer diagnosed in men, and in 2006 was responsible for 20.3% of all new male cancers and 9.2% of all male cancer deaths.[624]

Evidence that excess weight can cause prostate cancer is mixed. WCRF/AICR notes that evidence for an association between body fatness and prostate cancer is limited and inconclusive.[625] Pischon et al. report that, with few exceptions, the epidemiological studies that have examined the association between BMI and prostate cancer have not found significant associations except in advanced stages.[626] Although the reasons for this association between BMI and advanced prostate cancer are not clear, Pischon et al. note that one hypothesis is that detection and diagnosis are often difficult or delayed in obese men.[627]

In 2006, Robert MacInnis and Dallas English of the University of Melbourne found an increased risk associated with obesity mainly in advanced prostate cancer cases. They conducted a systematic review and meta-analysis of studies that examined the association between BMI and prostate cancer risk that were published between 1966 and October 2004.[628] They summarized reported risks from 56 studies—all except 9 of which were published between 1990 and 2004—and reported the results as a continuous variable (i.e. per 5 kg/m^2 increase in BMI.) The meta-analysis indicated that obesity is weakly associated with an increased risk of prostate cancer, with the RR being 1.05 per 5 kg/m^2 increase in BMI. When studies that reported prostate cancer by grade were analysed separately, the association between BMI and the risk of advanced prostate disease was positive (RR 1.12 per 5 kg/m^2 increase in BMI), and almost 17% higher

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[622] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[623] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[624] Pischon, Nothlings, and Boeing. "Obesity and Cancer."

[625] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[626] Pischon, Nothlings, and Boeing. "Obesity and Cancer."

[627] For a review of possible biological mechanisms involved in the pathology of prostate cancer, see Giovannucci, and Michaud. "The Role of Obesity and Related Metabolic Disturbances in Cancers of the Colon, Prostate, and Pancreas."

[628] MacInnis, Robert J., and Dallas R. English. "Body Size and Composition and Prostate Cancer Risk: Systematic Review and Meta-Regression Analysis," *Cancer Causes and Control*, 2006, vol. 17, no. 8: 989-1003.

than the risk of localized disease (RR 0.96 per 5 kg/m^2 increase in BMI), which showed a negative association between prostate cancer and BMI.

Carmen Rodriguez of the American Cancer Society et al. suggest several possible biological mechanisms that might potentially lead to the development of prostate cancer in obese men, though the identification of such pathways remains at the hypothetical level and still require proof:

> Some metabolic changes associated with obesity are consistent with the hypothesis that obesity differentially affects the development of nonaggressive and aggressive prostate cancer. … [T]estosterone contributes to the growth and progression of prostate cancer… Serum testosterone levels decrease with increasing obesity; thus, low testosterone levels may be associated with lower risk of nonaggressive prostate cancer and higher risk of less differentiated and more aggressive prostate cancer…. Other biological hypotheses that support the role of obesity in prostate cancer include alterations in insulin and bioavailable serum circulating insulin-like growth factor I (IGF-I) levels. Circulating insulin levels increase linearly with increasing obesity, and insulin has been implicated in prostate cancer biology, with higher risk of prostate cancer, and with higher recurrence of the disease. Circulating concentrations of total IGF-I have been associated with higher risk of prostate cancer.[629]

A recent groundbreaking Harvard-McGill study, led by Harvard researcher Jing Ma and published in the November 2008 issue of *Lancet Oncology*, has found convincing evidence that high insulin levels play a role in prostate cancer mortality.[630] The study used data from the long-term Physicians' Health Study of more than 22,000 doctors. During 24 years of follow-up, 2,546 men developed prostate cancer and 11% died from the disease. Compared with men of normal weight at the start of the study in 1982, overweight men were nearly 1.5 times (HR 1.47) more likely to die from prostate cancer, and obese men were 2.7 times more likely (HR 2.66) to die from the disease.[631] However, overweight and obese men who also had high C-peptide concentrations, which reflects insulin secretion, had a risk that was more than four times higher (HR 4.12) than the risk for men with normal weight and low C-peptide concentrations. Therefore, the study concludes: "Excess bodyweight and a high plasma concentration of C-peptide both predispose men with a subsequent diagnosis of prostate cancer to an increased likelihood of dying of their disease. Patients with both factors have the worst outcome."[632]

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[629] Rodriguez, Carmen, Stephen J. Freedland, Anusila Deka, Eric J. Jacobs, Marjorie L. McCullough, Alpa V. Patel, Michael J. Thun, and Eugenia E. Calle. "Body Mass Index, Weight Change, and Risk of Prostate Cancer in the Cancer Prevention Study II Nutrition Cohort," *Cancer Epidemiology Biomarkers & Prevention*, 2007, vol. 16, no. 1: 63-69.

[630] Ma, Jing, Haojie Li, Ed Giovannucci, Lorelei Mucci, Weiliang Qiu, Paul L. Nguyen, J. Michael Gaziano, Michael Pollak, and Meir J. Stampfer. "Prediagnostic Body-Mass Index, Plasma C-Peptide Concentration, and Prostate Cancer-Specific Mortality in Men with Prostate Cancer: A Long-Term Survival Analysis," *Lancet Oncology*, 2008, vol. 9, no. November: preview issue.

[631] HR = hazard ratio, which is an indication of relative risk, similar to RR.

[632] Ma, Li, Giovannucci, Mucci, Qiu, Nguyen, Gaziano, Pollak, and Stampfer. "Prediagnostic Body-Mass Index, Plasma C-Peptide Concentration, and Prostate Cancer-Specific Mortality in Men with Prostate Cancer: A Long-Term Survival Analysis." (preview issue, page 1).

Rodriguez et al. also report that the strongest associations between excess weight and prostate cancer have been found in metastatic or fatal prostate cancer.[633] They note that the early prospective studies of the relationship between BMI and the incidence of all prostate cancer, which had found a negative association, had examined prostate cancer incidence without considering the grade of the disease. In 2007, using data from the Cancer Prevention Study II for 69,991 men who began the study in 1982 and 1992 and were followed through June 2003, Rodriguez et al. documented 5,252 new cases of prostate cancer, which they classified at diagnosis into three grades: clinically non-metastatic low-grade prostate cancer, clinically non-metastatic high-grade prostate cancer, and metastatic or fatal prostate cancer. They then compared the BMIs that were self-reported in 1992 with the classified outcomes.

Rodriguez et al. found that compared with men with normal weights, obese men had an increased positive risk (RR 1.54) of developing metastatic or fatal prostate cancer, but a negative risk (RR 0.94) when all cases were considered without the grade classification.[634] When the other two grades were considered, obese men did not have a risk for non-metastic low-grade prostate cancer (RR 0.86) and in fact had a negative association with this grade of illness, but did have a positive risk for nonmetastic high-grade prostate cancer (RR 1.22).

Other studies have found similar results. In 2007, U.S. researchers Alyson Littman et al. reported results of a 2000–2004 prospective cohort study in Washington State, which included 34,754 men aged 50–76.[635] During the study 383 men developed aggressive prostate cancer and 437 developed non-aggressive prostate cancer. Compared with men of normal weight, men who were obese at age 30 years had a 20% increased risk (HR 1.2) of developing aggressive prostate cancer, but a substantially decreased risk (HR 0.62) of developing non-aggressive prostate cancer. For all ages, obese men had a 10% increased risk of aggressive prostate cancer, but again a substantially decreased risk (0.69) of the non-aggressive form of the disease.

It should be noted that these differential results for different grades and classifications of prostate cancer are consistent with the hypothesis referenced above that that detection and diagnosis of prostate cancer are often difficult or delayed in obese men. In that case, it stands to reason that associations with early, low-grade, and non-metastatic forms of the illness would be lower in obese men, since these are the forms most likely to be diagnosed at early stages, while associations with advanced, high-grade, and metastatic forms are higher since those are most likely to be revealed at later stages of diagnosis.

This raises the interesting question of whether it is possible to infer 'cause' in such a case. On the one hand, overall disease incidence may not vary significantly by level of BMI, and a particular physio-biological pathway between obesity and prostate cancer may not exist. On the other hand, if the hypothesis on difficulty of early diagnosis in obese men is true, then the presence of obesity would in fact be responsible for higher mortality rates due to prostate cancer.

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[633] Rodriguez, Freedland, Deka, Jacobs, McCullough, Patel, Thun, and Calle. "Body Mass Index, Weight Change, and Risk of Prostate Cancer in the Cancer Prevention Study II Nutrition Cohort."
[634] Ibid.
[635] Littman, Alyson J., Emily White, and Alan R. Kristal. "Anthropometrics and Prostate Cancer Risk," *American Journal of Epidemiology*, 2007, vol. 165, no. 11: 1271-1279.

4.5.7.1 Prostate cancer statistics in Alberta

Neither Katzmarzyk and Janssen nor Luo et al. included prostate cancer in their studies on obesity and chronic disease in Canada.[636, 637]

However, Pan et al. did include prostate cancer in their study of obesity and cancer risk in Canada in 2001.[638] Pan et al. attributed 6.02% of prostate cancer to overweight and 4.14% to obesity in men. The risk of prostate cancer was higher in obese men (OR 1.27) than in overweight men (OR 1.16).

According to the Alberta Cancer Registry, there were 1,905 new cases of prostate cancer in Alberta in 2005, accounting for 15% of all new cases of cancer.[639] Incidence rates per 100,000 Albertan men were low between the ages of 35–44 (0.9–8.2 per 100,000)—there were no cases among children aged 0–14 years—but the incidence rose to 36.7 per 100,000 in the 45–49 age group, and then began to rise dramatically in older age groups. The highest incidence rates were in the 70–74 age group (798.7 per 100,000), and in the 90+ age group (840.1 per 100,000).

Below the age of 50, there was only one new case among 35–39 year olds in Alberta in 2005, 11 new cases in 40–44 year olds, and 50 new cases in 45–49 year olds. After age 50, the number of cases rose to 367 in 65–69 year olds, and then began to decrease in the older age groups. The difference between the incidence results (per 100,000) by age group reported in the previous paragraph, and the number of cases by age group reported here is due to lower population numbers in the older age groups.[640]

As of January 1, 2005, there were 14,942 men living with prostate cancer in Alberta.[641] Therefore, in 2005, when the incidence of the 1,905 new cases is added, it can be estimated that 16,847 Albertan men were living with prostate cancer for all or part of 2005. This 16,847 estimate includes those Albertan men who died from prostate cancer during the year. These data, however, were not used in the cost estimates.

This study used PAFs for prostate cancer as estimated by Pan et al., who as noted above found that 6.02% of prostate cancer could be attributed to overweight and 4.14% to obesity.[642]

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[636] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[637] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[638] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[639] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

[640] Statistics Canada. *Annual Demographic Statistics 2005*, Catalogue no. 91-213-XIB, 2006; accessed November 2008; available from http://www.statcan.ca/english/freepub/91-213-XIB/0000591-213-XIB.pdf.

[641] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

[642] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

4.5.8 Pancreatic cancer

WCRF/AICR reports that cancer of the pancreas is the 13th most common type of cancer worldwide, is responsible for about 2% of cancers overall and 3% of cancer deaths, is almost always fatal and therefore has low survival rates, and is the 9th most common cause of cancer death.[643] The incidence of pancreatic cancer is highest in high-income countries and the risk increases with age—with most diagnoses made in adults aged 60–80 years. Berrington de Gonzalez et al. report that pancreatic cancer is the 6th most common cause of cancer death in European Union countries.[644]

Pischon et al. report that pancreatic cancer accounted for about 2.5% of cancer incidence in Europe in 2006.[645] They note that pancreatic cancer is difficult to study, particularly for its possible association with obesity, since it has a 5-year survival rate of only 5%, and it leads to substantial weight loss even before diagnosis. Therefore, the evidence from case-control studies is weak and potentially biased because of this high fatality rate and the necessity of relying on proxy interviews.

Despite these considerable challenges, there is a growing body of evidence showing associations between obesity and pancreatic cancer. According to WCRF/AICR, the "evidence that body fatness is a cause of cancer of the pancreas is convincing."[646] Larsson et al. note that the evidence indicates that abnormal glucose metabolism and insulin resistance may be a potential cause in the development of pancreatic cancer.[647] Edward Giovannucci of the Harvard School of Public Health and Dominique Michaud of Harvard Medical School find that long-standing diabetes (of five or more years) increases the risk of pancreatic cancer by about 50%.[648] As noted earlier, type 2 diabetes, which accounts for about 95% of all cases of diabetes, is strongly linked to obesity, with various studies attributing between 25% and 85% of the disease to overweight and obesity. This points to a potential indirect association between obesity and pancreatic cancer by virtue of their common link with diabetes as an intermediary factor.

In a 2007 review of the role of obesity in cancers of the colon, prostate, and pancreas, Giovannucci and Michaud report that the early studies, which showed no associations between excess weight and pancreatic cancer, had multiple flaws, including those resulting from high case fatalities and reports obtained by proxy from the next of kin. However, they point to more

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[643] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[644] Berrington de Gonzalez, Amy, Elizabeth A. Spencer, H. Bas Bueno-de-Mesquita, and 40 researchers from the EPIC study. "Anthropometry, Physical Activity, and the Risk of Pancreatic Cancer in the European Prospective Investigation into Cancer and Nutrition [EPIC]," *Cancer Epidemiology Biomarkers & Prevention*, 2006, vol. 15, no. 5: 879-885.

[645] Pischon, Nothlings, and Boeing. "Obesity and Cancer."

[646] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed. p. 271.

[647] Larsson, Susanna C., Nicola Orsini, and Alicja Wolk. "Body Mass Index and Pancreatic Cancer Risk: A Meta-Analysis of Prospective Studies," *International Journal of Cancer*, 2007, vol. 120: 1993-1998.

[648] Giovannucci, and Michaud. "The Role of Obesity and Related Metabolic Disturbances in Cancers of the Colon, Prostate, and Pancreas."

recent and reliable evidence that shows positive associations with obesity, with RRs for pancreatic cancer ranging between 1.2 and 3.0.

Giovannucci and Michaud note that the biological mechanisms for the BMI–pancreatic cancer association "remain speculative and deserve further study"[649]:

> The role of insulin appears to be indirect, through stimulation of islet cell proliferation and turnover. These mechanisms have been examined in hamster models, in which insulin resistance appears to play a key role; in these models, inhibition of insulin resistance prevents tumor development…. Further studies need to consider carefully the independent, joint, and interactive roles of the various hormones that are influenced by obesity.[650]

The WCRF/AICR meta-analysis of 17 cohort studies found that studies of the relationship between BMI and cancer of the pancreas yielded a summary relative risk of 1.14 per 5 kg/m^2 of increased BMI—indicating a 14% increased risk per 5 kg/m^2 (i.e. for every five additional units of BMI).[651] However, other studies have shown smaller risks.

In a 2006 study, Amy Berrington de Gonzalez of the University of Oxford and 42 other European researchers found that tobacco smoking is the only established cause of pancreatic cancer, though diabetes has been hypothesized as a probable cause, which in turn has led researchers to speculate that other factors like obesity associated with glucose intolerance may also be risk factors.[652]

Using data from the EPIC Study, Berrington de Gonzalez et al. found a "nonsignificant increased risk of pancreatic cancer with increasing body mass index".[653] The study included 438,405 adults aged 19–84 years who were followed for an average of 6 years, during which 324 incident cases of pancreatic cancer were diagnosed—152 cases among males and 172 cases among females, who were a median age of 61 and 63 years, respectively, at diagnosis.

Berrington de Gonzalez et al. found the unadjusted RRs for both males and females together to be 0.74 for overweight adults, 1.15 for obese class 1 adults (BMI 30–34.9), and 1.21 for obese classes 2–3 adults (BMI ≥35).[654] Risk ratios were not given by gender. Adjusting the RRs to account for smoking, diabetes, and height quartile, however, showed little difference between classes of obesity—RR 1.16 for obese class 1, and 1.19 for obese classes 2–3. For BMI as a continuous variable, the RR was 1.08 per 5 kg/m^2 in the unadjusted analysis and 1.09 per 5 kg/m^2 in the adjusted analysis. Although the authors stated that the increased risk of pancreatic cancer with increasing BMI was "nonsignificant," the RRs they found for the obesity-pancreatic

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[649] Ibid. p. 2219.

[650] Ibid. pp. 2219, 2220.

[651] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[652] Berrington de Gonzalez, Spencer, Bueno-de-Mesquita, and study. "Anthropometry, Physical Activity, and the Risk of Pancreatic Cancer in the European Prospective Investigation into Cancer and Nutrition [EPIC]."

[653] Ibid.

[654] Ibid.

cancer association are generally considered to be significant in other studies that have been described. Therefore, the reasons for the conclusions made by Berrington de Gonzalez et al. are not clear.

In addition, the authors also estimated risk ratios after excluding the first two years of follow-up, in order to assess whether pre-existing diseases might have influenced the results, and in doing so did not find statistically significant differences in risk by BMI level.

In 2007 in order to summarize the existing evidence, which they also note is uncertain, Swedish researchers Susanna Larsson et al. conducted a systematic review and meta-analysis of prospective cohort studies published between 1966 and November 2006 that have examined the association between BMI and pancreatic cancer.[655] Their study includes a number of new studies, and updates a 2003 systematic review that showed a weak positive association in a meta-analysis of 14 studies (—2% increase in risk per 1 kg/m^2 increase in BMI).[656]

Larsson et al. found 19 studies that fit their criteria, and transformed the RR estimates in the studies to an estimate of relative risk based on increase in BMI as a continuous variable. Their summary results showed a total RR of 1.12 per 5 kg/m^2 increase in BMI—1.16 for men and 1.10 for women—indicating an overall 12% increased risk of pancreatic cancer for every five unit increase in BMI. This RR was somewhat lower than the WCRF/AICR reported RR of 1.14 per 5 kg/m^2 increase in BMI for cohort studies, and higher than the RR of 1.08 per 5 kg/m^2 increase in BMI found in the EPIC study by Berrington de Gonzalez et al. described above. In conclusion, Larsson et al. note that a positive association between BMI and risk of pancreatic cancer is plausible.

4.5.8.1 Pancreatic cancer statistics in Alberta

Neither Katzmarzyk and Janssen nor Luo et al. included pancreatic cancer in their studies on obesity and disease in Canada.[657, 658]

Pan et al. included cancer of the pancreas in their study of the association of obesity and cancer risk in Canada.[659] They found that, in 2001, 7.11% of cancer of the pancreas in Canada could be attributed to obesity—6.44% among men and 8.11% among women—but being overweight showed no apparent association with pancreatic cancer. The OR for the association between

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[655] Larsson, Orsini, and Wolk. "Body Mass Index and Pancreatic Cancer Risk: A Meta-Analysis of Prospective Studies."

[656] Berrington de Gonzalez, Amy, S. Sweetland, and Elizabeth A. Spencer. "A Meta-Analysis of Obesity and the Risk of Pancreatic Cancer," *British Journal of Cancer*, 2003, vol. 89: 519-523. Cited in Larsson, Orsini, and Wolk. "Body Mass Index and Pancreatic Cancer Risk: A Meta-Analysis of Prospective Studies."

[657] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[658] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[659] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

pancreatic cancer and obesity in both genders was estimated to be 1.51—1.43 for men and 1.63 for women.

According to the Alberta Cancer Board, there were 351 new cases of pancreatic cancer in Alberta in 2005—183 among women and 168 among men.[660] These cases accounted for 2.8% of all new cancer cases. Female pancreatic cancer accounted for 1.4% of all new cancers, and male pancreatic cancer accounted for 1.3%.

In 2005, there were four new pancreatic cancer cases in Albertans under the age of 45 years (and none among children aged 0–14 years), after which the number of cases rose progressively until the 75–79 year age group, which saw 56 new cases, and then began to decline.

As of January 1, 2005, there were 233 people living with pancreatic cancer in Alberta.[661] Therefore, in 2005, when the incidence of the 351 new cases is added, it can be estimated that 584 Albertans were living with pancreatic cancer for all or part of 2005. This 584 estimate includes the people who died from pancreatic cancer during the year, who also incurred costs associated with the disease prior to their deaths.

In this case, it is notable that—due to the high mortality rate and very low 5-year survival rate for pancreatic cancer—the number of new cases reported in 2005 substantially exceeds the number living with the illness at the beginning of that year. Therefore the bulk of economic costs for pancreatic cancer are likely to be indirect costs attributable to mortality losses rather than costs associated with living with the illness. These data, however, were not used in the cost estimates.

This study used the PAFs for pancreatic cancer as estimated by Pan et al., who as noted above found that 7.11% of cancer of the pancreas in Canada could be attributed to obesity.[662]

4.5.9 Non-Hodgkin's lymphoma

According to WCRF/AICR, lymphoid and haemopoietic cancers, when taken together, are the 6th most common type of cancer globally, accounting for about 7% of all cancer cases.[663] These cancers include Hodgkin's and non-Hodgkin's lymphomas, leukemias, and multiple myelomas, Approximately 48% of lymphoid and haemopoietic cancers are lymphomas (83% of which are non-Hodgkin's, and 17% of which are Hodgkin's), 40% are leukemias, and 12% are multiple myelomas.[664]

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[660] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

[661] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

[662] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[663] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[664] Ibid., accessed.

WCRF/AICR reports that non-Hodgkin's lymphoma, which is actually a group of malignant cancers originating from lymphocytes, is the 11th most common cause of cancer incidence worldwide.[665] Rates are more than twice as high in high-income countries as in low- or middle-income countries. The 5-year survival rate is less than 35%.

WCRF/AICR reports that "body fatness" is associated with an increased risk of lymphoid and haemopoietic cancers.[666] However, the WCRF/AICR Panel concluded that there was not enough evidence to include these cancers in its extensive meta-analysis, although it did report that 20 studies have found an association between obesity and the three types of lymphoid and haemopoietic cancers. WCRF/AICR suggested a possible biological mechanism: "Obesity results in pathological states of inflammation and altered immune responses, both of which are factors that can influence lymphoid and haemopoietic cell function."[667]

In 2007, Swedish researchers Susanna Larsson and Alicja Wolk, who have recently conducted a number of meta-analyses of obesity and cancer risk, produced a meta-analysis to summarize the association between excess body weight and the risk of non-Hodgkin's lymphoma.[668] In a search of the epidemiological literature between 1966 and February 2007, they found 16 studies published between 1999 and 2006 that met their inclusion criteria, which included reporting risk estimates for the association.

Based on the findings of these 16 studies, Larsson and Wolk estimated summary RRs for non-Hodgkin's lymphoma of 1.07 for overweight adults and 1.20 for obese adults compared to individuals with normal weight, with the results not stratified by gender. They noted that the findings did not differ significantly between cohort and case-control studies. Four of the 16 studies further subdivided obesity by class 1 (BMI 30–34.9) and classes 2–3 (BMI ≥35).When results from these four studies were combined, the RRs for non-Hodgkin's lymphoma were 1.14 for obese class 1 and 1.23 for obese class 2–3, which indicates that the risk of non-Hodgkin's lymphoma increases with increasing BMI.

4.5.9.1 Non-Hodgkin's lymphoma statistics in Alberta

Neither Katzmarzyk and Janssen nor Luo et al. included non-Hodgkin's lymphoma in their studies on obesity and disease in Canada.[669,670]

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[665] Ibid., accessed.

[666] Ibid., accessed.

[667] Ibid., accessed. p. 320.

[668] Larsson, Susanna C., and Alicja Wolk. "Obesity and Risk of Non-Hodgkin's Lymphoma: A Meta-Analysis," *International Journal of Cancer*, 2007, vol. 121: 1564-1570.

[669] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[670] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

In 2004, Pan et al.— in the previously described population-based case-control study—estimated the risk of non-Hodgkin's lymphoma in 2001 among obese and overweight Canadian adults compared with normal-weight adults.[671] They found that overall, overweight adults were nearly 1.2 times as likely (OR 1.15) and obese adults were 1.5 times as likely (OR 1.46) to develop non-Hodgkin's lymphoma as were adults with normal weight.[672] When broken down by gender, overweight (OR 1.25) and obese (OR 1.42) men and obese women (OR 1.54) were more likely than normal weight men and women to develop non-Hodgkin's lymphoma, while overweight women (OR 0.98) were slightly less likely to develop the cancer.

Pan et al. found that, overall, 4.72% and 6.45% of non-Hodgkin's lymphoma cases could be attributable to overweight and obesity respectively. Interestingly, when broken down by gender, the percentage of cases that could be attributed to overweight among men (9.09%) was higher than the percentage attributable to male obesity (6.30%.). Among women, as noted, there was no association found between overweight and non-Hodgkin's lymphoma (-0.50% of cases), but the association with female obesity was significant, with 7.03% of cases among women attributable to obesity.

However, Pan et al. found slightly different results in a related 2006 study on obesity, physical activity, energy intake, and the risk of non-Hodgkin's lymphoma, in which they used the same 1994–1997 data from the National Enhanced Cancer Surveillance System that they used for the earlier study.[673] But in this case they did not use the CCHS data for the prevalence of BMI, because they did not estimate PAFs. Compared with normal weight adults, overweight and obese men were found in this study to have ORs of 1.29 and 1.59, respectively, while overweight and obese women had ORs of 1.16 and 1.36, respectively. The 2006 article did not describe or discuss the reasons for the discrepancy in rates between the two studies.

According to the Alberta Cancer Registry, there were 557 new cases of adult non-Hodgkin's lymphoma in Alberta in 2005, and 7 new cases among Albertan children aged 0–14 years—325 cases among males, including 5 children, and 239 cases among females, including 2 children.[674] New adult cases accounted for 4.4% of all new adult cancers—with male cases accounting for 2.6% of all new adult cancers, and female cases for 1.9%.

Non-Hodgkin's lymphoma is one of the few cancers to be found in children. Among children and youth aged 0–19, there were 10 new cases in Alberta in 2005, with the earliest age at diagnosis being four new cases in the 5–9 year old group. Every age group, with the exception of 0–4 year olds, had incident cases, with the number of cases in each age group increasing after age 40, but not progressively. Overall, the largest number of cases in Alberta (70 cases) was in the 70–74 year age group, after which the number of cases in each older age group began to decline, in large part because of declining population numbers in those older age groups. Among

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[671] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."
[672] Ibid.
[673] Pan, Sai Yi, Yang Mao, Anne-Marie Ugnat, and the Canadian Cancer Registries Epidemiology Research Group. "Physical Activity, Obesity, Energy Intake, and the Risk of Non-Hodgkin's Lymphoma: A Population-Based Case-Control Study," *American Journal of Epidemiology*, 2006, vol. 162, no. 12: 1162-1173.
[674] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

men, the largest number of cases (42) was in the 65–69 year age group, and among women, the largest number of cases (32) was in the 75–79 year age group.

As of January 1, 2005, there were 2,959 people living with non-Hodgkin's lymphoma in Alberta.[675] Of these, 210 were children aged 0–14, and 2,749 were adults aged ≥15. Therefore, in 2005, when the incidence of the 557 new adult cases is added, it can be estimated that 3,306 Albertan adults were living with non-Hodgkin's lymphoma for all or part of 2005. This 3,306 estimate includes the people who died from non-Hodgkin's lymphoma during the year. These data, however, were not used in the cost estimates.

This report uses PAFs as estimated by Pan et al., who as noted above found that 4.72% and 6.45% of non-Hodgkin's lymphoma cases could be attributed to overweight and obesity, respectively.[676]

4.5.10 Multiple myeloma

According to WCRF/AIRC, multiple myeloma is the 24th most common type of cancer globally, and the 19th most common cause of cancer death.[677] It occurs three times more frequently in high-income countries than in low- or middle-income countries. The 5-year survival rate is less than 50% in high-income countries.

U.S. researchers Dominik Alexander et al. report that the incidence of multiple myeloma increases with age, with about 99% of cases being diagnosed in persons older than 40.[678] In the U.S., the incidence of multiple myeloma is twice as high in African Americans as in white Americans, and the incidence in males is about 1.5 times higher than in females. They note that the disease is fairly rare, accounting for about 0.8% of all global cancer incidence and about 0.9% of cancer deaths worldwide, and that rates in Canada, the U.S. (for white Americans), and Europe are similar. Also in the U.S., multiple myeloma is the 9th most common cause of cancer mortality among females, and the 14th most common cause among males, and accounts for about 2% of cancer deaths in each gender.

In 2007, Alexander et al. conducted a review of the epidemiological literature to examine the relationship between multiple myeloma and various risk factors, including obesity.[679] They note that established risk factors include a family history of the disease, male gender, increasing age, and African-American ethnicity. They found six studies published between 2001 and 2005 and

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[675] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.
[676] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."
[677] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.
[678] Alexander, Dominik D., Pamela J. Mink, Hans-Olov Adami, Philip Cole, Jack S. Mandel, Martin M. Oken, and Dimitrios Trichopoulos. "Multiple Myeloma: A Review of the Epidemiologic Literature," *International Journal of Cancer*, 2007, vol. 120: 40-61.
[679] Ibid.

one study published in 1994 that analysed the relationship between multiple myeloma and various risk factors. Of the seven studies, one involved only postmenopausal women, three compared white and black racial groups, one was from Korea, one used data from the Cancer Prevention Study II of the American Cancer Society, and one was from Canada.

The U.S. study that used Cancer Prevention II data, by Eugenia Calle et al., found the risk of multiple myeloma mortality among obese men and women to be RR 1.71 and 1.44, respectively, compared to men and women of normal weight.[680] The Canadian study found by Alexander et al. was the previously described study by Pan et al., which found the risk for multiple myeloma among overweight and obese men to be OR 1.64 and 2.16, respectively, and among overweight and obese women to be OR 1.28 and 1.92, respectively.[681]

In 2007, Swedish researchers Susanna Larsson and Alicja Wolk also conducted a meta-analysis of BMI and the risk of multiple myeloma based on a review of studies published between 1966 and May 2007. The authors observed that theirs is the first meta-analysis of this relationship.[682] They note that, in addition to BMI, other suspected risk factors for multiple myeloma include chronic immune stimulation, autoimmune disorders, exposure to ionizing radiation, occupational exposure to pesticides or herbicides, and prolonged use of hair dyes. The 15 studies that met their inclusion criteria for the meta-analysis—which included the Canadian study by Pan et al.—were published between 1994 and 2007. The authors found that overall, overweight and obesity were associated with a statistically significant increased risk of multiple myeloma, and that the association was similar among men and women.

Larsson and Wolk summarized the relative risks separately for cohort and case-control studies, but not separately by gender, comparing both overweight and obese adults with those of normal weight.[683] Case-control studies showed higher relative risks of multiple myeloma for both overweight adults (RR 1.43) and obese adults (RR 1.82) than did cohort studies (RR 1.12 and RR 1.27, respectively).

4.5.10.1 Multiple myeloma statistics in Alberta

Neither Katzmarzyk and Janssen nor Luo et al. included multiple myeloma in their studies on obesity and disease in Canada.[684,685]

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[680] Calle, Eugenia E., Carmen Rodriguez, Kimberly Walker-Thurmond, and Michael J. Thun. "Overweight, Obesity, and Mortality from Cancer in a Prospectively Studied Cohort of U.S. Adults," *New England Journal of Medicine*, 2003, vol. 348: 1625-1638.

[681] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[682] Larsson, Susanna C., and Alicja Wolk. "Body Mass Index and Risk of Multiple Myeloma: A Meta-Analysis," *International Journal of Cancer*, 2007, vol. 121: 2512-2516.

[683] Ibid.

[684] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[685] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

Pan et al. found that 13.92% of multiple myeloma cases among adult Canadians could be attributed to overweight—20.38% of male cases and 6.54% of female cases—and another 13.72% of cases could be attributed to obesity—15.65% of male cases and 11.41% of female cases.[686] Pan et al. found the risk for multiple myeloma among overweight and obese men to be OR 1.64 and 2.16, respectively, and among overweight and obese women to be OR 1.28 and 1.92, respectively.

According to the Alberta Cancer Registry, there were 174 new cases of multiple myeloma in Alberta in 2005—102 among men, 72 among women, and none among children.[687] Overall, these cases accounted for 1.4% of all new cancer cases in the province. Among men, they represented 0.8% of all new cancer cases, and among women, they represented 0.6%.

In 2005, there were 17 new cases of multiple myeloma among Albertans aged 30–54, after which the number of incident cases rose in each age group, but not progressively—the were 4, 7, 23, 18, and 18 new cases among aged 45–49, 50–54, 60–64, and 65–69, respectively. The highest number of cases (33) was found in the 70–74 age group, after which the numbers began to decline in the older groups, largely due to smaller population numbers in those groups. Among men, the highest number of new cases (21) was found in the 70–74 year age group, and among women, the highest number (14) was in the 75–79 year age group.

As of January 1, 2005, there were 485 people living with multiple myeloma in Alberta.[688] Therefore, in 2005, when the incidence of the 174 new cases is added, it can be estimated that 659 Albertans were living with multiple myeloma for all or part of 2005. This 659 estimate includes the people who died from multiple myeloma during the year. These data, however, were not used in the cost estimates.

This study used PAFs for multiple myeloma as estimated by Pan et al., who as noted above found that 13.72% of multiple myeloma cases among adult Canadians could be attributed to obesity.[689]

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[686] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[687] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

[688] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

[689] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

4.5.11 Leukemia

WCRF/AIRC notes that leukemia, which is a group of cancers, is the 12th most common type of cancer worldwide, and the 10th most common cause of cancer death.[690] Rates are more than twice as high in high-income countries as in low- or middle-income countries. The five-year survival rate is approximately 40% in high-income countries, but survival rates are higher in children.

In 2008, Swedish researchers Susanna Larsson and Alicja Wolk conducted a meta-analysis of cohort studies on the association between BMI and leukemia that were identified through a review of literature published between 1966 and July 2007.[691] After excluding studies that did not meet their criteria, such as studies that did not report relative risk estimates, 9 studies published between 1994 and 2007 were found to be relevant for inclusion in the analysis. Height and weight were directly measured in five of the studies, but self-reported in the other four.

The summary RRs of leukemia were 1.14 for overweight individuals, and 1.39 for obese individuals, when compared with those with normal weight. The increased risk was statistically significant in both obese men (RR 1.46) and obese women (RR 1.19), but risk ratios were not given by gender for overweight individuals. On a continuous scale, a 5 kg/m^2 increase in BMI was associated with a 13% increased risk of leukemia.

Four of the studies used in the meta-analysis examined leukemia by four sub-types, and the summary RRs showed an increased risk associated with obesity in all four of the sub-types that was not significantly different from the risk for leukemia as a whole.[692] However, Larsson and Wolk note that the statistical power of using only four studies is low and further investigation is needed.

4.5.11.1 Leukemia statistics in Alberta

Neither Katzmarzyk and Janssen nor Luo et al. included leukaemia in their studies on obesity and disease in Canada.[693, 694]

Pan et al. included leukemia in their study of obesity and cancer risk in Canada in 2001.[695] For both genders, they estimated that 9.28% of adult leukemia cases could be attributed to

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[690] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[691] Larsson, Susanna C., and Alicja Wolk. "Overweight and Obesity and Incidence of Leukemia: A Meta-Analysis of Cohort Studies," *International Journal of Cancer*, 2008, vol. 122: 1418-1421.

[692] The four sub-types of leukemia found in the studies were chronic lymphocytic leukemia, acute lymphocytic leukemia, acute myeloid leukemia, and chronic myeloid leukemia.

[693] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[694] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

overweight and a further 8.38% to obesity. Among men, they estimated that 11.35% of leukemia cases could be attributed to overweight and a further 6.16% to obesity. And among women, they estimated that 6.54% of leukemia cases could be attributed to overweight and a further 12.39% to obesity.

Pan et al. also estimated the risk of leukemia to be higher for obese women (OR 2.01) than for obese men (OR 1.41), but slightly lower for overweight women (OR 1.28) than for overweight (OR 1.32) men.[696]

According to the Alberta Cancer Registry, there were 404 new cases of leukemia in Alberta in 2005—376 among adults aged ≥15, and 28 among children aged 0–14.[697] There were 244 new cases among males, including 21 new cases among boys, and 160 new cases among females, including 7 new cases among girls.[698] Among adults, leukemia accounted for 3.0% of all new cancer cases. Male leukemia accounted for 1.8% of new adult cases, and female leukemia accounted for 1.2%.

As with non-Hodgkin's lymphoma, leukemia is one of the major cancers found in children. In 2005, in Alberta, there were a total of 33 new cases of leukemia in children and youth between the ages of 0 and 19—17 new cases in the 0–4 age group, 10 in the 5–9 age group, 1 in the 10–14 age group, and 5 in the 15–19 year age group.[699] Among male children and youth aged 19 and under, there were 22 new cases, 12 of which were in the 0–4 age group. Among female children and youth aged 19 and under, there were half as many new cases (11) as among males, 5 of which were in the 0–4 age group.

New cases of leukemia in Alberta were found in every age group in 2005, with the number of cases in each age group beginning to rise after age 40, though not progressively. The largest number of new cases (50 cases out of the 404) was in the 70–74 year age group, after which the numbers began to decline gradually among the older ages, largely due to smaller population numbers in those age groups. The largest number of cases in males (30) was found in the 75–79 year age group, and in females (23) in the 65–69 year age group.

As of January 1, 2005, there were 2,400 people living with leukemia in Alberta.[700] Of these, 2,346 were among adults aged ≥15, and 54 were among children aged 0–14. Therefore, in 2005, when the incidence of the 376 new adult cases is added, it can be estimated that 2,722 Albertan adults were living with leukemia for all or part of 2005. This 2,722 estimate includes the people who died from leukemia during the year. These data, however, were not used in the cost estimates.

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[695] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."
[696] Ibid.
[697] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.
[698] Ibid., accessed.
[699] Ibid., accessed.
[700] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

This report used PAFs as estimated by Pan et al., who as noted above found that 8.38% of adult leukemia cases could be attributed obesity.[701]

4.5.12 Liver cancer (hepatocellular carcinoma)

WCRF/AICR reports that, globally, liver cancer is the 6th most common type of cancer, and accounts for about 6% of all cancers.[702] It occurs twice as often in low- and middle-income countries as in high-income countries, with about half of all cases occurring in China. Incidence rates range from 40 per 100,000 people in eastern Asia to less than 5 per 100,000 in North America and northern Europe. Rates are more than twice as high among men as among women.

Liver cancer is almost always fatal, and is the 3rd most common cause of cancer mortality worldwide, accounting for about 9% of all cancer deaths. Five-year survival rates are only about 5 percent. Although different types of tumours occur in the liver, between 75% and 90% of liver cancers are hepatocellular carcinoma, which start in hepatocytes—the most common type of liver cell.

According to WCRF/AICR, the main causes of liver cancer are toxic compounds found in foods—especially aflatoxins that contaminate mostly grains and legumes as a result of long storage in hot, wet conditions—and alcoholic drinks.[703] Other causes include hepatitis B or C viruses, cirrhosis, and liver flukes. Approximately 80% of hepatocellular carcinoma cases are found in livers that have developed cirrhosis.

In addition, WCRF/AICR notes that there is some evidence that suggests body fatness is also a cause of this cancer. Larsson and Wolk note that the pathogenesis of liver cancer associated with excess weight may be associated with the development of non-alcoholic fatty liver disease (NAFLD):

> NAFLD is characterized by a spectrum of liver tissue changes, ranging from accumulation of fat in the liver to non-alcoholic steatohepatitis (NASH), cirrhosis, and liver cancer at the most extreme end of the spectrum. Up to 90% of obese individuals have some degree of fatty liver, and approximately 25–30% have NASH.[704]

The WCRF/AICR systematic review of the literature found six cohort studies and two case-control studies that examined liver cancer in relation to BMI.[705] The two case-control studies did not find a statistically significant association. However, five of the cohort studies found an

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[701] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[702] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[703] Ibid., accessed.

[704] Larsson, Susanna C., and Alicja Wolk. "Overweight, Obesity and Risk of Liver Cancer: A Meta-Analysis of Cohort Studies," *British Journal of Cancer*, 2007, vol. 97: 1005-1008. p. 1007.

[705] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

increased risk for the obese group when compared with normal weight adults, but this was only statistically significant for men in four studies (RR 4.52, 1.56, 3.88, and 3.60), and for women in two studies (RR 1.68 and 1.70). Another cohort study found an increased risk in white men (RR 1.44) but not in black men (0.68).

Subsequent to the WCRF/AICR review, Swedish researchers Susanna Larsson and Alicja Wolk conducted a meta-analysis of cohort studies investigating the association between BMI and the risk of liver cancer.[706] In a literature search of studies published between 1966 and June 2007, Larsson and Wolk identified 11 relevant cohort studies, which included the 6 reviewed by WCRF/AICR.

The Larsson and Wolk meta-analysis found that adults who were overweight or obese had risks for liver cancer of RR 1.17 and RR 1.89, respectively, compared to adults with normal weight.[707] When three studies, in which BMI was derived from a hospital discharge diagnosis of obesity, were eliminated from the analysis, the RR for obese adults based on self-reported and directly measured BMI was 2.15. When all of the cohort studies were included, the risk among men (RR 2.42) was significantly higher than that among women (1.67).

Larsson and Wolk note that their study is the first one to summarize the epidemiological evidence quantitatively. Using the summarized RRs and the prevalence of excess weight in the U.S., Larsson and Wolk estimated that 28% of liver cancer cases among men and 27% among women in the U.S. could be attributed to excess weight (BMI ≥25). In conclusion, the authors note that "this meta-analysis supports evidence of an increased risk of liver cancer among overweight and obese persons. These findings indicate that liver cancer may, in part, be prevented by maintaining a healthy body weight."[708]

4.5.12.1 Liver cancer statistics in Alberta

Neither Katzmarzyk and Janssen nor Luo et al. included liver cancer in their studies on obesity and disease in Canada.[709, 710]

Pan et al. included liver cancer in their study of obesity and cancer risk in Canada in 2001.[711] However, they did not estimate the portion of liver cancer in the population that could be attributed to excess weight. They found that, for both genders, obese adults had a 17% increased risk (OR 1.17) of liver cancer compared with adults of normal weight, but overweight adults had no increased risk (OR 0.89). When stratified by gender, only obese men had an increased risk

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[706] Larsson, and Wolk. "Overweight, Obesity and Risk of Liver Cancer: A Meta-Analysis of Cohort Studies."
[707] Ibid.
[708] Ibid. p. 1008.
[709] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."
[710] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."
[711] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

(OR 1.30). They did not find elevated risks for liver cancer among overweight men (OR 0.99) or among overweight or obese women (ORs 0.61 and 0.94, respectively).

According to the Alberta Cancer Registry, in 2005, there were 166 new adult cases of liver cancer in Alberta, and one child case—119 cases among men, 47 among women, and one in a boy in the 10–14 year age group.[712] Overall, the cases accounted for 1.3% of all new cancer cases. Male liver cancer accounted for 0.9% of all new cases in Alberta and female liver cancer accounted for 0.4%.

Under the age of 50, there were only 14 new cases of liver case, with 10 of these in the 45–49 year age group. The highest number of new cases (26) was in the 70–74 year group. Among males, the highest number of new cases (19) was in the 50–54 year age group, and among females, the highest number (9) was in the 75–79 year age group.

As of January 1, 2005, there were 231 people living with liver cancer in Alberta.[713] Therefore, in 2005, when the incidence of the 166 new cases is added, it can be estimated that 397 Albertan adults were living with liver cancer for all or part of 2005. This 397 estimate includes the people who died from liver cancer during the year. These data, however, were not used in the cost estimates.

The PAFs for this study were estimated from the RRs estimated by Larsson and Wolk that were given above and the prevalence of obesity in Alberta data from 2004 CCHS cycle 2.2. Approximately 25.2% of liver cancer could be attributed to obesity. It was not possible to use the ORs from Pan et al. because the data needed to convert the ORs to RRs were not available.

4.5.13 Bladder cancer

WCRF/AICR reports that, globally, bladder cancer is the 10th most common type of cancer, and accounts for about 3% of all cancers.[714] It is five times more prevalent in men than in women, and the risk increases with age. Age-adjusted incidence rates in Europe and North America are 20–30 per 100,000 men. Rates are more than three times higher in high-income countries than in low- or middle-income countries. In the U.S., bladder cancer is the fourth most common cancer in men and the ninth most common in women.[715] It is also the 11th most common cause of death from cancer, and accounts for about 2% of all cancer deaths.[716]

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[712] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

[713] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

[714] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[715] Koebnick, Corinna, Dominique Michaud, Steven C. Moore, Yikyung Park, Albert Hollenbeck, Rachel Ballard-Barbash, Arthur Schatzkin, and Michael F. Leitzmann. "Body Mass Index, Physical Activity, and Bladder Cancer in a Large Prospective Study," *Cancer Epidemiology Biomarkers & Prevention*, 2008, vol. 17, no. 5: 1214-1221.

[716] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

According to WCRF/AICR, smoking is responsible for more than half of all bladder cases in men and about a third in women. It notes: "Dietary carcinogens, as well as those from tobacco smoke or other environmental sources, are often excreted in the urine, so the bladder lining is exposed to these toxins."[717]

The WCRF/AICR review of the epidemiological evidence for associations between bladder cancer and "body fatness" were inconclusive: "[T]he data were either of too low quality, too inconsistent, or the number of studies too few to allow conclusions to be reached."[718] However, this does not mean that no association exists. Early research on smoking impacts was also inconclusive on subsequently proven links with heart disease. Thus, the WCRF/AICR statement must be understood as a simple acknowledgement of the very early stage of research in this particular subject area and therefore of the need for more intensive investigation.

In fact, a subsequent and very recent large prospective study and review has found more conclusive evidence. In a report published in May 2008, U.S. researchers Corinna Koebnick of the National Cancer Institute et al. found that obesity was associated with a 28% increased risk for bladder cancer in the U.S.[719] Koebnick et al. refer to their study as "the largest study to date to examine BMI and physical activity in relation to this important malignancy."[720] They note that previous studies examining excess weight and bladder cancer, which have not found a statistically significant association, have been limited by small numbers of cases.

Koebnick et al. also note that the biological mechanisms that underlie the positive association between bladder cancer and BMI are speculative, but offer the following possibilities, which echo findings on mechanisms postulated for other cancers:

> Excess body fat is associated with elevated production of insulin, and insulin is a mitogenic factor that may also enhance tumor growth by increasing free insulin-like growth factor-I, which in turn stimulates cell proliferation and suppresses apoptosis and has been linked to bladder cancer. Although hyperinsulinemia per se has not been implicated in bladder carcinogenesis, type 2 diabetes is directly associated with bladder cancer. Adiposity is also accompanied by low-grade, systemic inflammation, which may play a role in bladder carcinogenesis as suggested by positive relations of circulating levels of inflammatory markers, such as C-reactive protein and interleukin-6, to bladder cancer mortality.[721]

Thus, the mechanisms linking obesity with bladder and other types of cancer may well be indirect. To the extent that obesity is strongly and demonstrably linked with type 2 diabetes, and that, in turn, "type 2 diabetes is directly associated with bladder cancer", obesity is likely a contributing factor to this cancer type.

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[717] Ibid., accessed. p. 313.
[718] Ibid., accessed. p. 314.
[719] Koebnick, Michaud, Moore, Park, Hollenbeck, Ballard-Barbash, Schatzkin, and Leitzmann. "Body Mass Index, Physical Activity, and Bladder Cancer in a Large Prospective Study."
[720] Ibid. p. 1214.
[721] Ibid. p. 1220.

Koebnick et al. used data from the prospective cohort NIH-AARP Diet and Health Study, which followed 471,760 adults, aged 50–71 years, from 1995 to 2003, during which 1,719 incident cases of bladder cancer—1,470 in men and 249 in women—were documented. They found that the risk for bladder cancer increased with increasing BMI. When compared to adults with normal weight, and after adjustment for age and gender, the RRs of bladder cancer for overweight (BMI 25–29.9), class 1 obesity (BMI 30–34.9), and classes 2–3 obesity combined (BMI ≥35), were 1.18, 1.29, and 1.34, respectively.

Koebnick et al. also note that their findings are consistent with results from 8 out of 11 prospective cohort studies on the topic. However, out of four case-control studies, only the large Canadian study by Pan et al. reported positive associations.[722] In conclusion, Koebnick et al. remark that they found "a modest but graded positive association between BMI and risk of bladder cancer…. Thus, bladder cancer may be added to the list of cancers potentially related to adiposity."[723]

4.5.13.1 Bladder cancer statistics in Alberta

Neither Katzmarzyk and Janssen nor Luo et al. included bladder cancer in their studies on obesity and disease in Canada.[724, 725]

Pan et al. included bladder cancer in their study of obesity and cancer risk in Canada in 2001.[726] However, they did not estimate the portion of bladder cancer that could be attributed to excess weight. They found that for both genders, obese adults had a 27% increased risk (OR 1.27) of bladder cancer when compared with adults of normal weight, and overweight adults had a 12% increased risk (OR 1.12). When stratified by gender, obese and overweight men had ORs of 1.35 and 1.18, respectively. The ORs for obese and overweight women were 1.15 and 1.03, respectively.

According to the Alberta Cancer Registry, there were 256 new adult cases of bladder cancer in Alberta in 2005, and 1 child case—with 188 cases among males and 69 among females (including the child).[727] Overall, bladder cancer accounted for 2.0% of all new cancer cases. New bladder cancer cases among males accounted for 1.5% of all new cancer cases, while new cases among females accounted for 0.5%.

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[722] Pan, DesMeules, Morrison, Wen, and Group. "Obesity, High Energy Intake, Lack of Physical Activity, and the Risk of Kidney Cancer."

[723] Koebnick, Michaud, Moore, Park, Hollenbeck, Ballard-Barbash, Schatzkin, and Leitzmann. "Body Mass Index, Physical Activity, and Bladder Cancer in a Large Prospective Study." pp. 1218, 1220.

[724] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[725] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[726] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

[727] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

In 2005, there were 20 new bladder cancer cases in Albertans below the age of 55, and one of these occurred in a girl in the 5–9 age group—the only new case among children and youth aged 19 and younger. Overall, the number of new cases remained low until age 55, when they began to increase progressively until the 75–79 year age group, which had 39 new cases. The number of new cases was high in all the older age groups, but began to decline in the 80+ age groups, largely due to smaller population sizes in those groups.

There were 10 new cases of bladder cancer in Albertan males under the age 55, with the number of new cases then increasing progressively to reach its highest level (31 cases) in the 70–74 year age group, after which they began to decline. Among Albertan females, there were also 10 new bladder cancer cases in age groups below age 55—including the above mentioned childhood case. The number of new female bladder cancer cases then rose to a high of 13 new cases in the 85–89 year age group.

As of January 1, 2005, there were 2,071 people living with bladder cancer in Alberta.[728] Therefore, in 2005, when the incidence of the 256 new adult cases is added, it can be estimated that 2,327 Albertan adults were living with bladder cancer for all or part of 2005. This 2,327 estimate includes the people who died from bladder cancer during the year. These data, however, were not used in the cost estimates.

This report used RRs from the Koebnick et al. study noted above and the prevalence of obesity in Alberta data from 2004 CCHS cycle 2.2 to estimate the portion of bladder cancer that could be attributed to obesity in Alberta (7.53%). It was not possible to use the ORs from Pan et al. because the data needed to convert the ORs to RRs were not available.

4.5.14 Stomach (gastric) cancer

WCRF/AICR reports that stomach cancer is the 4th most common type of cancer worldwide, and accounts for almost 9% of all cancers.[729] Incidence rates range from more than 60 per 100,000 people in Asia to less than 10 per 100,000 in North America, and are more than twice as high in men as in women, but reasons for these variations have not been identified. The risk for stomach cancer increases with age, and it is rarely diagnosed in people under the age of 50 years. It is the 2nd most common cause of death from cancer (after lung cancer), accounting for over 10% of all cancer deaths, and is usually fatal, with the five-year survival rate being approximately 20%.

There are two types of stomach cancer—distal gastric cancer of the lower portion of the stomach (noncardia gastric adenocarcinoma), and proximal gastric cardia of the gastro-esophageal junction (gastric cardia adenocarcinoma), which is often grouped with cancer of the esophagus in

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[728] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.
[729] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

epidemiological studies. According to D. Forman and V.J. Burley of the University of Leeds, most malignant tumours of the stomach are epithelial in origin and, consequently, "the overwhelming majority of cancers of the stomach are adenocarcinomas and most of the routine statistics about gastric cancer refer to this histological entity."[730]

WCRF/AICR notes that "food and nutrition play an important role in the prevention and causation of stomach cancer."[731] Among the causes of stomach cancer, it lists salt and salt-preserved foods as probable causes, and chilli, processed meat, smoked foods, and grilled or barbecued animal foods as possible causes. In addition WCRF/AICR notes that *Helicobacter pylori* bacterium infection "is established as a necessary cause of almost all cases of stomach cancer."[732]

> Changes in the stomach mucosa, brought about by a variety of environmental factors and ageing, can eventually lead to atrophic gastritis. The chronic form of this condition, and the resulting changes in the characteristics of the stomach cells, appear to be precursor conditions to the development of distal stomach cancer. Food carcinogens can also potentially interact directly with the epithelial cells that line the stomach. However, cancer can also develop without these precursors, particularly when the bacterium *H pylori* is present in the stomach.[733]

WCRF/AICR evaluated other exposures, including "body fatness," in order to identify other possible causes of stomach cancer, but it did not find enough high quality, consistent data or sufficient studies to draw a conclusion. As noted in our consideration of bladder cancer above, present data insufficiencies by no means indicate lack of association but point, rather, to the very early stage of research in this area and the need for more rigorous, differentiated studies with adequate sample sizes to draw more definitive conclusions.

For example, Forman and Burley note that most studies that do not differentiate types of stomach cancer generally have found no association between stomach cancer and excess weight.[734] However, studies that have reported risk according to tumour origin site have generally found that the risk of stomach cancer occurring in the proximal region of the stomach, or cardia, may be elevated in obese adults. They note that increased risks for cancer occurring in the noncardial, or distal region of the stomach, are not found among obese adults.[735]

Katherine Crew and Alfred Neugut of Columbia University also report that obesity is mainly a risk factor for the proximal type of stomach cancer—adenocarcinomas of the gastric cardia—

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[730] Forman, D., and V.J. Burley. "Gastric Cancer: Global Pattern of the Disease and an Overview of Environmental Risk Factors," *Best Practice & Research Clinical Gastroenterology*, 2006, vol. 20, no. 4: 633-649. p. 633.
[731] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed. p. 265.
[732] Ibid., accessed.
[733] Ibid., accessed. p. 266.
[734] Forman, and Burley. "Gastric Cancer: Global Pattern of the Disease and an Overview of Environmental Risk Factors."
[735] Ibid.

which has been increasing in Western countries since the 1970s, especially in males.[736] They note that gastric cardia tumours account for almost 50% of stomach cancers among men in the U.S. and U.K.

A large U.S. population-based case-control study, conducted by the National Cancer Institute, investigated patients aged 30–79 years who had a number of diseases, including gastric cardia adenocarcinoma, by comparison with control subjects.[737] The authors, Lawrence Engel et al., found that the majority of case patients for all types of gastric cancer were male—ranging from 69.0% of noncardia gastric adenocarcinoma patients to 85.4% of gastric cardia adenocarcinoma patients. The median ages for these two types of stomach cancer were 70 and 65 years, respectively.

For gastric cardia adenocarcinoma, the National Cancer Institute study found that the risk of excess weight among both genders was OR 1.3, compared to those with a BMI of 17.6–23.1. It attributed 19.2% of this type of stomach cancer in the U.S. to excess weight (22.6% among men and 8.7% among women). The authors did not estimate risk by BMI categories for noncardia gastric adenocarcinoma.

Swedish and Spanish researchers from the Karolinska Institutet, led by Mats Lindblad, investigated the association between BMI and both gastric cardia and noncardia adenocarcinoma in patients aged 40–84 years, using British data from the General Practitioners Research Database (GPRD), which they note "is one of the largest computerized databases of longitudinal patient records in the world, containing more than 35 million patient-years of British primary care data."[738]

Compared with normal weight adults, Lindblad et al. found that the risks for total stomach cancer were OR 1.09 and 1.21 for overweight and obese adults, respectively. When the two main types of stomach cancer were separated, an increased risk with increasing BMI was found for gastric cardia adenocarcinoma (total OR 1.46 for overweight and obese adults combined), but not for noncardia adenocarcinoma (total OR 0.97 for overweight and obese adults combined).[739] Interestingly, overweight adults showed an 11% increased risk for noncardia adenocarcinoma (OR 1.11), while obese adults had a decreased risk (OR 0.87)—indicating inconclusive results that could not be explained. Risks for noncardia adenocarcinoma were not reported by gender.

For gastric cardia adenocarcinoma, Lindblad et al. found that overweight (BMI 25-29.9), obese (BMI ≥30), and severely obese (BMI ≥35) adults all had elevated risks (OR 1.37,1.46, and 1.47

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[736] Crew, Katherine D., and Alfred I. Neugut. "Epidemiology of Gastric Cancer," *World Journal of Gastroenterology*, 2006, vol. 12, no. 3: 354-362.

[737] Engel, Lawrence S., Wong-Ho Chow, Thomas L. Vaughan, Marilie D. Gammon, Harvey A. Risch, Janet L. Stanford, Janet B. Schoenberg, Susan T. Mayne, Robert Dubrow, Heidrun Rotterdam, A. Brian West, Martin Blaser, William J. Blot, Mitchell H. Gail, and Joseph F. Fraumeni Jr. "Population Attributable Risks of Esophageal and Gastric Cancers," *Journal of the National Cancer Institute*, 2003, vol. 95, no. 18: 1404-1413.

[738] Lindblad, Mats, Luis A. Garcıa Rodrıguez, and Jesper Lagergren. "Body Mass, Tobacco and Alcohol and Risk of Esophageal, Gastric Cardia, and Gastric Non-Cardia Adenocarcinoma among Men and Women in a Nested Case-Control Study," *Cancer Causes and Control*, 2005, vol. 16: 285-294. p. 286.

[739] Ibid.

respectively) .[740] When stratified by gender, the risk of gastric cardia adenocarcinoma was found to be higher for overweight men (OR 1.41) than for obese men (OR 1.18), but the opposite was the case for overweight (OR 1.20) and obese (OR 1.91) women.[741] The counter-intuitive result for men again points to the need for further investigation in this new area of exploration.

In 2006, Kubo and Corley conducted a systematic review and meta-analysis of studies published between 1966 through July 2005 that evaluated the association between BMI and the risk of gastric cardia cancer.[742] They identified 11 relevant studies—seven that reported gastric cardia carcinoma alone, and four that combined cardia carcinoma with esophageal adenocarcinoma. Overall, the study found the risk for gastric cardia adenocarcinoma to be 20% higher in overweight adults (BMI 25–27.9) (OR 1.20) and 50% higher in obese adults (BMI ≥28) (OR 1.50) than in those with normal weight. When only studies from the U.S. and Europe were included, the overall risk was OR 1.5 for both overweight and obese adults combined—OR 0.6 for overweight adults, and OR 1.9 for obese adults. The results were not disaggregated by gender.

4.5.14.1 Stomach cancer statistics in Alberta

Neither Katzmarzyk and Janssen nor Luo et al. included stomach cancer in their studies on obesity and disease in Canada. [743, 744]

Pan et al. included stomach cancer as a whole in their study of obesity and cancer risk in Canada in 2001.[745] However, they did not estimate the portion of stomach cancer in the population that could be attributed to excess weight. They found that for both genders, obese adults had a 25% increased risk (OR 1.25) of stomach cancer when compared with adults of normal weight, while overweight adults had a slightly decreased risk (OR 0.97). When stratified by gender, overweight and obese men had increased risks (OR 1.01 and 1.36, respectively), but overweight and obese women (ORs 0.90 and 0.92, respectively) did not.

The sharp difference between Pan et al.'s results for stomach cancer among overweight and obese women and those of Lindblad et al. (ORs 1.20 and 1.91) for gastric cardia adenocarcinoma again point to the importance of differentiating the two kinds of stomach cancer in studies assessing associations with BMI. At this point, however, data are not available in Canada to differentiate between the two types of stomach cancer.

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[740] Ibid.

[741] Ibid.

[742] Kubo, and Corley. "Body Mass Index and Adenocarcinomas of the Esophagus or Gastric Cardia: A Systematic Review and Meta-Analysis."

[743] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[744] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[745] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."

According to the Alberta Cancer Registry, there were 229 new cases of stomach cancer in Alberta in 2005—151 among males, 78 among females, and none among children. Overall, the new adult stomach cancer cases accounted for 1.8% of all new adult cancer cases. Male stomach cancer cases accounted for 1.2% of all new cancer cases, and female stomach cancer accounted for 0.6%.

Overall, the number of new stomach cancer cases in Alberta was low in each age group from 25 to 50, and then began to rise. The highest number of new cases was found in the 75–79 year age group, which had 27 new stomach cancer cases. Among men, the highest number of new cases (29 cases) was in the 70–74 age group, and among women, the highest number (13 cases) was in the 75–79 year age group.

As of January 1, 2005, there were 683 people living with stomach cancer in Alberta.[746] Therefore, in 2005, when the incidence of the 229 new cases is added, it can be estimated that 912 Albertans were living with stomach cancer for all or part of 2005. This 912 estimate includes the people who died from stomach cancer during the year. These data, however, were not used in the cost estimates.

The PAFs for stomach cancer in this report were based on Pan et al.'s OR estimates and obesity prevalence data for Alberta from 2004 CCHS cycle 2.2. In the case of stomach cancer, which has a very low incidence rate, ORs can be considered to be reasonable proxies for RRs.[747]

4.5.15 Cancer statistics in Alberta

In 2004, Canadian researchers Sai Yi Pan et al. examined the association between BMI and many types of cancer in Canada—a study that was made possible through use of one large-sample data source that surveyed cancer patients.[748] The data were from the National Enhanced Cancer Surveillance System (NECSS), which—between 1994 and 1997—surveyed 21,022 Canadians, aged 20–76 years, who had one of 19 types of cancer, and 5,039 Canadians who served as population controls.[749]

Pan et al. used these data to estimate the degree to which risks of cancer overall and of particular site-specific cancers were attributable to overweight and obesity, by calculating adjusted odds ratios (OR) from the NECSS data as compared with those of normal weight. ORs were adjusted by 5-year age group, province of residence, education, pack-years of smoking, alcohol drinking, total caloric intake, vegetable intake, dietary fibre intake, recreational physical activity, menopausal status, number of live births, age at the onset of first menstruation, age at end of first

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[746] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.
[747] Zhang, Jun, and Kai F. Yu. "What's the Relative Risk? A Method of Correcting the Odds Ratio in Cohort Studies of Common Outcomes," *JAMA (Journal of the American Medical Association)*, 1998, vol. 280, no. 19: 1690-1691.
[748] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."
[749] Ibid.

pregnancy, and sex (for ORs for both sexes). Pan et al. also estimated overweight and obesity-related PAFs for various cancers in Canada, using BMI rates from the 2001 CCHS.

However, among the 19 cancers they investigated, Pan et al. did not include endometrial (uterine) cancer, which has been found to have one of the strongest associations with obesity among all cancers, and one of the highest risk ratios attributable to obesity among cancers. As will be described in more detail below, this report uses the PAFs (and one OR) provided by Pan et al.—to the extent possible—to estimate PAFs for cancer in the Alberta population, and uses other reliable sources wherever the PAFs provided by Pan et al. do not suffice for our purposes.

Pan et al. found that obese Canadians had a relative risk for all 19 cancers combined that was 34% higher than the risk for adults with normal weight, and overweight Canadians had a risk that was 9% higher. For all cancer cases in Canada, 2.88% of overall cancer was attributable to overweight, and an additional 4.85% of overall cancer was attributable to obesity.

Pan et al. found that obese adults had increased risks of non-Hodgkin's lymphoma, leukemia, multiple myeloma, and cancers of the kidney, colon, rectum, breast (postmenopausal), ovary, pancreas, liver, stomach, bladder, and prostate. Therefore, this report estimates separate overweight and obesity-related costs for all these cancers in Alberta as well as costs for endometrial cancer, which has strong, proven associations with obesity.

Cancers that, in Pan et al.'s analysis, showed no or only slight associations with overweight or obesity were cancers of the lung, brain, bone, and salivary glands for both men and women; testicular cancer for men; and premenopausal breast cancer for women, although the authors did find a 13% elevated risk of premenopausal breast cancer among obese women. This report does not provide cost estimates for any of these particular cancers.

Table 23 below shows the ORs of overall cancer and site-specific cancers associated with overweight and obesity for both men and women and for both sexes combined, as estimated by Pan et al.

Table 23. Odds ratios (OR) of overall cancer and site-specific cancers associated with overweight (BMI 25–<30) and obesity (BMI ≥30), National Enhanced Cancer Surveillance System, aged 20–76, by gender, Canada, 1997

Cancer site	Men		Women		Both	
	OR overweight	OR obese	OR overweight	OR obese	OR overweight	OR obese
All cancers	1.14	1.29	1.02	1.41	1.09	1.34
kidney	2.03	3.15	1.49	2.42	1.77	2.74
colon	1.54	2.16	1.22	1.77	1.40	1.93
rectum	1.41	1.75	1.28	1.50	1.36	1.65
non-Hodgkin's lymphoma	1.25	1.42	0.98	1.54	1.15	1.46
leukemia	1.32	1.41	1.28	2.01	1.31	1.61
multiple myeloma	1.64	2.16	1.28	1.92	1.49	2.06
pancreas	1.03	1.43	0.85	1.63	0.99	1.51
bladder	1.18	1.35	1.03	1.15	1.12	1.27
liver	0.99	1.30	0.61	0.94	0.89	1.17
stomach	1.01	1.36	0.90	0.92	0.97	1.25
prostate	1.16	1.27	na	na	na	na
premenopausal breast	na	na	0.89	1.13	1.10*	1.47*
postmenopausal breast	na	na	1.17	1.66		
ovary	na	na	1.16	1.95	na	na

Note: OR – Odds ratio, na – not applicable; * includes pre-and postmenopausal breast cancer.
ORs have been adjusted for adjusted for 5-year age group, province of residence, education, pack-years of smoking, alcohol drinking, total caloric intake, vegetable intake, dietary fiber intake, recreational physical activity, and for women: menopausal status, number of live births, age at menarche, and age at end of first pregnancy.

Source: Pan, Sai Yi, Kenneth C. Johnson, Anne-Marie Ugnat, Shi Wu Wen, Yang Mao, and the Canadian Cancer Registries Epidemiology Research Group. "Association of Obesity and Cancer Risk in Canada," *American Journal of Epidemiology*, 2004, vol. 159, no. 3: 259-268.

Table 24 below shows the portions of specific cancers that Pan et al. found to be attributable to obesity, among Canadian men and women aged 20–76 years in 2001. However, the authors only included PAFs for some of the types of cancer that had positive risks associated with obesity. As will be explained in Part 2 of this report, these proportions of site-specific cancers attributable to obesity were used in this study.

The portions of the specific cancers in men that could be attributed to obesity (BMI ≥30), as estimated by Pan et al., include 25.59% of kidney cancer, 15.65% of colon cancer, 10.71% of rectal cancer, 6.44% of pancreatic cancer, 15.65% of multiple myeloma, 6.30% of non-Hodgkin's lymphoma, 6.16% of leukemia, and 4.14% of prostate cancer.

The additional portions of specific cancer in men that could be attributed to overweight (BMI 25–<30) include 29.18% of kidney cancer, 17.76% of colon cancer, 14.09% of rectal cancer, 1.19% of pancreatic cancer, 20.38% of multiple myeloma, 9.09% of non-Hodgkin's lymphoma, 11.35% of leukemia, and 6.02% of prostate cancer.

The portions of the specific cancers in women that could be attributed to obesity, as estimated by Pan et al., include 16.58% of kidney cancer, 9.73% of colon cancer, 6.54% of rectal cancer, 8.11% of pancreatic cancer, 11.41% of multiple myeloma, 7.03% of non-Hodgkin's lymphoma, 12.39% of leukemia, 11.74% of ovarian cancer, and 8.46% of postmenopausal breast cancer.

The additional portions of the specific cancers in women that could be attributed to overweight, as estimated by Pan et al., include 10.91% of kidney cancer, 5.21% of colon cancer, 6.54% of rectal cancer, 6.54% of multiple myeloma, 6.54% of leukemia, 3.85% of ovarian cancer, and 4.08% of postmenopausal breast cancer. However, slight negative associations with overweight (BMI 25–29.9) were found for pancreatic cancer and non-Hodgkin's lymphoma—estimated by Pan et al. as –3.90% of pancreatic cancer and –0.50% of non-Hodgkin's lymphoma.

Table 24. Portions of specific cancers attributable to overweight and obesity (PAF–%), aged 20-76, by gender, Canada, 2001

Cancer site	All		Males		Females	
	Over-weight	Obesity	Over-weight	Obesity	Over-weight	Obesity
All cancers	2.88	4.85	5.30	4.43	0.50	5.43
kidney	20.26	20.7	29.18	25.59	10.91	16.58
colon	11.66	12.24	17.76	15.65	5.21	9.73
rectal	10.62	8.88	14.09	10.71	6.54	6.54
leukemia	9.28	8.38	11.35	6.16	6.54	12.39
pancreas	-0.33	7.11	1.19	6.44	-3.90	8.11
non-Hodgkin's lymphoma	4.72	6.45	9.09	6.30	-0.50	7.03
multiple myeloma	13.92	13.72	20.38	15.65	6.54	11.41
prostate	na	na	6.02	4.14	na	na
ovary	na	na	na	na	3.85	11.74
postmenopausal breast	na	na	na	na	4.08	8.46

Note: PAF – population attributable fraction; na – not applicable

Source: Pan, Sai Yi, Kenneth C. Johnson, Anne-Marie Ugnat, Shi Wu Wen, Yang Mao, and and the Canadian Cancer Registries Epidemiology Research Group. "Association of Obesity and Cancer Risk in Canada," *American Journal of Epidemiology*, 2004, vol. 159, no. 3: 259-268.

Figures 32–34 below show the percentage of all new adult (aged ≥15 years) cancer cases in Alberta in 2005 that are accounted for by the types of cancers attributable to obesity. Figure 32 provides these proportions for both genders combined, and Figures 33 and 34 indicate the proportions for males and females separately as proportions of total male and female cases respectively.

The 2005 data used to estimate these percentages come from the Alberta Cancer Registry (ACR) and, as of this writing, are the latest published data available.[750] The latest data from PHAC's Cancer Surveillance On-Line are from 2004. However, in reference to potential discrepancies found between different data sources, PHAC notes: "The provincial level files contain numbers taken directly from the Canadian Cancer Registry (CCR) and are the proper totals."[751] Released in 2008, the Alberta Cancer Registry's *2005 Annual Report of Cancer Statistics* notes that incidence and mortality rates that are used in its reports are based on Statistics Canada's

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[750] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.
[751] Public Health Agency of Canada (PHAC). *Cancer Surveillance on-Line*, accessed.

population figures.[752] In sum, the proportions provided here are based on the most recent and reliable data available for Alberta.

In 2005, the total number of incident cancer cases in Alberta among adults aged ≥15 years was 12,669 cases (excluding nonmelanoma skin cancer, as did ACR)—6,526 cases among males and 6,143 cases among females. Of total new adult cancer cases, 7,909 cases or *62.4% are of a type that has been partially attributable to obesity.*

Figure 32 below shows that, for both genders combined, the percentages of total adult cancers for the types of cancer attributable to obesity are highest for prostate (15.0%), colorectal (12.4%), and postmenopausal breast (10.5%) cancer. The other obesity-related cancers comprise fairly small percentages, ranging from 0.9–4.4% of all cases. Of the total number of adult cancer cases, 37.6% are of types that have no demonstrated link to obesity.

Figure 33 shows that, for among obesity-related cancers afflicting males, prostate cancer (29.2%) and colorectal cancer (12.0%) comprise the highest percentages of total male cancer cases, with the percentages of other cancers ranging from 1.4% to 4.9%, and cancers unrelated to obesity comprising 34.6%.

Figure 34 shows that, among obesity-related cancers afflicting females, postmenopausal breast cancer (21.7%) and colorectal cancer (10.4%) comprise the highest percent of total female cancers, with the percentages of other cancers ranging from 0.3-6.1%, and cancers unrelated to obesity comprising 43.0%.

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[752] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

Figure 32. Percentage of all new cancer cases accounted for by types of cancers that are associated with obesity, adults aged ≥15, both genders combined, Alberta, 2005
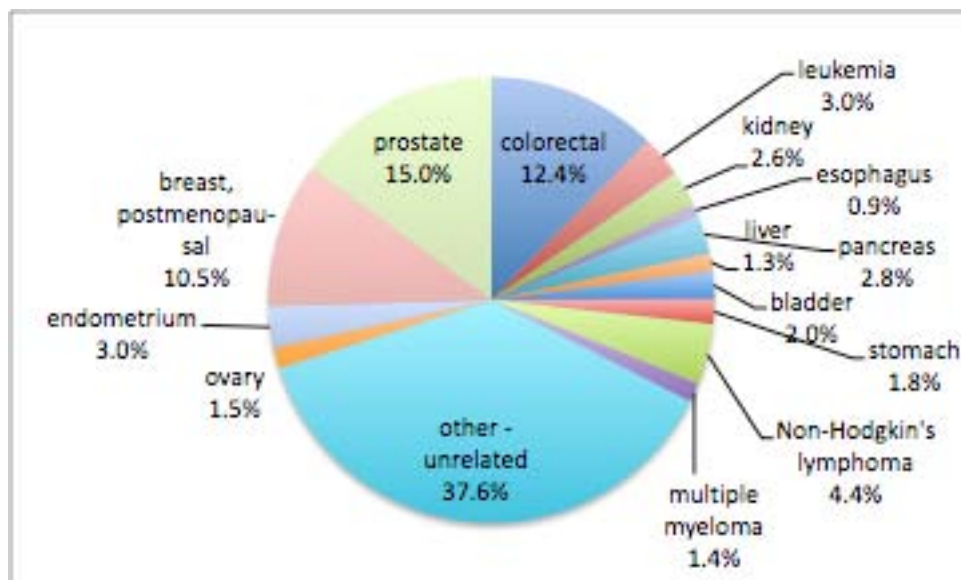
Source: Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics* Alberta Health Services, Alberta Cancer Board, 2008; accessed July 2008; available from http://www.cancerboard.ab.ca/NR/rdonlyres/2BADD738-9C4D-487F-B8C1-68D5D962CD81/0/AlbertaCancerRegistry200531Oct08.pdf.

Figure 33. Percentage of all new male cancer cases accounted for by types of cancers that are associated with obesity, males aged ≥15, Alberta, 2005
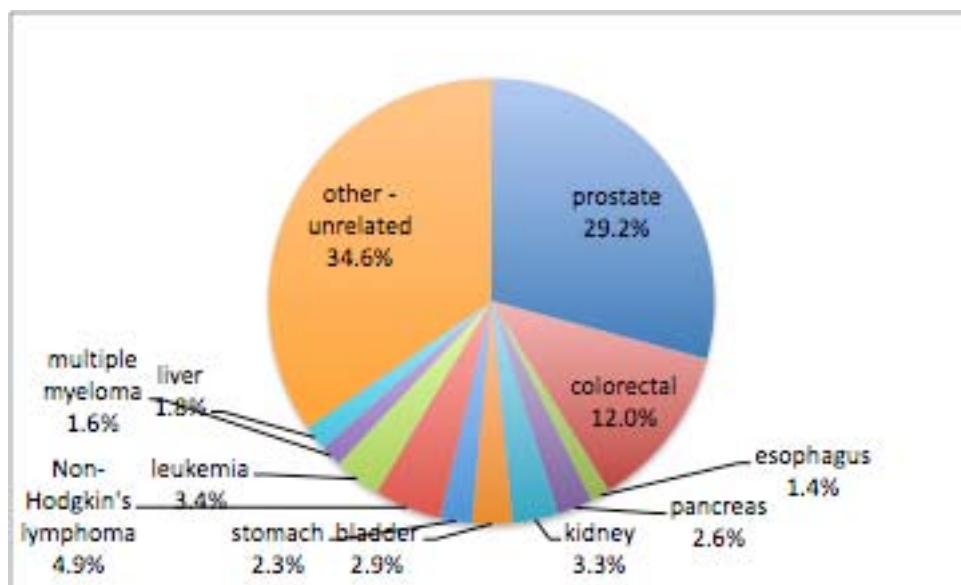
Source: Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics* Alberta Health Services, Alberta Cancer Board, 2008; accessed July 2008; available from http://www.cancerboard.ab.ca/NR/rdonlyres/2BADD738-9C4D-487F-B8C1-68D5D962CD81/0/AlbertaCancerRegistry200531Oct08.pdf.

Figure 34. Percentage of all new female cancer cases accounted for by types of cancers that are associated with obesity, females aged ≥15, Alberta, 2005
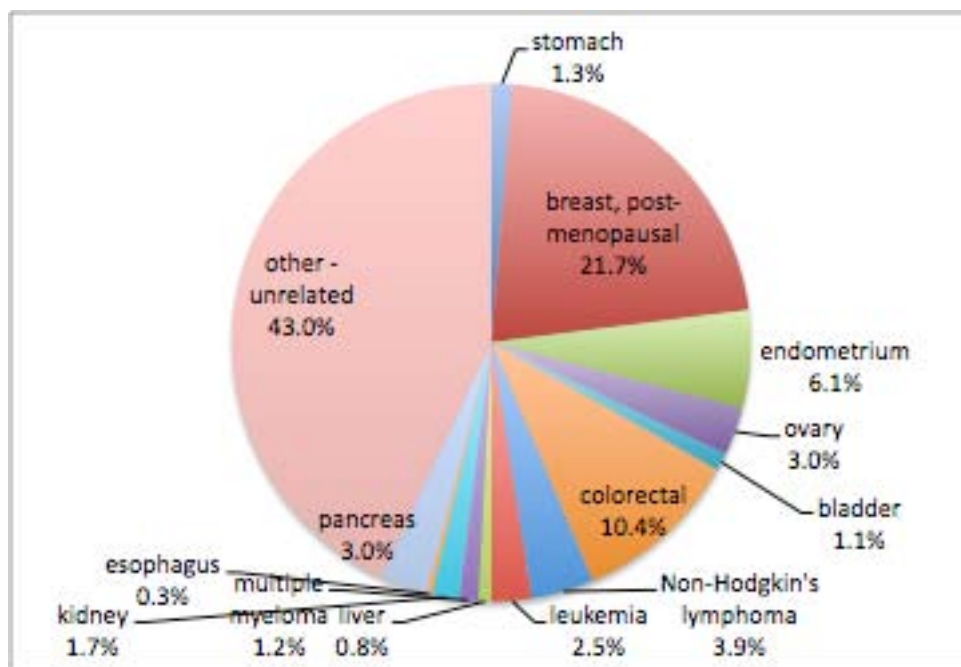
Source: Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics* Alberta Health Services, Alberta Cancer Board, 2008; accessed July 2008; available from http://www.cancerboard.ab.ca/NR/rdonlyres/2BADD738-9C4D-487F-B8C1-68D5D962CD81/0/AlbertaCancerRegistry200531Oct08.pdf.

In general, cancer incidence increases with age and remains fairly low among both genders until about age 30 years when it starts to increase gradually. Around age 55 years, cancer in both genders begins to increase dramatically and cancer among males becomes noticeably more prevalent than cancer in females.

Table 25 below illustrates the cancer incidence in Alberta in 2005 for all cancers by age group and gender—shown as the age-standardized incidence rate per 100,000 people. The figure points to the relatively low rate of cancer among young Albertans, and the escalating rates among older age groups, and it indicates that female cancer rates exceed male rates from ages 25 to 55, after which male rates exceed female rates.

These differential rates have important implications for the allocation of cancer costs by gender and age. While total cancer costs rise with age, indirect productivity losses due to premature death are greater on a per capita basis for young and female Albertans because more years of productive life are lost at early ages by these groups.

Table 25. Cancer incidence by age group, all cancer sites, age-standardized incidence rate per 100,000, Alberta, 2005

Age group	0-4	5-9	10-14	15-19	20-24	25-29	30-34
Males	23.5	18.8	9.5	24.7	35.9	46.6	49.9
Females	8.2	7.0	9.1	17.3	30.4	68.0	103.2
Both	16.0	13.1	9.3	21.1	33.2	57.3	76.4

Age group	35-39	40-44	45-49	50-54	55-59	60-64	65-69
Males	79.5	128.3	223.0	402.7	821.5	1249.8	1933.2
Females	135.7	249.0	356.3	535.0	686.5	1046.5	1249.6
Both	107.7	189.2	289.0	467.7	754.7	1148.5	1586.2

Age group	70-74	75-79	80-84	85–89	90+
Males	2410.7	2810.2	2924.6	3153.5	2774.9
Females	1566.5	1735.6	1916.6	2025.5	1642.3
Both	1973.5	2222.7	2317.8	2409.1	1962.1

Note: The above sites exclude nonmelanoma skin cancer, as do data from the Alberta Cancer Registry.

Source: Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics* Alberta Health Services, Alberta Cancer Board, 2008; accessed July 2008; available from http://www.cancerboard.ab.ca/NR/rdonlyres/2BADD738-9C4D-487F-B8C1-68D5D962CD81/0/AlbertaCancerRegistry200531Oct08.pdf.

It is not possible to use CCHS data to estimate relative risk ratios (RRs) for the association

between specific types of cancers and obesity. The 2004 CCHS, cycle 2.2, and 2005 CCHS, cycle 3.1, ask respondents whether or not they have been diagnosed with cancer by a health professional. The specific question is as follows: "We are interested in 'long-term conditions' which are expected to last or have already lasted 6 months or more and that have been diagnosed by a health professional… Do you have cancer?").

However, CCHS cycle 2.2 does not distinguish between different types of cancer,[753] and CCHS cycle 3.1 asks a few follow-up questions concerning the history of the cancer, but only for a few types of cancer: "Have you ever been diagnosed with cancer?"; "What type of cancer do/did you have? Breast (and prostate for men), Colorectal, Skin – Melanoma, Skin - Non-melanoma, Other."[754] Without information on the many specific cancer types linked to obesity, it is therefore not possible to use these data to estimate obesity-related RRs. Also, it is not recommended to use ORs to estimate PAFs when all of the data used to estimate the ORs are not available, which are needed to estimate RRs from ORs.[755]

Therefore, we have used the PAFs estimated by Pan et al. for 9 of the types of obesity-related cancers investigated in this report. This methodology is explained in detail in Part 2, Chapter 5.4.3.

4.5.15.1 Prevalence of cancer in Alberta

Although prevalence of cancer data are not used in this report to estimate the costs of obesity, the following is included for illustrative purposes. A costing study must account both for the significant number of premature deaths attributable to cancer—since these produce productivity losses to society—and for the ongoing care costs associated with living with cancer. Indeed, cancer cost studies have found that most of the costs of cancer occur in the first year of diagnosis and in the last year of life.[756, 757] However, as Statistics Canada notes below, cancer survivors may have continuing needs for cancer care resources and support services, the provision of which, in turn, has significant economic implications. According to Statistics Canada:

> Prevalence is a useful indicator of the burden cancer poses both at the personal level and at the level of the health care system. Although many individuals who survive cancer continue to live productive and rewarding lives, the cancer experience is difficult and presents many physical, emotional and spiritual challenges to patients and to their families and loved ones. These

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[753] Statistics Canada. *Canadian Community Health Survey (CCHS) Cycle 2.2 Nutrition Questionnaire*, 2004; accessed September 2008; available from http://www.statcan.ca/english/sdds/instrument/5049_Q1_V1_E.pdf.
[754] Statistics Canada. *Canadian Community Health Survey, Cycle 3.1, Final Questionnaire*, 2005; accessed September 2008; available from http://www.statcan.ca/english/sdds/instrument/3226_Q1_V3_E.pdf.
[755] Zhang, and Yu. "What's the Relative Risk? A Method of Correcting the Odds Ratio in Cohort Studies of Common Outcomes."
[756] Longo, Christopher J., Margaret Fitch, Raisa B. Deber, and A. Paul Williams. "Financial and Family Burden Associated with Cancer Treatment in Ontario, Canada," *Support Care Cancer*, 2006, vol. 14: 1077-1085
[757] Yabroff, K. Robin, William W. Davis, Elizabeth B. Lamont, Angela Fahey, Marie Topor, Martin L. Brown, and Joan L. Warren. "Patient Time Costs Associated with Cancer Care," *Journal of the National Cancer Institute*, 99, 2007, no. 14-23.

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challenges may persist beyond the point of physical recovery from the cancer itself, often requiring extensive use of rehabilitation and supportive care resources. A large number of Canadians live with the effects of cancer, require repeated active treatment and have continuing needs for cancer care resources and support services. This increased demand and the complexity of survivors' health needs must be considered in the planning and development of interdisciplinary health services.[758]

Statistics Canada makes a distinction between "complete prevalence" of cancer, which refers to those currently alive who have ever been diagnosed with cancer and which it does not report, and "limited-duration prevalence," which Statistics Canada does report in its annual cancer statistics for Canada for female breast, prostate, colorectal, lung, and other cancers. However, Statistics Canada does not report the limited-duration prevalence rates for provinces.[759] Cancer limited-duration prevalence data are reported as "the number of living Canadians who have received a diagnosis of cancer some time during the past 15 years."[760] For example, for 2005, the limited duration prevalence rate would be the number of patients alive in 2005 who had received a diagnosis of cancer in the 15-year period between 1990 and 2005.[761]

Because Statistics Canada does not have national prevalence data, it derives its 15-year prevalence rates for Canada indirectly by applying survival rate data from the Saskatchewan Cancer Registry for the period 1986 to 2001 to the Canadian incidence data.[762] This method assumes that cancer survival rates in Saskatchewan are representative of those for Canada.[763] According to Statistics Canada, there has been some improvement in cancer survival rates since 2001—the last year for which the Saskatchewan survival rate data are available—, and so the general Canadian limited-duration prevalence rates provided by the agency in 2008 for 2004 are likely to err on the low side. Statistics Canada also observes that it is not possible to obtain prevalence data or data for specific types of cancer from CCHS.[764]

According to Statistics Canada, the estimated 15-year prevalence of cancer in the Canadian population for the year 2004 was 2.7% of the population—2.5% of men and 2.8% of women— which marks a 21% overall increase from the rate reported for 1998.[765] The increase is attributed both to an increase in new cancer cases and to improved survival prospects. One percent of females in the Canadian population are survivors of breast cancer, and 0.8% of the male population are survivors of prostate cancer. Relative survival ratios are highest for thyroid, testicular, and prostate cancers and for melanoma, and lowest for pancreatic, esophageal, lung,

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[758] Statistics Canada. *Canadian Cancer Statistics 2008*, accessed. pp. 53-54.

[759] Statistics Canada. *Canadian Cancer Statistics 2008*, Canadian Cancer Society, National Cancer Institute of Canada, Statistics Canada, Provincial/Territorial Cancer Registries, Public Health Agency of Canada, 2008; accessed November 2008; available from http://www.cancer.ca/Canada-wide/About%20cancer/Cancer%20statistics/Canadian%20Cancer%20Statistics.aspx?sc_lang=en.

[760] Marrett, Loraine D., Prithwish De, Parisa Airia, and Dagny Dryer for the steering committee of Canadian Cancer Statistics 2008. "Cancer in Canada in 2008," *Canadian Medical Association Journal*, 2008, vol. 179, no. 11 - November 18: 1163-1170. p. 1163.

[761] Ibid. p. 1163.

[762] Statistics Canada. *Canadian Cancer Statistics 2008*, accessed.

[763] Ibid., accessed.

[764] Ibid., accessed.

[765] Ibid., accessed.

and liver cancers.[766]

Prevalence rates for specific cancers are not provided on the Alberta Cancer Board (ACB) website or in the available ACB reports.[767] For the purposes of this particular study, however, ACB did provide GPI Atlantic with a special tabulation of the prevalence estimates for specific cancer sites in Alberta, as of January 1, 2005.[768] According to ACB, 85,364 Albertans alive on January 1, 2005 were either living with cancer or had lived with cancer at some point in their lives—an estimate therefore based on "complete" cancer prevalence statistics rather than "limited-duration" rates.[769]

In a published report, ACB notes that approximately 12,000 Albertans are diagnosed with cancer each year, and approximately 5,000 die from cancer each year, leaving an additional 7,000 Albertans living with cancer each year.[770] According to the Alberta Cancer Registry, 12,749 new cancer cases were reported in 2005, and 5,332 died from cancer in that year.[771] While some of these deaths were attributable to cancers diagnosed prior to 2005, a simple subtraction of 2005 cancer deaths from new 2005 cancer cases indicates that the Alberta cancer prevalence for 2005 as a whole was 7,417 higher than the January 1, 2005, estimate—for a total of 92,781 cases.

It is important to differentiate cancer prevalence between that occurring in children and that occurring in adults, since premature death and disability at young ages incurs far higher social costs on a per capita basis—due to more years of productive life lost—than death and disability at older ages.

According to published and unpublished material from the Alberta Cancer Board, as of January 1, 2005, there were approximately 600 Albertan children in the 0–14 year age group who had been diagnosed with cancer at some point in their young lives, and there were 80 new cases of childhood cancer occurring in 2005.[772, 773] Among Albertan adults aged 15 years and older, there were 84,764 alive on January 1, 2005, who were either still living with cancer or who had been diagnosed with cancer at some point in their lives. An additional 12,669 new adult cancer cases were diagnosed in 2005.

Table 26 below indicates in column 2 the number of Albertan adults living on January 1, 2005, who had ever been diagnosed with the specific types of invasive cancers that have been found to be partially attributable to BMI, plus the number of new cases in 2005 (column 3), and the total prevalence (excluding mortalities) for the year 2005 (column 4). The breakdown of the total

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[766] Ibid., accessed.

[767] Alberta Cancer Board. *Cancer in Alberta: A Regional Picture*, 2007; accessed July 2008; available from http://www.cancerboard.ab.ca/NR/rdonlyres/125FDB29-3F4B-413B-BDB9-B4425F9BAFE0/0/ACBRegionalReport_2007.pdf.

[768] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

[769] Ibid., personal communication,

[770] Alberta Cancer Board. *Cancer in Alberta: A Regional Picture*, accessed.

[771] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

[772] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

[773] Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* accessed.

adult cancer prevalence for 2005, including cases diagnosed prior to 2005 and the new cases diagnosed in 2005, reveals that 55,071 Albertan adults had a prior diagnosis of one of the types of cancer that can be partially attributed to BMI, and that (after 2005 cancer mortality is subtracted from new cases that year) 62,980 Albertan adults were alive at the end of 2005 who had been diagnosed with one of these obesity-related cancer types.[774]

Breaking down Alberta's total cancer prevalence for 2005 by cancer type, Table 26 below indicates that the major obesity-related cancers were prostate cancer (16,847 cases), postmenopausal breast cancer (15,116 cases), and colorectal cancer (11,052 cases). These three cancer types alone amounted to 68% of the 62,980 Alberta adult cancer cases that that were partially attributable to excess weight, and to 44% of total 2005 Alberta cancer prevalence (counting both obesity-related and non obesity-related cancers).

Table 26 also indicates the prevalence among Albertan children of those specific cancers that have been associated with obesity. Among children, non-Hodgkin's lymphoma and leukemia were by far the main types of obesity-related cancers—accounting for more than 98% of the 304 cases of such cancers and for 44% of all childhood cancer prevalence in Alberta (counting both obesity-related and non obesity-related cancers.) Thus, in 2005, there were 210 Albertan children who had previously been diagnosed with non-Hodgkin's lymphoma plus 7 new cases that were diagnosed in 2005, and there were 54 Albertan children previously diagnosed with leukemia plus 28 new cases of leukemia diagnosed in 2005.

For other obesity-related cancers, there were two cases of kidney cancer, and one each for ovary, liver, and bladder cancer among Albertan children in 2005. However, these data sizes are too small for analysis, and there is insufficient evidence to indicate whether any cases of childhood cancer in Alberta are attributable to excess weight. For that reason, childhood cancer cases have not been included in this assessment of obesity-related cancer costs in Alberta.

Many researchers have noted that the key obesity-related issue in regard to childhood health is not that children are necessarily at greatest risk of health problems while young, but rather that the chronic illnesses associated with obesity frequently take time to develop, so that the dangers they face will be in the future. Ample research has demonstrated that excess weight in childhood often leads to excess weight in adulthood, which in turn elevates the risk of developing cancers attributable to excess weight as these young people age.[775]

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[774] Wang, Mengzhe, Manager, Surveillance analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

[775] Singh, A. S., C. Mulder, J. W. R. Twisk, W. van Mechelen, and M. J. M. Chinapaw. "Tracking of Childhood Overweight into Adulthood: A Systematic Review of the Literature," *Obesity Reviews*, 2008, vol. 9: 474-488.

Table 26. Cancer prevalence by types of cancer that can be partially attributed to obesity, as of January 1, 2005, and 2005 new cases, adults ≥15 and children aged 0–14, Alberta

Cancer site	Adults ≥15			Child	Child
	Prevalence on Jan 1, 05	New cases minus mortalities - 2005	Total prevalence for 2005	Prev	New
Colorectal	9,483	1,569	11,052	0	0
Breast, postmenopausal	13,786	1,330	15,116	0	0
Endometrium	4,006	377	4,383	0	0
Kidney	2,250	325	2,575	1	1
Esophagus	182	109	291	0	0
Ovary	1,624	185	1809	0	1
Prostate	14,942	1,905	16,847	0	0
Pancreas	233	351	584	0	0
Non-Hodgkin's lymphoma	2,749	557	3,306	210	7
Leukemia	2,346	376	2,722	54	28
Multiple myeloma	485	174	659	0	0
Liver	231	166	397	0	1
Bladder	2,071	256	2,327	0	1
Stomach	683	229	912	0	0
TOTAL Obesity-related	**55,071**	**7,909**	**62,980**	**264**	**40**
Other non-obesity-related	29,693	4,760	34,453	336	40
Total	84,764	12,669	97,433	600	80

Notes: Prevalence – number of living persons diagnosed with cancer; Numbers have been rounded; child prevalence numbers are estimated from the percentages of new cancers.

Sources: **Prevalence** – Wang, Mengzhe, Manager, Surveillance Analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, November 24, 2008. **New cases** – Alberta Cancer Board. _Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics_ Alberta Health Services, Alberta Cancer Board, 2008; accessed July 2008; available from http://www.cancerboard.ab.ca/NR/rdonlyres/2BADD738-9C4D-487F-B8C1-68D5D962CD81/0/AlbertaCancerRegistry200531Oct08.pdf.

Figure 35 below shows the proportion of total 2005 cancer prevalence among Albertan adults, aged ≥15, comprised of the types of cancer that are partially attributable to BMI. These obesity-related cancers accounted for 65% of total cancer prevalence in Alberta in 2005, with prostate cancer accounting for 17% of total cancer prevalence, postmenopausal breast cancer for 15%, and colorectal cancer for 11%. Endometrial cancer accounted for 5% of total cancer prevalence in Alberta in 2005, and other obesity-related cancers each accounted for 3% or less of the total.

Figure 35. Cancer prevalence by types of cancers that can be partially attributed to BMI, as percentage of total adult cancer prevalence, adults aged ≥15, Alberta, 2005
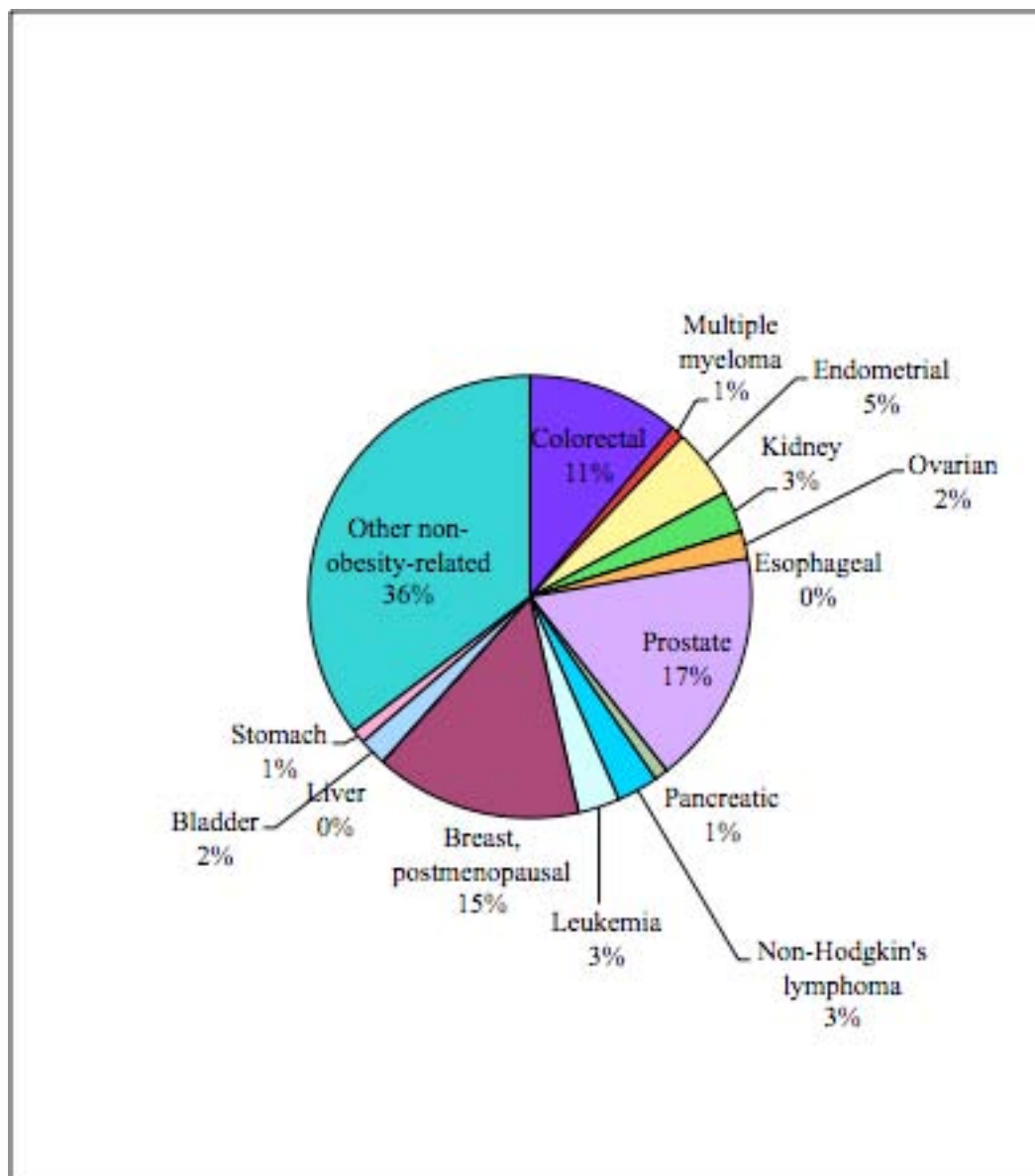
Notes: Prevalence includes the number of adults, aged ≥15 years, previously diagnosed with cancer in Alberta as of January 1, 2005, plus new cases diagnosed in 2005. Numbers have been rounded: Thus, cancer types marked as 0% actually have a percentage of 0.4% or less.

Sources: **Prevalence** – Wang, Mengzhe, Manager, Surveillance Analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, November 24, 2008. **New cases** – Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics* Alberta Health Services, Alberta Cancer Board, 2008; accessed July 2008; available from http://www.cancerboard.ab.ca/NR/rdonlyres/2BADD738-9C4D-487F-B8C1-68D5D962CD81/0/AlbertaCancerRegistry200531Oct08.pdf.

4.6 Osteoarthritis

The term "arthritis" refers to several diseases of the musculoskeletal system, of which osteoarthritis is the most common type.[776] Osteoarthritis accounts for approximately 85% of all arthritis cases, and affects over three million Canadians, or 1 in 10 individuals.[777, 778] It is a degenerative disease or joint disorder that damages and eventually destroys the cartilage of weight-bearing joints, mainly in the knees, hips, and lower back, and results in short- and long-term disability, loss of productivity, and joint-replacement surgery.[779] Although osteoarthritis can begin earlier, symptoms usually appear after age 40 and progressively worsen with age.[780] A recent study authored by French and Canadian researchers concludes that musculoskeletal diseases are one of the leading sources of disability, and represent a significant cost to society, "comparable to that of cancer."[781]

Obesity, which causes increased pressure on joints and a wearing away of protective cartilage, is one of the main, preventable risk factors for osteoarthritis.[782] James et al., noting that excess weight gain often precedes the development of osteoarthritis, explain the physical mechanisms linking excess weight with development of the disease:

> The physical burden associated with an increased load on the joints seems straightforward, but changes in movement and gravitational stresses as weight gain occurs are also a factor. Other mechanisms have, however, been invoked, including systemic changes in metabolism associated with hypertension, raised blood glucose and cholesterol concentrations, insulin resistance and elevated concentrations of blood uric acid, as well as hormonal changes induced by the metabolic effects of additional adipose tissue. Several of these factors could be acting on the metabolic integrity of the articular cartilage, as could other dietary factors, such as high fat intake, which have also been linked to this disease. The associations with hypertension tend to disappear once concomitant increased body weight is taken into account, and the link with hypercholesterolaemia is not sufficiently robust to warrant special consideration. Abnormal glucose metabolism is a more plausible mechanism, with the possible involvement of growth hormone, but epidemiological studies have not shown a consistent link between type II diabetes and osteoarthritis. Raised uric acid concentrations have been associated with osteoarthritis, but again data supporting the relationships are

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[776] Wilkins, Kathryn. "Incident Arthritis in Relation to Excess Weight," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2004, vol. 15, no. 1: 39-49.

[777] Miller, A. Lynn. *Action Plan for Arthritis*, Human Kinetics, 2003.

[778] The Arthritis Society. *Osteoarthritis*, 2008; accessed October 2008; available from http://www.arthritis.ca/types%20of%20arthritis/osteoarthritis/default.asp?s=1.

[779] House of Commons Health Committee (U.K.). *Obesity: Third Report of Session 2003-04, Annex 1: The Economic Costs of Obesity*, accessed.

[780] Wilkins. "Incident Arthritis in Relation to Excess Weight."

[781] Fautrel, Bruno, Ann E. Clarke, Francis Guillemin, Viviane Adam, Yvan St-Pierre, Tina Panaritis, Paul R. Fortin, Henri A. Menard, Cam Donaldson, and John R. Penrod. "Costs of Rheumatoid Arthritis: New Estimates from the Human Capital Method and Comparison to the Willingness-to-Pay Method," *Medical Decision Making*, Mar-Apr, 2007, 138-150.

[782] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

inconsistent. [783]

The WHO *Comparative Quantification of Health Risks* study found that in the America-A subregion (consisting of Canada, United States, and Cuba), 22% of osteoarthritis in males, and 24% in females, aged ≥30 years, could be attributed to a BMI of ≥30.[784] In one U.S. study using NHANES III data, 9.94% of females in obese class 1, 10.39% in obese class 2, and 17.19% in obese class 3 were found to have osteoarthritis, compared with 5.22% of women with normal weight.[785] Among males, 4.66% in obese class 1, 5.46% in obese class 2, and 10.04% in obese class 3 had osteoarthritis, compared with 2.59% of men with normal weight.[786]

Other studies have found that obese individuals are from 1.5 to 9.1 times more likely to have osteoarthritis than individuals with normal weight.[787] A recent Norwegian study that examined data from almost 1.2 million persons found that obese men were almost three and a half times (RR 3.4) as likely to have hip replacement surgery as men with normal weight.[788] Obese women were two and one third times (RR 2.3) as likely as normal weight women to have a hip replacement.

The Canadian Joint Replacement Registry (CJRR) notes that "[o]besity is one of the known factors associated with osteoarthritis," and that osteoarthritis is a primary reason for joint replacement surgeries.[789] CJRR reports that most of the hip and knee replacement surgeries are performed on patients in the 65 to 74 year age group, and observes:

> There is an established association between obesity and the development of osteoarthritis, and there is a window of time during which arthritis causes damage to joints requiring intervention. Those who are at an unhealthy weight at, or approaching, age 55 years can be expected then to form the predominant group of patients who will undergo joint replacements 10 years later, at or around, age 65 years. This is consistent with the long-time observed association between overweight and obesity and the need for joint replacement.[790]

According to Kathryn Wilkins of Statistics Canada: "Joint replacement, performed chiefly on people with arthritis, already accounts for sharply rising numbers of surgical procedures and hospital days of care."[791] Between 1994/95 and 2002/03, knee replacement rates in Canada

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[783] Ibid. p. 558.

[784] Ibid.

[785] Must, Spadano, and Coakley. "The Disease Burden Associated with Overweight and Obesity."

[786] Ibid.

[787] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[788] Flugsrud, Gunnar B., Lars Nordsletten, Birgitte Espehaug, Leif I. Havelin, Anders Engeland, and Haakon E. Meyer. "The Impact of Body Mass Index on Later Total Hip Arthroplasty for Primary Osteoarthritis: A Cohort Study in 1.2 Million Persons," *Arthritis and Rheumatism*, 2006, vol. 54, no. 3: 802-807.

[789] Canadian Institute for Health Information (CIHI). *Canadian Joint Replacement Registry (CJRR) 2006 Report: Total Hip and Total Knee Replacements in Canada*, 2006; accessed October 2008; available from http://secure.cihi.ca/cihiweb/products/CJRR_Annual_Report_Hip_Knee_Replacements_2006_e.pdf. p. 41.

[790] Ibid., accessed. p. 43.

[791] Wilkins. "Incident Arthritis in Relation to Excess Weight."

increased by 50%, while hip replacement rates increased by 11%.[792]

CJRR reports that in 2004-2005, there were a total of 58,714 hip and knee replacements performed in Canada.[793] Patients with knee replacements were more likely to be overweight or obese compared to hip replacement patients, and both hip and knee replacement patients were rarely underweight. According to Nicole DeGuia et al. of CIHI, among patients receiving knee replacements, 54% were obese, 33% were overweight, and only 12% had normal weight. Among those receiving hip replacements, 35% were obese, 38% were overweight, and 26% had normal weight.[794] There were no significant differences in BMI levels between males and females undergoing knee replacement surgery, but for hip replacements there was a higher proportion of overweight or obese males than females undergoing the procedure.

Among 1,458 joint replacement patients in Alberta in 2003-04, 709 or 48.6% were obese; 474 or 32.5% were overweight; and 255 or 17.5% had acceptable weights.[795] In 2003-2004 there were 2,763 hip and knee replacement procedures performed in Alberta, which rose sharply to 3,615 in 2004-2005.[796]

DeGuia et al. estimated relative risk ratios (RR) for joint replacement surgery in Canada by BMI, using 2004 CCHS data.[797] They found the risk to be RR 3.12 for obese persons and RR 1.53 for overweight persons, which indicates that obese persons were over three times more likely and overweight persons were about one and a half times more likely to undergo joint replacement surgery than those in the normal weight category.[798]

Kathryn Wilkins of Statistics Canada used data from the 2000/01 CCHS to quantify the contribution of obesity to the risk of developing arthritis (of any kind) in Canada.[799] In 2000/01, 19% of Canadian men and 31% of Canadian women, aged ≥40 years, reported having been diagnosed with arthritis. The prevalence increases with age and at ≥80 years, 40% of men and 57% of women reported having arthritis.

Wilkins found that obese individuals had higher rates of arthritis than did those of normal weight—with the odds of being diagnosed with arthritis 60% higher (OR 1.6) for obese patients

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[792] DeGuia, Nicole, Naishu Zhu, Margaret Keresteci, and Juqing Ellen Shi. "Obesity and Joint Replacement Surgery in Canada: Findings from the Canadian Joint Replacement Registry (CJRR)," *Healthcare Policy*, 2006, vol. 1, no. 3: 36-43.

[793] Canadian Institute for Health Information (CIHI). *Canadian Joint Replacement Registry (CJRR) 2006 Report: Total Hip and Total Knee Replacements in Canada*, accessed.

[794] DeGuia, Zhu, Keresteci, and Shi. "Obesity and Joint Replacement Surgery in Canada: Findings from the Canadian Joint Replacement Registry (CJRR)."

[795] Canadian Institute for Health Information (CIHI). *Table 1: BMI Category for Joint Replacement Patients, by Province and Territory, 2003–2004*, 2005; accessed October 2008; available from http://secure.cihi.ca/cihiweb/en/media_17aug2005_tab1_e.html.

[796] Canadian Institute for Health Information (CIHI). *Canadian Joint Replacement Registry (CJRR) 2006 Report: Total Hip and Total Knee Replacements in Canada*, accessed.

[797] DeGuia, Zhu, Keresteci, and Shi. "Obesity and Joint Replacement Surgery in Canada: Findings from the Canadian Joint Replacement Registry (CJRR)."

[798] Ibid.

[799] Wilkins. "Incident Arthritis in Relation to Excess Weight."

than for those in the normal, healthy weight range. However, Wilkins notes that these odds are probably lower than they would be if they were estimated on the basis of osteoarthritis alone. This is because there is little evidence that excess weight affects any form of arthritis other than osteoarthritis. Since osteoarthritis accounts for about 85% of all arthritis cases, however, Wilkins speculates that most respondents who reported arthritis probably had osteoarthritis.[800]

4.6.1 Osteoarthritis statistics in Alberta

Katzmarzyk and Janssen used a summary RR of 1.99 for the association between obesity and osteoarthritis in their study on the economic costs of obesity in Canada.[801] Birminham et al. did not include osteoarthritis in their cost of obesity in Canada study.[802]

The RRs of osteoarthritis attributable to excess weight used in this report were estimated using self-reported BMI data from the 2005 CCHS, since osteoarthritis prevalence data were not available from the 2004 CCHS. As previously noted, the self-reported BMI data were adjusted using a newly developed methodology from Statistics Canada to correct for reporting bias and more accurately align self-reported BMI rates with directly measured rates.[803]

Statistics Canada reports prevalence rates for "arthritis or rheumatism," rather than for osteoarthritis.[804] However, as noted above, osteoarthritis accounts for approximately 85% of all arthritis cases. According to Statistics Canada, 16.4% of Canadians aged 12 years and older had arthritis or rheumatism in 2005—12.5% of males and 20.1% of females.[805] This represented a very slight reduction from the 2003 rate of 16.8%—12.6% of males and 20.8% of females.[806]

As shown in Table 27 below, the 2005 prevalence of arthritis and rheumatism among Albertans aged 12 and older was 14.8%—11.3% of males and 18.4% of females.[807] This was about 10% lower than the Canadian rate, and 9% less than the 2003 Alberta rate of 16.3%—12.1% males and 20.7% females.[808] Statistics Canada also reports that in 2003, 425,560 Albertans 12 and

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[800] Ibid.

[801] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[802] Birmingham, C. Laird, Jennifer L. Muller, Anita Palepu, John J. Spinelli, and Aslam H. Anis. "The Cost of Obesity in Canada," *Canadian Medical Association Journal*, 1999, vol. 160, no. 4: 483-488.

[803] Gorber, Shields, Tremblay, and McDowell. "The Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity."

[804] Statistics Canada. *Arthritis or Rheumatism, by Age Group and Sex, Household Population Aged 12 and over, Canada, Provinces, Territories, Health Regions (June 2005 Boundaries) and Peer Groups, Every 2 Years*, Canadian Community Health Survey, CANSIM Table 105-0402, 2005.

[805] Statistics Canada. *Health Indicators*, accessed.

[806] Statistics Canada. *Health Indicators*, 2004; accessed July 2008; available from http://www.statcan.ca/english/freepub/82-221-XIE/00604/whatsnew.htm.

[807] Statistics Canada. *Health Indicators*, accessed.

[808] Statistics Canada. *Health Indicators*, accessed.

older (157,999 males and 267,561 females) had arthritis or rheumatism, which decreased by about 6% to 398,328 in 2005 (152,831 males and 245,497 females).[809]

Table 27 indicates that nearly half of Albertans 65 and older—55.4% of women and 40.5% of men—have been diagnosed with arthritis or rheumatism, with overall rates among seniors more than double those in the 45-64 year-old age group.

Alberta Health and Wellness reports that approximately 4.2% of Albertans received care related to arthritis in 2003.[810] It also notes that hospital separation rates for arthritis have declined significantly since 1979, which demonstrates that the condition is now much better managed and that "there has been a move away from inpatient care."[811]

Table 27. Arthritis and rheumatism prevalence, by age group and sex, Alberta, CCHS, 2005

Age Group	Population	No. & %	Both	Males	Females
12 and over	Total pop.	# persons	2,686,120	1,351,451	1,334,669
		percent	100.0	100.0	100.0
	with arthritis	# persons	398,328	152,831	245,497
		percent	14.8	11.3	18.4
	without arthritis	# persons	2,285,150	1,196,437	1,088,713
		percent	85.1	88.5	81.6
15-19	Total pop.	# persons	209,694	105,205	104,489
		percent	100.0	100.0	100.0
	with arthritis	# persons	3,022^E	F	F
		percent	1.4^E	F	F
	without arthritis	# persons	206,673	104,570	102,103
		percent	98.6	99.4	97.7
20-34	Total pop.	# persons	710,849	363,093	347,756
		percent	100.0	100.0	100.0
	with arthritis	# persons	24,907	12,631^E	12,276
		percent	3.5	3.5^E	3.5
	without arthritis	# persons	685,851	350,462	335,389
		percent	96.5	96.5	96.4
35-44	Total pop.	# persons	513,940	263,331	250,609
		percent	100.0	100.0	100.0
	with arthritis	# persons	43,518	21,099^E	22,419
		percent	8.5	8.0^E	8.9

[809] Statistics Canada, Catalogue no 82-221-X, CANSIM Tables 105-0202 and 105-0402.
[810] Alberta Health and Wellness. *Report on the Health of Albertans*, accessed.
[811] Ibid., accessed. p. 102.

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	without arthritis	# persons	470,370	242,180	228,190
		percent	91.5	92.0	91.1
45-64	Total pop.	# persons	785,752	396,708	389,045
		percent	100.0	100.0	100.0
	with arthritis	# persons	172,990	59,484	113,506
		percent	22.0	15.0	29.2
	without arthritis	# persons	611,892	336,353	275,539
		percent	77.9	84.8	70.8
65+	Total pop.	# persons	314,812	144,013	170,880
		percent	100.0	100.0	100.0
	with arthritis	# persons	152,967	58,312	94,656
		percent	48.6	40.5	55.4
	without arthritis	# persons	160,357	84,440	75,917
		percent	50.9	58.6	44.4

Notes: E - use with caution (coefficient of variation between 16.6% and 33.3%); F - too unreliable to be published (coefficient of variation >33.3%).
Source: Statistics Canada. *Arthritis or Rheumatism, by Age Group and Sex, Household Population Aged 12 and over, Canada, Provinces, Territories, Health Regions (June 2005 Boundaries) and Peer Groups, Every 2 Years*, Canadian Community Health Survey, CANSIM Table 105-0402, 2005.

The prevalence of osteoarthritis in the Canadian population 15 years of age and over based on 2005 CCHS cycle 3.1 data was 8.4% among obese males and 19.2% among obese females in 2005.

Comparing the obese population (i.e. the exposed population) (BMI ≥30) to the normal weight population (i.e. the unexposed) (BMI 18.5–24.9), the age-adjusted prevalence-based risk ratio for osteoarthritis was 1.8 for males and 1.7 for females, aged ≥15.

The portion (population attributable fraction–PAF) of osteoarthritis that can be attributed to obesity used in this report was 14.2% for males and 12.8% for females.

Applying these fractions to the number of cases of osteoarthritis in Alberta in 2005 shows that, for adults aged ≥20 years, 21,517 cases in males and 31,086 cases in females could be attributed to obesity.

4.7 Gallbladder disease

Gallbladder disease is generally defined as finding the presence of gallstones on ultrasound examinations of patients, or as requiring the removal of the gallbladder, which is called a cholecystectomy.[812] Gallbladder disease is reported to be one of the most costly and common digestive diseases requiring hospitalization—either for removal of gallstones or removal of the gallbladder.[813] According to Williams, cholecystectomy, which is one of the most common surgeries performed, is also one of the most costly digestive disease surgeries requiring hospitalization, although with laparoscopic cholecystectomy surgery, hospitalization is often not required.[814]

Laparoscopic surgeon Arun Prasad notes that the gallbladder stores bile that is secreted by the liver, which it then ejects into the small intestine to help make fats in the intestine soluble before digestion.[815] However, he comments that the gallbladder is not essential to human digestion, that many animals such as horses, rats, and pigeons do not have this organ, and that its importance is "something of a mystery."[816] The liver also secretes cholesterol and bilirubin into the bile, which both can crystallize to form stones in the gallbladder, and according to Dr. Prasad, this "explains almost all of the ills inflicted by this organ."[817]

Douglas Olsen, bariatric surgeon and medical director of the Centennial Center for the Treatment of Obesity, reports that although cholecystectomy is one of the most common surgeries in the U.S, two-thirds of adults who develop gallstones do not have symptoms and do not require surgery.[818] It is often reported that about 20 million Americans have gallstones, but only 1–3% of the population develop symptoms.[819] Women are more at risk for gallbladder disease than are men because estrogen stimulates the liver to divert more cholesterol into the bile, and the use of hormone replacement therapy doubles or triples the risk for gallstones.[820]

Writing in a 2006 issue of *The Lancet,* Italian physicians Piero Portincasa et al. note that more than one million people annually in the U.S. are diagnosed with gallstones, and about 700,000 adults have cholecystectomies. However, the data cited in that source come from an article

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[812] Everhart, James E., Meena Khare, Michael Hill, and Kurt R. Maurer. "Prevalence and Ethnic Differences in Gallbladder Disease in the United States," *Gastroenterology*, 1999, vol. 117: 632-639.
[813] Williams, Paul T. "Independent Effects of Cardiorespiratory Fitness, Vigorous Physical Activity, and Body Mass Index on Clinical Gallbladder Disease Risk," *American Journal of Gastroenterology*, 2008, vol. 103: 2239-2247.
[814] Ibid.
[815] Prasad, Arun. *Gallbladder Disease - Is Surgery Always Needed?* Apollo Hospital, New Delhi, n.d.; accessed October 2008; available from http://www.angelfire.com/ar/laparoscopy/gallstone.html.
[816] Ibid., accessed.
[817] Ibid., accessed.
[818] Olsen, Douglas O. *Gallbladder Disease and the Obese Patient*, Obesity Action Coalition, Centennial Center for the Treatment of Obesity, 2008; accessed October 2008; available from http://www.obesityaction.org/magazine/oacnews9/obesityrelateddiseases.php.
[819] University of Maryland Medical Center. *Gallstones and Gallbladder Disease*, 2008; accessed October 2008; available from http://www.umm.edu/patiented/articles/what_symptoms_of_gallstones_gallbladder_disease_000010_2.htm.
[820] Ibid., accessed.

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published in 1993.[821] In 2005, Eldon Shaffer of the University of Calgary reported the identical annual number of cholecystectomies (700,000) as occurring in the U.S., and also estimates that the cost of "gallstone disease" is approximately $6.5 billion dollars annually in the U.S.[822]

Because the population is aging in Western countries and the incidence of gallbladder disease is highest in the population aged 60 and over, and because of increasing rates of obesity, which is a key risk factor for gallbladder disease, the number of gallstone patients is increasing in Western countries.[823] Lammert and Juan-Francisco Miquel report that in Europe, between 10–20% of the population have gallstones, and more than 40% of patients aged 40 years and over develop severe complications.[824]

In order to obtain information on the prevalence of gallbladder disease in the United States, the Third National Health and Nutrition Examination Survey (NHANES III) incorporated a gallbladder ultrasonography into its physiological test procedures that took place between 1988 and 1994.[825] This is still the major U.S. source for widely reported gallbladder disease prevalence statistics.

According to James Everhart of the National Institute of Diabetes and Digestive and Kidney Diseases et al., over 14,000 U.S. adults aged 20–74 years were given an ultrasonography of the gallbladder in medical examination centres, along with other physical measurements and physiological tests.[826] The final nationally representative sample for analysis included 14,238 adults, of whom 1,149 had gallstones and 886 had undergone cholecystectomies—both indicating the presence of gallbladder disease.

Table 28 below shows the results of the Everhart et al. analysis of the data, by sex and age group, for the prevalence of gallbladder disease in general, for gallstones, and for cholecystectomies.[827] Results show that about 20.5 million American adults aged 20-74 had gallbladder disease, with rates more than twice as high among women as among men—with 7.9% of American men (6.3 million) and 16.6% of American women (14.2 million) estimated to have the disease. Among men, 5.5% had gallstones, and 2.4% had undergone a cholecystectomy; and among women 8.6% had gallstones, and 8.0% had undergone a cholecystectomy.

Gallbladder disease was also found to be considerably more prevalent among older than younger Americans, though women were relatively more likely to be afflicted at younger ages than men.

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[821] National Institutes of Health. "National Institutes of Health Consensus Development Conference Statement on Gallstones and Laparoscopic Cholecystectomy." *American Journal of Surgery*, 1993, vol. 165: 390-398. Cited in Portincasa, Piero, Antonio Moschetta, and Giuseppe Palasciano. "Cholesterol Gallstone Disease," *The Lancet*, 2006, vol. 368, no. July 15: 230-234. p. 230.

[822] Shaffer, Eldon A. "Epidemiology and Risk Factors for Gallstone Disease: Has the Paradigm Changed in the 21st Century?" *Current Gastroenterology Reports*, 2005, vol. 7: 132-140.

[823] Lammert, Frank, and Juan-Francisco Miquel. "Gallstone Disease: From Genes to Evidence-Based Therapy," *Journal of Hepatology*, 2008, vol. 48: S124-S135.

[824] Ibid.

[825] Everhart, Khare, Hill, and Maurer. "Prevalence and Ethnic Differences in Gallbladder Disease in the United States."

[826] Ibid.

[827] Ibid.

Thus, within the sample of 20-74 year-olds, 51% of male prevalence and 43% of female prevalence was in the 60–74 year age group. Among men aged 60–74 years, 17.2% had gallstones, 8.1% had undergone a cholecystectomy, and 25.3% had gallbladder disease; and among women aged 60–74 years, 16.4% had gallstones, 16.7% had undergone a cholecystectomy, and 33.1% had gallbladder disease.[828]

Everhart et al. note that the prevalence of gallbladder disease would have been higher if persons older than 74 years and persons in institutions had been included in the study sample. As well, the age breakdown proportions would have been even more strongly skewed to older age groups.

Table 28. Prevalence (and 95% CI) of gallstones, cholecystectomies, and gallbladder disease, by sex and age group, United States, 1988–1994

Sex, age group (yr)	Sample size	Gallstones		Cholecystectomy		Gallbladder disease		No. (millions)
		Prevalence (%)	95% CI	Prevalence (%)	95% CI	Prevalence (%)	95% CI	
Men								
Total	6688	5.5	4.7 – 6.4	2.4	1.8 – 3.1	7.9	6.7 – 9.1	6.3
20–29	1602	1.3	0.4 – 2.1	0.0	0.0 – 0.2	1.3	0.4 – 2.1	0.3
30–39	1426	1.1	0.1 – 2.2	0.8	0.2 – 1.4	1.9	0.8 – 3.1	0.4
40–49	1193	5.9	3.3 – 8.5	1.5	0.3 – 2.6	7.3	4.5 – 10.1	1.2
50–59	831	7.3	4.6 – 10.0	4.4	2.1 – 6.7	11.7	8.4 – 15.0	1.2
60–74	1636	17.2	14.8 – 19.7	8.1	6.2 – 10.0	25.3	21.8–28.8	3.2
Women								
Total	7550	8.6	7.4 – 9.7	8.0	7.2 – 8.9	16.6	15.0 - 18.1	12.2
20–29	1818	4.4	2.9 – 6.0	2.1	0.9 – 3.2	6.5	4.6 - 8.4	1.3
30–39	1805	5.2	3.3 – 7.2	4.9	3.3 – 6.6	10.2	7.6 - 12.7	2.2
40–49	1327	8.2	5.9 – 10.4	7.5	5.6 – 9.5	15.7	12.9–18.5	2.6
50–59	981	11.9	8.8 – 14.9	13.1	10.6–15.7	25	21.2–28.7	2.9
60–74	1619	16.4	14.2 – 18.6	16.7	14.3–19.2	33.1	29.7–36.5	5.2

Source: Everhart, James E., Meena Khare, Michael Hill, and Kurt R. Maurer. "Prevalence and Ethnic Differences in Gallbladder Disease in the United States," *Gastroenterology*, 1999, vol. 117: 632-639.

There is a strong association between gallbladder disease, especially gallstones, and obesity, mainly because the liver over-produces cholesterol in obese individuals. Thus, approximately one fourth of obese adults develop gallstones, which consist mainly of cholesterol, and often require surgery.[829] According to Haslam and James, this is mainly "due to supersaturation of bile

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[828] Ibid.

[829] University of Maryland Medical Center. *Gallstones and Gallbladder Disease*, accessed.

with cholesterol," which is often seen in situations of rapid weight loss that can exacerbate gallbladder disease.[830] For example, after obesity bariatric surgery, which reduces stomach size and severely limits the amount of food people can consume, gallstone formation has been seen to affect 38% of patients.

In general, studies of gallbladder disease have found that people with obesity are from 1.6 times to 4.8 times more likely to develop gallbladder disease than are people with normal weight.[831] Haslam and James note: "In women, the risk is three times higher with BMI of 32.0 kg/m^2 or above and seven times higher with BMI of 45.0 kg/m^2 or above than in those with lower BMI."[832] U.K. researcher Peter Kopelman confirms these excess risk rates compared with normal weight women.[833]

Paul Williams, of the Ernest Orlando Lawrence Berkeley National Laboratory in California, recently studied the effects of physical activity and BMI on physician-diagnosed gallbladder disease risk in a cohort of 29,110 men and 11,953 women who were surveyed first between 1991 and 1994, and then followed up between 1999 and 2002.[834] He found that the risk for gallbladder disease increased progressively for both males and females with increases in BMI, and that the risk accelerated sharply after a BMI ≥27.[835]

Alviva Must, of Tufts University, et al., who used NHANES III data to estimate the disease burden associated with obesity in adults aged 25 and older, found that among females 15.99% in obese class 1, 19.15% in obese class 2, and 23.45% in obese class 3 had gallbladder disease, compared with 6.29% with normal weight.[836] Among males, 5.38% in obese class 1, 5.80% in obese class 2, and 10.17% in obese class 3 had gallbladder disease, compared with 1.93% with normal weight.[837] The percentages in this case represent the prevalence of gallbladder disease, rather than the percentage of cases that could be attributed to obesity.

U.S. researchers Constance Ruhl and James Everhart also used NHANES III data to study the relationship between gallbladder disease and BMI in adults, aged 20–74 years.[838] They divided BMI rates into five quintiles and compared gallbladder disease prevalence in the lowest and highest BMI groups. Results showed ORs of 4.0 for females and 2.6 for men in the highest quintile compared with the lowest.[839]

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[830] Haslam, and James. "Obesity."
[831] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."
[832] Haslam, and James. "Obesity."
[833] Kopelman. "Health Risks Associated with Overweight and Obesity."
[834] Williams. "Independent Effects of Cardiorespiratory Fitness, Vigorous Physical Activity, and Body Mass Index on Clinical Gallbladder Disease Risk."
[835] Ibid.
[836] Must, Spadano, and Coakley. "The Disease Burden Associated with Overweight and Obesity."
[837] Ibid.
[838] Ruhl, Constance E., and James E. Everhart. "Relationship of Serum Leptin Concentration and Other Measures of Adiposity with Gallbladder Disease." *Heptology*, 2001, vol. 34, no. 5: 877-883.
[839] Ibid.

The Atherosclerosis Risk in Communities Study in the U.S., which involved more than 13,000 adults, aged 45–64 years, found an independent relationship between obesity and gallbladder disease.[840] The risk of hospitalization for gallbladder disease increased with increasing BMI, especially among women. For women, the RRs of being hospitalized with gallbladder disease rose progressively by BMI—i.e. from overweight (RR 1.45), obese class 1 (RR 1.58), to obese class 2–severe obesity (RR 1.80), to obese class 3–very severe obesity (RR 2.48), compared with normal weight women. Among men, however, only those in obese classes 2 and 3 (RRs 2.16 and 2.02, respectively) had elevated risks of being hospitalized with gallbladder disease.

Fields et al. estimated the risk of gallbladder disease by BMI category for middle-aged adults in a 10-year follow-up study of the Nurses Health Study and in the Health Professionals Follow-up Study in the U.S.[841] Compared with respondents with normal weight, the odds of developing gallbladder disease were 1.9, 2.5, and 3.0 for women who were overweight, obese (class 1), and severely obese (classes 2–3: BMI ≥35), respectively. For men who were overweight, obese, and severely obese, the odds of developing gallbladder disease were 1.4, 2.3, and 2.9, respectively.[842]

4.7.1 Gallbladder disease statistics in Alberta

Katzmarzyk and Janssen used a summary RR of 3.33 and a PAF of 25.5 for gallbladder disease in their report on the economic costs of obesity in Canada.[843] Luo et al. of PHAC used the same RR and obesity prevalence data from the 2004 CCHS, cycle 2.2, and found that 35.24% of gallbladder disease in Canadian men and 35.68% in Canadian women could be attributed to obesity.[844] Both of these studies, however, only included adults aged 20–64, but as noted above the incidence of gallbladder disease is highest in the population aged 60 and over.

According to Diane Kelsall, of the Institute for Clinical Evaluative Sciences (ICES), gallstone disease is a common problem in Canada, where about 20% of men and 33% of women will eventually develop gallstones, which are more common with increasing age.[845] She notes that although over two-thirds of gallstones do not produce symptoms, the widespread use of ultrasonography, which can detect very small stones, has resulted in an increased diagnosis of

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[840] Boland, Lori L., Aaron R. Folsom, and Wayne D. Rosamond. "Hyperinsulinemia, Dyslipidemia, and Obesity as Risk Factors for Hospitalized Gallbladder Disease: A Prospective Study," *Annals of Epidemiology*, 2002, vol. 12: 131-140.
[841] Field, Coakley, Must, Spadano, Laird, Dietz, Rimm, and Colditz. "Impact of Overweight on the Risk of Developing Common Chronic Diseases During a 10-Year Period."
[842] Ibid.
[843] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."
[844] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."
[845] Kelsall, Diane L. "Editor's Letter. What Galls You? Treatment Options in Gallstone Disease," *Informed*, Institute for Clinical Evaluative Sciences (ICES), 2005, vol. 11, no. 4: 1, 3.

asymptomatic gallstones. Symptoms or complications of gallstones, of which the mildest form is called biliary colic, occur in only 1–2% of persons who have gallstones each year.[846]

Prevalence rates for gallbladder disease are not readily available in Canada. However, Statistics Canada provides data on deaths from "cholelithiasis [presence of gallstones] and other disorders of gallbladder," which are relatively rare in Canada.[847]

In Canada, there were 337 deaths related to gallbladder disorders—157 male and 180 female—in 2004, the last year for which data available.[848] These deaths represent an age-standardized mortality rate of 1.1 per 100,000 population for both sexes—1.0 for males, and 1.1 for females. The vast majority of these deaths (84%) occurred in individuals aged 70 years and over. In 2004, twenty-two of the deaths (6.5%) occurred in adults under the age of 60 years. Between ages 60 and 69, there were 31 deaths (9%), between ages 70 and 79 there were 84 deaths (25%), between ages 80 and 89 there were 134 deaths (40%), and in adults aged 90 and over there were 66 deaths (20%) from gallbladder disorders in Canada.[849]

In Alberta there were 21 deaths related to gallbladder disorders in 2004—11 male and 10 female.[850] These deaths represent an age-standardized mortality rate of 0.6 per 100,000 population for both sexes—0.8 for males, and 0.5 for females. Statistics Canada did not stratify the provincial deaths by age group.

In 2005, David Urback and Therese Stukel of ICES and the University of Toronto used a number of administrative databases to find the changing rate of severe gallstone disease (separate from cholecystectomy patients) and elective cholecystectomy in Ontario between 1988 and 2000.[851] They found that the average annual rate of severe gallstone disease incidence in Ontario decreased from 127.8 per 100,000 population in 1988–1991 to 114.2 in the years 1992–2000. During the same time periods, the age-adjusted annual rate of elective cholecystectomy per 100,000 population in Ontario increased from 201.3 to 260.8. As seen below, this cholecystectomy rate estimate for 1992-2000 is considerably lower than that found by Tepper et al. for Ontario in 1999-2000.

If we assume that the rate of 114.2 per 100,000 population that Urback and Stukel found for severe gallstone disease in Ontario in 1992-2000 could be applied to the 2005 Alberta population of 3,222,191,[852] we might estimate that about 3,680 Albertans a year may suffer from severe gallbladder disease. If the elective cholecystectomy rate of 260.8 for Ontario held for Alberta,

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[846] Urbach, David R., and Therese A. Stukel. "Rate of Elective Cholecystectomy and the Incidence of Severe Gallstone Disease," *Canadian Medical Association Journal*, 2005, vol. 172, no. 8: 1015-1019.

[847] Statistics Canada. *Deaths, by Selected Grouped Causes and Sex, Canada, Provinces and Territories, Annual*, CANSIM Table 102-0552, 2007. ICD-9 diagnosis codes for gallbladder disease include 574 – Cholelithiasis, and 575 – Other disorders of the gallbladder.

[848] Ibid.

[849] Percentages are rounded.

[850] Statistics Canada. *Deaths, by Selected Grouped Causes and Sex, Canada, Provinces and Territories, Annual*.

[851] Urbach, and Stukel. "Rate of Elective Cholecystectomy and the Incidence of Severe Gallstone Disease."

[852] Alberta Health and Wellness. *Population Projections for Alberta and Its Health Regions 2006 to 2035*, Health Surveillance and Environmental Health Branch, 2007; accessed July 2008; available from http://www.health.alberta.ca/resources/publications/PopProj_06.pdf.

then about 8,400 Albertans a year might be undergoing this surgery.[853] As seen below, this is again considerably lower than the 1999-2000 cholecystectomy rate found by Tepper et al. for Alberta, but higher than the rate reported by Alberta Health and Wellness for 2003.

Kesall notes that after the introduction of laparascopic cholecystectomy in Canada in 1991—which has almost replaced open cholecystectomy—the rate of elective cholecystectomies increased by about 30% between 1992 and 2000.[854] The use of laparascopic cholecystectomy increased from less than 1% of cholecystectomy surgeries before 1990, to 31.6% in 1991, to 91.7% in 2000.[855] The surgeries have resulted in an overall 10% reduction in the incidence of severe gallstone disease, mainly because gallbladders were removed before the disease reached the acute stage.

According to Urback and Stukel: "Without cholecystectomy, nearly 50% of patients with biliary colic experience escalation of their symptoms, and 8% experience severe acute cholecystitis."[856] Biliary colic is the first major symptom of gallbladder disease and results in steady pain that usually resolves in between one to five hours, or can advance to acute cholecystitis, which is a severe inflammation.[857]

Utilization rates for cholecystectomy and 7 other common surgical procedures in rural and urban Alberta and Ontario in 1999/2000 were recently estimated by Joshua Tepper of ICES and colleagues at Alberta Health and Wellness and other institutions.[858] The Alberta data were gathered from the Discharge Abstract Database and the Alberta Health Insurance Plan database. Tepper et al. found that cholecystectomy utilization rates were significantly greater for rural than for urban adults in both Alberta and Ontario, and that rates for both were lower in Alberta than in Ontario. (The authors speculate that one reason for the rural / urban disparity could be that urban populations might have less access to common low-complexity procedures than rural populations, in part because large urban hospitals utilize competing complex province-wide surgical services that may take priority.[859])

Table 29 below shows the urban and rural cholecystectomy utilization rates in Alberta and Ontario in 1999/00, as estimated by Tepper et al., reported as the number of procedures per 100,000 population for adults aged ≥20 years. The Alberta rural and urban rates per 100,000 adults were 362 and 298, respectively, which are both lower than the Ontario rural and urban rates of 439 and 330, respectively.[860]

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[853] Ibid., accessed.
[854] Kelsall. "Editor's Letter. What Galls You? Treatment Options in Gallstone Disease."
[855] Urbach, and Stukel. "Rate of Elective Cholecystectomy and the Incidence of Severe Gallstone Disease."
[856] Ibid.
[857] Portincasa, Moschetta, and Palasciano. "Cholesterol Gallstone Disease."
[858] Tepper, Joshua, William Pollett, Yan Jin, Erik Ellehoj, Don Schopflocher, Brendan Barrett, and Stuart Iglesias. "Utilization Rates for Surgical Procedures in Rural and Urban Canada," *Canadian Journal of Rural Medicine*, 2006, vol. 11, no. 3: 195-203.
[859] Ibid.
[860] Ibid.

Table 29. Urban and rural cholecystectomy utilization rates in Alberta and Ontario in 1999/00, number of procedures per 100,000 population, adults aged ≥20 years

Procedure	Alberta		Ontario	
	Rural	Urban	Rural	Urban
Cholecystectomy	362 (344–380)	298 (289–306)	439 (413–466)	330 (317–342)

Note: Confidence Interval = 99.17%.

Source: Adapted from Tepper, Joshua, William Pollett, Yan Jin, Erik Ellehoj, Don Schopflocher, Brendan Barrett, and Stuart Iglesias. "Utilization Rates for Surgical Procedures in Rural and Urban Canada," *Canadian Journal of Rural Medicine*, 2006, vol. 11, no. 3: 195-203.

According to Alberta Health and Wellness, there were 7,048 cholecystectomy surgeries performed in Alberta in 2003. The crude rate for the surgery was 225.6 per 100,000 population, which is lower than both the rural and urban rates found by Tepper et al. and shown in Table 29 above.[861] Extrapolating to the 2005 Alberta population of 3,222,191 and using Alberta Health and Wellness' 225.6/100,000 crude cholecystectomy rate for 2003,[862] it can be estimated that approximately 7,269 cholecystectomies were performed in Alberta in 2005. Interestingly—by comparison with the 8,400 estimate extrapolated from Urback and Stukel's Ontario estimate above—this 7,269 Alberta estimate is almost precisely proportional to the difference between the Ontario and Alberta cholecystectomy rates found by Tepper et al. above.

This report used RRs as estimated by Must et al. for both genders aged <55 and ≥55 for overweight and obese classes 1–3 and obesity prevalence data for Alberta from 2004 CCHS cycle 2.2.[863] Approximately 67.3% of gallbladder disease among males and 38.3% among females could be attributed to total obesity.

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[861] Alberta Health and Wellness. *Table H-3: Cholestectomy Procedures and Rates Per 100,000 Population* 2003; accessed November 2008; available from http://www.health.alberta.ca/regions/h3.htm.
[862] Alberta Health and Wellness. *Population Projections for Alberta and Its Health Regions 2006 to 2035*, accessed.
[863] Must, Spadano, and Coakley. "The Disease Burden Associated with Overweight and Obesity."

4.8 Asthma

According to the Public Health Agency of Canada (PHAC), asthma is a chronic respiratory disorder that has symptoms of cough, shortness of breath, chest tightness, and wheezing that is characterized by periodic attacks of severe shortness of breath.[864] Attacks can be triggered by exposures to allergens, irritant fumes or gases, or viral respiratory infections that inflame the airway wall and narrow the airways.[865] PHAC notes: "An asthma attack can be a frightening event, with feelings of suffocation, breathlessness, and loss of control; it can also be life-threatening."[866] Aaron et al. note that obesity can increase the prevalence of asthma because it "decreases chest wall compliance, which results in reduced lung volumes, increased work of breathing and increased energy and oxygen costs of breathing."[867]

The Global Initiative for Asthma (GINA) 2004 study on the global burden of asthma—defined as "wheezing in the last 12 months"—reports that around 300 million people in the world currently have asthma, and the prevalence has been increasing in both adults and children in recent decades.[868] Urban rates are generally higher than rural rates. GINA also estimates that asthma accounts for about one in every 250 deaths globally, which for the most part are preventable.

According to GINA, Canadian asthma prevalence rates for both children and adults are among the highest in the world.[869] Thus, a GINA questionnaire found that 2004 prevalence rates were highest in the United Kingdom (Scotland – 18.4%, Wales – 16.8%, and England – 15.3%), New Zealand (15.1%), Australia (14.7%), Canada (14.1%), U.S. (10.9%), and a few South American countries. In addition, GINA reports that in Canada and the U.S., about 40% of all children and adults with asthma required urgent care such as hospitalization or emergency room treatment in the previous 12 months. Other studies, such as those reported below, have found lower Canadian prevalence rates for asthma. This may be because of the very general definition of asthma used by GINA, which could include more cases than the more specific definition used in Canada by PHAC and given above.

However, a very recent study published in November 2008 suggests that Canadian physicians

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[864] Public Health Agency of Canada (PHAC). *Life and Breath: Respiratory Disease in Canada*, Ottawa: PHAC, 2007; accessed October 2008; available from http://www.phac-aspc.gc.ca/publicat/2007/lbrdc-vsmrc/pdf/PHAC-Respiratory-WEB-eng.pdf.
[865] Ibid., accessed.
[866] Ibid., accessed.
[867] Aaron, Shawn D., Katherine L. Vandemheen, Louis-Philippe Boulet, R. Andrew McIvor, J. Mark FitzGerald, Paul Hernandez, Catherine Lemiere, Sat Sharma, Stephen K. Field, Gonzalo G. Alvarez, Robert E. Dales, Steve Doucette, and Dean Fergusson for the Canadian Respiratory Clinical Research Consortium. "Overdiagnosis of Asthma in Obese and Nonobese Adults," *Canadian Medical Association Journal*, 2008, vol. 179, no. 11, November 18: 1121-1131.
[868] Masoli, Matthew, Denise Fabian, Shaun Holt, and Richard Beasley. *Global Burden of Asthma*, Global Initiative for Asthma (GINA), 2004; accessed November 2008; available from http://www.ginasthma.com/ReportItem.asp?l1=2&l2%20=2&intId=94.
[869] Ibid., accessed.

may misdiagnose asthma by as much as one-third because of a lack of basic testing.[870] Shawn
Aaron of the Ottawa Health Research Institute et al. report that almost half of patients who have
asthma diagnosed by a physician have not been given a spirometry test—the most basic form of
testing for asthma.[871] Aaron et al. conducted a case-control study of a nationally representative
sample of 540 obese and non-obese individuals, aged 16 and over, across Canada who had been
diagnosed with asthma by a physician. Over a series of clinic visits, the individuals underwent
extensive medical testing. The study found that one-third of both obese and non-obese
individuals previously diagnosed with asthma did not in fact have asthma when objectively
assessed.

Obesity has been consistently associated with the likelihood of developing asthma. Aaron et al.
report that studies suggest obese adults are almost twice as likely to be diagnosed with asthma as
non-obese adults.[872] They note that in Canada and the U.S., between 8.8–9.2% of obese adults
have reported being diagnosed with asthma by a physician, compared with 4–5% of non-obese
adults.

Magali Poulain et al. of the University of Level in Quebec note that obesity was first shown to be
associated with respiratory symptoms in the 1980s, and that "it is now recognized that a causal
relation may exist between them."[873] To date, many epidemiological studies have linked obesity
with a risk of developing adult-onset asthma.[874] Globally, between 1980 and 2000, asthma
prevalence increased by 73%—in tandem with the increase in global obesity rates.[875] According
to U.S. researchers I.U. Eneli et al., "The simultaneous rise in the frequency of both conditions
may not be coincidental."[876] Eneli et al. note that evidence for the mechanisms linking obesity
and asthma is not consistent across studies, but generally include gastro-esophageal reflux,
genetic factors, dietary intake, inflammatory mediators (e.g., interleukins), abnormal chest wall
mechanics, and physical inactivity.[877]

In a review of the literature on weight loss and asthma, Eneli et al. found a consistent
improvement in asthma outcomes across all studies after weight loss—including reductions in
symptoms, use of medications, and hospitalization incidence. This provides evidence of
reversibility, which is an important epidemiological criterion of causality:—i.e. if high BMI is a
risk factor, then reducing BMI would decrease the prevalence of asthma, or reduce related
symptoms. Using this criterion, the authors note that there is clear evidence that obesity is a risk
factor for asthma.

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[870] Aaron, Vandemheen, Boulet, McIvor, FitzGerald, Hernandez, Lemiere, Sharma, Field, Alvarez, Dales, Doucette,
and Consortium. "Overdiagnosis of Asthma in Obese and Nonobese Adults."
[871] Ibid.
[872] Ibid.
[873] Poulain, Magali, Marieve Doucet, Genevieve C. Major, Vicky Drapeau, Frederic Series, Louis-Philippe Boulet,
Angelo Tremblay, and Francois Maltais. "The Effect of Obesity on Chronic Respiratory Diseases: Pathophysiology
and Therapeutic Strategies," *Canadian Medical Association Journal*, 2006, vol. 174, no. 9: 1293-1299.
[874] Vortmann, Michael, and Mark D. Eisner. "BMI and Health Status among Adults with Asthma," *Obesity*, 2008,
vol. 16, no. 1: 146-152.
[875] Eneli, Skybo, and Jr. " Weight Loss and Asthma: A Systematic Review."
[876] Ibid.
[877] Ibid.

The Nurses' Health Study of 85,911 female nurses, which was conducted in the U.S. in 1991 with a follow-up in 1995, was one of the first studies to report a strong positive association between high BMI and the risk of adult onset asthma.[878] The adjusted RR of having an asthma diagnosis and using an asthma medication for those with a BMI ≥30 was RR 3.0, when compared with those with normal weight. The study also found that females, aged 26 to 46, who had gained more than 25 kg after the age of 18 were almost five times as likely (RR 4.7) to develop diagnosed adult-onset asthma during the follow-up period as those who did not have weight gain.

In 2001, Australian researchers L.M. Schachter et al. analysed 1971 data from three large epidemiological studies for white adults aged 17–73 years.[879] They found the prevalence of asthma among those with moderate obesity (BMI 30.0–34.9) and severe obesity (BMI ≥35.0) to be significantly greater than in those with a normal BMI (18.5–24.9), with ORs for the prevalence of asthma of 1.21 for moderate obesity and 1.43 for severe obesity. Severely obese individuals also had a significantly higher prevalence of wheeze, shortness of breath on exertion, and medication use for asthma in the previous 12 months.

Michael Vortmann and Mark Eisner of the University of California conducted a telephone survey of 843 adults aged ≥18 who had been hospitalized for asthma between 2000 and 2004, for the purpose of assessing the impact of BMI on asthma-related health outcomes.[880] Of those surveyed, 44% were considered to be obese. The researchers found no statistical association between the obese and normal BMI groups for the risk of emergency room visits or hospitalization for asthma. However, obese asthma patients had more asthma symptoms, and a higher risk of daily or near daily asthma symptoms than the BMI group with normal weight. Also, compared with the normal weight group, obese patients had poorer general physical health, a lower asthma-specific quality of life, and a higher number of restricted activity days during the previous month.

In 2007, Louis-Philippe Boulet and Annick Des Cormiers of Université Laval examined the link between obesity and asthma in Canada using data from the 2000/01 CCHS.[881] They found that self-reported asthma rates increased with increasing BMI, especially among women. Rates for both men and women remained fairly stable until they began to increase in obese women (BMI ≥30) and in severely obese men (BMI ≥40). Although the report is mainly descriptive and does not provide risk ratios or specific data, it appears—based on Figure 3 of that study—that the prevalence of asthma in women increased from about 8% among women with normal weight, to about 10% among overweight women, to about 15% among women in obese class 1 (BMI 30–34.9), to about 18% among women in obese class 2 (BMI 35–39.9), to about 25% among women in obese class 3 (BMI ≥40). Among men, it appears that about 5% of men with normal weight

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[878] Camargo, Carlos A. Jr, Scott T. Weiss, Shumin Zhang, Walter C. Willett, and Frank E. Speizer. " Prospective Study of Body Mass Index, Weight Change, and Risk of Adult-Onset Asthma in Women," *Archives of Internal Medicine* 1999, vol. 159: 2582–2588.

[879] Schachter, L. M., C. M. Salome, J. K. Peat, and A. .J Woolcock. "Obesity Is a Risk for Asthma and Wheeze but Not Airway Hyperresponsiveness," *Thorax*, 2001, vol. 56: 4-8.

[880] Vortmann, and Eisner. "BMI and Health Status among Adults with Asthma."

[881] Boulet, Louis-Philippe, and Annick Des Cormiers. "The Link between Obesity and Asthma: A Canadian Perspective," *Canadian Respiratory Journal*, 2007, vol. 14, no. 4: 217-220.

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through obese class 2 had asthma, at which point the proportion appears to have increased dramatically to about 18% among men in obese class 3 (BMI ≥40).

4.8.1 Asthma statistics in Canada and Alberta

Neither Birmingham et al. nor Katzmarzyk and Janssen included asthma in their studies on obesity in Canada.[882,883]

According to a 2007 study from the Institute for Clinical Evaluative Sciences (ICES), asthma is the most common chronic respiratory disease in Canada, accounts for about 80% of chronic respiratory disease prevalence, and has risen sharply in prevalence in recent years.[884] The National Asthma Control Task Force (NACTF) of Health Canada also reports that asthma is one of the most prevalent chronic conditions affecting Canadians, and is responsible for increased health care expenditures, reduced productivity, and a poor quality of life both for individuals with asthma and for their families.[885] In addition, 35% of individuals with current asthma were restricted in their daily activities by asthma—with 22% of asthmatics reporting restrictions for one to five days in the previous year, and 13% reporting restrictions for more than five days in the previous year. However, the 75-page Task Force report, *The Prevention and Management of Asthma in Canada,* did not mention a connection between asthma and obesity, or include obesity as a risk factor for asthma.

As noted, asthma prevalence in Canada is generally reported as being considerably lower than the rate reported by the 2004 GINA study described above—14.1% of Canadians. The NACTF reports that data from the 1996/97 NPHS showed that the prevalence in the Canadian population of active asthma—defined as asthma diagnosed by a physician, and with respondents either on medication or having manifested symptoms in the past 12 months—was 6.2% overall. Rates were 5.0% among adults aged ≥20 and 9.9% among children and youth aged 0–19.[886]

The NACTF makes a distinction between "active asthma" and "physician diagnosed" asthma. Although not defined, presumably the latter does not include the use of medications or the incidence of symptoms in the past 12 months. It reports that rates of physician diagnosed asthma in the 1996/97 NPHS were 6.3% among adults aged ≥20 and 12.2% among children and youth aged 0–19.[887] The GINA study, however, defined asthma simply as "wheezing in the last 12

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[882] Birmingham, C. Laird, Jennifer L. Muller, Anita Palepu, John J. Spinelli, and Aslam H. Anis. "The Cost of Obesity in Canada."

[883] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[884] Gershon, Andrea, Chengning Wang, Lisa Cicutto, and Teresa To. "ICES [Institute for Clinical Evaluative Sciences] Report: The Burden of Asthma: Can It Be Eased?" *Healthcare Quarterly*, 2007, vol. 10, no. 1: 22-24.

[885] National Asthma Control Task Force. *The Prevention and Management of Asthma in Canada: A Major Challenge Now and in the Future*, Laboratory Centre for Disease Control (LCDC) of Health Canada, 2000; accessed October 2008; available from http://www.phac-aspc.gc.ca/publicat/pma-pca00/pdf/asthma00e.pdf.

[886] Ibid., accessed. The report did not specify whether the data used came from the longitudinal or cross-sectional samples.

[887] Ibid., accessed.

months" and did not include physician diagnosis in its questionnaire, so the data are not strictly comparable with those from the NPHS. The different methodologies also help explain the higher asthma prevalence rates reported by the GINA study.

In line with other sources, the Asthma Society of Canada also reported in 2005 that the prevalence of asthma among adults aged ≥15 has been increasing in Canada during the last 20 years.[888] The trend it reported were an increase in asthma from 2.3% of adults in 1979 to 6.1% in 1994. The source of the data was not given. A review of Statistics Canada CANSIM tables showed the prevalence of asthma increasing until 2000/01, after which the rates have remained fairly stable. According to the NPHS cross-sectional sample, among Canadians aged ≥12 years, the prevalence of diagnosed asthma was 6.5% in 1994/95, 7.2% in 1996/97, and 8.1% in 1998/99.[889] The CCHS shows that the prevalence of diagnosed asthma among Canadians aged ≥12 years was 8.4% in 2000/01, 8.4% in 2003, and 8.3% in 2005.[890]

However, PHAC reports that between 1994 and 2005, the prevalence of diagnosed asthma in Canada increased by 60% among women aged 35–44 years, by 80% among women aged 45–64 years, and by 41% among men aged 35–44.[891]

Using data from the National Longitudinal Survey of Children and Youth (NLSCY), Rochelle Garner and Dafna Kohen of Statistics Canada found that the diagnosed asthma prevalence among children aged 4–11 increased from 11.1% (518,000 children) in 1994/95 to 13.4% (586,000 children) in 2000/01, which they noted was "a statistically significant increase of nearly 70,000 children."[892] However, they also observed that, despite the increased prevalence of asthma among children, the percentage of children with asthma reported as having had an attack in the previous 12 months decreased from 51% of children with asthma in 1994/95 to 39% in 2000/01.

In 2006, Alberta Health and Wellness reported that the treated prevalence of asthma for all ages—meaning the percentage of those who actually received medical care for the condition in Alberta in 2003 based on administrative records—was 4.2%.[893] Statistics Canada, using self-reported data from 2005 CCHS, reports that the diagnosed asthma rate in Alberta for those aged 12 and over was 8.6% of the population—including 7.3% of males and 9.9% of females.[894] CANSIM tables show the diagnosed asthma prevalence in Alberta increasing from 6.7% in 1994/95, to 7.1% in 1996/97, 8.8% in 1998/99, 8.9% in 2000/01, and 9.1% in 2003. The 2005

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[888] Asthma Society of Canada. *Asthma Facts and Statistics*, 2005; accessed October 2008; available from http://www.asthma.ca/corp/newsroom/pdf/asthmastats.pdf. Although the Asthma Society revised its fact sheet in 2005, most of its data sources in this document were still from the 1990s.

[889] Statistics Canada. CANSIM Table 104-0001 – Asthma, by age group and sex, household population aged 4 and over, Canada, provinces, territories, health regions and peer groups, every 2 years.

[890] Statistics Canada. CANSIM Tables 105-0001, 105-0201, and 105-0401 – Asthma, by age group and sex, household population aged 12 and over, Canada, provinces, territories, health regions and peer groups, every 2 years.

[891] Public Health Agency of Canada (PHAC). *Life and Breath: Respiratory Disease in Canada*, accessed.

[892] Garner, Rochelle, and Dafna Kohen. "Changes in the Prevalence of Asthma among Canadian Children," *Health Reports*, Statistics Canada Catalogue no. 82-0003, 2008, vol. 19, no. 2: 1-6. p. 1.

[893] Alberta Health and Wellness. *Report on the Health of Albertans*, accessed.

[894] Statistics Canada. *Health Indicators*, accessed.

rate showed a decrease (8.6%) from 2003.[895] The above 1994–1999 rates are from the cross-sectional sample of NPHS, and rates since 2000 are from CCHS.

The population attributable fractions (PAFs) of asthma attributable to excess weight were estimated for this report using adjusted self-reported BMI data from 2005 CCHS cycle 3.1, since the 2004 CCHS, cycle 2.2, did not ask about asthma diagnosis. As previously noted, the self-reported BMI data were adjusted using a newly developed methodology from Statistics Canada to correct for reporting bias, and to more accurately align self-reported BMI rates with directly measured rates.[896]

The prevalence of asthma in the Canadian population aged ≥15 based on 2005 CCHS, cycle 3.1 was 7.3% among obese males and 13.5% among obese females in 2005.

Comparing the obese population (i.e. the exposed population) (BMI ≥30) to the normal weight population (i.e. the unexposed) (BMI 18.5–24.9), the age-adjusted prevalence-based risk ratio for asthma was 1.2 for males and 1.7 for females 15 years of age and over.

The portion (population attributable fraction–PAF) of asthma that can be attributed to obesity among Canadians aged ≥15 was 4.4% for males and 11.5% for females.

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[895] Statistics Canada. CANSIM Table 104-0001. Alberta – Asthma, by age group and sex, household population aged 4 and over, Canada, provinces, territories, health regions and peer groups, every 2 years; Statistics Canada. CANSIM Tables 105-0001, 105-0201, and 105-0401. Alberta – Asthma, by age group and sex, household population aged 12 and over, Canada, provinces, territories, health regions and peer groups, every 2 years.
[896] Gorber, Shields, Tremblay, and McDowell. "The Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity."

4.9 Mental health: mood disorders / depression

According to the U.K. House of Commons Health Committee, the psychological damage that is caused by obesity is a "huge health problem," especially in children and youth among whom chronic physical conditions have not yet developed, and for whom the first results of obesity are likely to be emotional and psychological.[897] Adverse psychological conditions arising from obesity range from lowered self-esteem and sense of isolation to anxiety and clinical depression, and even suicide.

Addressing the direction of causality, Hubert Lacey of the Royal College of Psychiatrists notes that depression tends to be caused by obesity, rather than obesity being caused by depression: "There is not a clear link between massive obesity and a pre-existing psychological problem; rather there is evidence of psychological sequelae from the massive obesity itself."[898] At the same time, as noted below, there is evidence that use of anti-depressants stimulates weight gain, and there has been a striking correlation between rising rates of depression and obesity.

James et al. note that, especially in Europe and North America, obesity carries an extreme social stigma, "which has clearly been related to poorer access to employment opportunities, lower earning power, a tendency to marry less affluent partners and a tendency to become personally distressed and socially isolated."[899] In one British study of obese girls, the girls "were perceived by their peers to be less active, less attractive, less healthy, weak-willed and having inferior physical abilities and poor self-control regarding dietary habits."[900] Thus, in addition to the social stigma associated with the appearance of the body, there is an additional stigma associated with "the character of the person for the perceived moral failure of not controlling one's weight."[901]

The World Health Organization (WHO) indicates that, globally among persons aged 15–44 years, major depressive disorder is among the top 10 leading causes of disability and premature death.[902] Depression has been found to be particularly common among women, single mothers, divorced persons, young adults, low-income earners, the unemployed, high school dropouts, those experiencing chronic health problems, and individuals experiencing chronic workplace or time stress.[903, 904]

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[897] House of Commons Health Committee (U.K.). *Obesity: Third Report of Session 2003-04, Annex 1: The Economic Costs of Obesity*, accessed.

[898] Lacey, Hubert. Cited in Ibid., accessed. p. 20.

[899] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)." p. 568.

[900] National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, accessed. p. 55. Citing McIntyre, A.M. "Burden of Illness Review of Obesity: Are the True Costs Realised?" *Journal of the Royal Society for the Promotion of Health*, 1998, vol. 118, no. 2.

[901] Ibid.

[902] World Health Organization. Cited in McIntyre, Roger S., Deborah A. Mancini, Mark M.Pearce, Peter Silverstone, Pierre Chue, Virginia L. Misener, and Jakub Z. Konarski. "Mood and Psychotic Disorders and Type 2 Diabetes: A Metabolic Triad," *Canadian Journal of Diabetes*, 2005, vol. 29, no. 2: 122-132.

[903] Patten, Scott, and Heather Juby. *A Profile of Clinical Depression in Canada*, Statistics Canada, Research Data Centre Network (RDC), RDC Synthesis Series, 2008; accessed October 2008; available from https://dspace.ucalgary.ca/bitstream/1880/46327/6/Patten_RSS1.pdf.

Roger McIntyre and colleagues of the University of Toronto define the disorder as follows:[905]

> Major depressive disorder is defined as recurrent periods of depressive episodes that interfere with psychosocial, interpersonal or vocational functioning. A depressive episode includes the presence of depressed mood and/or anhedonia for a duration of ≥2 weeks, with accessory symptoms such as sleep disturbance, diminished energy, poor concentration, thoughts of hopelessness and suicidal ideation.[906]

Statistics Canada defines depression somewhat differently:

> Population aged 12 and over with the probability of 0.9 or greater of having experienced a major depressive episode in the past 12 months based on responses to the short-form [WHO] Composite International Diagnostic Interview (CIDI). Respondents are classified according to the probability that they would have been diagnosed as having experienced a major depressive episode in the past 12 months, if they had completed the long-form CIDI.[907]

The data Statistics Canada uses for depression come from self-report surveys such as the CCHS, which uses the long-form CIDI, and the agency adds the following caveat concerning the use of these data:

> Data quality note on probability of depression from CCHS: The depression module used in CCHS Cycle 3.1 (as well as in Cycles 1.1 and 2.1 and in the NPHS) is based on a long form of the Composite International Diagnostic Interview (CIDI) scale, which was developed in the late 1980s/early 1990s. This scale was never fully validated by the CIDI research team and its psychometric properties are therefore not well understood. Statistics Canada is currently exploring strategies to complete such a validation. At this time, Statistics Canada recommends that analysis of data from this module be restricted to examination of depression as a correlate of other health behaviours and characteristics. For now, use of the data as an indicator for the probability of depression or to calculate simple population prevalence is discouraged.[908]

Clearly, this major caveat makes it difficult to use the CCHS data from the depression module, which uses the CIDI, to estimate RRs and assess PAFs for use in cost estimates linking obesity to depression. However, CCHS cycle 3.1 asks a basic question about depression diagnosed by a health professional that was not part of the module and is used in this study: "Do you have a

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[904] Shields, Margot. "Stress and Depression in the Employed Population," *Health Reports*, Statistics Canada, Catalogue no. 82-003 2006, vol. 17, no. 4: 11-29.
[905] McIntyre, Mancini, M.Pearce, Silverstone, Chue, Misener, and Konarski. "Mood and Psychotic Disorders and Type 2 Diabetes: A Metabolic Triad."
[906] Ibid. p. 123.
[907] Statistics Canada. *Health Indicators*, accessed. It is not clear how or when the short form of CIDI is used.
[908] Ibid., accessed.

mood disorder such as depression, bipolar disorder, mania or dysthymia?"[909] This methodology is discussed more fully in Part 2, Chapter 5.4.7 of this report.

Depression is generally measured by three main indicators that estimate the proportion of individuals with severe depression—
- at some point in their lives—called "lifetime prevalence";
- during the previous 12-month period—called "annual prevalence"; and
- at a given point in time (typically occurring in the 30 days preceding the survey)—called "point prevalence".[910]

McIntyre et al., who recently completed a review of the subject in the epidemiological literature published between 1966 and 2004, note that the lifetime prevalence of major depressive disorder is generally estimated to be from 10% to 25% in women and from 5% to 12% in men worldwide.[911]

Scott Patten of the University of Calgary and Heather Juby of Statistics Canada's Research Data Centre Network recently completed a review of studies of clinical depression in Canada that used Statistics Canada data from the NPHS or CCHS and that were published in scientific journals.[912] They report: "Recent estimates show that approximately one Canadian in ten experienced a major depressive episode at some point *(lifetime prevalence = 10%–12%)*, one in twenty in the course of a year *(annual prevalence = 4%–5%)*, and one in fifty at a particular point in time *(point prevalence = 2%)*."[913]

Katherine Smith of the University of Toronto et al. recently used 2000/01 CCHS data to study depression in urban Canada.[914] Although specific rates for the risk of depression were not given, they found that the lowest risk of depression was in Quebec, and the highest risk was in Alberta.[915]

Scott Patten and Cynthia Beck of the University of Calgary report that the use of medications for depression and anxiety increased dramatically in Canada during the 1990s.[916] Between 1994 and 2000, the proportion of individuals who had been identified as having clinical depression and who were taking antidepressants more than doubled. They found that in 2002, approximately 6% of Canadians were taking antidepressant medications, which are generally regarded as the first

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[909] Statistics Canada. *Canadian Community Health Survey (CCHS) Cycle 3.1, Final Questionnaire*, 2005; accessed September 2008; available from http://www.statcan.gc.ca/imdb-bmdi/instrument/3226_Q1_V3-eng.pdf. p. 25.

[910] Patten, and Juby. *A Profile of Clinical Depression in Canada*, accessed.

[911] McIntyre, Mancini, M.Pearce, Silverstone, Chue, Misener, and Konarski. "Mood and Psychotic Disorders and Type 2 Diabetes: A Metabolic Triad."

[912] Patten, and Juby. *A Profile of Clinical Depression in Canada*, accessed.

[913] Ibid., accessed.

[914] Smith, Katherine L. W., Flora I. Matheson, Rahim Moineddin, and Richard H. Glazier. "Gender, Income and Immigration Differences in Depression in Canadian Urban Centres," *Canadian Journal of Public Health*, 2007, vol. 98, no. 2: 149-153.

[915] Ibid.

[916] Patten, Scott B., and Cynthia A. Beck. "Major Depression and Mental Health Care Utilization in Canada: 1994-2000," *Canadian Journal of Psychiatry*, 2004, vol. 49, no. 5: 303-309.

line of treatment for depression and which include selective serotonin reuptake inhibitors (SSRIs) with brand-names such as Prozac, Zoloft, and Paxil.[917]

According to Maria Raeder et al. of the University of Bergen in Norway, there is evidence that many antidepressant drugs induce weight gain, perhaps because these drugs have a direct effect on fasting blood glucose and serum lipid-levels, and that drug-induced weight gain is a major problem in the treatment of psychiatric disorders.[918] Whether this increase in use of antidepressant drugs has increased the prevalence or severity of obesity is not known, but the correlation between the increase in the two factors is striking.

As noted, obese individuals have an increased risk for depression. McIntyre et al., who have written a number of articles examining the obesity / depression association in Canada, report that the association between major depressive disorders and obesity is established in the research.[919] However, a 2008 systematic review of epidemiological studies on the effects of obesity on depression found that most studies are cross-sectional, which limit their generalizability, and that the majority of studies are from the U.S., using samples mostly of middle-aged to older adults.[920]

Nevertheless, the authors, E. Atlantis and M. Baker of the University of Sydney in Australia, note: "Overall, the body of evidence systematically reviewed indicates that there is some level of support for the hypothesized association between obesity and incidence of depression outcomes (that is, depressive symptoms or nonclinical diagnosis of depression."[921] Again, it must be emphasized, as noted with other obesity-related disorders above, that research on these associations is in its infancy, and that support for the association between obesity and depression may well grow as further studies are undertaken. In the meantime, the evidence is mixed.

Haslam and James note that in the U.S., obesity in women increases the risk of being diagnosed with a major depression by 37%, but obesity in men *decreases* this risk by 37%.[922] However, other studies have not found the same outcome. Elizabeth Johnston of Acadia University et al. examined data from the 1995 Nova Scotia Health Survey to assess the relationship between measured BMI and risk of depression as measured by the Center for Epidemiological Studies Depression Scale (CES-D).[923] The scale, which was developed for use in population-based epidemiological studies, measures current levels (frequency and duration in the past week) of depressive symptoms. Scores range from 0–60, and an elevated risk of depression is indicated by a score of 16 or more.

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[917] Ibid.

[918] Raeder, Maria B., Johan Ferno, Audun O. Vik-mo, and Vidar M. Steen. "SREBP Activation by Antipsychotic- and Antidepressant-Drugs in Cultured Human Liver Cells: Relevance for Metabolic Side-Effects?" *Molecular and Cellular Biochemistry*, 2006, vol. 289, no. 1-2: 167-173.

[919] McIntyre, Roger S., Joanna K. Soczynska, Jakub Z. Konarski, Hanna O. Woldeyohannes, Candy W. Y. Law, Andrew Miranda, Don Fulgosi, and Sidney H. Kennedy. "Should Depressive Syndromes Be Reclassified as 'Metabolic Syndrome Type II'?" *Annals of Clinical Psychiatry*, 2007, vol. 19, no. 4: 257-264.

[920] Atlantis, E., and M. Baker. "Obesity Effects on Depression: Systematic Review of Epidemiological Studies," *International Journal of Obesity*, 2008, vol. 32: 881–891.

[921] Ibid.

[922] Haslam, and James. "Obesity."

[923] Johnston, Elizabeth, Shanthi Johnson, Peter McLeod, and Mark Johnston. "The Relation of Body Mass Index to Depressive Symptoms," *Canadian Journal of Public Health*, 2004, vol. 95, no. 3: 179-184.

In the Johnston et al. study, over 5,500 Nova Scotians aged ≥18 years were surveyed, of whom 2,482 both completed the CES-D and also had their height and weight directly measured. Results showed that obesity was associated with an unadjusted 53% increased risk of depression and a 41% risk of depression after controlling for income and education. Johnston et al. found that 18.4% of Nova Scotians had a BMI ≥30 and a CES-D score of ≥16, compared with 12.8% of the population with a BMI <30 and a CES-D score of ≥16. Women had a higher mean CES-D score than men (8.2 vs. 6.7).

Surprisingly, individuals with acceptable weight and obese weight both had higher depression scores in this study than those who were overweight. Concerning this finding, the authors remark: "Our finding that overweight individuals were less depressed than either the obese or acceptable weight groups may in part reflect the increasingly normative nature of being overweight, but this needs further exploration."[924]

McIntyre et al. have found that high rates of overweight and obesity are also consistently reported in individuals with mood disorders such as bipolar disorder.[925, 926] They note that mood and psychotic disorders are also associated with a sedentary lifestyle and with genetic factors, as well as with obesity.[927] However, they point out that, "a clear, comprehensive and coherent disease model in affective disorders does not currently exist," and they suggest that the metabolic system might be "a potential explanatory factor in mood disorders."[928]

McIntyre et al. refer to disturbances in the metabolic network that include insulin-glucose balance, inflammatory processes, and adipokine synthesis as being implicated in the biological pathways potentially leading to mood disorders. And they note that the "central nervous system, like the pancreas, is a critical modulator of the metabolic milieu and is endangered by chronic abnormalities in metabolic processes."[929] They also suggest that viewing mood disorders as metabolic syndromes might open new and innovative treatment for these disorders.

U.S. researchers Susan McElroy and colleagues reviewed almost 40 years of the medical literature for evidence of the relationship between mood disorders and obesity.[930] They found that mood disorder subtypes specifically related to obesity in the literature include major

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[924] Ibid. p. 183.

[925] McIntyre, Roger S., Joanna K. Soczynska, John Beyer, Hanna Woldeyohannes, Candy Law, Andrew Miranda, Jakub Z. Konarski, and Sidney H. Kennedy. "Medical Comorbidity in Bipolar Disorder: Reprioritizing Unmet Needs," *Current Opinion in Psychiatry*, 2007, vol. 20, no. 4: 406-416.

[926] McIntyre, Roger S., Susan L. McElroy, Jakub Z. Konarski, Joana K. Soczynska, Alexandra Bottas, Saulo Castel, Kathryn Wilkins, and Sidney H. Kennedy. "Substance Use Disorders and Overweight/Obesity in Bipolar I Disorder: Preliminary Evidence for Competing Addictions," *Journal of Clinical Psychiatry*, 2007, vol. 68: 1352-1357.

[927] McIntyre, Mancini, M.Pearce, Silverstone, Chue, Misener, and Konarski. "Mood and Psychotic Disorders and Type 2 Diabetes: A Metabolic Triad."

[928] McIntyre, Soczynska, Konarski, Woldeyohannes, Law, Miranda, Fulgosi, and Kennedy. "Should Depressive Syndromes Be Reclassified as 'Metabolic Syndrome Type II'?" p. 258.

[929] Ibid. p. 257.

[930] McElroy, Susan L., Renu Kotwal, Shishuka Malhotra, Erik B. Nelson, Paul E. Keck Jr., and Charles B. Nemeroff. "Are Mood Disorders and Obesity Related? A Review for the Mental Health Professional," *Journal of Clinical Psychiatry*, 2004, vol. 65: 634-651.

depressive disorder, major depressive disorder with juvenile onset, and bipolar disorder, especially when depressive features are more prominent than manic features.[931] The review found that obese individuals, and especially obese females, may have elevated rates of depressive disorders, but that most overweight or obese persons do not have mood disorders.

David Lau and members of the Obesity Canada Clinical Practice Guidelines Expert Panel also found that, in general, mood disorders, including major depression, are common in obese Canadians, and especially among obese women. Thus, they found that major depression occurs in 20–60% of women aged ≥40 with a BMI ≥30.[932]

U.S. researchers Gregory Simon et al. used data from the National Comorbidity Survey Replication—which surveyed 9,125 respondents using the CIDI form—to examine the associations between psychiatric disorders and obesity (BMI ≥30) compared with risks of psychiatric disorders among those with BMI <30.[933] In females, obesity was associated with an approximate 30% increase in the odds of having a mood disorder (OR 1.29) or anxiety disorder (OR 1.34) during their lifetimes. Obese males had an OR of 1.21 for lifetime mood disorder and an OR of 1.17 for lifetime anxiety disorder.

4.9.1 Mental health statistics in Alberta

Alberta Health and Wellness cites 2002 CCHS Mental Health and Well-being Survey estimates that 13.4% of Canadian adults have had a mood disorder at some point in their lifetimes.[934] McIntyre et al. used the self-reported data from the 2002 CCHS, cycle 1.2 on Mental Health and Wellbeing to estimate the association between obesity and mood disorders (i.e. major depressive disorder and bipolar disorder) in individuals, aged ≥15 years, in Canada.[935] They note that:

> This is the first Canadian epidemiologic investigation to specifically evaluate anthropometric indices and associated factors in people with MDs [mood disorders]. The results herein supplement substantial clinical evidence documenting the association between MDs and stress-sensitive somatic disorders (for example, obesity).[936]

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[931] Ibid.

[932] Lau, David C.W., James D. Douketis, Katherine M. Morrison, Irene M. Hramiak, Arya M. Sharma, and Ehud Ur for members of the Obesity Canada Clinical Practice Guidelines Expert Panel. "2006 Canadian Clinical Practice Guidelines on the Management and Prevention of Obesity in Adults and Children [Summary]," *Canadian Medical Association Journal*, 2007, vol. 176, no. Suppl. 8: S1-S13.

[933] Simon, Gregory E., Michael Von Korff, Kathleen Saunders, Diana L. Miglioretti, Paul K. Crane, Gerald van Belle, and Ronald C. Kessler. "Association between Obesity and Psychiatric Disorders in the Us Adult Population," *Archives of General Psychiatry*, 2006, vol. 63: 824-830. Simon et al. did not distinguish between the long and short forms of the CIDI.

[934] Alberta Health and Wellness Project Team. *Health Trends in Alberta*, accessed.

[935] McIntyre, Roger S., Jakub Z. Konarski, Kathryn Wilkins, Joanna K. Soczynska, and Sidney H. Kennedy. "Obesity in Bipolar Disorder and Major Depressive Disorder: Results from a National Community Health Survey on Mental Health and Well-Being," *Canadian Journal of Psychiatry*, 2006, vol. 51, no. 5: 274-280.

[936] Ibid. p. 274.

The authors found that 13.3% of Canadians (10.4% of males and 16.0% of females) had a history of mood disorders, and that 19.0% of individuals with a lifetime history of mood disorders were likely to be obese, compared with 15.4% of those without a lifetime history of mood disorders who were likely to be obese.[937] Females with lifetime mood disorders showed elevated odds of obesity (OR 1.22), but the difference was not as great in males (OR 1.10).

In 2003, Statistics Canada reported the self-reported prevalence of depression for selected provinces based on results from the 2002 CCHS.[938] In Alberta, 6.4% of the population, aged ≥12 years, had a probability of having experienced a major depressive episode in the past 12 months—4.6% of males and 8.1% of females.[939]

Alberta Health and Wellness notes that the main diagnoses of individuals seeking physician services for mental health problems in Alberta between 2002–2004 were anxiety disorders (56.7% of diagnoses) and mood disorders—including depressive and bipolar disorders (43.2% of diagnoses).[940] It notes that 11.9% of the population received care specifically related to anxiety disorders and depression in 2003.[941] Approximately 5% of Albertans sought physician services specifically to treat depression, including about 10% of women aged 30–49 years, "from 2004 to 2006."[942]

Alberta Health and Wellness found that administrative data showed a much higher prevalence of treated mental health disorders overall than did self-reported CCHS data:

> According to the Statistics Canada Canadian Community Health Survey, in 2005, approximately 8.4 per cent of Albertans reported consulting a medical professional for a mental health problem. However, rates from these self-report surveys are much lower than physician claims data for mental health problems in Alberta.The average rate over 3-years [2004–2006] based on physician claims for distinct individuals estimates that a little over 25 per cent of Albertans visited a physician for mental health related problems. Females, particularly in the adult years, are twice as likely as males to visit a physician for mental health; however differences between the sexes narrow in older adulthood.[943]

The possible discrepancy may indicate that individuals are likely to experience a stigma associated with mental health disorders, and may be reluctant to report that they have been treated for them.

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[937] Ibid.

[938] Statistics Canada. *Probability of Depression, by Age Group and Sex, Household Population Aged 12 and over, Selected Provinces, Territories and Health Regions (June 2003 Boundaries)*, CANSIM Table 105-0205, 2003.

[939] Ibid.

[940] Alberta Health and Wellness Project Team. *Health Trends in Alberta*, accessed.

[941] Alberta Health and Wellness. *Report on the Health of Albertans*, accessed.

[942] Alberta Health and Wellness Project Team. *Health Trends in Alberta*, accessed.

[943] Ibid., accessed. p. F-8. The AWH text does not make it clear whether the 25% average rate over three years refers to average *annual* rate or to numbers of Albertans who consulted a doctor about mental health at any time during these three years.

Respondents to 2005 CCHS, cycle 3.1 were asked whether they suffer from a mood disorder such as depression, bipolar disorder, mania or dysthymia, The prevalence of mood disorders in the Canadian population aged ≥15 years based on data from 2005 CCHS, cycle 3.1 was 5.7% among obese males and 11.4% among obese females in 2005.

Comparing the obese population (i.e. the exposed population) (BMI ≥30) to the normal weight population (i.e. the unexposed) (BMI 18.5–24.9), the age-adjusted prevalence-based risk ratio for mood disorders was 1.4 for males and 1.9 for females aged ≥15.

The portion (population attributable fraction–PAF) of mood disorders that can be attributed to obesity among Canadians aged ≥15 was 8.9% for males and 13.6% for females in 2005.

4.10 Functional limitations – long-term and short-term disability

With the notable exception of obesity-related cancers, obesity has generally been found to have a stronger impact on morbidity than on mortality.[944] New medical interventions are increasing survival prospects, especially for patients with obesity-related type 2 diabetes and cardiovascular diseases such as stroke, which often result in impaired neuro-musculoskeletal functions and mood disorders. Therefore, one of the costliest long-term burdens of obesity is its direct and significant effect on disability, since it can lead to a longer time living with disabling conditions.[945] Indeed, the medical interventions that increase the survival rates of obese individuals must themselves be considered part of the costs of obesity when used to prolong the lives of obese subjects who might otherwise have died of stroke or other afflictions.

Dutch researchers Tommy Visscher and Jacob Seidell note that because people are living longer with disabling conditions, the health impacts and costs of obesity will likely increase.[946] Economist Roland Sturm et al. have estimated that if the escalating obesity trend continues in the U.S. between 2005 and 2020 as it has in the last 20 years, without other changes in medical technology, behaviour, and other interventions, then the prevalence of daily activity limitation will increase by about 16% for women and 13% for men. In addition, the prevalence of work limitations will increase by about 10% for women and 6% for men.[947]

T.S. Han, of the University of Glasgow, et al. compared the measured BMI of over 4,000 Dutch adults aged 20–59 years with results for the same subjects from the standardized SF-36 Health Survey, which measures 9 health concepts, including physical functioning, or the ability to

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[944] Visscher, Tommy L.S., and Jacob C. Seidell. "The Public Health Impact of Obesity," *Annual Review of Public Health*, 2001, vol. 22: 355-375.
[945] Ibid.
[946] Ibid.
[947] Sturm, Roland, Jeanne S. Ringel, and Tatiana Andreyeva. "Increasing Obesity Rates and Disability Trends," *Health Affairs*, 2004, vol. 23, no. 2: 199-205.

perform daily tasks and activities.[948] Physical functioning includes ten items such as being able to perform vigorous or moderate activities, lifting or carrying groceries, climbing stairs, bending, walking various distances, and bathing or dressing. BMI was divided into terciles roughly corresponding to normal weight, overweight, and obesity.

Results showed that adults whose BMI was at tercile 3 level (obese) were more likely to have poor physical functioning (OR 2.37 for men and 2.11 for women) as were those whose BMI fell into tercile 1 (normal weight).

Martin Neovius et al. of Karolinska University Hospital in Sweden recently estimated the risk of receiving future disability pensions according to measured BMI in Swedish young men.[949] The researchers were able to link records from several national databases based on the unique personal identification numbers assigned to every Swedish citizen. This allowed them to use data on more than 80% of the total male population in Sweden over a third of a century. The men, who were born between 1951 and 1976, were physically examined for military conscription between the ages of 17–20 years. The follow-up period included 28.4 million person years, during which 60,024 men were granted a disability pension at an average age of 38.9 years.

Neovius et al. found that the median number of productive years of life lost increased from a median of 24.9 years for men with normal weight, to 26.4 years for moderately obese men, to 28.5 years for severely obese men.[950] In the study, 46% of all cases of disability pensions were for psychiatric causes, 24% for musculoskeletal causes, 9% for injuries, and 4% for circulatory causes. The relative risks for overweight and obese men were significantly elevated for each of these four causes when compared with normal weight men, but were especially high for musculoskeletal and circulatory causes.

Sturm et al. used nationally representative data for U.S. adults aged 50–69 years from the large U.S. Health and Retirement Study (HRS) as well as the Behavioral Risk Factor Surveillance Survey—which both use self-reported data for height and weight—to estimate the association between obesity and disability.[951] Two measures of disability or functional limitation were used—the first was based on limitations with activities of daily living such as bathing, eating, dressing, walking across a room, or getting in or out of bed; and the second was based on limitations that impair the kind or amount of paid work the individual is able to undertake.

The Sturm et al. study found that for both men and women, the percentages reporting that their impairment limits their ability to work were far higher than the rates of daily living activity limitations, but the effects of obesity on daily activity limitations were stronger.[952] The study also found that adults who were severely obese (BMI ≥35) had the highest disability rates.

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[948] Han, T. S., M. A. R. Tijhuis, M. E. J. Lean, and J. C. Seidell. "Quality of Life in Relation to Overweight and Body Fat Distribution," *American Journal of Public Health*, 1998, vol. 88, no. 12: 1814-1820.
[949] Neovius, Martin, M. Kark, and F. Rasmussen. "Association between Obesity Status in Young Adulthood and Disability Pension," *International Journal of Obesity*, 2008, vol. 32: 1319-1326.
[950] Ibid.
[951] Sturm, Ringel, and Andreyeva. "Increasing Obesity Rates and Disability Trends."
[952] Ibid.

Among U.S. men, the percentage of those with moderate obesity (BMI 30–34.9) who reported daily activity limitations (9.3%) was more than 50% higher than among men with normal weight (6.1%), while the rate among those with severe obesity (18.7%) was more than three times higher. The percentages of moderately and severely obese men reporting work limitations (27.6% and 40.1% respectively) were also significantly higher than among men with normal weight (22.6%).

Among U.S. women, the effect was even larger than for men. The percentages of moderately and severely obese American women reporting daily activity limitations (10.8% and 21.4% respectively) were more than double and quadruple the rate of women with normal weight (5.2%). The percentages of moderately and severely obese women reporting work limitations (27.3% and 45.7% respectively) were 55% and 160% higher than the rate among women with normal weight (17.6%).

4.10.1 Functional limitations statistics in Alberta

Kathryn Wilkins of Statistics Canada and Margaret de Groh of PHAC report that both cross-sectional and longitudinal data in Canada show that obese Canadians are more likely to experience physical disability than those with normal weight.[953] They examined the association between BMI and 'dependency' for adults aged ≥45 years using both cross-sectional data from the 2003 CCHS, and longitudinal data from the 1994/95–2002/03 NPHS. CCHS respondents were categorized as 'dependent' if they answered "yes" to at least one of the following questions:

> "Because of any physical or mental condition or health problem, do you need the help of another person with: Preparing meals? Getting to appointments and running errands such a shopping for groceries? Doing everyday housework? Personal care, such as washing, dressing, eating or taking medication? Moving about inside the house?"[954]

NPHS respondents were asked similar questions that had minor wording differences.

The study found that Canadian women had more than twice the rate of dependency than men (16.8% vs 8.2%), and were more dependent than men in all age groups, with particularly rates among women after the age of 75.[955] Study results showed that 10% of Canadian women and 4% of men aged 45–54 were dependent, as were 12% of women and 7% of men aged 55–64, 18% of women and 9% of men aged 65–74, 35% of women and 22% of men aged 75–84, and 64% of women and 47% of men aged ≥85.

Overall, the percentage of adults who were dependent rose progressively with increases in BMI, with this effect particularly marked for women. Among men, the rates of dependency actually

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[953] Wilkins, Kathryn, and Margaret de Groh. "Body Mass and Dependency," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2005, vol. 17, no. 1: 27-39.
[954] Ibid.
[955] Ibid.

remained fairly low until the most severe obesity category (BMI ≥40) where dependency rates were more than three times higher than in any other BMI class.

Thus, among women, 14% of those with normal weight, 16% of those overweight, 20% of those in obese class 1, 23% of those in obese class 2, and 31% of those in obese class 3 reported dependency. Among men, 8% of those with normal weight, 7% of those overweight, 9% of those in obese classes 1 and 2, and 28% of obese class 3 reported dependency. The study authors report that the NPHS longitudinal data also found obesity to be predictive of future dependency in both men and women.

In addition to examining dependency, the CCHS also inquires about 'functional health status', pain and discomfort, and activity limitations that prevent participation in daily activities. Further investigation is required to assess the association of BMI with these indicators.

Functional health status is measured in the CCHS by the Health Utility Index, which includes 8 dimensions of functioning (vision, hearing, speech, mobility, dexterity, feelings, cognition, and pain).[956] In 2005, 17.6% of Canadians and 18% of Albertans aged 12 and over had moderate or severe functional health problems—16.2% of Canadian males and 19.1% of Canadian females, and 15.5% of Albertan males and 20.6% of Albertan females. Quebec had the lowest rate in the country at 14.5%—13.5% of males and 15.5% of females.

In 2005, 16.7% of Canadians and 17.8% of Albertans aged 12 and over reported that they stayed in bed or cut down on normal activities because of illness or injury on one or more days in the past two weeks—14.3% of Canadian males and 19.2% of Canadian females, and 15.1% of Albertan males and 20.5% of Albertan females.[957] Quebec again reported the lowest rate in the country at 13.3%—11.8% of males and 14.8% of females.

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[956] Statistics Canada. *Health Indicators*, accessed.
[957] Ibid., accessed.

4.11 Other health conditions associated with obesity

The following health conditions have been associated with obesity but have not been included in the costs of obesity tabulated in this report due to a lack of suitably comparable data and other uncertainties that prevented reliable estimations of relative risks and PAFs for these diseases in Canada. The omission of obesity-related costs associated with the following conditions indicates that the total cost estimates in this report are likely to be conservative. Nevertheless, a few words on these illnesses is in order here to indicate their demonstrated associations with obesity.

4.11.1 Gallbladder cancer

WCRF/AICR reports that gallbladder cancer is a relatively rare, but usually fatal, malignancy that is commonly diagnosed at an advanced stage and, therefore, has a global 5-year survival rate of less than 10%.[958] Gallbladder cancer accounts for approximately 2% of cancer cases worldwide and 2% of cancer deaths, and is the 17th most common cause of cancer death. The risk increases with age, with more than two-thirds of cases occurring in adults aged 65 years and older. Women are about two to six times more likely than men to be diagnosed with gallbladder cancer.[959]

Ignacio Wistuba of the University of Texas' M.D. Anderson Cancer Center and Adi Gazdar of the University of Texas' Southwestern Medical Center note that gallbladder cancer is relatively uncommon and accounts for about 2,000 new cases per year in the U.S. This compares, for example, with the 150,000 new cases of colon cancer per year in the U.S.[960]

The U.S. and Canada both recorded low rates of gallbladder cancer in the mid 1990s—below 3 per 100,000 for women, and 1.5 per 100,000 for men.[961] Between 1993 and 1997, the age standardized incidence rates of gallbladder cancer among Canadian adults aged 35–74 years were 2.13 per 100,000 for women and 1.22 per 100,000 for men. U.S. rates in that same period were even lower at 1.78 per 100,000 for women and 1.03 per 100,000 for men.[962]

However, in both North and South America, indigenous Indian populations have much higher rates of gallbladder cancer. For example, the rate for First Nations people in New Mexico is 11.3 per 100,000. For gallbladder cancer, rates between 4 and 9 per 100,000 are considered to be 'moderately high', and rates over 9 per 100,000 are considered to be 'high'. Globally, the highest

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[958] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.
[959] Randi, Giorgia, Silvia Franceschi, and Carlo La Vecchia. "Gallbladder Cancer Worldwide: Geographical Distribution and Risk Factors," *International Journal of Cancer*, 2006, vol. 118: 1591-1602.
[960] Wistuba, Ignacio I., and Adi F.Gazdar. "Gallbladder Cancer: Lessons from a Rare Tumor," *Cancer*, 2004, vol. 4: 695-706.
[961] Randi, Franceschi, and Vecchia. "Gallbladder Cancer Worldwide: Geographical Distribution and Risk Factors."
[962] Ibid.

incidence rates have been reported among women in Delhi, India, who have a gallbladder cancer incidence rate of 21.5 per 100,000.[963]

Wistuba and Gazdar note that relatively little is known about gallbladder cancer, and that it is rarely diagnosed before surgical removal of the gallbladder. They explain that there are two main physio-biological pathways to gallbladder cancer, but that patients usually have symptoms of the first:

> Two main pathways of gallbladder carcinoma pathogenesis have been identified. The most common is associated with gallstones and chronic inflammation of the gallbladder, whereas a second, less frequent pathway is associated with a congenital abnormality of the pancreatic bile-duct junction, which is particularly common in Japan. A multistage sequence of histopathological and molecular changes has been identified for gallbladder carcinoma, which is especially well-defined for tumorigenesis associated with gallstones. Molecular abnormalities commence in normal-appearing epithelium in chronically inflamed gallbladders. … Relatively little is known about gallbladder cancer, and a significant influx of research funding is required for this to be remedied. In particular, the identification of susceptibility genes, elucidation of the role of inflammation and an increased understanding of the molecular changes that occur during multistage pathogenesis should be important goals for the future.[964]

WCRF/AICR notes that it is probable that body fatness is a factor in the development of gallbladder cancer through the formation of gallstones, but the evidence is not yet convincing.[965] The WCRF/AICR meta-analysis showed a 23% increased risk of gallbladder cancer per 5 kg/m^2 for cohort studies, and a 19% increased risk per 5 kg/m^2 for case-control studies. (Thus, the summary RR estimates provided by WCRF/AICR were 1.23 and 1.19 per 5 kg/m^2 for cohort and case-control studies, respectively.)

French and Italian researchers Giorgia Randi et al. note that a history of gallstones is the strongest risk factor for development of gallbladder cancer (RR 4.9).[966] However, only one to three percent of individuals worldwide with a history of gallstones develop gallbladder cancer. WCRF/AICR notes that in high-income countries about one in ten adults have mainly asymptomatic gallstones—a very common afflication, but gallbladder cancer is diagnosed only in around one in 50,000 cases of gallstones.[967]

In 2005 Randi et al. conducted a systematic review of studies published between 1980 and March 2005 on the association between gallbladder cancer and various risk factors, including obesity. They found eight studies published during this period that related obesity and gallbladder cancer, but they did not summarize the risk ratios to produce a single number that

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[963] Ibid.

[964] Wistuba, and F.Gazdar. "Gallbladder Cancer: Lessons from a Rare Tumor." p. 696.

[965] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

[966] Randi, Franceschi, and Vecchia. "Gallbladder Cancer Worldwide: Geographical Distribution and Risk Factors."

[967] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.

could be used in this report—primarily because the studies produced such a wide range of estimates depending on gender and ethnic differences and where they were conducted.

Thus, in studies that showed positive associations between obesity and gallbladder cancer, RRs ranged from 1.4 for obese women in Denmark and the Netherlands to 4.5 for obese women in Japan. Studies in which the RRs showed no association between obesity and gallbladder cancer all involved male participants. Two studies were from the U.S., and one of those compared white (RR 1.7) and black (RR 0.9) men.[968] The other compared risks of gallbladder cancer among obese class 1 men and women (BMI 30–34.9) compared with those of normal weight, and found the RRs to be 1.8 for men and 2.1 for women.[969]

In 2007 Swedish researchers Susanna Larsson and Alicja Wolk reviewed 11 studies on the association between excess weight and the risk of gallbladder cancer found in a search of studies published between 1966 and February 2007.[970] Their meta-analysis found an increasing risk of gallbladder cancer with increasing BMI, and they therefore concluded that their study "indicates that excess body weight is a risk factor for gallbladder cancer."[971] The association was significantly stronger among women than among men.

Using summary risk ratios from the meta-analysis and obesity prevalence data from the U.S., Larsson and Wolk estimated that 12% of gallbladder cancer cases among men and 30% among women could be attributed to overweight and obesity (BMI ≥25).

Compared with adults of normal weight, the Larsson and Wolk study found that obese adults had a 66% increased risk of gallbladder cancer, and overweight adults had a 15% increased risk. The authors found that summary RRs from studies that used self-reported height and weight measures were higher than those based on studies that directly measured height and weight (RR 1.82 versus 1.60, respectively). The overall summary risk estimates comparing obese adults with those of normal weight were RR 1.88 for women and RR 1.35 for men. For overweight adults, the RRs for women and men were 1.28 and 1.05, respectively.

Neither Birmingham et al., Katzmarzyk and Janssen, Luo et al., nor Pan et al. included gallbladder cancer in their studies on obesity and disease in Canada.[972][973, 974, 975] However,

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[968] Samanic, C., Gridley. G., W.H. Chow, J. Lubin, R.N. Hoover, and J.F. Fraumeni Jr. "Obesity and Cancer Risk among White and Black United States Veterans," *Cancer Causes and Control*, 2004, vol. 15: 35-43. Cited in Randi, Franceschi, and Vecchia. "Gallbladder Cancer Worldwide: Geographical Distribution and Risk Factors."

[969] Calle, Rodriguez, Walker-Thurmond, and Thun. "Overweight, Obesity, and Mortality from Cancer in a Prospectively Studied Cohort of U.S. Adults." Cited in Randi, Franceschi, and Vecchia. "Gallbladder Cancer Worldwide: Geographical Distribution and Risk Factors."

[970] Larsson, Susanna C., and Alicja Wolk. "Obesity and the Risk of Gallbladder Cancer: A Meta-Analysis," *British Journal of Cancer*, 2007, vol. 96, no. 9: 1457-1461.

[971] Ibid. p. 1460.

[972] Birmingham, C. Laird, Jennifer L. Muller, Anita Palepu, John J. Spinelli, and Aslam H. Anis. "The Cost of Obesity in Canada."

[973] Katzmarzyk, and Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update."

[974] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

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Birmingham et al., Katzmarzyk and Janssen, and Luo et al. did include gallbladder disease—a possible precursor of gallbladder cancer—which is discussed in Section 4.7 above.

4.11.2 Obstructive sleep apnea (OSA)

Obstructive sleep apnea (OSA), or obstructive sleep apnea syndrome (OSAS), which causes an intermittent cessation of breathing during sleep lasting from 10 to 30 seconds, and which can occur hundreds of times during the night, is actually a serious respiratory disorder (because of its potential consequences) that has been related to obesity.[976] OSA happens when soft tissue collapses in the back to the throat and blocks the airway, which in turn results in sleep interruption, lack of oxygen, and airflow cessation. Factors associated with obesity, such as relaxed throat muscles, a narrow airway, a large tongue, or extra fatty tissue in the throat, are among the main causes of OSA episodes.[977] OSA is serious because it can lead to pulmonary hypertension, hypertensive disease, heart failure, stroke, arrhythmias, insulin resistance, and accidents caused by daytime somnolence.[978]

In a recent 2008 report, Robert Carter III and Donald Watenpaugh note that "[o]besity is the most established and primary risk factor given that body mass index, visceral fat, and neck circumference are major predictors in the clinical expression of OSA."[979] A weight loss of 10% can result in a 26% decrease in the severity of sleep apnea.[980] Despite strong evidence pointing to an association between obesity and OSA, J. Garvey and W.T. McNicholas of St. Vincent's University Hospital in Dublin report that "obesity research has largely ignored the contribution of obstructive sleep apnea syndrome to the pathogenesis of cardiovascular risk in overweight patients."[981]

According to a 2007 PHAC report, there is a lack of information on the prevalence of OSA in Canada.[982] However, PHAC notes that prospective studies on middle-age adults have found that moderate to severe OSA, which is more common in men than in women, is present in about 4% of men and 2% of women between the ages of 30 and 60 years.[983]

The highest prevalence of OSA is seen in men aged 45–64 years, and in women over the age of 65. However, it has been estimated that from 60% to 80% of adults with OSA have not been

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[975] Pan, Johnson, Ugnat, Wen, Mao, and Group. "Association of Obesity and Cancer Risk in Canada."
[976] National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, accessed.
[977] Public Health Agency of Canada (PHAC). *Life and Breath: Respiratory Disease in Canada*, accessed.
[978] Wolk, and Somers. "Obesity-Related Cardiovascular Disease: Implications of Obstructive Sleep Apnea."
[979] Carter III, Robert, and Donald E. Watenpaugh. "Obesity and Obstructive Sleep Apnea: Or Is It OSA and Obesity?" *Pathophysiology, doi:10.1016/j.pathophys.2008.04.009*, Article in Press, 2008. p. 1.
[980] Ibid.
[981] Garvey, J., and W.T. McNicholas. "Effect of Obesity in Patients with Coronary Artery Disease," *The Lancet*, 2006, vol. 368, no. 9548: 1645. p. 1645.
[982] Public Health Agency of Canada (PHAC). *Life and Breath: Respiratory Disease in Canada*, accessed.
[983] Young, T., M. Palta, J. Dempsey, J. Skatrud, S. Weber, and S. Badr. "The Occurrence of Sleep Disordered Breathing among Middle-Aged Adults," *New England Journal of Medicine*, 1993, vol. 328: 1230-1235. Cited in Public Health Agency of Canada (PHAC). *Life and Breath: Respiratory Disease in Canada*, accessed.

diagnosed with the condition.[984, 985] R. Wolk and V.K. Somers of the Mayo Clinic note that OSA "should be suspected in obese individuals presenting with a history of loud snoring, daytime somnolence, fatigue or otherwise unexplained personality changes (such as irritability and depression)."[986]

Carter and Watenpaugh report that significant OSA is present in 40% of obese individuals, that between 40% and 90% of those who are severely obese (BMI ≥40) have diagnosed OSA, and that over 70% of OSA patients are obese.[987] Brazilian researchers A.G.P. de Sousa et al. report that an increase of 6 kg/m^2 BMI can result in a fourfold risk of developing OSA.[988] And Reena Mehra and Susan Redline report that a 10% weight gain in middle-aged adults predicts about a 32% increase in the severity of OSA and a 6-fold increase in OSA incidence.[989]

OSA can result in significant direct costs to the health care system; indirect costs related to workplace productivity, work accidents, and motor vehicle crashes; and non-financial costs of the burden of disease.[990] Thus, PHAC reports that patients with sleep disordered breathing use health care services prior to diagnosis at approximately twice the rate of those without the disorder.[991] University of British Columbia researchers Nayef AlGhanim et al. found evidence that men suffering from OAS have 50% more occupational accidents than those without the syndrome, and that individuals with OAS "have a three- to sevenfold increased risk of motor vehicle crashes."[992]

In 2006, David Hillman et al. estimated the overall cost of sleep disorders—mainly OSA, insomnia, and periodic limb movements—in Australia (population 20.1 million) in 2004 to be approximately $7.5 billion in U.S. dollars.[993] This was comprised of a total financial cost of $4.524 billion, which represents 0.8% of the Australian gross domestic product (GDP)—including direct health care and associated costs of $459 million, and indirect financial costs of $4.065 billion—plus estimated non-financial costs of suffering of $2.970 billion.

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[984] Carter III, and Watenpaugh. "Obesity and Obstructive Sleep Apnea: Or Is It OSA and Obesity?"

[985] Haslam, and James. "Obesity."

[986] Wolk, and Somers. "Obesity-Related Cardiovascular Disease: Implications of Obstructive Sleep Apnea." p. 254.

[987] Carter III, and Watenpaugh. "Obesity and Obstructive Sleep Apnea: Or Is It OSA and Obesity?"

[988] de Sousa, A. G. P. , C. Cercato, M. C. Mancini, and A. Halpern. "Obesity and Obstructive Sleep Apnea-Hypopnea Syndrome," *Obesity Reviews*, 2008, vol. 9: 340-354.

[989] Mehra, Reena, and Susan Redline. "Sleep Apnea: A Proinflammatory Disorder That Coaggregates with Obesity," *Journal of Allergy and Clinical Immunology*, 2008, vol. 121, no. 5: 1096-1102.

[990] Hillman, David R, Anita Scott Murphy, Ral Antic, and Lynne Pezzullo. "The Economic Cost of Sleep Disorders," *Sleep*, 2006, vol. 29, no. 3: 299-305.

[991] Public Health Agency of Canada (PHAC). *Life and Breath: Respiratory Disease in Canada*, accessed.

[992] AlGhanim, Nayef, Vikram R. Comondore, John Fleetham, Carlo A. Marra, and Najib T. Ayas. "The Economic Impact of Obstructive Sleep Apnea," *Lung*, 2008, vol. 186: 7-12. p. 9.

[993] Hillman, Murphy, Antic, and Pezzullo. "The Economic Cost of Sleep Disorders."

4.11.3 Non-alcoholic fatty liver disease (NAFLD)

According to Ogden et al., obesity is also a common risk factor for non-alcoholic fatty liver disease (NAFLD) and non-alcoholic steatohepatitis, which are diseases "in which normal liver architecture is disrupted by the presence of fat, or in the case of steatohepatitis, fat-induced inflammation and injury to the hepatocyte that may progress to fibrosis and cirrhosis."[994] One report estimated that between 69% and 100% of persons with NAFLD are obese.[995] According to Harvard Medical School researchers, extreme obesity increases fat accumulation in the liver—a condition observed in over 80% of obese individuals with a BMI of ≥40.[996]

Ogden et al. note that there are no good population-based data on the prevalence of NAFLD, mainly because most patients are asymptomatic, although one review estimated the prevalence in the U.S. to be between 3% and 24%.[997] David Haslam and Philip James of the International Obesity Task Force in the U.K. estimate that the prevalence of non-alcoholic steatohepatitis in the general population is between 2% and 9%.[998]

4.12 Mortality

The World Health Organization (WHO) estimates that chronic diseases contribute to more than 60% of global deaths, and to approximately 90% of all deaths in Canada.[999]

Because of an aging population and other factors such as increasing prevalence of obesity and diabetes, mortality rates have been steadily rising in Canada since about 1990.[1000] According to Statistics Canada, the annual number of deaths in Canada rose by 20% between 1990 and 2005—from 191,973 to 230,132.[1001] Between 2004 and 2005 alone, the total number of deaths in Canada increased by 1.6%—from 226,584 to 230,132.[1002]

Statistics Canada also reports that between 1990 and 2005, the number of deaths among females has increased more than twice as fast as the number among males—by 30% and 12%,

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[994] Ogden, Yanovski, Carroll, and Flegal. "The Epidemiology of Obesity." p. 2095.

[995] Sheth, S.G., F.D. Gordon, and S. Chopra. "Nonalcoholic Steatohepatitis," *Annals of Internal Medicine*, 1997, vol. 126: 137–145. Cited in Ogden, Yanovski, Carroll, and Flegal. "The Epidemiology of Obesity."

[996] Khaodhiar, L., K.C. McCowen, and G.L. Blackburn. "Obesity and Its Comorbid Conditions," *Clinical Cornerstone: A Clinical Journal for Primary Care*, 1999, vol. 2: 17-31.

[997] Clark, J.M. "The Epidemiology of Nonalcoholic Fatty Liver Disease in Adults," *Journal of Clinical Gastroenterology*, 2006, vol. 40, no. Suppl 1: S5–S10. Cited in Ogden, Yanovski, Carroll, and Flegal. "The Epidemiology of Obesity."

[998] Haslam, and James. "Obesity."

[999] World Health Organization. *The Impact of Chronic Disease in Canada*, accessed. andWorld Health Organization (WHO). *Preventing Chronic Diseases: A Vital Investment*, accessed.

[1000] Statistics Canada. "Deaths." *The Daily*, January 14, 2008, accessed November 2008; available from http://www.statcan.gc.ca/daily-quotidien/080114/dq080114b-eng.htm.

[1001] Ibid.

[1002] Ibid.

respectively.[1003] In 2005, the mean age at death was 74.2 years—71.1 years for males and 77.4 years for females. The number of male deaths was slightly higher than the number of female deaths:—thus, for every 100 female deaths there were 102 male deaths.[1004] Changing demographics clearly account for a significant portion of these mortality trends, but there is strong evidence that rising rates of obesity and diabetes play an important role.

Thus, WHO predicts that, by 2015, deaths from chronic diseases in Canada will increase by 15%, with deaths from diabetes increasing by 44%.[1005] A team of scientists supported in part by the National Institute of Aging recently reported in the *New England Journal of Medicine* that unless major efforts are made to slow the rising rates of obesity, especially in children, the life expectancy of Americans could decline by as much as five years over the next few decades.[1006] According to the authors S. Jay Olashansky et al.:

> [T]he life-shortening effect of obesity could rise from its current level of about one third to three fourths of a year to two to five years, or more, in the coming decades, as the obese who are now at younger ages carry their elevated risk of death into middle and older ages.[1007]

As shown in Figure 36 below, of all the 226,584 Canadian deaths that occurred in 2004—the latest Statistics Canada data on mortality causes available at the time of writing—32.0% were caused by cardiovascular diseases, 29.5% by cancer, 4.4% by chronic respiratory disease, 3.5% by diabetes, 4.4% by communicable and nutritional deficiencies, 4.1% by injuries, and 22.1% by other chronic and acute diseases.[1008]

[1003] Ibid.

[1004] Ibid.

[1005] World Health Organization. *The Impact of Chronic Disease in Canada*, accessed.

[1006] Olshansky, S. Jay, Douglas J. Passaro, Ronald C. Hershow, Jennifer Layden, Bruce A. Carnes, Jacob Brody, Leonard Hayflick, Robert N. Butler, David B. Allison, and David S. Ludwig. "A Potential Decline in Life Expectancy in the United States in the 21st Century," *New England Journal of Medicine*, 2005, vol. 352, no. 11: 1138-1145.

[1007] Ibid. p. 1138.

[1008] Alberta mortality data are included at the end of this section.

Figure 36. Mortality by selected causes, percentage of total causes of death, Canada, 2004
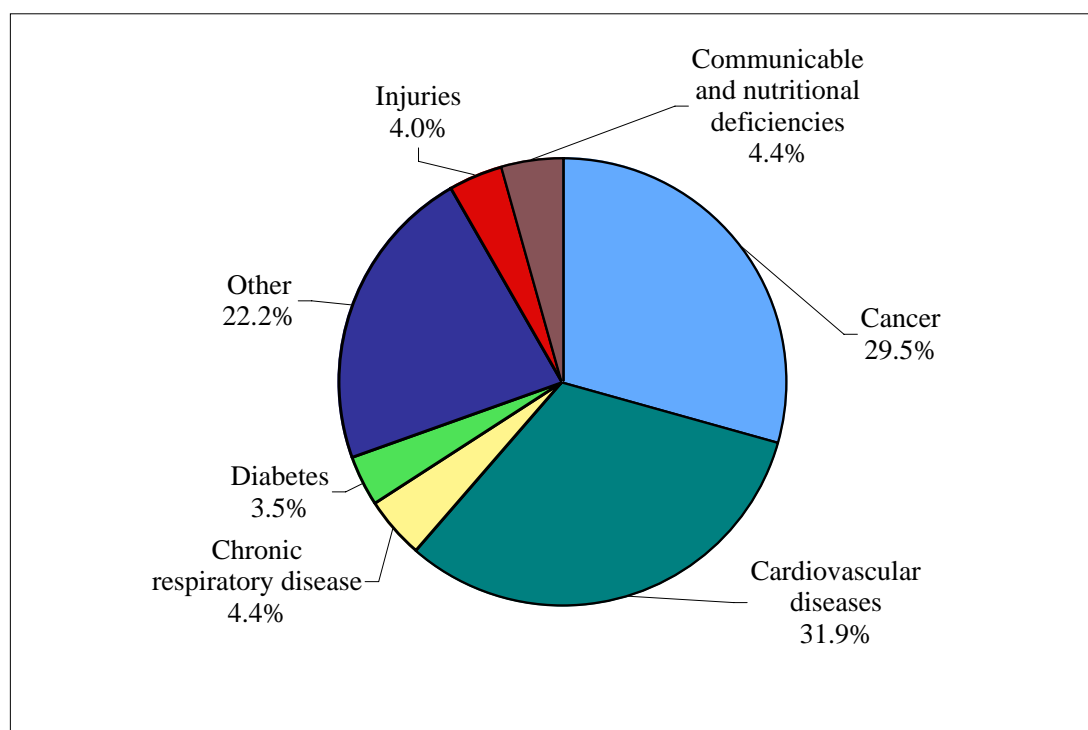
Source: Statistics Canada. *Mortality, Summary List of Causes 2004*, Catalogue no. 84F0209XIE, 2007; accessed November 2008; available from http://www.statcan.gc.ca/pub/84f0209x/84f0209x2004000-eng.pdf.

Obesity is considered to be a risk factor for premature mortality from a number of the chronic diseases that are the main causes of death in Canada—including CVD, cancer, and type 2 diabetes. At the age of 40, obesity has been shown to decrease life expectancy by 7 years, and the magnitude of reduced life expectancy associated with obesity remains substantial until age 75 and older.[1009] A U.S. study found that obese individuals (BMI = ≥30) have a 10–50% increased risk of death from all causes compared with healthy-weight individuals (BMI = 18.5–24.9), with most of the increased risk due to cardiovascular disease and certain cancers.[1010]

The WHO *Comparative Quantification of Health Risks* study found that in 2000 in the America-A subregion (consisting of Canada, United States, and Cuba), the percentages of disease-specific deaths attributable to obesity (BMI ≥30), in adults aged ≥30 years, were: 49.8% of coronary

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[1009] Haslam, and James. "Obesity."
[1010] National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). *Statistics Related to Overweight and Obesity*, 2006; accessed September 2008; available from http://www.niddk.nih.gov/health/nutrit/pubs/statobes.htm.

heart disease deaths, 22.9% of diabetes deaths, 10.3% of stroke deaths, 8.1% of hypertensive disease deaths, 4.4% of colon cancer deaths, 3.0% of postmenopausal breast cancer deaths, and 1.5% of endometrial cancer deaths.[1011]

Estimating the relative risk of mortality associated with BMI is more challenging than estimating the risk of morbidity associated with BMI. As Donna Stroup noted in a U.S. Institute of Medicine workshop: "[S]ignificant scientific questions remain regarding the best methods for assessing the number of deaths and the overall burden of disease from specific modifiable risks and causes. Different investigators examining the same set of issues have used different approaches."[1012]

And as June Stevens notes in an editorial for the *International Journal of Obesity*, the complexity of the BMI-mortality association has "turned out to be deceptively difficult to study," and, especially, that attempting to estimate an association that is free of confounding is a complex task.[1013] However, she argues:

> Confounding is less of a concern if your interest is knowing the association between BMI and mortality, given the naturally occurring assortment of ages, smoking, education, gender, ethnicity, minor illness, major illness and so on existing in the population.[1014]

Analysts have pointed to a number of specific methodological challenges in quantifying this association. For example, longitudinal studies are needed that, preferably, have directly measured the height and weight of participants who are then followed over a period of time, during which the number and causes of death are recorded. The number of deaths occurring over the time span, and the causes of death, can then be estimated by BMI. However, such studies are rare.

Because the current study was not able to estimate relative risk ratios in this way due to data limitations, we have had to rely on mortality-related RRs and PAFs from the epidemiological literature in order to estimate likely mortality risks by BMI level for Alberta. The description below, therefore, provides somewhat detailed information on the range of RRs reported in the literature.

However, Flegal et al. note that even a modest difference between relative risk ratios can make a very substantial difference in obesity-attributable mortality estimates. In a study specifically designed to estimate possible bias in estimating deaths attributable to obesity, they found that a difference of 0.20 between relative risk ratios almost doubled the number of deaths attributed to obesity (97% overestimation).[1015] Therefore, although we have consistently tried to be

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[1011] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."
[1012] Stroup, Donna. "Rationale for Actual Causes of Death, 2000," in Institute of Medicine. *Estimating the Contributions of Lifestyle-Related Factors to Preventable Death: A Workshop Summary*, accessed. p. 19.
[1013] Stevens, June. "Editorial: BMI and Mortality: Sorting through the Data to Find the Public Health Message," *International Journal of Obesity*, 2008, vol. 32: 727-729.
[1014] Ibid. p. 727.
[1015] Flegal, Graubard, and Williamson. "Methods of Calculating Deaths Attributable to Obesity."

conservative in our own estimates of the portion of mortality attributable to excess weight in Alberta in this study, these estimates must be interpreted with caution—primarily because they are based on RRs and PAFs from the epidemiological literature that in turn are conditioned by a wide range of potentially confounding factors underlying those studies and that therefore show a fairly wide range.

In particular, researchers suggest that when gender and age are not considered in estimations of obesity-attributable mortality, considerable bias can occur, since both gender and age significantly influence the outcomes. Flegal et al. found that when the methodology they used in their hypothetical study did not completely adjust for confounding of the obesity-mortality relation by age and sex, deaths due to obesity were overestimated by 17%.[1016] We have therefore taken this potential bias into consideration in the methodology used in this report for estimating obesity-attributable mortality.

Another key issue in this equation is that higher mortality rates are frequently seen—in the epidemiological literature—in underweight people compared to those with normal weights. This has led researchers to the conclusion that smoking prevalence or undiagnosed diseases might also be confounding the rates. According to James et al.:

> There has been extensive discussion over the last 30 years regarding the repeated finding of higher mortality rates associated with lower BMIs. It was recognized that the original inclusion of data from smokers in such calculations had a marked effect because smokers are at greater risk of mortality, but tend to be thinner because of their reduction in appetite and their increased metabolic rate, that is, increased total energy expenditure, which leads to lower body weights when these effects are in energy balance. Thus the excess of smokers in the group of "thin" adults imposes higher mortality rates on the group overall, despite the lower BMIs.

> Further, the mortality rates of the groups with low BMIs may be enhanced by the presence of individuals with as yet undiagnosed diseases, for example, cancer, who may have lost weight before symptoms emerged or a diagnosis was made. A convention has therefore developed whereby the early deaths are excluded and only those deaths that occur 2–5 years after the initiation of any study are considered. By doing this, it is frequently found that the U-shaped curve converts to a J-shaped curve or log-linear relationship.[1017]

However, Katzmarzyk et al. note that excluding deaths from the first few years of follow-up is likely to be ineffective at controlling for confounding:

> For example, the results of a meta-analysis of 29 studies and 1,954,345 subjects indicated that eliminating early deaths shifted the BMI associated with minimum mortality only 0.4 units for men and 0.6 units for women at age 50. Although the results were statistically

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[1016] Ibid.

[1017] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)." p. 565

significant, the clinical significance of the shift is quite small.[1018]

In addition, as Luo et al. note, when the purpose of the study is to estimate the burden of obesity for the entire population, it is not appropriate to exclude portions of the population. Therefore, "it is most appropriate to use relative risks that apply to the entire population."[1019] In other words, the best intentioned adjustments for potentially confounding factors raise other methodological challenges in estimations of obesity-attributable mortality. And yet, failing to make such adjustments is hardly an option, as the following analysis demonstrates.

A recent study conducted by British researchers Debbie Lawlor et al. examined the differences in relative risks of mortality attributable to excess weight when the analysis included the total sample compared to when it only included never-smokers and deaths that occurred after the first few years of follow-up.[1020] The authors found significantly higher BMI-attributable mortality risks in the latter analysis.

The Lawlor et al. study—one of the few that report mortality by specific causes of death—used data from a large prospective study in Scotland (the Renfrew/Paisley study cited earlier) that included 8,327 women and 7,017 men.[1021] Participants who were aged 45–64 at the beginning of the study in 1970–1976 were followed for 28 years until 2004. Their height and weight were directly measured at the beginning of their enrolment in the study. During the 28-year follow-up period, 5,242 of the men and 5,019 of the women in the sample died. As noted, Lawlor et al. first conducted their analysis for the whole sample, and then again including only never-smokers and excluding the first five years of deaths—as in the convention cited by James et al. above.[1022]

The researchers first calculated the proportions of never-smokers and current cigarette smokers in each BMI category. In the underweight category, 83% of men and 78.8% of women were current smokers, and 7.5% and 19.0% respectively were never-smokers. In the normal weight category, 68.4% of men and 55.4% of women were current smokers, and 13.0% and 37.1% respectively were never-smokers. In the overweight weight category, 49.8% of men and 39.1% of women were current smokers, and 18.6% and 52.9% respectively were never-smokers. And in the obese weight category, 42.9% of men and 33.8% or women were current smokers, and 22.3% and 59.0%,respectively were never-smokers. These adjustments are reflected in the absolute number of deaths provided for the second analysis, as seen in Table 30 below.

Table 30 below shows the results of both analyses, both for all-cause mortality and for specific causes of mortality by gender and BMI. In the whole sample (unadjusted for smoking and year of death), both men and women who were underweight had the highest risk of mortality both for

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[1018] Katzmarzyk, Peter T., Ian Janssen, and Christopher I. Ardern. "Physical Inactivity, Excess Adiposity and Premature Mortality," *Obesity Reviews*, 2003, vol. 4: 257-290.

[1019] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada." p. 141.

[1020] Lawlor, Debbie A., Carole L. Hart, David J. Hole, and George Davey Smith. "Reverse Causality and Confounding and the Associations of Overweight and Obesity with Mortality," *Obesity*, 2006, vol. 14, no. 12: 2294-2304.

[1021] Ibid.

[1022] Ibid.

all-cause mortality and for every type of cause investigated. The sharply increased risks of death from lung cancer and respiratory deaths for underweight subjects, revealed in Table 30 below, reflects the strong influence of including the greater number of smokers in this category in the first analysis. The researchers found no, or very little, association between overweight and mortality, and only "weak to modest associations" between obesity and mortality.[1023]

In the analysis that included only never-smokers and removed the first five years of deaths, both overweight and obesity were associated with an increase in all-cause mortality among both men and women.[1024] The risk among underweight adults was not reported for the second analysis. Among overweight men, the RR increased significantly from 0.90 in the first analysis to 1.38 in the second analysis, and among obese men, the RR increased from 1.80 to 2.10. Among overweight women, the RR increased from 0.97 in the first analysis to 1.28 in the second analysis, and among obese women, the RR increased from 1.12 to 1.56. The changed risks for specific causes of death showed a similar pattern.

Table 30. Age-adjusted relative risks of all-cause and cause-specific mortality by BMI categories, for total sample and never-smoker sample excluding first five years of deaths, by gender, as estimated by Lawlor et al. from the Renfrew/Paisley study in Scotland, 1976–2004

	Total Sample				Never Smokers		
	UW	Norm	OW	Ob	Norm	OW	Ob
MEN							
Number of participants	53	2805	3410	749	347	605	160
All-cause mortality							
No. of deaths	45	2123	2483	591	175	374	117
RR	1.34	1.00	0.90	1.08	1.00	1.38	2.10
CVD							
No of deaths	13	968	1307	355	100	205	73
RR	0.85	1.00	1.04	1.42	1.00	1.32	2.27
CHD							
No. of deaths	9	623	876	246	48	132	47
RR	0.91	1.00	1.09	1.52	1.00	1.73	2.84
Stroke							
No. of deaths	4	216	275	63	38	53	16
RR	1.21	1.00	0.97	1.16	1.00	0.94	1.48
Cancer							
No. of deaths	10	655	713	138	35	92	23
RR	0.97	1.00	0.84	0.82	1.00	1.68	1.98
Lung cancer							
No. of deaths	6	209	259	30	–	–	–
RR	1.26	1.00	0.67	0.39	–	–	–

[1023] Ibid.
[1024] Ibid.

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All smoking-related cancers							
No. of deaths	7	461	449	81	–	–	–
RR	0.96	1.00	0.75	0.68	–	–	–
Respiratory disease							
No. of deaths	14	269	194	38	–	–	–
RR	3.37	1.00	0.55	0.56	–	–	–
WOMEN							
Number of participants	189	3824	3061	1253	1385	1569	698
All-cause mortality							
No. of deaths	144	2177	1804	894	660	849	477
RR	1.80	1.00	0.97	1.28	1.00	1.12	1.56
CVD							
No of deaths	57	1024	895	499	318	431	278
RR	1.50	1.00	1.00	1.48	1.00	1.15	1.84
CHD							
No. of deaths	29	539	489	270	155	222	142
RR	1.43	1.00	1.06	1.53	1.00	1.23	1.93
Stroke							
No. of deaths	20	344	282	154	130	141	95
RR	1.57	1.00	0.91	1.32	1.00	0.90	1.52
Cancer							
No. of deaths	38	640	506	206	172	226	97
RR	1.58	1.00	0.96	1.05	1.00	1.17	1.26
Lung cancer							
No. of deaths	16	181	88	28	–	–	–
RR	2.38	1.00	0.60	0.52	–	–	–
All smoking-related cancers							
No. of deaths	23	307	208	69	–	–	–
RR	2.03	1.00	0.83	0.75	–	–	–
Respiratory disease							
No. of deaths	31	231	151	61	–	–	–
RR	3.85	1.00	0.75	0.83	–	–	–

Notes: UW – Underweight (BMI <18.5), Norm – Normal (BMI 18.5–<25), OW – Overweight (25 – <30), Ob – Obese (BMI ≥30); Never-smokers – Partial sample includes only never-smokers, and excludes the first 5 years of deaths; – indicates that no data was available.

Source: Adapted from: Lawlor, Debbie A., Carole L. Hart, David J. Hole, and George Davey Smith. "Reverse Causality and Confounding and the Associations of Overweight and Obesity with Mortality," *Obesity*, 2006, vol. 14, no. 12: 2294-2304.

Between 1982 and 1996, the American Cancer Society conducted one of the most comprehensive studies ever undertaken on obesity and mortality.[1025, 1026] The study examined data from over one million participants aged ≥30 years at the time of their enrolment in 1982 in the U.S. Cancer Prevention Study II. In the initial interview, participants reported both their current height and weight and their height and weight one year previously. Deaths that occurred between enrolment and the end of 1996—accounting for 20.1% of the enrolled participants—as well as the causes of these deaths, were determined first by personal inquiries by volunteers and then confirmed through linkage with the National Death Index.

Not surprisingly, the study found that the relative risk for all causes of death was generally higher in men than in women and that it declined with age. But the absolute risk of death associated with excess weight was found to be highest in the oldest age groups. The study also found that obese individuals had a higher rate of premature death from all causes even if they did not smoke and were otherwise healthy. Severely obese men (BMI ≥40) who didn't smoke or have a history of disease were still over 2.5 times more likely to die prematurely than men with normal weight (BMI 18.5–24.9) who didn't smoke or have a history of disease, and severely obese women in this category were 2.0 times more likely to die prematurely than were women with normal weight.

Harvard University endocrinologist, Jo Ann Manson, concludes from the study, "The evidence is now compelling and irrefutable. Obesity is probably the second-leading preventable cause of death in the United States after cigarette smoking, so it is a very serious problem."[1027]

In addition to all-cause mortality, the American Cancer Society study also examined the relationship between BMI and death from all cancers and from specific types of cancer.[1028] It found that overweight and obesity in U.S. adults accounted for 4.2% of cancer mortality in men and 14.3% in women. Among those adults who had never smoked, the percentages were significantly higher, with overweight and obesity responsible for 14.2% of all cancer deaths in men and 19.8% of those in women.

Table 31 below provides the RRs of mortality that were estimated in the study for both men and women, both for all cancers and for specific cancer sites, by BMI category when compared to normal weight adults. In general, there was seen to be an increase in risk for all types of cancer mortality with each increase in BMI level from overweight through obese class 3, with the exception of leukemia, multiple myeloma, and stomach cancer in females in the obese class 2 category.

[1025] Calle, Eugenia E., Michael J. Thun, Jennifer M. Petrelli, Carmen Rodriguez, and Clark W. Heath Jr. "Body-Mass Index and Mortality in a Prospective Cohort of U.S. Adults," *New England Journal of Medicine*, 1999, vol. 341, no. 16: 1097-1106.

[1026] Calle, Rodriguez, Walker-Thurmond, and Thun. "Overweight, Obesity, and Mortality from Cancer in a Prospectively Studied Cohort of U.S. Adults."

[1027] Associated Press. "The Fatter You Are, the Shorter Your Life: Study." *Jet*, October 28, 1999, accessed September 2008; available from http://findarticles.com/p/articles/mi_m1355/is_21_97/ai_57564173/print?tag=artBody;col1.

[1028] Calle, Rodriguez, Walker-Thurmond, and Thun. "Overweight, Obesity, and Mortality from Cancer in a Prospectively Studied Cohort of U.S. Adults."

For all cancers, there was no excess risk for overweight men (RR 0.97), and only a slight excess risk for overweight women (RR 1.08). Among men in obese classes 1, 2, and 3, however, the all-cancer mortality relative risks increased from 1.09, to 1.20, to 1.52, respectively, compared to normal weight men. Among women in obese classes 1, 2, and 3, the risks increased from 1.23, to 1.32, to 1.62, respectively.

As might be expected, adults in the most severe obesity category—class 3 (BMI $\geq$40)—had the highest RRs of any weight category for specific cancer sites, with the highest risks found for cancers of the kidney (RR 4.75) and uterus (RR 6.25). However, small sample sizes prohibited estimations for this group for most of the cancer sites. In obese class 2, the highest relative risk was found among males for liver cancer (RR 4.52). In the obese class 1 category, with the exception of cancer of the uterus (RR 2.53), the risk for all cancer sites was lower than 2.00—ranging from RR 1.12 for leukemia in females to RR 1.90 for male liver cancer.

Table 31. Relative risks of cancer mortality for all cancers and specific cancer sites by body mass index and gender, aged ≥30 years, from the U.S. Cancer Prevention Study II, 1982–1998

Type of Cancer Death	Gender	Body Mass Index – RR			
		Overweight 25.0-29.9	Obese, class 1 30.0-34.9	Obese, class 2 35.0-39.9	Obese, class 3 ≥40.0
All cancers	male	0.97	1.09	1.20	1.52
	female	1.08	1.23	1.32	1.62
esophagus	male	1.15	1.28	1.63	—
	female	1.20	1.39	—	—
stomach	male	1.01	1.20	1.94	—
	female	0.89	1.30	1.08	—
colorectal	male	1.20	1.47	1.84	—
	female	1.10	1.33	1.36	1.46
liver	male	1.13	1.90	4.52	—
	female	1.02	1.40	1.68	—
pancreas	male	1.13	1.41	1.49	—
	female	1.11	1.28	1.41	2.76
bladder	male	1.03	1.14	—	—
	female	1.02	1.34	1.34	—
kidney	male	1.18	1.36	1.70	—
	female	1.33	1.66	1.70	4.75
non-Hodgkin's lymphoma	male	1.08	1.56	1.56	—
	female	1.22	1.20	1.95	—
multiple myeloma	male	1.18	1.44	1.44	—
	female	1.12	1.47	1.44	—
leukemia	male	1.14	1.37	1.37	—
	female	1.05	1.12	0.93	—
prostate	male	1.08	1.20	1.34	—
breast	female	1.34	1.63	1.70	2.12
uterus	female	1.50	2.53	2.77	6.25
ovary	female	1.15	1.16	1.51	—

Notes: The BMI categories with blank cells did not have a sufficient number of deaths to enable an estimation of the relative risk ratio. All of the RRs have been estimated by comparing the number of deaths within each BMI category with the number of deaths in the normal weight category (BMI 18.5-24.9), which therefore all have a RR of 1.00.

RRs have been adjusted for age, education, smoking status and number of cigarettes smoked, physical activity, alcohol use, marital status, race, aspirin use, fat consumption, and vegetable consumption.

Source: Calle, Eugenia E., Carmen Rodriguez, Kimberly Walker-Thurmond, and Michael J. Thun. "Overweight, Obesity, and Mortality from Cancer in a Prospectively Studied Cohort of U.S. Adults," *New England Journal of Medicine*, 2003, vol. 348: 1625-1638.

In 2007, Katherine Flegal et al. of the U. S. Centers for Disease Control and Prevention conducted a very important study that investigated the relationship between BMI and mortality from cardiovascular disease, cancer, and all other non-cancer and non-CVD deaths in U.S. adults aged ≥25 years, which updated an earlier 2005 study.[1029, 1030] The study's importance, including its adjustment for age, is explained below. Illness subcategories that were included by Flegal et al. were:

- for CVD — coronary heart disease, and 'other CVD';
- for cancer — lung cancer, 'obesity-related cancers' (cancers of the colon, breast, esophagus, uterus, ovary, kidney, and pancreas combined), and all other cancers; and
- for all other deaths–non-cancer and non-CVD— subdivided into diabetes and kidney disease combined, chronic respiratory disease, acute respiratory and infectious disease, injuries, and miscellaneous.

Unfortunately, Flegal et al.'s 2007 article did not give the actual RR numbers for cause-specific deaths. However, Flegal et al.'s earlier 2005 study did provide RRs for all-cause mortality by BMI category for three age groups.[1031] The RRs obtained from the earlier 2005 study were used in Luo et al.'s study of the burden of adult obesity in Canada described below.[1032] Because the Flegal et al. findings have been applied specifically to Canada and are therefore directly relevant to this obesity cost study, the results are described here in some detail.

Luo et al. explain their rationale for using the RRs as estimated by Flegal et al.:

> 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; as a result, Flegal estimated fewer deaths attributable to obesity. 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. In other words, the obese of today are less likely to die of coronary heart disease than the obese of 40 years

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[1029] Flegal, Katherine M., Barry I. Graubard, David F. Williamson, and Mitchell H. Gail. "Cause-Specific Excess Deaths Associated with Underweight, Overweight, and Obesity," *JAMA - Journal of the American Medical Association*, 2007, vol. 298, no. 17: 2028-2037.

[1030] Flegal, Katherine M., Barry I. Graubard, and David F. Williamson. "Excess Deaths Associated with Underweight, Overweight, and Obesity," *JAMA - Journal of the American Medical Association*, 2005, vol. 293, no. 15: 1861-1867.

[1031] Ibid.

[1032] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

ago because of advances in the treatment of comorbid conditions such as dyslipidemia and hypertension, and because of improved treatments such as cardiac revascularization.[1033]

As explained above by Luo et al., the data that Flegal et al. used to estimate relative risks came from the National Health and Nutrition Examination Survey (NHANES) program of the National Center for Health Statistics, which directly measured the height and weight of respondents. The total number of deaths came from U.S. vital statistics. Flegal et al. were able to use the NHANES data to estimate RRs for mortality since the samples include more than 15 years of follow-up data and include mortality statistics as well as BMI data.

Relative risks, which were adjusted for smoking status, race, and alcohol consumption, were estimated for the BMI categories of underweight (BMI <18.5), overweight (BMI 25–<30), obese class 1 (BMI 30-<35), and obese classes 2 and 3 combined (severe obesity—BMI ≥35), compared to normal weight, for each category of cause of death, and for three age groups. Flegal et al. then applied the relative risks for a given age group to the current distribution of BMI in that age group in the general population, as estimated from the NHANES 1999–2002 data.

The RRs for all-cause mortality by BMI and for three age groups—25–59 years, 60–69 years, and ≥70 years, as calculated by Flegal et al. are shown in Table 32 below. In general, both obesity and severe obesity were associated with increased risk—most markedly in the youngest age group. And relative risks were generally lowest in the oldest age group. Those aged 25–59 had the highest relative risk in both the obese class 1 category (RR 1.20) and the severely obese category (RR 1.83) compared to normal weight adults in the same age group. Those aged ≥70 years had the lowest relative risks in both the obese class 1 (RR 1.03) and the obese classes 2–3 categories (RR 1.17) compared to normal weight individuals in the same age group.

The researchers found that adults aged 60–69 years and ≥70 years had the highest risks of all-cause mortality if they were underweight (RR 2.30 and RR 1.69, respectively). Overweight adults (BMI 25–<30) showed no risk in any age group.

According to the authors, when the analysis was repeated with the exclusion of participants who had reported a prior history of the disease in question, had ever smoked, died in the first three years of follow-up, or were older than 70 years when their height and weight were measured, there was only a small effect on the estimates (data not shown).[1034]

[1033] Ibid. p. 141.
[1034] Flegal, Graubard, and Williamson. "Excess Deaths Associated with Underweight, Overweight, and Obesity."

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Table 32. Relative risk for all-cause mortality by BMI for three age groups, U.S., 2004

BMI level	Relative Risk (95% Confidence Interval) by Age Category		
	25 - 59 years	60 - 69 years	≥70 years
<18.5	1.38 (0.82-2.32)	2.30 (1.70-3.13)	1.69 (1.38-2.07)
18.5 - <25	1.00	1.00	1.00
25 - <30	0.83 (0.65-1.06)	0.95 (0.80-1.13)	0.91 (0.83-1.01)
30 - <35	1.20 (0.84-1.72)	1.13 (0.89-1.42)	1.03 (0.91-1.17)
≥35	1.83 (1.27-2.62	1.63 (1.16-2.30)	1.17 (0.94-1.47)

Source: Flegal, Katherine M., Barry I. Graubard, and David F. Williamson. "Excess Deaths Associated with Underweight, Overweight, and Obesity," *JAMA - Journal of the American Medical Association*, 2005, vol. 293, no. 15: 1861-1867.

Flegal et al. estimated PAFs by BMI category for all-cause mortality and for each cause of death.[1035] The number of deaths for each cause that could be attributed to excess weight was then calculated by multiplying to total number of deaths in the age group in 2004 by the corresponding PAF. The authors found that the association between excess weight and mortality varies considerably by cause of death.

Flegal et al. found that cardiovascular disease (CVD) accounted for 37% of adult deaths that occurred in the U.S. in 2004, with 13% of those total CVD deaths attributable to obesity (BMI ≥30). For the CVD mortality sub-groups—coronary heart disease and 'other CVD' including stroke—obesity, but not underweight or overweight, was significantly associated with excess mortality.

In 2004, cancer accounted for approximately 24% of total deaths in the U.S. For the total cancer, lung cancer, and 'all other cancer' categories, there was no significant association between excess mortality and any BMI category, but there was a significant association between obesity and the listed cluster of obesity-related cancers in particular. Obesity-related cancers—cancers of the colon, breast, esophagus, uterus, ovary, kidney, and pancreas combined—accounted for approximately 32% of cancer deaths. Obesity was found to be related to 11.4% of deaths from the obesity-related cancers combined, but overweight showed no association.

Flegal et al. note that there is some evidence that overweight may actually improve prognosis and survival during recovery from some medical procedures, infections, and other adverse conditions, perhaps due to greater nutritional reserves.

Other non-cancer and non-CVD deaths in 2004 accounted for approximately 39% of total deaths in the U.S. Both underweight (mainly from respiratory causes) and obesity classes 2–3 combined were associated with increased mortality from other all non-CVD, non-cancer causes combined.

[1035] Flegal, Graubard, Williamson, and Gail. "Cause-Specific Excess Deaths Associated with Underweight, Overweight, and Obesity."

Among sub-categories of these other deaths, only diabetes and kidney disease combined were significantly and positively associated with increased mortality in both the overweight and obese categories.

However, Flegal et al. note that diabetes may be underreported as a cause of death. For example, they cite one study by McEwen et al. that showed that only 39% of death certificates for individuals who had diabetes and died of CVD actually reported diabetes.[1036] Other studies confirm that, because diabetes so often leads to other complications and illnesses, it is generally under-reported on death certificates. According to Health Canada:

> There were 5,447 deaths in 1996 for which diabetes was certified as the underlying cause. This ranks diabetes as the seventh leading cause of death in Canada. However, the actual number of deaths for which diabetes was a contributing factor is probably five times this number.[1037]

The U.S. Centers for Disease Control similarly report:

> Actually diabetes contributes to a much larger proportion of mortality, since it is reported on only about half of the death certificates for persons who die with the disease and is listed as the underlying cause on only one-quarter of the certificates on which it appears. The most frequent causes of death among persons with diabetes are ischemic and other forms of heart disease, cerebrovascular disease, and other forms of atherosclerosis; renal disease, including nephritis/nephrosis and uremia; respiratory disease; and infection.[1038]

In sum, the association between obesity and diabetes-related mortality may actually be larger than indicated in the results of Flegal et al., which relied on the official record of numbers of deaths by cause as reported in the U.S. vital statistics and NHANES databases.

One of the most recent studies investigating the BMI-mortality connection was conducted in Sweden by Gunilla Weitoft et al., of the Swedish National Board of Health and Welfare and Umeå University, with results reported in 2008.[1039] A sample of 23,580 random respondents in the Swedish population, who were aged 16–74 years in 1980–81 and 1988–89, and who were interviewed and reported their height and weight in those years, were then followed for 12 years to estimate all-cause mortality, and mortality from circulatory diseases in particular. Sweden's use of personal identification numbers for all citizens enables this longitudinal follow-up for all

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[1036] McEwen, Laura N., Catherine Kim, Mary Haan, Debashis Ghosh, Paula Lantz, Carol Mangione, Monika Safford, David Marrero, Theodore Thompson, and William Herman. "Diabetes Reporting as a Cause of Death: Results from the Translating Research into Action for Diabetes (TRIAD) Study," *Diabetes Care*, 2006, vol. 29, no. 2: 247-253. Cited in Flegal, Graubard, Williamson, and Gail. "Cause-Specific Excess Deaths Associated with Underweight, Overweight, and Obesity."

[1037] Health Canada, *Diabetes in Canada*, available at: http://www.hc-sc.gc.ca/hpb/lcdc/publicat/diabet99/d02_e.html

[1038] Centers for Disease Control, op. cit., page 34. *MMWR,* 21 November, 1986, 35. 46: pp. 711-714, available at http://www.cdc.gov/epo/mmwr/preview/mmwrhtml/00000824.htm.

[1039] Weitoft, Gunilla Ringback, Mats Eliasson, and Mans Rosen. "Underweight, Overweight and Obesity as Risk Factors for Mortality and Hospitalization," *Scandinavian Journal of Public Health*, 2008, vol. 36: 169-176.

causes of death. As was found by Flegal et al., both underweight and obesity, but not overweight, were associated with increased risks of all-cause mortality.

Gunilla Weitoft et al. found that—when compared to those with normal weight—underweight men (RR 3.1) and women (RR 2.2) and obese men (RR 1.5) and women (RR 1.5) all had increased risks of mortality from any cause.[1040] For mortality from circulatory diseases, underweight men (RR 1.4) and women (RR 1.8) and obese men (RR 1.8) and women (RR 1.7) all had increased risks. The RRs for both overweight men (RR 1.0) and women (RR 1.1) for total mortality, and for overweight men (RR 1.1) for circulatory disease mortality did not show an increased risk. However, overweight women (RR 1.4) had a 40% increased risk for circulatory disease mortality.

A large number of U.S. and international investigators, including Daniel McGee and approximately 35 members of the Diverse Populations Collaboration, recently conducted a meta-analysis of 26 studies in their database that were from the U.S. and other (mainly European) countries to estimate the risks associated with BMI for all-cause mortality and three specific causes of death—cardiovascular disease in general, coronary heart disease, and cancer.[1041] Basically, they found that, relative to adults with normal weight, there was no or only slight excess risk of mortality among overweight adults. But adult obesity (BMI ≥30) was associated with a summary RR of 1.22 for all-cause mortality, 1.57 for coronary heart disease mortality, 1.48 for cardiovascular disease mortality in general, and 1.07 for cancer mortality.[1042]

Table 33 below provides the results of this particular analysis stratified by gender. Among obese adults, the risks for all-cause mortality were similar for males and females (RR 1.201 and 1.275, respectively). Among both obese males and females, risks were somewhat higher for coronary heart disease (RR 1.508 and 1.624, respectively) than for cardiovascular disease in general (RR 1.453 and 1.529, respectively). And risks of cancer showed much lower relative risk ratios among both obese males and females (RR 1.055 and 1.103, respectively) than for the other causes of mortality examined.

[1040] Ibid.

[1041] McGee, and Diverse Populations Collaboration. "Body Mass Index and Mortality: A Meta-Analysis Based on Person-Level Data from Twenty-Six Observational Studies."

[1042] Ibid.

Table 33. Summary relative risks of death for all-cause mortality, cardiovascular disease, coronary heart disease, and cancer, by BMI category (overweight—BMI 25–<30, and obesity—BMI ≥30) and by gender, as estimated by McGee et al. (2005)

Cause of death	Gender	BMI	Summary RR
All-cause	male	overweight	0.965
		obese	1.201
	female	overweight	0.968
		obese	1.275
Cardiovascular disease	male	overweight	1.096
		obese	1.453
	female	overweight	1.029
		obese	1.529
Coronary heart disease	male	overweight	1.159
		obese	1.508
	female	overweight	1.097
		obese	1.624
Cancer	male	overweight	0.932
		obese	1.055
	female	overweight	0.985
		obese	1.103

Note: RRs are a summary of risk ratios found in 26 studies.
Source: McGee, D.L., and Diverse Populations Collaboration. "Body Mass Index and Mortality: A Meta-Analysis Based on Person-Level Data from Twenty-Six Observational Studies," *Annals of Epidemiology*, 2005, vol. 15: 87-97.

Results from a massive new European study that examines the risk of death related to a larger-than-usual range of BMI categories were recently published in the November 18, 2008, issue of *The New England Journal of Medicine.*[1043] European investigators T. Pischon of the German Institute of Human Nutrition and 47 other international colleagues examined the association of BMI and abdominal adiposity with the risk of death among 359,387 participants from nine countries in the European Prospective Investigation into Cancer and Nutrition (EPIC) study.

The study participants, who were aged 25–70 years of age at the time of their enrolment in the study—from 1992 through 2000—all underwent anthropometric measurements including directly measured height and weight. Participants were excluded from the study is they reported a history of cancer, heart disease, or stroke at the time of enrolment. They were followed for a mean of 9.7 years, during which 14,723 participants died—5,429 from cancer (neoplasms), 3,443

[1043] Pischon, T., H. Boeing, K. Hoffmann, and 45 Other Colleagues. "General and Abdominal Adiposity and Risk of Death in Europe," *The New England Journal of Medicine*, 2008, vol. 359, no. 20: 2105-2120.

from circulatory causes, 637 from respiratory causes, 2,209 from other causes, and 3,005 from unspecified causes.

Table 34 below shows the results of the study, which is one of the very few studies that have reported both unadjusted and adjusted relative risks for the overall cause of death. In addition, RRs are provided for the following general disease categories—neoplasms, circulatory diseases, respiratory diseases, and 'other' diseases, and also specifically for ischemic heart disease, cerebrovascular disease, colorectal cancer, prostate cancer, and breast cancer.

Pischon et al. found a significant association of BMI with risks of death—with increased risks indicated among those who were in both the underweight and obese categories. With the exception of the risk of death from ischemic heart disease among women (RR 3.28) in the most obese category (≥35), men otherwise had a significantly greater risk of death than did women in all other BMI categories and for all other causes of death. (Although not shown in Table 34 below, after adjustments for BMI, waist circumference and waist-to-hip ratios showed even higher risk ratios for mortality than those for BMI alone.)

Among the general causes of death, circulatory diseases had the strongest association between obesity and risk of death for both men (RR 2.7 for BMI ≥35, and RR 1.62 for BMI 30–<35), and women (RR 2.27 for BMI ≥35, and RR 1.31 for BMI 30–<35). Circulatory diseases also had a significant association with underweight among men (RR 1.84) but not among women (RR 1.09). Respiratory disease mortality had the strongest associations with underweight for both men (RR 6.53) and women (RR 4.74).

Among specific diseases, there were strong associations between obesity and risk of death for:
- ischemic heart disease among both men (RR. 2.64 for BMI ≥35, and RR 1.72 for BMI 30–<35) and women (RR. 3.28 for BMI ≥35, and RR 1.44 for BMI 30–<35),
- colorectal cancer among men (RR 2.55 for BMI ≥35, and RR 2.66 for BMI 30–<35), but not among women,
- breast cancer among women (RR. 1.79 for BMI ≥35, and RR 1.55 for BMI 30–<35),
- cerebrovascular disease among men (RR 1.86 for BMI ≥35, and RR 1.27 for BMI 30–<35) and among severely obese women (RR 1.4), and
- prostate cancer among severely obese men (RR 2.04).

Table 34. Relative risk of death among men and women in the European Prospective Investigation into Cancer and Nutrition (EPIC) study, by BMI category, cause of death, gender, and age group, as estimated by T. Pischon et al. (2008)

Relative Risk	BMI								
	<18.5	18.5-<21.0	21.0-<23.5	23.5<25	25.0-<26.5	26.5-<28.0	28.0-<30.0	30.0-<35.0	≥35.0
Men									
Person years	4,504	45,494	176,701	188, 823	215,370	186,758	177,458	157,899	24,794
Deaths (no.)	92	390	1,071	1,144	1,209	1,118	1,212	1,256	380
Overall RR									
Unad-justed	2.86 (2.30-3.57)	1.64 (1.46-1.84)	1.08 (0.99-1.17)	1.00	0.90 (0.83-0.98)	0.95 (0.88-1.03)	1.09 (1.01-1.18)	1.28 (1.18-1.39)	2.06 (1.81-2.34)
Adjust-ed *	2.30 (1.84-2.86)	2.30 (1.84-2.86)	1.39 (1.24-1.57)	1.00	0.91 (0.84-0.99)	0.96 (0.88-1.04)	1.08 (1.00-1.17)	1.24 (1.14-1.35)	1.94 (1.71-2.20)
RR by sub-group									
Age									
<55 yr	4.43 (3.01-6.54)	1.66 (1.33-2.07)	1.07 (0.90-1.26)	1.00	1.09 (0.93-1.28)	0.91 (0.77-1.08)	1.16 (0.98-1.37)	1.41 (1.20-1.66)	1.98 (1.55-2.53)
55-<65	2.27 (1.55-3.33)	1.30 (1.09-1.55)	1.03 (0.91-1.17)	1.00	0.83 (0.73-0.93)	0.97 (0.86-1.09)	1.06 (0.94-1.19)	1.22 (1.08-1.37)	2.02 (1.69-2.41)
≥65	1.56 (1.07-2.27)	1.32 (1.06-1.65)	1.00 (0.85-1.17)	1.00	0.91 (0.77-1.06)	0.99 (0.84-1.16)	1.06 (0.90-1.24)	1.10 (0.93-1.30)	1.63 (1.21-2.19)
Cause of death									
Neo-plasms	1.20 (0.73-1.97)	1.27 (1.03-1.55)	0.92 (0.80-1.07)	1.00	0.82 (0.72-0.95)	0.91 (0.79-1.04)	0.93 (0.81-1.07)	0.94 (0.82-1.09)	1.24 (0.97-1.60)
Circula-tory	1.84 (1.19-2.87)	1.05 (0.81-1.36)	0.97 (0.81-1.15)	1.00	0.95 (0.80-1.12)	1.01 (0.85-1.20)	1.28 (1.09-1.51)	1.62 (1.38-1.90)	2.70 (2.13-3.42)
Respira-tory	6.53 (3.56-11.97	2.29 (1.46-3.59)	1.21 (0.84-1.76)	1.00	0.66 (0.44-0.99)	0.74 (0.49-1.10)	0.74 (0.50-1.11)	0.90 (0.60-1.34)	1.65 (0.90-3.03)
Other	4.67 (2.98-7.31)	1.02 (1.54-2.66)	1.30 (1.05-1.60)	1.00	0.99 (0.80-1.22)	0.94 (0.75-1.17)	1.12 (0.90-1.40)	1.26 (1.01-1.57)	2.15 (1.57-2.96)
Ische-mic heart disease	1.17 (0.54-2.56)	1.10 (0.77-1.57)	1.02 (0.80-1.31)	1.00	1.11 (0.89-1.40)	1.13 (0.89-1.43)	1.50 (1.20-1.88)	1.72 (1.37-2.16)	2.64 (1.86-3.73)
Cerebro vascular disease	1.41 (0.49-4.12)	1.39 (0.78-2.46)	0.90 (0.58-1.39)	1.00	0.99 (0.66-1.49)	1.14 (0.76-1.70)	0.88 (0.58-1.36)	1.27 (0.84-1.92)	1.86 (0.97-3.58)
Colo-rectal	F	1.64	1.83	1.00	1.22	1.95	2.13	2.66	2.55

cancer		(0.74-3.67)	(1.10-3.05)		(0.73-2.04)	(1.21-3.16)	(1.32-3.44)	(1.65-4.29)	(1.17-5.57)
Prostate cancer	0.52 (0.07-4.14)	1.11 (0.52-2.37)	0.95 (0.57-1.58)	1.00	0.97 (0.60-1.57)	0.96 (0.58-1.60)	0.93 (0.55-1.59)	1.07 (0.62-1.85)	2.04 (0.83-4.99)
Women									
Person years	41,623	286,794	574,777	344,116	287,155	221,840	209,676	250,494	85,953
Deaths (no.)	3,941	27,900	56,541	33,972	28,468	22,131	21,030	25,364	8,770
Overall RR									
Unadjusted	1.96 (1.66-2.32)	1.27 (1.15-1.40)	1.01 (0.93-1.10)	1.00	1.01 (0.92-1.11)	1.08 (0.98-1.19)	1.10 (1.00-1.21)	1.18 (1.08-1.29)	1.68 (1.49-1.89)
Adjusted*	1.71 (1.44-2.01)	1.22 (1.10-1.34)	1.00 (0.92-1.09)	1.00	1.01 (0.92-1.11)	1.07 (0.97-1.18)	1.11 (1.00-1.22)	1.17 (1..07-1.29)	1.65 (1.46-1.85)
RR by sub-group									
Age									
<55 yr	1.69 (1.26-227)	1.15 (0.97-1.37)	0.96 (0.82-1.11)	1.00	1.04 (0.87-1.25)	1.06 (0.87-1.28)	1.16 (0.96-1.41)	1.24 (1.03-1.50)	1.66 (1.31-2.11)
55-<65	1.92 (1.49-2.48)	1.26 (1.08-1.47)	1.01 (0.89-1.15)	1.00	0.97 (0.84-1.12)	1.03 (0.89-1.19)	1.16 (1.01-1.33)	1.19 (1.03-1.36)	1.68 (1.42-2.00)
≥65	1.40 (1.01-1.96)	1.28 (1.05-1.56)	1.04 (0.89-1.23	1.00	1.04 (0.87-1.24)	1.15 (0.96-1.37)	1.00 (0.83-1.21)	1.10 (0.93-1.31)	1.56 (1.24-1.97)
Cause of death									
Neoplasms	1.16 (0.85-1.58)	1.17 (1.00-1.36)	0.98 (0.86-1.11)	1.00	1.00 (0.87-1.15)	1.06 (0.91-1.23)	1.21 (1.05-1.40)	1.12 (0.97-1.29)	1.38 (1.14-1.68)
Circulatory	1.09 (0.69-1.75)	1.10 (0.87-1.41)	1.01 (0.83-1.22)	1.00	1.13 (0.92-1.39)	1.15 (0.92-1.43)	1.24 (1.01-1.54)	1.31 (1.07-1.61)	2.27 (1.78-2.90)
Respiratory	4.74 (2.60-8.64)	1.70 (1.05-2.76)	1.29 (0.85-1.97)	1.00	0.64 (0.37-1.11)	1.29 (0.80-2.09)	0.83 (0.48-1.44)	1.18 (0.73-1.91)	1.57 (0.83-2.95)
Other	2.97 (2.07-4.27)	1.43 (1.10-1.84)	1.11 (0.89-1.39)	1.00	1.02 (0.79-1.31)	1.13 (0.87-1.46)	0.95 (0.72-1.26)	1.35 (1.05-1.73)	1.79 (1.30-2.46)
Ischemic heart disease	1.02 (0.44-2.39)	0.88 (0.55-1.40)	1.02 (0.72-1.44	1.00	1.26 (0.87-1.81)	1.27 (0.87-1.86)	1.58 (1.10-2.28)	1.44 (1.00-2.07)	3.28 (2.20-4.89)
Cerebrovascular disease	1.02 (0.44-2.38)	1.16 (0.78-1.73)	1.18 (0.86-1.62)	1.00	0.94 (0.65-1.36)	1.00 (0.68-1.45)	1.06 (0.73-1.54)	0.97 (0.67-1.40)	1.40 (0.88-2.24)
Colorectal cancer	0.52 (0.16-1.65)	0.88 (0.57-1.36)	0.64 (0.45-0.91)	1.00	0.60 (0.40-0.91)	0.95 (0.64-1.41)	1.02 (0.69-1.51)	0.64 (0.41-0.98)	1.14 (0.67-1.94)
Breast cancer	1.11 (0.47-2.60)	0.87 (0.56-1.35)	0.79 (0.55-1.13)	1.00	1.12 (0.76-1.66)	1.38 (0.93-2.06)	1.62 (1.09-2.40)	1.55 (1.05-2.28)	1.79 (1.07-2.99)

Notes: F – not estimable because the number of incident cases was too low in this category.

* As explained by Pischon et al.: "Multivariable adjusted relative risks calculated using Cox proportional hazard regression using age as the underlying time variable and stratified by center and age at recruitment with additional adjustment for smoking (never, former with quit≥10y, former with quit<10y, former with time since quit unknown, current with <15 cig/d, current with 15-24 cig/d, current with ≥25 cig/d, current with number of cig unknown, and missing); education (no school degree, primary school degree, technical or professional school degree, secondary school degree, university degree, and missing); alcohol consumption (nondrinker, 0.1-4.9 g/d, 5.0-14.9 g/d, 15.0-29.9 g/d, and ≥30 g/d), activity (inactive, moderately inactive, moderately active, active, and missing), and height (quintiles)."

Source: Adapted from Pischon, T., H. Boeing, K. Hoffmann, and 45 Other Colleagues. "General and Abdominal Adiposity and Risk of Death in Europe," *The New England Journal of Medicine*, 2008, vol. 359, no. 20: 2105-2120.

In Canada, Meera Jain of the University of Toronto used data from the Canadian National Breast Screening Study (NBSS) to estimate the association between BMI and the risk of premature all-cause mortality in Canadian women.[1044] The study used data obtained between 1980–1985 at the time of enrolment in the study, which directly measured the height and weight of the women. The 49,165 women in the study were aged 40–59 years at recruitment and were followed for a mean of 16.5 years. The researchers were able to link the records from the cohort of women to the National Mortality Database, which is maintained by Statistics Canada. The linkage created data on all-cause mortality to 1999 for most regions of the country, and identified 2,566 deaths.

Jain et al. expanded the BMI categories to account for the previous Canadian BMI classification. They found that the risk of all-cause premature mortality generally increased with increasing BMI levels, with the slight exception that risks were somewhat more elevated for overweight than obese class 1 women. Thus, women who had "some excess weight' (BMI 25.0–27.9) had a 28% increased risk of premature mortality; those who were overweight (BMI 28.0–29.9) had a 34% increased risk; those who were obese class 1 (BMI 30.0–34.9) had a 30% increased risk; and those who were in obese classes 2–3 (BMI ≥35.0) had a 40% increased risk of all-cause mortality.

Cause-specific mortality data linkage was only available to December 31, 1993, and only 1,223 deaths had occurred by that time.[1045] Jain et al. report that in their cause-specific analysis of these data, "BMI showed a positive, statistically significant association with each of the major causes of death: cancers of the colon, pancreas, lung, and breast, heart disease (myocardial infarction and ischemic heart disease), and stroke."[1046] However, they did not provide the actual data or relative risk ratios in their report of results. Jain et al.'s results for Canada are generally consistent with those found in other studies elsewhere, but according to WCRF/ACIR, most

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[1044] Jain, Meera G., A.B. Miller, T.E. Rohan, J.T. Rehm, S.J. Bondy, M.J. Ashley, J.E. Cohen, and R.G. Ferrence. "Body Mass Index and Mortality in Women: Follow-up of the Canadian National Breast Screening Study Cohort," *International Journal of Obesity*, 2005, vol. 29: 792-797.
[1045] Ibid.
[1046] Ibid. p. 795.

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studies have not found an association between lung cancer and excess weight.[1047]

Peter Katzmarzyk et al. of Queen's University reviewed longitudinal prospective studies of BMI and all-cause mortality that were published in the epidemiological literature between 1965 and February 2003.[1048] They found 36 studies, which included 81 analyses, that fit their criteria, and they noted that it is important to consider that the association between BMI and mortality is particularly influenced by age, pre-existing comorbidities, and smoking.

Based on results from the 36 studies, Katzmarzyk et al. produced a summary RR of 1.24 for the risk of obesity for all-cause mortality, when compared with normal weight as the reference group.[1049] They remarked that this represents their best estimate of the 'independent' effect of excess adiposity on all-cause mortality. The estimate was difficult to determine, however, because of the large number of covariates that were used in the various studies to estimate relative risk. In terms of age effects, they found mixed results, but estimated a summary RR for adults aged 65 and older of 0.97, which is consistent with the literature that finds obesity less likely to affect mortality outcomes in older adults.

In their literature review, the only Canadian study that Katzmarzyk et al. found at the time that examined the relationship between BMI and mortality in the Canadian population was one in which Katzmarzyk himself was the lead investigator.[1050] (It should be noted that the results of this Katzmarzyk literature review were published in 2003, prior to the 2007 publication of Luo et al's Canadian study previously cited and also referenced below, and the 2005 publication of Jain's results for Canadian women cited above.)

The earlier (2001) Katzmarzyk study used data from the longitudinal 1981 Canadian Fitness Survey (CFS), and included a nationally representative sample of 10,725 adult participants aged 20–69 years (out of a total sample of 23,400 people aged 7–69) who were followed for 13 years. The height and weight of the total sample were directly measured.

A total of 593 deaths (353 men and 240 women) occurred during the 13-year follow-up period, which was determined by linking the CFS database to Statistics Canada's Canadian Mortality Database. Because of the limited sample size for deaths, the researchers could not determine disease-specific mortality risk. The authors noted that when the analysis was repeated excluding deaths that occurred within the first two years of follow-up, the results were not significant. Therefore, only results that included all participants in the analysis and all deaths that occurred in the follow-up period were reported.

Table 35 below shows the results of the 2001 Katzmarzyk et al. study, which estimate hazard ratios by BMI category for both genders combined, and for males and females separately. The

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[1047] World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, accessed.
[1048] Katzmarzyk, Janssen, and Ardern. "Physical Inactivity, Excess Adiposity and Premature Mortality."
[1049] Ibid.
[1050] Katzmarzyk, Peter T., Cora L. Craig, and Claude Bouchard. "Underweight, Overweight and Obesity: Relationships with Mortality in the 13-Year Follow-up of the Canada Fitness Survey," *Journal of Clinical Epidemiology*, 2001, vol. 54: 916-920.

risk ratios were adjusted for sex (in the total sample), age, smoking status, and alcohol consumption. Smoking prevalence in the sample included 45% among males and 37% among females, but these adjusted results could not be stratified by BMI because of limited sample sizes.

The results show increased risks of all-cause mortality in the total sample in the underweight category (HR 1.63), and increasing risks with increasing BMI across the overweight (HR 1.16), obese class 1(HR 1.25), and obese classes 2–3 (HR 2.96) categories, when compared to normal weight individuals.[1051] Females had higher risks than males in all categories except underweight, but males had significant risks only in the underweight (HR 2.29) and obese class 2–3 (HR 2.52) categories. Severely obese Canadian women (BMI ≥35.0) had more than three times the risk (HR 3.13) of death from all causes than normal weight women, while obese class 1 women (BMI 30-<35) had a nearly 60% greater risk (HR 1.59).

Table 35. Risk for all-cause mortality by BMI and gender, aged 20–69 years, Canadian Fitness Survey, 1981–1993

BMI Class	Number	Person-years of follow-up	Number deaths	Hazard ratio	95% CI
Total sample					
Underweight	314	3,882	16	1.63	0.93-2.85
Normal weight	6,173	76,887	236	1.00	Reference
Overweight	3,279	40,151	250	1.16	0.96-1.39
Obese Class 1	770	9,313	74	1.25	0.96-1.65
Obese Class 2-3	189	2,282	17	2.96	1.39-6.29
Males					
Underweight	64	745	9	2.29	1.06-4.93
Normal weight	2,491	30,790	134	1.00	Reference
Overweight	2,035	24,846	169	1.08	0.86-1.36
Obese Class 1	386	4,662	36	1.00	0.68-1.47
Obese Class 2-3	63	757	5	2.52	0.80-7.99
Females					
Underweight	250	3,137	7	1.24	0.54-2.83
Normal weight	3,682	46,097	102	1.00	Reference
Overweight	1,244	15,305	81	1.24	0.93-1.67
Obese Class 1	384	4,651	38	1.59	1.08-2.34
Obese Class 2-3	126	1,525	12	3.13	1.15-8.52

Source: Katzmarzyk, Peter T., Cora L. Craig, and Claude Bouchard. "Underweight, Overweight and Obesity: Relationships with Mortality in the 13-Year Follow-up of the Canada Fitness Survey," *Journal of Clinical Epidemiology*, 2001, vol. 54: 916-920.

In a related 2004 study undertaken subsequent to the literature review, Peter Katzmarzyk and

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[1051] Ibid.

Christopher Ardern estimated the effects of overweight and obesity (class 1 and classes 2 and 3 combined) on Canadian premature mortality rates between 1985 and 2000/01.[1052] The age range of the study subjects—aged 20–64 years—did not allow inclusion of elderly mortality results because of data limitations. BMI was calculated from self-reported height and weight data found in six Canadian health surveys conducted between 1985 and 2000, including National Population Health Surveys and the 2000/01 Canadian Community Health Survey.

The BMI-related relative risk ratios for all-cause mortality that were used in the study—1.16 for overweight, 1.25 for obese class 1, and 2.96 for obese class 2–3— were taken from the previous 2001 study by Katzmarzyk et al., who, as noted above, calculated these hazard ratios by linking data from the 1981 Canadian Fitness Survey (CFS) database with Statistics Canada's Canadian Mortality Database.[1053] PAFs for all-cause mortality were calculated separately for each of the BMI categories, and summed together. The PAFs for each survey year (1985, 1990, 1994, 1996, 1998, and 2000) were multiplied by the total annual number of deaths among 20–64 year olds from all causes in each of the years.[1054]

Katzmarzyk and Ardern found that the estimated percentage of deaths among 20–64 year-old Canadians that could be attributed to overweight and obesity increased from 5.1% of all deaths in 1985 to 9.3% in 2000—primarily due to a dramatic nationwide increase in the prevalence of obesity. This translates to an increase from 2,514 deaths in 1985 to 4,321 deaths in 2000—which amounts to almost 1 in 10 premature deaths in 2000 that can be attributed to overweight and obesity.[1055]

As previously noted, Wei Luo et al. of the Public Health Agency of Canada recently estimated the risks for all-cause mortality in Canada in 2004 that were attributable to obesity, for the same three age groups used by Flegel et al.—ages 25–59, 60–69, and ≥70.[1056, 1057] Luo et al. applied the RRs for the risk of death due to obesity (BMI ≥30) by age obtained from the Flegal et al. study, to the age- and gender-specific prevalence of obesity found in the 2004 CCHS, cycle 2.2, which directly measured the height and weight of the respondents.

The RRs had been adjusted by Flegal et al. for race, gender, smoking status, and alcohol consumption. However, Luo et al. note that they "were not able to adopt Flegal's multi-risk approach to estimate the RR and number of excess deaths associated with obesity."[1058] They also

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[1052] Katzmarzyk, Peter, and Christopher I. Ardern. "Overweight and Obesity Mortality Trends in Canada, 1985-2000," *Canadian Journal of Public Health*, 2004, vol. 95, no. 1: 16-20.

[1053] Katzmarzyk, Craig, and Bouchard. "Underweight, Overweight and Obesity: Relationships with Mortality in the 13-Year Follow-up of the Canada Fitness Survey." The RRs were adjusted for age, gender, cigarette smoking, and alcohol consumption.

[1054] The authors used a PAF equation (PAF = $\sum$[P(RR-1)/RR]) that they comment, "produces an internally valid estimate when adjusted relative risks are used." They also note that the PAF commonly used (PAF = [$\sum$(P)(RR-1)]/[1+$\sum$(P)(RR-1)]) would have produced a greater, but biased, impact.

[1055] Katzmarzyk, and Ardern. "Overweight and Obesity Mortality Trends in Canada, 1985-2000."

[1056] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

[1057] Flegal, Graubard, and Williamson. "Excess Deaths Associated with Underweight, Overweight, and Obesity."

[1058] Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada." p. 141.

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chose not to include the overweight category in their estimation because of uncertainties among researchers on whether or not excess risks of mortality are attributable to overweight.

Since 2002 was the most recent year for which mortality data were available in the Canadian Mortality Database for 2007 when the study was conducted, Luo et al. applied the 2002 mortality rate to the 2004 population. The formula used to estimate the total number of deaths attributable to obesity was: $Y = \sum D_{i,j} * F_{i,j,k}$, where Y is the total number of 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 prevalence of obesity, RRs, PAFs, 95% confidence intervals, and number of deaths attributable to obesity for the three age groups in Canada in 2004, as estimated by Luo et al., are shown in Table 36 below. The results show that individuals aged 25–59 have the highest risks of mortality attributable to obesity (RR 1.83 for severely obese Canadians and RR 1.2 for those in obese class 1), while those aged ≥70 have the lowest relative risk.

Luo et al. estimated the total number of deaths attributable to obesity in Canada in 2004 to be 8,414 deaths, or four percent of total deaths. This is significantly higher than the 4,321 deaths in 2000 estimated by Katzmarzyk and Ardern (described above). However, the authors note that the two studies differed in age ranges— Katzmarzyk and Ardern used ages that ranged from 20 to 64, while Luo et al. included all adults aged 25 and over, which will necessarily produce a considerably higher number of deaths. In addition, Katzmarzyk and Ardern used surveys with self-reported heights and weight to determine BMI, while Luo et al. used surveys with directly measured heights and weights, which produce higher (and much more accurate) obesity rates.

The influence of age is also seen in comparing results from these two Canadian studies. As noted, Luo et al. found the risks of death attributable to obesity to be considerably higher among younger Canadians aged 25–59 than among older Canadians. This helps explain why Luo et al.'s estimate that 4% of Canadian deaths among those 25 and older in 2004 were attributable to obesity is less than Katzmarzyk et al.'s estimate that 9.3% of deaths among those aged 20–64 in 2000 could be attributed to overweight and obesity. As seen in Table 36 below, Luo et al. found that 3% of all deaths among Canadians in obese class 1 aged 25–59 were attributable to obesity, while 6.9% of all deaths among severely obese Canadians were attributable to obesity.

Of the 8,414 Canadian deaths that Luo et al. estimated could be attributed to obesity in 2004, 3,350 deaths, or about 40%, occurred in obese adults aged 25–59; 2,339 deaths, or about 28%, occurred in obese adults aged 60–69; and 2,725 deaths, or about 32%, occurred in obese adults aged ≥70 years. In addition, 32% of obesity-related deaths occurred among individuals in obese class 1, and 68% occurred among individuals in obese classes 2–3.

Table 36. Prevalence of obesity, RRs, PAFs, and deaths attributable to obesity in Canada, 2004 (95% confidence interval), by age group and obesity class, estimated by Luo et al.

Age in years	BMI (kg/m2) 30 to <35 (obese class 1)		BMI (kg/m2) ≥35 (obese class 2–3)		BMI (kg/m2) ≥30 – Total obese	
25-59						
prevalence of obesity (%)	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	(2,922–3,773)
60-69						
prevalence of obesity (%)	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	(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 of obesity (%)	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)**	**5,705.00**	**(4,541–6,850)**	**8,414.00**	**(6,881–9,927)**

Note: PAF = population attributable fraction; RR = risk ratios obtained by Luo et al. from Flegal, Katherine M., Barry I. Graubard, and David F. Williamson. "Excess Deaths Associated with Underweight, Overweight, and Obesity," *JAMA - Journal of the American Medical Association*, 2005, vol. 293, no. 15: 1861-1867.

Source: Adapted from Luo, Wei, Howard Morrison, Margaret de Groh, Chris Waters, Marie DesMeules, Elaine Jones-McLean, Anne-Marie Ugnat, Sylvie Desjardins, Morgan Lim, and Yang Mao. "The Burden of Adult Obesity in Canada," *Chronic Diseases in Canada*, 2007, vol. 27, no. 4: 135-144.

4.12.1 Mortality statistics in Canada and Alberta

In 2004, there were a total of 226,584 deaths in Canada—2,515 among children aged 1–14 years, and 224,069 among adults aged ≥15 years.[1059, 1060] According to PHAC, the leading causes of death for different age groups in Canada are:

- perinatal mortality for infants under the age of one year,
- unintentional injuries for those aged 1–34 years,
- cancer for those aged 35–64 years, and
- circulatory system diseases for those aged 65 years and over.[1061]

Of the total adult deaths in Canada in 2004, 99,680 deaths, or approximately 44.5% of the total,[1062] were caused by a type of health condition that is partially attributable to excess body weight, and 55.5% of deaths were caused by health conditions that have no demonstrated association with excess weight. Figure 37 below illustrates key causes of adult death in Canada in 2004, as percentages of all adult deaths, for those causes that have been found to be partially attributable to excess body weight,. These are the diseases that have been reliably associated with obesity in the epidemiological literature, and the health impacts and relative risks of which have been discussed in the sections above.

Figure 37 shows that 17.5% of total adult deaths in Canada were caused by coronary heart disease, 16.1% by those particular cancers that have been reliably associated with excess weight, 6.5% by stroke, 3.5% by diabetes, 0.6% by hypertension, 0.1% by gallbladder disease, and 0.1% by asthma. Needless to say, these are not the proportions of Canadian deaths attributable to excess weight, but represent the total deaths attributable to those particular conditions that have a demonstrated association with obesity, and of which a *portion* of deaths is therefore attributable to obesity.

[1059] Statistics Canada. *Deaths, by Selected Grouped Causes, Age Group and Sex, Canada, Annual*, CANSIM Table 102-0551, 2007.

[1060] Statistics Canada. *Mortality, Summary List of Causes 2004*, Catalogue no. 84F0209XIE, 2007; accessed November 2008; available from http://www.statcan.gc.ca/pub/84f0209x/84f0209x2004000-eng.pdf.

[1061] Public Health Agency of Canada (PHAC). *Leading Causes of Death and Hospitalization in Canada*, 2005; accessed November 2008; available from http://www.phac-aspc.gc.ca/publicat/lcd-pcd97/index-eng.php.

[1062] 44.5 is rounded from 44.48629.

Figure 37. Mortality by types of death that can be partially attributed to BMI, percentage of all adult deaths, adults aged 15+, Canada, 2004

Source: Statistics Canada. *Mortality, Summary List of Causes 2004*, Catalogue no. 84F0209XIE, 2007; accessed November 2008; available from http://www.statcan.gc.ca/pub/84f0209x/84f0209x2004000-eng.pdf.
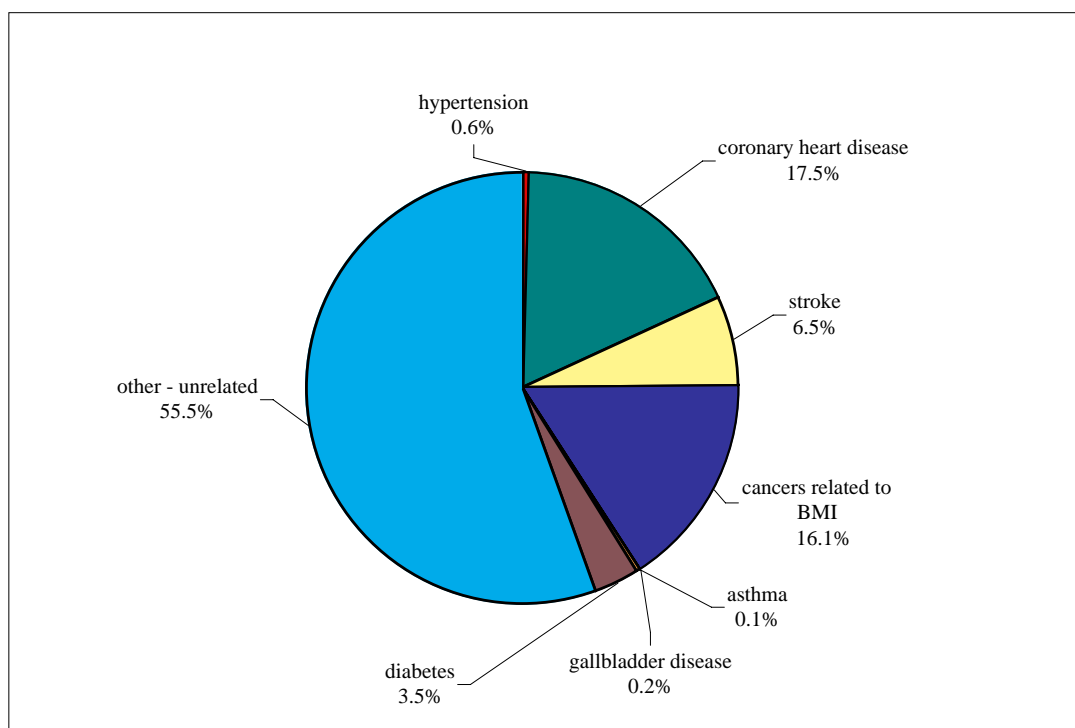
Statistics Canada. Deaths, by Selected Grouped Causes, Age Group and Sex, Canada, Annual, CANSIM Table 102-0551, 2007.

In Canada, there were a total of 66,947 deaths caused by cancer in 2004—141 among children aged 0–14 years, and 66,806 among adults aged ≥15 years.[1063, 1064] Of the total number of Canadian adult cancer deaths, 35,972—or approximately 53.8%—were of a type that can be partially attributable to excess body weight, and 30,834—or approximately 46.2%—were of a type that has no demonstrated association with obesity.

Figure 38 below shows the percentages of total adult cancer deaths in Canada in 2004 attributable to those particular types of cancer for which a partial association with excess body weight has been demonstrated. The main cancer types associated with excess weight are seen to account for the following percentages of total Canadian adult cancer deaths: colorectal cancer —

[1063] Statistics Canada. *Deaths, by Selected Grouped Causes, Age Group and Sex, Canada, Annual*.
[1064] Statistics Canada. *Mortality, Summary List of Causes 2004*, accessed. Category for cancer is malignant neoplasms.

11.0%, female breast cancer —7.4%, prostate cancer — 5.5%, cancer of the pancreas — 5.4%, non-Hodgkin's lymphoma — 4.0%, leukemia — 3.3%, stomach cancer — 2.9%, bladder cancer — 2.4%, ovarian cancer — 2.4%, liver cancer — 2.3%, kidney cancer — 2.2%, esophageal cancer — 2.2%, multiple myeloma — 1.9%, and endometrial cancer — 1.1%.

Figure 38. Cancer mortality, by types of cancer partially attributable to BMI, percentage of total adult cancer deaths, aged ≥15, Canada, 2004
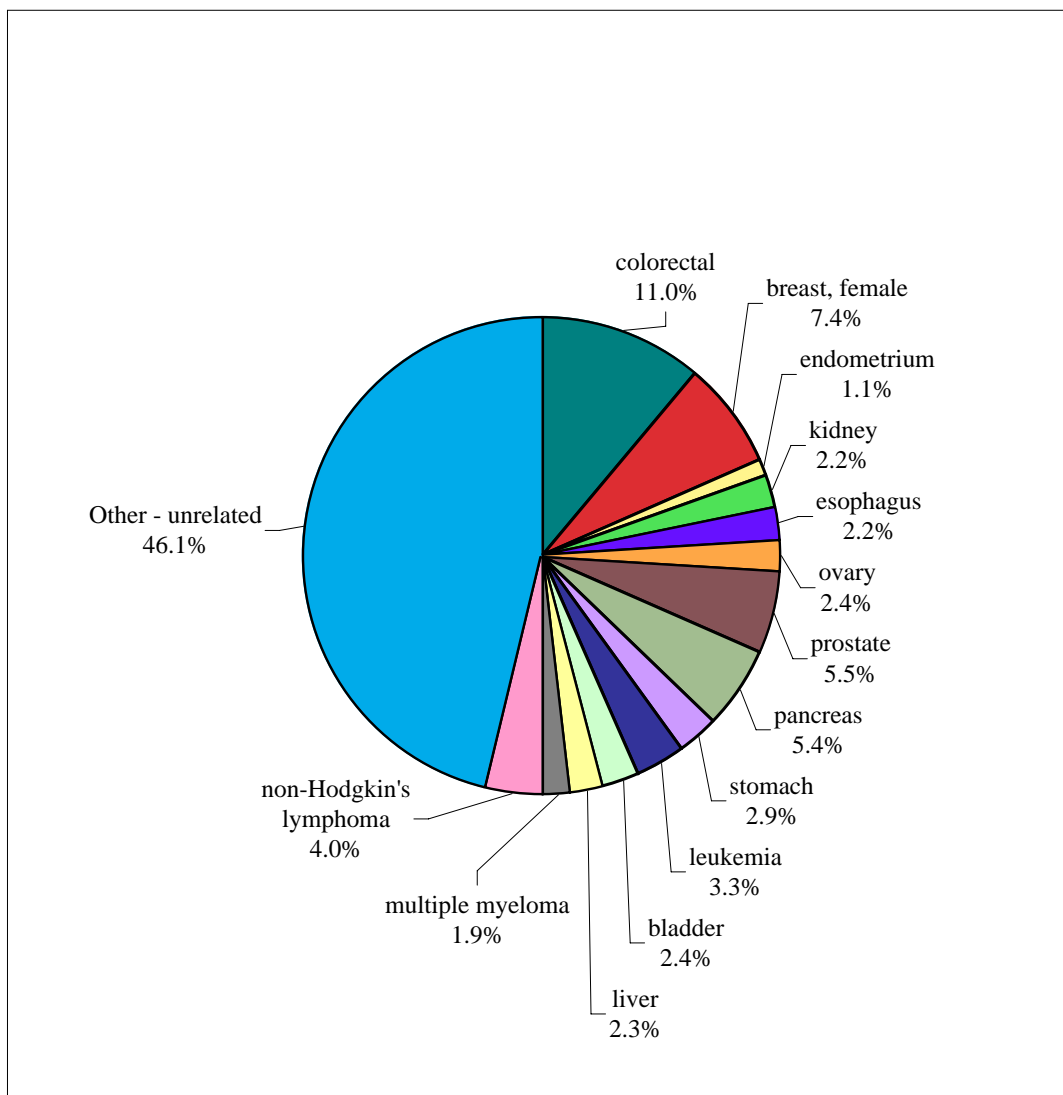
Sources: Statistics Canada. *Mortality, Summary List of Causes 2004*, Catalogue no. 84F0209XIE, 2007; accessed November 2008; available from http://www.statcan.gc.ca/pub/84f0209x/84f0209x2004000-eng.pdf. Statistics Canada. Deaths, by Selected Grouped Causes, Age Group and Sex, Canada, Annual, CANSIM Table 102-0551, 2007.

In Alberta, there were a total of 19,071 deaths in 2005 (excluding non-residents), of which 18,682 were among adults, aged ≥15 years, and 389 were among children, aged 0–14 years.[1065] Approximately 41.5% of adult deaths in Alberta (7,749 deaths) were caused by a type of health condition that can be partially attributed to excess weight, and 58.5% (10,933 deaths) were of a type unrelated to excess weight.[1066]

Figure 39 below illustrates the causes of adult death in Alberta in 2005 for those health conditions that have been found to be partially attributable to excess body weight, and for other unrelated deaths, as percentages of all adult deaths. With the exception of coronary heart disease, which in Alberta accounts for a somewhat larger proportion of total deaths than nationwide, other key obesity-related conditions—BMI-related cancers, stroke, diabetes, and hypertension—are seen to account for somewhat smaller percentages of total deaths in Alberta than nationwide.

Figure 39 shows that 19.3% of total adult deaths in Alberta in 2005 were caused by coronary heart disease (3,611 deaths), 14.8 % by cancers that are associated with excess weight (2,763 deaths), 3.9% by stroke (734 deaths), 2.5% by diabetes (471 deaths), 0.4% by hypertension (83 deaths), 0.1% by gallbladder disease (25 deaths), and 0.1% by asthma (16 deaths). In addition, 33 adult Albertans are recorded in Alberta government records specifically as dying from "obesity and other hyperalimentation."

[1065] Alberta Government Services. *Alberta Vital Statistics Annual Review 2005, Death Related Statistics*, Service Alberta, 2006; accessed November 2008; available from http://www.servicealberta.gov.ab.ca/pdf/vs/2005deaths.pdf.
[1066] Ibid., accessed.

Figure 39. Mortality by types of death that can be partially attributed to BMI, percentage of all adult deaths, adults aged ≥15, Alberta, 2005
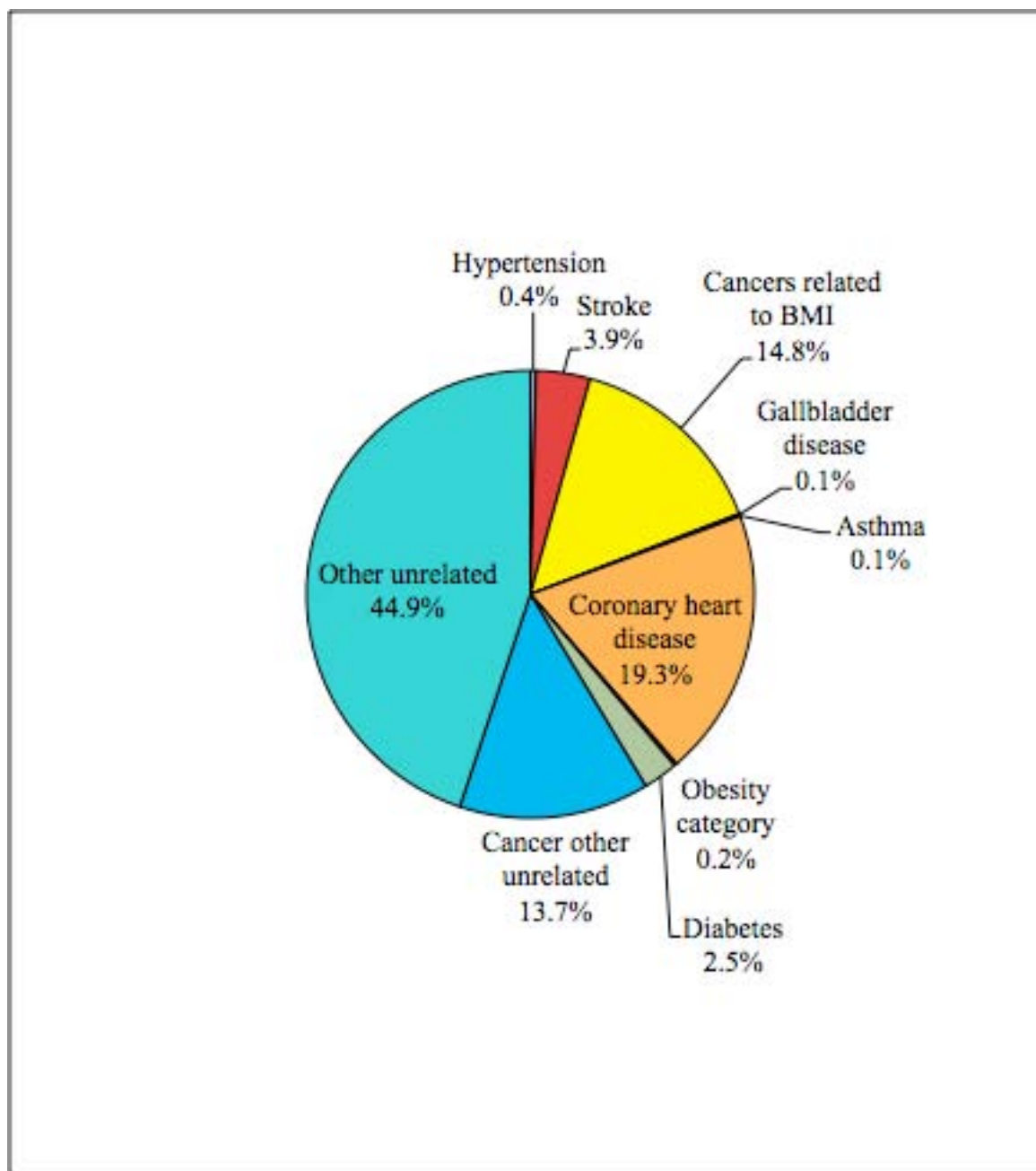
Notes: "Obesity category" in Figure 39 above is actually listed in the Alberta Government source below as (Alberta code) "132 Obesity and other hyperalimentation."

Source: Alberta Government Services. *Alberta Vital Statistics Annual Review 2005, Death Related Statistics*, Service Alberta, 2006; accessed November 2008; available from http://www.servicealberta.gov.ab.ca/pdf/vs/2005deaths.pdf.

In Alberta there were a total of 5,332 adult (aged ≥15 years) deaths caused by cancer in 2005, (excluding non-residents).[1067] In addition, three children died from leukemia, but none died from the other types of cancer that have been related to BMI. Of the total number of adult cancer deaths, 2,763—or approximately 52%—were from types of cancers that can be partially attributed to excess body weight, and 2,569—or approximately 48%—were of types of cancer with no proven association with BMI.

Figure 40 below shows the percentages of total adult cancer deaths in Alberta in 2005 attributable to those particular types of cancer for which a partial association with excess body weight has been demonstrated. For the most part, the proportions are close to those for Canada as a whole in 2004. With the exceptions of prostate cancer and multiple myeloma, which show slightly higher percentages of total cancer deaths than nationwide, the rest of the obesity-related cancer types account for the same or slightly lower percentages of total cancer deaths than nationwide.

As shown in Figure 40 below, the main cancer types associated with excess weight are seen to account for the following percentages of total Canadian adult cancer deaths. Colorectal cancer — 9.7% (518 deaths), female breast cancer — 7.2% (382 deaths), prostate cancer — 6.4% (340 deaths), cancer of the pancreas — 5.4% (285 deaths), non-Hodgkin's lymphoma — 3.7% (196 deaths), leukemia — 3.3% (178 deaths), multiple myeloma — 2.4% (127 deaths), stomach cancer — 2.3% (124 deaths), ovarian cancer — 2.3% (121 deaths), esophageal cancer — 2.2% (115 deaths), bladder cancer — 2.1% (113 deaths), kidney cancer — 2.1% (110 deaths), liver cancer — 2.0% (105 deaths), and endometrial cancer — 1.0% (49 deaths).

[1067] Ibid., accessed.

Figure 40. Cancer mortality, by types of cancer partially attributable to BMI, percentage of total adult cancer deaths, adults aged ≥15, Alberta, 2005
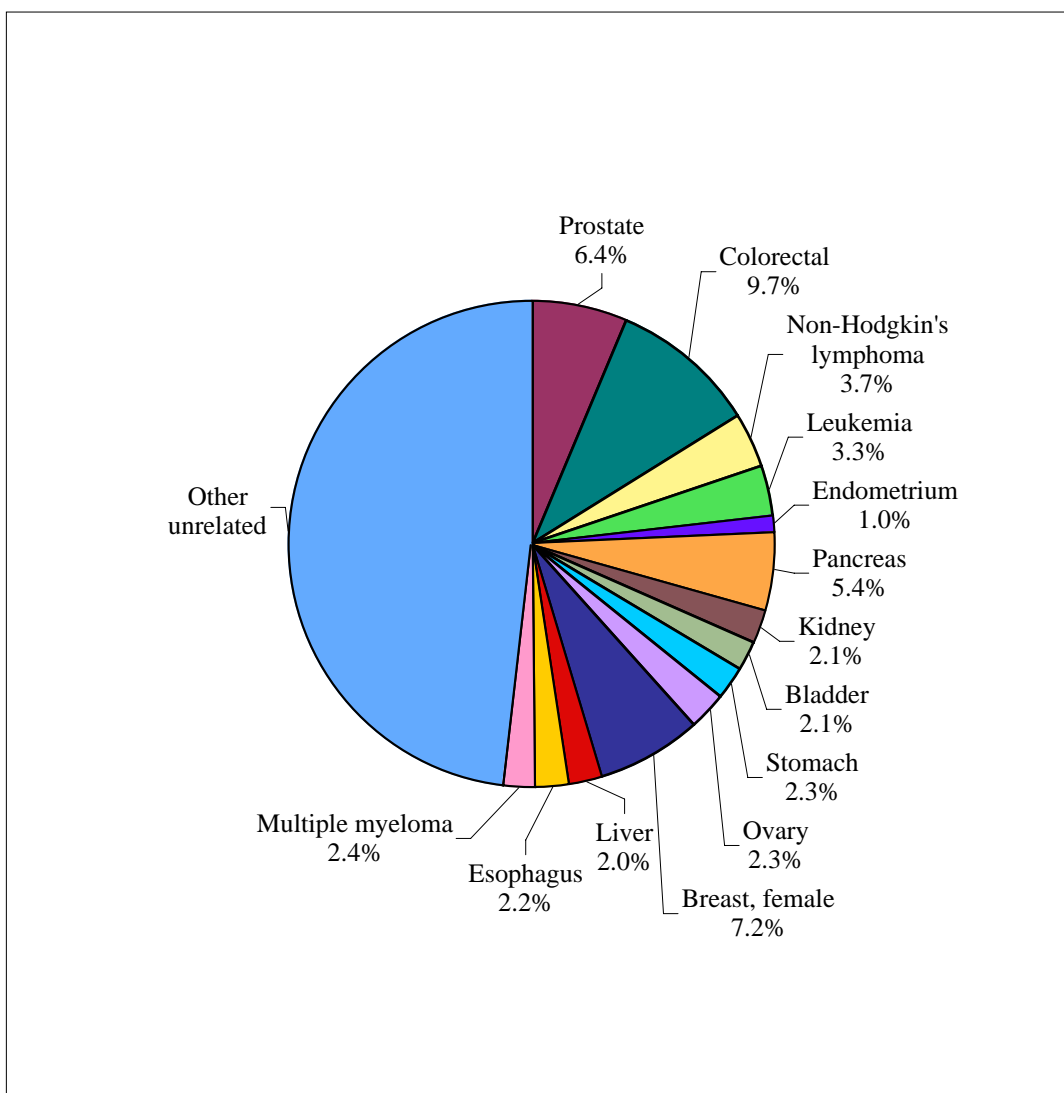
Source: Alberta Cancer Board. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics* Alberta Health Services, Alberta Cancer Board, 2008; accessed July 2008; available from http://www.cancerboard.ab.ca/NR/rdonlyres/2BADD738-9C4D-487F-B8C1-68D5D962CD81/0/AlbertaCancerRegistry200531Oct08.pdf.

Table 37 below shows the specific number of deaths that occurred in Alberta in 2005, by age group and gender, for the types of health conditions partially attributable to BMI. The age ranges were chosen to correspond with those used by *The Economic Burden of Illness in Canada* (EBIC), which is the key Canadian source for disease cost estimates, and by Luo et al.[1068] As seen in Table 37 below, there were very few deaths among young Albertans below the age of 35 related to health conditions that have a demonstrated association with obesity. For those particular obesity-related illnesses, the vast majority of deaths occurred in adults aged 60 and over.

Among obesity-related conditions in Alberta, coronary heart disease—which includes myocardial infarction or heart attack—was the leading cause of death (3,611 deaths), followed by those cancers that are partially attributable to high BMI (2,766 deaths), stroke (734 deaths), diabetes (471 deaths), hypertension (83 deaths), gallbladder disease (25 deaths), asthma (16 deaths), and mood disorders (13 deaths). In addition, 33 deaths among Albertan adults are listed in Alberta government records as being caused by "obesity and other hyperalimentation."

In 2005, there were more male than female deaths in Alberta caused by the type of health conditions related to excess body weight—approximately 4,150 versus 3,600, respectively. More Albertan males than females also died from cancer types that can be partially attributed to excess weight—approximately 1,495 versus 1,270, respectively. When gender-specific cancers like prostate, breast, ovarian, and endometrial/uterine cancers are excluded, it is seen that more Albertan males than females also died from each of the non-gender-specific types of cancer that have been associated with obesity.

More Albertan males also died from coronary heart disease and diabetes than did females, but more females than males died from stroke, hypertension, gallbladder disease, asthma, and the category described in Alberta government records as "obesity and other hyperalimentation." Gallbladder disease and mood disorders had an equal number of deaths in each gender.

Again, it must be emphasized that all the death statistics and percentages cited in these pages, including age and gender breakdowns, so far refer only to total deaths attributable to each type of health condition that has been reliably associated with obesity, ***not*** to deaths specifically attributable to obesity. Clearly, as seen by the relative risk ratios described above, obesity only accounts for a portion of deaths among the total number attributable to each health condition, with markedly different relative risks for males and females and by BMI category and age group. Thus, for example, the gender ratios may be very different than indicated above and in Table 37 below when those Albertan deaths that are specifically attributed to obesity itself are considered, as they will be below.

[1068] *Economic Burden of Illness in Canada (EBIC), 2000*.
Luo, Morrison, Groh, Waters, DesMeules, Elaine Jones-McLean, Ugnat, Desjardins, Lim, and Mao. "The Burden of Adult Obesity in Canada."

Table 37. Number of deaths, by type of health condition partially attributable to BMI, by age group and gender, Alberta, 2005

Cause	Gender	0–14	15–19	20–24	25–34	35–59	60–64	65–69	70–74	75+	Total
Diabetes	Male	0	0	0	4	39	23	16	26	147	255
	Female	0	0	1	1	18	7	15	27	147	216
	Both	0	0	1	5	57	30	31	53	294	471
Hypertension	Male	0	0	0	1	2	2	4	2	16	27
	Female	0	0	0	0	0	3	3	3	47	56
	Both	0	0	0	1	2	5	7	5	63	83
Coronary heart disease*	Male	0	0	0	12	359	132	156	343	1045	2047
	Female	0	0	0	0	64	45	70	291	1094	1564
	Both	0	0	0	12	423	177	226	634	2099	3611
Stroke	Male	0	0	0	0	12	7	12	28	229	288
	Female	0	0	0	0	8	10	8	21	399	446
	Both	0	0	0	0	20	17	20	49	628	734
Colorectal cancer*	Male	0	0	1	2	50	34	34	44	134	299
	Female	0	0	0	0	32	18	19	33	117	219
	Both	0	0	1	2	82	52	53	77	251	518
Breast cancer*	Male	0	0	0	0	1	1	0	2	4	8
	Female	0	0	0	3	114	46	34	50	135	382
	Both	0	0	0	3	115	47	34	52	139	390
Endometrial cancer*	Male	0	0	0	0	0	0	0	0	0	0
	Female	0	0	0	0	9	8	5	7	20	49
	Both	0	0	0	0	9	8	5	7	20	49
Kidney cancer	Male	0	0	0	0	16	6	9	11	38	80
	Female	0	0	0	0	6	0	7	3	14	30
	Both	0	0	0	0	22	6	16	14	52	110
Esophageal cancer	Male	0	0	0	0	22	18	9	14	34	97
	Female	0	0	0	0	1	1	3	2	12	19
	Both	0	0	0	0	23	19	12	16	45	115
Ovarian cancer	Male	0	0	0	0	0	0	0	0	0	0
	Female	0	0	0	1	29	12	10	19	50	121
	Both	0	0	0	1	29	12	10	19	50	121
Prostate cancer	Male	0	0	0	0	19	12	28	41	240	340
	Female	0	0	0	0	0	0	0	0	0	0

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	Both	0	0	0	0	19	12	28	41	240	340
Pancreatic cancer	Male	0	0	0	0	33	14	25	21	58	151
	Female	0	0	0	0	23	15	20	19	57	134
	Both	0	0	0	0	56	29	45	40	115	285
Non-Hodgkin's lymphoma	Male	0	1	1	1	22	14	15	18	42	114
	Female	0	0	0	0	11	8	5	9	49	82
	Both	0	1	1	1	33	22	20	27	91	196
Leukemia*	Male	1	2	2	1	15	7	20	16	51	115
	Female	2	0	0	1	4	3	10	12	34	66
	Both	3	2	2	2	19	10	30	28	85	181
Multiple myeloma	Male	0	0	0	1	9	2	12	12	31	67
	Female	0	0	0	0	4	5	7	16	28	60
	Both	0	0	0	1	13	7	19	28	59	127
Liver cancer	Male	0	0	0	0	21	9	10	10	24	74
	Female	0	0	0	1	8	0	2	8	12	31
	Both	0	0	0	1	29	9	12	18	36	105
Bladder cancer	Male	0	0	0	0	13	8	11	9	45	86
	Female	0	0	0	0	1	0	3	2	21	27
	Both	0	0	0	0	14	8	14	11	66	113
Stomach cancer	Male	0	0	0	1	13	8	4	16	30	72
	Female	0	0	0	0	9	3	4	8	28	52
	Both	0	0	0	1	22	11	8	24	58	124
Osteoarthritis	Male	0	0	0	0	0	0	0	0	0	0
	Female	0	0	0	0	0	0	0	0	0	0
	Both	0	0	0	0	0	0	0	0	0	0
Gallbladder disease	Male	0	0	0	0	0	1	1	1	10	13
	Female	0	0	0	0	3	0	1	1	7	12
	Both	0	0	0	0	3	1	2	2	17	25
Asthma	Male	0	0	0	0	0	1	0	1	2	4
	Female	0	0	1	0	2	0	1	1	7	12
	Both	0	0	1	0	2	1	1	2	9	16
Mood (affective) disorders*	Male	0	0	0	0	1	1	0	0	4	6
	Female	0	0	0	0	2	0	0	0	5	7
	Both	0	0	0	0	3	1	0	0	9	13
Obesity	Male	0	0	0	1	12	1	0	0	0	14

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category*										
	Female	0	0	0	1	12	1	2	2	1	19
	Both	0	0	0	2	24	2	2	2	1	33

Total deaths, all causes, excluding non-residents – 19,071
Total adult deaths, aged ≥15 years – 18,682.
Total child deaths, aged 0–14 years – 389.
Total deaths for types of health conditions that are partially attributable to excess body weight as a risk factor – 7,760

Notes: Total number of Albertan deaths exclude 350 non-resident deaths. Age categories were chosen to correspond to those used in:

Economic Burden of Illness in Canada (EBIC), 2000, Policy Research Unit, Knowledge Information and Data Systems Division, Office of Public Health Practice, Public Health Agency of Canada. Unpublished material supplied by Alan Diener, Manager, to GPI Atlantic, October 2008, 2008. (0–14, 15–34, 35–64, 65–74, and 75+), and in:

Luo, Wei, Howard Morrison, Margaret de Groh, Chris Waters, Marie DesMeules, Elaine Jones-McLean, Anne-Marie Ugnat, Sylvie Desjardins, Morgan Lim, and Yang Mao. "The Burden of Adult Obesity in Canada," *Chronic Diseases in Canada*, 2007, vol. 27, no. 4: 135-144. (25–59, 60–69, and 70+)

*Breast cancer – Only postmenopausal breast cancer (not premenopausal breast cancer) has been reliably associated with obesity. It is therefore important to note that the number of deaths for female breast cancer in the 50-59 year age group in 2005 was 59 deaths, and that 324 out of the total of 382 female breast cancer deaths in Alberta in 2005 occurred among women aged 50 or older.

* Coronary heart disease includes the following specific conditions as listed with code numbers in Albertan government death statistics records: **"**165 acute myocardial infarction", "166 other acute ischemic heart diseases", "167 atherosclerotic cardiovascular disease", and "168 all other forms of chronic ischemic heart disease".

Colorectal cancer includes the following specific conditions as listed with code numbers in Albertan government death statistics records: "63 Malignant neoplasms of colon", and "64 Malignant neoplasms of rectoigmoid junction and rectum".

Endometrial cancer refers to the following specific condition as listed with code number in Albertan government death statistics records: "85 Malignant neoplasms of corpus utereri and uterus, part unspecified".

Leukemia includes the following specific conditions as listed with code numbers in Albertan government death statistics records: "105 Lymphoid leukemia", "106 Myeloid leukemia", "107 Monocytic leukemia", and "108 Other unspecified leukemia".

Mood disorders refers to the following specific condition as listed with code number in Albertan government death statistics records: "141 Mood (affective) disorders".

Obesity category refers to the following specific condition as listed with code number in Albertan government death statistics records: "132 Obesity and other hyperalimentation".

Source: Alberta Government Services. *Alberta Vital Statistics Annual Review 2005, Death Related Statistics*, Service Alberta, 2006; accessed November 2008; available from http://www.servicealberta.gov.ab.ca/pdf/vs/2005deaths.pdf.

4.13 Health impacts related to obesity in children and youth

Obesity in children and youth is increasing at an alarming rate in the Western world. Abundant evidence now indicates that overweight children are more likely to continue to gain weight and become obese in adulthood.[1069] Thus, childhood obesity is a concern not only for its more immediate impacts on children's health and wellbeing, but for its long-term implications and impacts on adult health and risks of premature death.

According to Ogden et al., there are "considerable gaps in knowledge of the links between childhood weight and future health outcomes."[1070] They cite a 2005 report of the Childhood Obesity Task Force of the U.S. Preventive Services Task Force:

> We do not know the best way to identify children who are at risk for future adverse health outcomes due to obesity or overweight. Although BMI is a convenient and widely agreed-on measure of obesity, it is not clear what BMI at any given age is associated with future good health.[1071]

According to James et al., obese children have a propensity for a number of diseases and health disorders, such as bone and joint deformation during growth, asthma, and sleep apnea in extreme cases.[1072] This is in addition to the psychological trauma associated with the social stigma frequently attached to childhood obesity, which was referenced earlier in the section on obesity-related mental health conditions above. James et al. note:

> It is now clear that overweight children also have higher blood pressure, serum lipid abnormalities and increasing insulin resistance, all of which are hallmarks of early metabolic disease and susceptibility to atherosclerosis and other cardiovascular problems.[1073]

Below, we look very briefly at five obesity-related health conditions that may appear in childhood. However, this brief overview of these five specific conditions should not obscure the reality that the largest health impact of childhood obesity actually manifests in adulthood rather than childhood, and is especially due to the propensity of obese children to carry excess weight in adulthood. As well, the chronic conditions associated with obesity take time to develop and

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[1069] Dietz, W.H. "Health Consequences of Obesity in Youth: Childhood Predictors of Adult Disease," *Pediatrics*, 1998, vol. 101, no. 3: 518–525.

[1070] Ogden, Yanovski, Carroll, and Flegal. "The Epidemiology of Obesity." p. 2094.

[1071] Moyer, V.A., J.D. Klein, J.K. Ockene, S.M. Teutsch, M.S. Johnson, J.D. Allan, and Childhood Obesity Working Group of the U.S. Preventive Services Task Force. "Screening for Overweight in Children and Adolescents: Where Is the Evidence? A Commentary by the Childhood Obesity Working Group of the U.S. Preventive Services Task Force," *Pediatrics*, 2005, vol. 116: 235–238. Cited in Ogden, Yanovski, Carroll, and Flegal. "The Epidemiology of Obesity." p. 2094.

[1072] James, Jackson-Leach, Mhurchu, Kalamara, Shayeghi, Rigby, Nishida, and Rodgers. "Overweight and Obesity (High Body Mass Index)."

[1073] Ibid. p. 581.

are therefore far more likely to produce disease, suffering, premature death, and economic costs many years after initial weight gain than in childhood.

4.13.1 Cardiovascular disease

Childhood obesity is considered to be the leading cause of hypertension in children and of other risk factors leading to myocardial infarction, stroke, and other cardiovascular-related conditions in children.[1074] Researchers using data from the Bogualusa (Louisiana) Heart Study, which is an ongoing longitudinal study of CVD risk factors begun in 1972, found that overweight children, aged 5–17 years, were 2.4 times as likely as children with normal weight to have elevated diastolic blood pressure and 4.5 times as likely to have high systolic blood pressure.[1075]

4.13.2 Asthma

Recent evidence shows that asthma prevalence is increasing in Canada and other countries, and is a major cause of morbidity and mortality among children.[1076] A chronic inflammatory disorder of the airways, asthma can cause wheezing, difficulty in breathing, and chest pain, and is the most common chronic disease among children in North America.[1077]

Asthma is a major cause of hospitalization for young children in Canada, contributing—according to one Canadian government report—to 12% of all hospital admissions in the birth to 4 years age group.[1078] The Public Health Agency of Canada gives somewhat different figures, indicating that asthma accounted in 2004 for 10% of all hospital admissions among children aged 0–4 years, and for 8% of admissions among children aged 5–14 years.[1079]

A British study also found asthma to be the leading cause of absenteeism from school in the U.K.[1080] A 2006 report by the Commission for Environmental Co-operation found that asthma

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[1074] Tauman, Riva, and David Gozal. "Obesity and Obstructive Sleep Apnea in Children," *Paediatric Respiratory Reviews*, 2006, vol. 7: 247-259.

[1075] Freedman, D.S., W.H. Dietz, S.R. Srinivasan, and G.S. Berenson. "The Relation of Overweight to Cardiovascular Risk Factors among Children and Adolescents: The Bogalusa Heart Study," *Pediatrics* 1999, vol. 103: 1175-1182.

[1076] Flaherman, V., and G. W. Rutherford. "A Meta-Analysis of the Effect of High Weight on Asthma," *Archives of Disease in Childhood*, 2006, vol. 91: 334-339.

[1077] US EPA. *America's Children and the Environment: Measures of Contaminants, Body Burdens, and Illness, Second Edition*, cited in Government of Canada, *The Well-Being of Canada's Young Children*, 2003; accessed December 2007; available from http://socialunion.gc.ca/ecd/2003/report2_e/chapter02_e.html.

[1078] Government of Canada. *The Well-Being of Canada's Young Children*, 2003; accessed December 2007; available from http://socialunion.gc.ca/ecd/2003/report2_e/chapter02_e.html.

[1079] Public Health Agency of Canada (PHAC). *Life and Breath: Respiratory Disease in Canada*, accessed.

[1080] Harrison, B.W.D, and M.G. Pearson. "Audit in Acute, Severe Asthma – Who Benefits?" *Journal of Royal College of Physicians of London*, 1992, vol. 27: 387-390. cited inAsthma Society of Canada. *Asthma Facts and Statistics*, accessed December 2007; available from http://www.asthma.ca/corp/newsroom/pdf/asthmastats.pdf.

rates among children in some parts of North America are four times higher than they were 20 years ago, and that poor urban children are at greater risk.[1081] Exposure to second-hand smoke and air pollution has been found to contribute to asthma incidence and severity.[1082]

It is noteworthy that children and youth have the highest prevalence of asthma of any age group in Canada and the highest hospitalization rates due to asthma. A Health Canada study found that:

- Since 1994, asthma prevalence has been increasing among Canadian children (except for boys aged 4–7 years)
- Boys of all ages have a higher prevalence of asthma than girls—in contrast to the adult asthma data noted above, which indicate that females *over 12* have higher asthma rates than males.
- Currently, approximately 20% of boys aged 8–11 have been diagnosed with asthma, the highest prevalence group among children.[1083]

According to the National Longitudinal Survey on Children and Youth (NLSCY), in 2000-01, 13% of Canadian children aged 0-15 years have been diagnosed with asthma (16% of boys and 11% of girls). British Columbia and the Prairie provinces were found to have the lowest childhood asthma rates in the country at 11.4% and 11.1% respectively, and the Atlantic provinces had the highest rate (15.5%).[1084]

Although they did not examine childhood asthma in relation to body weight, Rochelle Garner and Dafna Kohen of Statistics Canada estimated changes in the prevalence of asthma in Canadian children aged 0–11 using data from four cycles of the National Longitudinal Survey of Children and Youth (NLSCY).[1085] Between 1994/95 and 2000/01 the rate of asthma among Canadian children increased by 20.7% from 11.1% (or 518,400 children nationwide) to 13.4% (or 586,000 children). Among these asthma sufferers, however, the authors found that the proportion of children with high-severity symptoms actually decreased from 41% to 36%, and that the prevalence of asthma attacks during the previous year decreased from 51% to 39%. The authors speculate that the decrease in asthma attacks might be associated with the increased use of medications to control asthma.

Also using data from the cross-sectional component of the 2000/01 NLSCY, PHAC found that 15.6% of Canadian children aged 4–11 years had ever been diagnosed with asthma, and that

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[1081] CBC News. *Asthma Rates in Children Have Jumped Fourfold: Report*, 27 January, 2006; accessed December 2007; available from http://www.cbc.ca/canada/story/2006/01/27/asthma-report060127.html.
[1082] Physicians for a Smoke-free Canada. accessed April 2008; available from http://www.smoke-free.ca/Second-Hand-Smoke/health_kids.htm. Health Canada. *Respiratory Disease in Canada*, 2001; accessed April 2008; available from http://www.phac-aspc.gc.ca/publicat/rdc-mrc01/pdf/rdc0901e.pdf.p. 33.
[1083] Health Canada. *Respiratory Disease in Canada*, accessed. p. 18.
[1084] Childhood asthma rates for Canada and regions are for 2000/2001 and are from Garner, Rochelle, and Dafna Kohen. "Changes in the Prevalence of Asthma among Canadian Children," *Health Reports*, Statistics Canada Catalogue no. 82-0003, 2008, vol. 19, no. 2: 1-6. Findings are based on the 2000/2001 National Longitudinal Survey of Children and Youth (NLSCY).
[1085] Ibid.

8.3% of children aged ≥12 have been diagnosed with asthma.[1086] As noted above, asthma rates are consistently higher among boys than girls, but this changes in adulthood when women have a higher prevalence of asthma than men. PHAC notes that, although asthma is often considered to be a childhood disease, and while children do have the highest prevalence of asthma and the highest hospitalization rates of any age group in the country, the disease is common among all age groups and actually affects more adults than children in terms of absolute numbers.

V. Flaherman and G.W. Rutherford of the University of California San Francisco report that "weight is not one of the largest contributors to the development of asthma, and the effect of weight on asthma is likely to be relatively weak compared to other risk factors."[1087] These other factors include diet quality, air pollution, environmental tobacco smoke, family history of asthma, and decreased exposure to infectious agents.

However, Flaherman and Rutherford conducted a systematic review and meta-analysis of the literature from 1966 to 2004 to estimate the future risk of asthma for school-aged children with a high BMI. They found that most early studies showed a negative association, but studies published in 2000 and after were positive. Their results suggest that a high body weight among school-age children, aged 6–19, increases the risk of future asthma by approximately 50%—the adjusted summary RR was 1.5 (95% CI 1.2 to 1.8). They also found that 6.6% of all cases of childhood asthma in the U.S. could be due to overweight.

In 2001, J. I. Figueroa-Muñoz et al. used data from the nationally representative 1993/94 National Study of Health and Growth in England and Scotland to estimate the association between high BMI and asthma in almost 15,000 children aged 4–11. Asthma was prevalent in 17.3% of the total sample, and the odds (OR 1.29) for the association of childhood asthma with high BMI were statistically significant in both sexes.

E. von Mutius et al. analysed a representative sample of U.S. children and youth aged 4–17 using data from the Third National Health and Nutrition Examination Survey (NHANES III), which was conducted in two phases (1988–1991 and 1991–1994).[1088] BMI was divided into quartiles, and the authors found that asthma prevalence rose significantly with increasing quartiles of BMI—i.e. from 8.7% among children aged 4-17 with the lowest BMI to 9.3% in the second quartile to 10.3% in the third quartile to 14.9% among children and youth with the highest BMI. Compared with the 1st quartile BMI (OR 1.00), the odds ratios (ORs) of having asthma for 2nd, 3rd, and 4th quartile BMI children and youth were 1.19, 1.36, and 1.98, respectively. The study found no correlation between asthma and television viewing hours or physical activity levels.

[1086] Public Health Agency of Canada (PHAC). *Life and Breath: Respiratory Disease in Canada*, accessed.

[1087] Flaherman, and Rutherford. "A Meta-Analysis of the Effect of High Weight on Asthma." p. 338.

[1088] von Mutius, E. , J. Schwartz, L. M. Neas, D. Dockery, and S. T. Weiss. "Relation of Body Mass Index to Asthma and Atopy in Children: The National Health and Nutrition Examination Study III," *Thorax*, 2001, vol. 56, no. 835-838.

4.13.3 Type 2 diabetes

The prevalence of type 2 diabetes among obese children and youth has also been increasing. Type 2 diabetes has previously been referred to as "adult-onset" diabetes, since it normally develops in adults after the age of 35. However, new evidence indicates that type 2 diabetes is now being diagnosed at ever younger ages where it is strongly linked to obesity. Indeed, it is now estimated that approximately 45% of child diabetes cases in the U.S. are type 2.[1089]

However, Ogden et al. observe that type 2 diabetes in children is still "a very low prevalence condition," that occurs primarily in children who have a strong history of diabetes, who are extremely obese by adult standards (with a BMI range of 35–40), and who are from certain ethnic groups such as First Nations.[1090] They record that over 90% of youth with type 2 diabetes in all racial and ethnic groups in the U.S. are overweight.

A House of Commons Health Committee report in the United Kingdom notes that children with type 2 diabetes, which is more difficult to control than type 1 diabetes, are at risk for a myriad of chronic health problems throughout their lifetimes.[1091] This is confirmed by one long-term Canadian study of 51 individuals aged 18–33 years who had been diagnosed with type 2 diabetes before the age of 17 years, which found that:

> Seven had died; three others were on dialysis; one became blind at the age of 26; and one had had a toe amputation. Of 56 pregnancies in this cohort, only 35 had resulted in live births (62.5%).[1092]

4.13.4 Obstructive sleep apnea (OSA)

Obstructive sleep apnea (OSA)—also know as obstructive sleep apnea syndrome (OSAS)—in children is characterized by partial or total upper airway obstruction during sleep that recurs throughout the night.[1093] According to Riva Tauman and David Gozal of the University of Louisville: "Despite recognition of OSAS in the late 1800s, this complex, yet relatively frequent disorder is only now being recognized as a major public health problem in the pediatric age range."[1094] They note that most of the sleep apnea studies among children that have taken place to date have had small sample sizes, and that there is a need for a large-scale assessment of the

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[1089] Fagot-Campagna, A. "Emergence of Type 2 Diabetes Mellitus in Children: Epidemiological Evidence," *Journal of Paediatric Endocrinology and Metabolism*, 2000, vol. 13, no. supplement 6: 1395-1402
[1090] Ogden, Yanovski, Carroll, and Flegal. "The Epidemiology of Obesity." p. 2094.
[1091] House of Commons Health Committee (U.K.). *Obesity: Third Report of Session 2003-04, Annex 1: The Economic Costs of Obesity*, accessed.
[1092] Dean, H., and B. Flett. "Natural History of Type 2 Diabetes Diagnosed in Childhood: Long Term Follow-up in Young Adult Years," *Diabetes*, 2002, vol. 51, no. supplement 2: A24-25. Cited in House of Commons Health Committee (U.K.). *Obesity: Third Report of Session 2003-04, Annex 1: The Economic Costs of Obesity*, accessed. Some individuals had multiple pregnancies.
[1093] Tauman, and Gozal. "Obesity and Obstructive Sleep Apnea in Children."
[1094] Ibid. p. 248.

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obesity–OSA association in children. The prevalence of OSA in U.S. children aged 2–8 has been estimated to be approximately 3%.[1095]

Carter and Watenpaugh report that untreated sleep apnea "compromises physical, behavioral, and cognitive development in children."[1096] More severe cases of OSA in children have been associated with hypertension, failure to thrive, developmental delay, and sudden unexpected death.[1097] Although the prevalence of OSA is lower in children than in adults, the impact on behaviour, mood, and excessive daytime sleepiness or hyperactivity may be more severe in children than in adults.[1098] OSA has been associated with enlarged adenoids or tonsils in children, and children and youth who carry excess weight have been found to be at increased risk of developing OSA.[1099]

U.S. researchers Susan Redline et al. examined risk factors for sleep disordered breathing (SDB) in children aged 2–18 years.[1100] SDB was defined as "the occurrence of repetitive episodes of complete or partial obstruction of the upper airway during sleep," but the definition included asthma, bronchitis, and other conditions such as sinus problems in addition to OSA.

Redline et al. found that obese children (defined in this case by a BMI of ≥28) were four to five times more likely to have SDB than were non-obese children.[1101] The unadjusted OR for the obesity-SDB association was 4.59, and SDB was found to occur in 28.0% of the obese children. In addition to using BMI cutoff values (≥28) to identify obesity in children, Redline et al. also repeated the analysis for BMI as a continuous variable. Using this method, they found that for each increase in BMI by 1 kg/m^2 above the mean BMI in the study sample, the risk of SDB increased by 12%.[1102]

Tauman and Gozal found that among children referred for evaluation of suspected sleep-disordered breathing, between 45% and 55% were obese.[1103] They note: "It is expected that the increased prevalence of obesity in children and adolescents in our society and worldwide will be accompanied by a steady increase in the incidence of OSAS."[1104]

4.13.5 Mental health

In addition to the physical health risks associated with being overweight, obese children and

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[1095] Ibid.

[1096] Carter III, and Watenpaugh. "Obesity and Obstructive Sleep Apnea: Or Is It OSA and Obesity?" p. 3.

[1097] Tauman, and Gozal. "Obesity and Obstructive Sleep Apnea in Children."

[1098] Carter III, and Watenpaugh. "Obesity and Obstructive Sleep Apnea: Or Is It OSA and Obesity?"

[1099] Public Health Agency of Canada (PHAC). *Life and Breath: Respiratory Disease in Canada*, accessed.

[1100] Redline, Susan, Peter V. Tishler, Mark Schluchter, Joan Aylor, Kathryn Clark, and Gregory Graham. "Risk Factors for Sleep-Disordered Breathing in Children: Associations with Obesity, Race, and Respiratory Problems," *American Journal of Respiratory and Critical Care Medicine*, 1999, vol. 159, no. 5: 1527-1532.

[1101] Ibid.

[1102] Ibid.

[1103] Tauman, and Gozal. "Obesity and Obstructive Sleep Apnea in Children."

[1104] Ibid.

youth may be at risk for significant psychological disorders. J.L. Tang-Peronard and B.L. Heitman of Denmark published a review of the literature on the stigmatization of children and youth in a 2008 issue of *Obesity Reviews*.[1105] Stigmatization was defined as "a psychological consequence of overweight and obesity that was originally defined as 'the devaluation of individuals who have unwanted personal characteristics' but, with time, it has come to include both attitudes and actions of prejudice, such as discrimination."[1106] The review found that overweight girls seemed to experience a higher degree of stigmatization than overweight boys. Overweight girls were more often verbally and physically bullied and socially isolated— having few friends or romantic relationships—than overweight boys. The result was that overweight girls developed low self-esteem, depression, eating disorders, and generally poor health.

Alan J. Zametkin et al. of the U.S. Institutes of Health reviewed the epidemiological literature on the psychiatric aspects of child and adolescent obesity published between 1993 and 2003.[1107] Obesity was defined as having a BMI greater than the 95th percentile for age and sex and overweight as a BMI greater than or equal to the 85th percentile. In general, the evidence found by Zametkin et al. showed mixed results and "no clear indication of higher rates of psychiatric comorbidity in the general population of obese children."[1108] Obese children and youth seeking clinical treatment, however, had lower self-esteem and increased levels of depression, anxiety, and eating disorders than obese children and youth in the general population. The authors note that obese children and youth seeking clinical treatment are generally those who are severely obese.

Sarah Mustillo et al. note that most of the research on obesity and psychiatric disorders in children has used clinical samples recruited because of obesity, and that the risk of psychiatric disorders in these samples may not reflect what is found in the general population.[1109]

Gilbert Vila et al. evaluated the frequency of psychiatric disorders in 155 obese children and youth, including 98 girls and 57 boys aged 5 to 17 years, who were outpatients in the Department of Pediatric Nutrition at Necker-Enfants-Malades Hospital in Paris in 2003.[1110] Psychiatric disorders were assessed through standardized diagnostic interviews and self-reported questionnaires completed by the children or their parents. Approximately 58% of the obese children and youth showed significant psychiatric disorders, of which the most prominent were anxiety disorders such as social phobias and separation anxiety disorders, affective disorders such as major depressions, and disruptive behaviour disorders such as defiant oppositional disorders and attention deficit hyperactivity disorders. The obese children were compared with

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[1105] Tang-Peronard, J.L., and B.L. Heitmann. "Stigmatization of Obese Children and Adolescents, the Importance of Gender," *Obesity Reviews*, 2008, vol. 9: 522-534.

[1106] Ibid. p. 522.

[1107] Zametkin, Alan J., Christine K. Zoon, Hannah W. Klein, and Suzanne Munson. "Psychiatric Aspects of Child and Adolescent Obesity: A Review of the Past 10 Years," *Child and Adolescent Psychiatry*, 2004, vol. 43, no. 2: 134-150.

[1108] Ibid.

[1109] Mustillo, Sarah, Carol Worthman, Alaattin Erkanli, Gordon Keeler, Adrian Angold, and E. Jane Costello. "Obesity and Psychiatric Disorder: Developmental Trajectories," *Pediatrics*, 2003, vol. 111, no. 4: 851-859.

[1110] Vila, Gilbert, Ewa Zipper, Myriam Dabbas, Catherine Bertrand, Jean Jacques Robert, Claude Ricour, and Marie Christine Mouren-Simeoni. "Mental Disorders in Obese Children and Adolescents," *Psychosomatic Medicine*, 2004, vol. 66: 387-394.

diabetic children who were not obese and were found to have more severe and frequent psychiatric disorders than the diabetic children. The authors noted that their sample cannot be generalizable to all obese children and must be limited to children seeking treatment for obesity.

Mustillo et al. followed a representative sample of 991 mainly rural white children aged 9 to 16 years in the U.S. over an 8-year period. During this time the children were interviewed, measured, and weighed annually, and the association between obesity and psychiatric disorders—including conduct disorder, oppositional defiant disorder, attention deficit hyperactivity disorder, depression, anxiety, bulimia, and substance abuse—was assessed.

Mustillo et al. found that by age 16, about 20% of the sample was obese, defined as above the 95th percentile for age and sex according to the 2000 (U.S.) Centers for Disease Control tables.[1111] About 72.8% of the sample were never obese, while 14.6% of the sample were "chronically obese," or obese throughout the study, 7.5% had normal weight in young childhood but became obese in adolescence, and 5.1% were obese during late childhood but lost weight during adolescence and dropped below the obesity threshold. The increased risk of 7 psychiatric disorders in the 3 obesity groups was tested, relative to the group which was never obese. The study found that only chronic obesity was associated with a psychiatric disorder—oppositional defiant disorder in both boys and girls and depressive disorders in boys. No significant associations were seen between obesity and the other psychiatric disorders assessed. The authors concluded that while their results could suggest that obesity increases the risk of psychopathology for certain psychiatric disorders, further research is needed in order to draw definitive conclusions.

Despite the seriousness of all of the childhood health conditions briefly reviewed above, the major health impacts of childhood obesity will show up in adulthood. We have not been able to include childhood obesity costs in this analysis for Alberta due to data limitations and methodological challenges, and that the issue requires more careful investigation. However, these obesity costs are likely to be considerable. Zametkin et al. noted in 2004 that the only U.S. study estimating obesity-associated costs in children was conduced by Wang and Dietz who used hospital-discharge data for obesity-related diseases among children—particularly diabetes, gallbladder disease, sleep apnea, asthma, and other health conditions where obesity was listed as a secondary diagnosis.[1112] Wang and Dietz found that the obesity-associated annual hospital costs among obese children to have increased from $35 million ($US2001) during 1979–1981 to $127 million ($US2001) during 1997–1999.[1113] The authors conclude that the increased costs reflect the increasing prevalence and severity of obesity in the U.S. It is likely that obesity among children is also responsible for increased health care costs in Canada.

[1111] Mustillo, Worthman, Erkanli, Keeler, Angold, and Costello. "Obesity and Psychiatric Disorder: Developmental Trajectories."
[1112] Zametkin, Zoon, Klein, and Munson. "Psychiatric Aspects of Child and Adolescent Obesity: A Review of the Past 10 Years."
[1113] Wang, Zheng, Heath, Macera, Pratt, and Buchner. "Economic Burden of Cardiovascular Disease Associated with Excess Body Weight in U.S. Adults."

Note: This report is completed in Part 2, which contains the economic costs of obesity in Alberta. Part 2 is located in the accompanying document.

REFERENCES

Aaron, Shawn D., Katherine L. Vandemheen, Louis-Philippe Boulet, R. Andrew McIvor, J. Mark FitzGerald, Paul Hernandez, Catherine Lemiere, Sat Sharma, Stephen K. Field, Gonzalo G. Alvarez, Robert E. Dales, Steve Doucette, and Dean Fergusson for the Canadian Respiratory Clinical Research Consortium. "Overdiagnosis of Asthma in Obese and Nonobese Adults," *Canadian Medical Association Journal*, 2008, vol. 179, no. 11, November 18: 1121-1131.

Access Economics. *The Economic Costs of Obesity*, Diabetes Australia, 2006; accessed June 2008; available from http://www.accesseconomics.com.au/publicationsreports/showreport.php?id=102.

_____. *The Growing Cost of Obesity in 2008: Three Years On*, Diabetes Australia, 2008; accessed September 2008; available from http://diabetesaustralia.com.au/PageFiles/7832/FULLREPORTGrowingCostOfObesity2008.pdf.

Alberta Cancer Board. *Cancer in Alberta: A Regional Picture*, 2007; accessed July 2008; available from http://www.cancerboard.ab.ca/NR/rdonlyres/125FDB29-3F4B-413B-BDB9-B4425F9BAFE0/0/ACBRegionalReport_2007.pdf.

_____. *Alberta Cancer Registry. 2005 Annual Report of Cancer Statistics,* Alberta Health Services, Alberta Cancer Board, 2008; accessed July 2008; available from http://www.cancerboard.ab.ca/NR/rdonlyres/2BADD738-9C4D-487F-B8C1-68D5D962CD81/0/AlbertaCancerRegistry200531Oct08.pdf.

Alberta Diabetes Surveillance System. *Alberta Diabetes Atlas 2007*, Institute of Health Economics, 2007; accessed September 2008; available from http://www.albertadiabetes.ca/pdf/atlas/AlbertaDiabetesAtlas2007_001.pdf.

Alberta Government Services. *Alberta Vital Statistics Annual Review 2005, Death Related Statistics*, Service Alberta, 2006; accessed November 2008; available from http://www.servicealberta.gov.ab.ca/pdf/vs/2005deaths.pdf.

Alberta Health and Wellness. *Alberta Diabetes Strategy 2003-2013*, 2003; accessed September 2008; available from http://www.health.alberta.ca/public/dis_diabetesstrategy.pdf.

_____. *Table H-3: Cholestectomy Procedures and Rates Per 100,000 Population* 2003; accessed November 2008; available from http://www.health.alberta.ca/regions/h3.htm.

_____. *Keeping Albertans Healthy*, 2004; accessed September 2008; available from http://www.health.alberta.ca/public/in_healthy.pdf.

_____. *Report on the Health of Albertans*, Edmonton: Alberta Health and Wellness, 2006; accessed July 2008; available from http://www.health.alberta.ca/resources/publications/Health-of-Albertans-2006.pdf.

_____. *Population Projections for Alberta and Its Health Regions 2006 to 2035*, Health Surveillance and Environmental Health Branch, 2007; accessed July 2008; available from http://www.health.alberta.ca/resources/publications/PopProj_06.pdf.

Alberta Health and Wellness Project Team. *Health Trends in Alberta*, Surveillance and Environmental Health Branch, Alberta Health and Wellness, 2007; accessed July 2008; available from http://www.health.alberta.ca/resources/publications/trends_2007.pdf.

Alexander, Dominik D., Pamela J. Mink, Hans-Olov Adami, Philip Cole, Jack S. Mandel, Martin M. Oken, and Dimitrios Trichopoulos. "Multiple Myeloma: A Review of the Epidemiologic Literature," *International Journal of Cancer*, 2007, vol. 120: 40-61.

AlGhanim, Nayef, Vikram R. Comondore, John Fleetham, Carlo A. Marra, and Najib T. Ayas. "The Economic Impact of Obstructive Sleep Apnea," *Lung*, 2008, vol. 186: 7-12.

Allison, David B., Morgan Downey, Richard L. Atkinson, Charles J. Billington, George A. Bray, Robert H. Eckel, Eric A. Finkelstein, Michael D. Jensen, and Angelo Tremblay. "Obesity as a Disease: A White Paper on Evidence and Arguments Commissioned by the Council of the Obesity Society," *Obesity*, 2008, vol. 16: 1161-1177.

Assembly of First Nations / First Nations Information Governance Committee. *First Nations Regional Longitudinal Health Survey (RHS) 2002/03: Results for Adults, Youth and Children Living in First Nations Communities*, RHS National Team, 2007; accessed August 2008; available from http://www.rhs-ers.ca/english/pdf/rhs2002-03reports/rhs2002-03-technicalreport-afn.pdf.

Associated Press. "The Fatter You Are, the Shorter Your Life: Study." *Jet*, October 28, 1999, accessed September 2008; available from http://findarticles.com/p/articles/mi_m1355/is_21_97/ai_57564173/print?tag=artBody;col1.

Asthma Society of Canada. *Asthma Facts and Statistics*, accessed December 2007; available from http://www.asthma.ca/corp/newsroom/pdf/asthmastats.pdf.

_____. *Asthma Facts and Statistics*, 2005; accessed October 2008; available from http://www.asthma.ca/corp/newsroom/pdf/asthmastats.pdf.

Atlantis, E., and M. Baker. "Obesity Effects on Depression: Systematic Review of Epidemiological Studies," *International Journal of Obesity*, 2008, vol. 32: 881–891.

Benichou, Jacques. "A Review of Adjusted Estimators of Attributable Risk," *Statistical Methods in Medical Research*, 2001, vol. 10: 195-216.

Bergstrom, A., C-C. Hsieh, P. Lindblad, C-M Lu, N.R. Cook, and A. Wolk. "Obesity and Renal Cell Cancer - a Quantitative Review," *British Journal of Cancer*, 2001, vol. 85, no. 7: 984-990.

Berrington de Gonzalez, Amy, Elizabeth A. Spencer, H. Bas Bueno-de-Mesquita, and 40 researchers from the EPIC study. "Anthropometry, Physical Activity, and the Risk of Pancreatic Cancer in the European Prospective Investigation into Cancer and Nutrition [EPIC]," *Cancer Epidemiology Biomarkers & Prevention*, 2006, vol. 15, no. 5: 879-885.

Berrington de Gonzalez, Amy, S. Sweetland, and Elizabeth A. Spencer. "A Meta-Analysis of Obesity and the Risk of Pancreatic Cancer," *British Journal of Cancer*, 2003, vol. 89: 519-523.

Birmingham, C. Laird, Jennifer L. Muller, Anita Palepu, John J. Spinelli, and Aslam H. Anis. "The Cost of Obesity in Canada," *Canadian Medical Association Journal*, 1999, vol. 160, no. 4: 483-488.

Bjorge, Tone, Anders Engeland, Steinar Tretliand, and Elisabete Weiderpass. "Body Size in Relation to Cancer of the Uterine Corpus in 1 Million Norwegian Women," *International Journal of Cancer*, 2006, vol. 120: 378-383.

Blakely, Tony, Simon Hales, Charlotte Kieft, Nick Wilson, and Alistair Woodward. "Distribution of Risk Factors by Poverty," in *Comparative Quantification of Health Risks: Global and Regional Burden of Disease Attribution to Selected Major Risk Factors*, ed. Ezzati, Majid, Alan D. Lopez, Anthony Rodgers and Christopher J.L. Murray, Volume 1, Chapter 24: 1941-2127. Geneva: World Health Organization, 2004; accessed June 2008; available from http://www.who.int/publications/cra/chapters/volume2/1941-2128.pdf.

Blakely, Tony, Simon Hales, and Alistair Woodward. *Poverty: Assessing the Distribution of Health Risks by Socioeconomic Position at National and Local Levels*, Environmental Burden of Disease Series No. 10, World Health Organization, 2004; accessed June 2008; available from http://www.who.int/quantifying_ehimpacts/publications/en/ebd10.pdf.

Boland, Lori L., Aaron R. Folsom, and Wayne D. Rosamond. "Hyperinsulinemia, Dyslipidemia, and Obesity as Risk Factors for Hospitalized Gallbladder Disease: A Prospective Study," *Annals of Epidemiology*, 2002, vol. 12: 131-140.

Boulet, Louis-Philippe, and Annick Des Cormiers. "The Link between Obesity and Asthma: A Canadian Perspective," *Canadian Respiratory Journal*, 2007, vol. 14, no. 4: 217-220.

Briggs, Andrew H., Bernie J. O'Brien, and Gordon Blackhouse. "Thinking Outside the Box: Recent Advances in the Analysis and Presentation of Uncertainty in Cost-Effectiveness Studies," *Annual Review of Public Health*, 2002, vol. 23, no. 377-401.

Burkhauser, Richard V., and John Cawley. "Beyond BMI: The Value of More Accurate Measures of Fatness and Obesity in Social Science Research," *Journal of Health Economics*, 2008, vol. 27: 519-529.

Calle, Eugenia E., Carmen Rodriguez, Kimberly Walker-Thurmond, and Michael J. Thun. "Overweight, Obesity, and Mortality from Cancer in a Prospectively Studied Cohort of U.S. Adults," *New England Journal of Medicine*, 2003, vol. 348: 1625-1638.

Calle, Eugenia E., Michael J. Thun, Jennifer M. Petrelli, Carmen Rodriguez, and Clark W. Heath Jr. "Body-Mass Index and Mortality in a Prospective Cohort of U.S. Adults," *New England Journal of Medicine*, 1999, vol. 341, no. 16: 1097-1106.

Camargo, Carlos A. Jr, Scott T. Weiss, Shumin Zhang, Walter C. Willett, and Frank E. Speizer. " Prospective Study of Body Mass Index, Weight Change, and Risk of Adult-Onset Asthma in Women," *Archives of Internal Medicine* 1999, vol. 159: 2582–2588.

Canadian Diabetes Association. "Canadian Diabetes Association 2008 Clinical Practice Guidelines for the Prevention and Management of Diabetes in Canada," *Canadian Journal of Diabetes*, 2008, vol. 32, no. Supplement 1: Si-S201.

Canadian Hypertension Education Program (CHEP). *CHEP Recommendations for the Management of Hypertension*, 2007; accessed September 2008; available from http://www.hypertension.ca/chep/wp-content/uploads/2007/10/chep-2007-spiral-mar16.pdf.

Canadian Institute for Health Information (CIHI). *Table 1: BMI Category for Joint Replacement Patients, by Province and Territory, 2003–2004*, 2005; accessed October 2008; available from http://secure.cihi.ca/cihiweb/en/media_17aug2005_tab1_e.html.

_____. *Canadian Joint Replacement Registry (CJRR) 2006 Report: Total Hip and Total Knee Replacements in Canada*, 2006; accessed October 2008; available from http://secure.cihi.ca/cihiweb/products/CJRR_Annual_Report_Hip_Knee_Replacements_2006_e.pdf.

Carter III, Robert, and Donald E. Watenpaugh. "Obesity and Obstructive Sleep Apnea: Or Is It Osa and Obesity?" *Pathophysiology,doi:10.1016/j.pathophys.2008.04.009*, Article in Press, 2008.

Caterson, I.D. "Obesity, Part of the Metabolic Syndrome," *Clinical Biochemist Reviews*, 1997, vol. 18, no. 1: 11-21.

CBC News. *Asthma Rates in Children Have Jumped Fourfold: Report*, 27 January, 2006; accessed December 2007; available from http://www.cbc.ca/canada/story/2006/01/27/asthma-report060127.html.

Centers for Disease Control and Prevention (CDC). *Preventing Obesity and Chronic Diseases through Good Nutrition and Physical Activity*, United States Department of Health and Human Services, 2008; accessed September 2008; available from http://cdc.gov/nccdphp/publications/factsheets/Prevention/obesity.htm.

Child Health Surveillance Project Data Group. *Alberta Child Health Surveillance Report 2005*, Alberta Health and Wellness, 2005; accessed August 2008; available from http://www.health.alberta.ca/resources/publications/ACH_Surveillance.pdf.

Clark, J.M. "The Epidemiology of Nonalcoholic Fatty Liver Disease in Adults," *Journal of Clinical Gastroenterology*, 2006, vol. 40, no. Suppl 1: S5–S10.

Cole, T.J., M.C. Bellizzi, K.M. Flegal, and W.H. Dietz. "Establishing a Standard Definition for Child Overweight and Obesity Worldwide: International Survey," *British Medical Journal*, 2000, vol. 320: 1240-1243.

Colman, Ronald. *Cost of Obesity in Alberta*, Halifax, N.S.: Genuine Progress Index Atlantic (GPI Atlantic), 2000.

_____. *Cost of Obesity in Nova Scotia*, GPI Atlantic and Cancer Care Nova Scotia, 2000; accessed June 2008; available from http://www.gpiatlantic.org/pdf/health/obesity/ns-obesity.pdf.

_____. *The Cost of Chronic Disease in Nova Scotia*, GPI Atlantic and Atlantic Region Population and Public Health Branch, Health Canada, 2002; accessed Nov 2007; available from http://gpiatlantic.org/pdf/health/chronic.pdf.

Connor Gorber, S., M. Tremblay, D. Moher, and B. Gorber. "A Comparison of Direct Vs. Self-Report Measures for Assessing Height, Weight and Body Mass Index: A Systematic Review," *Obesity Reviews*, 2007, vol. 8: 307-326.

Crew, Katherine D., and Alfred I. Neugut. "Epidemiology of Gastric Cancer," *World Journal of Gastroenterology*, 2006, vol. 12, no. 3: 354-362.

Danaei, Goodarz, Stephen Vander Hoorn, Alan D. Lopez, Christopher J.L. Murray, Majid Ezzati, and the Comparative Risk Assessment collaborating group (Cancers). "Causes of Cancer in the World: Comparative Risk Assessment of Nine Behavioural and Environmental Risk Factors," *Lancet*, 2005, vol. 366: 1784-1793.

de Sousa, A. G. P. , C. Cercato, M. C. Mancini, and A. Halpern. "Obesity and Obstructive Sleep Apnea-Hypopnea Syndrome," *Obesity Reviews*, 2008, vol. 9: 340-354.

Dean, H., and B. Flett. "Natural History of Type 2 Diabetes Diagnosed in Childhood: Long Term Follow-up in Young Adult Years," *Diabetes*, 2002, vol. 51, no. supplement 2: A24-25.

DeGuia, Nicole, Naishu Zhu, Margaret Keresteci, and Juqing Ellen Shi. "Obesity and Joint Replacement Surgery in Canada: Findings from the Canadian Joint Replacement Registry (CJRR)," *Healthcare Policy*, 2006, vol. 1, no. 3: 36-43.

Dietz, W.H. "Health Consequences of Obesity in Youth: Childhood Predictors of Adult Disease," *Pediatrics*, 1998, vol. 101, no. 3: 518–525.

Economic Burden of Illness in Canada (EBIC), 1998. Policy Research Division, Health Canada, 2002; accessed July 2008; available from http://www.phac-aspc.gc.ca/publicat/ebic-femc98/pdf/ebic1998.pdf

Economic Burden of Illness in Canada (EBIC), 2000. Policy Research Unit, Knowledge Information and Data Systems Division, Office of Public Health Practice, Public Health Agency of Canada. Unpublished material supplied by Alan Diener, EBIC Manager to GPI Atlantic, October, 2008.

Elliott, Victoria Stagg. "Is Obesity a Disease? Clinicians Disagree." *AMNews*, Feb. 6, 2006.

Eneli, I .U., T. Skybo, and C.A. Camargo Jr. "Weight Loss and Asthma: A Systematic Review," *Thorax*, 2008, vol. 63: 671-676.

Engel, Lawrence S., Wong-Ho Chow, Thomas L. Vaughan, Marilie D. Gammon, Harvey A. Risch, Janet L. Stanford, Janet B. Schoenberg, Susan T. Mayne, Robert Dubrow, Heidrun Rotterdam, A. Brian West, Martin Blaser, William J. Blot, Mitchell H. Gail, and Joseph F. Fraumeni Jr. "Population Attributable Risks of Esophageal and Gastric Cancers," *Journal of the National Cancer Institute*, 2003, vol. 95, no. 18: 1404-1413.

Engeland, Anders, Steinar Tretli, and Tone Bjorge. "Height and Body Mass Index in Relation to Esophageal Cancer; 23-Year Follow-up of Two Million Norwegian Men and Women," *Cancer Causes and Control*, 2004, vol. 15, no. 8: 837-843.

Everhart, James E., Meena Khare, Michael Hill, and Kurt R. Maurer. "Prevalence and Ethnic Differences in Gallbladder Disease in the United States," *Gastroenterology*, 1999, vol. 117: 632-639.

Ezzati, Majid, Stephen Vander Hoorn, Alan D. Lopez, Goodarz Danaei, Anthony Rodgers, Colin D. Mathers, and Christopher J.L. Murray. "Comparative Quantification of Mortality and Burden of Disease Attributable to Selected Risk Factors," in *Global Burden of Disease and Risk Factors*, ed. Lopez, Alan D., Colin D. Mathers, Majid Ezzati, Dean T. Jamison and Christopher J. L. Murray, Chapter 4: 241-396. New York: Oxford University Press; The Disease Control Priorities Project is a joint enterprise of the Fogarty International Center of the National Institutes of Health, the World Health Organization, the World Bank, and the Population Reference Bureau, 2006; accessed December 2008; available from For the chapter: http://files.dcp2.org/pdf/GBD/GBD04.pdf; For the book: http://www.dcp2.org/pubs/GBD; For detailed Excel files for individual risk factors: http://www.who.int/healthinfo/global_burden_disease/risk_factors/en/index.html.

Ezzati, Majid, Alan D. Lopez, Anthony Rodgers, Stephen Vander Hoorn, Christopher J. L. Murray, and the Comparative Risk Assessment Collaborating Group. "Selected Major Risk Factors and Global and Regional Burden of Disease," *Lancet*, 2002, vol. 360, no. November 2: 1347-1360.

Fagot-Campagna, A. "Emergence of Type 2 Diabetes Mellitus in Children: Epidemiological Evidence," *Journal of Paediatric Endocrinology and Metabolism*, 2000, vol. 13, no. supplement 6: 1395-1402

Fautrel, Bruno, Ann E. Clarke, Francis Guillemin, Viviane Adam, Yvan St-Pierre, Tina Panaritis, Paul R. Fortin, Henri A. Menard, Cam Donaldson, and John R. Penrod. "Costs of Rheumatoid Arthritis: New Estimates from the Human Capital Method and Comparison to the Willingness-to-Pay Method," *Medical Decision Making*, Mar-Apr, 2007, 138-150.

Field, Alison E., Eugenie H. Coakley, Aviva Must, Jennifer L. Spadano, Nan Laird, William H. Dietz, Eric Rimm, and Graham A. Colditz. "Impact of Overweight on the Risk of Developing Common Chronic Diseases During a 10-Year Period," *Archives of Internal Medicine*, 2001, vol. 161: 1581-1586.

Finkelstein, Eric, and Phaedra Corso. "Cost-of-Illness Analyses for Policy Making: A Cautionary Tale of Use and Misuse," *Expert Review of Pharmacoeconomics Outcomes Research*, 2003, vol. 3, no. 4: 367-369.

Flaherman, V., and G. W. Rutherford. "A Meta-Analysis of the Effect of High Weight on Asthma," *Archives of Disease in Childhood*, 2006, vol. 91: 334-339.

Flegal, K.M., D.F. Williamson, E.R. Pamuk, and H.M. Rosenberg. "Estimating Deaths Attributable to Obesity in the United States," *American Journal of Public Health*, 2004, vol. 94, no. 9: 1486-1489.

Flegal, Katherine M. "Editorial: Estimating the Impact of Obesity," *Sozial Praventivmedizin / Social and Preventive Medicine*, 2005, vol. 50: 73-74.

Flegal, Katherine M., Barry I. Graubard, and David F. Williamson. "Methods of Calculating Deaths Attributable to Obesity," *American Journal of Epidemiology*, 2004, vol. 160, no. 4: 331-338.

_____. "Excess Deaths Associated with Underweight, Overweight, and Obesity," *JAMA - Journal of the American Medical Association*, 2005, vol. 293, no. 15: 1861-1867.

Flegal, Katherine M., Barry I. Graubard, David F. Williamson, and Mitchell H. Gail. "Cause-Specific Excess Deaths Associated with Underweight, Overweight, and Obesity," *JAMA - Journal of the American Medical Association*, 2007, vol. 298, no. 17: 2028-2037.

Flegal, Katherine M., David F. Williamson, and Barry I. Graubard. "Using Adjusted Relative Risks to Calculate Attributable Fractions," *American Journal of Public Health*, Letter to the editor, 2006, vol. 96, no. 3: 398.

Flugsrud, Gunnar B., Lars Nordsletten, Birgitte Espehaug, Leif I. Havelin, Anders Engeland, and Haakon E. Meyer. "The Impact of Body Mass Index on Later Total Hip Arthroplasty for Primary

Osteoarthritis: A Cohort Study in 1.2 Million Persons," *Arthritis and Rheumatism*, 2006, vol. 54, no. 3: 802-807.

Forman, D., and V.J. Burley. "Gastric Cancer: Global Pattern of the Disease and an Overview of Environmental Risk Factors," *Best Practice & Research Clinical Gastroenterology*, 2006, vol. 20, no. 4: 633-649.

Freedman, D.S., W.H. Dietz, S.R. Srinivasan, and G.S. Berenson. "The Relation of Overweight to Cardiovascular Risk Factors among Children and Adolescents: The Bogalusa Heart Study," *Pediatrics* 1999, vol. 103: 1175-1182.

Galassi, Andrea, Kristi Reynolds, and Jiang He. "Metabolic Syndrome and Risk of Cardiovascular Disease: A Meta-Analysis," *The American Journal of Medicine*, 2006, vol. 19: 812-819.

Garner, Rochelle, and Dafna Kohen. "Changes in the Prevalence of Asthma among Canadian Children," *Health Reports*, Statistics Canada Catalogue no. 82-0003, 2008, vol. 19, no. 2: 1-6.

Garvey, J., and W.T. McNicholas. "Effect of Obesity in Patients with Coronary Artery Disease," *The Lancet*, 2006, vol. 368, no. 9548: 1645.

Gershon, Andrea, Chengning Wang, Lisa Cicutto, and Teresa To. "ICES [Institute for Clinical Evaluative Sciences] Report: The Burden of Asthma: Can It Be Eased?" *Healthcare Quarterly*, 2007, vol. 10, no. 1: 22-24.

Gerstman, B. B. *Stratified Analysis: Introduction to Confounding and Interaction*, 2000; accessed November 2008; available from http://www.sjsu.edu/faculty/gerstman/StatPrimer/stratified.PDF.

Giovannucci, Edward, and Dominique Michaud. "The Role of Obesity and Related Metabolic Disturbances in Cancers of the Colon, Prostate, and Pancreas," *Gastroenterology*, 2007, vol. 132: 2208-2225.

Gorber, S. C., M. Tremblay, D. Moher, and B. Gorber. "A Comparison of Direct Vs. Self-Report Measures for Assessing Height, Weight and Body Mass Index: A Systematic Review," *Obesity Reviews*, 2007, vol. 8: 307-326.

Gorber, Sarah Connor, Margot Shields, Mark S. Tremblay, and Ian McDowell. "The Feasibility of Establishing Correction Factors to Adjust Self-Reported Estimates of Obesity," *Health Reports*, Statistics Canada, 2008, vol. 19, no. 3: 71-82. accessed September 2008; available from http://www.statcan.ca/english/freepub/82-003-XIE/2008003/article/10680-en.htm.

Government of Canada. *The Well-Being of Canada's Young Children*, 2003; accessed December 2007; available from http://socialunion.gc.ca/ecd/2003/report2_e/chapter02_e.html.

Graubard, Barry I., and Thomas R. Fears. "Standard Errors for Attributable Risk for Simple and Complex Sample Designs," *Biometrics*, International Biometric Society, 2005, vol. 61, no. 3: 847-855.

Gregg, Edward W., Betsy L. Cadwell, Yiling J. Cheng, Catherine C. Cowie, Desmond E. Williams, Linda Geiss, Michael M. Engelgau, and Frank Vinicor. "Trends in the Prevalence and Ratio of Diagnosed to Undiagnosed Diabetes According to Obesity Levels in the U.S.," *Diabetes Care*, 2004, vol. 17, no. 12: 2806-2812.

Hallqvist, J., A. Ahlbom, F. Diderichsen, and C. Reuterwall. "How to Evaluate Interaction between Causes: A Review of Practices in Cardiovascular Epidemiology," *Journal of Internal Medicine*, 1996, vol. 239: 377-382.

Han, T. S., M. A. R. Tijhuis, M. E. J. Lean, and J. C. Seidell. "Quality of Life in Relation to Overweight and Body Fat Distribution," *American Journal of Public Health*, 1998, vol. 88, no. 12: 1814-1820.

Hanley, J.A. "A Heuristic Approach to the Formulas for Population Attributable Fraction," *Journal of Epidemiology and Community Health*, 2001, vol. 55: 508-514.

Harrison, B.W.D, and M.G. Pearson. "Audit in Acute, Severe Asthma – Who Benefits?" *Journal of Royal College of Physicians of London*, 1992, vol. 27: 387-390.

Hart, C. L., D. J. Hole, D. A. Lawlor, and G. Davey Smith. "How Many Cases of Type 2 Diabetes Mellitus Are Due to Being Overweight in Middle Age? Evidence from the Midspan Prospective Cohort Studies Using Mention of Diabetes Mellitus on Hospital Discharge or Death Records," *Diabetic Medicine*, 2007, vol. 24, no. 1: 73-80.

Haslam, David W., and W. Philip T. James. "Obesity," *The Lancet*, 2005, vol. 366, no. October 1: 1197-1209.

Haydon, Emma, Michael Roerecke, Norman Giesbrecht, Jurgen Rehm, and Marianne Kobus-Matthews. *Chronic Disease in Ontario and Canada: Determinants, Risk Factors and Prevention Priorities*, Ontario Chronic Disease Prevention Alliance and the Ontario Public Health Association, 2006; accessed June 2008; available from http://www.ocdpa.on.ca/docs/CDP-FullReport-Mar06.pdf.

Health Canada. *Canadian Handbook on Health Impact Assessment. Vol 3: Roles for the Health Practitioner*, 1999; accessed Dec 2007; available from http://dsp-psd.communication.gc.ca/Collection/H46-2-99-235-3E-9.pdf.

_____. *Respiratory Disease in Canada*, 2001; accessed April 2008; available from http://www.phac-aspc.gc.ca/publicat/rdc-mrc01/pdf/rdc0901e.pdf.

_____. *Economic Burden of Illness in Canada, 1998*, 2002; accessed Nov 2007; available from http://www.phac-aspc.gc.ca/publicat/ebic-femc98/pdf/ebic1998.pdf.

_____. *Canadian Guidelines for Body Weight Classification in Adults*, 2003; accessed June 2008; available from http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/nutrition/weight_book-livres_des_poids-eng.pdf.

_____. *Canadian Community Health Survey Cycle 2.2, Nutrition (2004). Income-Related Household Food Security in Canada*, Office of Nutrition Policy and Promotion, Health Products and Food Branch, 2007; accessed March 2008; available from http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/surveill/income_food_sec-sec_alim_e.pdf.

Heart and Stroke Foundation of Canada. *Incidence of Cardiovascular Disease*, 2002; accessed November 2008; available from http://ww2.heartandstroke.ca/Page.asp?PageID=33&ArticleID=1077.

_____. *The Growing Burden of Heart Disease and Stroke in Canada*, Health Canada, Canadian Cardiovascular Society, and Heart and Stroke Foundation of Canada, 2003; accessed September 2008; available from http://www.cvdinfobase.ca/cvdbook/CVD_En03.pdf.

_____. *Tipping the Scales of Progress: Heart Disease and Stroke in Canada 2006*, 2006; accessed June 2008; available from http://ww2.heartandstroke.ca/images/english/Tipping_the_Scales.pdf.

Heshka, Stanley, and David B. Allison. "Is Obesity a Disease?" *International Journal of Obesity*, 2001, vol. 25: 1401-1404.

Hillman, David R, Anita Scott Murphy, Ral Antic, and Lynne Pezzullo. "The Economic Cost of Sleep Disorders," *Sleep*, 2006, vol. 29, no. 3: 299-305.

Holcomb, Jr., William L. , Tinnakorn Chaiworapongsa, Douglas A. Luke, and Kevin D. Burgdorf. "An Odd Measure of Risk: Use and Misuse of the Odds Ratio," *Obstetrics and Gynecology*, 2001, vol. 98, no. 4: 685-688.

House of Commons Health Committee (U.K.). *Obesity: Third Report of Session 2003-04, Annex 1: The Economic Costs of Obesity*, The United Kingdom Parliament, 2004; accessed July 2008; available from http://www.publications.parliament.uk/pa/cm200304/cmselect/cmhealth/23/23.pdf.

Hu, Jinfu, Yang Mao, Kathy White, and The Canadian Cancer Registries Epidemiology Research Group. "Overweight and Obesity in Adults and Risk of Renal Cell Carcinoma in Canada," *Sozial- und Präventivmedizin / Social and Preventive Medicine*, 2003, vol. 48: 178-185.

Hubert, H.B., M. Feinleib, P.M. McNamara, and W.P Castelli. "Obesity as an Independent Risk Factor for Cardiovascular Disease: A 26-Year Follow-up of Participants in the Framingham Heart Study," *Circulation. Journal of the American Heart Association*, 1983, vol. 67: 968-977.

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Human Resources and Social Development Canada (HRSDC). *Indicators of Well-Being in Canada: Obesity*, 2008; accessed August 2008; available from http://www4.hrsdc.gc.ca/indicator.jsp?lang=en&indicatorid=6.

Institute of Medicine. *Estimating the Contributions of Lifestyle-Related Factors to Preventable Death: A Workshop Summary*, The National Academies Press, 2005; accessed August 2008; available from http://www.iom.edu/?id=27316.

Jain, Meera G., Geoffrey R. Howe, and Thomas E. Rohan. "Nutritional Factors and Endometrial Cancer in Ontario, Canada," *Cancer Control*, 2000, vol. 7, no. 3: 288-296.

Jain, Meera G., A.B. Miller, T.E. Rohan, J.T. Rehm, S.J. Bondy, M.J. Ashley, J.E. Cohen, and R.G. Ferrence. "Body Mass Index and Mortality in Women: Follow-up of the Canadian National Breast Screening Study Cohort," *International Journal of Obesity*, 2005, vol. 29: 792-797.

James, W. Phillip T., Rachel Jackson-Leach, Cliona Ni Mhurchu, Eleni Kalamara, Maryam Shayeghi, Neville J. Rigby, Chozuru Nishida, and Anthony Rodgers. "Overweight and Obesity (High Body Mass Index)," in *Comparative Quantification of Health Risks – Global and Regional Burden of Diseases Attributable to Selected Major Risk Factors*, ed. Ezzati, Majid, Alan D. Lopez, Anthony Rodgers and Christopher J.L. Murray, Vol 1, Chapter 8, 497-596, World Health Organization, 2004; accessed September 2008; available from http://www.who.int/publications/cra/chapters/volume1/0497-0596.pdf.

Jensen, Majken K., Stephanie E. Chiuve, Eric B. Rimm, Claus Dethlefsen, Anne Tjonneland, Albert M. Joensen, and Kim Overvad. "Obesity, Behavioral Lifestyle Factors, and Risk of Acute Coronary Events," *Circulation. Journal of the American Heart Association*, 2008, vol. 117, no. 24: 3062-3069.

Johnston, Elizabeth, Shanthi Johnson, Peter McLeod, and Mark Johnston. "The Relation of Body Mass Index to Depressive Symptoms," *Canadian Journal of Public Health*, 2004, vol. 95, no. 3: 179-184.

Kaaks, Rudolf, Annekatrin Lukanova, and Mindy S. Kurzer. "Obesity, Endogenous Hormones, and Endometrial Cancer Risk: A Synthetic Review," *Cancer Epidemiology, Biomarkers & Prevention*, 2002, vol. 11, no. 12: 1531-1543.

Katzmarzyk, Peter, and Christopher I. Ardern. "Overweight and Obesity Mortality Trends in Canada, 1985-2000," *Canadian Journal of Public Health*, 2004, vol. 95, no. 1: 16-20.

Katzmarzyk, Peter T. "Obesity and Physical Activity among Aboriginal Canadians," *Obesity*, 2008, vol. 16, no. 1: 184-190.

Katzmarzyk, Peter T., Cora L. Craig, and Claude Bouchard. "Underweight, Overweight and Obesity: Relationships with Mortality in the 13-Year Follow-up of the Canada Fitness Survey," *Journal of Clinical Epidemiology*, 2001, vol. 54: 916-920.

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Katzmarzyk, Peter T., and Ian Janssen. "The Economic Costs Associated with Physical Inactivity and Obesity in Canada: An Update," *Canadian Journal of Applied Physiology*, 2004, vol. 29, no. 1: 90-115.

Katzmarzyk, Peter T., Ian Janssen, and Christopher I. Ardern. "Physical Inactivity, Excess Adiposity and Premature Mortality," *Obesity Reviews*, 2003, vol. 4: 257-290.

Katzmarzyk, Peter T., and Caitlin Mason. "Prevalence of Class I, II, and III Obesity in Canada," *Canadian Medical Association Journal*, 2006, vol. 174, no. 2: 156-157.

Kelsall, Diane L. "Editor's Letter. What Galls You? Treatment Options in Gallstone Disease," *Informed*, Institute for Clinical Evaluative Sciences (ICES), 2005, vol. 11, no. 4: 1, 3.

Kenchaiah, S., J.M. Gaziano, and R.S. Vasan. "Impact of Obesity on the Risk of Heart Failure and Survival after the Onset of Heart Failure," *Medical Clinics of North America*, 2004, vol. 88: 1273–1294.

Kendall, Ora, Tammy Lipskie, and Shauna MacEachern. "Canadian Health Surveys, 1950-1997," *Chronic Diseases in Canada*, Public Health Agency of Canada, 1997, vol. 18, no. 2: 1-20. accessed July 2008; available from http://www.phac-aspc.gc.ca/publicat/cdic-mcc/18-2/b_e.html.

Key, Timothy J., Pia K. Verkasalo, and Emily Banks. "Epidemiology of Breast Cancer," *The Lancet*, 2001, vol. 2, no. March: 133-140.

Khaodhiar, L., K.C. McCowen, and G.L. Blackburn. "Obesity and Its Comorbid Conditions," *Clinical Cornerstone: A Clinical Journal for Primary Care*, 1999, vol. 2: 17-31.

King, Deborah S., and Marion R. Wofford. " Obesity and Hypertension," *Drug Topics*, 2000, vol. 144, no. 1: 59-66.

Koebnick, Corinna, Dominique Michaud, Steven C. Moore, Yikyung Park, Albert Hollenbeck, Rachel Ballard-Barbash, Arthur Schatzkin, and Michael F. Leitzmann. "Body Mass Index, Physical Activity, and Bladder Cancer in a Large Prospective Study," *Cancer Epidemiology Biomarkers & Prevention*, 2008, vol. 17, no. 5: 1214-1221.

Kopelman, Peter G. "Health Risks Associated with Overweight and Obesity," *Obesity Reviews*, 2007, vol. 8, no. Suppl. 1: 13-17.

Krauss, Ronald M., Mary Winston, Barbara J. Fletcher, and Scott M. Grundy. "Obesity: Impact on Cardiovascular Disease," *Circulation. Journal of the American Heart Association*, 1998, vol. 98: 1472-1476.

Kubo, Ai, and Douglas A. Corley. "Body Mass Index and Adenocarcinomas of the Esophagus or Gastric Cardia: A Systematic Review and Meta-Analysis," *Cancer Epidemiology Biomarkers & Prevention*, 2006, vol. 15, no. 5: 872-878.

Lagergren, J., R. Bergstrom, and O. Nyren. "Association between Body Mass and Adenocarcinoma of the Esophagus and Gastric Cardia," *Annals of Internal Medicine*, 1999, vol. 130: 883-890.

Lagergren, Jesper. "Controversies Surrounding Body Mass, Reflux, and Risk of Oesophageal Adenocarcinoma," *The Lancet*, 2006, vol. 7, no. April: 347-349.

Lammert, Frank, and Juan-Francisco Miquel. "Gallstone Disease: From Genes to Evidence-Based Therapy," *Journal of Hepatology*, 2008, vol. 48: S124-S135.

Larsson, Susanna C., Nicola Orsini, and Alicja Wolk. "Body Mass Index and Pancreatic Cancer Risk: A Meta-Analysis of Prospective Studies," *International Journal of Cancer*, 2007, vol. 120: 1993-1998.

Larsson, Susanna C., and Alicja Wolk. "Body Mass Index and Risk of Multiple Myeloma: A Meta-Analysis," *International Journal of Cancer*, 2007, vol. 121: 2512-2516.

_____. "Obesity and Colon and Rectal Cancer Risk: A Meta-Analysis of Prospective Studies," *American Journal of Clinical Nutrition*, 2007, vol. 86: 556-565.

_____. "Obesity and Risk of Non-Hodgkin's Lymphoma: A Meta-Analysis," *International Journal of Cancer*, 2007, vol. 121: 1564-1570.

_____. "Obesity and the Risk of Gallbladder Cancer: A Meta-Analysis," *British Journal of Cancer*, 2007, vol. 96, no. 9: 1457-1461.

_____. "Overweight, Obesity and Risk of Liver Cancer: A Meta-Analysis of Cohort Studies," *British Journal of Cancer*, 2007, vol. 97: 1005-1008.

_____. "Overweight and Obesity and Incidence of Leukemia: A Meta-Analysis of Cohort Studies," *International Journal of Cancer*, 2008, vol. 122: 1418-1421.

Lau, David C.W., James D. Douketis, Katherine M. Morrison, Irene M. Hramiak, Arya M. Sharma, and Ehud Ur for members of the Obesity Canada Clinical Practice Guidelines Expert Panel. "2006 Canadian Clinical Practice Guidelines on the Management and Prevention of Obesity in Adults and Children [Summary]," *Canadian Medical Association Journal*, 2007, vol. 176, no. Suppl. 8: S1-S13.

Lawlor, Debbie A., Carole L. Hart, David J. Hole, and George Davey Smith. "Reverse Causality and Confounding and the Associations of Overweight and Obesity with Mortality," *Obesity*, 2006, vol. 14, no. 12: 2294-2304.

Le Petit, Christel, and Jean-Marie Berthelot. *Obesity: A Growing Issue*, Statistics Canada, component of Catalogue no 82-618MWE2005003, 2005; accessed July 2008; available from http://www.statcan.ca/english/research/82-618-MIE/2005003/pdf/82-618-MIE2005003.pdf.

Levin, M.L. "The Occurence of Lung Cancer in Man," *Acta Unio Internationalis Contra Cancrum*, 1953, vol. 9: 531-535.

Lindblad, Mats, Luis A. Garcıa Rodrıguez, and Jesper Lagergren. "Body Mass, Tobacco and Alcohol and Risk of Esophageal, Gastric Cardia, and Gastric Non-Cardia Adenocarcinoma among Men and Women in a Nested Case-Control Study," *Cancer Causes and Control*, 2005, vol. 16: 285-294.

Littman, Alyson J., Emily White, and Alan R. Kristal. "Anthropometrics and Prostate Cancer Risk," *American Journal of Epidemiology*, 2007, vol. 165, no. 11: 1271-1279.

Longo, Christopher J., Margaret Fitch, Raisa B. Deber, and A. Paul Williams. "Financial and Family Burden Associated with Cancer Treatment in Ontario, Canada," *Support Care Cancer*, 2006, vol. 14: 1077-1085

Luo, Wei, Howard Morrison, Margaret de Groh, Chris Waters, Marie DesMeules, Elaine Jones-McLean, Anne-Marie Ugnat, Sylvie Desjardins, Morgan Lim, and Yang Mao. "The Burden of Adult Obesity in Canada," *Chronic Diseases in Canada*, 2007, vol. 27, no. 4: 135-144.

Ma, Jing, Haojie Li, Ed Giovannucci, Lorelei Mucci, Weiliang Qiu, Paul L. Nguyen, J. Michael Gaziano, Michael Pollak, and Meir J. Stampfer. "Prediagnostic Body-Mass Index, Plasma C-Peptide Concentration, and Prostate Cancer-Specific Mortality in Men with Prostate Cancer: A Long-Term Survival Analysis," *Lancet Oncology*, 2008, vol. 9, no. November: preview issue.

MacInnis, Robert J., and Dallas R. English. "Body Size and Composition and Prostate Cancer Risk: Systematic Review and Meta-Regression Analysis," *Cancer Causes and Control*, 2006, vol. 17, no. 8: 989-1003.

Magnusson, Cecilia, John Baron, Ingemar Persson, Alicja Wolk, Reinhold Bergstro, Dimitrios Trichopoulos, and Hans-Olov Adami. "Body Size in Different Periods of Life and Breast Cancer Risk in Post-Menopausal Women," *International Journal of Cancer*, 1998, vol. 76: 29-34.

Malo, Shaun. *Chronic Disease Projections 2006 to 2035. Ischemic Heart Disease*, Alberta Health and Wellness, 2008; accessed July 2008; available from http://www.health.alberta.ca/resources/publications/CDP_Ischemic-HD-2006-35.pdf.

Mann, J.I. "Diet and Risk of Coronary Heart Disease and Type 2 Diabetes," *The Lancet*, 2002, vol. 360, no. Sept 7: 783-789.

Mao, Yang, Saiyi Pan, Shi Wu Wen, Kenneth C. Johnson, and The Canadian Cancer Registries Epidemiology Research Group. "Physical Inactivity, Energy Intake, Obesity and the Risk of Rectal Cancer in Canada," *International Journal of Cancer*, 2003, vol. 105: 831-837.

Marrett, Loraine D., Prithwish De, Parisa Airia, and Dagny Dryer for the steering committee of Canadian Cancer Statistics 2008. "Cancer in Canada in 2008," *Canadian Medical Association Journal*, 2008, vol. 179, no. 11 - November 18: 1163-1170.

Masoli, Matthew, Denise Fabian, Shaun Holt, and Richard Beasley. *Global Burden of Asthma*, Global Initiative for Asthma (GINA), 2004; accessed November 2008; available from http://www.ginasthma.com/ReportItem.asp?l1=2&l2%20=2&intId=94.

Mathers, C., T. Vos, and C. Stevenson. *The Burden of Disease and Injury in Australia*, Australian Institute of Health and Welfare, Catalogue no PHE17, 1999; accessed July 2008; available from http://www.aihw.gov.au/publications/index.cfm/title/5180.

Mathers, Colin, Theo Vos, Alan Lopez, Josh Salomon, and Majid Ezzati. *National Burden of Disease Studies: A Practical Guide*, Edition 2.0. Global Program on Evidence for Health Policy. Geneva: World Health Organization, 2001; accessed June 2008; available from http://www.who.int/healthinfo/nationalburdenofdiseasemanual.pdf.

Matheson, Flora I., Rahim Moineddin, and Richard H. Glazier. "The Weight of Place: A Multilevel Analysis of Gender, Neighborhood Material Deprivation, and Body Mass Index among Canadian Adults," *Social Science & Medicine*, 2008, vol. 66: 675-690.

McCullough, Marjorie L., Alpa V. Patel, Roshni Patel, Carmen Rodriguez, Heather Spencer Feigelson, Elisa V. Bandera, Ted Gansler, Michael J. Thun, and Eugenia E. Calle. "Body Mass and Endometrial Cancer Risk by Hormone Replacement Therapy and Cancer Subtype," *Cancer Epidemiology Biomarkers and Prevention*, 2008, vol. 17, no. 1: 73-79.

McElroy, Susan L., Renu Kotwal, Shishuka Malhotra, Erik B. Nelson, Paul E. Keck Jr., and Charles B. Nemeroff. "Are Mood Disorders and Obesity Related? A Review for the Mental Health Professional," *Journal of Clinical Psychiatry*, 2004, vol. 65: 634-651.

McEwen, Laura N., Catherine Kim, Mary Haan, Debashis Ghosh, Paula Lantz, Carol Mangione, Monika Safford, David Marrero, Theodore Thompson, and William Herman. "Diabetes Reporting as a Cause of Death: Results from the Translating Research into Action for Diabetes (TRIAD) Study," *Diabetes Care*, 2006, vol. 29, no. 2: 247-253.

McGee, D.L., and Diverse Populations Collaboration. "Body Mass Index and Mortality: A Meta-Analysis Based on Person-Level Data from Twenty-Six Observational Studies," *Annals of Epidemiology*, 2005, vol. 15: 87-97.

McIntyre, A.M. "Burden of Illness Review of Obesity: Are the True Costs Realised?" *Journal of the Royal Society for the Promotion of Health*, 1998, vol. 118, no. 2.

McIntyre, Roger S., Jakub Z. Konarski, Kathryn Wilkins, Joanna K. Soczynska, and Sidney H. Kennedy. "Obesity in Bipolar Disorder and Major Depressive Disorder: Results from a National Community Health Survey on Mental Health and Well-Being," *Canadian Journal of Psychiatry*, 2006, vol. 51, no. 5: 274-280.

McIntyre, Roger S., Deborah A. Mancini, Mark M.Pearce, Peter Silverstone, Pierre Chue, Virginia L. Misener, and Jakub Z. Konarski. "Mood and Psychotic Disorders and Type 2 Diabetes: A Metabolic Triad," *Canadian Journal of Diabetes*, 2005, vol. 29, no. 2: 122-132.

McIntyre, Roger S., Susan L. McElroy, Jakub Z. Konarski, Joana K. Soczynska, Alexandra Bottas, Saulo Castel, Kathryn Wilkins, and Sidney H. Kennedy. "Substance Use Disorders and Overweight/Obesity in Bipolar I Disorder: Preliminary Evidence for Competing Addictions," *Journal of Clinical Psychiatry*, 2007, vol. 68: 1352-1357.

McIntyre, Roger S., Joanna K. Soczynska, John Beyer, Hanna Woldeyohannes, Candy Law, Andrew Miranda, Jakub Z. Konarski, and Sidney H. Kennedy. "Medical Comorbidity in Bipolar Disorder: Reprioritizing Unmet Needs," *Current Opinion in Psychiatry*, 2007, vol. 20, no. 4: 406-416.

McIntyre, Roger S., Joanna K. Soczynska, Jakub Z. Konarski, Hanna O. Woldeyohannes, Candy W. Y. Law, Andrew Miranda, Don Fulgosi, and Sidney H. Kennedy. "Should Depressive Syndromes Be Reclassified as "Metabolic Syndrome Type II?" *Annals of Clinical Psychiatry*, 2007, vol. 19, no. 4: 257-264.

McKeown, T. *The Role of Medicine - Dream, Mirage or Nemesis?*, Princeton, New Jersey: Princeton University Press, 1979.

MedicineNet.com. *Definition of Dyslipidemia*, 2008; accessed November 2008; available from http://www.medterms.com/script/main/art.asp?articlekey=33979.

Mehra, Reena, and Susan Redline. "Sleep Apnea: A Proinflammatory Disorder That Coaggregates with Obesity," *Journal of Allergy and Clinical Immunology*, 2008, vol. 121, no. 5: 1096-1102.

Miettinen, O.S. . "Proportion of Disease Caused or Prevented by a Given Exposure, Trait or Intervention," *American Journal of Epidemiology*, 1974, vol. 99: 325-332.

Miller, A. Lynn. *Action Plan for Arthritis*, Human Kinetics, 2003.

Moghaddam, Alireza Ansary, Mark Woodward, and Rachel Huxley. "Obesity and Risk of Colorectal Cancer: A Meta-Analysis of 31 Studies with 70,000 Events," *Cancer Epidemiology Biomarkers & Prevention*, 2007, vol. 16, no. 12: 2533-2547.

Mokdad, Ali H., Earl S. Ford, Barbara A. Bowman, William H. Dietz, Frank Vinicor, Virginia S. Bales, and James S. Marks. "Health Risk Factors, 2001 Prevalence of Obesity, Diabetes, and Obesity-Related Health Risk Factors, 2001," *Journal of the American Medical Association (JAMA)*, 2003, vol. 289, no. 1: 76-79.

Moyer, V.A., J.D. Klein, J.K. Ockene, S.M. Teutsch, M.S. Johnson, J.D. Allan, and Childhood Obesity Working Group of the U.S. Preventive Services Task Force. "Screening for Overweight in Children and Adolescents: Where Is the Evidence? A Commentary by the Childhood Obesity

Working Group of the U.S. Preventive Services Task Force," *Pediatrics*, 2005, vol. 116: 235–238.

Mudur, G.S. *Overnight, Many Overweight Indian Figures Revised to Tone up Flab Fight*, Calcutta, India, *The Telegraph*, 2008; accessed February 2009; available from http://www.telegraphindia.com/1081126/jsp/frontpage/story_10165735.jsp.

Must, A., J. Spadano, and E.H. Coakley. "The Disease Burden Associated with Overweight and Obesity," *Journal of the American Medical Association*, 1999, vol. 282: 1523–1529.

Mustillo, Sarah, Carol Worthman, Alaattin Erkanli, Gordon Keeler, Adrian Angold, and E. Jane Costello. "Obesity and Psychiatric Disorder: Developmental Trajectories," *Pediatrics*, 2003, vol. 111, no. 4: 851-859.

National Asthma Control Task Force. *The Prevention and Management of Asthma in Canada: A Major Challenge Now and in the Future*, Laboratory Centre for Disease Control (LCDC) of Health Canada, 2000; accessed October 2008; available from http://www.phac-aspc.gc.ca/publicat/pma-pca00/pdf/asthma00e.pdf.

National Audit Office Comptroller and Auditor General (U.K.). *Tackling Obesity in England*, National Audit Office, 2001; accessed July 2008; available from www.nao.org.uk/publications/nao_reports/00-01/0001220.pdf.

National Cancer Institute (U.S.). *Fact Sheet: Obesity and Cancer: Questions and Answers*, 2004; accessed July 2008; available from http://www.cancer.gov/cancertopics/factsheet/Risk/obesity.

National Health and Medical Research Council (Australia). *Clinical Practice Guidelines for the Management of Overweight and Obesity in Adults*, 2004; accessed July 2008; available from http://www.health.gov.au/internet/main/publishing.nsf/Content/obesityguidelines-guidelines-adults.htm.

National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). *Statistics Related to Overweight and Obesity*, 2006; accessed September 2008; available from http://www.niddk.nih.gov/health/nutrit/pubs/statobes.htm.

National Institutes of Health. "National Institutes of Health Consensus Development Conference Statement on Gallstones and Laparoscopic Cholecystectomy." *American Journal of Surgery*, 1993, vol. 165: 390-398.

Neovius, Martin, M. Kark, and F. Rasmussen. "Association between Obesity Status in Young Adulthood and Disability Pension," *International Journal of Obesity*, 2008, vol. 32: 1319-1326.

Ng, Edward, Kaberi Dasgupta, and Jeffrey A. Johnson. "An Algorithm to Differentiate Diabetic Respondents in the Canadian Community Health Survey," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2008, vol. 19, no. 1: 1-9.

352

Nicodemus, Kristin K., Carol Sweeney, and Aaron R. Folsom. "Evaluation of Dietary, Medical and Lifestyle Risk Factors for Incident Kidney Cancer in Postmenopausal Women," *International Journal of Cancer*, 2004, vol. 108: 115-121.

Nutrition Committee of the Canadian Paediatric Society. "The Nutrition Canada Survey: A Review," *Canadian Medical Association Journal*, 1976, vol. 115: 775-777.

Obesity Society Council. "Obesity as a Disease: The Obesity Society Council Resolution," *Obesity*, 2008, vol. 16: 1151.

Ogden, Cynthis L., Susan Z. Yanovski, Margaret D. Carroll, and Katherine M. Flegal. "The Epidemiology of Obesity," *Gastroenterology*, 2007, vol. 132: 2087-2102.

Olsen, Catherine M., Adele C. Green, David C. Whiteman, Shahram Sadeghi, Fariba Kolahdooz, and Penelope M. Webb. "Obesity and the Risk of Epithelial Ovarian Cancer: A Systematic Review and Meta-Analysis," *European Journal of Cancer*, 2007, vol. 43: 690-709.

Olsen, Catherine M., Christina M. Nagle, David C. Whiteman, David M. Purdie, Adele C. Green, Penelope M. Webb, and Australian Cancer Study (Ovarian Cancer) and Australian Ovarian Cancer Study Group. "Body Size and Risk of Epithelial Ovarian and Related Cancers: A Population-Based Case-Control Study," *International Journal of Cancer*, 2008, vol. 123: 450-456.

Olsen, Douglas O. *Gallbladder Disease and the Obese Patient*, Obesity Action Coalition, Centennial Center for the Treatment of Obesity, 2008; accessed October 2008; available from http://www.obesityaction.org/magazine/oacnews9/obesityrelateddiseases.php.

Olshansky, S. Jay, Douglas J. Passaro, Ronald C. Hershow, Jennifer Layden, Bruce A. Carnes, Jacob Brody, Leonard Hayflick, Robert N. Butler, David B. Allison, and David S. Ludwig. "A Potential Decline in Life Expectancy in the United States in the 21st Century," *New England Journal of Medicine*, 2005, vol. 352, no. 11: 1138-1145.

Organisation for Economic Co-operation and Development (OECD). *Health at a Glance: OECD Indicators 2003: Chart 8. Increasing Obesity Rates among the Adult Population in OECD Countries*, 2003; accessed July 2008; available from http://www.oecd.org/searchResult/0,3400,en_2649_34631_1_1_1_1_37407,00.html.

_____. *OECD Factbook 2008: Economic, Environmental and Social Statistics: Obesity*, 2008; accessed July 2008; available from http://puck.sourceoecd.org/vl=1680972/cl=12/nw=1/rpsv/factbook/110103.htm.

Orpana, Heather M., Mark S. Tremblay, and Philippe Fines. "Trends in Weight Change among Canadian Adults," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2007, vol. 18, no. 2: 9-16.

Pan, Sai Yi, Marie DesMeules, Howard Morrison, Shi Wu Wen, and the Canadian Cancer Registries Epidemiology Research Group. "Obesity, High Energy Intake, Lack of Physical Activity, and the Risk of Kidney Cancer," *Cancer Epidemiology Biomarkers & Prevention*, 2006, vol. 15, no. 12: 2453-2460.

Pan, Sai Yi, Kenneth C. Johnson, Anne-Marie Ugnat, Shi Wu Wen, Yang Mao, and Canadian Cancer Registries Epidemiology Research Group. "Association of Obesity and Cancer Risk in Canada," *American Journal of Epidemiology*, 2004, vol. 159, no. 3: 259-268.

Pan, Sai Yi, Yang Mao, Anne-Marie Ugnat, and the Canadian Cancer Registries Epidemiology Research Group. "Physical Activity, Obesity, Energy Intake, and the Risk of Non-Hodgkin's Lymphoma: A Population-Based Case-Control Study," *American Journal of Epidemiology*, 2006, vol. 162, no. 12: 1162-1173.

Patel, Alpa V., Heather Spencer Feigelson, Jeffrey T. Talbot, Marjorie L. McCullough, Carmen Rodriguez, Roshni C. Patel, Michael J. Thun, and Eugenia E. Calle. "The Role of Body Weight in the Relationship between Physical Activity and Endometrial Cancer: Results from a Large Cohort of Us Women," *International Journal of Cancer*, 2008, vol. 123: 1877-1882.

Patra, Jayadeep, Svetlana Popova, Jurgen Rehm, Susan Bondy, Robynne Flint, and Norman Giesbrecht. *Economic Cost of Chronic Disease in Canada 1995-2003*, Ontario Chronic Disease Prevention Alliance and the Ontario Public Health Association, 2007; accessed July 2008; available from http://www.ocdpa.on.ca/docs/OCDPA_EconomicCosts.pdf.

Patten, Scott B., and Cynthia A. Beck. "Major Depression and Mental Health Care Utilization in Canada: 1994-2000," *Canadian Journal of Psychiatry*, 2004, vol. 49, no. 5: 303-309.

Patten, Scott, and Heather Juby. *A Profile of Clinical Depression in Canada*, Statistics Canada, Research Data Centre Network (RDC), RDC Synthesis Series, 2008; accessed October 2008; available from https://dspace.ucalgary.ca/bitstream/1880/46327/6/Patten_RSS1.pdf.

Physicians for a Smoke-free Canada. accessed April 2008; available from http://www.smoke-free.ca/Second-Hand-Smoke/health_kids.htm.

Pischon, T., H. Boeing, K. Hoffmann, and 45 Other Colleagues. "General and Abdominal Adiposity and Risk of Death in Europe," *The New England Journal of Medicine*, 2008, vol. 359, no. 20: 2105-2120.

Pischon, Tobias, Ute Nothlings, and Heiner Boeing. "Obesity and Cancer," *Proceedings of the Nutrition Society*, 2008, vol. 67: 128-145.

Portincasa, Piero, Antonio Moschetta, and Giuseppe Palasciano. "Cholesterol Gallstone Disease," *The Lancet*, 2006, vol. 368, no. July 15: 230-234.

Poulain, Magali, Marieve Doucet, Genevieve C. Major, Vicky Drapeau, Frederic Series, Louis-Philippe Boulet, Angelo Tremblay, and Francois Maltais. "The Effect of Obesity on Chronic

Respiratory Diseases: Pathophysiology and Therapeutic Strategies," *Canadian Medical Association Journal*, 2006, vol. 174, no. 9: 1293-1299.

Prasad, Arun. *Gallbladder Disease - Is Surgery Always Needed?* Apollo Hospital, New Delhi, n.d.; accessed October 2008; available from http://www.angelfire.com/ar/laparoscopy/gallstone.html.

Public Health Agency of Canada (PHAC). *Leading Causes of Death and Hospitalization in Canada*, 2005; accessed November 2008; available from http://www.phac-aspc.gc.ca/publicat/lcd-pcd97/index-eng.php.

_____. *Life and Breath: Respiratory Disease in Canada*, Ottawa: PHAC, 2007; accessed October 2008; available from http://www.phac-aspc.gc.ca/publicat/2007/lbrdc-vsmrc/pdf/PHAC-Respiratory-WEB-eng.pdf.

_____. *Cancer Surveillance on-Line*, 2008; accessed November 2008; available from http://dsol-smed.phac-aspc.gc.ca/dsol-smed/cancer/index_e.html.

_____. *Cardiovascular Disease Surveillance on-Line*, 2008; accessed November 2008; available from http://dsol-smed.phac-aspc.gc.ca/dsol-smed/cvd/c_quik_e.html.

_____. *Chronic Disease Infobase*, 2008; accessed November 2008; available from http://cvdinfobase.ca/ncdasp/Profile_e.aspx.

_____. *Diabetes in Canada: Highlights from the National Diabetes Surveillance System 2004 - 2005*, 2008; accessed October 2008; available from http://www.phac-aspc.gc.ca/publicat/2008/dicndss-dacsnsd-04-05/pdf/dicndss-04-05-eng.pdf.

_____. *Diabetes. Facts and Figures*, n.d.; accessed July 2008; available from http://www.phac-aspc.gc.ca/ccdpc-cpcmc/diabetes-diabete/english/index.html.

Purdie, David M., Christopher J. Bain, Penelope M. Webb, David C. Whiteman, Sandi Pirozzo, and Adele C. Green. "Body Size and Ovarian Cancer: Case-Control Study and Systematic Review (Australia)," *Cancer Causes and Control*, 2001, vol. 12: 855-863.

Raeder, Maria B., Johan Ferno, Audun O. Vik-mo, and Vidar M. Steen. "SSREBP Activation by Antipsychotic- and Antidepressant-Drugs in Cultured Human Liver Cells: Relevance for Metabolic Side-Effects?" *Molecular and Cellular Biochemistry*, 2006, vol. 289, no. 1-2: 167-173.

Randi, Giorgia, Silvia Franceschi, and Carlo La Vecchia. "Gallbladder Cancer Worldwide: Geographical Distribution and Risk Factors," *International Journal of Cancer*, 2006, vol. 118: 1591-1602.

Raphael, Dennis. *Inequality Is Bad for Our Hearts: Why Low Income and Social Exclusion Are Major Causes of Heart Disease in Canada*, Toronto: North York Heart Health Network, 2001;

355

accessed Nov 2007; available from
http://action.web.ca/home/narcc/attach/Inequality%20is%20Bad%20for%20Our%20Hearts%20-%202001%20Report%5B1%5D.pdf.

Reddy, K. Srinath, and Martijn B. Katan. "Diet, Nutrition and the Prevention of Hypertension and Cardiovascular Diseases," *Public Health Nutrition*, 2004, vol. 7, no. 1A: 167–186.

Redline, Susan, Peter V. Tishler, Mark Schluchter, Joan Aylor, Kathryn Clark, and Gregory Graham. "Risk Factors for Sleep-Disordered Breathing in Children: Associations with Obesity, Race, and Respiratory Problems," *American Journal of Respiratory and Critical Care Medicine*, 1999, vol. 159, no. 5: 1527-1532.

Renehan, Andrew G, Margaret Tyson, Matthias Egger, Richard F Heller, and Marcel Zwahlen. "Body-Mass Index and Incidence of Cancer: A Systematic Review and Meta-Analysis of Prospective Observational Studies," *Lancet*, 2008, vol. 371: 569-578.

Rockhill, Beverly, Beth Newman, and Clarice Weinberg. "Use and Misuse of Population Attributable Fractions," *American Journal of Public Health*, 1998, vol. 88, no. 1: 15-19.

Rockhill, Beverly, Clarice Weinberg, Robert A. Hahn, Nancy D. Barker, Steven M. Teutsch, Elaine Eaker, Waldemar Sosniak, and Nancy Krieger. "Error in Population Attributable Risk Calculation," *Epidemiology*, 1996, vol. 7, no. 4: 453-454.

Rodriguez, Carmen, Stephen J. Freedland, Anusila Deka, Eric J. Jacobs, Marjorie L. McCullough, Alpa V. Patel, Michael J. Thun, and Eugenia E. Calle. "Body Mass Index, Weight Change, and Risk of Prostate Cancer in the Cancer Prevention Study II Nutrition Cohort," *Cancer Epidemiology Biomarkers & Prevention*, 2007, vol. 16, no. 1: 63-69.

Romero-Corral, A., V.K. Somers, J. Sierra-Johnson, R.J. Thomas, M.L. Collazo-Clavell, J. Korinek, T.G. Allison, J.A. Batsis, F.H. Sert-Kuniyoshi, and F. Lopez-Jimenez. "Accuracy of Body Mass Index in Diagnosing Obesity in the Adult General Population," *International Journal of Obesity*, 2008, vol. 32: 959-966.

Ruhl, Constance E., and James E. Everhart. "Relationship of Serum Leptin Concentration and Other Measures of Adiposity with Gallbladder Disease," *Heptology*, 2001, vol. 34, no. 5: 877-883.

Sabry, Z.I., E. Campbell, J.A. Campbell, and D.F. Bray. *Nutrition Canada National Survey; 1970-1972*, (Original publishers: Nutrition Canada and Department of National Health and Welfare, Food and Drug Directorate), Public Archives of Canada, 2005.

Samanic, C., Gridley. G., W.H. Chow, J. Lubin, R.N. Hoover, and J.F. Fraumeni Jr. "Obesity and Cancer Risk among White and Black United States Veterans," *Cancer Causes and Control*, 2004, vol. 15: 35-43.

Schachter, L. M., C. M. Salome, J. K. Peat, and A. .J Woolcock. "Obesity Is a Risk for Asthma and Wheeze but Not Airway Hyperresponsiveness," *Thorax*, 2001, vol. 56: 4-8.

Schmidt, Carsten Oliver, and Thomas Kohlmann. "When to Use the Odds Ratio or the Relative Risk?" *International Journal of Public Health*, 2008, vol. 53: 165-167.

Schopflocher, Donald. *Self-Reported Body Mass Index and Its Correlates in Alberta: A Portrait from Survey and Administrative Data Sources*, Alberta Health and Wellness, 2005; accessed July 2008; available from http://www.health.alberta.ca/public/NT_ObesityV6.pdf.

Scott, Keith G., Craig A. Mason, and Derek A. Chapman. "The Use of Epidemiological Methodology as a Means of Influencing Public Policy," *Child Development*, 1999, vol. 70, no. 5: 1263-1272.

Shaffer, Eldon A. "Epidemiology and Risk Factors for Gallstone Disease: Has the Paradigm Changed in the 21st Century?" *Current Gastroenterology Reports*, 2005, vol. 7: 132-140.

Sheth, S.G., F.D. Gordon, and S. Chopra. "Nonalcoholic Steatohepatitis," *Annals of Internal Medicine*, 1997, vol. 126: 137–145.

Shields, Margot. *Nutrition: Findings from the Canadian Community Health Survey: Measured Obesity. Overweight Canadian Children and Adolescents*, Statistics Canada, Catalogue no. 82-620-MWE2005001, 2005; accessed June 2008; available from http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/cobesity.pdf.

_____. "Overweight and Obesity among Children and Youth," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol. 17, no. 3: 27-42.

_____. "Stress and Depression in the Employed Population," *Health Reports*, Statistics Canada, Catalogue no. 82-003 2006, vol. 17, no. 4: 11-29.

_____, personal communication with Karen Hayward, August 21, 2008.

Shields, Margot, Sarah Connor Gorber, and Mark S. Tremblay. "Effects of Measurement on Obesity and Morbidity," *Health Reports*, Statistics Canada, Catalogue no. 82-003-X, 2008, vol. 19, no. 2: 1-8. accessed June 2008; available from http://www.statcan.ca/english/freepub/82-003-XIE/2008002/article/10564-en.pdf.

_____. "Estimates of Obesity Based on Self-Report Versus Direct Measures," *Health Reports*, Statistics Canada, Catalogue no. 82-003-X, 2008, vol. 19, no. 2: 1-16. accessed June 2008; available from http://www.statcan.ca/english/freepub/82-003-XIE/2008002/article/10569-en.pdf.

Shields, Margot, and Michael Tjepkema. "Regional Differences in Obesity," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2006, vol. 17, no. 3: 61-67.

357

_____. "Trends in Adult Obesity," *Health Reports*, Statistics Canada, Catalogue 82-003, 2006, vol. 17, no. 3: 53-59.

Shugart, Ian. *Summary of Discussion, Science Advisory Board Meeting, June 6-7, 2000 - The Precautionary Approach in Health Canada*, Health Canada, 2000; accessed February 2008; available from http://www.hc-sc.gc.ca/sr-sr/pubs/advice-avis/sab-css/meetings-reunions/june-juin00_e.html#12.

Silventoinen, Karri, Patrik K.E. Magnusson, Martin Neovius, Johan Sundstrom, G. David Batty, Per Tynelius, and Finn Rasmussen. "Does Obesity Modify the Effect of Blood Pressure on the Risk of Cardiovascular Disease?: A Population-Based Cohort Study of More Than One Million Swedish Men," *Circulation. Journal of the American Heart Association*, 2008, vol. 118, no. 16: 1637-1642.

Simon, Gregory E., Michael Von Korff, Kathleen Saunders, Diana L. Miglioretti, Paul K. Crane, Gerald van Belle, and Ronald C. Kessler. "Association between Obesity and Psychiatric Disorders in the Us Adult Population," *Archives of General Psychiatry*, 2006, vol. 63: 824-830.

Singh, A. S., C. Mulder, J. W. R. Twisk, W. van Mechelen, and M. J. M. Chinapaw. "Tracking of Childhood Overweight into Adulthood: A Systematic Review of the Literature," *Obesity Reviews*, 2008, vol. 9: 474-488.

Smith, Katherine L. W., Flora I. Matheson, Rahim Moineddin, and Richard H. Glazier. "Gender, Income and Immigration Differences in Depression in Canadian Urban Centres," *Canadian Journal of Public Health*, 2007, vol. 98, no. 2: 149-153.

Statistics Canada. *Health Indicators: Definitions and Data Sources: Body Mass Index (BMI-Canadian Standard)*, Health Canada, Statistics Canada, 2001; accessed June 2008; available from http://www.statcan.ca/english/freepub/82-221-XIE/00401/defin1.htm.

_____. *Probability of Depression, by Age Group and Sex, Household Population Aged 12 and over, Selected Provinces, Territories and Health Regions (June 2003 Boundaries)*, CANSIM Table 105-0205, 2003.

_____. *Canadian Community Health Survey (CCHS) Cycle 2.2 Nutrition Questionnaire*, 2004; accessed September 2008; available from http://www.statcan.ca/english/sdds/instrument/5049_Q1_V1_E.pdf.

_____. *Health Indicators*, 2004; accessed July 2008; available from http://www.statcan.ca/english/freepub/82-221-XIE/00604/whatsnew.htm.

_____. *Arthritis or Rheumatism, by Age Group and Sex, Household Population Aged 12 and over, Canada, Provinces, Territories, Health Regions (June 2005 Boundaries) and Peer Groups, Every 2 Years*, Canadian Community Health Survey, CANSIM Table 105-0402, 2005.

_____. *Canadian Community Health Survey 2004 User Guide for the Public Use Microdata File*, Catalogue no. 82M0024GPE, 2005; accessed February 2009; available from http://www.statcan.gc.ca/imdb-bmdi/document/5049_D8_T9_V1-eng.pdf.

_____. *Canadian Community Health Survey (CCHS) Cycle 3.1, Final Questionnaire*, 2005; accessed September 2008; available from http://www.statcan.gc.ca/imdb-bmdi/instrument/3226_Q1_V3-eng.pdf.

_____. *Diabetes, by Age Group and Sex, Household Population Aged 12 and over, Canada, Provinces, Territories, Health Regions (June 2005 Boundaries) and Peer Groups, Every 2 Years*, Canadian Community Health Survey, CANSIM Table 105-0411, 2005.

_____. *Gross Domenstic Product (GDP) at Basic Prices. Provincial Gross Domestic Product by Industry. Alberta*, CANSIM Table 379-0025, 2005.

_____. *High Blood Pressure, by Age Group and Sex, Household Population Aged 12 and over, Canada, Provinces, Territories, Health Regions (June 2005 Boundaries) and Peer Groups, Every 2 Years*, Canadian Community Health Survey, CANSIM Table 105-0410, 2005.

_____. *Annual Demographic Statistics 2005*, Catalogue no. 91-213-XIB, 2006; accessed November 2008; available from http://www.statcan.ca/english/freepub/91-213-XIB/0000591-213-XIB.pdf.

_____. *Canadian Community Health Survey - Nutrition*, 2006; accessed May 2008; available from http://www.statcan.ca/english/sdds/5049.htm.

_____. *Health Indicators*, 2006; accessed July 2008; available from http://www.statcan.gc.ca/pub/82-221-x/2006001/4198117-eng.pdf.

_____. *Canadian Community Health Survey (CCHS)*, 2007; accessed May 2008; available from http://www.statcan.ca/cgi-bin/imdb/p2SV.pl?Function=getSurvey&SDDS=3226&lang=en&db=IMDB&dbg=f&adm=8&dis=2.

_____. *Deaths, by Selected Grouped Causes and Sex, Canada, Provinces and Territories, Annual*, CANSIM Table 102-0552, 2007.

_____. *Deaths, by Selected Grouped Causes, Age Group and Sex, Canada, Annual*, CANSIM Table 102-0551, 2007.

_____. *Mortality, Summary List of Causes 2004*, Catalogue no. 84F0209XIE, 2007; accessed November 2008; available from http://www.statcan.gc.ca/pub/84f0209x/84f0209x2004000-eng.pdf.

_____. *National Longitudinal Survey of Children and Youth*, 2007; accessed June 2008; available from http://www.statcan.ca/cgi-

bin/imdb/p2SV.pl?Function=getSurvey&SDDS=4450&lang=en&db=imdb&dbg=f&adm=8&dis=2.

_____. *Aboriginal Identity (8), Area of Residence (6), Age Groups (12) and Sex (3) for the Population of Canada, Provinces and Territories, 2006 Census of Population*, Catalogue no. 97-558-XCB2006006, 2008; accessed August 2008; available from http://www12.statcan.ca/english/census06/data/topics/RetrieveProductTable.cfm?Temporal=2006&PID=89121&METH=1&APATH=3&PTYPE=88971&THEME=73&AID=&FREE=0&FOCUS=&VID=0&GC=99&GK=NA&RL=0&d1=0&d2=0&GID=614144.

_____. "Aboriginal Peoples in Canada in 2006: Inuit, Metis and First Nations, 2006 Census." *The Daily*, January 15, 2008, accessed August 2008; available from http://www.statcan.ca/Daily/English/080115/d080115a.htm.

_____. *Aboriginal Population Profile, 2006 Census: Alberta*, 2008; accessed August 2008; available from http://www12.statcan.ca/english/census06/data/profiles/aboriginal/Details/Page.cfm?Lang=E&Geo1=PR&Code1=48&Geo2=PR&Code2=01&Data=Count&SearchText=Alberta&SearchType=Begins&SearchPR=01&B1=All&GeoLevel=&GeoCode=48.

_____. *Body Mass Index (BMI), by Sex, Household Population Aged 18 and over Excluding Pregnant Females, Canada, Provinces and Territories, Occasional, 1994 - 2007*, CANSIM Table 105-4009, 2008.

_____. *Canadian Cancer Statistics 2008*, Canadian Cancer Society, National Cancer Institute of Canada, Statistics Canada, Provincial/Territorial Cancer Registries, Public Health Agency of Canada, 2008; accessed November 2008; available from http://www.cancer.ca/Canada-wide/About%20cancer/Cancer%20statistics/Canadian%20Cancer%20Statistics.aspx?sc_lang=en.

_____. "Deaths." *The Daily*, January 14, 2008, accessed November 2008; available from http://www.statcan.gc.ca/daily-quotidien/080114/dq080114b-eng.htm.

_____. *Health Indicators*, Health Statistics Division, Statistics Canada, Catalogue no. 82-221-X, 2008; accessed June 2008; available from http://www.statcan.ca/english/freepub/82-221-XIE/82-221-XIE2008001.pdf.

_____. *National Population Health Survey (NPHS)*, 2008; accessed July 2008; available from http://www.statcan.ca/cgi-bin/imdb/p2SV.pl?Function=getSurvey&SDDS=3225&lang=en&db=IMDB&dbg=f&adm=8&dis=2.

Steenland, Kyle, and Ben Armstrong. "An Overview of Methods for Calculating the Burden of Disease Due to Specific Risk Factors," *Epidemiology*, 2006, vol. 17, no. 5: 512-519.

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Stevens, June. "Editorial: BMI and Mortality: Sorting through the Data to Find the Public Health Message," *International Journal of Obesity*, 2008, vol. 32: 727-729.

Sturm, Roland, Jeanne S. Ringel, and Tatiana Andreyeva. "Increasing Obesity Rates and Disability Trends," *Health Affairs*, 2004, vol. 23, no. 2: 199-205.

Tang-Peronard, J.L., and B.L. Heitmann. "Stignatization of Obese Children and Adolescents, the Importance of Gender," *Obesity Reviews*, 2008, vol. 9: 522-534.

Tate, R.B., J. Manfreda, and T.E. Cuddy. "The Effect of Age on Risk Factors for Ischemic Heart Disease: The Manitoba Follow-up Study, 1948–1993," *Annals of Epidemiology*, 1998, vol. 8: 415–421.

Tauman, Riva, and David Gozal. "Obesity and Obstructive Sleep Apnea in Children," *Paediatric Respiratory Reviews*, 2006, vol. 7: 247-259.

Tepper, Joshua, William Pollett, Yan Jin, Erik Ellehoj, Don Schopflocher, Brendan Barrett, and Stuart Iglesias. "Utilization Rates for Surgical Procedures in Rural and Urban Canada," *Canadian Journal of Rural Medicine*, 2006, vol. 11, no. 3: 195-203.

Terry, P. D., A. B. Miller, and T. E. Rohan. "Obesity and Colorectal Cancer Risk in Women," *Gut*, 2002, vol. 51, no. 2: 191-194.

The Arthritis Society. *Osteoarthritis*, 2008; accessed October 2008; available from http://www.arthritis.ca/types%20of%20arthritis/osteoarthritis/default.asp?s=1.

Thomas, Steven. *Combining Cycles of the Canadian Community Health Survey*, Statistics Canada, Proceedings of Statistics Canada Symposium 2006: Methodological Issues in Measuring Population Health 2006; accessed July 2008; available from http://www.statcan.ca/english/freepub/11-522-XIE/2006001/article/10411-en.pdf.

Tjepkema, Michael. *Measured Obesity - Adult Obesity in Canada: Measured Height and Weight*, Analytical Studies and Reports, Statistics Canada, Catalogue no. 82-620-MWE2005001, 2005; accessed June 2008; available from http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/aobesity.pdf.

_____. "Adult Obesity," *Health Reports*, Statistic Canada, catalogue no. 82-003, 2006, vol. 17, no. 3: 9-25.

Tremblay, Mark, and Sarah Connor Gorber. "Canadian Health Measures Survey - Brief Overview," *Canadian Journal of Public Health*, 2007, vol. 98, no. 6: 453-456.

Tu, Karen, Zhongliang Chen, and Lorraine L. Lipscombe. "Prevalence and Incidence of Hypertension from 1995 to 2005: A Population-Based Study," *Canadian Medical Association Journal*, Canadian Hypertension Education Program Outcomes Research Taskforce, 2008, vol. 178, no. 11: 1429-1435.

Tu, Shihfen. "Developmental Epidemiology: A Review of Three Key Measures of Effect," *Journal of Clinical Child and Adolescent Psychology*, 2003, vol. 32, no. 2: 187-192.

University of Maryland Medical Center. *Gallstones and Gallbladder Disease*, 2008; accessed October 2008; available from http://www.umm.edu/patiented/articles/what_symptoms_of_gallstones_gallbladder_disease_000 010_2.htm.

Urbach, David R., and Therese A. Stukel. "Rate of Elective Cholecystectomy and the Incidence of Severe Gallstone Disease," *Canadian Medical Association Journal*, 2005, vol. 172, no. 8: 1015-1019.

US. EPA. *America's Children and the Environment: Measures of Contaminants, Body Burdens, and Illness, Second Edition*, cited in Government of Canada, *The Well-Being of Canada's Young Children*, 2003; accessed December 2007; available from http://socialunion.gc.ca/ecd/2003/report2_e/chapter02_e.html.

van den Brandt, Piet A. , Donna Spiegelman, Shiaw-Shyuan Yaun, Hans-Olov Adami, Lawrence Beeson, Aaron R. Folsom, Gary Fraser, R. Alexandra Goldbohm, Saxon Graham, Larry Kushi, James R. Marshall, Anthony B. Miller, Tom Rohan, Stephanie A. Smith-Warner, Frank E. Speizer, Walter C. Willett, Alicja Wolk, and David J. Hunter. "Pooled Analysis of Prospective Cohort Studies on Height, Weight, and Breast Cancer Risk," *American Journal of Epidemiology*, 2000, vol. 152: 514-527.

Verret, Francois. *Methodological Challenges in Analyzing Nutrition Data from the Canadian Community Health Survey - Nutrition*, Statistics Canada, Proceedings of Statistics Canada's Symposium 2006: Methodological Issues in Measuring Population Health, 2006; accessed July 2008; available from http://www.statcan.ca/english/freepub/11-522-XIE/2006001/article/10394-en.pdf.

Vila, Gilbert, Ewa Zipper, Myriam Dabbas, Catherine Bertrand, Jean Jacques Robert, Claude Ricour, and Marie Christine Mouren-Simeoni. "Mental Disorders in Obese Children and Adolescents," *Psychosomatic Medicine*, 2004, vol. 66: 387-394.

Visscher, Tommy L.S., and Jacob C. Seidell. "The Public Health Impact of Obesity," *Annual Review of Public Health*, 2001, vol. 22: 355-375.

von Mutius, E. , J. Schwartz, L. M. Neas, D. Dockery, and S. T. Weiss. "Relation of Body Mass Index to Asthma and Atopy in Children: The National Health and Nutrition Examination Study III," *Thorax*, 2001, vol. 56, no. 835-838.

Vortmann, Michael, and Mark D. Eisner. "BMI and Health Status among Adults with Asthma," *Obesity*, 2008, vol. 16, no. 1: 146-152.

Wang, Guijing, Zhi-Jie Zheng, Gregory Heath, Carol Macera, Mike Pratt, and David Buchner. "Economic Burden of Cardiovascular Disease Associated with Excess Body Weight in U.S. Adults," *American Journal of Preventive Medicine*, 2002, vol. 23, no. 1: 1-6.

Wang, Mengzhe, Manager, Surveillance Analytic Team, Division of Population Health and Information, Alberta Cancer Board, personal communication with Karen Hayward, e-mail, November 24, 2008.

Wang, Y., X. Chen, Y. Song, B. Caballero, and L.J. Cheskin. "Association between Obesity and Kidney Disease: A Systematic Review and Meta-Analysis," *Kidney International*, 2008, vol. 73: 19-33.

Weitoft, Gunilla Ringback, Mats Eliasson, and Mans Rosen. "Underweight, Overweight and Obesity as Risk Factors for Mortality and Hospitalization," *Scandinavian Journal of Public Health*, 2008, vol. 36: 169-176.

Whitlock, Gary, Sarah Lewington, and Cliona Ni Mhurchu. "Coronary Heart Disease and Body Mass Index: A Systematic Review of the Evidence from Larger Prospective Cohort Studies," *Seminars in Vascular Medicine*, 2002, vol. 2: 369–381.

Wilkins, Kathryn. "Incident Arthritis in Relation to Excess Weight," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2004, vol. 15, no. 1: 39-49.

Wilkins, Kathryn, and Margaret de Groh. "Body Mass and Dependency," *Health Reports*, Statistics Canada, Catalogue no. 82-003, 2005, vol. 17, no. 1: 27-39.

Williams, Paul T. "Independent Effects of Cardiorespiratory Fitness, Vigorous Physical Activity, and Body Mass Index on Clinical Gallbladder Disease Risk," *American Journal of Gastroenterology*, 2008, vol. 103: 2239-2247.

Wistuba, Ignacio I., and Adi F.Gazdar. "Gallbladder Cancer: Lessons from a Rare Tumor," *Cancer*, 2004, vol. 4: 695-706.

Wolk, R., and V. K. Somers. "Obesity-Related Cardiovascular Disease: Implications of Obstructive Sleep Apnea," *Diabetes, Obesity and Metabolism*, 2006, vol. 8: 250-260.

World Cancer Research Fund and the American Institute for Cancer Research (WCRF/AICR). *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, 2007; accessed August 2008; available from http://www.dietandcancerreport.org/?p=ER.

_____. *Policy and Action for Cancer Prevention. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective*, Washington, D.C.: AICR, 2009; accessed February 2009; available from http://www.dietandcancerreport.org/downloads/Policy_Report.pdf?JServSessionIdr008=kts074vm65.app46a.

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World Commission on the Ethics of Scientific Knowledge and Technology (COMEST) Expert Group. *The Precautionary Principle*, Paris: UNESCO, 2005; accessed December 2005; available from http://unesdoc.unesco.org/images/0013/001395/139578e.pdf.

World Health Organization (WHO). *Global Burden of Disease Summary Tables*, 2008; accessed December 2008; available from http://www.who.int/healthinfo/global_burden_disease/en/index.html.

_____. *Obesity and Overweight*, 2008; accessed August 2008; available from http://www.who.int/dietphysicalactivity/publications/facts/obesity/en/.

_____. *Preventing Chronic Diseases: A Vital Investment*, 2005; accessed September 2008; available from http://www.who.int/chp/chronic_disease_report/full_report.pdf.

_____. *Reducing Risks, Promoting Healthy Life*, 2002; accessed Dec 2007; available from http://www.who.int/whr/2002/Overview_E.pdf.

_____. *The Impact of Chronic Disease in Canada*, 2005; accessed September 2008; available from http://www.who.int/chp/chronic_disease_report/media/canada.pdf.

Yabroff, K. Robin, William W. Davis, Elizabeth B. Lamont, Angela Fahey, Marie Topor, Martin L. Brown, and Joan L. Warren. "Patient Time Costs Associated with Cancer Care," *Journal of the National Cancer Institute*, 99, 2007, no. 14-23.

Young, T., M. Palta, J. Dempsey, J. Skatrud, S. Weber, and S. Badr. "The Occurrence of Sleep Disordered Breathing among Middle-Aged Adults," *New England Journal of Medicine*, 1993, vol. 328: 1230-1235.

Yusuf, Salim, Steven Hawken, Stephanie Ounpuu, Leonelo Bautista, Maria Grazia Franzosi, Patrick Commerford, Chim C. Lang, Zvonko Rumboldt, Churchill L. Onen, Liu Lisheng, Supachai Tanomsup, Paul Wangai Jr., Fahad Razak, Arya M. Sharma, and Sonia S. Anand. "Obesity and the Risk of Myocardial Infarction in 27,000 Participants from 52 Countries: A Case-Control Study," *Lancet*, 2005, vol. 366: 1640-1649.

Zametkin, Alan J., Christine K. Zoon, Hannah W. Klein, and Suzanne Munson. "Psychiatric Aspects of Child and Adolescent Obesity: A Review of the Past 10 Years," *Child and Adolescent Psychiatry*, 2004, vol. 43, no. 2: 134-150.

Zhang, Jun, and Kai F. Yu. "What's the Relative Risk? A Method of Correcting the Odds Ratio in Cohort Studies of Common Outcomes," *JAMA (Journal of the American Medical Association)*, 1998, vol. 280, no. 19: 1690-1691.