

Obesity and its Relation to Mortality and Morbidity Costs

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Abstract

We reviewed almost 500 research articles on obesity and its relation to mortality and morbidity, focusing primarily on papers published from January 1980 to June 2009. There is substantial evidence that obesity is a worldwide epidemic and that it has a significant negative impact on health, mortality and related costs. Overweight and obesity are associated with increased prevalence of diabetes, cardiovascular disease, hypertension and some cancers. There also is evidence that increased weight is associated with kidney disease, stroke, osteoarthritis and sleep apnea. Moreover, empirical studies report that obesity significantly increases the risk of death.

We used the results to estimate costs due to overweight and obesity in the United States and Canada. We estimate that total annual economic cost of overweight and obesity in the United States and Canada caused by medical costs, excess mortality and disability is approximately \$300 billion in 2009.

JEL Classification: H10, H11, J11, J32

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1. Introduction

Obesity and overweight have been shown to increase the rate of several common adverse medical conditions, resulting in economic costs of \$300 billion per year in the United States and Canada. These costs result from an increased need for medical care and the loss of economic productivity resulting from excess mortality and disability. This paper reviews the literature on overweight and obesity and summarizes the evaluation of economic costs.

Medical conditions with a statistically significant relationship to obesity and overweight include cardiovascular impairments, diabetes, hypertension, cancer, kidney disease, strokes, osteoarthritis and sleep apnea. The causal relationship of most of these conditions to overweight and obesity has been demonstrated by research projects in which individuals were assisted in losing weight and the degree of the conditions was reduced or eliminated (Ross et al., 2000; Anderson et al., 2007).

Determining causation is a complex process that goes far beyond analysis of statistical data (Hitchcock, 2002). A condition such as diabetes may be caused by several concurring conditions, such as age, genetic predisposition and obesity. To measure the economic effects of obesity, we evaluated the cost difference in the absence of obesity. We believe the types of controlled studies reviewed here permit such an evaluation. It is not necessary, for our purpose, to draw an inventory of conditions that bear on the probability of occurrence of morbid conditions.

The fact that the level of obesity in the population may be influenced by education and public policy and that the reduction in obesity, other things being equal, would lead to a decrease in morbidity and mortality, justify the isolation of obesity as one of the causes of morbidity. The papers on which our estimates of excess morbidity and mortality are based – all published in peer-reviewed journals – insist on the adequate control of confounding variables. The evidence they provide in support of a strong statistical association and the elimination of other potential causes points to a causal relationship between obesity and certain diseases. Whether obesity is the primary cause of a disease or simply an aggravating factor, we believe the cost increases and economic effects estimated in this paper would not arise but for obesity in the population.

There are significant issues that affect the quantitative effect of obesity on the conditions noted in this review. For example, some health problems cause a loss of weight, so there are elements of reverse causation in the relationship between low weight and certain health problems. In addition, self-reported body weight is somewhat unreliable and the difference between actual and reported weight is not random, but tends to have an increased negative value as weight increases. There is evidence that some of the papers we reviewed did not make adequate consideration of these issues and, as a result, may have understated the effects of overweight and obesity. The purpose of this review was not to challenge peer-reviewed papers but rather to summarize their findings for potential users of the literature. It is important for users of the research results to consider the possible lack of consideration of issues that may have caused some errors in the results determined.

1.1. Definition of Obesity and Overweight

According to Spence-Jones (2003), body mass index (BMI) is the most commonly used indicator of body fat¹, providing the basis for determining whether someone may be defined as obese, overweight, etc. BMI is the person's weight in kilograms divided by the square of the height in meters. As an example, the Centers For Disease Control and Prevention (CDC) website calculates that a person with weight 68 kilograms and height 1.65 meters has a BMI of $68/(1.65)^2 = 24.98$. The value in terms of English measurement is approximately 703 times the weight in pounds divided by the square of the height in inches. (Some sources use an incorrect multiple of 704.5, which results from deriving a four-digit multiple on the basis of the two-digit approximation: 1kilogram \approx 2.2pound. Multiplying by 700 would be sufficiently accurate for most purposes.)

The definitions of overweight and obesity have become accepted since the 1980s and were based on the 85th and 95th percentiles of the adult population BMI from 1971 to 1974. Excess rates of medical conditions in relation to BMI are not limited to a BMI in excess of 25. Moderate increases in several conditions with relations to obesity are found in people with a BMI in excess of 22.

The definition of BMI itself goes back at least 160 years, when it was defined by Adolphe Quetelet, who provided mathematical evaluations of medical risks to insurance companies in France (Eknoyan, 2008). No significant challenge to this definition has been made since the time of its establishment, but we are not aware of any attempts to test the basic structure of the BMI formula, to see whether it is the most appropriate way to relate body mass to health.

The National Heart Lung and Blood Institute organized an expert panel to develop and publish clinical guidelines for identifying and treating overweight and obesity (Pi-Sunyer, 2000, 1998). The guidelines, and the definition of obesity in terms of BMI, have been widely accepted. The CDC, the World Health Organization and the great majority of researchers use these BMI-based definitions, as shown in Table 1.

In this review, we will use these definitions of weight-status categories. If a cited paper uses an alternative definition, we will note the difference; otherwise, we use the accepted definitions. We have found that more recent research in Asia often uses different definitions, which some researchers claim are more appropriate for Asian populations. However, Gu et al. (2006) conclude from a large study of adults in China from 1991 to 2000 that their findings are consistent with studies of Western populations and that a single standard for overweight and obesity is justified.

Although BMI is the most commonly used measure of body fat, perhaps because of the influence of the clinical guideline published in 1998, it may not be the best measure. Burkhauser and Cawley (2008) claim there is "wide agreement in the medical literature" that BMI is seriously flawed because it does not distinguish fat from fat-free body mass such as muscle and bone. They advocate strongly for researchers to use more accurate measures such as bioelectrical impedance analysis, fat-free mass, total body fat and percentage body fat. The differences they provide are dramatic. For example, using the National Health and Nutrition Examination Survey (NHANES) III data, obesity preva-

¹The Centers for Disease Control and Prevention has an easy-to-follow discussion of BMI on its website: <http://www.cdc.gov/healthyweight/assessing/bmi/>

Table 1
Centers for Disease Control and Prevention BMI Categories

BMI	Weight Status
Below 18.5	Underweight
18.5–24.9	Normal
25.0–29.9	Overweight
30.0 and above	Obese
Above 40.0	Extremely obese

Definitions apply to all adult men and women. Definitions for children vary by sex and age. The World Health Organization uses the same definitions. *Source:* <http://www.cdc.gov/healthyweight/assessing/bmi/>

lence using BMI is 23.7 percent for men and 19.0 percent for women. Using percentage body fat, the prevalence is 70.4 percent for men and 43.4 percent for women. Romero-Corral et al. (2008) find similar results in their study of BMI versus body fat percentage for a cross-sectional study of 13,601 U.S. adults. According to Romero-Corral et al., using BMI as a measure dramatically understates the magnitude of the obesity epidemic. They discuss alternatives to BMI, including biometrical impedance, hydrostatic weighing, dual-energy X-ray absorptiometry and air displacement plethysmography. They recommend using bioelectrical impedance for measuring body fatness because it is more accurate than BMI, it is easy to obtain, it does not use radiation and it is relatively low cost.

Janssen (2007) shows BMI is less useful for diagnosing obesity for older adults, over 65, than in young and middle-age adults. On the other hand, Ryan et al. (2008) find that metabolic syndrome and cardiovascular disease risk factor status did not vary substantially when subjects were categorized by waist circumference or BMI.

Waist circumference and waist-to-hip ratio are other indicators of body fatness. Waist circumference is simply the person's waist circumference in centimeters. Yusuf et al. (2005) have shown that waist circumference and waist-to-hip ratio offer better estimates of myocardial infarction attributable to obesity than BMI, even though BMI is much more widely used as a measure of weight status. In a large cohort study of Chinese women, Zhang et al. (2007) found a positive monotonic dose-response relationship between waist-hip ratio and the risk of death. This positive association was independent of BMI and socio-demographic and lifestyle factors.

Although waist circumference may indeed be a better measure with respect to heart disease, Cumming and Pinkham (2008) found that "BMI and waist circumference are essentially equivalent in their ability to predict mortality risk in a male insurance population." A majority of the papers we found use BMI rather than body fat percentage, waist circumference or waist-to-hip ratio.

Roudebush et al. (2006) studied 241,966 life insurance policies, using BMI calculated at the time of issue and standardized mortality ratios. They found a U-shaped relation-

ship. The ratio of mortality to the standard table rates increases as BMI increases from the normal level.

In a 2004 survey, Mokdad et al. (2004) estimate that “roughly 400,000 deaths now occur annually due to poor diet and physical inactivity. The gap between deaths due to poor diet and physical inactivity and those due to smoking has narrowed substantially.” Their article generated a series of corrections and comments (Barnoya and Glantz, 2004; Blair et al., 2004; Anstadt, 2004; Gandjour, 2004; McGinnis and Foege, 2004; Flegal et al., 2005), without altering the main conclusion of Mokdad et al.: Poor diet and physical inactivity lead to more deaths than every other cause except tobacco. In discussing the Mokdad et al. paper in a *Journal of the American Medical Association* editorial McGinnis and Foege (2004) conclude that deaths due to poor diet and inactivity “are in fact likely greater contributors to mortality than tobacco”. Flegal et al. obtain lower estimates perhaps because of differences in statistical methods. They estimated that 111,909 excess deaths were associated with obesity, very much lower than the the earlier studies. They suggest that “the possibility that improvements in medical care, particularly for cardiovascular disease, the leading cause of death among the obese, and its risk factors may have led to a decreased association of obesity with total mortality” (Flegal et al., 2005).

Not all of the studies we reviewed reported negative effects for obesity. In fact, most research suggests that, while severe obesity and underweight significantly increase all-cause mortality, overweight does not appear to be a similar risk factor. For example, Arndt et al. (2007) held that among nonsmoker, heavily working men, normal and overweight men experienced similar mortality. Similarly, Kaestner and Grossman (2008) reported that data from the U.S. National Longitudinal Survey of Youth indicated that overweight and obese children had about the same academic achievement scores as those of normal weight children. However, it has been hypothesized that excess weight is a risk factor for delayed recovery from neck pain, such as from whiplash injuries. Yet in a recent study of 4,395 persons with whiplash injuries, Yang et al. (2007) found no evidence that the overweight or obese have longer recovery rates.

Puhl and Heuer (2009) described the high degree of prejudice and discrimination that obese individuals suffer in employment, education and health care. The studies they reviewed showed that obese patients were less likely to get screenings for breast, cervical and colorectal cancer, among other examples. The stigma of obesity evidently compounds its effect on health status and mortality. Mitchell et al. (2008) found that, based on data from the Canadian Community Health Survey 2003, overweight and obesity were associated with a markedly lower use of cervical cancer screening.

Obesity has a variety of significant effects on the body. For example, excess weight puts additional pressure on tissue in joints, such as the knees, causing increased risk of arthritis and damage to tendons and ligaments. It also inhibits healing of joints damaged by accidents or surgery. Higher weight increases the effort of movement, causing stress on the heart and muscles. An increased volume of tissue in the torso and abdomen can inhibit the normal function of organs. For example, excess abdominal tissue can cause esophageal reflux and other digestive problems, which excess consumption of food may exacerbate.

Alternatively, obesity may result from reduced physical activity, and certain excess

morbidity associated with obesity, such as cardiovascular problems, may be caused by the lack of exercise. Fatty tissue affects the chemical balance of the body, interacting with hormones such as estrogen and insulin and thus altering their effect on the body (Rosin, 2007). While this paper does not investigate the reasons for the relationships between obesity and morbidity, the direct effects of obesity show the statistical relationships between obesity and morbidity may generally be assumed to indicate the presence of causal relationships.

There is further support for the causal relationship between obesity and morbidity in studies that have followed groups of obese people whose controlled diets reduced their degree of overweight, thereby significantly diminishing their health problems (Anderson et al., 2007).

1.2. Effect of BMI on Medical Conditions

Research on the effect of excess BMI on various medical conditions tends to relate BMI to the increase in rate of the conditions by using either an odds ratio or relative risk in relation to the level of BMI. Some measurements of excess risk are evaluated in terms of hazard ratio. Relative risk, odds ratio and hazard ratio each measure the increase in risk of the particular condition but in a somewhat different manner. The specifics of these measurements are explained in Appendix 1. We have used relative risk for our analyses, and have converted odds ratio and hazard ratio to relative risk when using results from papers that evaluated the effects in terms of odds ratio or hazard ratio. The choice of measurement basis has no effect on the final results, but relative risk makes the intermediate calculations less complicated.

The various studies of medical conditions are not consistent in terms of the level of BMI included in the measurement of excess risk. For example, some studies compute risks based on overweight separately from obesity, while some combine them. Others use levels of BMI that do not match the specific levels that currently define overweight and obesity. To combine the results of these various studies, it is necessary to apply the proportion of the population with various levels of BMI. We have based our evaluation of the effects of various levels of BMI on the population distribution obtained from the NHANES 2007-08 survey, which identified the BMI of over 6,000 individuals in a random sample of the population.

The shape of the abdomen is related to BMI, but the degree of abdominal adiposity, while strongly positively correlated to BMI, can have independent effects in people of the same BMI. Abdominal adiposity has been shown to have independent negative health effects (Zhang et al., 2008). The fact that abdominal adiposity is strongly related to BMI, as well as the fact that BMI has significant effects, whether or not abdominal adiposity is considered, as well as the fact that most of the published studies are based on BMI without considering abdominal adiposity separately, has caused us to base the results of this literature review on BMI, without attempting to separate the effects of abdominal adiposity.

2. Prevalence of Obesity

The proportion of the United States and Canada population over age 20 in the standard BMI groups is shown in Table 2. We used the specific distribution of BMI for the U.S.

Table 2
Population Distribution of BMI, Percentage of Adult Population by BMI Level

BMI	United States	Canada	Combined
< 18.5	1.9	2.1	1.9
18.5–25	33.0	34.0	33.1
25–30	34.3	34.2	34.3
30–35	19.2	17.4	19.0
35–40	7.4	7.6	7.4
≥ 40	4.2	4.7	4.3
Total	100.0	100.0	100.0

Source: Tjepkema (2006, Table 3); NCHS (2009); CDC (2008)

population for our calculations because the total percentages for the U.S. were very close to the percentages for the U.S. and Canada, and the U.S. results were available at a detailed level that allowed us to determine population percentages and average BMI for each relevant portion of the population.

2.1. More Results on Prevalence of Overweight and Related Disease

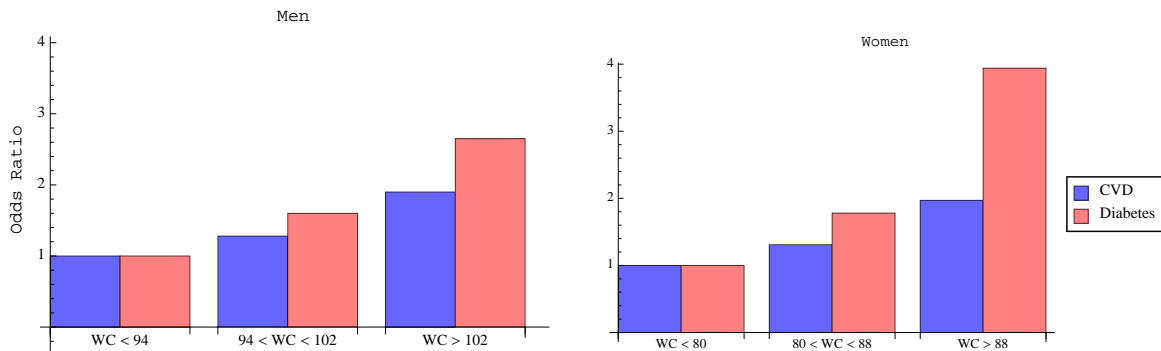
Balkau et al. (2007) reported on their study of data collected in 63 countries during 2005 through primary care physicians. Participating physicians recruited patients, aged 18 to 80, on two pre-specified half-days for a total of 69,409 men and 98,750 women. For each participating patient, the physician collected BMI and waist circumference, and recorded presence (or not) of cardiovascular disease (CVD) and diabetes.

The study is interesting in several respects. First, it covers Northwest Europe, South Europe, East Europe, North Africa, South Africa, Middle East, East Asia, South Asia, Australia, Canada and Latin America (but not the U.S.). A majority of adults (64 percent of men and 57 percent of women) in the study were either overweight or obese. Moreover, in all regions except South and East Asia, a majority of adults were overweight or obese.

Second, the study found a statistically significant graded increase in the frequency of cardiovascular disease and diabetes mellitus with both BMI and waist circumference. We illustrate some of their results in Figure 1.

We note that this study selected only members of the population who had access to and a willingness to visit their primary-care physician. Since this is not a population study, the results cannot apply to the entire population. On the other hand, this study

Figure 1
Odds Ratios for Increased Risk of CVD and Diabetes



The graphs show the odds ratios for increased risk of cardiovascular disease (CVD) and diabetes for all 69,409 men and 98,750 women based on the waist circumference in centimeters. The average waist circumference for men was 95.8 centimeters with a standard deviation of 14.0 centimeters. For women, the average waist circumference was 88.7 centimeters with a standard deviation of 14.9 centimeters. *Source:* Balkau et al. (2007, Table 2)

supports the idea that obesity has become a worldwide epidemic and that increased BMI and waist circumference are associated with significantly higher odds ratios for cardiovascular disease and diabetes mellitus. We also note that this is measured BMI and waist circumference while the national surveys in the U.S. and Canada have included both measured BMI and the participants' reported values. In a Dutch study, Visscher et al. (2006) found significant underreporting of BMI in adults and under-estimation of obesity prevalence in the period between 1998 and 2001.

Stamatakis et al. (2005) studied economic factors related to obesity trends among children in England from 1974 to 2003. They determined that obesity prevalence had been accelerating and that it varied by socioeconomic class. Children from lower socioeconomic classes had an odds ratio of 1.14 for obesity, compared to an odds ratio of 0.74 for children from higher socioeconomic classes.

2.2. Obesity in the United States

In 2001, the U.S. Surgeon General's Call To Action to Prevent and Decrease Overweight and Obesity declared, "The United States is experiencing substantial increases in overweight and obesity that cut across all ages, racial and ethnic groups, and both genders" (Satcher, 2001). Call to Action's description of the trend in overweight and obesity for adults and children is based on NHANES for 1976-80, 1988-94 and 1999. Ogden et al. updated the statistics based on NHANES for 2003-04 and added NHANES 1971-74 (Ogden et al., 2007b). Table 3 is from their article.

The CDC website has a table showing obesity prevalence and costs attributable to obesity by state (CDC, 2008). It also shows aggregate medical spending attributable to overweight and obesity in 1998. The prevalence data is updated in (NCHS, 2009).

Table 3
**Trends of Percentages of Overweight and Obese Adults, Ages 20–74 in the
 United States for 1960–2004**

Sex	Survey Period	Overweight BMI \geq 25	Obese BMI \geq 30	Extremely obese BMI \geq 40
Men and women	1960–1962	44.8	13.3	0.9
	1971–1974	47.2	14.5	1.3
	1976–1980	47.1	15.0	1.4
	1988–1994	55.8	23.2	3.0
	1999–2000	64.5	30.9	5.0
	2001–2002	65.7	31.3	5.4
	2003–2004	66.3	32.9	5.1
Men	1960–1962	49.4	10.7	0.3
	1971–1974	53.8	12.1	0.6
	1976–1980	52.6	12.7	0.4
	1988–1994	60.8	20.5	1.8
	1999–2000	66.9	27.7	3.3
	2001–2002	69.9	28.4	3.9
	2003–2004	71.1	31.7	3.0
Women	1960–1962	40.5	15.8	1.4
	1971–1974	40.9	16.6	2.0
	1976–1980	41.9	17.0	2.2
	1988–1994	51.0	25.9	4.1
	1999–2000	62.0	34.0	6.6
	2001–2002	61.4	34.1	6.8
	2003–2004	61.4	34.0	7.3

The trends are significantly increasing overall and for men and women separately. The source document includes standard errors of the estimates. *Source:* Ogden et al. (2007b, Table 1)

While the percentage overweight, obese and extremely obese exhibited increases in each study, the greatest increases for men occurred in the three studies beginning in the period 1976–80 and ending with 1999–00. The situation for women was similar, although the percentages were a little lower. A recent study makes projections from the same data (Ruhm, 2007). By 2020, according to Ruhm’s analysis, 77.6 percent of men will be overweight and of those overweight 40.2 percent will be obese. For women, the percentages are 71.1 percent and 43.3 percent, respectively. In a subsequent study, Ogden et al. (2007a) found that there was no significant change in obesity prevalence between 2003–04 and 2005–06.

Haas et al. (2003) studied the distribution of BMI of children in the U.S. as it relates to race, socioeconomic status and health insurance status. They found significant racial disparities in the prevalence of overweight among different ethnic groups, and they documented a significant association of health insurance status with the prevalence of adolescent overweight.

Cumming and Pinkham (2008) studied BMI and waist circumference for a large sample of life-insured males and their relation to mortality. The percentage of lives with BMI ≥ 30 was 19.0 percent. This is lower than the prevalence in population studies (31.7 percent in NHANES 2003–04, see Table 3). However, this is to be expected because insured lives are selected after underwriting, and extremely obese candidates are typically not accepted (nor are candidates with very low BMI). The difference also is consistent with population studies, in that candidates for life insurance typically have higher income than average. Population studies have found lower obese prevalence with higher income groups.

2.3. Prevalence of Obesity in Canada

Our review uses four national health surveys to describe the trend prevalence of obesity in Canada (Torrance et al., 2002; Raine, 2005; Tjepkema, 2005, 2006). Some of the results are summarized in Table 4.

Torrance et al. (2002) found that lower education is associated with higher BMI. However, lower education is underreported, so the estimate could be biased. Smoking cessation was not a major factor in rising rates of overweight and obesity since BMI increased in smokers as well as never-smokers and former smokers. The trend in prevalence of overweight and obesity is shown in Table 4.

Raine (2005) found that obesity varies regionally. Atlantic Canada has the highest prevalence of obesity. In Western Canada, rural men and women were significantly more likely to be obese than urban men and women. In the 2000–01 survey, northern Ontario, the Atlantic provinces, the Prairie provinces, Nunavut and the Northwest Territories had significantly higher obesity rates than the national average of 15 percent.

Tjepkema (2006) found that 23.1 percent of Canadians were obese in 2004 (compared to 29.7 percent in the U.S. based on 1999–2002 NHANES) and another 36.1 percent are overweight. This is a large increase over the 1978–79 Canada Health Survey estimate of the prevalence of obesity as 13.8 percent. This study also estimates the distribution of BMI across the population. In addition, the author reports the relationships between obesity and physical activity. As one would expect, those reporting a sedentary lifestyle

had a higher rate of obesity. The study also found that, generally, obesity varies inversely with educational attainment.

Table 4
Percentage of Canadian Adults with BMI \geq 25

	Men	Women
Nutrition Canada Survey 1970–1972	47.0	33.9
Canada Health Survey 1978–1979	55.6	42.3
Canada Heart Health Survey 1986–1992	58.1	40.6
Canadian Community Health Survey 2004	65.0	53.4

Trends in prevalence of overweight and obesity among Canadian adults.

Source: Torrance et al. (2002, Figure 2); Tjepkema (2006, Table 3)

3. Obesity and Cardiovascular Disease

Calle et al. (1999) use data from the Cancer Prevention Study II to study the relation of BMI to all-cause mortality, cardiovascular disease and cancer. This is a large prospective 14-year cohort study of more than 1 million adults in the U.S. We discuss this again in Section 8 where we take up BMI and all-cause mortality. Calle et al. found a curvilinear relation between BMI and the relative risk of death from cardiovascular disease. Relative to normal weight, the relative risk of death due to cardiovascular disease increases for overweight and obese men and women. However, it is also higher for BMI below normal. According to the authors, this is explained by increased risk of death among lean men and women as a result of cerebrovascular disease, pneumonia and diseases of the central nervous system.

Oster et al. (2000) estimate health care costs attributable to obesity for a hypothetical U.S. managed care health plan. They use survey data from 3,400 adults collected in 1996, as well as prevalence and relative risk data from published sources such as NHANES III. For each of eight conditions, they estimated the annual medical costs associated with obesity as

$$\frac{P(RR - 1)}{1 + P(RR - 1)} \times N \times C$$

where P is the prevalence of obesity in the population, RR is the relative risk for the condition, N is the number of cases of the condition in the population, and C is the estimated annual cost of the condition per case. Table 5 shows the relative risks they used; the costs are shown in the section on costs. The relative risk for coronary heart disease is 2.4 for men and 3.0 for women. This study uses 29.0 as the BMI level for obesity, rather than the standard level 30.0, but that would not matter very much as it uses the standard definition for the normal BMI range.

In a large study, Bassuk and Manson (2008) reviewed the epidemiologic data on risk factors for cardiovascular disease and diabetes. Cardiovascular disease is the leading cause of death for American men and women, accounting for nearly half of all deaths,

Table 5
Relative Risk by BMI

Obesity-Related Disease	BMI < 25	25 ≤ BMI < 29	BMI ≥ 29
Men			
Hypertension	1.0	1.9	2.6
Hypercholesterolemia	1.0	–	1.8
Diabetes	1.0	2.6	10.4
Coronary heart disease	1.0	1.4	2.4
Stroke	1.0	1.2	1.3
Gallbladder disease	1.0	2.0	3.5
Osteoarthritis of the knee	1.0	1.0	2.8
Women			
Hypertension	1.0	2.3	3.8
Hypercholesterolemia	1.0	–	1.8
Diabetes	1.0	4.4	48.9
Coronary heart disease	1.0	1.7	3.0
Stroke	1.0	1.4	1.9
Gallbladder disease	1.0	2.0	3.5
Osteoarthritis of the knee	1.0	1.7	1.6
Endometrial cancer	1.0	1.0	2.0

Relative risks of selected obesity-related diseases, by sex, disease and BMI. *Source:* Oster et al. (2000, Table 1)

and diabetes is a major risk factor for cardiovascular disease. This paper cites data from the Nurse's Health Study. The authors suggest that 74 percent of cardiovascular disease cases among women could be prevented by not smoking, engaging in regular physical activity, eating healthier food and drinking alcohol in moderation. Galal et al. (2007) conducted a study on the relationship between BMI and mortality in patients with known or suspected coronary artery disease, and found that BMI was inversely related to long-term mortality in the patients. This result is different from most of the results in the research documents. The incidence of long-term mortality in underweight, normal, overweight and obese was reported to be 39 percent, 35 percent, 24 percent and 20 percent, respectively. They state that, compared with a normal BMI, the hazard rate for mortality in underweight patients was 2.4 (95 percent confidence interval 1.7 to 3.7), and that overweight and obese patients had a significantly lower mortality than the normal BMI (hazard rate 0.65, 95 percent confidence interval 0.6 to 0.7 for overweight; hazard rate 0.61, 95 percent confidence interval 0.5 to 0.7 for obese patients).

Yan et al. (2006) studied 17,643 workers who were recruited at ages 31 to 64 from 1967 to 1973 to assess the relation of mid-life BMI to morbidity and mortality at older ages. They found that among individuals with no cardiovascular risk factors, those who were obese in middle age have a significantly higher risk of death from cardiovascular disease than those who had normal weight.

Yusuf et al. (2005) studied obesity and myocardial infarction in 27,000 participants in 52 countries. They were trying to determine if the alternative obesity markers, waist-to-hip ratio, waist circumference and hip circumference, would be stronger indicators of myocardial infarction than BMI. They found that while BMI showed a modest association with myocardial infarction, it became insignificant after adjustment for other risk factors. On the other hand, the alternative markers were closely associated with myocardial infarction even after adjustment. They conclude that using waist-to-hip ratio rather than BMI increases the estimate of myocardial infarction attributable to obesity.

Eisenstein et al. (2002) studied the effect of BMI on acute coronary syndrome and patients' long-term clinical and economic outcomes. They found that increased BMI was associated with younger age multi-morbidity, and less severe coronary artery disease. It was also associated with more clinical events, higher cumulative inpatient medical costs, and significant differences in unadjusted survival at 10 years. However, it was not associated with differences in 10-year survival, after adjusting for baseline characteristic differences.

Abdulla et al. (2008) investigated the impact of obesity on disease-specific mortality in high-risk patients with myocardial infarction or chronic heart failure. They found a U-shaped association between BMI and mortality with highest mortality in underweight and extremely obese groups (BMI < 18.5, BMI > 35 respectively) among patients with myocardial infarction but not heart failure. They state that compared with normal weight, they found the following:

- Underweight patients were in increased death risk regardless of myocardial infarction or heart failure, with myocardial infarction hazard rate of 1.54 with 95 percent confidence interval 1.35 to 1.75 and heart failure hazard rate of 1.37 with 95 percent confidence interval 1.18 to 1.59.

- Obese patients were not associated with increased risk of death in myocardial infarction or heart failure.
- Overweight was associated with decreased death risk in myocardial infarction but not heart failure. The hazard rate for myocardial infarction was 0.91 with 95 percent confidence interval 0.87 to 0.96. The hazard rate for heart failure was 1.04 with 95 percent confidence interval 0.97 to 1.12.
- Extreme obesity was associated with increased risk of death in patients with myocardial infarction (hazard rate 1.23 with 95 percent confidence interval 1.06 to 1.44) and heart failure (hazard rate 1.13 with 95 percent confidence interval 0.95 to 1.36).

Rogers et al. (2003) studied the effect of obesity on all-cause mortality, circulatory disease and diabetes as well as the change in BMI distribution in the U.S. from 1987 to 1997. Between 1987 and 1997, the population got heavier; the graph of the distribution shifted to the right and indicates a higher standard deviation. Based on an analysis of hazard ratios of death due to diabetes, circulatory disease and all-cause mortality, they conclude that obesity significantly contributes to increased mortality for all-cause, circulatory-specific and diabetes-specific mortality, even after accounting for socioeconomic and geographic differences. They comment that as the population becomes more obese, the relationship between obesity and risk factors may change. For example, the relationship between obesity and income levels and obesity and education levels may change as more people become obese. Rogers et al. also discuss the U-shape relationship between BMI and hazard ratios. Individuals in the overweight class have slightly less risk of all-cause death than individuals of normal weight, but nevertheless have slightly higher risk of death for circulatory disease and much higher risk of death for diabetes. Moreover, higher than normal mortality of underweight individuals could be an artifact of cigarette smoking or underlying chronic conditions that cause weight loss, such as cancer, infectious diseases or heart disease.

Flegal et al. (2007b) studied the cause-specific relative risks of mortality using the NHANES I, II and III. Here is a summary of their conclusions:

- Underweight was associated with significantly increased mortality from non-cancer, noncardiovascular disease causes (23,455 excess deaths; 95 percent confidence interval 11,848 to 35,061), but not associated with cancer or cardiovascular disease mortality.
- Overweight was associated with significantly decreased mortality from non-cancer, noncardiovascular disease causes (69,299 excess deaths; 95 percent confidence interval 100,702 to 37,897), but not associated with cancer or cardiovascular disease mortality.
- Obesity was associated with significantly increased cardiovascular disease mortality (112,159 excess deaths; 95 percent confidence interval, 87,842 to 136,476), but not with either cancer mortality or with noncancer, noncardiovascular disease mortality.

Another study using NHANES data, conducted by Flegal et al. (2007a), indicated that smoking or pre-existing illness had little effect on estimates of relative risk, and that the relative risk for BMI categories did not show large or systematic changes after simultaneous exclusion of persons who had ever smoked, persons with a history of cancer or cardiovascular disease, and persons who died early in the follow-up period or had their heights and weights measured at older ages.

Gregg et al. (2005) use the five NHANES from 1960 to 2000 to document the substantial decline in the prevalence of key cardiovascular disease risk factors over the past three to four decades, affecting obese, overweight and lean segments of the population. The prevalence of high cholesterol, high blood pressure and smoking levels decreased for all BMI categories, while the prevalence of diabetes increased for the overweight and obese groups. In 2005, the prevalence of high cholesterol, high blood pressure and smoking for obese people were 21, 18 and 12 percentage points lower, respectively, than 30 to 40 years earlier.

Bogers et al. (2007) conducted a meta-analysis of 21 cohort studies including more than 300,000 lives. They determined relative risks for cardiovascular disease of 1.17 and 1.49 for overweight and obese persons relative to normal weight persons, with adjustment for blood pressure and cholesterol levels.

Weitofte et al. (2008) showed an association between overweight and increased risks for circulatory diseases and musculoskeletal disease, where the population attributable risks were large (13 percent for men and 8.1 percent for women for circulatory disease, and 12.7 percent for men and 12.9 percent for women for musculoskeletal disease).

Silva et al. studied adult obesity and survival with and without cardiovascular disease using the Framingham Heart Study data (Silva et al., 2006). They found that 45-year-old obese men with no cardiovascular disease live six years fewer than normal weight counterparts. For women, the figure was 8.4 years. On the other hand, obese men and women with cardiovascular disease lived longer than normal weight men and women with cardiovascular disease, 2.7 and 1.4 years, respectively.

Bibbins-Domingo et al. (2007) estimated the impact of U.S. adolescent overweight in 2000 on the incidence of coronary heart disease in those attaining age 35 in 2020. They estimated that 30 to 37 percent of 35-year-old men will be obese and 34 to 44 percent of 35-year-old women will be obese. The higher prevalence of obesity would be expected to increase the overall prevalence of coronary heart disease by 2 percent in 2020 and by 11 percent in 2035. Capewell and Critchley (2008) suggested that Bibbins-Domingo et al. underestimated the impact on coronary heart disease mortality, citing their own work as well as Bogers et al. (2007).

Falkner et al. (2006) studied BMI and blood pressure of U.S. children age 2 to 19; they concluded that overweight begins at a very young age and that the blood pressure gradient is associated with overweight throughout childhood. They urge adoption of strategies for prevention of childhood obesity.

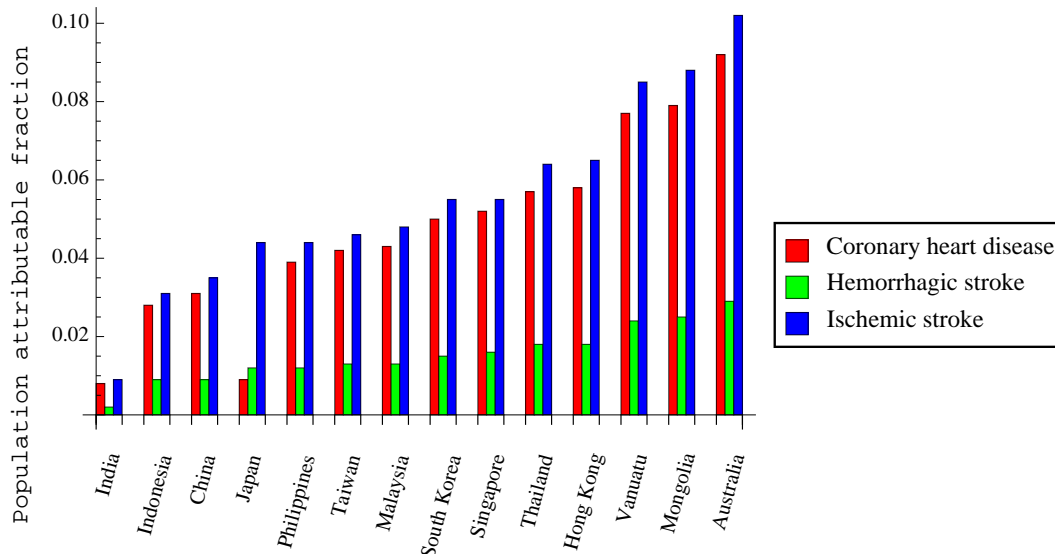
Abell et al. (2008) analyzed cardiovascular disease mortality risk associated with obesity using meta-analysis of data from the Black Pooling Project for Black and White individuals, which is based on four studies including NHANES I and II. They find that the association between obesity and cardiovascular disease mortality is significantly greater in white individuals. Nevertheless, the obesity should be considered a significant

risk factor in both black and white people. A recent large prospective study of patients with a certain type of myocardial infarction found that the obese and very obese had higher survivor rates.

Buettner et al. (2007) investigated the impact of obesity on mortality in unstable angina/non-ST-segment elevation myocardial infarction (UA/NSTEMI). Their conclusion was that obesity was associated with improved outcome after UA/NSTEMI treated with early revascularization. They reported that cumulative three-year mortality rates were 9.9 percent for normal BMI, 7.7 percent for overweight, 3.6 percent for obese, and 0 (no deaths) for very obese. Obese and very obese patients had less than half the long-term mortality when compared with normal BMI patients. The hazard ratio was 0.38, with 95 percent confidence interval 0.18 to 0.81.

In a large longitudinal study in Taiwan, Fu et al. (2008) found that the impact of higher BMI on costs rose for six metabolic syndrome diseases, including hypertension. In their study of middle-aged Korean women (ages 40 to 64), Song et al. (2007) reported a positive linear relationship between BMI and coronary heart disease in women at premenopausal status and a U-shaped relationship in women at postmenopausal status. A meta-analysis-based study of 14 countries in the Asia Pacific region found increasing prevalence of overweight and obesity. It also provides estimates of population attributable fractions for fatal coronary heart disease and stroke associated with the overweight and obesity classes (Lee et al., 2007). Some of this study's results are illustrated in Figure 2.

Figure 2
Distribution of CHD and Stroke in the Asia Pacific



Population attributable fractions of fatal coronary heart disease and stroke associated with overweight and obesity. *Source:* Lee et al. (2007, Figure 2)

Zhou et al. (2008) conducted one of the largest prospective studies of the relationship between BMI and mortality from stroke, involving 212,000 relatively lean Chinese men.

BMI was strongly related to blood pressure throughout the range of BMI levels. While blood pressure was strongly related to stroke mortality, BMI was strongly associated with stroke mortality only in the range BMI above 25. Most men in the study had BMI below 25 and among them BMI had little relation to risk of stroke mortality. For BMI below 25, an inverse association between BMI and some other stroke risk factors counterbalance the effects of BMI on blood pressure.

Goméz-Ambrosi et al. (2008) studied the relation between BMI and acute-phase protein serum amyloid A circulating in the blood of children and adolescents. They found significant positive correlation between serum amyloid A and body fat. Increased serum amyloid A levels may provide a direct link between obesity and comorbidities such as diabetes and cardiovascular disease.

4. Obesity and Diabetes

According to a recent editorial in *The Lancet*, at least 246 million adults worldwide have diabetes mellitus, and the figure is escalating to epidemic proportions (Lancet, 2008). Moreover, the “inexorable rise of diabetes parallels that of the obesity pandemic spreading throughout both industrialized and developing countries.” In an editorial in *Diabetes*, Runge wrote that 60 percent of diabetes² is directly attributable to weight gain (Runge, 2007).

Ford et al. (1997) use a cohort of 14,407 people who were 25 or older at the NHANES I baseline examination between 1971 and 1975, followed through 1992. They found weight gain over a 10-year period was strongly associated with increased risk for diabetes. Table 6 shows, in the last column, that adjusted hazard rates for diabetes increase steadily with increased weight gain during the study period.

Detournay et al. (2000) used a sample of 14,670 from the adult population of France, age 18 and older. The results show that, with the reference to BMI at the normal level, obese subjects had a relative risk of 3.8 for diabetes. Moreover, the paper estimated the direct cost attributable to obesity to be in the range of 4.2 billion to 8.7 billion 1992 French francs, which accounted for about 0.7 percent to 1.5 percent of total health expenditures.

Oster et al. (2000) used relative risk for diabetes of 10.4 for men and 48.9 for women. They estimated that for a hypothetical U.S. managed care health plan, 86 percent of diabetes related costs are associated with obesity.

Bloomgarden (2008) surveyed recent literature on BMI and diabetes. We reviewed the main sources for this survey. Colditz et al. (1995) used data from a cohort study of 114,281 female registered nurses, starting in 1976 with follow-up to 1990. They found, after adjustment for age, that BMI was the dominant predictor of risk for diabetes mellitus. The risk increased with increasing BMI even for normal weight women. The results are independent of family history.

Based on data retrieved from the Bureau of National Health Insurance of Taiwan, Fu et al. (2008) examined the costs of diseases and outpatient and inpatient services

²There are several forms of diabetes. We use diabetes to mean type-2 diabetes. This is the form associated with overweight and inactivity.

Table 6
Age-Adjusted Hazard Rate as a Function of Weight Change

Weight change (kg)	No. of cases of diabetes	Person-years of follow-up	Hazard rate, adjusted for age
Entire sample			
Loss \geq 11	24	1,909	1.99
5 \leq Loss $<$ 11	49	5,475	1.66
-5 \leq Loss $<$ 5	218	39,420	1.00
5 \leq Gain $<$ 8	81	10,607	1.90
8 \leq Gain $<$ 11	31	6,123	1.09
11 \leq Gain $<$ 20	60	6,139	2.85
Gain \geq 20	24	1,239	4.60
30- to 50-year-old participants			
Loss \geq 11	11	769	2.05
5 \leq Loss $<$ 11	21	2,486	1.52
-5 \leq Loss $<$ 5	110	22,504	1.00
5 \leq Gain $<$ 8	60	6,756	2.14
8 \leq Gain $<$ 11	24	4,101	1.23
11 \leq Gain $<$ 20	49	4,251	3.13
Gain \geq 20	21	538	5.28

Ford et al. concluded that weight gain over the 10-year period was strongly associated with increased risk for diabetes. Data from the National Health and Nutrition Examination Survey Epidemiological Follow-up Study, 1971–92. *Source:* Ford et al. (1997, Table 2)

caused by obesity. Their sample consisted of 4,318 subjects from 2000–02. However, they defined BMI categories differently from the existing literature. For Asians, $24 \leq \text{BMI} < 27$ is overweight, $27 \leq \text{BMI} < 30$ is first-degree obesity, and $\text{BMI} \geq 30$ is second-degree obesity. The study found that the relative risk for diabetes increased as BMI increased. With diabetes, the relative risk of being overweight is 1.49, but 1.87 (2.35) in the first- (second-) degree obesity category. Furthermore, the population-attributable risks for diabetes were 7.0%, 11.9% and 17.4% for overweight and first- and second-degree obesity, respectively. The results also show that 0.42% of Taiwan's annual medical costs were attributable to diabetes mellitus caused by overweight and first- and second-degree obesity.

Janssen (2007) studied data retrieved from the Cardiovascular Health Study, one sample of 4,968 American males and females age 65 and older, from 1989–99. He organized his subjects into three classes: normal, overweight and obese, based on the CDC definitions, with the exception of “normal.” Normal weight was defined as $20 \leq \text{BMI} \leq 24.9$ while the CDC definition is $18.5 \leq \text{BMI} \leq 24.9$. Janssen observed the incidence of 10 health issues related to weight for the subjects he studied for nine years. Compared to normal-weight peers, the risk for developing diabetes grew by 78 percent within the overweight BMI group. Specifically, the relative risks for diabetes for overweight and obese groups were 1.78 and 4.15, respectively. In terms of sex-specific hazards ratios, the diabetes hazard ratio for overweight men (obese men) stood at 2.91 (5.96), but 1.12 (3.26) for overweight women (obese women). Furthermore, when he analyzed two age groups (the first age group consisting of 63- to 74-year-olds, the second age group 75-year-olds and older), Janssen found the hazard ratios for the first overweight (obese) age group and the second overweight (obese) age group were 1.16 (4.29), and 2.21 (2.96) respectively.

Fonarow et al. (2007) studied 108,927 subjects from October 2001 to December 2004 and divided them into four BMI quartiles, QI, QII, QIII and QIV with BMI 16.0–23.6, 23.7–27.7, 27.8–33.3 and 33.4–60.0, respectively. The data came from the Acute Decompensated Heart Failure National Registry (U.S.), with documented admission heights and weights. They found that subjects who were younger and had a higher prevalence of diabetes history were in the higher BMI quartiles. However, they concluded that in-hospital mortality risk for heart failure was inversely related to BMI. The results showed that with QII as the mortality-risk BMI referent, the odds ratios for QI, QIII and QIV were 1.21, 0.84 and 0.83, respectively. In other words, those with higher BMI were less likely to suffer in-hospital heart failure.

Field et al. (2001) conducted a 10-year longitudinal study from 1986–96 on 77,690 middle-aged women from the Nurses' Health Study and 46,060 men from the Health Professionals Follow-up Study. They used subjects with normal BMI as a referent and compared the results with three other categories: overweight, class 1 obese (the same as CDC obese but not extremely obese) and class 2 obese (CDC extremely obese). They concluded that, as BMI increases, so the relative risk of getting diabetes increases, regardless of gender. The relative risk values for overweight, class 1 obese and class 2 obese women (men) were 4.6 (3.5), 0.0 (11.2) and 17.0 (23.4), respectively. However, after changing the reference group to BMI between 18.5 and 21.9, the relative risk values for the remaining part of CDC normal, $22.0 \leq \text{BMI} \leq 24.9$, overweight, class 1 obese

and class 2 obese women (men) were 2.2 (1.8), 8.1 (5.6), 17.8 (18.2) and 30.1 (41.2), respectively.

In a study for the Texas Department of Health, McCusker et al. (2004) examined 1,501,876 routine discharges among Texas adults age 18 and older in 2001. The data, obtained from the Texas Health Care Information Council, showed that 31.6 percent (22.3 percent) of the discharges were attributable to overweight (obese) men with type II diabetes, while 22.3 percent (22.0 percent) were attributable to overweight (obese) women with diabetes. The findings further showed that, compared to normal weight status, the relative risks for diabetes for overweight, obese but not extremely obese and extremely obese men (women) were 3.5 (4.6), 11.2 (10.0) and 23.4 (17.0), respectively. The results also indicated that in 2001, among Texas adults age 18 and older, 2.1 percent of the total morbidity-related loss of productivity was attributable to overweight, 9.1 percent of which was attributable to obesity.

In 2002–03, Tsai et al. (2004) conducted a one-and-a-half-year cross-sectional study of 3,540 men and 2,778 women in southern Taiwan. They found the prevalence of type II diabetes was higher among men than women. They categorize the subjects as normal ($BMI < 23.0$), overweight ($23.0 \leq BMI \leq 24.9$), first-degree obese ($25.0 \leq BMI \leq 29.9$, which is CDC overweight), and second-degree obesity ($BMI \geq 30.0$, which is CDC obese) according to the definition of obesity for the Asia-Pacific region. For type II diabetes, the odds ratios for overweight men (women), first-degree obese men (women) and second-degree obese men (women) were 1.2 (2.0), 1.3 (2.2), and 3.7 (5.1), as compared to normal weight status subjects.

Using 2003 data obtained from the Medical Expenditure Panel Survey, Bhattacharya and Bundorf (2009) examined the relationship between obesity and medical expenditures in the United States. Their results showed that obese women (men) were 3.49 percent (5.38 percent) more likely than nonobese women (men) to develop diabetes. In addition, obese women (men) diagnosed with diabetes spent \$1,522 more (\$802 less) in medical bills than nonobese women (men).

In a recent survey, Malnick and Knobler (2006) concluded there is a strong association between obesity and diabetes, in both genders and all ethnic groups. Even in healthy young men, obesity is an important risk factor for diabetes. They report on a survey of young men in the Israel Defense Forces that found $BMI > 30$ was strongly associated diabetes, compared to those men with a $BMI < 25$.

Alley and Chang (2007) retrieved two sets of data (1988-1994 and 1999-2004) from the NHANES and examined 5,724 and 4,984 adults 60 and older for the first and second time periods, respectively. The results showed that, over time, the prevalence of diabetes increased among obese but not among nonobese subjects.

Hamman et al. (2006) studied the effects of intensive lifestyle intervention for 1,079 participants in the Diabetes Prevention Program. They found weight loss was the dominant determinant in reduced risk for diabetes. A 5-kilogram weight loss over time could account for a 55 percent reduction in the risk of diabetes over the 3.2-year follow-up period of their study.

Urbanski et al. (2008) studied recently published articles on the cost-effectiveness of diabetes prevention and treatment. Four of five studies found diabetes prevention was cost saving. Another four studies found that diabetes prevention is more cost-

effective than treatment. Diabetes self-management training is likely cost-effective but more research is needed. A solid body of evidence shows that medical nutrition therapy changes dietary behaviors resulting in improvement in metabolic control, blood glucose, hemoglobin A1c and cardiovascular risk factors. A small body of evidence indicates medical nutrition therapy is cost-effective in diabetes treatment.

Farin et al. (2006) studied insulin resistance as a function of BMI and waist circumference in 330 healthy volunteers. They found that the more overweight, with respect to both measures, the greater the degree of insulin resistance. They found no difference in the relation between the degree of insulin resistance and either index of adiposity.

Hart et al. (2007) studied the relationship between weight and development of diabetes after age 45 in Scotland. Over 19,000 entered the study from 1970–76, with no reported diabetes, and were followed until March 31, 2004. Hospital discharge records and death certificates were used to identify cases of diabetes. They concluded that overweight and obesity account for about 60 percent of the cases of diabetes.

5. Obesity and Cancer

The American Institute for Cancer Research website has a brief description of the obesity-cancer link (AICR, 2006). The AICR is funding research to examine how fatty acids affect insulin-like growth factors, which may promote cancer. According to this article, obesity results from long-term energy imbalance resulting in increases in storage fat and fatty acid levels. Higher levels of fatty acid result in insulin resistance, which has been linked to diabetes, heart disease and cancer. We found many articles providing empirical evidence of a link between increased BMI and these diseases. For example, Renehan et al. (2008a) review the epidemiological and clinical evidence associating excess body weight with increased cancer risk and its impact on mortality in patients with a diagnosis of certain cancers. A number of possible mechanisms explaining these epidemiological observations are listed, focusing on the three most studied: insulin and insulin-like growth factor, sex steroids and adipokine systems.

A paper by Renehan et al. (2008b) based on a meta-analysis of 141 articles covering 221 data sets and more than 133 million person-years of exposure shows a significant increase in cancer incidence in relation to obesity for 15 sites. This paper covers all prospective studies that could be identified at the time, the majority of which were published after the Surgeon General's 2001 report on the effects of obesity. The Renehan et al. paper noted a causal link to obesity was suggested by the fact that the link to obesity varied significantly by site. Further evidence is provided by some of the specific sites with strong relationships, such as adenocarcinoma of the esophagus, which is related to esophageal reflux that can be associated with obesity.

Renehan et al. (2008a) reported the relative risk of cancer in relation to obesity by sex. In addition to the sites for which the relative risk of cancer increased in relation to obesity, a reduction in risk was indicated for three cancers, lung cancer, esophageal squamous cell carcinoma and premenopausal breast cancer. The first two results appear to be based on the confounding effect of smoking and will be ignored in this study. In the case of lung cancer, the negative effect of obesity did not exist for nonsmokers. The

inverse relationship between BMI and the degree of smoking can explain the confounding effect of smoking measured in a nonquantitative manner, or with smoking intensity in broad categories. The number of studies of esophageal squamous cell carcinoma is much smaller than the number of studies of lung cancer, so it is not possible to perform the same level of analysis of the negative relationship for this cancer, but smoking has a strong association with this cancer, increasing its confounding effect. Thus, the negative relationship between BMI and esophageal squamous cell carcinoma may not be demonstrated to be the result of confounding, but this explanation is reasonable. The reduction of premenopausal breast cancer in relation to obesity may be a valid relationship, in view of the hormonal effects of obesity. Therefore, this reduction in cancer has been included in our study as an offset to the economic costs of obesity.

The significant relative risks identified by Renehan et al. (2008b) are shown in Table 7. These results are the basis for the number of cases used for our estimates of the economic costs of obesity-related cancer. Malignant melanoma and gastric cancer were not included in the analysis because of the opposite effects on males and females.

Table 7
Mean Relative Risk per 5 Increase in BMI

Cancer Site or Type	Female	Male
Gallbladder	1.59	1.09
Endometrium	1.59	
Esophageal Adenocarcinoma	1.51	1.52
Renal	1.34	1.24
Thyroid	1.14	1.33
Colon	1.09	1.24
Liver	1.07	1.24
Leukemia	1.17	1.08
Malignant Melanoma	0.96	1.17
Postmenopausal Breast	1.12	
Pancreas	1.12	1.07
Multiple Myeloma	1.11	1.11
Rectum	1.02	1.09
Non-Hodgkin's Lymphoma	1.07	1.06
Gastric	1.04	0.97
Prostate		1.03
Ovarian	1.03	
Premenopausal Breast	0.92	

Source: Renehan et al. (2008b, Figures 3, 4)

In a comment on the Renehan et al. article, Larsson and Wolk (2008) point out several unanswered questions. For example, does excess fat in adolescence increase cancer risk? Does weight loss in obese people reduce cancer risk?

A recent large meta-analysis of nutrition and non-Hodgkin's lymphoma failed to find

a significant association with severe obesity, defined as BMI ≥ 40 , but suggested further investigation of severe obesity and some sub-types of non-Hodgkin's lymphoma (Willett et al., 2008).

A number of studies have shown an increased risk of death in relation to obesity once cancer is diagnosed. Only renal cancer has shown a decreased risk of death in relation to obesity following diagnosis. The data relating the risk of cancer death to obesity is not yet sufficient to form a basis for adjusting the economic results of obesity-related cancer. Therefore, our economic analysis assumes the outcome of cancers related to obesity is the same as the general results for the specific site.

5.1. Other Literature on Cancer Risk

We mentioned the large prospective study by Calle et al. (1999) in the section on cardiovascular disease. They also studied BMI and cancer using the same cohort. The risk of death from cancer increases steadily with BMI at all ages. The graphs are in Figure 4 of Section 7.2.

Efstathiou et al. (2007) studied 945 men with locally advanced prostate cancer. Greater BMI was significantly associated with higher prostate cancer-specific mortality. For overweight men, the hazard ratio was 1.52; for obese men, the hazard ratio was 1.64. They concluded, "Greater baseline BMI is independently associated with higher prostate cancer-specific mortality in men with locally advanced prostate cancer." Freedland et al. (2008) found that obese men have significantly lower prostate-specific antigen concentrations and larger prostate volumes. While obesity was not significantly related to prostate cancer in univariate analysis, after multivariate adjustment, obesity was significantly associated with increased prostate-cancer risk, with an odds ratio of 1.98.

Larsson and Wolk (2007) performed a meta-analysis of the relation between BMI and gallbladder cancer based on 11 published studies with a total of 3,288 cases. Overweight or obese men had a relative risk of 1.15 for this cancer, compared to normal weight men. The relative risk for women was 1.35. They conclude that excess body weight is a risk factor for gallbladder cancer.

Kabat et al. (2007) studied the relationship between obesity and lung cancer in women 40-59 years old. After adjustment for pack-years of smoking and other covariates, they found inverse associations in current smokers and in former smokers. However, for never-smokers, BMI was positively associated with lung cancer. The results for current and former smokers were not altered by exclusion of cases diagnosed within the first five years of follow-up; however, in never-smokers, the strength of the association was reduced. Calle et al. (1999) found a similar relationship for BMI and all-cause mortality in that the association of increased BMI with the risk of death was stronger for never-smokers than it was for former-smokers or smokers.

A study on risk factors in relation to survival after endometrial cancer diagnosis (Chia et al., 2007) found that a history of obesity and diabetes increases the risk of mortality after endometrial cancer diagnosis. This study found obese women prior to endometrial cancer diagnosis had a hazard rate ratio of 1.6 (95 percent confidence interval 1.0 to 2.5) for all-cause mortality and a hazard rate ratio 2.0 (95 percent confidence interval 0.8 to 5.1) for mortality of endometrial cancer, compared with women who were not

overweight.

Takata et al. (2007) found in a population of 80-year-olds that overweight was associated with longevity and underweight with short life, due to lower and higher mortality, respectively, from cardiovascular disease and cancer. They argued that mortality due to cardiovascular disease in the underweight group was 4.6 times (hazard rate 4.64 with 95 percent confidence interval 1.68 to 12.80) higher than in the normal weight group, and that mortality due to cancers was 88 percent lower (hazard rate 0.12, 95 percent confidence interval 0.02 to 0.78) in the overweight group than in the underweight group.

Schottenfeld and Beebe-Dimmer (2005) reviewed several aspects of cancer in the worldwide population, summarizing articles on related areas. Obesity plays an important role. They find, for example, that the preventive impact of maintaining a BMI below the overweight level was 15-20 percent of cancer deaths in women and 10-14 percent in men.

6. Obesity and Other Conditions

6.1. Osteoarthritis

Allman-Farinelli et al. (2008) wrote a short letter to the editor describing the population attributable risk for osteoarthritis associated with obesity for Australian men at 25 percent and 22 percent for women. Farooqui and Jawad (2007) mainly cited other works and were omitted. All but one of the articles deals with osteoarthritis of the knee.

Cooper et al. (1998) focused on osteoarthritis of the hip, and presented an odds ratio of 1.7 for obese individuals. The frequency of this condition was low enough to make the odds ratio a reasonable approximation to the relative risk; the relative risk provided the basis for estimating obesity-related frequency and costs. We have not measured the frequency and economic costs associated with obesity-related osteoarthritis of the hip.

The remaining articles deal with osteoarthritis of the knee. The low frequency of this condition (1.5 percent) made the odds ratio a reasonable approximation to the relative risk. The odds ratios associated with obesity in several of these articles were reasonably consistent. Davis et al. (1989) presented an odds ratio of 6.6 and Hart and Spector (1993) found an odds ratio of 6.17. Coggon et al. (2001) offered a formula for computing the odds ratio that associated these odds ratios with a BMI of 34, reasonably close to average BMI for obese individuals.

The article by Noskova et al. (2007) found osteoarthritis to be strongly associated with cholesterol and c-reactive protein, rather than with just obesity, but the summary gave no indication of how they controlled for the fact that cholesterol and obesity were highly correlated in reaching this conclusion. In view of the majority of evidence, we ignored this article (its text was excluded from our database).

Coggon et al. (2001) showed that almost all knee osteoarthritis was associated with body weight. An individual with a BMI of 20 had an odds ratio of 0.1. In other words, only about one very thin person in 600 would have this condition, compared to the population average of 1.5 percent. This meant that the portion associated with obesity was less than the portion associated with body mass. The Coggon et al. article estimated

that 57.1 percent of osteoarthritis of the knee would be eliminated if obese people could fall to normal weight. It reviewed 525 cases with 525 controls.

The cost of obesity-related osteoarthritis of the knee has been studied in Italy and found to have direct costs of €934 and indirect costs of €1,236 per patient (a total of about \$3,000). We do not know the corresponding cost in the U.S. and Canada but assume it to be higher in the U.S. Coggon et al. stated that 1.5 percent of people will have knee surgery for osteoarthritis at some point in their lives. Assuming this to stand for about 90,000 cases per year in the U.S. and Canada, the total cost would be about \$270 million. Of this amount, \$154 million would be related to obesity. Anecdotal evidence indicates that recovery from knee surgery is slower for people who are more obese. Therefore, the above estimate may well be understated, especially in relation to indirect costs. To date, however, we have found no scientific evidence to support this observation.

Hart and Spector (1993) used BMI greater than 26.4 (upper tertile), rather than the $\text{BMI} \geq 25$ definition of overweight, to obtain an odds ratio of 6.17. Their odds ratio was for single-knee osteoarthritis. For double-knee osteoarthritis, the odds ratio was 17.99. Both odds ratios were compared to the bottom tertile, BMI less than 23.4.

Manninen et al. (1996) found the relationship between BMI and the incidence of disabling knee osteoarthritis was linear. The relative risk was 1.4. They found also overweight is a strong risk factor for disabling knee osteoarthritis.

Rosemann et al. (2008) studied obesity and quality of life, health service utilization and physical activity in a large sample of primary care patients with osteoarthritis. In this cohort study of 1,029 osteoarthritis patients, they concluded that increased BMI had a strong association with increased use of the health care system. The study emphasized the need for appropriate approaches in primary care to break the vicious circle of overweight, depression, decreasing physical inactivity and diminishing quality of life.

Oster et al. (2000), in the study we mentioned earlier, found that 30 percent of health costs due to knee osteoarthritis are associated with obesity. They used a relative risk of 2.9 for men and 1.6 for women.

6.2. Asthma

Luder et al. (2004) studied asthma-related risk factors in a cross-sectional survey of 5,524 New York state adults. Women showed a steadily increasing association between BMI and asthma. On the other hand, men showed a U-shaped relation, with an increased prevalence of asthma compared to normal associated with the low BMI category and obese category. Luder et al. conjecture that the gender difference may indicate obesity influences sex hormones, which in turn influences incidence of asthma. They cited related longitudinal studies that show BMI predicts development of asthma.

Vargas et al. (2007) examined BMI and asthma for children in a small cross-sectional study of children in the U.S. Head Start Program. Although they found no clear explanation for the link between asthma and BMI, the authors suggested an increased BMI significantly affected the well being of young asthmatic patients and should be studied further. Moore (2005), who surveyed several studies on obesity and asthma, points to an association of asthma with obesity in adults and children. His survey includes a

prospective study of 85,911 women in the Nurses' Health Study that reported a relative risk for asthma of 2.7 for obese women relative to nonobese women.

Chen et al. (2002) used the longitudinal data from the first and second cycles of the National Population Health Survey, conducted in Canada in 1994–95 and 1996–97, to study BMI and development of asthma. In the two-year follow up of 9,149 subjects who reported no asthma initially, 1.6 percent of men and 2.9 percent of women developed asthma. Baseline BMI was a significant predictor of asthma for women but not for men.

6.3. Renal Disease

Hsu et al. focused on the relationship between obesity and end-stage renal disease in a cohort study of 320,252 adults, who were observed for 15 to 35 years (Hsu et al., 2006). The rate of end-stage renal disease increased in a stepwise manner as BMI increased. Age-, sex- and race-adjusted rates of end-stage renal disease rose from 10 per 100,000 person-years among those with normal weight to 108 per 100,000 among those with BMI ≥ 40 . Blood pressure levels or diabetes did not affect this relationship. They reported these values for relative risk of end-stage renal disease for overweight and obese, compared with normal BMI, as shown in Table 8.

Table 8
Relative Risk of End-Stage Renal Disease as a Function of BMI

BMI status	Relative Risk
Overweight, nonobese	1.87
Obese class I	3.57
Obese class II	6.12
Extreme obesity	7.07

Source: Hsu et al. (2006, Figure on page 23)

6.4. In-Hospital Infection

Smith et al. (2007) explored the association between BMI and mortality in patients with infections. They found that increasing BMI is not associated with heightened mortality rate in surgical/trauma intensive care unit patients.

7. Excess Mortality Caused by Obesity

7.1. Overview

As would be expected for a condition that increases the risk of heart disease, diabetes and other serious medical conditions, overweight and obesity increase the rate of mortality. All studies we reviewed found a significant increase in mortality associated with adult

obesity, and most of the studies found a significant increase associated with overweight. As explained in a paper by Greenberg et al. (2007), the measurement of the effect of overweight and obesity on mortality is subject to potential biases resulting from the variation in an individual's BMI over time, and the reverse causation resulting from the reduction in BMI associated with adverse medical conditions. Greenberg et al. found significant increases in the effects of overweight and obesity if the results were corrected for these biases, and also found that some of the published reports on the relationship between obesity and mortality did not make these corrections. For this reason, the mortality effects and related costs we have determined on the basis of published results should be considered a conservative estimate of the economic productivity effects of overweight and obesity.

There is a strong association between very low BMI and increased mortality. For example, individuals with a BMI less than 18.5 have approximately double the mortality of individuals with a BMI between 20 and 25. Since many serious health problems cause weight to decrease, this relationship is not necessarily an indication that low weight causes increased mortality. In fact, it may be almost the opposite: certain conditions causing higher mortality also cause low weight. In any case, the purpose of this research project is to evaluate the effects of overweight and obesity. The higher mortality of people with extremely low weight does not affect the conclusions about the effect of excess weight in relation to normal weight, and therefore in our analysis we have not included any consideration of the effects of extremely low weight.

7.2. Population Studies of Excess Mortality due to Obesity

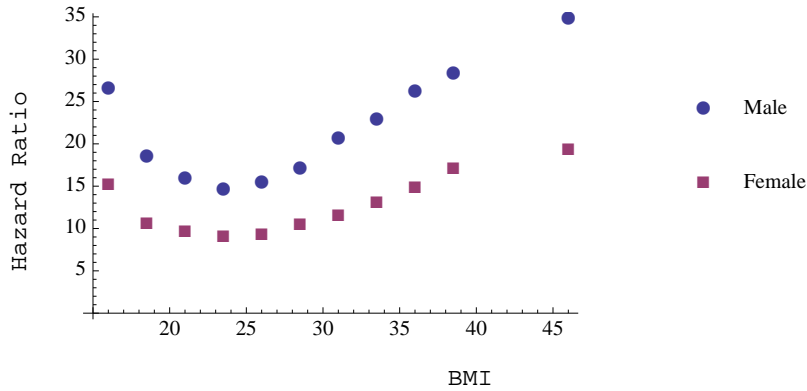
In a recent analysis of 57 prospective studies with 894,576 participants, primarily in western Europe and North America, Whitlock et al. (2009) found that lowest mortality was associated with BMI of 22.5 to 25. This impressive study also found the J-shape or U-shape for BMI and all-cause mortality. They found that each 5-unit increase of BMI was associated, on average, with an increase of 30 percent in the hazard ratio for all-cause mortality. Below the 22.5 to 25 range, BMI was inversely associated with all-cause mortality, mainly because of the strong inverse associations with respiratory disease and lung cancer. Figure 3 shows their graphic presentation of the relationship between BMI and annual deaths per 1,000.

We already mentioned the work of Calle et al. (1999) in the sections on cardiovascular disease and cancer. As noted earlier, this is a large 14-year cohort study of more than 1 million U.S. residents. The authors reported results for all-cause mortality as well as cardiovascular disease and cancer. In addition, they reported results related to smoking habit, race and disease history.

For example, in Figure 4, we plot data from their study showing the relative risk of death for all-causes as a function of BMI for participants who had never smoked and had no history of disease at entry into the study (for each sex and race). The relationship is J-shaped or U-shaped for all-causes and cardiovascular disease (not shown here), while, for cancer, it is increasing at all ages (not shown here).

Pednekar et al. (2008) found that cohort members with BMI < 16 were at highest risk for death due to tuberculosis, with relative risk 7.20 for men and 14.94 for women.

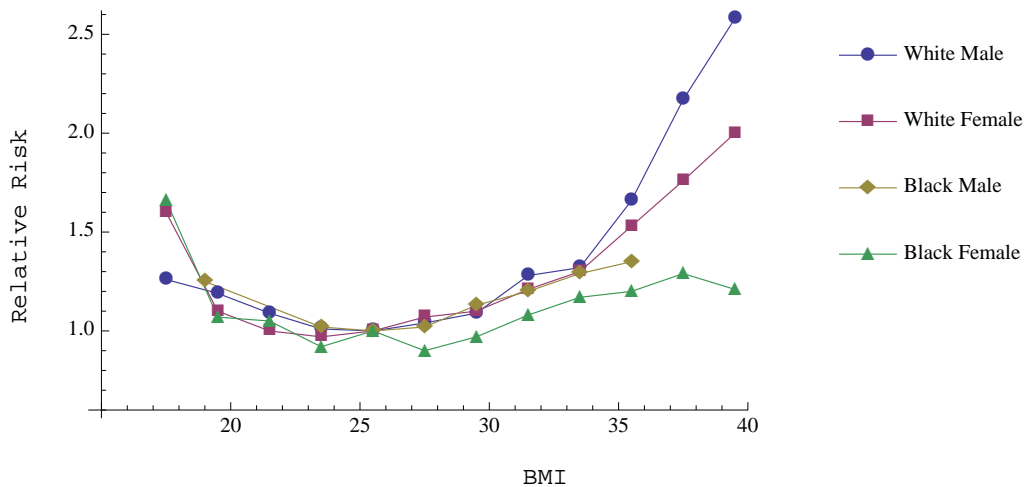
Figure 3
Hazard Ratio for All-Cause Mortality Versus BMI



Note: Hazard ratio for all-cause mortality (annual expected deaths per 1,000 in the U.S.) for ages 35-89 combined versus BMI for male and females in the BMI range 15-50 (excluding the first five years of follow-up). The source article has much more data, such as 95 percent confidence intervals for the estimates.

Source: Whitlock et al. (2009, Figure 2)

Figure 4
Relative Risk of Death Versus BMI in the United States



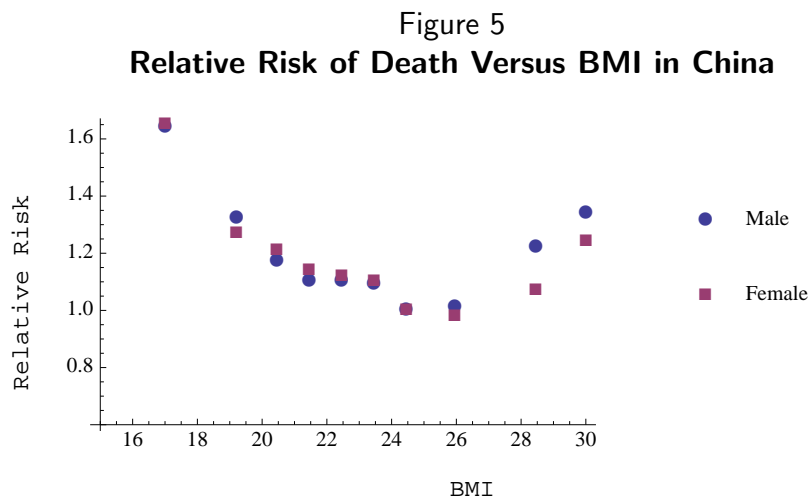
Note: Multivariate relative risk of death from all-causes among men and women who have never smoked and who had no history of disease at enrollment, according to BMI.

Source: Calle et al. (1999, Table 2)

The cancer relative risks were 1.87 and 2.44, respectively, and the respiratory diseases relative risks were 3.46 and 4.35, respectively. Subjects with above normal BMI had lower mortality risk than those with normal BMI values.

Gu et al. (2006) studied a sample of 154,736 adults in China from 1991–2000 to determine the relation between BMI and all-cause mortality. Their results indicate that both underweight and obesity were associated with increased mortality – that is, they also found the U-shaped relation. Their study is unusual in that it is very large and it has a large proportion of participants with BMI < 23, which allows for more precise and more powerful statistical estimates of the relationship between BMI and mortality rates. The U-shaped relation shows up in the subgroups of participants at ages less than 65 as well as the over age 65 subgroup. It also shows up in both healthy and unhealthy subgroups.

Figure 5 is based on data from the Gu et al. article; it illustrates the relationship between BMI and relative risk of death due to all causes for men and women. The article has a lot more data and graphics. The authors found that the U-shaped relation between BMI and mortality remains even after adjustment for factors such as age, sex, smoking habit and so on.



Note: Relative risk of death from all causes as a function of BMI for men and women. The reference group for the relative risk calculation is the group with BMI between 22.0 and 23.9, which had the lowest all-cause mortality rate.

Source: Gu et al. (2006, Table 2)

Reuser et al. (2008) analyzed the life expectancy of adults age 55 and older in the Health and Retirement Survey, a U.S. prospective longitudinal study. They found no statistically significant difference between survival of overweight women or obese men and women, on the one hand, and the corresponding normal weight group, on the other. They found severe obesity (BMI \geq 35) costs three years (95 percent confidence interval 2.2 – 3.8) for men and 5.2 years (95 percent confidence interval 4.4 – 6.1) for women, and that underweight shortened the life of men by 2.4 years compared with men of normal weight.

Allison et al. (1999b) calculated the annual number of deaths attributable to obesity. The estimated number of annual deaths attributable to obesity among U.S. adults is

approximately 280,000 based on hazard ratios from all subjects and 325,000 based on hazard ratios from only nonsmokers and never-smokers.

Weitof et al. (2008) investigated the effects of different BMI levels on mortality and hospitalization for a Swedish population age 16–74. They observed that the relative risks adjusted for age, longstanding illness, smoking and educational level at baseline for all-cause mortality was 2.4 (95 percent confidence interval 1.6 to 3.6) for underweight men, and 2.0 (95 percent confidence interval 1.5 to 2.7) for underweight women. They did not associate overweight with increased all-cause mortality but found obese men and women had about 50 percent higher risk of all-cause mortality than normal weight people.

In a cohort study of Americans 65 or older, Al Snih et al. (2007) observed that disability-free life expectancy was greatest among overweight subjects. The lowest hazard rate was in the overweight group, with a hazard rate of 0.78 (95 percent confidence interval 0.72 to 0.85), and obese but not severely obese, with a hazard rate of 0.80 (95 percent confidence interval 0.72 to 0.90). They also found a U-shaped relationship. The underweight and severely obese experienced higher hazard rates for all-cause mortality than normal weight.

A study of an 80-year-old population in Japan found different results (Takata et al., 2007). Takata et al. found that overweight in the 80-year-old population was associated with longevity and underweight was associated with higher mortality than normal weight. The lower mortality of overweight was associated with lower mortality from cardiovascular disease and cancer.

Flegal et al. (2005) concluded that underweight and obesity were associated with increased mortality relative to the normal weight group in the National Health and Nutrition Examination Surveys for 1971–75, 1976–80 and 1988–84. They estimated the excess deaths to be 111,909 (95 percent confidence interval 53,754 to 170,064) for obesity, 33,746 (15,726 to 51,766) for underweight, and -86,094 (-161,233 to -10,966) for overweight. They reached similar results using estimated relative risks. They reported that the relative risk of death for underweight and extreme obesity were significantly higher than for normal weight.

Manson et al. (2007) subsequently published a critique of the estimates of Allison et al. (1999b), Mokdad et al. (2004), Mokdad et al. (2005) and Flegal et al. (2005). A critique by Greenberg et al. (2007) is described below.

Using data from the Physicians' Health Study, Ajani et al. (2004) estimated the relative risk of death for different BMI groups as follows. This is a cohort study of 85,078 men age 40 to 84 with five-year follow-up. The relative risk for the obese group was statistically higher than that of the reference group (shown in Table 9). There were no significant relative risk differences among the other BMI groups and the BMI group with BMI 22.5 to 24.9.

Kulminski et al. (2008) investigated the relationship of BMI and nine-year mortality in Americans age 65 or older in the National Long Term Care Survey. Their findings showed that overweight or mild obesity ($25 \leq \text{BMI} \leq 34.9$) was not a risk factor for nine-year mortality. They observed a nonsymmetric U-shaped pattern, with larger risks associated with underweight ($\text{BMI} < 22$) and minimal risks for BMI of 25 to 34.9 compared with the normal group ($22 \leq \text{BMI} \leq 24.9$). Arndt et al. (2007) found a

Table 9
Relative Risk of Death Estimates by BMI for the United States

BMI	Men who never smoked	All men
< 20	0.86	1.57
20.0–22.4	0.98	1.07
22.5–24.9 (Referent)	1.00	1.00
25.0–27.4	1.05	1.09
27.5–29.9	1.22	1.19
≥ 30	1.67	1.61

This is based on the Physicians' Health Study. The source article has much more information, including confidence intervals for the estimates. *Source:* Ajani et al. (2004, Table 2)

strong U-shaped association between BMI and all-cause mortality in a cohort study with a 10-year follow-up for men doing heavy physically work.

However, Greenberg et al. (2007) argued that all-cause mortality hazard ratios increased after correcting for putative biases. This is a very convincing argument that the U-shape and J-shaped earlier results are the result of bias induced by regression dilution and reverse causation. This helps explain some of the discrepancy between previous estimates of mortality attributable to obesity. The Allison et al. (1999b) estimate of 325,000 excess deaths attributable to obesity in the United States is 97 percent larger than the Flegal et al. (2005) estimate of 164,836, when the same reference category is used. Allison et al. attempted to correct for reverse causation by excluding smokers. The Flegal et al. (2005) estimates were not corrected for regression dilution or reverse causation. A second important finding is that correcting for the putative biases changed the hazard ratios for overweight from less than 1.00 to greater than 1.00. This was true for all three ideal-weight categories tested. It is possible, therefore, that Flegal et al. found negative rather than positive estimates of mortality attributable to overweight because they did not correct for the putative biases.

The lowest all-cause mortality was also observed in overweight by Takata et al. (2007) in a cohort study with four-year follow-up for the 80-year-old population; by Janssen (2007) in a cohort of 65-year-old men and women; by the Study of Osteoporotic Fractures in 65-year-old women (Dolan et al., 2007); and by some Asian studies (Pednekar et al., 2008; Song et al., 2007). Janssen argued that the all-cause mortality risk was 11 percent lower in the overweight group. Song et al. found a U-shaped association between BMI and all-cause deaths, with the lowest risk for BMI between 25 and 26.9 for middle-aged Korean women.

Corrada et al. (2006) explored the relationship between BMI and all-cause mortality in the elderly (average age 73 at study entry) using data from a large California-based cohort study. They used self-reported BMI at study, BMI at age 21, and weight change between age 21 and study entry. Relative to normal BMI, the relative risk of death for underweight was 1.51. The relative risk for obesity was 1.25. Those who were either overweight or obese at age 21 had increased relative risk of death of 1.17. This study

found a U-shaped relationship between BMI and relative risk for all-cause mortality, like the Calle et al. (1999) and Flegal et al. (2005) studies. Of course the mortality rate for smokers is higher than the rate for non-smokers with the same BMI. The BMI with lowest mortality for smokers is in the overweight range in the Calle et al. and Flegal et al. papers. But as stated in the Calle et al. paper, the analysis of mortality in relation to BMI among smokers is difficult to evaluate because of the interaction of health problems and weight loss among smokers. Calle et al. suggest that the best way to evaluate the effect of BMI is to study the effects among non-smokers without pre-existing health problems.

Adams et al. (2006) conducted a cohort study of 527,265 U.S. men and women, ages 50 to 71 in 1995–96. After 10 years, 61,317 had died. The analysis, limited to healthy nonsmoker participants, revealed that overweight and obesity were significantly associated with the risk of death for both men and women. For healthy 50-year-olds, the risk of death increased 20 to 40 percent for overweight persons and 200 to 300 percent for obese persons.

Artham and Ventura (2007) contributed evidence in support of a U-shaped relationship between BMI and increased risk for cardiovascular disease and all-cause mortality. They asserted, “These data suggest that obesity should not be ruled out as a risk factor just because of the existence of the obesity paradox and it continues to be a risk factor for heart failure or coronary disease.”

Jeffreys et al. (2003) studied the mortality of 692 men in Scotland for which BMI was measured in early adulthood and middle age. They found that overweight in early adulthood is more strongly associated with increased risk of death than is overweight at mid-adulthood. Men who were overweight at both ages had twice the risk of death as men who were normal weight at both ages. The change in BMI between ages was not associated with mortality.

7.3. Excess Mortality due to Obesity in Life Insurance Studies

In one of the few recent studies based on insured populations, Niverthi and Ivanovic (2001) describe results of a study of 356,926 individual life insurance applicants, from date of issue from 1975-1998 and followed until death, lapse or survival to the end of 1999. In general, their results agree with general population studies. For example, for most groups defined by age, sex and smoking habit, the relative risk of death has the familiar J-shape, as shown in Table 10. The exception is the group of 40- to 59-year old male smokers.

As Niverthi and Ivanovic note, smokers over age 60 seem to benefit from extra weight. Moreover, because of the low expected death rates, healthy male nonsmokers appear to have the greatest relative risk, and this is a group where competition for preferred issues is high. The authors assert that even small deviations from the expected can have a significant impact on profits.

Baldinger et al. (2006) reported on a long-term study of mortality of 22,927 life-insured Swiss males with high levels of sums insured. The prevalence of overweight was 35.7 percent and prevalence of obese was 6.2 percent, about the same as the general population. BMI and all-cause mortality showed a U-shaped relationship with the nadir

Table 10
Relative Risk of Death for Male Insured Lives in the United States

	BMI Range	Age Range			
		All	18–39	40–59	≥ 60
Smoker	19–22	1.20	1.82	0.90	1.83
	22–24	1.15	1.23	1.08	1.07
	25–27 (Referent)	1.00	1.00	1.00	1.00
	28–30	1.06	1.20	0.97	1.58
	≥ 31	1.49	2.32	1.45	0.35
Nonsmoker	< 22	1.37	1.06	1.69	0.94
	22–24 (Referent)	1.00	1.00	1.00	1.00
	25–27	1.18	0.91	1.37	1.08
	28–30	1.23	1.17	1.45	1.02
	31–33 (≥ 31)	1.68	1.67	1.84	2.23
	≥ 34	1.84			

Source: Niverthi and Ivanovic (2001, Table 3)

at 22.0–23.9. Some results from this study are illustrated in Figure 6.

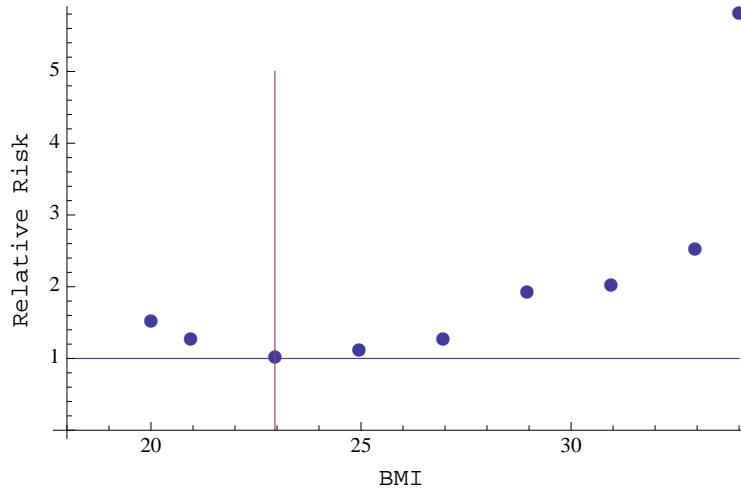
Those with BMI above the normal range tended to be older than subjects with BMI in the normal or underweight ranges. Hypertension was more prevalent and more severe as BMI increased. The authors note that the participants are from high socioeconomic classes and yet all-cause mortality increases significantly with BMI. They concluded, “life insurers clearly share the same goals as public health authorities.”

Roudebush et al. (2006) studied 241,966 life insurance policies submitted from 1989–2003 with 10-policy-year follow-up, comparing actual BMI and standardized mortality based on the 2001 Valuation Basic Table (VBT). They found a U-shaped pattern with the minimum attained for the group with BMI in the 25–29 range, as illustrated in Figure 7. Roudebush et al. make the following conclusions:

- Extra mortality for obesity amounts to 25–75 percent. For underweight and extreme obesity it is 75–150 percent.
- There was excess mortality for the underweight and obese, even at the shortest durations.
- The pattern of standardized mortality ratios by BMI was not materially different by any significant underwriting factor. For example, the relationship is found in the smoker and nonsmoker subgroups.
- There is an ongoing need for more extensive research like the historical sweeping investigations.

Cumming and Pinkham studied 54,387 male insurance policies, issued by a single company during 1994–2004, and followed through 2005. Measured height, weight and

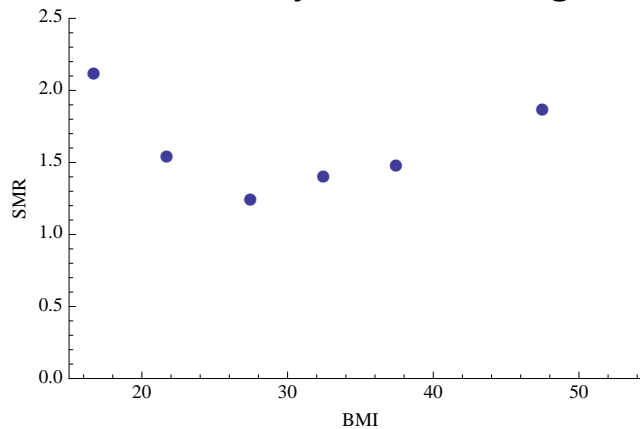
Figure 6
Relative Risk of Death Versus BMI for Swiss Male Insured Lives



Note: This relative risk of death from all causes according to body mass index, adjusted for age and calendar year, for Swiss males with high levels of life insurance. The reference group has BMI between 24.0 and 25.9.

Source: Baldinger et al. (2006, Figure on page 49)

Figure 7
Standardized Mortality Ratios According to BMI



Note: These are the overall results for the entire group of policies. The standardized mortality ratios are based on the 2001 Valuation Basic Table (VBT). BMI was calculated from measurements of height and weight. The source article has much more data, such as 95 percent confidence intervals for the estimates.

Source: Roudebush et al. (2006, Figure 1)

waist circumference was available for all of these policies. Their main objective was to determine whether BMI or waist circumference is a better predictor of future all-cause mortality. The relative risk of increased mortality due to obesity is 1.33 when obesity is defined by $\text{BMI} \geq 30$. The relative risk is 1.20 when obesity is defined as waist circumference ≥ 40 inches (101.6 cm). They found that obesity has significant implications for underwriting and pricing life insurance, regardless of which measure is used to define obesity. Mortality risk increased with both BMI and waist circumference, and BMI and waist circumference are highly correlated. Both BMI and waist circumference predict mortality well, at least in the male insured lives population.

7.4. Effect of Overweight and Obesity on Mortality Rates

We have used the results of seven published studies to estimate the average effect of overweight and obesity on mortality of individuals at an age from 20 to 65, which we consider the typical working lifetime. In addition, we have used the results of a study that showed a declining effect of overweight and obesity with increasing age. The research results show that overweight and obesity are responsible for approximately 16 percent of population mortality from age 30 to 44, 17 percent from age 45 to 54, and 14% from age 55 to 64.

While our analysis is based on empirical research, we found two articles based on models, rather than empirical analysis, that we should mention (Olshansky et al., 2005; Chatterjee et al., 2008).

Olshansky et al. (2005) adjusted a U.S. population mortality table, removing the effect of obesity. In effect, the result is a mortality table for a population that is the same as the original population except that each member with $\text{BMI} \geq 30$ is replaced by an otherwise identical life with $\text{BMI} = 24$. The conclusions from this model analysis are relatively pessimistic. The authors anticipate that life expectancy at birth and at older ages will level off or even decline within the next 50 years as a result of the increase in obesity. Perhaps their approach can be applied to obtain estimates of mortality costs due to overweight and obesity, but they did not do it. (And we took another approach.) It is also possible that their approach could be adapted to insured life tables, provided overweight and obesity prevalence data is available for the same population of insured lives.

Chatterjee et al. (2008) used a very detailed stochastic model of an individual's lifetime that includes the effect of BMI on diabetes, heart disease and mortality (as well as other factors). The model parameters were estimated with data from the Framingham Heart Study and the Health Survey for England. Their conclusions are relatively optimistic. For example, they find that even if the prevalence of obesity increases to an extreme degree, the effect on expected future lifetime would be small. However, this model does not work like most of the empirical studies in which data (age, sex, BMI etc.) is collected at the beginning of the study, the person is observed during the study period, and results (onset of disease, treatment, death etc.) are recorded. This model simulates the life of the person over time with transitions between different states of health. The results for someone age 20 would not be based only on their current BMI, but rather on a simulation of their future BMI. For example, the authors state that a

person who was obese, but as a result of health problems became underweight and then died, would be considered an example of the death of an underweight person. This does not provide an evaluation of the causal effect of obesity. This approach, while interesting and valuable, does not really focus as much on the effect of BMI as on the effect of the pattern of change.

7.5. Costs Related to Loss of Productivity Because of Excess Mortality

We have estimated the costs of overweight and obesity related to the loss of productivity caused by excess mortality. Our estimates are based only on labor productivity using average earnings, and do not include the loss of an individual's services to his or her family. We have made no assumption of any difference between the earnings level of obese people who are working compared to nonobese people who are working. There is some evidence that obese individuals who are working may earn slightly less income than normal-weight individuals, but the large percentage of people who are obese or overweight makes it unlikely that this represents a large difference between the average earnings of working individuals who are not overweight versus those who are overweight or obese. In addition, to the extent that such a difference might reduce the economic effect of mortality or disability, the reduction would be offset by the economic cost represented by the lower income itself.

Worklife expectancy is defined as the expected number of years that an individual will be employed during their future lifetime. This is affected by their life expectancy, and by the probability of working at each future age. Worklife expectancy has been found to vary significantly on the basis of age, sex, education, and whether or not the person is actively employed at the time of determination (Millimeta et al., 2003). To determine average working lifetime for the population as a whole, we used the worklife expectancy for individuals age 20 published by the Bureau of Labor Statistics in 1986. While worklife data for individuals with various characteristics have been published by researchers since 1986, the Bureau of Labor Statistics report is the most recent evaluation of worklife for the population as a whole. We were unable to find a population worklife estimate for Canada, but the percentage of the population that is employed is very similar to that of the United States, which implies a similar worklife.

The average worklife expectancy for people age 20 in the United States is 36.8 years. For the purpose of evaluating the loss of productivity from obesity, we assumed people typically work a level percentage of their time from age 20 to 65. On the basis of the population mortality rates published by the Social Security Administration, the average number of years lived by a 20-year-old to age 65 is 43.1 years, so the number of years worked (i.e., 36.8) is 85 percent of the potential working lifetime while they are alive. The ratio of years lived from 20 to 65 to the potential 45 total years is 95 percent. This means that when comparing worklife to the potential maximum total of 45 years, it is reduced 5 percent by mortality and 15 percent by the fact that, on average, 85 percent of the living population in that age group is working at any time.

The population mortality table published by the Social Security Administration in-

cludes any excess mortality caused by obesity, so, to determine the effect on worklife, we reduced the rate of mortality at each age to eliminate the excess mortality we have computed. We used the percentage of deaths caused by overweight or obesity as stated above to provide an age-based distribution of the excess mortality caused by obesity during the working lifetime.

We computed the years that would be lived from age 20 to 65 if the adverse effects of overweight and obesity were eliminated, and used that increase to estimate the productivity gained if overweight and obesity were eliminated. This amount is the cost of the loss of productivity (excluding services to one's family) caused by excess mortality related to overweight and obesity.

According to the research of Stevens et al., the relative effect of overweight and obesity on mortality declines with age (Stevens et al., 1998). On the other hand, the degree of obesity increases with age. We have combined the decreasing mortality effect of BMI by age, and the pattern of increasing BMI by age, to produce an age-related mortality effect. We then applied this effect to the base rates of mortality by age to determine the effect on worklife. The effect of overweight and obesity excess mortality is to reduce the population average worklife by 3.8 months. This is consistent with the research of Olshansky et al. (2005), which found the overall effect of obesity on life expectancy in the U.S. to be a reduction of 1/3 of a year to 3/4 of a year. That study adjusted for obesity only; it did not adjust for overweight. To have this population effect caused by 65 percent of the adult population that is overweight or obese, the loss of worklife caused by excess mortality for overweight or obese individuals is an average of 5.8 months. On the basis of average earnings of \$35,700, and average employee benefits of 19.4 percent, this creates an average lifetime cost of \$20,600 per overweight or obese individual, which is equivalent to an annual cost of \$49 billion for the working-age population of the United States and Canada as a whole. Since the levels of overweight and obesity in Canada are less than those of the United States, the cost in the United States is \$44 billion³. The cost in the United States and Canada related to overweight is \$6 billion, and the cost of obesity is \$43 billion.

8. Disability

8.1. Overview

Disability rates are increased by obesity. For example, a significant portion of disability cases are caused by the medical conditions identified as having an increased rate as a result of obesity. In addition to these conditions, however, there are conditions, such as osteoarthritis, that have little or no effect on mortality, but have significant disability effects, and for which obesity has a significant causal relationship. In addition to higher rates of these conditions, there is evidence that obesity significantly increases the time for recovery from disabling medical conditions.

³\$44 billion \approx \$35,700 \times 119.4% \times (5.8/12) \times 65.1% overweight or obese \times 148,000,000 working age individuals \times (1 year / 45 years)

8.2. Disability Costs of Overweight and Obesity

To evaluate the economic effects of disability, the disability considered in this evaluation is employment disability, which is a subset of general disability. Specifically, we consider employment disability that is caused by physical disability resulting from overweight or obesity.

There are other forms of disability, such as mental disability, that reduce employment, but we have not considered these, because the effect of overweight or obesity on these types of disability is minimal or absent. While there appears to be general agreement about the increase in employment disability as a result of obesity, there is limited quantitative evidence of the degree of increase in employment disability caused by overweight or obesity. The majority of the studies relating obesity to disability are concerned with physical function, rather than employment disability, and tend to focus on the elderly.

We have focused on four studies that deal with disability among individuals who could be employed if not disabled. Cawley et al. (2007) and Bungum et al. (2003) studied the absenteeism caused by overweight or obesity among employed individuals. A study by Ferraro et al. (2002) evaluates the effect of overweight and obesity on disability in the sense that the individual is unable to perform various physical activities, rather than specifically having limited employment ability. A study by Østbye et al. (2007) evaluates the increase in workers' compensation claims caused by overweight or obesity. The study by Ferraro et al. includes a definition of disability broader than the type of disability that would reduce productivity, and the study by Østbye et al. is based on a more narrow definition of disability. We were unable to obtain studies of employment disability associated with overweight or obesity in Canada, so our analysis of employment disability is limited to the United States.

While the two studies of worker absenteeism both found a statistically significant increase in absenteeism associated with obesity, they produced somewhat different numerical results. The study of Cawley results in an estimate that 0.75 percent of potential work time is lost as a result of absenteeism associated with overweight or obesity. The study of Bungum et al. results in an estimate that 0.19 percent of potential work time is lost. The study by Bungum et al. was based on responses of 471 municipal employees who responded to a survey request mailed to 3,300 employees and requested in meetings with portions of a 13,000 employee group. There was no adjustment in the Bungum et al. study for any bias that might have existed between the responses of employees who were willing to respond to a survey about their absences and the absenteeism of those who did not respond. On the other hand, the study of Cawley et al. was based on the Medical Expenditure Panel Survey for 2002-04, which covered 55,000 individuals, and had a much higher response rate. For these reasons, we have used the Cawley et al. study to produce the numerical results for worker absenteeism caused by overweight or obesity.

Using the average earnings per year and the average employee benefit rate cited above, the absenteeism identified by the Cawley et al. study results in a loss of productivity of \$43 billion per year.

The two studies of worker absenteeism deal with the absenteeism of active employ-

ees. The number of days of work missed per year as a result of sickness or accident in both studies is approximately one week for all employees, including those in all weight classes. While these studies produce results for the effect of overweight and obesity on the productivity of active employees, they do not include a more significant reduction in productivity caused by long-term disability. The 2006 American Community Survey conducted by the U.S. Census Bureau found that 23.9 million individuals in the U.S. from age 16 to 64 had physical disabilities. Of this group, 15.0 million, or 62.8 percent were not employed. Among people who were not disabled, 25.6 percent were not employed. In other words, a reduction of 37.2 percent in employment can be attributed to physical disability, which represents 8.9 million individuals who are not employed. This represents a loss of 6.0 percent of existing productivity. This is eight times the loss of productivity identified in the above studies as caused by physical problems among employed individuals. Therefore, to evaluate the total effect of obesity, the above studies must be supplemented with an estimate of the percentage of potential workers with employment disability caused by overweight or obesity who are not active employees because of their disability. Using a relative risk of 1.24, which is the relative risk identified for disability of active employees applied to the total 6 percent loss of productivity caused by all physical disabilities, the annual cost caused by overweight and obesity is \$72 billion per year. This means the total loss of productivity caused by employment disability and increased absence is approximately \$115 billion in the United States. Based on the ratio of the number of overweight and obese individuals in Canada compared to the United States, the total annual cost for the United States and Canada would be about \$128 billion. We did not obtain any research data that split the cost of overweight and obesity between those two categories. If the relative rates are similar to the rates for medical problems, the cost in the United States and Canada would be approximately \$39 billion for overweight and \$89 billion for obesity.

An important element of the study of workers' compensation claims is the duration of claims based on BMI. There is a significant increase in the duration with increasing body mass, with employees with a body mass over 40 having double the number of days per claim compared to employees with a BMI between 18.5 and 25. This relationship would appear to be applicable to some degree to disability other than workers' compensation claims, which would provide support for the use of extended disability periods to estimate a portion of the overall reduction in productivity caused by conditions resulting from overweight or obesity.

Workers' compensation claims are based on work-related injuries. Except for relatively rare situations in which an illness can be ascribed to employment, workers' compensation claims do not include employment disability resulting from illness. This means that the reduction in productivity indicated by workers' compensation claims is a fraction of the total loss of productivity caused by the increase in illness or injury caused by overweight or obesity. The increase in workers' compensation claim costs caused by overweight and obesity is \$10 billion per year, approximately 10 percent to 15 percent of the total cost of workers' compensation coverage.

In summary, the total cost caused by the loss of productivity due to excess deaths and disability as a result of overweight and obesity, as well as the cost of work-related injury associated with overweight and obesity, is approximately \$177 billion per year in the

United States. Based on the population ratio between the United States and Canada, and the relatively similar percentages of overweight and obesity, and the currently similar value of Canadian and U.S. dollars, we would estimate a total cost in Canada of excess mortality, disability and absenteeism associated with overweight and obesity at \$18 billion.

8.3. Effect of Juvenile Obesity

Our analysis of lost productivity associated with overweight and obesity has been carried out to identify current costs. Therefore, we have not included effects of obesity on juvenile individuals because they are not currently working. On the other hand, there is a clear relationship between juvenile obesity and a loss of productivity in the future. The most obvious reason is that obesity tends to continue, i.e., an obese juvenile is much more likely to be obese as an adult than a juvenile of normal weight. A second reason, which has recently been demonstrated, is that obesity of juveniles tends to produce health problems that can continue into adulthood and cause future losses of productivity of those individuals.

9. Economic Costs of Obesity

9.1. Articles on Obesity Costs

Wolf and Colditz (1998) used the prevalence-based approach to estimate an update to earlier estimates of the economic costs of obesity in the U.S. The authors applied population-attributable risk percentages to total annual direct costs of diabetes, coronary heart disease, hypertension, gallbladder disease, cancer and osteoarthritis to determine corresponding costs due to obesity. The authors also describe how indirect cost indices (bed days, restricted-activity days and work-loss days) increase with BMI. They were able to convert these measures to annual costs. Table 11 summarizes direct and indirect costs from their article. They concluded that direct costs associated with obesity represent 5.7 percent of U.S. aggregate health expenditure in 1995. In a similar analysis, Seidell and Deerenberg determined that about 4 percent of total health care costs in the Netherlands are associated with obesity (Seidell and Deerenberg, 1994). They cited similar figures from other countries: direct costs of obesity amount to about 5 percent of total costs in industrialized societies in 1994.

Katzmarzyk and Janssen (2004) also used the prevalence-based approach to estimate economic health care costs attributable to inactivity and obesity in Canada. They found the economic burden of physical inactivity was \$5.3 billion (\$1.6 billion in direct costs and \$3.7 billion in indirect costs) while the cost associated with obesity was \$4.3 billion (\$1.6 billion of direct costs and \$2.7 billion of indirect costs). The total economic costs of physical inactivity and obesity represented 2.6 percent and 2.2 percent, respectively, of the total health care costs in Canada. The costs are not additive because some obesity is due to inactivity.

Table 11
Annual Direct and Indirect Costs due to Obesity

l	Direct Cost	Indirect Cost
Diabetes	32.400	30.740
Coronary heart disease	6.990	—
Hypertension	3.230	—
Gallbladder disease	2.590	0.150
Breast cancer	0.840	1.480
Endometrial cancer	0.286	0.504
Colon cancer	1.010	1.780
Osteoarthritis	4.300	12.900
Total	51.646	47.564

Annual costs attributable to obesity in billions of 1995 U.S. dollars for the indicated conditions. *Source:* Wolf and Colditz (1998, Table 2)

Thompson et al. (1998) estimated the economic costs of obesity to U.S. businesses in 1994, using the prevalence-based approach. They estimated that at least 43 percent of all health care spending by U.S. businesses in 1994 was associated with obesity. The impact varies considerably by disease with a low of about 16 percent for hypercholesterolemia and a high of 86 percent for diabetes.

Arterburn et al. (2005) reported the following relationship between BMI and health care expenditures. Table 12 shows some of their results.

Table 12
Adult Total per Capita Health Care Expenditures According to BMI

BMI	Expenditures
< 18.5	2,547
18.5–24.9	2,424
25–29.9	2,664
30–34.9	2,984
35–39.9	3,511
≥ 40	4,399

Annual costs per person in 2002 U.S. dollars.
Source: Arterburn et al. (2005, Table 2)

Østbye et al. (2007) determined the relationship between BMI and workers' compensation claim frequency, associated claim costs and lost workdays. Table 13 summarizes the annual cost per employee.

Based on data collected in 1999 for a large Minnesota health plan, Anderson et al. estimated the proportion of total health care charges associated with physical inactivity,

Table 13
Medical and Indemnity Claims According to BMI

BMI	Medical Claims Costs	Indemnity Claims Costs
< 18.5	71.09	39.24
18.5–24.9	75.03	53.96
25–29.9	133.38	135.69
30–34.9	196.61	236.33
35–39.9	233.73	342.93
≥ 40	510.91	591.78

Annual claims costs per worker in U.S. dollars determined from 1997–2004. *Source:* Østbye et al. (2007, Table 3)

overweight and obesity among U.S. populations age 40 and older (Anderson et al., 2005). Table 14 shows the average annualized charges per participant as a function of BMI. The authors concluded that 27 percent of U.S. national health care costs were associated with physical inactivity, overweight and obesity.

Table 14
Average Annual Health Care Charges in the U.S. According to BMI

BMI	Average Annual Cost
< 25.0	3,994
25.0–29.9	5,239
≥ 30.0	6,146

Annual costs per person in U.S. dollars determined from 1996–99. *Source:* Anderson et al. (2005, Table 2)

Wang et al. (2003) conducted a cross-sectional study of 177,971 General Motors employees and adult dependents in the U.S. from 1996–97 to explore the relation between BMI groups and concurrent medical costs. Overall, they found the normal group had lowest median costs. The underweight group had higher medical costs than the normal group. Median medical costs increased steadily from normal to overweight, and from overweight to obese. The results differ slightly for subgroups defined by age, but a clear J-shape pattern is evident in the data overall. The study confirms that increased BMI, above normal, is associated with increased medical costs, as illustrated in Table 15.

We found several reports by state agencies. For example, according to Powell (2007), the CDC estimated Georgia spends \$2.1 billion per year in costs associated with obesity. A Louisiana report describes obesity as one of the state’s greatest and most pressing public health challenges.

Table 15
Median Annual Health Care Costs per Person According to BMI

BMI	Annual Median Cost
< 18.5	3,184
18.5–24.9	2,225
25.0–29.9	2,388
30.0–34.9	2,801
35.0–39.9	3,182
≥ 40	3,753

Annual costs per person in U.S. dollars determined from 1996-97. *Source:* Wang et al. (2003, page 186)

Daviglus et al. (2004) reported on U.S. Medicare costs for cardiovascular disease and diabetes for men and women 65 and older compared to the levels of BMI at younger ages, average age 46.0 for men and average age 48.4 for women. The results were similar for three different models. Table 16 gives annual health costs in 2002 dollars per person as a function of BMI.

Table 16
Average Annual U.S. Medicare Charges per Person According to BMI

BMI	18.5–24.9	25–29.9	30–34.9	≥ 35
Male	7,339	8,319	10,155	13,531
Female	6,637	7,525	9,270	11,985

These are average annual U.S. Medicare charges per person in 2002 dollars for inpatient and outpatient care from 1984–2002 by baseline BMI determined in 1967–73. *Source:* Daviglus et al. (2004, Table 2)

The study by Rosamond et al. (2008) gives total U.S. costs for medical conditions whose relative-risk was increased by obesity. There is some overlap among the conditions cited. The 2008 costs in billions, by condition, are shown in Table 17.

Bhattacharya and Bundorf (2009) determined medical expenditures based on more than 88,000 person-year observations of workers in the U.S. from 1989–2002. Table 18 shows their estimates of total annual medical costs for nonobese and obese persons.

Wang et al. (2005) examined the influence of physical activity and BMI on health care utilization and costs among U.S. Medicare retirees. They concluded that promoting active lifestyles would improve well-being of Medicare participants and save a substantial amount of health care costs. Some of their results are summarized in Table 19.

Borg et al. (2005) studied hospital days and BMI to determine annual aggregate excess hospitalization and additional indirect costs due to obesity. They estimate that ag-

Table 17
U.S. Annual Health Care Costs

Condition	Total Cost
Heart Diseases	287.3
CHD	156.4
Stroke	65.5
Hypertensive Diseases	69.4
Heart Failure	34.8
Total	448.5

Aggregate annual costs in the U.S. in billions of dollars for various conditions in 2008. *Source:* Rosamond et al. (2008, Table 19-1)

Table 18
Total U.S. Annual Medical Costs for Nonobese and Obese Persons

Women				
Age	Nonobese	Obese	Difference	
18-64	2,718	4,176	1,458	
20-50	2,406	3,193	787	
20-50 Privately Insured	2,586	3,169	583	
Men				
Age	Nonobese	Obese	Difference	
18-64	2,498	2,904	406	
20-50	1,719	1,881	162	
20-50 Privately Insured	1,896	1,949	53	

Aggregate annual costs based on data for U.S. workers from 1989–2002, according to sex, age and obesity status determined by BMI. *Source:* Bhattacharya and Bundorf (2009)

Table 19
U.S. Annual Health Care Costs per Person by Level of Physical Activity

	BMI		
	18.5–24.9	25–29.9	≥ 30
Level of PA (times/week)			
0	12,528	12,583	12,586
1-3	10,439	10,567	11,306
≥ 4	9,178	9,979	10,207

Annual U.S. Medicare health care costs (outpatient, inpatient and drug costs) per person in 2002 dollars, according to BMI and three levels of physical activity (PA). *Source:* Wang et al. (2005, Table 2)

gregate indirect lost production due to obesity-related early deaths at 2.9 billion Swedish krona. Table 20 summarizes their results.

Table 20
Present Value of Excess Aggregate Annual Health Care Costs in Sweden

Category	Hospital Cost	Indirect Cost	Total
Overweight			
Male	39,526	26,663	66,189
Female	40,215	9,193	49,408
Total Obese			
Male	1,320,654	2,387,908	3,708,562
Female	768,904	511,690	1,280,594

The present value is based on a discount rate of at 3 percent. The costs are in Swedish krona (approximately 7 krona per dollar). *Source:* Borg et al. (2005, Table 7)

Buescher et al. (2008) studied the relationship between BMI and Medicaid costs of North Carolina adolescents during 2004. They found that children who receive public health services are more likely to be overweight than children in a representative national sample (27.2 percent versus 17 percent). They found that overweight children in the study had greater risk of diabetes, asthma and mental disorders than normal weight children.

Bungum et al. (2003) studied the relationship between body mass index, medical costs and job absenteeism. They used self-reported data from 577 of the 1,300 employees they asked to participate. The results provide evidence that employee BMI classification predicts high health care costs and high absenteeism in the U.S. The average annual health care cost for the normal class (\$114) increased to \$573 for overweight and to \$620 for obese. The bigger increase was from normal to overweight. Smoking habit and

gender did not predict health care costs or absenteeism.

Finkelstein et al. (2003) used a regression model with U.S. data from the 1998 Medical Expenditure Panel Survey (MEPS) and the 1996 and 1997 National Health Interview Surveys (NHIS) to estimate aggregate medical spending attributable to overweight and obesity. They concluded that (in 1998) 9.1 percent of annual medical spending was attributable to overweight and obesity. That rivals the cost attributable to smoking, which ranges between 6.5 and 14.4 percent, depending on the source.

Finkelstein et al. (2005) discussed the economics of the obesity epidemic, using published data and articles. They found economic medical costs for obese individuals are 36 percent higher than for nonobese individuals. Absenteeism attributable to obesity in the U.S. cost \$2.4 billion in 1998.

Sturm (2002) compares the effects of obesity, overweight, smoking and problem drinking on health care costs based on a national telephone survey of about 10,000 U.S. households from 1997-98. He found obesity has roughly the same association with chronic disease as twenty years' aging, and this greatly exceeds the associations of smoking or problem drinking. Overweight was associated with average increase of \$125 in annual health costs. The cost increases associated with obesity, smoking and problem drinking were \$395, \$230 and \$225, respectively.

Oster et al. (2000) estimated the health and economic burden of obesity to a hypothetical U.S. health plan with 1 million members between ages 35 and 84. This paper has more information, some of which we cited earlier. In Table 21 we summarize the results on obesity-related costs.

Table 21
Obesity Related/Total Costs of Managed Care per Member

Condition	Male	Female	Total
Hypertension	30.9/73.3 (0.42)	41.4/85.3 (0.49)	72.3/158.6 (0.46)
Cholesterol	3.7/19.5 (0.19)	4.3/24.6 (0.17)	8.0/44.1 (0.18)
Type 2 Diabetes	49.7/64.9 (0.77)	80.8/86.8 (0.93)	130.5/151.7 (0.86)
CHD	54.2/153.0 (0.35)	29.3/88.2 (0.33)	83.5/241.2 (0.35)
Stroke	8.3/71.9 (0.12)	15.4/83.7 (0.18)	23.7/155.6 (0.15)
Gallbladder	3.3/7.4 (0.45)	6.9/16.8 (0.41)	10.2/24.2 (0.42)
Osteoarthritis, Knee	7.1/20.2 (0.35)	10.1/37.0 (0.27)	17.2/57.2 (0.30)
Endometrial Cancer	–	0.5/2.2 (0.23)	0.5/2.2 (0.23)
Total	157.2/410.2 (0.38)	188.7/424.6 (0.44)	345.9/834.8 (0.41)

Obesity related costs over total costs, in millions of U.S. dollars, according to condition. We added the numerical value of the ratio in parentheses. *Source:* Oster et al. (2000, Table 5)

Costs associated with obesity include obesity-related work absenteeism. Cawley et al. (2007) documented job absenteeism associated with obesity in the U.S. They found a strong association. Costs are much greater for women than men, as shown in Table 22.

Colditz (1999) surveyed the National Library of Medicine's MEDLINE database for studies reporting on economic costs of obesity or inactivity. The survey results include

Table 22
Aggregate Costs from Absenteeism in the U.S.

	Women			Men		
	Obese	Morbidly Obese	Total	Obese	Morbidly Obese	Total
Overall	1830.1	1337.7	3167.8	453.2	637.8	1091.0
Managers	446.3	211.1	657.4	81.9	317.2	399.1
Professionals	452.1	432.8	884.9	190.5	109.7	300.2
Sales workers	186.8	130.0	316.8	73.0	39.6	112.6
Office workers	301.8	278.8	580.6	14.4	32.0	46.4
Equipment and transportation operators	178.3	132.5	310.8	58.9	96.2	155.1

These are the aggregate costs per year associated with obesity and morbid obesity, by gender and occupation (in millions of 2004 dollars).

Source: Cawley et al. (2007, Table 6)

population-attributable risk for inactivity, which the author multiplied by annual costs of illnesses caused by physical inactivity. Overall, physical inactivity costs the U.S. \$24.3 billion per year (in 1995) for direct health care delivery costs. That is 2.4 percent of all health care costs.

Similarly, Colditz estimated that health care costs associated with obesity were about 7 percent of total health care costs in 1995. Thus, direct health care costs associated with obesity and inactivity amounted to 9.4 percent of total direct costs in 1995. Indirect costs are given, but it is not clear how they were estimated. The author stated: “The indirect costs attributable to obesity amount to at least \$48 billion. The major contributor to these costs is CHD (48 percent), which accounts for the large portion of premature mortality. Other indirect costs were non-insulin dependent diabetes mellitus (17.5 percent), and osteoarthritis (17.1 percent); the latter largely resulted from excess bed days, work days lost, and restricted activity days.”

Allison et al. (1999a) showed that the “prevalence-based” approach overestimates costs because it does not take into account the fact that those with higher BMI have higher mortality rates. They concluded that previous estimates overstate the cost attributable to obesity, although obesity nevertheless increases health care costs.

The report on overweight and obesity in Canada we mentioned earlier (Raine, 2005) also discussed economic costs associated with obesity. The total direct cost of obesity-related health care in Canada in 1997 exceeded \$1.8 billion, about 2.4 percent of total health care costs (Birmingham et al., 1999). In Canada in 1997, about 50.7 percent costs of type 2 diabetes were attributable to obesity. For hypertension, the percentage was 31.6 percent, for pulmonary embolism it was 29.8 percent and for endometrial cancer it was 26.6%. There seems to be no national study, but a study in Nova Scotia calculated that direct costs of obesity were \$128 billion while indirect costs (productivity, absenteeism and disability) were another \$140 billion, for a total of \$268 billion. The report concluded: “In this Canadian context indirect costs of obesity to the economy

exceed direct health care costs.”

Detournay et al. (2000) studied a large sample from the 1991–92 National Household Survey in France to determine the relation of obesity to health care costs. They found the extra cost of obesity is in the range 0.7 to 1.5 percent of total French health care costs (in 1992). The direct health care cost of obesity as a percentage of total cost varied by age. It ranged upward from 3 percent at age 20 to 8 percent at age 55, before moving downward by about 0.3 percent per year. It was actually negative above age 80.

Wang et al. (2004) examined the relationship of body mass index and physical activity to health care costs among 196,000 employees of General Motors Corp. from 1995–96. They found that more physically active employees had significantly lower health costs. If obese sedentary employees adapted an active lifestyle, the total health care costs would be reduced by 1.5 percent.

Visscher and Seidell (2001) stated: “The direct costs of obesity are now estimated to be around 7 percent of total health care costs in the United States and around 1 to 5 percent in Europe. Narbro calculated that approximately 10 percent of the total costs of loss of productivity due to sick leave and work disability might be attributable to obesity-related diseases.”

In a study of Swiss health costs for 2001, Schmid et al. (2005) found that overweight and obesity contributed equally to health costs, although overweight was much more prevalent. Moreover, they went on, the latter was increasing and may even have been underreported. This observation led them to claim that overweight constituted a financial time bomb.

Visscher et al. (2004) studied 19,518 adults in Finland for 15 years from entry in 1973–77. They were able to quantify the number of years lived in an unhealthy state relative to BMI. Obese men age 25–64 who have never smoked lived 0.63 more years of work disability than normal weight counterparts. The figure was 0.52 for women. Excess risk of morbidity and disability due to obesity were the highest in the youngest age groups.

Mansson et al. (1996) studied BMI and its relation to disability and mortality for more than 7,500 Swedish male workers. Some of their results are shown in Table 23. The incidence of disability was lowest for normal BMI and increased as BMI increased to overweight and obese. The incidence was also higher for underweight, probably due to higher incidence of musculoskeletal and circulatory diseases and alcohol dependence.

Table 23
Rates of Disability by BMI for Male Workers in Sweden

BMI	Disability Rate
< 20.0	18.8
20.0–24.9	9.9
25.0–29.9	12.6
> 30.0	23.9

Source: Mansson et al. (1996, page 82)

Schmier et al. (2006) surveyed the literature on worker absenteeism, sick leave and disability associated with overweight and obesity. They found several studies showing overweight and obese employees take more sick leave and have higher absenteeism than normal weight employees. For example, one study found that approximately 10 percent of total sick leave and disability cost in Sweden is associated with overweight and obesity. Several studies report the rate of work-place injuries is significantly higher among overweight and obese workers. For example, a study of 10,000 oil company workers in Texas found the rate of lower back injuries is 1.42 times higher for overweight than normal workers.

9.2. Risk Estimates

We combined the relative risk data from the published studies we reviewed by using the population percentage for each of the BMI groups covered by each study, and by weighting the results by the square root of the number of individuals included in each study. Our reason for using the square root of the number, rather than the number itself, was based on our observation that the variance in results between studies was much higher than would be indicated by the variance of results based on the statistical uncertainty caused by the number of individuals forming the basis of the study. If a series of identical studies found varying levels of effects, it would be appropriate to weight the results of each study by the number of individuals covered by the study. But when the variance of results is much higher than expected, it indicates that the procedures followed by the studies were not identical and some judgment is therefore required in weighting the results. We felt the square root of the population provided increased weight for larger studies while reflecting the fact that smaller studies could be of significant interest because of the variation in methodology. This decision does not have an overwhelming effect on the ultimate results.

In the case of cancer, we used a different methodology because a meta-analysis by Renehan et al. (2008b) provided excess cancer rates caused by overweight or obesity on the basis of 141 published studies, covering almost all types of cancers identified as being related to obesity. The increase in the rate of uterine (endometrial) cancer was not covered in the Renehan et al. study, so we based our results for that cancer on a study published by the World Cancer Research Fund (Marmot, 2007).

9.3. Excess Medical Costs Caused by Overweight and Obesity

Using the total medical costs reported by the Medical Expenditure Panel Survey (MEPS)⁴ combined with the base rate and relative risks of overweight and obesity determined as stated above, we have estimated the excess medical costs of cancer, cardiovascular disease, diabetes, kidney disease and strokes caused by overweight and obesity. The MEPS costs were reported as of 2006, so we increased these costs by 11.4 percent to reflect the medical cost inflation from 2006 to 2009. Because the documented relationship of

⁴Medical Expenditure Panel Survey, Table 3: Total Expenses for Selected Conditions by Type of Service: United States, 2006, published at website http://www.meps.ahrq.gov/mepsweb/data_stats

cancer to obesity relates only to certain specific types of cancer, we used other sources to estimate the medical costs for these types of cancer. The data upon which our cost estimate is based for cancer, cardiovascular conditions, diabetes and hypertension are shown in the Table 21. The relative risk values are based on the overweight and obese portion of the population.

Twenty-five percent of kidney-stone disease is caused by overweight or obesity (Taylor et al., 2005) with a cost of \$0.6 billion (Pietrow and Karellas, 2006). More than 60 percent of end-stage renal disease is caused by overweight or obesity (Hsu et al., 2006) with a cost of \$22.6 billion⁵. Based on population ratios, we estimate the cost in Canada at \$2.7 billion.

According to Silva et al., the rate of strokes in the population is increased by overweight and obesity (Silva et al., 2006). Approximately 22 percent of the strokes in the U.S. are caused by overweight or obesity. The direct cost caused by the newly occurring strokes was found to be \$40.6 billion in 1990 (Taylor et al., 1996). Since much of this amount is based on non-medical costs, such as nursing home care, we used nonmedical inflation to project a value of \$66.6 billion in 2009. The portion caused by overweight or obesity is approximately \$6.2 billion. Based on population ratios, we estimate the cost in Canada at \$0.7 billion.

The cost of osteoarthritis is much less than that of the other conditions noted above, so it is not included in our estimate; however, osteoarthritis is discussed in more detail below because its relationship to BMI provides an interesting issue to consider when defining normal and excess BMI.

Table 24
Estimated Economic Costs of Overweight and Obesity by Condition

Condition	Case Count (millions)	Relative Risk	Base Rate	Total Cost (billions)	BMI \geq 25 Cost (billions)
Cancer	0.6	1.23	0.24%	48.0	\$7
Cardiovascular	26.8	1.30	8.15%	116.2	27
Diabetes	18.3	3.15	3.26%	53.8	37
Hypertension	45.8	1.79	13.35%	54	24
Kidney Disease					25
Stroke					7
Total					127

We have estimated the portion of the excess medical costs caused by overweight and the portion caused by obesity. The overweight but not obese ($25 \leq \text{BMI} < 30$) portion is \$38 billion and the portion caused by obesity ($\text{BMI} \geq 30$) is \$89 billion. Case count is the annual rate of new cases. *Source:* Medical Expenditure Panel Survey, Table 3: Total Expenses for Selected Conditions by Type of Service: United States, 2006, published online at http://www.meps.ahrq.gov/mepsweb/data_stats

⁵U.S.Renal Data System 2008 Annual Data Report

9.4. Medical Costs of Overweight in Children

The increase in medical costs associated with obesity occurs primarily at older ages. We have not found evidence of increased costs for children of a magnitude that would be significant in relation to the total for adults. Therefore, we have not included the effects of obesity of children in the above estimates. On the other hand, there is a clear relationship between juvenile obesity conditions that could lead to medical problems in the future. In addition, obesity of children tends to continue into the future, so the current increase in juvenile obesity can be expected to lead to even higher future medical costs if juvenile obesity is not reduced.

9.5. Effect of the Definition of Overweight on the Estimated Excess Medical Cost

As noted above, most of the analyses of excess rates of medical conditions are based on the definitions of overweight and obesity, which were based on population BMI rates in the 1980s, rather than on analyses of the level of BMI that starts increasing these conditions. The few papers that evaluate the effect of BMI below 25 indicate that BMI levels below the current overweight cutoff can have an adverse effect on these conditions. For example, Tsai et al. found a relative risk of diabetes equal to 1.57 for individuals with a BMI from 23 to 25 (Tsai et al., 2006). There is not enough data for these levels of BMI to support an accurate overall analysis, but it is clear an increase in relative risk at these levels would increase the estimated excess medical costs in two ways. First, the number of individuals with excess risk would be increased to include some individuals not currently considered to be overweight. Second, if the rate for lower BMI is even less than currently considered the base for a BMI less than 25, the relative risk for overweight individuals in relation to this lower rate would be more than is estimated in relation to the rate for a BMI less than 25. For example, if the result of the Tsai et al. paper is used to determine the portion of the population without excess risk and the relative risk of the remaining population, the excess cost of diabetes would be increased from \$31 billion to \$41 billion.

9.6. Overweight and Osteoarthritis

While the medical costs of osteoarthritis are much less than the costs of the other conditions we considered related to overweight and obesity, osteoarthritis is of interest because of the very specific relationship between osteoarthritis of the knee and BMI. Virtually all osteoarthritis of the knee is caused by body weight.

For individuals with a BMI of 20, the rate of osteoarthritis of the knee is 0.1 percent compared to the population average of 1.5 percent (Coggon et al., 2001; Hart and Spector, 1993). The relative risk of osteoarthritis of the knee for obese individuals is between 6 and 7 (Hart and Spector, 1993). This means that an obese individual has more than 60 times as much risk of having osteoarthritis of the knee as a thin individual with a BMI of 20. The total cost of surgery for osteoarthritis of the knee is about \$270 million in the U.S. and Canada. Approximately \$150 million of this amount is related

to obesity, and almost all of the rest is related to overweight or body weight in excess of a BMI of 20.

9.7. Obesity in Older Populations

We found several studies that focus on older age populations. The major findings on the impact of overweight on morbidities in Janssen's research for a 65 or older U.S. population are as follows (Janssen, 2007):

- The risks for arthritis and physical disability increased modestly in the overweight group.
- The risk for diabetes increased by 78 percent in the overweight group.
- Overweight and obesity were not associated with an increased risk for myocardial infarction.
- Overweight and obesity were not associated with increased risk for sleep apnea.
- Overweight and obesity were not associated with increased risk for urinary incontinence.
- Overweight was not associated with increased risk for cancer.
- Moderately elevated BMI may have a slight effect on physical disability risk.

Zablotsky and Mack (2004) studied BMI for U.S. women from large sample surveys taken in 1990 and 2000. The prevalence of obesity increased from 14.4 percent in 1990 to 21.7 percent in 2000. Prevalence of underweight declined from 3.1 percent to 2.4 percent. Factors related to obesity in older women are consistent with factors previously identified in younger women. The study is based on self-reported survey data, the Behavioral Risk Factor Surveillance System, which is thought to be reliable. The study identified diabetes with the question, "Have you been told you have diabetes?" This led to an odds ratio of 2.55 for obese women age 50–69 and 2.19 for women age 70 or older.

Wray et al. studied self-reported diabetes and weight loss for overweight healthy adults age 51–61 (Wray et al., 2004). They found that these middle-aged adults were 50 percent more likely to lose at least 10 pounds if they had been diagnosed with diabetes than if they had not. The weight loss was not predicted by other variables such as education or net worth.

Krueger et al. studied mortality associated with BMI for the U.S. elderly (60 and older) population (Krueger et al., 2004). They found that the general claims of the protective or harmful effects of obesity at older ages may be misguided. For example, normal weight, overweight and obese class I individuals have equivalent or lower risks of overall, cancer and respiratory disease mortality, but increased risks of circulatory disease mortality. Moreover, overweight and obese class I individuals have increased risks of diabetes mortality, relative to normal-weight individuals. These results are important, given the increased prevalence of obesity among the elderly. Circulatory disease, cancer and diabetes are the first, second and fourth leading causes of death, respectively, among those 65 and older.

9.8. Obesity and Insurance

We found relatively few research articles based on insurance data. In Section 7.3, we mentioned four of them (Niverthi and Ivanovic, 2001; Roudebush et al., 2006; Baldinger et al., 2006; Cumming and Pinkham, 2008). In addition, there are several insurance articles that survey research and discuss implications for insurance. For example, the reinsurer reports (Eng, 2004) and (Hopkins and Bright, 2005) show that life and health insurers take obesity seriously and are trying to get this information to clients. However, these are descriptive articles; they do not model or quantify the impact of obesity and overweight trends on life and health insurance.

The Eng et al. (2005) article is a very interesting comparison of public and insurance industry responses to obesity and smoking, based on peer-reviewed articles, news sources and government publications. However, the article itself is intended for insurance industry and public officials; it is not peer-reviewed and it presents no new scientific results or models.

The Hopkins and Bright article appears in a Hanover Re in-house publication, which is not peer-reviewed. Nevertheless, the article is a very good review of scientific papers on BMI and its relation to life insurance, critical illness and disability income risks. There are no new results or models; however, they do describe how BMI is used in underwriting. For example, they write that standard rates for life, critical illness and disability insurance are not available for applicants with $BMI \geq 30$. Applications for life insurance with $BMI \geq 40$ are declined and applications for critical illness and disability insurance with $BMI \geq 45$ are declined. They also note that, while obesity is more prevalent among lower socio-economic groups, its prevalence is significant in typical insurance applicant groups.

In a report published by Swiss Re, Eng discusses the impact of obesity on life insurance. It is a thorough and convincing presentation of increased mortality due to overweight and obesity, based on peer-reviewed articles and government reports. This is a survey paper; it does not present new results or models. However, it explains very well the impact on the life insurance industry (as of 2004), including this important point: Not only is the prevalence of obesity increasing in insured populations, the relative mortality risk is higher in insured populations than in the general population. The increasing obesity trend could reduce the profitability of existing life insurance business. Evidently, the trend has an impact on disability profitability also. In 2004, Unum Provident, a large U.S. insurer, reported a tenfold increase in obesity-related, short-term disability claims over the preceding decade (Unum, 2004).

Hartwig and Wilkinson (2004) give an overview of obesity and its impact on health insurance, life insurance, disability insurance, workers compensation, liability and excess casualty insurance. It has an excellent discussion of obesity litigation, including the example of food-related advertising on children's television. They also discuss the difference between "fast food" litigation and tobacco litigation. It is very interesting, but there are no new research results.

Gutterman (2008a) wrote a monograph that synthesizes the research on the prevalence and impacts of obesity and related factors on the future health of the population, with an emphasis on the United States. He summarized this monograph in a brief article

(Gutterman, 2008b). The monograph describes future obesity trends and the resulting impact on mortality, morbidity, disability, health care costs and quality of life. His conclusions are somewhat pessimistic. He warns against “a simple extrapolation of recent overall mortality trends” without taking into account changes in human behaviors (eating more fat, exercising less). This is an excellent discussion of the literature and future possible implications, but there are no new research results.

9.9. What to Do?

A recent preliminary study by Bleich et al. (2008) tries to answer the question “Why is the developed world obese?” by studying the relative contribution of increased caloric intake and reduced physical activity. The introduction of the paper has interesting data on trends and levels of obesity in various developed countries. They offer these summary conclusions:

- The rapid increase in obesity across the developed world suggests a common cause.
- Increased caloric intake is primarily responsible for adult weight gain in developed countries.
- The shift toward increased caloric intake is associated with technological innovations such as reduced food prices as well as changing socio-demographic factors such as increased urbanization and increased female labor force participation.
- Efforts should be made to reduce consumption and encourage low-calorie diets.

In a recent JAMA editorial, Gregg and Guralnik (2007) stated: “Disability represents in part the collective effects of multiple obesity-related conditions, which bodes poorly for any simple clinical or public health solutions to modify the obesity-associated disability trends. This challenge is compounded by the lack of commonly practiced interventions directly aimed at reducing disability in at-risk populations. Structured exercise and weight-loss programs may be among the most promising unifying interventions, because they appear to help prevent type 2 diabetes, reduce arthritis symptoms, and improve physical functioning – i.e., they can reduce each of the outcomes of obesity that have persisted over time. In the end, however, reducing the effect of obesity on morbidity by simply altering its course or accommodating its presence may never have an impact equal to a successful public health strategy to prevent obesity.”

Several sources recommend improving diet and increasing physical activity. However, according to a study by Bauman et al. (2008), population-effective weight-loss and weight-maintenance intervention requires more than leisure-time physical activity. They recommend serious efforts be made to promote active commuting, work-related activity, active living and domestic incidental energy expenditure. They found that in China, increases in obesity are clearly related to increasing sedentary lifestyles. They recommend using public transportation, limiting car use, designing cities appropriately and offering socioeconomic incentives to engage in everyday leisure time activities. James (2008) also discussed the rise of overweight and obesity in China. He asserted policymakers do not understand that the obesity epidemic is a normal population response to reduction

in the demand for physical activity and major changes in the food supply, which is seen clearly in China. He concluded the study with this: “How can the community of doctors and others concerned with nutrition and physical activity engage the policy makers at the highest government level? It is probably only at this level that major reforms can be made.”

According to Mehrotra et al. (2005), there are few population-based studies of weight-loss surgeries in the United States, despite the rapid adoption of weight-loss surgery. No study has assessed the economic costs and complications associated with weight-loss surgery. Mehrotra et al. used Wisconsin hospital discharge data to examine the trends, costs and complications for all types of weight loss surgery from 1990 to 2003. From 1990–92 and 2000–02, the total number of weight-loss surgeries increased by 600 percent, from 269 to 1,884. Total costs of weight-loss surgeries in Wisconsin were \$3.3 million in 1990-92 and \$44.5 million in 2000–02.

Anderson et al. (2007) reported on a long-term follow-up of 118 obese patients, arguing that intensive behavioral intervention can be very effective. Brownell and Frieden (2009) proposed a tax on sugar-sweetened beverages. Fitzner et al. (2006) suggest a managed-care approach to treating obesity.

Several states have reported on obesity, its impact on health care, and programs to encourage better diets and more physical activity. Alaska (Alaska, 2003), California (California, 2005), Georgia (Georgia, 2007), Louisiana (Louisiana, 2004) and Texas (McCusker et al., 2004) published results on prevalence of obesity, its relationship to health care and other costs, and the states’ responses. Washington established an Obesity Program in its Department of Health & Social Services (Washington, 2007). In 2003, Georgia initiated a program to prevent obesity and related chronic diseases through improved nutrition and physical activity (Powell, 2007).

Roux and Donaldson (2004) argued that the abundance of studies on cost of illness merely confirmed that obesity was a serious societal issue, but that those studies did not lead to remedial policies. They asserted that an appropriate role of economics would be to focus on formal evaluation of different strategies for the prevention and treatment of obesity.

Flynn et al. studied hundreds of articles and reports on childhood obesity to make a synthesis of evidence of best practice recommendations to funding agencies, program developers, agencies serving children and governments (Flynn et al., 2006). We do not have space to discuss all of this very thorough study. Here are key recommendations for funding agencies:

- Develop obesity prevention and treatment programs for critical population subgroups currently being underserved: immigrants, children younger than 5 and males.
- Develop obesity prevention programs with rigorous evaluation components in the home and community settings.
- Develop population-based obesity interventions to balance, support and extend current emphasis on individual-based programs.

- Implement long-term follow-up to determine program impacts as relates to normal body weight. For agencies serving youth, they say that intervening to prevent obesity is worthwhile. They encourage schools to increase physical activity and healthy eating.

According to Hill et al., recent efforts to address population obesity have been unsuccessful because they focus on individual interventions to prevent or treat obesity through individual changes in diet and physical activity (Hill et al., 2008). They propose prevention as an alternative to obesity treatment over a very long term, by first stopping the increasing obesity rates. Gradually, over a few generations, reduce levels to those of previous decades. The America On the Move initiative was started to translate their theory into practice. America On the Move promotes small changes to the environment to help support and sustain the small behavior changes.

Cohen et al. recommend that future interventions to control weight address the social environment at the community level (Cohen et al., 2006). Their study supports the need to re-conceptualize the obesity epidemic to include group-level phenomena in addition to individual choice and free will.

As part of his monograph, Gutterman (2008a) surveyed articles on obesity management and prevention, with somewhat pessimistic results. He found that attempts to decrease obesity which rely on changing individual behavior have been “pretty much ineffective.” In addition, he said there seems to be no single best approach, and proposes a combination of two big approaches, diet and exercise, with others such as education and treatments such as bariatric surgery and drugs.

10. Summary

10.1. Prevalence of Obesity

Obesity has become a world wide epidemic. Almost 10 years ago, the U.S. Surgeon General declared that the United States was experiencing substantial increases in overweight and obesity. While there is recent evidence the rate of increase in obesity is slowing, the current prevalence of BMI ≥ 30 is now approximately 30 percent of the population of the U.S. (CDC, 2008). The prevalence in Canada was 23.1 percent (Tjepkema, 2006). Similar increases in obesity have occurred in many countries in Europe and Asia.

10.2. Impact of Obesity on Disease and Mortality

Perhaps the most convincing results concern the effect of overweight and obesity on cardiovascular disease and diabetes. Relative to normal weight, the relative risk of death from CVD increases significantly for overweight men and women (Balkau et al., 2007; Calle et al., 1999; Oster et al., 2000). About 60 percent of diabetes is directly related to weight gain (Runge, 2007). As of 2008, studies showed about 246 million adults world wide have diabetes, and the figure is increasing (Lancet, 2008).

Several papers provide empirical evidence that obesity is significantly related to increased risk for certain cancers (Renehan et al., 2008b). The relationship is complex

and depends on the site of the cancer. The American Institute for Cancer Research is funding examination of how fatty acids affect insulin-like growth factors, which may be linked to heart disease, diabetes and various cancers (AICR, 2006).

Overweight and obesity are significantly related to a variety of negative effects on the body, such as delayed healing of joint injuries, increased risk of arthritis, impaired function of internal organs, and interference with hormone balances. For example, one study showed that more than 50 percent of osteoarthritis of the knee is due to obesity (Coggon et al., 2001). The negative effects play a role in a cycle of overweight, depression and decreasing physical activity, which results in increased use of the health care system (Rosemann et al., 2008). Increasing BMI also is related to development of asthma (Luder et al., 2004) and renal disease (Hsu et al., 2006).

Many empirical studies found that obesity significantly increases the risk of death, that is, all-cause mortality (Whitlock et al., 2009; Calle et al., 1999; Gu et al., 2006; Flegal et al., 2005). The relationship between BMI and all-cause mortality is often found to be U-shaped or J-shaped, since underweight also is associated with increased mortality. The few studies of insured groups generally agree with population results (Niverthi and Ivanovic, 2001; Roudebush et al., 2006; Baldinger et al., 2006; Cumming and Pinkham, 2008). One article has relatively optimistic conclusions based on a very detailed stochastic model with parameters based on the Framingham data (Chatterjee et al., 2008).

10.3. Economic Cost Conclusions

The total economic cost of overweight and obesity we have estimated for the United States and Canada is the sum of excess medical costs and the cost of the loss of productivity caused by excess disability and mortality due to overweight and obesity. As shown in Table 24, the total cost of excess medical care caused by overweight and obesity was estimated at \$127 billion for 2009. The economic loss of productivity caused by excess mortality is estimated at \$49 billion per year in the United States and Canada, as explained in Section 7.5. The economic loss of productivity caused by disability is estimated at \$43 billion per year for active workers, as discussed in Section 8.2. In the same section, we discuss the costs for workers unable to work at all because of disability. We estimated the economic loss of productivity caused by overweight or obesity for totally disabled workers at \$72 billion. In summary, the total economic cost of overweight and obesity in the United States and Canada caused by medical costs, excess mortality and disability is approximately \$300 billion per year. The portion of this total due to overweight is approximately \$80 billion, and approximately \$220 billion is due to obesity. The portion of the total in the United States is approximately 90 percent of the total for the United States and Canada.

A. Relative Risk, Hazard Ratio and Odds Ratio

Analyses of the effects of BMI on health status use three different measures of the effect of BMI; relative risk, hazard ratio and odds ratio. Relative risk is the ratio of the frequency of a given condition for a given group to the frequency for the base or referent group. For example, if people with normal weight have a frequency of 3 percent for diabetes, and obese people have a frequency of 12 percent, then the relative risk for obese individuals would be 12 percent/3 percent = 4. Hazard ratio is the limit of relative risk as the time period approaches zero. For chronic conditions, relative risk and hazard ratio are the same. For acute conditions, the relationship between relative risk and hazard ratio depends on the duration of the medical events. For example, suppose the probability of a given short-term medical event for individuals with normal BMI is 1/365 per day, and that the hazard ratio for overweight and obesity is 2. Suppose that when the condition occurs, it lasts for one month. Suppose, further, that the Poisson distribution applies to the incidence of the event when the individual is healthy. Then the expected number of cases per year for normal weight individuals would be close to 1 (actually 0.92), but the expected number for overweight and obese individuals would be less than two times the probability of 1 for the nonoverweight individuals (actually $1.73 = 1.9 \times 0.92$), because there would only be an 11-month healthy period for the second occurrence, and less time for higher numbers of occurrences. For the conditions considered in this study, the difference between the results of relative risk and hazard ratio is either zero or small enough that using hazard ratio as relative risk is an acceptable approximation.

On the other hand, numerous articles use the odds ratio to measure the effects of BMI. Odds ratio is the ratio of the odds of having a given health problem for people in a given group to those in the referent group. For example, if one-fourth of people with normal weight have hypertension, the odds are 1-to-3, i.e., one person with hypertension compared to three people without hypertension. If half of the obese individuals have hypertension, then their odds are 1-to-1. The odds ratio in this case would be $1 : 1/1 : 3 = 3$, in other words, one divided by one-third. There is a direct relationship between relative risk and odds ratio, but the relationship uses the value of the base rate, which is the rate of the normal population having the given condition. The formulas providing these relationships are as follows:

$$\begin{aligned} \text{Relative Risk} &= \frac{\text{Odds Ratio}}{1 - \text{Base Rate} + \text{Odds Ratio} \times \text{Base Rate}} \\ &= \frac{\text{Odds Ratio}}{1 + \text{Base Rate} \times (\text{Odds Ratio} - 1)} \\ &= \frac{\text{Odds Ratio}}{1 - \text{Base Rate} \times (1 - \text{Odds Ratio})} \end{aligned}$$

and

$$\text{Odds Ratio} = \text{Relative Risk} \times \frac{1 - \text{Base Rate}}{1 - \text{Relative Risk} \times \text{Base Rate}}$$

The base rate can be determined from the total percentage rate with a given condition combined with the percentage of obese individuals in the population and either the relative risk or odds ratio. The formula for determining base rate in relation to relative risk is as follows:

$$\begin{aligned} \text{Base Rate} &= \frac{\text{Population Rate}}{1 - \text{Obese Percentage} + \text{Obese Percentage} \times \text{Relative Risk}} \\ &= \frac{\text{Population Rate}}{1 + \text{Obese Percentage} \times (\text{Relative Risk} - 1)} \\ &= \frac{\text{Population Rate}}{1 - \text{Obese Percentage} \times (1 - \text{Relative Risk})} \end{aligned}$$

The formula based on odds ratio is more complex, because it involves the solution of a quadratic equation. To determine the base rate on the basis of odds ratio, first determine the following three values:

$$\begin{aligned} A &= (1 - \text{Obese Percentage}) \times (\text{Odds Ratio} - 1) \\ B &= 1 + (\text{Odds Ratio} - 1) \times (\text{Obese Percentage} - \text{Population Rate}) \\ C &= -\text{Population Rate} \end{aligned}$$

Then

$$\text{Base Rate} = \frac{-B + \sqrt{B^2 - 4AC}}{2A}$$

Because of the more simple formulas derived from the use of relative risk, we have used relative risk for the determinations in this paper, and have converted odds ratios to relative risk. This has no effect on the numerical results for frequency and cost of medical conditions.

B. Cost Calculation Procedures for Medical Care

We have estimated the cost to society of the effects of obesity on health problems and mortality by combining three components. These are the additional costs of medical care, the loss of productivity caused by disability and the loss of productivity caused by increased mortality. This appendix summarizes the methods we used to calculate these estimates.

The cost of excess medical care is based on the application of the average cost per case times the number of excess cases. This is based on an assumption that the average cost for cases caused by overweight or obesity is similar to the average cost of other cases. This seems reasonable on the basis of the fact that overweight and obesity cause some

existing cases to become more serious, but also cause a particular medical condition in individuals who would not otherwise have the condition and consequently may be less serious than average. When obesity increases the rate of occurrence of a condition, this is an overall evaluation, and it may not be possible to identify particular cases specifically caused by obesity. Therefore, it may not be possible to identify specific costs. In our literature review, we did not find any studies that provided this information.

For each article that identified the rates of increase in a medical condition for BMI groups, we combined the results to obtain a population total. For example, the paper of Fu et al. (2008) found relative risks for cardiovascular conditions of 1.52, 1.71, and 2.04 for the BMI groups 24–27, 27–30, and 30+, respectively. The population percentages for these groups are 22.25 percent, 19.08 percent, and 30.78 percent respectively, resulting in a population relative risk of 1.57. The population relative risks from the various articles were combined by determining a weighted average based on the square-root of the size of the sample count for each article. In this case the overall relative risk was approximately 1.30. This means that 23.06 percent (i.e., $(1.30 - 1)/1.30$) of the population cases result from overweight or obesity. We estimate the total population medical cost of cardiovascular conditions at \$116.2 billion in 2009, which means the cost related to obesity is 23.06 percent of \$116.2 billion, or \$26.8 billion. In retaining three- or four-digit numbers in these calculations, we do not intend to indicate that the results are accurate to within a tenth of 1 percent. In fact, the results may be uncertain by 10 percent or more, but we have chosen not to round results until the end, so that the effects of rounding can be easily seen on an overall basis.

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