

Obesity: Status and Effects

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1. Executive Summary

Body fat historically enabled people and animals to survive food shortages by tapping into their internal energy reserves. Today, abundant calorie-rich food often overwhelms the body's weight regulatory system, with many individuals' genetic makeup unable to control this input, resulting in massive societal weight gains. The current era of obesity and inactivity is threatening the substantial progress made in postponing illness and death and with its reduction in cardiovascular disease made possible in part because of a decrease in smoking.

This paper first explores the current state and contributing causes of obesity. It then explores the mortality and health care costs of obesity. It ends with a discussion of its management.

The increase in obesity over the past 30 years has been a result of changes in the environment that have simultaneously lowered the cost of food production, lowered the time and monetary cost of food consumption, increased the real cost of being physically active, and decreased the perceived cost of the adverse consequences of habits that add weight. These changes have in part been in response to a demand for labor-saving technology and desire for convenient, affordable food.

The resulting prevalence of obesity (for adults, a greater than 30 body mass index, or BMI) of over one-third of the adult population in the United States should be a significant and growing concern to society. Not only has average weight increased, but the percentage in excess of any given weight level has increased in all age and ethnic categories, reflecting a shift in the prevalence distribution itself, as the fastest growing weight segment of population has been the morbidly obese, associated with more extreme mortality and health care cost effects. The trend in children's obesity is also particularly troubling, as it will likely add more challenges as they age.

Even though what seemed to be a never-ending rise in the percent of overweight and mildly obese appears to be stabilizing somewhat in the United States, the current level for all population segments is staggering. Unfortunately, the rest of the world is, at different speeds and degrees, catching up to Americans, although the severely obese is where Americans stand out from others so far.

Although several studies have placed a spotlight on a possible obesity paradox, where the mortality of those overweight or moderately obese appears somewhat better or not significantly worse than that of those with a "normal" BMI, many other studies have shown that, particularly given a long-term lag period subsequent to measurement, mortality increases with growing BMI. As result, although being overweight and moderately obese constitutes a significant mortality risk remains somewhat controversial, the obese are certainly associated with greater health care costs and those morbidly obese represent both a growing percent of the population and a group with significantly greater mortality and health care costs. In addition, whether those in the huge overweight population segment are looked at as (1) being at-risk of becoming obese, (2) experiencing adverse mortality prospects itself, or (3) experiencing significant additional morbidity and health care risks and costs, this population segment constitutes a societal concern as well.

Obesity has been a significant contributing factor to the continuing increase of U.S. health care costs. Although some earlier studies relating to health care costs indicated that obesity-related health care expenditures have been between 5 and 7 percent of annual health care expenditures in the United States, two recent studies have estimated that as much as 9.1 percent or 16.5 percent of total health care costs can be attributed to being overweight or obese.

The last section of this paper that describes what might be done for the prevention and management of obesity highlights that there is no silver bullet to solve the “problem” of obesity. Since being obese is commonly the result of or lack of healthy behaviors, the challenges of reducing the incidence of obesity have to be addressed on a personal, business and societal/government level to succeed. Since it is normal to have other risk factors in addition to being obese, action is needed to address more than just weight.

2. Issue Background

The effects of human behavior and changes in this behavior have significant effects on all aspects of life, including mortality and morbidity, as well as its quality. This paper focuses on the consequences of obesity and the two primary risk factors that have contributed to its unprecedented increase, nutrition and physical activity. Obesity is an excessive accumulation of fat (adipose tissue) that can impair health.

The so-called “obesity epidemic” has been underway for more than 30 years, with significant adverse health ramifications. This has been particularly troubling because all segments of the population have on average gained weight, with an especially large percentage increase in those more severe obese categories with its correspondingly large financial and personal impact.

The objective of this paper is to provide a synthesis of observed trends and results of recent studies regarding the effects of obesity and related factors on the future health of the population, with an emphasis on that of the United States. The growth and breadth in the number of studies and literature on this topic has more than matched the underlying trends in obesity. What both complicates and makes this issue fascinating are the inconsistent findings reached by these studies and the difficulty in confirming causal relationships among risk factors and with personal and societal costs that in turn can be important in designing mitigation and public policy strategies that have, at least so far, experienced limited success, at best.

The fundamental causes of the increase in obesity are rooted in the nature of current western culture, in which incentives exist to live a significant portion of life in a sedentary manner and to consume a high-fat, energy-dense diet, while spending an ever smaller share of income on food. Although historically people were mostly consumed by simply obtaining food, people now think more about how to enjoy it as a result of the ability of choosing their diet. The results of these trends have and will likely continue to result in profound changes in society and in personal behavior patterns throughout the world.

For many people, being overweight or obese primarily results from a combination of excess calorie consumption and/or inadequate physical activity, consistent with the following fairly obvious formula:

$$\text{Current Weight} = \text{Previous Weight} + \text{Energy Inputs} - \text{Energy Outputs}$$

Despite this simple equation, the factors involved are numerous, wide-ranging, complex and interrelated. Genetic factors have been found to affect and predispose individuals to becoming obese; for example, more than 250 genes have been linked to obesity and more will likely be found. Possibly between 40 and 50 percent of the incidence of obesity have genetic origins; there can be genetic defects involved, but these are relatively rare. For genetically susceptible individuals, both conveniently available inexpensive energy-dense food and reduced opportunities for energy expenditure have contributed to the increased levels of obesity. However, just as—if not more—important are the factors involved in changes over the past 30 years, which point to a major role played by environmental, lifestyle and nutritional changes and

behavioral shifts. These factors, possibly interacting with genetic susceptibility, can contribute directly to an individual's weight and also can contribute to future chronic disease. Economic growth, urbanization, globalization of food markets and ineffective personal control mechanisms all have and will affect personal health around the world, the inevitable result of our modern post-industrial and post-agricultural world.

Since the late 20th century, the best metric to measure obesity has been somewhat controversial. Body mass index (BMI) that relates weight to height, with standardized categories recommended by the World Health Organization (WHO) is now used worldwide, in part because its convenience. See the appendix for the correspondence between weights and heights to BMI. The controversy arises in part because weight only represents one dimension of body composition, albeit an important one, as relevant body composition consists of many inter-related factors. The relatively easy-to-measure BMI should only be viewed as one such metric, as it only captures certain characteristics of weight and adiposity, ignoring such factors as fat content and how adiposity tissues are carried in a person's body. For example, in some cases a person who is obese according to BMI and physically fit might be in better overall health than someone else who is of normal weight but not physically fit. In any case, it is considered better to be fit and lean, rather than unfit and fat.

BMI categories include overweight and obese. Overweight for an adult is defined as having a BMI of between 25.0 and 29.9, while an adult is considered to be obese with a BMI of 30.0 and greater. Because of recent overall gains in weight, the obese category is now sometimes split into class I obese with a BMI of 30.0 to 34.9, class II obese with a BMI of 35.0 to 39.9, class III obese with a BMI of 40.0 to 44.9, class IV obese with a BMI of 45.0 to 49.9, and class V obese with a BMI of 50.0 and upwards. "Normal" weight is usually indicated by an 18.5 to 24.9 BMI, with anyone at a BMI less than 18.5 being considered "underweight."

Although these cutoffs have been applied to the total population, they do not represent equivalent health risk categories for all population segments. For example, Asians, particularly those from Southeast Asia, may have a lower cutoff for the equivalent effect of overweight and obesity, while for older individuals (e.g., over 75), higher cutoffs might better characterize equivalent health conditions.

Prevalence rates of obesity typically increase with age, generally peaking in the 50s or 60s, followed by a decrease at older ages. The typical American gains between 22 and 33 pounds between ages 20 and 50; this "natural" increase is not due to caloric intake, as nutrition surveys indicate that caloric input actually declines with age. Increasing obesity until the 60s is not explained by increases in fat-free mass, as bone mass peaks around age 30 and muscle mass plateaus and later declines without strengthening exercises. The changes in body weight and body composition are attributable, in part, to the natural declines in certain hormones, as well as changes in metabolism. Although in some age categories in tables 1 and 2, the largest prevalence is at ages 60 and older, the percent of obese or overweight decreases considerably at ages 70 and older compared with ages 60 to 69; the percent of obese at ages greater than 80 can be about half that of ages 60 to 79.

The data from tables 1 and 2 are derived the National Health and Nutrition Examination Survey (NHANES), a nationally representative survey with professionally measured height and weight, while Table 3 is based on the National Health Interview Survey (NHIS) that relies on self-assessed height and weight. BMIs generated from self-assessments are generally lower than those based on professional measurement, thus the values in Table 3 are lower than Tables 1 and 2. Although self-reported obesity prevalence is smaller than those based on professional measurement, the trend based on the NHIS survey has continued to increase, although at a somewhat slower rate than in earlier periods.

Table 1 shows the very different obese prevalence rates by age, gender and ethnic group, with black females exhibiting the highest percent of obesity. Based on NHANES (2007-08), about 34 percent of the adult population is obese. The most noticeable increase is in the morbidly obese, as shown in Table 2. Based on prevalence rates in Table 1 and the 2000 population of American adults, there are more than 75 million obese and 15 million morbidly obese adults in the United States, with about 150 million adults either obese or overweight. Note that several of the tables and papers used as sources for this paper include more specific data regarding Mexican Americans, a more homogeneous population category, than the total Hispanic population; on the whole, they have a slightly higher average BMI than that of Hispanics as a whole.

TABLE 1
Adult Obesity (BMI >30) as a Percent of U.S. Population
1999-2000 Compared with 2007-08

1999-2000 Ages	Males				Females			
	All	White (nonH)	Black (nonH)	Mexican American	All	White (nonH)	Black (nonH)	Mexican American
20-39	23.7%	22.0%	27.4%	30.4%	28.4%	24.4%	46.2%	30.6%
40-59	28.8	28.5	29.9	27.0	37.8	34.2	53.2	48.5
≥ 60	31.8	34.3	26.4	29.7	35.0	33.3	50.2	41.0
Total Age-Adjusted	27.5	27.3	28.1	28.9	33.4	30.1	49.7	39.7
2007-08 Ages								
20-39	27.5	26.3	34.7	33.8	34.0	31.3	47.2	39.6
40-59	34.3	34.0	39.7	38.2	38.2	35.7	51.7	48.9
≥ 60	37.1	38.4	38.0	35.8	33.6	31.4	50.5	48.1
Total Age-Adjusted	32.2	31.9	37.3	35.9	35.5	33.0	49.6	45.1

Source: National Health and Nutrition Examination Surveys, Flegal (2010); “nonH” refers to non-Hispanic.

TABLE 2
2007-2008 Adults as a Percent of U.S. Population
Overweight and All Obese (BMI >25), Class II Obese (BMI >35) and
Class III Obese (BMI >40) Obese

Overweight and Obese Ages	Males				Females			
	All	White (nonH)	Black (nonH)	Mexican American	All	White (nonH)	Black (nonH)	Mexican American
20-39	63.5%	62.6%	61.5%	75.0%	59.5%	54.9%	78.0%	70.3%
40-59	77.8	77.7	73.5	88.0	66.3	63.8	78.4	80.3
≥ 60	78.4	81.4	72.5	75.8	68.6	67.6	78.2	82.6
Total Age-Adjusted	72.3	72.6	68.5	80.0	64.1	61.2	78.2	76.9
Class II+ Obese Ages								
20-39	9.4	8.5	14.2	12.5	18.9	17.2	30.2	20.9
40-59	11.6	11.6	13.8	13.8	19.5	18.7	28.1	19.0
≥ 60	11.6	12.0	15.5	9.8	13.3	12.3	22.0	19.6
Total Age-Adjusted	10.7	10.5	14.4	12.4	17.8	16.6	27.9	19.9
Class III+ Obese Ages								
20-39	4.2	3.4	7.5	7.0	7.6	6.8	15.0	6.8
40-59	4.2	4.4	5.6	3.7	8.4	7.3	17.7	5.9
≥ 60	4.2	4.4	8.2	N/A	4.7	4.1	7.2	7.6
Total Age-Adjusted	4.2	4.0	7.0	4.4	7.2	6.4	14.2	6.7

Source: National Health and Nutrition Examination Surveys, Flegal (2010); “nonH” refers to non-Hispanic.

TABLE 3
Prevalence of Obesity Among Adults Ages 20 and Over on a Self-Reported Basis

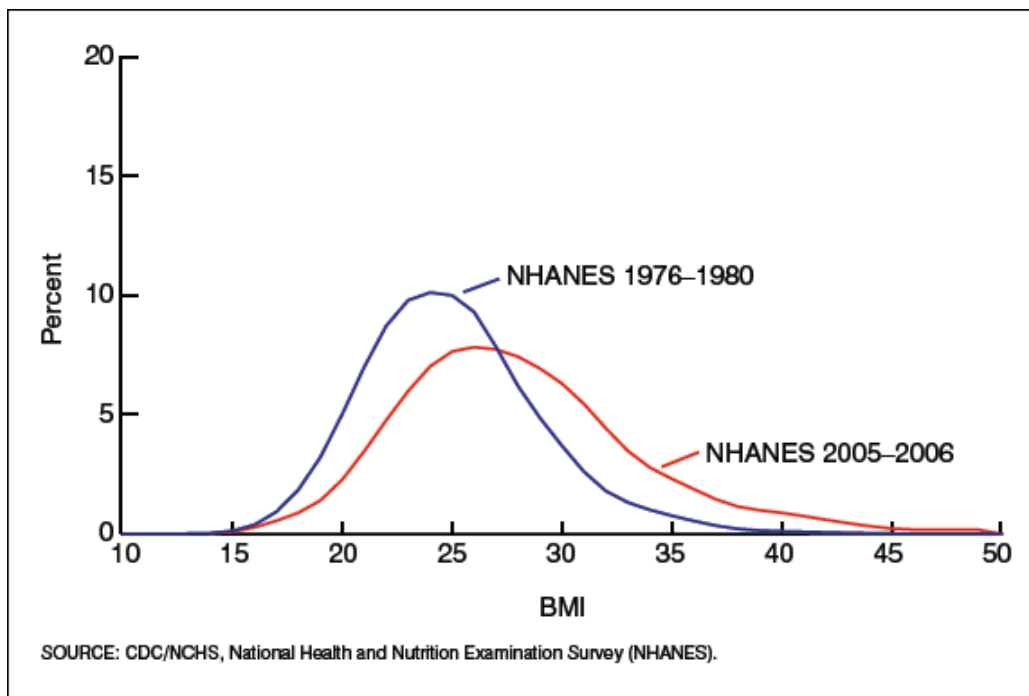
Year	Age-Adjusted
1997	19.5%
1998	20.6
1999	21.5
2000	21.8
2001	22.9
2002	23.8
2003	23.5
2004	24.3
2005	25.3
2006	26.2
2007	26.6
2008	27.5
2009	27.9

Source: National Health Interview Survey (NHIS) self-reported weights and heights.

Among states participating in the Behavioral Risk Factor Surveillance Survey (BRFSS) that uses self-reported weights and heights, in 1990, 10 states had a prevalence of adult obesity less than 10 percent and no states had a prevalence rate equal to or greater than 15 percent. By 1998, no state had a prevalence rate less than 10 percent, seven states had a prevalence of adult obesity equal to or greater than 20 percent, and no state had a prevalence rate equal to or greater than 25 percent. In contrast, in 2009, only one state had an average prevalence rate of adult obesity less than 20 percent (Colorado) and nine had a prevalence rate equal to or greater than 30 percent (Alabama, Arkansas, Kentucky, Louisiana, Mississippi, Missouri, Oklahoma, Tennessee and West Virginia). The large over-representation of obesity prevalence in Southern states is positively correlated with relatively high levels of diabetes and hypertension and low levels of physical activity.

Possibly more important than the shift to the right in the average BMI prevalence curve is the shift in its distribution, particularly among the morbidly or significantly obese (class II and greater), as can be seen in the curve shifts in Figure 4 as well as in the second chart in Figure 9. Increasingly Americans are becoming a bipolar society: fit thin and inactive fat. Although admittedly a generalization and most likely unfair, you rarely see someone obese running in the park and the fattest among us are not the ones that eat salads for lunch.

FIGURE 4
Changes in the Distribution of BMI of U.S. Adults
Between 1976-80 and 2005-06



For children, a somewhat different weight benchmark is now in common use. Obese refers to a BMI at or above the 95th percentile for the 2000 Centers for Disease Control and Prevention (CDC) Growth Chart for the United States for the applicable gender and age, while

overweight refers to a BMI between the 85th and 95th percentile of this chart. For youth, a history of the upward trend over the past 30 years is shown in Table 5 and Figure 7.

TABLE 5
Trends in Prevalence in Obese U.S. Children and Adolescents
Between 1971 and 2008

Age (Years)	NHANES I 1971-74	NHANES II 1976-80	NHANES III 1988-94	NHANES IV 1999-2000	NHANES 2001-2002	NHANES 2003-2004	NHANES 2007-2008
2-5	5.0%	5.0%	7.2%	10.3%	10.6%	13.9%	10.4%
6-11	4.0	6.5	11.3	15.1	16.3	18.8	19.6
12-19	6.1	5.0	10.5	14.8	16.7	17.4	18.1

Source: National Center for Health Statistics and Ogden (2010), >95th percentile CDC 2000 growth chart.

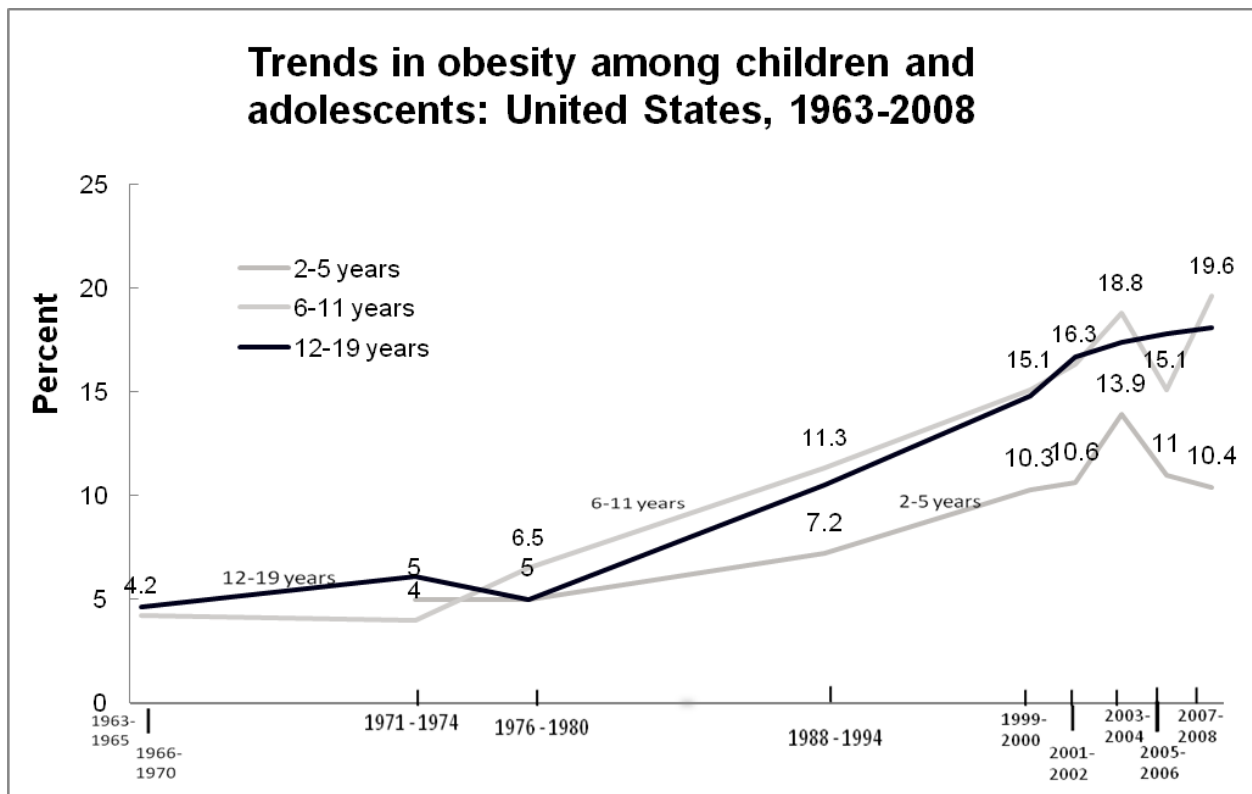
For youth, an age/ethnic group/gender comparison from the NHANES 2007-2008 is shown in Table 6. Particularly noteworthy are the high obesity prevalence rates for Mexican American boys and non-Hispanic black adolescent girls.

TABLE 6
Percentage Prevalence in Obese U.S. Children and Adolescents by
Age/Ethnic Group/Gender in 2007-08

Ethnic Group	Gender	Ages 2-19	Ages 2-5	Ages 6-11	Ages 12-19
Mexican-American	Boys	24.9%	19.3%	27.1%	26.8%
	Girls	16.5	7.5	22.3	17.4
	All	20.8	13.7	24.7	22.2
White	Boys	15.7	6.6	20.5	16.7
	Girls	14.9	12.0	17.4	14.5
	All	15.3	9.1	19.0	15.6
Black	Boys	17.3	11.4	17.7	19.8
	Girls	22.7	11.1	21.1	29.2
	All	20.0	11.7	19.4	24.4
All	Boys	17.8	10.0	21.2	19.3
	Girls	15.9	10.7	18.0	16.8
	All	16.9	10.4	19.6	18.1

Source: National Center for Health Statistics and Ogden (2010), >95th percentile CDC 2000 growth chart.

Figure 7

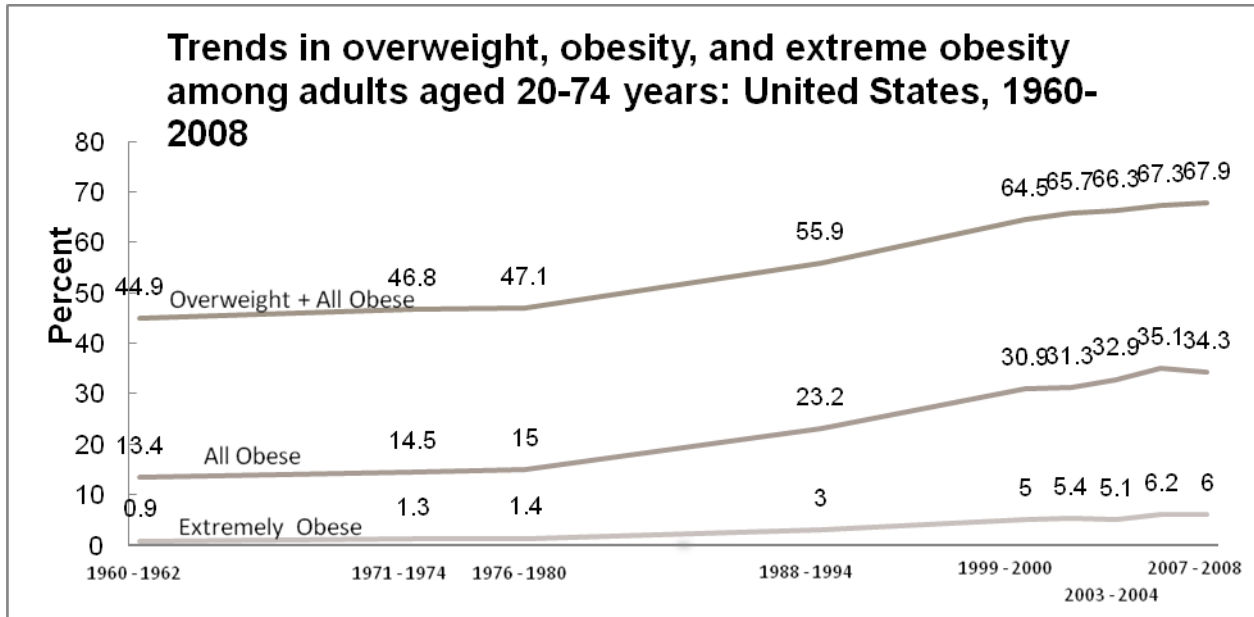


Source: National Center for Health Statistics/CDC National Health and Nutrition Examination Surveys.

Although most studies have incorporated obesity in terms of BMI, other studies have found that other weight-related metrics are just as good, if not better, for measuring obesity. For example, waist circumference and waist-to-hip ratios have been found in some studies to be better predictors of adverse health results, particularly in certain population segments such as those older than 70, in whose bodies central adiposity may be more of a health concern than overall weight.

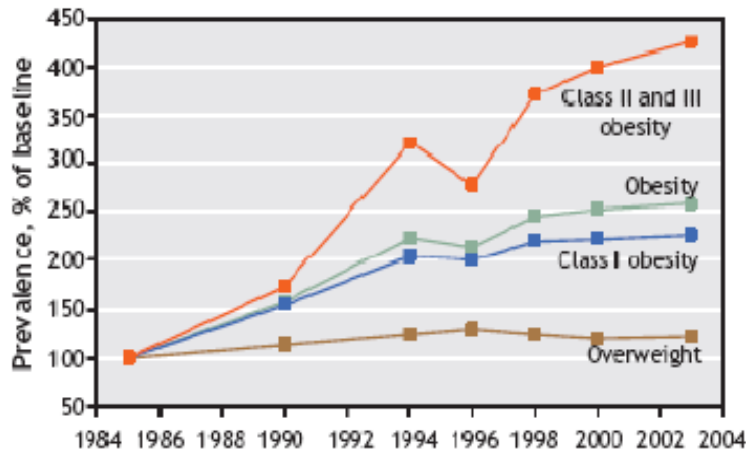
The dramatic growth in obesity prevalence rates since the 1970s seems to be tapering off. Although there remains some room for further deterioration, the limit to growth in overweight and obesity for both adults and children does not seem far away. In contrast, the percentage of those who are class II and III obese continues to increase. According to NHANES (2007-08), the prevalence of class II and III obese adults was 14.3 percent, with women having higher prevalence (17.8 percent) than men (10.7 percent), and non-Hispanic blacks having a higher prevalence (21.9 percent) than non-Hispanic whites (13.6 percent), with non-Hispanic black women having the highest prevalence of 27.9 percent. Unfortunately, the ultimate level of the morbidly obese does not yet appear to have been reached.

Figure 8



Source: National Health and Nutrition Examination Surveys.

Figure 9
Adult Overweight and Obesity Trends



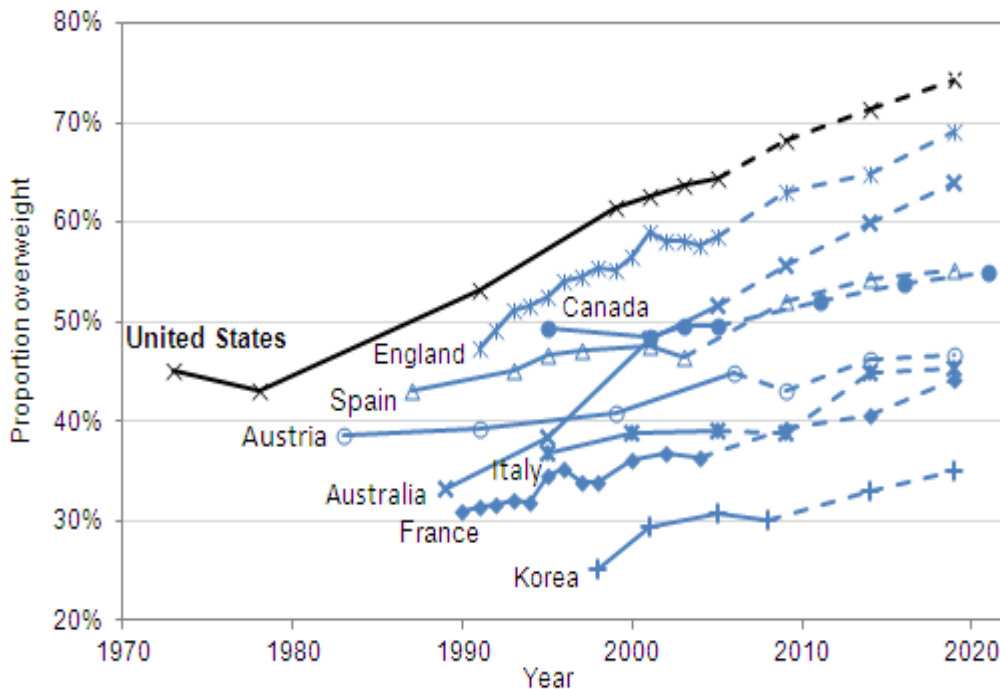
Source: National Health and Nutrition Examination Surveys.

Based on the experience of the past 30 years, it appears the obesity epidemic is solely a recent phenomenon. However, Helmchen and Henderson (2004) found in their study of a randomly drawn group of Civil War Union veterans this is not the first period of adverse obesity trends in U.S. history. They wrote, “In relative terms, then, obesity was spreading at least as fast at the beginning of the 20th century as at the end of the 20th century.” For example, for a similar 50 to 59 age group, the prevalence rate of obese males increased from about 1.0 percent in 1880-84 to 3.5 percent in 1890-94 and to 5.5 percent in 1900-04. The annual rate of growth of median BMI was about 0.3 percent between 1900 and 1976, which almost doubled to 0.5 percent

between 1988 and 2000. Komlos and Brebec (2010) estimated, studying birth cohort groups since 1882, that BMI has been increasing among birth cohort groups since early in the 20th century and possibly earlier, punctuated by upsurges, particularly after the two world wars, generally attributable to the gradual increase in affluence and well-nourished lifestyles, which persisted between birth and adulthood. They observed a 71 percent increase in average BMI in black females born between 1955 and 1965 (possibly 30 to 40 years ahead of the trend in the other major ethnic/gender groups). They also found that this long-term trend was accompanied by a substantial increase in distribution skewing toward the more obese range, indicating a greater increasing trend at higher levels of BMI. When assessing trends in skin-fold thicknesses rather than BMI, Burkhauser et al. (2009) indicated the current rise in the prevalence of obesity in both adults and youths as measured by BMI was detectable 10 to 20 years earlier.

This is not just a U.S. problem, as seen by the Organization for Economic Co-operation and Development (OECD) statistics and projections in Figure 10. Although the level of obesity by country will likely continue to differ as a result of cultural and eating habits, there has already been a convergence of trends as many countries move through their nutrition and sedentary transitions. Unless radical changes occur, the seemingly unstoppable spread of Western-style diets and convenience-facilitating technology will lead others to reaching U.S. level obesity levels.

Figure 10
Past and Projected Overweight and Obese Rates



Source: *Health at a Glance 2009*, Organization for Economic Co-operation and Development.

Katzmarcyk et al. (2006) indicated that based on self-reported heights and weights in seven nationally representative surveys in Canada, although the trends in obesity growth in Canada are similar to that in the United States, the absolute level of the different obesity classes remains lower than that in the United States. An analysis of the National Population Health Survey data indicates that almost a quarter of Canadians who were overweight in 1994-95 had become obese by 2001-03. As a result, although the number of overweight may have stabilized, the number of obese may continue to increase.

In Europe, similar concerns have been raised, because, although obesity levels are still below those of the United States, the increasing trend is not dissimilar, e.g., in Pichon et al. (2008). In Latin America, obesity and overweight prevalence rates are growing steadily and reaching levels close to and possibly surpassing that of the United States. For example, obesity prevalence for Mexican women older than 30 increased from 9 percent in 1988 to 24 percent in 1999. Monteverde et al. (2010) found higher excess mortality risks among those overweight and obese 60 and older in Mexico than in the United States, although the rate of obesity-related chronic diseases for those with a high BMI is now larger for the U.S. elderly than in Mexico. In addition, the relative risk of death for those experiencing these diseases is higher in Mexico, possibly because they are more concentrated in those of lower income (who most likely have less and poorer quality of health care services and higher prevalence of infectious and parasitic diseases).

How Did We Get to This Point?

Many theories have been presented to explain how the current obesity epidemic began and has developed. It can be viewed as a physiological disease with limited available cures and treatments, likely to have been caused by multiple interacting factors. Much of the current discussion relates to the factors contributing to the obesity surge and how to manage them, most suggesting that behavioral and environmental influences rather than genetic ones dominate the causes, although the mix can vary significantly by individual.

Studies of contributing causes of obesity and their risk factors indicate this is a multifaceted and complex issue, with a partial discussion following.

- **Nutrition.** Current American food habits are not compatible with healthy nutrition. Although in most cultures the young have been taught to “clean their plate,” there has been a fundamental change in the consumption of an unhealthy mix and the quantity of food, as the expectations of convenience and consumption expands, even for those experiencing unemployment and recession challenges.

After remaining relatively stable during most of the 20th century, average energy consumption increased by roughly 12 percent between 1985 and 2000, mostly due to increased carbohydrates and consumption of grains and added fats and sugars. This is in part because of lack of time or availability of less expensive food lacking in nutritional quality — once people ate tortilla chips, then tortilla chips with cheese, and now with a factory-made topping that looks like cheese but

mostly contains oil, flavoring and chemicals. Ingredients often are made of what consumers crave most — fat, sugar and salt.

In 2009, 67.5 percent of adults ate less than two servings of fruits daily and 73.7 percent ate less than three vegetable servings daily, far short of official national health objectives. Technological factors have led to increased food consumption due to lower food prices and less time available and taken for its preparation. Portion size and food industry pricing are related trends difficult to change without external interference. Many fast food restaurants' items and snack foods include a large fixed-price component, with the charge for larger sizes being quite small (particularly in "supersizing"). These relatively small incremental prices for large additional quantities of food form a powerful incentive to over-buy quantity in excess to fundamental food needs. In addition, all-you-can-eat restaurants and buffets are always a test of one's self-control and ability to stop eating when full. These test one's willpower and desire to take advantage of apparent value.

The United States Department of Agriculture (USDA) reported that between 1985 and 2000 the retail price of carbonated soft drinks rose by 20 percent and sugars and sweets by 46 percent, while there was a 118 percent increase in the price of fresh fruits and vegetables. Serving sizes in some cases have increased between two and 10 times. Increases in mass preparation of food in part due to increases in food production and preparation efficiency, the availability of fast food restaurants, and calorie-rich foods and serving size, as well as changes in relative food prices, all have an effect on consumption.

Sometimes blamed is the increase in the availability of fast food restaurants. Anderson and Matsa (2011) found that at least in rural areas, the causal link between fast food or full-service restaurant consumption and obesity is minimal at best, although other studies have indicated a close correlation between eating out (due in part to restaurant density) and obesity. However, Anderson and Matsa suggested that many consumers offset calories from restaurant meals by eating less at other times.

In general, patterns of diet are shaped by price and availability (i.e., convenience), although the consumption of some food such as staples do not vary much with price, while purchases of meat, eggs, milk, sugary drinks and fruits are more price sensitive. Those in low income households are particularly sensitive to price changes.

Increases in labor force participation for mothers and two-income households in which neither parent remains at home to look after the house, have resulted in reduced time to prepare food, which tends to increase the number of restaurants and take-out meals. This can also lead to a "TV dinner" microwave approach.

The relative prices of food in fast food outlets and full-service restaurants and the price of food prepared at home are considered by many every day. In fact, according to Bleich et al. (2007), average food prices in the developed world fell by 12 percent from 1980 to 2002, ranging from 26 percent in the United Kingdom to 0.2 percent in France, with the United States reduction being 8 percent (although the consumer price index for food items increased only 3 percent slower than the CPI for nonfood items). Nevertheless, Cutler et al. (2003) observed that it is not clear that eating out should be assumed to always increase caloric intake, as restaurants can cook low-calorie food almost as easily as high-calorie food, and food prepared at home can be just as junky as that eaten in a fast food restaurant.

Conditioned hyper-eating is now often a hard habit to break among those overweight and obese. Not only this, those who are obese tend to eat faster, there being a correlation between behavior and body weight, as the faster you eat, the more full signals can be missed, intentionally or not.

The increased use of high-fructose sugar-flavored soft drinks is a prime suspect in the growth of obesity. Bray et al. (2004) found a clear distinction between the absorptive process for fructose and glucose. Fructose is 73 percent sweeter than sucrose. In addition, the significantly larger portion size of these beverages may also be contributing to weight gain in America. The daily intake of sodas and fruit drinks for U.S. adults increased by at least three times between 1975 and 2005. The Nurses' Health Study II (an eight-year follow-up study reported on by Weinstein et al. 2004) indicated that women who increased their consumption of sugar-sweetened soft drinks gained 17.6 pounds between 1991 and 1999, whereas women who decreased their consumption during this period gained only 6.2 pounds.

Nevertheless, others have found the link between the use of soft drinks and childhood obesity is weak at best. Forshee et al. (2007) indicated that several of the major ecological, epidemiologic and randomized controlled trials addressing the effects of sugar-flavored soft drinks are either inconclusive or unreliable. They claim it is unclear why high-fructose corn syrup would affect satiety or absorption and metabolism differently than sucrose, and a recent expert panel convened by the Center for Food, Nutrition and Agriculture Policy concluded that corn syrup does not appear to contribute to overweight and obesity any differently than do other energy sources. They did indicate that further study is warranted and that many other factors contribute to the obesity epidemic. According to U.S. Food Supply Series data, there has been an increase in the use of both high-energy-containing and low-energy-containing sweeteners. According to Harnack (2000), this suggests that, although the percentage of low-energy sweeteners has increased, these are perhaps being consumed in addition to rather than in place of high-energy-containing sweeteners. The controversy continues regarding cause-and-effect, although it is clear the increased use of fructose and the obesity epidemic did occur at the same time.

Many studies have shown that a significant percentage of those dieting are unsuccessful and will terminate their dieting programs before successful results are achieved and sustainable. In fact, dieting may result in weight gain through possible erratic delivery of nutrients that might trigger physiological responses conducive to gaining, rather than losing weight. The elasticity of personal appetite is often difficult to control.

Social and cultural forces also can play a role. For example, Agras and Mascola (2005) found parental overweight was the most potent risk factor in determining a child's overweight status. The obese tend to marry other obese. This can amplify bad habits in current and future generations that may result in a certain level of genetic vulnerability over a long period of time. In addition, having friends who are obese tends to "promote" the belief that obesity is acceptable, although if the friends experience health problems in the future, this belief might change.

- **Lack of physical activity.** Modern living has facilitated a more sedentary lifestyle for many Americans. Contributing factors have included the increase in labor-saving devices at work, home, transit and play, while leisure time activities increasingly involve passive entertainment.

Sustained physical activity helps protect against weight increase, overweight and obesity, as well as enhancing fitness. In addition, inadequate physical activity can itself be an independent health risk factor. When men and women were hunters and farmers, physical activity was a fact of life; in fact, it was life. Now most Americans not only do not rely on physical activity at work and have not for several decades, but they also have a relatively sedentary lifestyle outside work. Technology has been harnessed to make life easier through such gadgets as remote controls, microwaves, garage door openers, electric lawn mowers and snowblowers, but, as an intended or unintended consequence, has decreased the total amount of personal resources involved in physical activity. Social networking and surfing the Internet cannot help either.

The lack of physical activity is evident in all population segments and the trends are not favorable either. Indeed, more than half of U.S. adults do not meet the recommended amount of physical activity to obtain the optimal amount of healthy benefits. Whether this is attributed to laziness, too many other important things to do or just not allocating the time necessary does not matter.

The 2004 NHIS found 59 percent of American adults do no vigorous physical activity in their leisure time, with only 26 percent engaging in vigorous leisure-time physical activity for at least 10 minutes three or more times per week. Michaud et al. (2007) found, based on the 2004 U.S. Health and Retirement Study, that about 52 percent of men older than 50 hardly ever engaged in physical exercise, while 61 percent of females older than 50 hardly ever did (in comparison, the percentages for Europeans older than 50 were 50 percent and 40 percent, respectively). Based on the 2004 NHIS, the highest prevalence of

inactivity is for those older than 75 (about 57 percent of males and 66 percent of females), with 52 percent of both Mexican-Americans and non-Hispanic blacks being inactive. With respect to education, 29 percent of those with at least some college education are inactive, while 49 percent of those with at least high school completed and 64 percent of those without a full high school education are inactive. We can certainly do better than this.

Today's youth are considered by some to be the most inactive generation in history, with 57 percent of American adolescents watching two or more hours of television or its equivalent daily. This is in part due to reductions in school physical education programs, parental security concerns due to seemingly unsafe community recreation facilities and neighborhoods, and substitution of sitting-on-a-chair activities for physically active leisure. It is also common to place blame on soft drink manufacturers and fast food restaurants. Advertising budgets and the scale of use of what is considered unhealthy food and drink are indeed huge. Sixty percent of American middle and high schools sell soft drinks in vending machines, although efforts continue to change this practice. In 2002, it was estimated that 240 U.S. school districts had entered "pouring rights" contracts with soft drink companies, giving the schools cash and other incentives in exchange for the right to sell sodas in vending machines and to include public advertisements for their products.

Physical inactivity is both a cause and a consequence of obesity. For example, black women with a high obesity level report less frequent participation in exercise than white women. This points out a more general problem, the interconnection of these factors — a higher level of obesity is in part due to a lack of exercise while at the same time obesity contributes to this relative lack of exercise.

- **Socio-economic factors.** According to Ogden (2010b), obesity prevalence among adult men is currently similar at all income levels with a tendency to be slightly higher at higher income levels, although among adult females, obesity prevalence increases as household income decreases. There appears to be no significant trend between educational level and obesity prevalence for men, while among women obesity prevalence increases as attained education level decreases. Changes in obesity prevalence between 1988-94 and 2007-08 as indicated in NHANES increased at all income and educational levels. Historically in the West, it was more common to find obesity more concentrated in the poor. As George Orwell said, "The less money you have, the less inclined you feel to spend it on wholesome food. A millionaire may enjoy breakfasting off orange juice and Ryvita biscuits; an unemployed man does not." Well-planned nutritious meals are more difficult to plan and maintain when finances are tight. More recently, the more well-off have placed added value in convenience, with a feeling of entitlement to fully satisfy their nutritional desires.

According to Ogden (2010c), among boys and girls, obesity prevalence generally decreases as household income increases and as the education of the head of household increases, although these relationships are not consistent across race and ethnic groups, especially in the case of non-Hispanic blacks and Mexican Americans. However, between 1988-94 and 2005-08, as was true for adults, childhood obesity increased at all household income and head-of-household education increases.

- **Do obese children become obese adults?** Evidence that obese adolescents are likely to develop into obese adults suggests that the current adult weight problem may get worse before it gets better, as adolescent obesity has increased in prevalence. Many public policy programs, including “Let’s Move” sponsored by first lady Michelle Obama, have recently been focused on controlling youth obesity, in part due to this long-term concern. This is particularly important as poor eating habits established during childhood can be difficult to change as adults.

Although it is not possible to determine which children will become obese as adults, being overweight when young appears to predispose a person to being overweight in adulthood. While the correlation between BMIs in childhood (e.g., ages through 5) and adulthood is not particularly strong, it is higher between adolescence and adulthood. Based on a review of eight prospective studies, one-third of obese preschool children and about one-half of obese grade school children become obese as adults; the more obese a child, the more likely he or she remains obese as an adult. In two long-term studies, 80 percent of overweight children remained overweight when re-examined 20 or 30 years later.

Other studies have confirmed this relationship, especially in morbidly obese adolescents. Thé et al. (2010), based on the National Longitudinal Study of Adolescent Health with a mean follow-up of 13 years, found that a large percentage (about 70 percent of severely obese adolescents) remain severely obese in adulthood, while less than 8 percent of those non-severely obese adolescents become severely obese in adulthood, with the highest rates for non-Hispanic black women. Obese adolescents were significantly more likely (by a factor of about 15) to develop severe obesity in young adulthood than normal-weight or overweight adolescents. Thus, there was a strong persistence of severe obesity from adolescence to young adulthood. In addition, there is a relatively high incidence rate of severe obesity during the transition from adolescence to adulthood, and those who were obese as adolescents were significantly more likely to become severely obese in adulthood.

3. Effect of Obesity and Related Factors on Mortality

A randomized controlled trial can deepen the understanding of the cause and effect of related factors. Unfortunately in the case of phenomena such as mortality, perfect trials or biogenetic analysis that can distinguish between statistical correlations and cause-and-effect relationships cannot easily be conducted. As a result, the scientific approach of learning through observation has to be applied. Although various studies of relationships have indicated that health care costs have been affected by obesity as is discussed in Section 4 of this paper, the case for obesity causing adverse mortality has either not been proven conclusively or has only been demonstrated to be the case for certain population segments.

Contributing and mitigating factors, including nutrition, physical activity, genes and other physical conditions can significantly influence the extent that a risk factor such as obesity can affect a person's and population's health and living status. Certainly the amount and type of nutrition and energy consumption not only contribute to an increased amount of obesity but are also mortality risk factors themselves.

Nevertheless, even though the prevalence of obesity has skyrocketed over the past 30 years, the level of mortality rates has moved in the other direction. Two significant reasons for this apparent divergence in trends are the reduction in smoking and the effect of mitigating medical advances in treating cardiovascular diseases and their risk factors, such as blood pressure and cholesterol. A key issue in assessing future mortality trends is the extent to which these potentially offsetting contributions to mortality will continue, including both because of the continuing underlying trends and time lags involved. This has resulted in expectations of future mortality of those who are overweight and mildly obese being lower in some areas than the mortality of those in the normal weight category several decades ago.

It is difficult to attribute a given level of mortality to a specific cause, in part because of the multiple causative factors often involved, the complex nature of and inter-relationships among underlying health processes and mortality, and the difficulties in assigning causation, attribution and measurement. Not only are those overweight or obese more likely to suffer from chronic diseases, but their physical condition (e.g., weight) can further contribute to poor nutrition and exercise, in a vicious circle. According to Manson et al. (2007), adipose tissue has been increasingly recognized as an active endocrine organ, capable of releasing a large number of cytokines and bioactive mediators that play important roles in the pathogenesis of many obesity-related diseases.

The prevalence and effects of higher BMIs vary by age. As a result, although many observations relate to those of all ages, some of these effects vary by major age group categories, i.e., adults, older adults and children. Overall, obesity, particularly at the morbid level, has been associated with a higher level of mortality at all ages, although the primary ages it affects appear to be nonelderly adults and evidence indicates it can affect individuals in different population segments in diverse ways.

Some of the health hazards associated with being overweight or obese may be more strongly related to the pattern of body fat distribution, composition of weight or to fluctuations in

weight than to weight in excess of a given level per se. Those with an excess accumulation of abdominal adipose tissue are at increased risk for several medical conditions, in part because fat stored in different body locations can have different characteristics. Metabolic changes can result from an increase in fat stores in which the fat cells themselves enlarge and produce chemicals that increase the risk for several diseases. In addition, increased mass itself can cause disabling conditions and injury.

Specific Hazards

Obesity appears to directly affect some diseases, such as diabetes and hypertension, while it acts on others indirectly through (or as a second-order effect on) intermediary factors, such as cardiovascular risk factors that in turn affect stroke and heart disease. Diseases and conditions often associated with obesity include:

- **Type 2 diabetes** mellitus (referred to in this paper simply as diabetes), impaired glucose tolerance and insulin (a key hormone in the use of sugar) resistance.

Diabetes is one of the most costly and burdensome chronic diseases, as well as being one of the fastest growing public health problems in the United States and globally. In the United States, 19 million adults reported receiving treatment for diabetes, more than double the 9 million who indicated they received such care in 1996. Wilde has projected there will be 366 million diabetics in the world by 2030 compared with 171 million in 2000. Currently, about 6.4 percent of the world population has diabetes, the percentage growth of which is similar and has been linked to the growth of obesity.

According to BRFSS surveys, 4.8 percent of American adults in 1995-97 and 9.1 percent in 2005-07 were reported to have diabetes, a continuation of the trend of the prior 20 years. The results of NHANES III indicated that while those who are overweight are twice as likely as those of normal weight to develop diabetes, those who are obese had three times the risk. The North American Association for the Study of Obesity found that 85 percent of those with diabetes are type 2 and of those almost 90 percent are overweight or obese. Corresponding percentages found in NHANES (1999-2002) were 86.3 percent. One of the most significant changes in reported diabetes incidence rates has been the result of a gradual improvement in its diagnosis, particularly in the obese, as they have been so highly related.

Its incidence generally increases with advance in age. It has been estimated to represent a substantial economic burden, as reflected by diabetes consuming about 14 percent of the U.S. total health expenditure. Thus, even if obesity itself is not as significant a health factor at those ages, diabetes, one of its effects, is.

Diabetes is in turn associated with a doubling of the risk of heart disease and stroke and is the leading cause of blindness, kidney failure and non-traumatic amputations. Diabetes can also lead to hypertension and high blood pressure, as

well as to severe long-term disability. Diabetes in conjunction with obesity has been found to be associated with chronic stress, depression, sleeping troubles and a reduced quality of life.

Obesity, due to its excess adipose tissue, especially when centrally distributed, predisposes an individual to diabetes by means of increased portal delivery of fatty acids to the liver. This process induces both hepatic insulin resistance and reduced insulin clearance. Because waist measurements may be more indicative of susceptibility to diabetes and because of the time lag involved (about 12 years according to the Framingham study), a focus on current levels of BMI may underestimate the future risks associated with the increase in obesity and the future trend in this disease. Recent genomic studies of the relationship between obesity and diabetes have identified what may be causal genes (e.g., FTO, a fat-mass and obesity-related gene), although most such studies can only discern correlations rather than cause and effect. According to McCarthy (2010), a person's risk of diabetes or obesity reflects the joint effects of genetic predisposition and relevant environmental exposures.

The Nurses' Health Study indicates that the relative risk to women of developing diabetes increases from a low point at a BMI of 22. Note that since many women tend to underreport their weight, its self-reported findings may underestimate this effect. The relative risks of diabetes were 2.7 for those of normal weight (23 to 24.9 BMI), 7.6 for those overweight, 20.1 for class I obese and 39 for those heavier than class I. This study found that being overweight or obese is the most important predictor of diabetes.

Similar results were found by Weinstein et al. (2004) in the Women's Health Study that also studied the relationship of weight and physical activity. Weinstein observed that, although weight had a greater influence on diabetes incidence than the level of physical activity, the latter was seen to modestly reduce the risk of diabetes. However, for those of a given level of BMI, physical activity had a more significant effect; Weinstein speculated, "Although they are viewed as independent variables, they may be influencing each other and contributing to the same causal pathway. Obesity is known to increase peripheral insulin resistance and reduces beta cell sensitivity to glucose. Although physical activity, among other things, increases insulin sensitivity and has complex effects that can improve glucose metabolism, it may not fully reverse the effects of obesity. Weight loss may therefore be a key mechanism to reduce the secretion of these factors by decreasing adipose tissue volume and subsequently reducing the risk of diabetes."

Hu et al. (2006) observed similar results in the Health Professionals' Follow-up Study. This study indicated that waist circumference was a better predictor of diabetes than BMI. Although both BMI and waist circumference appear related to the incidence of diabetes, waist circumference is a somewhat better metric to use for this purpose. The Nurses' and Professionals' studies also showed progressive

reductions in the multivariate-adjusted relative risk of diabetes with increases in physical activity.

Hu et al. (2006) indicated that in a study of Finnish men and women age 35 to 64 over a 12-year follow-up period, the relationship in diabetes incidence and obesity, physical activity and glucose levels were as shown in Table 11.

TABLE 11
Relative Risk of Type 2 Diabetes by Level of Physical Activity,
BMI and Glucose Levels

Level of Physical Activity	Glucose Level	Less Than 30 BMI	More Than 30 BMI
Low	Normal	1.1	13.2
Low	Impaired glucose regulation	15.5	30.2
Medium	Normal	2.2	7.3
Medium	Impaired glucose regulation	12.7	30.1
High	Normal	1.0	3.8
High	Impaired glucose regulation	5.5	19.0

Source: Hu et al. (2006).

Hu et al. concluded their study with the observation, “The best long-term results may be achieved when physical activity produces an energy expenditure of at least 2,500 kcal per week. The optimal approach in weight reduction programs appears to be a combination of regular physical activity and caloric restriction. A minimum of 60 minutes, but most likely 80 to 90 minutes of moderate-intensity physical activity per day may be needed to avoid or limit weight regain in formerly overweight or obese individuals. Regular moderate-intensity physical activity, a healthy diet and avoiding unhealthy weight gain are effective and safe ways to prevent and treat type 2 diabetes, as well as cardiovascular disease, and to reduce premature mortality in all population groups.”

Although total fat intake has not been directly associated with the increased incidence of diabetes, increased levels of saturated fats have been associated with higher impaired glucose levels, while intake of vegetable fats and polyunsaturated fat has been associated with a lower level. In addition, eating more fruits and vegetables has been associated with a reduced risk of diabetes, while typical Western-style diets generally have had the opposite relationship. The Nurses’ Health Study II found that an increase in sugar-sweetened beverages may be associated with an increased risk of diabetes, possibly through excessive calories and large amounts of rapidly absorbable sugars.

Overall mortality rates of male diabetics have decreased by 43 percent over the past few decades between NHANES I and NHANES III. In contrast, mortality rates increased even more for female diabetics (hazard rates¹ increased from 1.31 to 2.84). The overall diabetic prevalence rate for males and females is similar in a more recent NHANES. Note that this comparison should be viewed with caution because of the self-reported nature of diabetic status and as NHANES only reports diagnosed cases (since females visit the doctor more often than males, some of this differential may be due to a higher diabetes diagnosis rate) and because of the relatively small size of its gender-specific study.

The burden of diabetes and cardiovascular disease has fallen disproportionately on racial and ethnic minority groups, especially blacks and Hispanics, even after socioeconomic status and conventional heart disease risk factors are considered. Although some of these differences may have genetic sources, other factors may also be involved, e.g., under- or over-nutrition, including breast-feeding at critical stages of fetal development, which can induce permanent changes in metabolism or body composition that result in insulin resistance.

- **Cardiovascular and Heart Disease**

In addition to an altered metabolic profile, a variety of adaptations and alterations in cardiac structure and function occur as adipose tissue accumulates in excess amounts, even in the absence of comorbidities. The obese generally have a higher cardiac output, mostly attributable to increased stroke volume, and a lower total peripheral resistance than do those who are lean. Ventricular chamber dilation may then lead to increased wall stress, which in turn may lead to an increase in myocardial mass and ultimately to left ventricular hypertrophy. In addition, excess body fat may cause stiffness in the aorta. On the whole, through its effect on the cardiovascular system, an excess amount of weight generally predisposes to or is associated with numerous cardiac complications such as coronary heart disease (CHD), congestive heart failure and sudden death.

Obesity has been shown to be an independent risk factor for cardiovascular disease and a contributing factor to several of the key risk factors for cardiovascular disease as indicated below, as well as in combination with other risk factors, e.g., through the metabolic syndrome². In addition to obesity that measures overall weight, central adiposity has also been found to be related to these risk factors.

¹ Multiple of benchmark mortality rates for the applicable demographic characteristic.

² A cluster of several key risk factors for cardiovascular disease and diabetes, including three of the following five factors: (1) elevated triglycerides, at least 150 mg per deciliter, the most common type of fat in the blood; (2) high blood sugar, a sign of insulin resistance; (3) enlarged waist circumference, generally above 40 inches for men and 35 inches for women; (4) elevated blood pressure, at least 130/85 mm of mercury; and (5) a low level of “good” HDL cholesterol, lower than 40 mg/dL for men and 50 mg/dL for women.

Most, but not all studies have indicated that obesity as assessed by BMI is associated with an increased risk of coronary heart disease and the incidence and consequential premature mortality associated with cardiovascular disease. Coronary heart failure, for which obesity has been found to be a significant risk factor, is the only common cardiovascular condition that is currently increasing in incidence, prevalence and resulting mortality rates, with the overall five-year survival rate being no better than 50 percent.

NHANES III indicated that those who are overweight were 40 percent more likely to develop heart disease, class I obese were twice as likely, and class II+ obese had a risk nearly 70 percent higher than those of normal weight.

Analysis of the Framingham Heart Study (Peeters et al. 2003) indicates that during its 14-year follow-up period for those who were obese, the relative risk of heart failure doubled after correction for other known risk factors. Results indicated that a 40-year-old female nonsmoker lost 7.1 years of life and a similar male nonsmoker lost 5.8 years of life because of obesity. Although the study indicated that BMI level at ages 30 to 49 was indicative of higher mortality between ages 50 to 69, since the study could not identify cause-and-effect relationships, excess premature death would not necessarily be prevented through obesity reduction.

As reported by Kenchaiah et al. (2002), who also used Framingham data, the increase in relative risk of heart failure was 5 percent for men and 7 percent for women for each increase in body weight equivalent to a 1 BMI unit across the entire range of BMIs, with no minimum BMI threshold. Increments of BMI had a smaller effect on the risk of heart failure for those with hypertension. With a median follow-up period of 33 years, BMI was found to be linearly and independently associated with cardiovascular disease mortality (a hazard rate of 1.22 per 5-unit increase). According to Kenchaiah, this was probably due to a decreased contribution of obesity to the risk of heart failure in the presence of this major risk factor. It suggests that approximately 11 percent of heart failure cases among men and 14 percent among women were attributable to obesity alone. These findings are particularly of interest due to the very long follow-up period included in this study.

The Chicago Heart Association Detection Project in Industry indicated that, adjusted for systolic blood pressure and total cholesterol level, the hazard rates for those obese compared with those of normal BMI was 1.43 for those with low cardiovascular risk factors and 2.07 for those with moderate cardiovascular risk factors. With its long follow-up period (32 years), the Chicago study indicated that those with three or more elevated risk factors in middle age had a median survival period more than nine years shorter than men with none or one risk factor, with a corresponding ratio for women of a seven-year shorter survival period.

Djoussé et al. (2010)'s study of 20,900 men in the Physicians' Health Study I indicated that the lifetime risk of heart failure (based on a mean follow-up period of 22.4 years) was, unsurprisingly, higher in men with hypertension and no prior CHD than in those without hypertension. Healthy lifestyle habits, including body weight, smoking, exercise, moderate alcohol intake, and consumptions of breakfast cereals and fruits and vegetables were individually and jointly associated with a lower lifetime risk of heart failure. Maintenance of these habits remains critical to lowering the risk of heart failure. Thus, the Chicago and the Framingham studies suggest cardiovascular disease as related to BMI may only develop after a long follow-up period. As a result, without sufficiently long follow-up periods, studies may not observe the full effect of the relationship between obesity and premature heart failure. This effect is further discussed later in this section.

Findings from the Framingham Heart Study as indicated in Eng (2003) suggest the following relationships:

- a 10 percent increase in weight corresponds to about a 30 percent increase in the incidence of heart disease;
- an increase in a unit of BMI corresponds to a 5 percent increase in the likelihood of heart failure for men and 7 percent for women; and
- 40-year-old male and female nonsmokers can expect to lose about six and eight years of their life, respectively, because of being obese.

Nevertheless, based on the results of the Framingham study, it appears that the reduction in the primary proximate cause of many premature deaths (e.g., cardiovascular disease) resulting from obesity, together with the trend in several of the risk factors for cardiovascular disease (see below, e.g., cholesterol and blood pressure levels), may have decreased the overall mortality risk associated with being overweight or obese as well. Nevertheless, the higher prevalence of obesity, particularly severe obesity, will constitute a significant risk to future mortality levels.

Adequate physical activity can protect against premature cardiovascular heart disease in those overweight and obese. As a result, it is useful to study both of these factors, preferably simultaneously.

The results of Nemetz et al. (2008), in a study of autopsy results (the gold standard in assessing causes of death) from 1981-2004 of non-natural deaths of Olmsted County, Minn., residents ages 16 through 64, indicated that the increase in CHD incidence may have ended after 1995 and possibly has reversed after 2000. Any increased prevalence resulting from improved survival from CHD has been offset by reductions in disease incidence. These findings suggest that the decline in CHD prevalence may have ended and that the significant reductions in cardiovascular disease experienced over the past 40 years may be ending. This is a significant finding for future possible trends in overall mortality levels as well.

The authors end their paper by indicating that “the extent to which recent trends are attributable to the epidemics of obesity and diabetes mellitus awaits further investigation.” This study’s limitations are its relatively small sample size and limitation to the study of a single county in the United States that primarily consists of non-Hispanic whites.

Nevertheless, Olshansky and Persky (2008) wrote, “What this observation may foretell is that in the coming decades the age at onset of coronary artery disease could shift to younger ages and the death rate rise. ... If so, the reversal in trends in young adults today could precede that in older individuals in the future.” They further hypothesize, “It is possible that obesity has a stronger negative effect on coronary artery disease when the disease is expressed early in life because the late-onset expression may be attenuated more effectively with aggressive therapies for hyperlipidemia and hypertension.”

Bibbins-Domingo et al. (2007) projected future CHD deaths in 2035 using U.S. Census population data, National Center for Health Statistics (NCHS) mortality and Framingham Heart Study CHD incidence rates, together with CHD risk factor prevalence from the NHIS 2000. They projected excess CHD deaths of 35-year-olds of 33,000 CHD deaths in 2035 (a 14 percent increase from current levels). The range in their estimate was between 14,000 and 45,000, equivalent to a 5 to 16 percent increase in these deaths.

- **Cardiovascular Risk Factors**

In assessing the effect of obesity upon mortality due to cardiovascular and related diseases, an assessment of two sets of relationships may be useful: (1) underlying factors including obesity and cardiovascular risk factors and (2) all of these factors and resultant mortality. On the surface, particularly looking at the second set of relationships, it seems counter-intuitive that while the rate of obesity has skyrocketed over the past few decades, the rates of premature death from ischemic heart disease and key cardiovascular disease risk factors have experienced a gradual and long reduction. In addition, according to various NHANES (as reported by Gregg et al. 2005), there have also been large reductions (33 to 52 percent in the past 30 to 40 years) in the prevalence of adverse cholesterol levels, high blood pressure and smoking, offset somewhat by an increase by 55 percent in diagnosed and undiagnosed diabetes.

In fact, during the last part of the 20th century, overall mortality trends have been quite favorable. In large part, this has been due to the significant (e.g., by more than 40 percent between 1980 and 2000) reduction in cardiovascular heart disease. Table 12 shows an attribution of the sources of this change developed by Ford et al. (2007).

TABLE 12
Attribution of Sources of Mortality Improvement Between 1980 and 2000

Source	Proportion
Medical treatments	47%
Reduced total cholesterol levels	24
Reduced blood pressure levels	20
Reduced smoking prevalence	12
Increased physical activity	5
Increase in diabetes prevalence	-10
Increase in BMI	-8

Source: Ford (2007).

One conclusion that could be reached is that the reduction in premature mortality would have been greater had adverse trends in obesity and diabetes not occurred. However, considering the time lag between the increase in additional body weight and consequential cardiovascular risk factors and premature death, these relationships and the time lags involved indicate this counter-intuitive trend may not continue.

According to Brown (2000) reporting NHANES III results, “The importance of increasing BMI as a determinant of these conditions (positive relationships with systolic and diastolic blood pressures, total cholesterol, as well as the negative relationship with HDL-C) is clear.” However, elsewhere it has been noted that in some studies it has been observed that up to a certain point at some ages more weight may even be associated with greater survival for those with coronary artery disease.

As reported in NHANES 1999-2000, the prevalence rates of high cholesterol, high blood pressure and smoking among those obese was 21 percent, 18 percent and 12 percent less than those of corresponding obese individuals surveyed in the earlier NHANES I, while its prevalence declined between 12 percent and 14 percent for those in the normal BMI category. Gregg et al. (2005) found that, over the last 40-year period, with the exception of diabetes, the prevalence of the major risk factors has declined over recent decades among all BMI groups. This is consistent with the trend in the overall treatment of these factors by key risk mitigation (drug) techniques.

In contrast, according to Ezzati et al. (2007) based on NHANES and BRFSS, this trend appears to have changed, with the prevalence of uncontrolled hypertension throughout the 1990s among U.S. men beginning to stabilize or decline at a slower rate (19 percent to 17 percent between the early 1990s and early 2000s), while it has increased for females (17 percent to 22 percent). Unless this recent trend is reversed, the rate of decline in cardiovascular disease may also begin to decelerate, consistent with the indications noted in Nemetz et al. (2008).

While obesity remains associated with elevated levels of several risk factors compared to those who are lean, the fact that the level of risk factors has now diminished such that some of them are lower than those of lean individuals of 30 years ago has often been overlooked. In addition, BMI, as one of the several factors associated with cardiovascular disease risk, may have been treated in a more appropriate or aggressive way recently, reducing its ultimate effect on this risk.

Bogers et al. (2007) is a meta-analysis of 28 studies of the relationship between CHD and overweight and obesity consisting of about 300,000 subjects with follow-up periods between 4.8 and 35 years that included multiple adjustments for age, sex, physical activity and smoking, with simultaneous adjustment for blood pressure and cholesterol levels. It found that even for those moderately overweight, there is a significant increased risk of CHD, independent of traditional risk factors. However, adjustments for blood pressure and cholesterol level lowered the excess risk identified by about 45 percent, to a hazard rate of 1.16 for those overweight (before adjustment it was 1.32), although the authors noted that some of this reduction may be due to confounding factors such as differences in nutrition. The corresponding hazard rate for those obese after blood pressure and cholesterol level were adjusted for was 1.39 (before adjustment it was 1.69). In addition, the study indicated that overweight is significantly associated with increased risk of diabetes.

The Atherosclerosis Risk in Communities study of 9,514 adults between ages 45 and 64 with a nine-year follow-up was reported on in Lutsey et al. (2008). Participants who had high intakes of red meat, fried foods and refined grains experienced an 18 percent increase in the metabolic syndrome, with each of the food types individually associated with the increase. These were also studied in combination, in what might be characterized as a Western-style diet. At the same time, those who ate a diet dominated by fruits, vegetables, fish and poultry experienced no change in their metabolic syndrome levels. A 25 percent increase in risk was observed in those who ate two or more servings of red meat a day compared with those who only ate meat twice a week. In addition, dairy consumption appeared to confer protection against developing the syndrome. Thus, in this population, the overall Western-style diet had a significant adverse effect on the average metabolic syndrome. Interestingly, yet unexplained, was a finding that the use of diet soft drinks also resulted in an adverse effect on the metabolic syndrome. This latter finding warrants further investigation.

An extensive literature review prepared by the National Heart Foundation of Australia for the Australian Institute of Health and Welfare and the National Heart Foundation of Australia (2004) concluded that evidence connecting excess body weight and the major risk factors for cardiovascular disease existed in Australia for atherosclerosis, high blood pressure, particularly in children and adolescents, high total cholesterol, high LDL cholesterol, low HDL cholesterol, diabetes in

adults with central adiposity, cardiovascular disease arising in young to middle-aged adults and CHD in adults.

Based on the 1998 Health Survey for England that studied those age 35 to 74 and the Framingham study, Nanchahal et al. (2005) wrote, “Much of the CHD risk associated with overweight is mediated by its association with hypertension, lowered HDL, and increased triglyceride concentration and insulin resistance. Moreover, weight gain increases risk of CHD and weight loss significantly improves risk factors for CHD. Every kilogram of weight gain after high school increased risk of CHD by 3.1 percent in men, while every kilogram of weight loss resulted in a reduction of 0.7 percent in LDL-cholesterol, 0.5 percent in systolic blood pressure, 0.2mM in blood glucose, and an increase of 0.2 percent in HDL-cholesterol. The loss of 5 to 10 percent in initial body weight among overweight and obese adults can lead to reductions in various chronic disease risk factors, clinical benefits and improvements in health.” This may imply that obesity’s adverse effects work through these risk factors, rather than being an independent risk factor itself.

Although most studies have used BMI as an indicator of obesity, as noted elsewhere in this paper, some of the studies of obesity may not have used the best metric of excess body weight. For example, the National Heart Foundation of Australia (Dhaliwal and Welborn 2009) found that obesity measures of waist circumference and waist-to-hip ratio were both significant univariate predictors of cardiovascular deaths, while BMI was not.

Two cardiovascular disease risk factors are discussed in the following:

- Hypertension or high blood pressure. Hypertension can lead to premature atherosclerosis, coronary artery disease, heart attacks, abnormally large hearts and strokes. Obesity may blunt certain actions of insulin that open blood vessels and may cause structural changes in the kidney and abnormal handling of sodium. Because those who are heavier generally consume more calories, they also are likely to take in more sodium. It also is associated with alterations in the systems that regulate blood flow and cardiac output. Must et al. (1999) found that NHANES II showed the obese had high blood pressure twice as often as those individuals of normal weight.

Obesity-related hypertension also is commonly associated with other elements of the metabolic syndrome (in the aggregate often shown to be a cardiovascular risk factor), such as insulin resistance and glucose intolerance. The hypertension effect of blood lipid levels not effectively mitigated by drugs can contribute to a higher mortality rate from CHD. Furthermore, obesity as a significant risk factor for diabetes also increases cardiovascular risk through diabetes.

According to Brown (2000), on an age-adjusted basis, high blood pressure is twice as prevalent for the obese (both males and females) compared with that for those with a BMI of less than 25. After adjustments to NHANES III made for age, gender and race/ethnicity, BMI was independently and positively associated with high blood pressure levels and was found to contribute to more than half of the increase in these levels. Narkiewicz (2005) found that at least 75 percent of hypertension cases are reported to be directly attributed to obesity.

The association of low-risk factors for hypertension, including a BMI of less than 25, vigorous exercise, a high score of the Dietary Approaches to Stop Hypertension diet, modest alcohol intake, non-narcotic analgesics less than once per week and a low amount of supplemental folic acid, was studied in the Nurses' Health Study II. Although all six of these modifiable risk factors were independently associated with new hypertension during follow-up after adjusting for age, race, family history, smoking and the use of oral contraceptives, it was found that BMI was the most powerful factor. Although multiple low-risk factors were significantly associated with lower risk among normal weight and overweight women, there was no association among obese women; based on this, obese women may not benefit from other low-risk behaviors without weight loss.

A Finnish study (Jousilahti et al. 1996) found that with effective long-term intervention, voluntary weight loss has been shown to be effective in the prevention of hypertension. Since obesity is the strongest determinant of hypertension, weight control may be the most effective means to prevent hypertension.

U.S. non-Hispanic adult blacks have a significantly higher prevalence of hypertension (high blood pressure and/or taking anti-hypertensive medication) than non-Hispanic adult whites or Mexican Americans (rates for males of 41.4 percent compared with 31.5 percent and 26.3 percent, respectively, and for females of 44.4 percent compared with 28.1 percent and 26.2 percent, respectively, for those 20 and older at 2005-08, according to Health, United States, 2010 on an age-adjusted basis). The overall trend in the prevalence of hypertension has been increasing, although that has not been true in all racial, gender and age categories.

The Copenhagen General Population Study as reported in Timpson et al. (2009) found that BMI and blood pressure were related. Including those taking antihypertensive drugs, systolic blood pressure was seen to increase by 3.85 mm Hg for each 10 percent increase in BMI, with diastolic blood pressure showing an increase of 1.79 mm Hg for a similar increase. Timpson estimated the causal association through use of both rs9939609 (FTO) and rs17782313 (MC5R) genotypes that was verified by DNA

sequencing as instruments for BMI, the former possibly having a role in the hypothalamic regulation of appetite and food intake. This relationship was maintained after adjustment for education, income, smoking and drinking. These associations are large, supporting findings elsewhere that a reduction of weight by even relatively small levels can reduce the risk of hypertension. Note that the relationship between BMI and blood pressure declined at older ages, approximately half in those older than 75 compared with those younger.

- Cholesterol and triglycerides (fat) levels. Obesity has been shown to be associated with adverse levels of cholesterol. Although cholesterol has been treated effectively with medicine, reduced weight, proper nutrition and physical exercise have also been shown to have a favorable effect. During the 1980s and most of the 1990s, there were significant favorable changes in cholesterol levels, with smaller reductions in the late 1990s, even with the increased use of cholesterol-lowering drugs. Although it is unclear whether this trend will continue, growth in the prevalence in obesity will not help.

The pattern of fat distribution also affects the level of cholesterol, particularly relative to those with predominant abdominal obesity. This pattern of extra weight can also lead to growth in coronary artery disease, heart attacks and strokes. Triglyceride levels in adipose tissues represent the cumulative effect over time of differences between energy intake and expenditures, a key risk factor for heart disease that is usually higher in those who are obese.

- **Cancer**

According to the American Cancer Society, “Except for quitting smoking, the best way to cut your risk of cancer is to achieve and maintain a healthy weight, to be physically active on a regular basis, and to make healthy food choices.” These risk factors have been associated with, and in some cases are direct causal factors for, certain types of cancers. In *Current Facts & Figures, 2010*, the American Cancer Society indicated that overweight and obesity contributed to between 14 and 20 percent of all cancer-related mortality.

In the World Cancer Research Fund (2009) report, an estimate is made that between 34 and 39 percent of cancers are convincingly or can probably be modified by food, nutrition and physical activity (based on a study of 12 major types of cancer). The report indicated that since these factors also have been associated with other cancers, a broad estimate of about a quarter to a third of cancers can be prevented in higher income countries (about a fifth to a quarter in lower-income countries). The report concluded that a major proportion of the cancers attributable to food, nutrition, physical activity and body fatness can be

prevented by avoiding being overweight and obesity alone. They also emphasized the vital importance of control of smoking, the other major behavioral risk factor. The international panel convened by the World Cancer Research Fund (2007) performed a six-year evaluation of a wide range of original studies from around the world and observed that cancer patterns are primarily determined by environmental factors and not genetics, and in principle are preventable. The theme of the report was “correlations between changes in patterns of diet, physical activity, body composition and changes in patterns of cancer provide evidence that these factors are important modifiers of cancer risk,” and these factors influence fundamental body processes that may promote or inhibit cancer development and progression. The panel emphasized that “the risk of cancer is modified, not only by obesity, as usually defined, but by overweight as well, and even by degrees of body fatness generally regarded as healthy.” Possibly the most surprising finding was the degree to which being somewhat overweight is a risk for certain cancers.

The expert panel, based on their literature review, indicated through the following characterized relationships that there was: robust evidence that body fatness resulted in an increase in post-menopausal and endometrial breast cancer; evidence that esophageal adenocarcinoma, pancreas, colorectal and kidney cancers represented a plausible mechanism and the cause-and-effect relationship was convincing; a plausible cause for gallbladder and pre-menopausal cancers; speculative cause for liver cancer; and limited evidence for lung cancer.

In addition, this panel found that regular sustained physical activity protects against cancers of some sites, including colon cancer (at a convincing level) and female hormone-related cancers (probably relating to post-menopausal breast cancer) independently of other factors such as body fatness. The panel was impressed by the overall consistency of the evidence and concluded that relatively high (but not extreme) levels of physical activity protect or may protect against cancers of the colon, post-menopausal breast cancer and endometrium. In addition, since physical activity can protect against overweight, obesity and weight gain, it also indirectly protects against the cancers indicated above as well. Conversely, it indicated that a sedentary lifestyle may increase these risks. The report notes the panel was aware that average physical activity levels are continuing to decrease throughout the world.

Calle et al. (1999) found the risk of cancer was monotonically upward sloping relative to increasing BMI levels. Li et al. (2009) confirmed that obesity contributes to both earlier onset of pancreatic cancer and worsened survival. Studies have shown that among older, long-term survivors of various cancers, diet and exercise intervention reduced the rate of self-reported functional decline.

Calle et al. (2003), based on the results of the Cancer Prevention Study II, showed the proportion of deaths from all forms of cancer in the United States due to being overweight or obese was between 4.2 and 14.2 percent for men and from 14.3 to

19.8 percent among females, with the lower end of the ranges generally representing those who had never smoked. NHANES through 2004 indicated that morbidly obese men and women who were class III+ obese experienced mortality due to cancer 52 percent and 62 percent, respectively, greater than those in the normal BMI category. For female class III obese who had never smoked, the relative risk was 88 percent higher. BMI was shown to be significantly associated with higher rates of death due to cancer of the esophagus, colon and rectum, liver, gallbladder, pancreas and kidney, non-Hodgkin's lymphoma and multiple myeloma. In addition, significant number of premature deaths was observed in men of cancers of the stomach and prostate and in deaths of women from cancers of the breast, uterus, cervix and ovary.

The Nurses' Health Study showed that women who gained more than 20 pounds from age 18 to midlife doubled their risk of breast cancer compared with those with stable weight. It has been found that processing calories affects the activity of BRCA1, a gene that encodes a tumor-suppression protein, thus linking calorie intake and this type of cancer. This indicates that gene research is an increasing and fertile area of research that may provide further insight into epidemiological processes.

The Ning et al. (2009) meta-regression of 56 studies that analyzed the association of BMI and colorectal cancer indicated a 14 percent, 19 percent, 24 percent and 41 percent greater mortality hazard rates for BMI groupings of 23.0 to 24.9, 25.0 to 27.4, 27.5 to 29.9 percent and obese relative to those with normal BMIs, respectively. It also found that the association was stronger for colon rather than rectal cancer, men rather than women and North Americans rather than Europeans, and studies with self-reported rather than directly measured BMI and those adjusting rather than not adjusting for level of physical activity. Asian populations showed more steeply increased hazard rates than other population segments.

- **Kidney and Liver Diseases**

Although obesity is the No. 1 preventable risk factor for chronic kidney disease, it also appears associated with improved survival in patients with end-stage renal disease. Obesity may result in an increased risk of chronic kidney disease, especially when additional adverse factors are present, such as diabetes or lipid abnormalities. Structural damage of the kidneys may further increase blood pressure and predispose an individual to cardiovascular events.

Possibly three-quarters of those who are obese suffer from fatty liver (also known as steatohepatitis) and obesity is its most common risk factor. At least one-third of those affected by nonalcoholic fatty liver disease are estimated to occur in the extremely obese. Although whether obesity causes fatty liver is not yet known, it may also lead to more serious liver disease.

- **Psychological disorders**

Although more related to health care than mortality, psychological disorders affected by obesity include depression, anxiety, stress, bipolar disorder, schizophrenia, sleep apnea, sex disorders, weight stigma, dementia and other mental conditions, possibly even ending in suicide. These particularly affect the extremely obese. Whitmer et al. (2008), in a long-term (average 36 years) follow-up study of 6,583 members of Kaiser Permanente of Northern California, compared a group of 40- to 45-year-olds who had their sagittal abdominal diameter (SAD) measured between 1964 and 1973 with a diagnosis of dementia from medical records from 1994 to mid-2006. This demonstrated that overall, BMI is a strong predictor of dementia, including Alzheimer's, with a 75 percent increased risk for those with the highest BMI.

- **Other**

In addition to the specifically discussed conditions above, other conditions associated with obesity include respiratory difficulties (a large waist circumference has been shown to be the strongest predictor for impaired lung function), arthritis, asthma, gallbladder disease, chronic muscular-skeletal problems caused by stress on joints, osteoarthritis, heat injuries and heat disorders, skin problems, infertility, reproductive complications and complications from hospital stays. Waist size of middle-aged females has also been found to be related to an increase in the rate of strokes. Some of these conditions result in more adverse morbidity (see Section 4 of this paper) rather than mortality effects. And of course, overall quality of life issues can result from obesity as well.

Although these associations are generally related to all of the obese, they are particularly applicable to the extremely obese. Mokdad et al. (2003) found the BRFSS has revealed that, compared with those in the normal BMI category, extremely obese adults had seven times the risk of having diabetes, six times the risk of having hypertension, four times the risk of having arthritis, three times the risk of having asthma and four times the risk of having fair or poor health.

It should be noted it can be difficult to attribute premature mortality and health-related costs directly to obesity, as a quarter of those who are obese had six or more adverse medical conditions during 2002. This complexity affects the study of the sources of both mortality and medical costs.

Being sedentary has been found to increase the incidence of CHD, stroke, hypertension, obesity, diabetes, osteoporosis, depression, endometrial cancer, and, probably, breast and colon cancers. Sedentary behaviors and obesity are viewed as being independent risk factors for diabetes, cardiovascular and musculoskeletal diseases, and the metabolic syndrome. Cardio-respiratory fitness resulting from

regular physical activity can result in increased fitness, decreased fatness, enhanced metabolism and a stronger immune system, leading to a reduction in risks of such conditions as diabetes and cardiovascular disease.

Overall Effects on Aggregate Mortality

The diseases and conditions discussed above relate to those overweight as well as the obese, although Flegal et al. (2005) reported that, based on NHANES through 2002, there was about 85,000 fewer annual deaths of those in the overweight BMI category than those of the normal BMI (18.5 to 24.9) weight category, even after a 10-year follow-up period. This contrasts with CDC warnings against being overweight as well as obesity. Flegal et al. (2007b), also based on NHANES through 2002, estimated the number of cause-specific excess deaths due to various weight categories in the United States in 2004: those overweight were associated with significantly decreased mortality from non-cancer and non-cardiovascular disease (CVD) causes (about 69,300 fewer deaths) with no association with cancer or CVD, while those obese were associated with significantly increased mortality due to CVD (112,159 more deaths) not associated with cancer or non-cancer and non-CVD mortality. They concluded that excess mortality varied by cause, suggesting there has been a decrease in the association of obesity with CVD mortality over time, thus reducing its impact.

Results from Romero-Corral et al. (2006) that analyzed 40 studies with relatively limited follow-up periods (average of 3.8 years) that did make adjustments for various confounding factors, were consistent with Flegal et al.'s results. The hazard rates for all-cause mortality in these studies compared with standard BMI (between 20 and 25) were 0.87 for those overweight, 0.93 for those class I obese, and only 1.10 for those at least class II obese. Similar hazard rates were indicated for just cardiovascular mortality, although the cardiovascular hazard rate for those at least class II obese was 1.88 (those for overweight were 0.88 and class I obese 0.97). Those underweight (less than a 20 BMI) had a 1.37 hazard rate for all-cause mortality. The relatively favorable results for those overweight and in class I obesity of this aggregation of studies contrast with many other studies on this topic. The authors speculated that these findings might be explained by the lack of discriminatory power of the BMI to differentiate between body fat and lean mass, in addition to the relatively short follow-up periods involved in the studied included.

In addition to Flegal and Romero-Corral, there are at least three other American studies in which there is no observed adverse relationship between obesity (no matter what level) and mortality: the Veterans Exercise Testing Study (VETS, McAuley et al. 2010), the Aerobics Center Longitudinal Study (a series of papers including Sui et al. 2007), and the Lipid Research Clinics Study. Fitness level (in contrast with the level of physical activity) was studied in the first two of these studies by means of exercise testing results. The VETS study of male military veterans with cardiovascular problems and a mean age of 57 excluded current smokers and deaths in the study's first two years, with a follow-up period of a median about 7.7 years. This study experienced almost flat hazard rates over all BMIs, except for those underweight (VETS did not exclude those currently with a cancer diagnosis). In fact, both BMI and fitness were independently and inversely associated with mortality risk — when highly fit patients were compared by various BMI categories, those who were overweight and obese experienced

dramatic reductions in mortality risk (hazard rates 0.43 and 0.52, respectively). Explanations for this unusual result included possible reverse causation in clinically referred patients, increased coronary artery size due to the so-called “veteran effect” (a high percent of obese who are physically fit) and the survival effect. In other words, confounding variables might be involved; including the type of the people included in this study, and those in the upper range of the age group studied might be less susceptible to the negative effects of overweight. These two studies demonstrated that in the populations they studied, relative fitness level was a stronger predictor of mortality than BMI, that is, higher fitness levels eliminated the mortality risk of elevated BMI (demonstrating the fat-but-fit hypothesis).

The Canadian National Health Survey (Orpana et al. 2010) also indicates a significantly lower risk of death from those overweight over a 12-year follow-up period and a hazard rate close to 1.0 for class I obese compared with that of BMI between 18.5 and 24.9. About 18 percent in the studied population was older than 65. The fact that the hazard rates were so close to 1 for all higher classes of obesity leads one to wonder whether an unrecognized variable was omitted from the study, as it is otherwise inconsistent with most other reported experience of those morbidly obese. Other analysis of this survey data indicated that about a quarter of those overweight in 1994-99 became obese by 2002-03.

The findings of Mahta and Chang (2009), using findings from the Health and Retirement Study (1997-2004), were consistent with Flegal and Orpana. They studied middle-aged adults born between 1931 and 1941. Although they found adverse experience for those underweight, the BMI/hazard rate curve was at a minimum in the overweight category, with relatively small additional deaths for those class I obese. Although they found extra mortality hazard rates for those in obese classes II+, these extra mortality rates were in the range of 68 percent for females and 45 percent for males, much less than in many other studies. They attempted to eliminate the effect of pre-existing diseases and other confounding factors using several methods and models — none of these attempts supported any confounding by pre-existing illnesses or association between weight status and mortality, although they indicated that given the complex pathways among body weight, disease and death, they could not rule this out. In this study, they found the effect of confounding by socio-economic status (SES) appeared modest and most pronounced in the highest BMI category. I will note that the births in the Depression and early World War II era represent an unusual period with its reduced level of food available, although such effects have not been as pronounced in the United States as in the United Kingdom.

In a compilation of nine studies of the mortality of patients with chronic heart failure, Oreopoulos et al (2008) found lower mortality associated with overweight and obesity (a 0.88 percent and 0.93 risk-adjusted hazard rate, respectively). Protective mechanisms that would lead to these results have not yet been identified.

Significant controversy and in fact confusion has arisen because of these seemingly inconsistent results. The following provides further contrasting results.

Berrington de Gonzalez et al. (2010)’s study of pooled data from 19 large-scale prospective studies comparing BMI and mortality among white adults (excluding Hispanics) came to opposite conclusions. They found J-curve or U-curve relationships between BMI and

mortality hazard rates compared with a benchmark (1.0) of BMIs between 22.5 and 24.9 in all of the 19 studies. The pooled experience consisted of 1.46 million adults with a median follow-up period of 10 years (minimum of five years), with primarily self-reported heights and weights at the baseline of the studies. The results, according to Berrington de Gonzalez, were similar to the 900,000 Prospective Studies Collaboration.

Although the BMI/hazard rate curves varied somewhat for the individual studies included in Berrington de Gonzalez, they all found that in general both overweight and obese had experienced extra mortality than the benchmark BMI range. Most incorporated information on pre-existing conditions, especially cancer, as many were primarily designed to study cancer. Most excluded pre-existing cancers (to correct for resultant weight loss); this exclusion reduced observed additional mortality from those who were underweight, but increased the resultant mortality of those overweight and obese. They studied applicable adjustments needed for other confounders, such as physical activity level, alcohol consumption, educational level and marital status that reduced the hazard rates somewhat, but they did not affect the results significantly.

In contrast, both NHANES and the Canadian National populations included smokers and those with pre-existing diseases. Arguments for these inclusions are that their exclusion would bias and make results of any analyses difficult to extrapolate to the general population; the counterargument is that these contribute to weight loss and are thus powerful confounders, with inclusion lacking validity and comparability across BMI levels, thus biasing the results. Nevertheless, stratification or exclusion rather than adjustment may be appropriate because smoking is strongly related to obesity and mortality. In the studies covered in Berrington de Gonzalez, the long-term follow-up period strengthened rather than weakened the association between BMI and all-cause mortality, the expected result if pre-existing illness confounds this association. In addition, the relationship between low BMI and all-cause mortality is stronger among former smokers who quit less than 20 years ago than among current smokers.

Manson et al. (2007) criticized the use of the NHANES for the study of mortality because (1) the number of participants is not sufficiently large to reach its conclusions; (2) the follow-up period is currently too short; (3) due to reverse causation, those with chronic diseases have not been excluded, although secondary analyses that excluded smokers and those with recent weight loss were studied simultaneously; (4) use of 18.5 to 24.9 rather than 23 to 24.9 as the referent group would have increased the number of excess deaths by 45,000; and (5) a large percent of reported deaths occurred among those older than 70 at the time of BMI assessment resulting in the possible existence of illness-induced weight loss and loss of muscle mass. Responding to this criticism, Flegal et al. (2007b) noted they believed the effects found did not introduce a significant bias but did agree the health effects of overweight and obesity are complex and multifaceted.

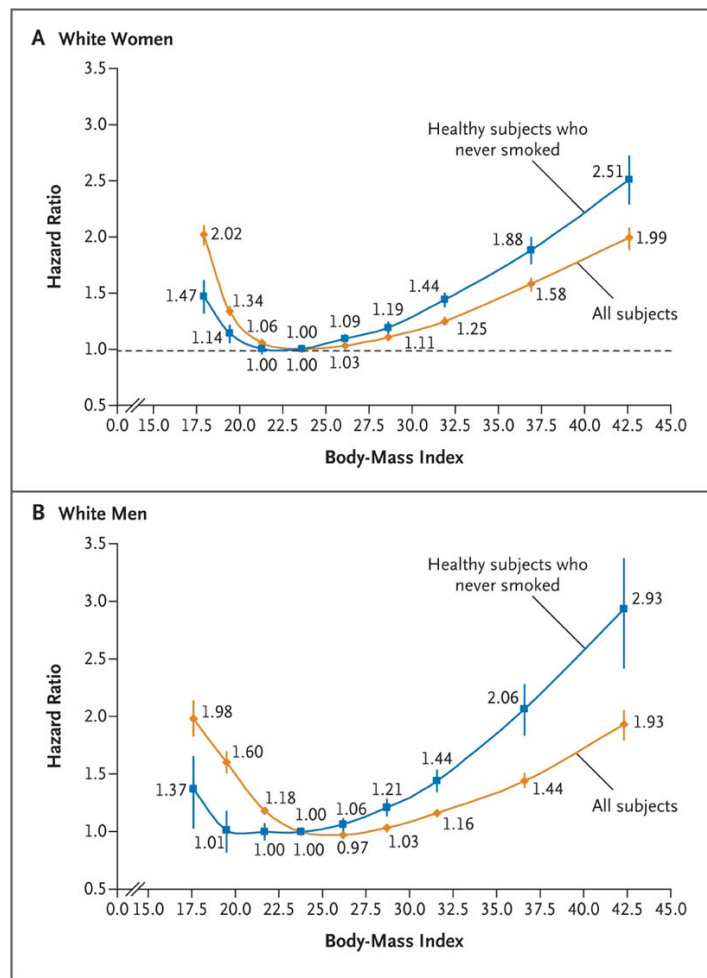
Berrington de Gonzalez further found the NHANES and Canadian studies covered smaller populations than theirs, so may not be as credible. Since the adjustment for cardiorespiratory fitness, a proxy for physical wellness, has blunted the paradoxical association between BMI and total mortality in these studies, it is possible one or more other variables may not have been reflected in studies in which the obesity paradox exists. An example of results where adjustments were made is the Physicians' Health Study, one of the studies included in this

pooled data set with a wide range of adult ages (44 to 84) that studied 100,000 males, in which a linearly increasing pattern of extra mortality beginning in the overweight category was found, having accounted for several potential sources of bias, including age, smoking, alcohol consumption, physical activity, prior disease, and interactions between BMI and both prior disease and smoking.

As can be seen in Table 14, the hazard rates in the pooled Berrington de Gonzalez study decreased by age, but the additional deaths per 1,000 studied increased. Also as can be seen in Table 15, the hazard rates for the class II+ obese categories increased with years of follow-up, although those for overweight and class I obese did not. This corroborates the long-term lagged effect of morbid obesity, and may indicate why some studies with limited follow-up periods have not shown significant additional mortality, as the percent morbidly obese has increased dramatically lately. For those underweight (not shown here), the hazard rates decrease with follow-up period, probably indicating the effect of recent weight loss or those lean and fit in this category.

Figure 13

Estimated Hazard Rates for Death from Any Cause According to Body-Mass Index for (1) All Study Participants and (2) Healthy Subjects Who Never Smoked



Source: Berrington de Gonzalez (2010).

TABLE 14
Hazard Rates and Extra Deaths per 1,000 Exposed Lives by Age, Years of Follow-up and Cause of Death

Age Group Hazard Rates	BMI Range				
70-84	1.04	1.15	1.24	1.59	1.91
70-84	0.7	2.5	3.9	9.7	14.9
>14 years	1.12	1/19	1.41	2.04	3.11
Other	1.02	1.13	1.29	2.00	3.00

Source: Berrington de Gonzalez (2010).

In the Build and Blood Pressure Study of 1959, a large-scale mortality study of insured lives sponsored by the Society of Actuaries, those moderately overweight experienced extra mortality of about 20 percent compared with those of the standard class for the first five years after policy issue, rising slightly in the next five years and continuing upwards over a period of another 10 years to about 35 percent additional mortality, with those markedly overweight males rising to nearly 85 percent after 15 years had elapsed.

In the 1979 version of this study, the hazard rates for those 30 to 60 percent overweight improved by 10 to 15 percent compared with those of similar weight in the 1959 study, thus sharing in the overall population's mortality improvement. As reported by Brackenridge and Elder (1998), men weighing 20 percent above average experienced mortality from all causes 20 percent more than those of standard weight. These men experienced a 15 percent higher mortality rate due to CHD, 150 percent higher rate due to diabetes and 20 percent higher rate due to digestive diseases. The effect of elevated weight then increased significantly as weight increased. There was a slight J-curve effect at body weights for both men and women who were less than 20 percent of average weight of about 110 percent of mortality of those at average weight, although these included smokers. The results of the 1979 study have also been used to argue that optimal body weights increase at higher ages.

Life insurance statistics indicate the extra mortality associated with being overweight when accompanied by one or more additional impairment is more than additive in nature. Any significant deviation from what would be normal weight, particularly associated with a recent rapid decrease or increase, has been considered to usually be a signal of the existence or emergence of a serious disease.

It has been recognized by those in the life insurance industry that individuals with different builds (somatotypes) have different mortality characteristics. In particular, the relative proportions of fat, muscle and bone that contribute to excess weight is important. Nevertheless, insurance studies have not generally reflected somatotypes, although a subjective adjustment for large abdominal girth is usually taken into account. Those whose excess weight consists mainly of muscle or bone have usually been considered better mortality risks than those of the same weight. Separately, "pear-shaped" people tend to be healthier than "apple-shaped" people.

According to Roudebush et al. (2006), life insurance experience for policies issued between 1989 and 2003 with an average duration of 2.5 years generated from the Impairment

Study Capture System produced standardized mortality rates (SMRs) that rose modestly as BMI increased until reaching severe obesity levels. Nonsmoker insureds whose elevated build was the only reported impairment experienced SMRs of 265 percent for BMIs less than 18.5, 130 percent at BMIs of 30 to 34.9, 160 percent at BMIs of 35 to 39.9, and 239 percent at BMIs greater than 40, although given the short follow-up period with this relatively new insurance database it is unlikely all the adverse effects of overweight and obesity have had time to reveal themselves. Mortality rates were greatest in policy durations 1 and 2, especially for those underweight and the extremely obese, possibly due to anti-selection.

Calle et al. (1999) found that, in general, the relative excess mortality risk of being obese expressed in terms of additional deaths per thousand of population at older ages was greater than at younger ages, but when expressed as a hazard rate (that is, as a multiple of the benchmark mortality rate, at say BMIs between 23.5 and 24.9) it decreased. In general, they found that hazard rates took the shape of a J-curve, with the lowest hazard rates at BMIs in the range of 23.5 and 24.9, although at some age groups the lowest hazard rate was at somewhat lower BMIs. For class II+ obese, the hazard rate varied between 1.86 and 2.75 for those younger than 75, depending on age group and gender, and between 1.41 and 1.53 for those 75 and older.

Observations from the Framingham study found “overweight can be an independent, long-term predictor of cardiovascular disease” and both being overweight and obese for adults are associated with large decreases in life expectancy and increases in early mortality, similar to those seen with smoking (Peeters et al. 2003). Many large-scale studies focused on cardiovascular disease risks have usually come to similar conclusions.

Fontaine et al. (2003) estimated that, based on NHANES III, the life expectancy of those severely obese is on average five to 20 years less than that of those of normal weight. Olshansky et al. (2005) estimated that, using the 2000 U.S. Life Table as a base, the expected effect of the elimination of obesity (he assumed that population segment experienced mortality similar to mortality of a BMI of 24) to be an increase in life expectancy at birth of one-third to three-quarters of a year. The Olshansky paper has been criticized by some who have pointed out that since those who are obese also have other risk factors, it is inappropriate to assume the total difference in life expectancy indicated by this single factor can be attributed solely to obesity. On the other hand, since the time Olshansky prepared his estimate, the obesity prevalence rate reported by NHANES increased by about 10 percent for males; at the same time, there has been a reduction in mortality rates from cardiovascular disease, a principal cause of excess mortality of the obese.

Ezzati et al. (2008) indicated that, based on U.S. mortality statistics at a county level, an increased mortality rate inequality is emerging among population segments. Life expectancy declined in 11 counties for men and 180 counties for women, corresponding to 4 percent and 19 percent of the male and female U.S. population, respectively. This decrease was concentrated in the Deep South, along the Mississippi River and in Appalachia, extending to the southern part of the Midwest and Texas. This differential trend did not occur in the prior two decades. The differential was primarily due to an increase in cancers, diabetes and chronic obstructive pulmonary disease. This trend is consistent with the geographic patterns and trends in smoking, high blood pressure and obesity.

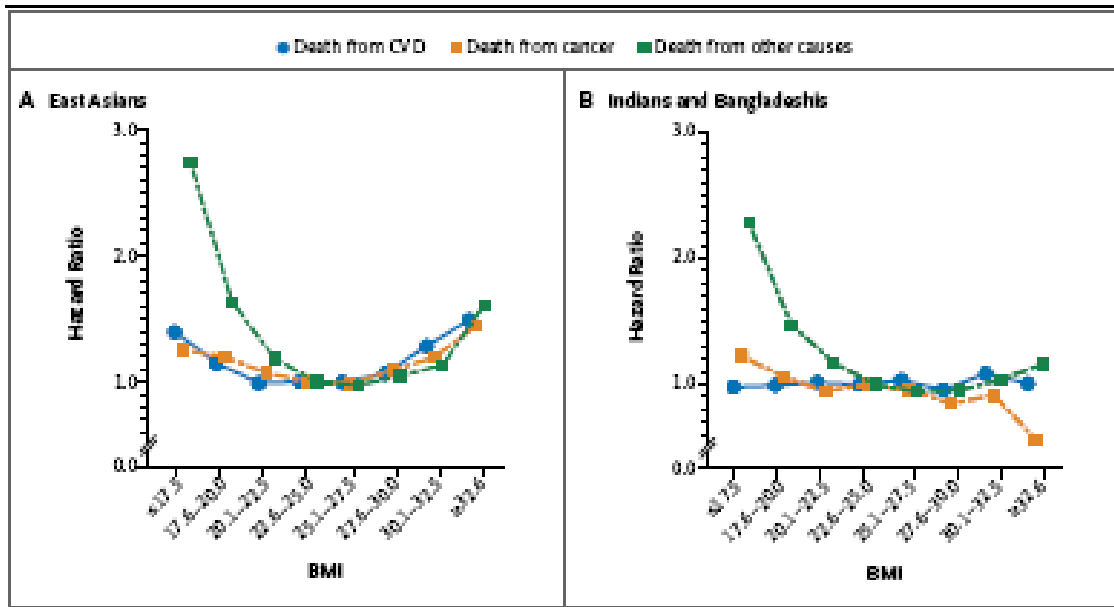
Cutler et al. (2007), projecting mortality over the next 10 years, incorporated estimates of changes in smoking, education, drinking, hypertension, cholesterol and obesity in developing two alternative scenarios: (1) current levels of medication continue (NHANES 1999-2002 indicates that 60 percent of those with hypertension take anti-hypertensive medication and 35 percent of those with adverse cholesterol levels take cholesterol-lowering medication) and (2) all those with adverse blood pressure and cholesterol levels take medication and they are 75 percent effective with respect to this treatment (percentage diagnosed and taking medication as directed). In the first scenario, the effect of projected continued increases in obesity is expected to more than offset the continued favorable effect of the recent reductions in smoking and by itself is expected to result in a 13 percent increase in age-adjusted mortality. In contrast, in the second scenario in which the mitigating factors are more effective than current experience, the two factors (obesity and effective medical treatment) offset each other. Cutler indicated the magnitude of the effect of obesity was reflected through the use of the nonlinear relationships between BMI and weight increase, and between BMI and health risks.

Stewart et al. (2009) forecasted life expectancy (and quality-adjusted life expectancy) for an 18-year-old, assuming continuation of historical trends in smoking and obesity. The 2003 Medical Expenditure Panel Survey (MEPS) was used to assess the effects of smoking and BMI on health-related quality of life. Stewart et al. found that the negative effects of increasing BMI more than offset the positive effects of declines in smoking. In this base case, the increase in the life expectancy of the 18-year-old was reduced by 0.71 years between 2005 and 2020. In contrast, if adults became nonsmokers and of normal weight by 2020, life expectancy would increase by 3.76 years or 5.16 quality life years. Since the increase in the percentage of obesity has been decelerating (although the rate of smoking has also stabilized recently), Stewart et al. estimated the threshold rate of increase where its adverse effects would be equal to the beneficial effects of the decline in the rate of smoking (where about 45 percent of the population would be obese by 2020). This trend reduces the gains in life expectancy by 1.02 years, while the gain through smoking would lead to an increase in life expectancy of 0.31 years, with the effect becoming greater over time. This increase in obesity prevalence rates is expected by the authors to be due to the long-term effects of the current all-time high rate of obesity of children. They note that even modest weight loss and reductions in smoking at the individual level can lead to substantial effects on the overall health of the population.

In a pooled mortality analysis of more than 1.1 million Asians and BMI, Zheng et al. (2011) grouped 19 age groups according to East Asians (primarily Chinese and Japanese, and some South Koreans) and South Asians (primarily Indian, but also some Bangladeshi), with a mean follow-up period of 9.2 years. In both groups, significant extra deaths were associated with those underweight (BMIs of less than 15), primarily due to respiratory diseases (the dominant cause of the “other” in Figure 17), although the authors caution the reader that reverse causation might exist because infectious diseases could not be ruled out at benchmark, because information regarding the existence of infections or chronic lung disease diagnosis was not available at that time. Although the pattern of additional mortality with increasing BMI is broadly similar to that of North Americans and Europeans (a hazard rate of upwards of 1.5 for class II+ obese compared with those with 22.6 to 25.0 BMIs), no additional deaths were apparent from overweight or obese individuals. Note that there was also far less experience in those categories, and there was some additional mortality for the class II+ obese (a 1.27 hazard rate). The hazard rate curves by

cause of death for the two categories of Asians are shown in Figure 15. A study of the relationship of waist circumference was not made; such a study may be particularly important in Asian populations. One conclusion from this pooled study is that the combination of racial populations needs in such studies should be performed with caution.

Figure 15
Association Between BMI and Risk of Cause-Specific Death in Two Asian Populations



Source: Zheng et al. (2011).

Two studies of the joint effect of smoking and obesity as measured by BMI show some interesting results. In a study of Asians, the joint effect of smoking and obesity are somewhat greater than if they were assessed separately. In this study, Huxley et al. (2009) evaluated a pooled analysis of 378,519 adults. The adverse effect shown in that study began above a BMI of 22, consistent with other findings that Asians are more affected by extra weight than others. Freedman et al. (2006) also found that smoking and obesity interacted. In that study, obese current smokers younger than 65 experienced mortality at a level of six to 11 times that of nonsmokers, while smokers older than 65 experienced mortality at a level of two to four times. Obese former smokers younger than 65 had notably lower mortality rates than current smokers. The study found that being a current smoker constituted a higher mortality risk than being obese.

The Nurses' Health Study II (with a 24-year follow-up period) studied the attribution of deaths to various factors. Van Dam et al. (2008) reported 28 percent of deaths could be attributed to smoking and 55 percent to the combination of smoking, being overweight, lacking adequate physical activity and a low diet quality. Each of these lifestyle factors independently and significantly predicted mortality.

For several types of situations and demographic groups, some excess body fat can be protective, although these contribute only a very small amount of total mortality rates:

- In women, a reduction in the incidence of premenopausal breast cancer and the rates of hip fractures has been found.
- In older women, some excess fat may produce extra estrogen that helps slow bone loss and insulate bones from fall-related injuries that occur about 9 percent annually for men and women older than 75. As the aged inevitably lose weight, additional weight to start from may provide some protection against extreme frailty.
- Conditioned athletes may be overweight due to being fit and having dense muscle tissue. Being fit and overweight in these cases can be protective.
- Some ethnic groups, possibly including blacks, may have a “best” BMI somewhere greater than the current “normal” BMI category, which contrasts with the best BMI level of Asians that at least for cardiovascular diseases are somewhat less than the normal BMI category, as noted above. A relatively weak relation between weight and mortality has been observed in some studies of mortality of black females, the American ethnic-gender category with the highest prevalence of overweight and obesity. Possible hypotheses include less access to or less frequent use of health care services along with less adequate communication with physicians; different patterns of carrying their extra weight with whites having greater visceral adipose tissue, despite the greater overall obesity prevalence in black women; and different exposure to competing mortality risks (e.g., different levels of homicide that would be constant across all BMI categories). Further research is needed to confirm these hypotheses.
- Children may have higher normal fat levels during growth spurts and around puberty.

Although the value of weight management and weight loss is often discussed, it is not often explicitly studied. This is due in part because the causes of weight loss should be distinguished to ensure a useful study. Reporting on the results of the Copenhagen City Heart Study with a long follow-up period that studied overweight and obese participants without pre-existing diagnosis of diabetes, stroke, heart disease or cancer, and who were categorized as either being physically active or inactive, Ostergaard et al. (2010) indicated that while losing weight, those who were physically inactive experienced excess mortality compared to those who were active (a hazard rate of 2.25 for men and 1.43 for women). However, losing weight while remaining physically active was also associated with excess mortality compared with those who were physically active whose weight was stable (1.72 for men and 1.57 for women). For those who were physically inactive, excess mortality was also associated with those who lost weight compared with those whose weight was stable (men with a hazard rate of 2.25 and women a hazard rate of 1.43). To be physically active is important for your health whatever the BMI.

Lags and Follow-up Periods

The long period between becoming obese and development of chronic health problems or consequential death has made the study of these interconnections difficult. Recognition of the effects of such time lags is important in the proper assessment of both smoking and obesity. Trends in such factors as smoking and obesity have moved in different directions, as the largest declines in smoking occurred between 1960 and 1980, whereas the largest increase in the prevalence of obesity occurred between 1980 and 2000. In part because of favorable trends in smoking and the continuing favorable effect of technology, it is likely we have not yet been able to fully discern the long-term consequences of trends in other aspects of human behavior, particularly because of the huge upswing in obesity and the shifts in causes of death, e.g., the reduction in cardiovascular disease. The limited information regarding the nature, timing and severity of the long-term effects of the many factors involved represents a challenge for those involved in long-term mortality projections.

In some cases, it is the cumulative exposure to additional weight and adiposity tissue rather than its status at a particular point in time that more significantly contributes to higher mortality rates. If, for example, someone was obese at age 40, became diabetic at age 50, had a heart attack at age 60, then lost a lot of weight and died at age 65, this person's BMI at 40 would be more significant than that at age 62. This long latency period raises concerns with respect to the effects of the recent increase in obesity prevalence, in that recent reported studies may not have had a sufficiently long follow-up experience period to properly recognize the effect of the overweight and obesity surge over the past 30 years.

Some believe it may take 10 to 20 years or more for obesity to have its full impact on, for example, diabetes and cardiovascular mortality, although Allison et al. (1999) found that in the six studies they reviewed, there did not appear to be such a relationship. Dyer et al. (2004), in reporting on results of the Chicago Heart Association Detection Project in Industry study in which deaths from the first 15 years were excluded in its 25-year follow-up study, found a positive association in all age/gender subgroups between BMI and mortality of those overweight and obese, indicating the importance of that follow-up period for cardiovascular disease. This long manifestation period affects the ability to directly measure at the current time, but some studies have reflected this, e.g. the Nurses Study. This also emphasizes the importance of health condition in childhood/adolescence.

Mortality experience from the life insurance industry has confirmed the importance of this lag effect. A mortality study of those overweight conducted by Cologne Reinsurance Co. reported in 1969, indicated that mortality rates associated with being overweight as the sole impairment remained relatively low for eight years and only then started to show a modest rise in comparison with those of standard weight. Lew and Gajewski (1990) found the adverse effects on mortality of being overweight appear to be delayed, sometimes for 10 years or longer. In one of the longer study periods, reported mortality rates of overweight men rose over a 35-year period after insurance policy issuance based on an earlier investigation conducted by Provident Mutual Life.

The results from Berrington de Gonzalez in Table 15 above showed a significant increase in hazard rates for results after a 10-year follow-up period compared with those for earlier periods, particularly for cardiovascular mortality and for those morbidly obese. In addition, Adams et al. (2006) separately studied experience into its first five years and more than five-year follow-up period. For men during the less-than-five-year follow-up period, the hazard rates were 0.93, 0.99, 1.17 and 1.54 for those who were overweight, obese class I, obese class II and obese class III+, respectively, while for those with more than a five-year follow-up, the rates were 0.99, 1.19, 1.52 and 2.11, respectively. For females, the corresponding rates were 1.03, 1.10, 1.30 and 1.65, compared with 1.06, 1.25, 1.66 and 2.20. This suggests that a long lag period exists between a given categorization of BMI and resultant mortality, and that results of studies with limited follow-up periods should be viewed considering these lag effects.

Another example of a large-scale long follow-up period study, more than 250,000 Danish schoolchildren (initially measured between ages 7 and 13 for boys and ages 10 and 13 for girls, born between 1930 and 1976) were followed through their early adulthood (Baker et al. 2007). This study covered all socioeconomic groups, although all were white due to the nature of the population of Denmark. They subsequently underwent mandatory annual physical examinations. Fatal and nonfatal CHD events were studied. The rate of both fatal and nonfatal coronary heart disease (CHD) increased monotonically by BMI level in childhood, that is, the risk was higher for higher BMIs at a given childhood age with no J-curve relationship. Girls showed the same increasing pattern relating to BMIs as boys, but at a lower level.

Even when adult BMI was factored in, a Norwegian study of women who were obese as teenagers found they were about 30 percent more likely to die by middle age than those with an average teenage BMI.

In summary, the effect of obesity on certain chronic diseases can have a long latency period, only fully identifiable through the use of a long follow-up period. Current and future studies are encouraged to continue their follow-up period for as long as possible so these latency effects, if they do exist, can be further studied.

Older Ages

Many studies that have analyzed the mortality of those of older ages have shown the relationship between BMI and mortality weakens (at least on a multiplicative basis) at advanced ages, in some cases over age 75. BMI, a surrogate or indirect estimate of adiposity, may underestimate the extent of adverse adiposity tissues in older adults compared to the effect of the BMI of younger adults. This may in part be due to the loss in muscle and bone mass that can be the result of inactivity or illness. Thus, the interpretation of the effect of measures based on weight and height can be complicated.

Note that studies such as the one reported on in Calle et al. (1999) have found that the absolute (not proportional) additional risk of death associated with adiposity was highest at the oldest ages.

An example, similar to the example given in the section of this paper immediately above relating to follow-up period, can illustrate a reason for this lower correlation where an insufficient follow-up period is provided for: an individual might be obese in her 40s with an onset of diabetes in her 50s, which in turn might lead to a myocardial infarction in her 60s, heart failure and weight loss at age 70 with death shortly thereafter. In this case, an epidemiological study that only measures BMI at age 70 after the weight loss occurs would not be able to identify the original cause of the premature death without a very long follow-up period.

Such a causal pathway problem can lead to misleading study results. This also involves reverse causation that can contribute significant problems in interpreting the results of studies of mortality of the older segment of the population, in that many chronic diseases lead to weight loss at those ages, particularly when relatively short follow-up periods are involved. Studies with longer follow-up periods and exclusions of pre-existing conditions (e.g., those who smoke) can provide a better perspective on the problem being assessed than studies of mortality and obesity in the elderly measured on a concurrent basis. To obtain more useful results, it is often desirable to segment experience by major age categories.

Janssen and Mark's (2007) meta-analysis of 26 studies examining the effect of elevated BMI on mortality risk of those 65 and older found that for those overweight (not obese), an average hazard rate (compared with normal BMIs) was 1.00, while for those of moderate obesity it was 1.10 (note that 10 percent greater mortality at older ages can be considerably greater in terms of mortality rates expressed in terms of number of deaths per 1,000 population than produced by a much higher hazard rate at younger ages).

The Cardiovascular Health Study that followed 4,968 people older than 65 for a follow-up period of up to nine years was reported on by Janssen (2007). In it, the all-cause mortality risk for those overweight (defined in terms of BMIs for those greater than age 65) was 11 percent lower in contrast with those in the normal BMI category, in contrast with several of the morbidity outcomes that were less favorable than those of normal BMI, particularly for diabetes (78 percent higher) and arthritis, for both of which weight is a significant risk or causative factor. The higher rates for diabetes are of concern, due to the significant increase in diabetes prevalence at older ages in recent years, even after adjustments are made for possible covariates (e.g., age, gender, race, socioeconomic status and prior condition [for myocardial infarction, stroke and cancer]). Janssen suggested a BMI greater than 25 may be a more appropriate upper range cut-off point for a "standard" class as applied to older adults.

Biggs et al (2010) also reported on the Cardiovascular Health Study, then with a follow-up period of an average of 12.4 years. They found that, although both BMI and waist circumference were independently related to diabetes, the use of a joint model of BMI and waist circumference indicated a strong association of waist circumference and diabetes. BMI, waist circumference and hip-waist ratio had similar hazard rates for diabetes. These rates were appreciably lower for those 75 and older compared with those between ages 65 and 74. They indicated that standard anthropometric measures may not adequately quantify body fat due to age-related changes in body composition, including decreases in skeletal muscle mass and height. In addition, they concluded that fat distribution within a person's body may be more important than absolute fat mass for older adults. The effect of weight loss was not associated

with a reduction in diabetes risk over a three-year period, unlike the results in younger populations.

According to a meta-analysis conducted by Heiat et al. (2001) of 13 studies reporting on those at least age 65 with follow-up periods of between three and 23 years, only two (the Framingham Heart Study and the American Cancer Society Cancer Prevention Study) indicated a positive relationship between all-cause mortality and BMI. These two studies showed a higher optimal BMI of at least 27. The other studies showed either no or a negative relationship between mortality and BMI. For cardiovascular disease, there was a U-shaped BMI mortality curve, with BMI at the lowest mortality level not reached until BMI hit 31 or 32, with a less steep upward slope than at younger ages. Various analysts have hypothesized that: (1) a higher percent of those previously obese had already died by that time, (2) those of greater weight left were stronger and more healthy, (3) excess body fat may be less important in the elderly and may provide important protective reserves, and (4) those older had far more frequent multiple health hazards that might mask the underlying relationships. These hypotheses are similar to findings in other studies of the aged.

Crimmons and Saito (2005) found that in a study of 7,000 participants over age 70, the remaining life expectancy of the obese was quite similar to the life expectancy of those of lower weight, 12.3 years for non-obese males compared with 12.4 years for obese males. However, for those who were not disabled (measured by activities of daily living they are capable of managing independently), the nondisabled life expectancy was 9.8 years for the non-obese, compared with only 8.4 years for the obese, indicating a significant disability risk for the overweight and obese aged. Corresponding life expectancy values for females were 15.3 years compared with 15.4 years, along with 8.1 and 7.4 nondisabled years, respectively.

McTigue et al. (2006) reported on a study of 90,185 participants in the Women's Health Initiative Observational Study with an average follow-up period of seven years. They demonstrated that at older ages, hazard rates decreased by age, but the hazard rates increased by degree of obesity. For example, for white women without adjustment for smoking, education, region and physical activity, the hazard rates are 1.17, 1.30, 2.05 and 3.34 for ages 50 to 59, compared with 1.15, 1.34, 1.62 and 2.35 for ages 60 to 69, and with 0.88, 1.04, 1.28 and 1.42 for ages 70 to 79. After adjustments for the just named factors, all of the values were reduced somewhat, but not by a significant amount. They also found that the risk of developing a comorbidity increases as the degree of obesity increases, although the extent of this increase varies by sex, racial/ethnic group and genetic factors.

Lozonczy et al. (1995) found that in the Established Populations for Epidemiologic Studies of the Elderly that followed 6,387 whites 70 and older during the 1980s, the reduction in hazard rates between those in their 50s and their 70s was explained in part by weight change. Compared with those with stable weight during this period, those who lost 10 percent or more of their weight after adjustment for changes in health status, eliminated the extra risk of death associated with low weight. Based on the results of this study, the inverse association of weight and mortality in old age appears to reflect illness-related weight loss from heavier weight in middle age.

Krueger et al. (2004) found, using NHIS data for U.S. adults 60 and older, that those obese have higher risks of overall mortality and mortality due to circulatory disease and diabetes. Smoking tended to suppress the relationships between obesity and overall mortality and mortality due to circulatory disease and cancer.

Rimm et al. (1995) and others have observed that BMI may be a less useful indicator of adiposity for those at advanced ages, whose fat tends to shift from peripheral to central body sites, with a resultant increase in their waist-to-hip ratio but with no change in BMI. Although Rimm found that men younger than 65 had a relative risk (compared to those with BMI of less than 25) of 1.72 for BMIs between 25 and 29, 2.61 for BMIs between 29 and 33, and 3.44 for those greater than 33 BMIs, corresponding relative risks for those older than 65 were much lower. In this study, the use of the waist-to-hip ratio metric provided a much stronger predictor of risk than did BMIs (a 2.76 hazard rate between the highest and lowest quintiles of the waist-to-hip distribution). Folsom et al. (2000), reporting on the Iowa Women's Health Study of 55- to 69-year-olds, likewise found that an abdominal-based metric was superior to the BMI in predicting mortality over its five-year follow-up period, that is, a multivariable-adjusted hazard rate for the highest quintile relative to the lowest one of 1.2, compared with 1.1 using waist circumference and 0.91 using BMI. In the Iowa Women's Health Study, all three metrics (BMI, waist-to-hip and waist circumference) were strongly correlated with the incidence of diabetes and hypertension, with waist-hip measure being less consistent with the other two with respect to cancer incidence.

Jenkins (2004), using data from the Asset and Health Dynamic Among the Oldest Old Survey, found that those overweight or obese are more likely to have more functional impairments. Jenkins found that obesity independently affected the onset of strength impairment, lower body mobility and activities of daily living problems. Separately, based on the increase in dementia found by Whitmer et al. (2008) associated with BMI and visceral adiposity, the use of long-term care facilities by those who become obese will likely increase in the future as those with greater weight reach old ages.

The effect of income, education and occupation also appear weaker at older ages, although income has been shown to have a greater effect among females. In addition, the flattening of the BMI/mortality curve at these ages may be in part the result of the "selective survivor" effect, that is, that many at higher risk (those most sensitive to adverse health efforts or burdened with other conditions) may have died at younger ages, with only the healthier obese individuals living at these ages. Note that a fixed BMI cutoff across all ages may lead to miscategorization of individuals whose body mass and skeletal structure change due to the aging process rather than due to their underlying health conditions.

The results of the following two studies relate the importance of physical activity and fitness in the elderly. Sui et al. (2007), reporting on the 12-year follow-up results of those older than 65 enrolled in the Aerobics Center Longitudinal Study, found fitness was a significant indicator of mortality (that is, a hazard rate for the first quintile of about three times that of the fifth quintile), independent of overall or abdominal adiposity. Those class I obese experienced about 30 percent higher rate of mortality while those class II or III obese mortality experienced 130 percent higher mortality rates, with those with a waist circumference of greater than 88 cm

for women and 102 cm for men having about 30 percent higher mortality rates. Thus, functional capacity was determined to be important for older individuals, including both those of normal and overweight. Manini et al. (2006) studied a randomly selected group of 70- to 82-year-old Medicare recipients in the Health, Aging and Body Composition Study with an eight-year follow-up period whose energy expenditure was measured over a two-week period. Those who reported low physical activity levels experienced elevated mortality, in that the highest tertile of energy expenditure through physical activity experienced about half the mortality rate than those in the lowest tertile.

Although this paper focuses on obesity, it should be noted that being underweight can represent a more significant hazard for the older old (particularly for those 85 and older), due to frailty that may be both a result of one or more diseases or exposures and may provide limited protection against others. The larger the number or severity of health conditions or diseases, the more difficult is the assessment. The mitigating effect of fitness may be even more important for older adults than for those at younger ages.

4. Morbidity and Health Care

Morbidity, a measure of poor quality health, can result in both human suffering and adverse financial consequences, including the cost of medical care services and loss of income, as well as requiring assistance in performing certain activities of daily living (ADLs). Although the risk of premature death, discussed in Section 3 of this paper, due in part to or as a result of conditions that obesity and overweight is serious enough, being obese and overweight may have an even greater adverse effect on health care costs and suffering. The threshold for adverse morbidity resulting from the conditions that being overweight and obese exposes a person is lower than the corresponding threshold for mortality risk, increasing the cost for many affected. Although some studies indicate that being overweight may not constitute a significant mortality risk, being overweight is both a necessary stage in developing obesity and a health risk factor in and of itself.

There has been significant improvement in overall mortality rate due to improvement in treatment of cardiovascular disease risk factors over the past several decades. However, this mortality improvement has come at the cost of increased health care costs due in part to more aggressive treatment of cardiovascular disease risk factors.

The Surgeon General (2001) indicated that morbidity due to obesity in the United States may be as great as that due to poverty, smoking or problem drinking. Thorpe et al. (2007) wrote, “The only way to get health care costs under control is to find ways to reduce obesity. ... We have to manage patients with chronic conditions more effectively, and we have to find a way to prevent this rise in obesity.”

Physical disabilities resulting from obesity include skeletal and joint problems such as orthopedic disorders and carpal tunnel syndrome, which are not usually associated with mortality, as well as respiratory problems such as sleep apnea, as those who are obese tend to have an increased demand for ventilation and breathing workload, respiratory muscle inefficiency, decreased functional reserve capacity and expiratory reserve volume, and closure of peripheral lung units. Others possibly less obvious non-mortality risks include increased use of cesarean deliveries, and pre-existing and gestational diabetes for pregnant women.

In general, three factors contribute to the health care cost associated with obesity: (1) the increase in the number of obese, (2) the aging of the obese population that will inevitably result in increases in co-morbidities and health care costs, and (3) the increase in the cost of treatments for obesity-related illnesses.

Studies of the cost of specific disease or health conditions such as obesity have taken several forms, including (1) a “disease cost” or “prevalence” approach that measures the direct and indirect economic impact of a risk factor or disease on the health care system and society, (2) the “number of years of life” and years of healthy living lost as a result of the risk factor or disease, and (3) an “economic evaluation” or a cost-effectiveness approach, estimating the cost per year of life lost or cost/utility given alternative action choices.

The disease cost approach can be addressed in at least two ways. One involves estimating current costs associated with a condition in a given year based on the current population, and what those costs would be if no one had the condition, then estimating those two sets of costs for each subsequent year. The second involves estimating future costs for a given population at a given age, say 20, if that person either had or did not have the condition. The differences between these two scenarios are a result of differences in the characteristics of the population being evaluated. In some studies, the effect of the difference in expected mortality is taken into account (e.g., if the obese are expected to have a greater mortality rate, there will be fewer of them alive in later years) and thus lower costs in those years.

In a study that attempts to project additional costs at current cost levels or at their present value, the determination of appropriate discount rates and health cost inflation can significantly affect its conclusions. In addition, incorporating numerical values for the value of a person's life and healthy life, which are subjective at best, can also prove to be problematic. Appropriate sensitivity to alternative values should be included.

Studies of the overall cost of obesity have usually separately evaluated (1) direct health care costs that include preventive, diagnostic and treatment services, including the cost of associated bariatric surgery and cost associated with dieting, and (2) indirect health care costs that include wages lost, decreased productivity, absenteeism and value of future lost earnings caused by premature death. It should be noted that neither of these sets of factors include intangible costs, such as quality of life and psychological harm or public costs/benefits, such as a reduction in future social insurance or related benefits whose amount is affected by premature death. These can be useful in cost-benefit analyses of public policy decision making; however, due to the nature of the assumptions used, it can be relatively easy to skew the results if care is not taken. Sensitivity testing the results to alternative key assumptions adds value to such studies. Because of this, it is always important to disclose the assumptions used in communicating the results of such a study.

Roux and Donaldson (2003) compared the advantages of using a cost-of-illness approach to a solutions-based approach. They indicated that the cost-of-illness approach aims to quantify all direct and indirect costs attributable to a disease. It includes opportunity costs associated with the allocation of resources to the interventions aimed at managing and alleviating the conditions. The solutions-based approach (often used for decision-making and priority setting) is driven by a comparison of incremental costs and incremental benefits resulting from interventions aimed at controlling the illnesses and their consequences. Opportunity costs in the latter approach include lost earnings and differences in productivity costs between the populations compared. According to the MEPS, treatment costs for diabetes more than doubled, from \$18.5 billion in 1996 (in 2007 dollars) to \$41 billion in 2007.

Studies have come to different conclusions regarding the source of differences in health care costs. However, most have attributed higher health care costs to the use of more prescription drugs and primary physician visits, although some have also found more emergency department/outpatient clinic visits, specialty care clinics, number of diagnostic services, and hospital visits (Bertakis and Azari 2005) are made by the obese. Peytremann-Bridevaux and Santos-Eggimann (2007) indicated that based on the Survey of Health, Aging and Retirement in

Europe, additional costs were primarily due to more medication use, ambulatory care and, for women, increased hospitalization.

The approach taken to estimate the cost of obesity (or any other condition) depends on the problem for which models are developed. As a result, it is important to be clear about the basis for the quantifications performed. The following summarizes the results of many of the major studies conducted to date:

- Colditz (1992) evaluated the major consequential diseases associated with obesity using the results of other studies as input. First, he estimated the additional cost associated with obesity by condition (diabetes, gall bladder, cardiovascular disease excluding hypertension, hypertension and cancer) using a disease approach to be about 5.5 percent of total U.S. health care costs. To supplement this, he added an assumed percentage of costs for other relatively minor conditions. In addition, he estimated the costs attributable to severe obesity (class III and IV) for a 34-year-old man and a 34-year-old woman, in part to estimate the total cost of bariatric surgery, including the cost of the surgery and savings due to consequential improved health effects that in total he believed to be minor in comparison. He then added to this half the cost associated with muscular-skeletal disorders to derive a total of about 7.8 percent. Separately, he estimated that \$33 billion was spent in 1992 on weight reduction products and services (including low-calorie food and diet sodas), although this did not include their possible adverse side effects.
- Wolf and Colditz (1998) estimated a direct cost of \$51.6 billion and indirect cost of \$47.6 billion in the United States in 1995 (equivalent to 5.7 percent of U.S. national health spending in that year).
- Quesenberry et al. (1998), based on a study of Kaiser Northern California (a health maintenance organization) members over a one-year period, reported a 44 percent greater amount of health care costs for class II and heavier obese and a 25 percent greater cost for class I obese compared with those with BMIs in the standard BMI range. They also reported that class I obese had 14 percent and class II+ obese have 25 percent more physician visits than those of normal weight.
- Allison et al. (1999), using a prevalence-based approach, estimated per capita health costs by age, reflecting the additional expected mortality for the obese that, due to fewer obese living in the later years of the projection period, can reduce aggregate longer-term costs. Allison compared expected future health care costs of individuals age 20 to 85. Two scenarios were compared, using a constant relative cost ratio for each (health care costs of the obese to that of the non-obese) of 2.15 and 1.35, corresponding to the assumed ratio at BMIs between 29.0 and 31.9, and 25.0 to 26.9, respectively. Their resulting estimate was a reduction of about 5.7 percent and 4.3 percent for the two scenarios compared to what the costs would be had there been no obese in the population.

- Thompson et al. (2001) found that obese adults had 48 percent more inpatient days per year and 1.8 times more pharmacy dispenses, including six times the number for diabetes and 3.4 times the number of cardiovascular medication dispenses.
- Sturm (2002) estimated that the overall health status of being obese is roughly equivalent to the health status of someone 20 years older with a normal BMI, the effect of which greatly exceeds the association of either smoking or problem drinking. Obesity was associated with a 36 percent increase in inpatient and outpatient spending and a 77 percent increase in the cost of medications compared with corresponding spending in the normal weight range, and a 21 percent and 28 percent increase in spending in these two health care cost categories, respectively, comparing current and prior smokers to those who never smoked. He associated various conditions with the following annual increases in cost for a person with normal weight:
 - obese: \$395
 - overweight: \$125
 - current or ever smoker: \$230
 - problem drinking: \$150
 - aging: \$225

A primary reason that obesity was assumed to have a larger effect than smoking is that obesity has a more significant effect on heart disease, hypertension and diabetes, all of which tend to be chronic conditions with long-term drug regimens, while smoking has its strongest effects on cancer and lung disease that, although costly, tend to be less common and lead to death more quickly than does the chronic conditions more associated with obesity.

- Finkelstein et al. (2003) estimated the effect of the overweights and obese on Medicare, Medicaid, insurance and uninsured sources of health care cost funding separately. They estimated this effect to be about 5.3 percent of total annual medical expenditures and contributed about 9.1 percent of the total increase in U.S. medical care costs in 1998, and as high as \$92.6 billion of health care costs measured in 2002 dollars, with obesity accounting for an almost equal amount of indirect costs (mostly due to reduced productivity resulting from obesity-related morbidity). Finkelstein et al. (2009) updated their study based on the 2006 Medical Expenditure Panel Survey (MEPS), estimating that almost \$40 billion of increased spending through 2006 was the result of the increase in obesity prevalence. They thus estimated that the medical expenditures of obesity rose to \$147 billion per year by 2008, representing 12.9 percent of private payer, 8.5 percent of Medicare and 11.8 percent of Medicaid expenditures. They also reported that outpatient and physician office expenditures were 26.9 percent greater and prescription drug expenditures were 80 percent greater than with those of normal weight.

- Thorpe et al. (2004) estimated the increase in the prevalence and corresponding health care spending of the obese relative to those of normal weight accounted for 27 percent of the increase in real per capita spending between 1987 and 2001, while the corresponding percent increase for hyperlipidemia was 22 percent, diabetes 38 percent and heart disease 41 percent. They estimated the increase in obesity prevalence alone accounted for 12 percent of the growth in total health care spending between those years, primarily due to increases in the cost of treatment for diabetes and hypertension. Treatment cost for diabetes, paid by all sources, more than doubled from \$18.5 billion in 1996 (in 2007 dollars). Others have estimated that obesity accounted for about a third of the growth in health care spending over the past 20 years. They projected that by 2020, treating those who are obese will cost an estimated 61 percent more than the cost of treating healthy weight people.
- Daviglius et al. (2004) reported on annual fee-for-service Medicare charges of participants of the Chicago Heart Association Detection Project in Industry (with a 32-year mean follow-up period). After adjusting for age and race, severely obese men were 98.3 percent, those obese class I 54.4 percent and those overweight 23.0 percent greater than those in the standard BMI category. Corresponding percentages in excess of standard BMI for females were 90.0 percent, 40.6 percent and 16.4 percent, respectively. Relative additional health care costs were larger for those younger than 65 compared with those older than 65.
- Raebel et al. (2004) in a one-year study of Kaiser Permanente of Colorado found that although the primary driver of additional cost was more prescription drug use (e.g., by a 1.81 factor, primarily for cardiovascular, intranasal allergic rhinitis, asthma, ulcer, diabetes, thyroid and analgesic drugs), obese patients also had more (by a factor of 3.85) hospitalizations and outpatient visits (1.5 times more). They found that for the obese there was a total health care cost of \$585.44, compared with \$333.24 for the non-obese. In addition, each unit of increase in the BMI increased the risk of hospitalization by about 11 percent, while each additional chronic disease increased this risk by an additional 40 percent.
- Yan et al. (2006) reported that the Chicago Heart study's participants who were obese and were otherwise at low risk (based on favorable blood pressure, cholesterol level and smoking status) at baseline measurement had a hazard rate of 4.25 for coronary heart disease and 2.32 for cardiovascular disease related hospitalizations compared with those at standard BMI and a hazard rate of 2.04 for coronary heart disease and 1.61 for cardiovascular disease at moderate risk relative to those risk factors. Corresponding hazard rates for those overweight relative to those at standard BMIs were less but still generally greater than 1.0. Hazard rates for mortality in those categories were generally less, for example 1.43 for coronary heart disease for those at low risk. Hazard rates for diabetes were uniformly greater than those for coronary or cardiovascular disease. Based on these results, obesity has a relatively greater relative effect on hospitalization

use than on mortality, and coexistence of other risk factors is quite important to overall health when obese or overweight. These results also suggest that (1) there may be a need for longer-period follow-up than has been used for most health care studies and (2) this may help to explain the relatively low effect of concurrent or recent obesity levels on the health of older age individuals. Those who are obese in their middle ages with no or a few cardiovascular risk factors may have a higher risk of hospitalization and mortality from coronary heart disease, cardiovascular disease and diabetes when they become older than those of normal BMI.

- Anderson et al. (2005) estimated the combined effect of obesity, overweight and physical inactivity on health care charges of a single medium-size Minnesota health insurer (with insureds primarily white and over age 40) from 1996-99 to be about 23.5 percent of total health care costs. By adjusting this estimate to reflect more nationally representative health care charges, they derived an estimate of 27 percent on a national basis (due to a different expected demographic mix). Although they estimated that the charges associated with these risk factors were greatest for the oldest group (65 and older), nearly half of the total charges came from the 40 to 64 age group who did not have chronic diseases. This suggests that a broad, populationwide approach to address physical inactivity and obesity may be a more effective strategy than a more focused one to address health care costs. It emphasizes the importance of prevention and effective management of the risk factors as a health care cost containment strategy. This study addressed the combined effect of weight and physical activity because of their behavioral and physiological interrelationship.
- Sutocly (2005) indicated that annual direct health care costs in California associated with obesity were estimated by the California Department of Health Services to be about \$4.11 billion in 2000, with indirect associated costs exceeding \$2.25 billion.
- Hart et al. (2006), reporting on the results of the Renfrew/Paisley study in Scotland whose follow-up period was quite long (between 28 and 32 years), indicated that men in the study experienced higher than expected bed day rates. In contrast, women experienced a U-shaped admission rate relationship, with rates of admission greater than expected for both those underweight and those obese, with normal weight women having the lowest admission rate.
- Monheit et al. (2007) found that, based on an econometric analysis of data from the 2001-03 MEPS, adolescent bodyweight and overweight conditions are strongly associated with parental bodyweight, parental education, parental smoking behavior and neighborhood attributes such as the availability of fresh food markets, convenience/snack food outlets, neighborhood safety and material deprivation. Overweight females were estimated to have annual health care expenditures that exceeded those of normal weight by \$622, while for those at risk of becoming overweight there was only a \$68 difference in annual health care

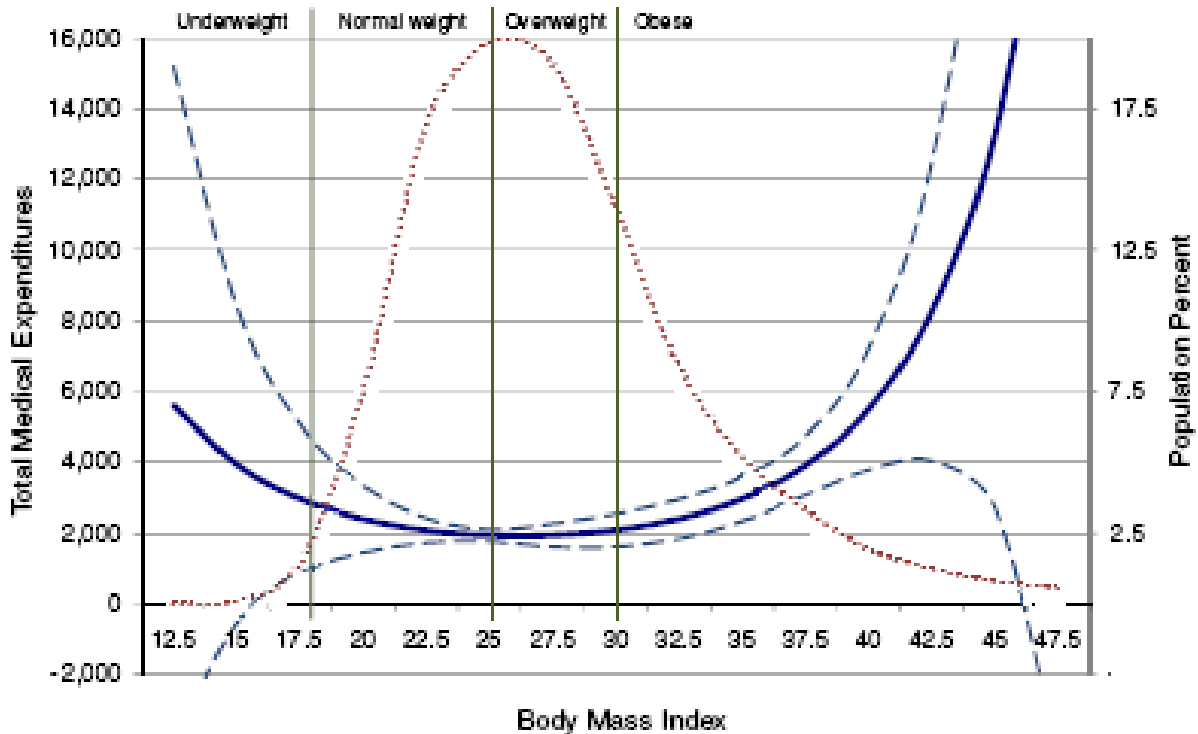
expenditures, with part of this difference due to differences in mental health expenditures. Corresponding differences were not found for male adolescents.

- Van Baal et al. (2008), using a simulation model based on relative medical costs per condition affected, estimated in a Dutch study that the annual and lifetime medical costs attributable to the obese, the smoker, and those who weren't obese or a smoker. They found that until age 56 the obese have higher health care costs, while after age 56 smokers incur higher health care costs. However, due to differences in life expectancy, lifetime health care costs were higher for non-obese and non-smokers (healthy) people and lowest for smokers. The conclusion reached was that it costs less for health care over an average lifetime for an individual who is obese or a smoker, but only due to their shorter life expectancy. The assumption used was that on average smokers live about 77 years; those obese live about 80 years; and the healthy live about 84 years. The cost of care for the obese was \$371,000 compared with \$326,000 for smokers. They also determined the break-even discount rate that would equate the costs for the three population segments to be 4.7 percent to equate the cost of the healthy and obese, and 5.7 percent to equate the cost of the healthy and smoker.
- Lackdawalla et al. (2005) indicated that, although obese 70-year-olds live about as long as those at that age of normal weight, they will spend \$39,000 more on health care, have fewer disability-free life years and experience higher rates of diabetes, hypertension and heart disease. In this study, the additional health care costs were not expected to be offset by health care savings due to higher mortality rates.
- Thorpe (2009), using the 2006 Household Component to the MEPS and the average annual change in the percent of the population by underweight, normal weight, overweight and obese changes between the 2003 and 2008 BRFSS, projected health care spending by state in 2013 and 2018. The resulting health care spending for obesity-attributable health care was about \$79.4 billion in 2008, and projected to be \$139.1 billion in 2013 and \$343.9 billion in 2018. In contrast, if obesity prevalence stays at current levels, the 2018 level would be reduced by about \$200 billion.
- In a projection of the benefits of risk factor prevention in Americans 51 and older, Goldman et al. (2009) estimated there would be a gain in average life expectancy from successful treatment of smoking, diabetes, hypertension and obesity of 3.44, 3.17, 2.05 and 0.85 years, respectively, and there would be a corresponding decrease in expected health and medical spending of about \$198,000, \$138,000, \$119,000 and \$52,000 from these risk factors.
- Trasande et al. (2009) reported there was a near doubling (21,717 to 42,429) of hospitalizations with a diagnosis of obesity between 1999 and 2005, with an increase in related costs from \$125.9 million to \$237.6 million (2005 dollars). The

authors also noted undercoding for obesity as a cause of treatment, so that these reported results may be understated.

- Anis et al. (2009) indicated that, based on recent studies of health care costs and obesity in Canada, the cost of obesity was currently running at 4.1 percent of overall health expenditures, in contrast with prior estimates of 2.2 to 2.4 percent. This increase was primarily due to recent increases in those overweight and obese and to several comorbidities that had previously not been identified as resulting from obesity. The authors expected this trend to increase further in the future.
- Stagnitti (2009), based on the Household Component of the 2001 and 2006 MEPS, found that the proportion of total health care expenditures for obese adults increased from 28.1 percent to 35.3 percent during that period, while the corresponding proportion of adults of normal weight decreased from 35.0 percent to 30.3 percent. The average annual health care expenditure increased for the obese population from \$3,458 to \$5,148 (49 percent), while the corresponding increase for those overweight was from \$2,792 to \$3,636 (30 percent) and those of normal weight from \$2,607 to \$3,315 (27 percent).
- Cawley and Meyerhoefer (2010) studied the impact of obesity of those between ages 20 and 64 with biological children, based on the 2000-05 wave of the MEPS. They found the impact of obesity on health care costs was about \$2,826 per year (\$3,696 for females and \$1,171 for males, expressed in 2005 dollars), or \$3,115 in 2008 dollars, about twice that of Finkelstein (2009)'s widely quoted estimate. They attributed this large difference to the use of additional variables that other studies did not reflect and reporting errors that may have biased the results of other studies. In particular, they reflected the nonlinear nature of the relationship between class of obesity and health care costs. Generalizing their findings to all non-institutionalized adults, they estimated that about \$268.5 billion (in 2005 dollars) or 16.5 percent of U.S. national health expenditures is spent treating obesity-related illness. The additional observed costs from this study took the form of a J-curve (somewhat higher costs for those who are underweight) with sharp increases for those of class II+ obese, as shown in Figure 16. This indicates the significant health care costs associated with the morbidly obese, particularly the effect of the nonlinear relationship between BMI and health care costs.

Figure 16
2005 Average Health Care Costs by BMI (Cawley and Meyerhoefer)



Notes: Data: MEPS 2000-2005. Expenditures are in 2005 USD. Dashed lines represent the 90% confidence interval, which has been adjusted for the complex design of the MEPS. Medical expenditures are denoted by the solid line and are measured on the left axis, while the distribution of individuals in the population is indicated by the dotted line and is measured on the right axis. BMI is calculated using self-reports or proxy reports of weight and height.

- Bhattacharya and Sood (2011), based on the Health and Retirement Study and the Future Elderly Model, estimated the additional lifetime medical care costs for a 50-year-old due to the existence of obesity was about \$15,000; for a 65-year-old this additional cost was about \$5,000 and for a 75-year-old there would be a modest \$1,000 health care cost saving.

Although some of the earlier studies indicated that obesity-related health expenditures ran between 5 and 7 percent of annual health care expenditures in the United States, the more recent studies have estimated them as much as 9.1 percent (Finkelstein et al. 2009) or 16.5 percent (Cawley and Meyerhoefer 2010). These large percentages suggest that this is an area in which significant health care focus and further research is needed. This is particularly true in view of the extremely large health care costs in the United States (for example, according to National Health Expenditures as published by the Centers of Medicare and Medicaid Services in 2009 hospital costs were about \$759 billion, physician and clinical services were about \$506 billion and prescription drugs were about \$250 billion. Note that these more recent estimates of percent of health expenditures focus on direct costs and do not include indirect costs, which have been estimated in certain earlier studies to be between 50 to 100 percent of the direct costs. In addition, there is also a need for further analysis and research into relativities between morbidity/health care costs and subsequent mortality.

Disability and Employment

Rates of disability, particularly those of the elderly, have generally improved over time. Nevertheless, there is a risk this trend may not continue. According to several reports in Madrian et al. (2007), baby boomers age 51 to 56 in the Health and Retirement Study in 2004 were not in better health than those at the same age a decade older, with a higher proportion reporting being in poorer health and having more difficulty performing daily tasks.

Based on the National Longitudinal Survey of Youth, 1979 Cohort, as reported on by Burkhauser and Cawley (2004), the probability that men report work limitations rose 0.7 percent per extra 10 pounds of weight and 5.4 percent if obese, although other datasets do not necessarily support this amount of increase by weight. For women, use of this source and the results of the Panel Survey of Income Dynamics also suggest that weight may increase the probability of work limitations. In addition, it was observed that the relationship between body weight and disability is nonlinear (that is, the probability of disability increases sharply as BMI increases after a point). They found it is likely obesity contributes to disability, although they note “that even nationally representative datasets collected over similar time periods can generate results that differ in important ways underscores the need to test hypotheses using multiple datasets in order to determine which results are truly robust.”

Tucker and Friedman (1998) found that obese employees (defined in this case as males with greater than 25 percent body fat and 30 percent for women) are 1.74 and 1.61 times more likely to experience higher (defined as seven or more absences due to illness per six-month period) and moderate levels (three to six) of absenteeism, respectively, than their lean counterparts.

Finkelstein et al (2010b), based on the 2006 MEPS and 2008 National Health and Wellness Survey, found that excess per capita medical expenditures and the value of lost productivity ranged from -\$322 for overweight males to \$6,087 for class III obese males and from \$797 for overweight females to \$6,694 for class III obese females. In aggregate, they calculated that the annual cost attributable to obesity among full-time employees was \$73.1 billion. Those class II+ obese constituted 37 percent of the obese population, but were responsible for 61 percent of these excess costs. Similar to other studies, it found these costs are linear with respect to BMI.

Bhattacharya and Bundorf (2009) found that incremental health care and productivity costs associated with obesity are passed on to obese workers through employer-sponsored insurance in the form of lower cash wages. They estimated that obese men earn \$1.21 an hour less than non-obese men, while obese women earn \$1.66 less than non-obese women, although the wage penalty is even higher in firms where employers provide health insurance, where the obese earn \$2.64 an hour less. They also observe that the difference in expenditure grows with age and is greater for women than for men.

Lakdawalla et al. (2004) indicated that according to the NHIS of 1984 to 2000, the rate of the more severe personal care-limitations increased by 50 percent. Lakdawalla reasoned that the deterioration in health could be due to: (1) the tendency of the obese to have more disabilities,

together with the significant increase in the percent of obese; (2) lifesaving medical techniques may result in a higher percentage of disabled who might otherwise have died; and (3) the less-than-average wage growth for less-skilled workers that can provide a greater incentive to claim disability insurance coverage. According to NHIS results, obesity accounts for about one-half of the increased rates of disability among those age 18 to 29, one-quarter for those age 30 to 39 and one-tenth for those age 40 to 49. The two most important causes of disability among the nonelderly are musculoskeletal problems and mental illness, and together with the small but growing contribution of diabetes, suggest the increasing contribution of obesity to those disabled.

Using then current trends in the rate of disability for different BMI categories, Sturm et al. (2004) projected rates of disability for those between ages 50 and 69 to increase by 17.7 percent for men and by 21.8 percent for women from 2000 to 2020 due to prevalence of ADL limitations. This also will mean greater claims costs associated with disability and workers compensation. They concluded that as obesity becomes more prevalent among the elderly, it will be more difficult for other societywide trends to counter its adverse health effects. Unless factors other than obesity underlying past trends become stronger, Americans between ages 50 and 69 may not have better health and functionality than those currently in that age group.

According to Alley et al. (2007), among the obese age 60 and older between the period of NHANES III and NHANES 1999-2004, the prevalence of functional impairments increased 5.4 percent (from 36.8 percent to 42.2 percent), although ADL impairments did not change. In NHANES III, the hazard rate for those obese compared to normal BMI individuals was 1.78, but that increased to 2.75 in NHANES 1999-2004. With respect to ADL impairments, the hazard rate between obese and normal BMI increased from 1.31 to 2.05 as reported in these two surveys because although the rate of impairment did not change for the obese, they decreased by 34 percent for the non-obese. Alley concluded that over the 10-year period between the two surveys (1) the obese were more likely to report functional impairments, and (2) reductions in ADL impairment for older non-obese individuals did not occur in those who were obese.

Stallard (2011) found, based on the data from the 2004 National Long-Term Care Survey, the effects of current levels of self-reported obesity were associated with large increases in diabetes, substantial decreases in mortality and nonsignificant increases in disability among the elderly. Obesity at age 50 was associated with large increases in diabetes and disability, and nonsignificant increases among the obese elderly. Categorization at age 50 measurements eliminated the obesity paradox that might exist using then current measurement. This effect also held at the oldest age group. Stallard found the effects of obesity and diabetes were consistent with the initial or intermediate stages in a complex multistage/multipath disablement process leading to early disability and death. The joint adverse effects of obesity and diabetes were indicated to be greater than if they occurred alone.

5. Prevention and Management

There are four basic approaches to controlling (including both prevention and management of) adiposity tissue: (1) reducing caloric input, e.g., eating less, using drugs to impair absorption or reduce appetite; (2) enhancing the mix of caloric input, e.g., increasing relative consumption of nutritious foods; (3) increasing energy expenditure, e.g., increasing exercise; and (4) removing adiposity tissue or impairing its metabolism, e.g., bariatric surgery.

However, easier said than done! At least at this time, there is no single best method or motivation that will prevent the onset of or effectively manage obesity. No large-scale trial has demonstrated reduction in clinical events through a practical weight management strategy (although bariatric surgery has shown some recent success, its long-term risks have not yet been fully assessed). The most effective strategies, including those that follow below, have been multifaceted with a long-term focus. It is important to note that unhealthy behavior does not just emerge in adulthood. It usually starts in one's youth and is more likely to continue if it occurs as an adolescent, shaped by the influences of family, friends, peer groups, schools, and the broader social and physical environment.

- **Diet.** There have been, are and will be a wide range of diets and weight-reduction techniques available. Although many diets consist simply of reducing caloric intake, others involve the management or mix of foods consumed, such as being low in fat or carbohydrates. But diets themselves rarely “cure” obesity, can be quite costly, and can sometimes be dangerous in and of themselves, with the latest magical cure rarely magical and rarely a cure. Philipson and Posner (1999) indicated that weight management involves “not information but incentives; everyone knows how to lose weight — either you eat less or exercise more, but few want to pay the price, in effort, expense, or forgone pleasure, of doing it.” According to Rosenbaum et al. (1997), since both protein and carbohydrate can be metabolically converted to fat, there is no evidence that changing the relative proportions of protein, carbohydrate and fat in the human diet without reducing caloric intake promotes weight loss — it is thus more important to control the amount of food eaten than the type of diet undertaken. In fact, dieting makes the dieter focus more on food than ever before, often increasing the appreciation of food. Diets, either using a carrot, stick or education, are easy to try but difficult to maintain. Many people lack sufficient self control over their dietary habits to consistently make the healthy choice.
- **Food, beverage and restaurant industries.** These industries and individual firms have shown they can develop strategies that address both business and health objectives. Various elements of the food industry could improve their products and packaging or labeling innovations to help consumers make healthy choices. Enhancement of product development, and promotion and advertising of healthy foods and snacks may prove beneficial to all involved. People can and do respond to simple innovations, such as smaller servings of snack food or in company cafeterias, although possible substitution effects also need to be considered.

Giving an advantage to healthy choices through pricing, achieved either through subsidies, convenience or taxation can influence behavior.

The food industry already has significant incentives to provide healthy foods if they taste good, are affordable and have a good public image. It simply has to provide and effectively promote those types of food. Healthy “junk food” and snacks might help in some cases, although marketing them may prove challenging. A successful example has been the bottled water industry, whose product has gained significant acceptance and may be healthier than what it in part has substituted for, e.g., soft drinks. If food better addresses consumers’ needs and concerns, current adverse opinions may change. Promotion of quality rather than quantity at an affordable price would certainly be beneficial.

Fast food and full-service restaurants could expand their healthier food options and provide more and transparent nutritional information on a voluntary basis, with government requirements likely without private action. More effective promotion of smaller portion size might help. For example, at a local ice cream store, a single scoop is now often referred to as a “kiddie” size, not particularly psychologically conducive to adult males ordering it. Healthy substitutes for ingredients such as “transfat” and similar nonfat ingredients should be developed, either through government rules or voluntarily, although care may be needed to ensure that substitutes are superior nutritionally. And, of course, even stricter rules for advertising to children could be adopted.

Based on a study of purchase decisions in Starbucks after mandatory calorie posting (first required in the United States by New York in 2008) was implemented, Bollinger et al. (2011) indicated that average calories per transaction fell by 6 percent, almost entirely related to changes in food and not beverage choices, with three quarters due to the purchase of fewer items. That may not fully apply to full-service restaurants. This modest impact may imply that consumers care more about convenience, price and taste than calories. However, regular exposure to this information may modify buying habits over the long term.

Although the food industry has made significant amounts of relevant information available on companies’ websites, it is rarely used by the average consumer, and unfortunately little of that type of information is easily available at the time of food selection. More effective delivery approaches are needed.

Firms within these industries have often argued you can’t tell the net health contribution of a particular food unless the totality of a person’s diet is considered. They claim that failure to achieve a healthy food regimen is the personal responsibility of individuals, not those that facilitate their application, as individuals will find other means of satisfying their needs if a specific product doesn’t. To some extent this claim is true; however, if no one does their part, such an enormous systemic problem will not be successfully addressed.

The leisure industry has inherent incentives to promote physical fitness and age-specific physical activities. There are many segments of the population that this objective and feature may benefit.

- **Physical activity.** A regular, moderate and sustainable physical activity program can help both weight management and improved overall health and fitness. An increase in physical activity, although not usually as effective by itself as decreased caloric intake, can help prevent weight regain, particularly when combined with proper nutrition. Although a healthy weight or BMI is desirable, even those overweight who exercise sufficiently can experience lower premature mortality rates. Various studies support that 30 minutes of daily exercise is just as effective as 60 minutes per day, so, if appropriately planned, it can prove not to be inconvenient. For example, Hankinson et al. (2010), in the Coronary Artery Risk Development in Young Adults, a prospective longitudinal study conducted in several large U.S. cities with 20 years of follow-up, found that maintenance of high physical activity levels (at least 30 minutes daily) through young adulthood, particularly for women, may lessen weight gain (measured by means of BMI and waist circumference). Although home health equipment has the potential to enhance physical fitness, it is not uncommon that such purchased equipment is not used.
- **Education.** Although frequent articles in the consumer media have highlighted the obesity epidemic, useful information is not usually conveniently available to consumers when action is needed, such as when purchasing food. This might include easily accessible calorie food labeling in restaurants or in supermarkets (the 1991 Nutrition Labeling and Education Act has provided more information to consumers, but the benefits seem to have been limited to certain demographic groups, mostly non-Hispanic white females, and even there the effect does not appear noticeable to date). In addition, more creative approaches to provide relevant and timely information regarding the health effects of being overweight and obese are needed, although general education has not been shown to result in weight loss. Although seeking health information is currently the third most prevalent activity among Internet users, the application of this information to generally improve overall health conditions, such as controlling weight, remains far more difficult than making the information more accessible. Social marketing, with a focus on voluntary change, is a well-used technique. However, most of the population already understands that more food consumed results in more body weight; effective education that results in changed habits is the difficult part. But even when people are aware of the long-term risks, there is a limit to its effectiveness if the right type and size of food and physical activity are not conveniently available.
- **Bariatric surgery.** The National Institutes of Health (1998) recommended bariatric surgery for those with a BMI of 40 or more or a BMI of at least 35 with comorbid conditions. Such a procedure involves reducing the size of the stomach (gastric banding) or bypassing part of the intestines (gastric bypass). Even though

this may only be recommended in extreme cases, the number of such procedures has increased significantly over the past decade, from about 10,000 in 1996-98, to 100,000 in 2002-04, and about 220,000 in 2009, with a cost upwards of \$25,000 per operation, although the current average is closer to \$15,000. However, since resulting weight loss can lead to a 30 to 40 percent reduction (and one study even up to 70 percent) in weight and complete resolution of certain comorbid conditions for up to 10 years, it is not surprising that the rate of these procedures is steadily increasing in popularity, although in some cases it can have major potential complications, including leakage, pneumonia, and band slippage and erosion. Other surgical “solutions,” such as liposuction by which outer fat layers are removed, have not resulted in the health benefits.

Finkelstein et al. (2010a) found that in a study of more than 7,000 U.S. health care claims, laparoscopic adjustable gastric banding, with a mean cost of about \$20,000, resulted in modestly lower health care costs in the post-operative period compared with a matched control group, with the net cost reduced to zero after four years and a reduction to zero after two years for those with diabetes. In a meta-analysis of 26 studies of open and laparoscopic bariatric surgery by Picot et al (2009), bariatric surgery was found to be a more effective intervention for weight loss than nonsurgical options. In one study, weight loss was still apparent 10 years after surgery. Some, but not all measures of quality of life improved after this surgery, but with a higher remission rate of diabetes than in the nonsurgical group. Three out of six comorbidities were reduced after 10 years, with adverse event reporting that varied, with mortality ranging from none to 10 percent. In summary, these studies indicated that surgical management was more costly but with improved outcomes. Further research was called for.

- **Pharmacotherapy.** Drugs currently available to control weight can be placed into two major categories: (1) those that act on the central nervous system to influence eating behavior and appetite and (2) those that target the gastrointestinal system and inhibit absorption or enhance a feeling of fullness. Neither approach has had great success to date, with at best modest weight loss achieved in some cases. In sum, effective weight-management drugs have yet to be developed, although it is possible that someday a magic bullet might be found, possibly from an idea currently under development. Basic scientific investigations into obesity continue and eventually may reveal new knowledge regarding the relationships between health and obesity and nutrition and physical activity to enable effective drugs to be developed.
- **Schools and communities.** Peer pressure, public acceptability, personal body image and the fashion industry are all sources of psychological inputs that are often more important than other educational efforts. Where practical, these need to be steered in healthy directions.

Nevertheless, nutrition education can and should continue to emphasize food mix; for example, fat comprises an average of 35 percent of total caloric intake for

youths age 2 to 19, and almost two-thirds did not eat the currently recommended daily amount of fruit and vegetables (according to the CDC in 2010, only 14 percent of U.S. adults and 9.5 percent of adolescents consumed recommended levels of fruits and vegetables on a daily basis).

About 25 million students currently make use of the National School Lunch Program and 7 million use the National School Breakfast Program. Implementing the minimum requirements of these programs for all school meals might help promote a nutritional mix. Although better rules and enforcement of what can be offered in vending machines may prove useful, studies such as Forshee et al. (2005) have indicated that, based on findings from the Continuing Survey of Food Intake by Individuals (1994-96, 1998), NHANES 1999-2000 and the National Family Opinion WorldGroup Share of Intake Panel, there would be limited impact on BMI from removing regular carbonated soft drinks from schools due to substitution effects. In any event, offering water bottles in such vending machines may provide a healthier alternative.

Annual measurement and reporting of student BMI level through so-called obesity report cards are being provided on a confidential basis to parents or guardians in 16 states. However, unless care is taken, this may have negative consequences, e.g., it could lead to eating and psychological disorders and unwarranted social stigmatization, although it may also lead to acknowledgement on the part of applicable parents and children that there is a problem that hasn't been effectively dealt with.

Communities can increase access to recreational facilities and put pressure on schools to enhance their physical education programs and improve access to nutritional food.

- **The family.** For children, both positive parental role models and an overall healthy family lifestyle promote healthier choices. Parents have to take an effective role in recognizing the existence of a weight problem in their children and a sufficiently active role and responsibility in addressing the causes, be it nutritional or exercise related. Nevertheless, as indicated in the report card discussion above, care is needed to avoid unwanted side effects, including body hatred, inappropriate weight loss attempts, eating disorders and weight stigma while avoiding oppositional reactions that may produce actions the opposite those intended.

Exclusive breast-feeding for the first four to six months has been recommended based on several studies finding its inherent protective effects, possibly due to enhanced learning of satiety by the infant, the composition of breast-milk and less insulin secretion post-breast-feeding. But in contrast, Lawlor and Chaturvedi (2006) indicated that “while mean BMI in later life was lower among breast-fed subjects, the difference was small and likely to have been strongly influenced by publication bias and confounding factors. ... Evidence to date does not support

infancy as a critical period during which interventions might have long-term effects on the risk of obesity and its associated diseases.”

- **The workplace.** Although not every place of work requires or facilitates physical activity, many employers, particularly the larger ones, can facilitate wellness programs and can increase awareness of and an environment in which healthy habits are encouraged, especially as they increasingly recognize that not only is it good for their employees, but it can also reduce health care and disability costs for them. An employer can also offer low cost healthier options in cafeterias and vending machines. Companies can also offer nutritious food programs in the workplace’s neighborhood.
- **Insurance and health care services industries.** Although insurance tends to spread the cost of certain activities and conditions across pools of individuals, to the extent that these industries can charge for expected costs, they can increase personal incentives to take on healthy and safe behaviors. These industries can provide incentives for healthy and cost effective behavior, e.g., through lower deductibles or copays for preventive care, treating obesity as a chronic health disease, and sponsoring wellness programs and ongoing health awareness programs that include more effective and active counseling. They can also provide parents enhanced education and more accessible and action-oriented information including possible techniques to address them. Paying for performance through wellness rebates or premium reductions, frequent health activity points, gym membership discounts or high deductible health insurance plans can be effective in some instances. Other approaches include enhanced counseling and more effective monitoring of patients’ actions, and improved training of health care professionals in best prevention practices and management of healthy weight and lifestyles. Health care professionals need to be more actively involved in education, prevention and treatment of obesity and obesity-related conditions.
- **Behavior therapy.** Psychological reinforcement by means of support groups such as Weight Watchers can in some cases facilitate weight control. Television shows glorifying and supporting large weight loss have recently grown in popularity. Goal-setting, self-monitoring, frequent contact, feedback, and continuous motivation and support are important components of any such program.

To study the effectiveness of intensity of weight-loss interventions, Levine et al. (2007) studied three groups of healthy women between the ages of 25 and 45 who, during a three-year period, received different levels of intervention: a more active, clinic-based group that met bi-monthly and a group that received instruction through a correspondence course, both compared with a group only provided an information booklet about weight management. Neither of the groups with intervention was better at preventing weight gain than the control group; both gained at least some weight, with about 60 percent gaining at least 2 pounds during the period studied. Those on a diet prior to the commencement of the study

period were less likely to be successful in controlling their weight, possibly indicating the difficulty that many people have in controlling their weight. However, Levine indicated the study did find that for high-risk groups, intensive, structured interventions can be successful in preventing weight gain. Goodpaster et al. (2010) conducted a similar study of a one-year intensive lifestyle intervention consisting of diet and physical activity of severely obese adults and achieved clinically significant weight loss and favorable changes in cardiometabolic risk factors, including abdominal fat.

- **Government and public policy.** Although many resent a “nanny state,” a wide range of proposals have been made that would have government dictate or restrict actions by people and food-related industries. In any event, such proposals could help ensure an adequate supply of affordable and healthy food, limit portion serving size, and make sufficient information available for consumers to make informed food choices. However, ultimately, only through personal motivation can change occur; one only has to look at the almost 20 percent of the population who still smoke as a warning of the limits of the effectiveness of government action in directing personal behavior.

If obesity affected others, as in the case of second-hand smoke, its significant increase would have led to government action to help people take more personal responsibility to change their lifestyle or risk paying for the results by themselves. To the extent desirable, the additional public costs associated with obesity, such as health care costs, have not so far been transparent enough to sufficiently change public policy. At least so far, general public health recommendations for weight reduction have not proven to be an effective approach.

Requiring caloric labeling in restaurants has recently begun in the United States. Nutritional content labeling of purchased food has long existed, but the labels are often not a sufficient consideration in most food purchases, although it may be worth pursuing in some form, possibly similar to the European traffic light system (e.g., red could mean high fat or sugar contents). Other examples include reduced availability of poor nutrition snack vending machines or increased healthy public cafeteria offerings and smaller portion size, restrictions on toy giveaways with unhealthy food purchases, grocery stores in poor areas and food stamp requirements.

It has been suggested that some current U.S. programs (known as “checkoff” programs) for which there are 35, according to Wilde (2005), promote the wrong types of food through lower prices, e.g., beef, pork and dairy products, and certain energy dense foods. The incentives resulting from these programs may not be consistent with the government’s *Dietary Guidelines for Americans* that promotes healthier foods, such as fruit, vegetables, fish and whole grains.

Additional taxes on selected foods or drinks can be effective, although depending on their implementation, such taxes can be viewed as being regressive (although

over a long time period subsequent health care costs may offset these short-term costs). To be effective, such taxes have to be relatively large (apparent in the case of cigarettes), which can be highly unpopular at best. In addition, these can be attractive to governments in their search for more revenue, justified in part as a recoupment of public health care dollars and to further incentive for healthy living. Tax incentives, such as certain currently offered local programs that provide property or other tax relief if food establishments meet certain minimum “healthy” guidelines and national programs that provide agricultural subsidies to redistribute crops to a healthier food mix, might prove effective.

Providing physical activity infrastructure, such as redesigning roads and walkways to promote walking and cycling, more and more attractive parks and open spaces, healthy food retailers and farmers markets, as well as more activity-incenting school activity, might also be used to make physical activity more acceptable.

- **Technology.** The use of technology should be emphasized in carrying out any public health program. It has been suggested that devices, possibly implantable, that would be less severe than bariatric surgery, may prove more effective than diets and drugs. Overall, new technologies may be developed to treat obesity and obesity-attributable comorbidities.

The goal should not be for everyone to reach the elusive American ideal of being slim and fit and remaining young forever. Nevertheless, maintaining as healthy a body as the individual’s genetic situation practically allows remains a worthwhile goal. Because the causes of obesity are so heterogeneous, a multifaceted program to achieve a healthy body has to be tailored to the individual. It should focus not only on weight, but also the contributing behaviors, primarily relating to nutrition and physical activity, although BMI and other weight-related measures remain reasonable metrics by which to assess progress. The key is not just weight loss in those obese, but whether it can stay lost. The development of effective weight loss and management programs over the long term will remain a challenge. Human behavior may be the toughest part of any program to change.

Those in developed and in developing countries are sometimes overwhelmed by the easy availability of high-fat, energy-dense foods and finding the time and motivation for physical activity. It is not surprising that education-based interventions promoting behavior changes have had limited success. There is a need for interventions aimed at facilitating a supportive population-based environment promoting improved nutrition, the availability and accessibility of a variety of attractive low-fat, high-fiber foods and physical activity habits that stand a chance of continuing after the end-of-year resolutions are discarded by Jan. 8. Fortunately, physical activity and food consumption involve mutually reinforcing behaviors that can often be influenced by the same measures and policies.

In any case, sufficient motivation and incentives are needed for any approach to work over a long period. With modern societal incentives and built-in mechanisms to satisfy short-term desires and preferences, it is difficult for the individual to meet healthy objectives at the

same time with their long-term rewards. Too heavy a focus on weight may demonize and demoralize those who are currently obese, thus creating significant psychological hurdles to good health and long life. Progress for children and adolescents is unlikely to be made without active intervention and assistance from both schools and parents, for without starting young, health conditions may get worse before they get better.

So much public emphasis has been placed on preventing childhood obesity because (1) the likelihood that habits formed in childhood are difficult to break when an adult, (2) the difficulty of “curing” obesity in adults and (3) the many long-term adverse effects of childhood and adolescent obesity. Adolescents will have to become involved in helping themselves; imposed solutions will often not work.

Since weight-cycling can be even more harmful in certain cases than having a somewhat higher but stable weight, long-term programs should be emphasized. In a society in which food is plentiful and affordable and the need for exercise is no longer necessary but just desirable for health, we will likely see a lot of fat people trying to get thin for a long time to come.

Some have expressed the view that some of the strategies used to reduce the amount of smoking, including steep increases in the price and taxation of tobacco products, implementing smoke-free laws in workplaces and public places, aggressive anti-tobacco media campaigns, and support for quitting smoking, should be employed to reduce the prevalence of obesity. However, it is far more difficult to follow any of these strategies to control obesity due to the differences between the nature of tobacco and nutrition. In an attempt to raise public awareness, community and national goals and strategies have been established. This may prove to be an effective approach. Prevention rather than treatment may hold the highest potential for reversing the trend toward increased obesity and its unwanted costs in the United States and worldwide.

A major cultural/social shift may be needed, both on a national and local level, as opinions must be embedded more deeply than at present to influence individuals’ decision-making processes through an environment that discourages overeating and encourages more physical activity. We are spoiled; we seemingly have it all and we feel “more is better,” even when eating. As individual efforts have not succeeded in the past, the need for population-level intervention strategies, possibly including communities, governments, the media and the food industry, as well as the individual, may be needed to prevent avoidable premature deaths. Maintained intensive intervention groups have proven successful in some cases, although the individuals involved have to be committed to the program, and make the results and habits ingrained into future daily activities and lifestyle — not easy to accomplish in practice. As Michelle Obama has said, “We’re talking about changing habits that have been formed over generations. It’s not going to be easy.” Nevertheless, we have seen changes in smoking habits, so change in a healthy direction is possible.

The likely future adverse effects on mortality, morbidity and health care of the huge percent of the population that is now overweight and obese should not be ignored. Although much of the cost and suffering is born by the individuals affected, the additional resulting health care costs are in part shared with other members of the public. It will remain a significant challenge to public policy, the health of the nation, and the actuarial profession in all practice

areas. Further research, especially that covering long-term follow-up periods, will help to better assess the implications of this issue. It took decades of intensive government effort to gain a modest control over smoking; it will take at least as long to obtain improvements from the fight against obesity and sedentary living. Only long-term solutions taken on both an individual and societal level will likely be effective in contributing to the solutions to the current condition and possible adverse trends. No magic bullet is yet in sight.

Appendix

The most commonly used metric in the study of obesity, body mass index (BMI), is used throughout this paper. It is equal to weight (kilograms)/height (meters). Its calculation applies to both females and males, and is given in Table 17 (shown in feet/inches and pounds for the convenience of those more used to those measures).

TABLE 17
Height (Feet/Inches) and Weight (Pounds) for Adults

Height / Weight	Body Mass Index (BMI)						
	Underweight < 18.5	Normal 18.5-24.9	Overweight 25.0-29.9	Obese 1 30.0-34.9	Obese 2 35.0-39.9	Obese 3 40.0-44.9	Obese 4 ≥45.0
5' 0"	< 95	95-127	128-152	153-178	179-203	204-229	≥ 230
5' 2"	< 101	101-135	136-163	164-190	191-217	218-245	≥ 246
5' 4"	< 107	107-144	145-173	174-205	206-231	232-261	≥ 262
5' 6"	< 114	114-154	155-185	186-217	218-246	247-277	≥ 278
5' 8"	< 121	121-163	164-196	197-231	232-261	262-294	≥ 295
5' 10"	< 129	129-173	174-208	209-242	243-277	278-312	≥ 313
6' 0"	< 136	136-183	184-220	221-257	258-293	294-330	≥ 331
6' 2"	< 143	143-193	194-232	233-271	272-310	311-349	≥ 350

The BMI benchmark has been used internationally and adopted by the World Health Organization (WHO) in part due to its ease in measurement (or estimate), although differences often arise between self-reported BMI and that measured by a medical professional. Extension of this benchmark are now used for children and adolescents. Obese refers to a BMI at or above the 95th percentile for the 2000 CDC Growth Chart for the U.S. for the applicable gender and age, while overweight refers to a BMI between the 85th and 95th percentile of this chart.

There are several limitations to the use of the BMI. For example, a simple weight-based measure cannot reflect the degree of fitness (in a small-scale study of patients in which chronic heart failure, body composition and risk factors were studied, the use of BMI misclassified body fat status in 41 percent of the individuals studied). In addition, Romero-Corralet al. (2008), based on NHANES III, found that BMI failed to discriminate between body fat percent and lean mass. Obesity, according to the BMI definition, was present in 19.1 percent of men and 24.7 percent of women, while an obesity measured by body fat percent (greater than 25 percent in men and greater than 35 percent in women, according to WHO reference standards defining obesity) by bioelectrical impedance analysis was present in 43.9 percent of men and 52.3 percent of women. In this case, the accuracy of BMI in diagnosing obesity was shown to be limited, particularly for individuals in the intermediate BMI ranges, in men and in the elderly. They hypothesized that these results may help to explain the obesity paradox found in some studies referred to in Section 3 of this paper.

BMI also does not differentiate where weight is carried, with visceral or abdominal adiposity in some cases being a more serious health hazard than overall weight level. Other

practical measurement benchmarks may prove more useful in measuring health conditions or as valuable supplements to BMI for certain population segments.

Women tend to have a higher percent of body fat stored in subcutaneous rather than visceral adipose tissue. Because of the difference, women will tend to have a higher percentage of body fat than men at the same BMI. A given BMI level may be the same for men and women and for people of different ages, but may not represent the same percentage of body fat, the same degree of risk or even the same degree of overweight in comparison with a weight standard.

Because direct measurement of fat is difficult, several measures have been used. Other metrics have also been used to benchmark overweight and obesity, either in place of or supplementary to the BMI. None so far have yet proven as easy to apply and consistently measured as a benchmark. These have included waist circumference (sometimes with 102 cm [40 inches] for males and 88 cm [35 inches] for females representing an at-risk cutoff), waist-to-hip and waist-to-height ratios, and skin-fold thickness. Based on available research, these alternative obesity metrics may be superior to BMI for certain segments of the population or to supplement BMI, especially at the youngest and oldest ages.

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Other relevant references, some of which are referred to in the body of this paper, are included in the list of references in Gutterman, S. 2008, "Human Behavior: an Impediment to Future Mortality Improvement, a Focus on Obesity and Related Matters." In many cases, it is important to note that useful and relevant information may be included in the source documents that are summarized in this paper, including possible limitations to their findings and the basis for the findings summarized here and statistical confidence intervals that may be important to recognize the significance of quantitative findings indicated in this paper.

