2014 International Congress of Actuaries
Obesity and Mortality, by Sam Gutterman, FSA, CERA, FCAS, MAAA

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ABSTRACT

The percentage of the population who are obese has grown dramatically on a worldwide basis over the last several decades, with the prevalence of obesity stabilizing recently in the United States. Although there have been numerous studies of the effect of this trend on mortality, their findings have been inconsistent and controversial, in part because of methodological differences and the complexity of the relationship between obesity and mortality.

The objective of this paper is to discuss the issues surrounding this relationship and to shed some light on the likely effects of this epidemic. In particular, the so-called obesity-mortality paradox, where mortality experience is lower for those individuals who are overweight and in some cases those who are obese compared with mortality experience for those who are in the normal weight category, is examined in some detail. Numerous issues associated with this reported paradox are discussed. Although more recently reported studies of the relationship between mortality and obesity seem to indicate that obesity is related to a reduced percent of additional mortality, this may in part be due to the shorter average time that those currently obese have been exposed to their condition. Additional premature deaths may arise in the future as more individuals who have become obese are exposed for a longer period to excess adiposity tissue.

Nevertheless, the amount of health care costs and rates of disability associated with being obese are significant, contributing to the increase in overall health care costs as those who are overweight and obese become more numerous and age. Although the issues surrounding obesity are being addressed with a great deal of activity and publicity, they have and will continue to prove quite challenging for both individuals and society to manage and overcome.

1. BACKGROUND

Our weight regulatory systems are being overwhelmed by the sheer quantity and unhealthy mix of available nutritional inputs and inadequate energy use; to such an extent that an increasing number of individuals have been unable to fully adapt. One result has been a massive societal (especially, but not exclusively in the U.S.) weight gain over the last third of a century, with an increased number of adults and children becoming obese in all population segments. This trend is threatening the substantial worldwide progress made in the postponement of illness and death.

Although many factors have contributed to the massive increase in obesity, its fundamental causes are rooted in the nature of current Western culture, with its behavioral incentives to live an increasingly sedentary life (e.g., screen

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[1] Also presented at the 2014 Living to 100 Symposium

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watching and driving a car) and to consume a high-fat, energy-dense diet. Becoming obese has never been easier. While historically people were mostly occupied with obtaining food, they are now more concerned with enjoying it, leading to passive over-consumption of an unhealthy mix of food and drink that is more processed and effectively marketed, while at the same time more affordable and appealing.

Even some of what many feel are beneficial trends, such as the increase in mothers' (of all socioeconomic levels) working hours, can have a negative consequential effect on the entire family's body mass index (BMI) and extent of obesity. This can lead to being more reliant on processed and less healthy food, foods prepared away from home, as well as increases in snacking and sedentary activities such as time spent watching screens, offset to some extent by being slightly more physically active.

Larger serving sizes, the economics of food including simultaneous increases in the cost (and inadequate supply in some neighborhoods) of fruits and vegetables while the cost of processed foods has decreased, as well as volume discounts for increasing size of food packaging in restaurants and grocery stores, excessive use of fructose sugar-flavored drinks, low levels of brown adiposity tissues, and the almost-inevitable dieting failures is a long list of current facts of life in many areas. Today's modern computer-dependent, sleep-deprived, and physically inactive society of food abundance, together with convenient, and economically driven eating habits, are just some of the big trends that have adversely affected human behavior and in turn often lead to higher BMIs. In addition, financial stress can lead to obesity, as for example caused by unemployment that can make it more difficult to afford healthy foods.

Is weight gain an inevitable consequence of economic development? But in spite of this long recitation of adverse factors, hope has not been extinguished, as there remains a very health conscious and physically fit segment of society. We will see how strong and effective this cultural backlash becomes.

Similar trends in nutrition and physical activity have emerged everywhere, although cultural and genetic differences have resulted in a wide range of levels and distribution of obesity prevalence. In lower-income countries, obesity has mostly affected middle-aged adults, especially females, from wealthy and urban environments, while in higher-income countries it has affected both genders and those of all ages, although disproportionately concentrated in certain disadvantaged groups. Many individuals have chosen to accept a greater body weight rather than to limit eating and to devote time to things other than home-cooked meals and exercise.

Even though genetics has been the source of an estimated 40 to 50 percent of obesity prevalence, the rapid upturn in weight points to a major role played by shifts in behavior. Although genes may determine the individual's metabolic rates and thus may influence how efficiently the body burns fat to produce energy, metabolism can only explain modest differences in weight—not the huge changes that have driven this epidemic. Individuals' bodies react to different types of calories in diverse ways. For example, it has been found that a variation in the FTO gene can predispose people to weigh an average of seven pounds more than people without the variation, although there is no clear indication so far why this occurs. It can also be affected by a person's gut bacteria, as well as ineffective personal control mechanisms and environmental, lifestyle and nutritional factors.

In summary, genetic, biological, and behavioral factors, as well as their complex inter-connections, have contributed to today's high prevalence of obesity.

Obesity is popularly defined for adults in terms of a body mass index (BMI, weight in kilograms divided by height in meters squared) greater than 30, with "overweight" being between 25 and 30. The current prevalence of obesity is a little more than 35 percent of the adult population in the United States, as seen in Figure 1 and Tables 1 and 2. This index will be discussed in depth later in this paper.
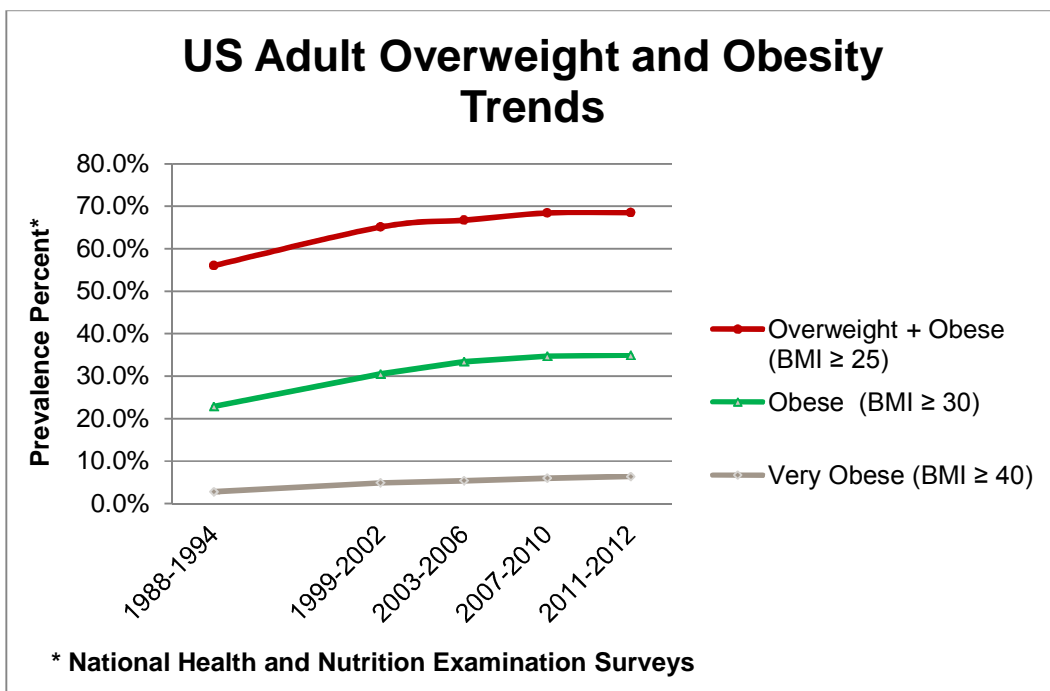
Table 1 shows the changes in adult obesity over the decade of the 2000's for gender and age and racial/ethnic categories between the 1999-2000 and 2009-2010 National Health and Nutrition Examination Surveys (NHANES), while Table 2 indicates, based on the more recent NHANES, the gender, age and racial/ethnic makeup of various obese classes.

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Independently, a semi-annual survey of self-reported weights and heights conducted by Gallop between 2008 through the 2013 indicated relative stability over this period in obesity prevalence of between 25.5 percent and 27.1 percent (as discussed below, self-reported BMIs tend to be lower than professionally measured BMIs. The 27.1 percent reported in 2013 was an increase from 26.2 percent in 2012.
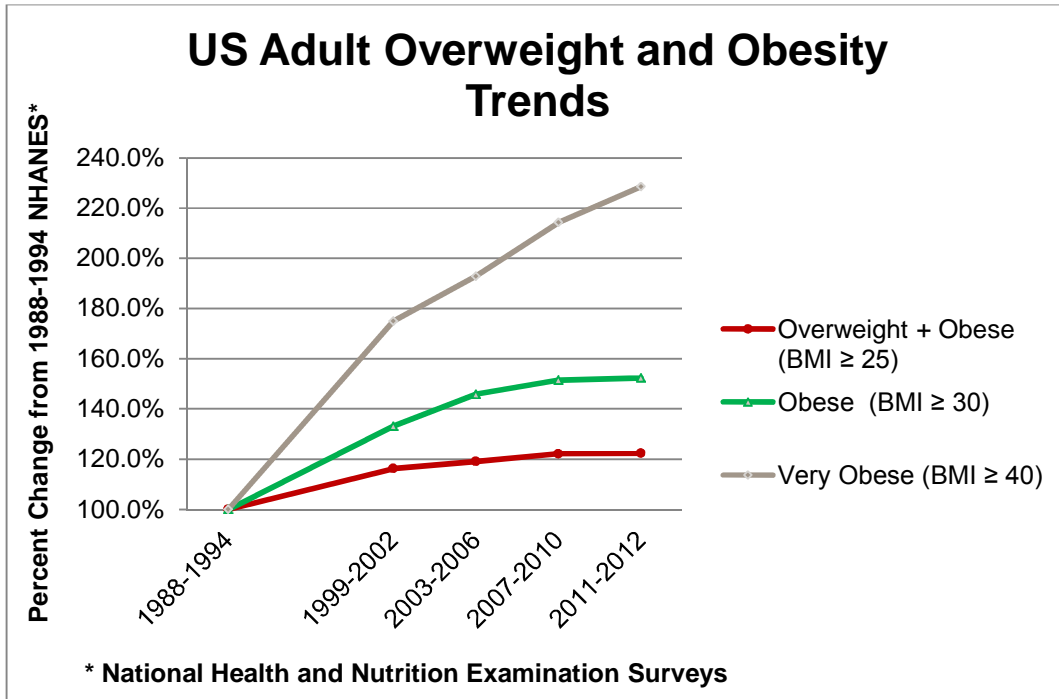
Not only has average weight and BMI increased over the last few decades, but its distribution has also shifted, significantly increasing the percentage of those extremely obese. This BMI prevalence curve shift (highlighted in Figures 2 and 3, especially noticeable in the very obese) has resulted in about 6.3 percent of adult Americans in the class 3+ obese category in 2009-2010, of significant concern because as discussed below the morbidly obese experience extremely high mortality and health care costs. The percent of those in class 2+ obese (BMI of 35 and above) was 15.4 percent of the adult population in the 2009-2010 NHANES. This is unfortunately an area in which Americans stand out.

Figure 1

Figure 2

US Adult Overweight and Obesity Trends

* National Health and Nutrition Examination Surveys

Table 1 – Adult obesity as a percent of U.S. population
2009-2010 compared with 1999-2000 by age and gender

Ages	Males				Females			
2009-2010	**All**	**White (non-H)**	**Black (non-H)**	**Mexican American**	**All**	**White (non-H)**	**Black (non-H)**	**Mexican American**
20-39	33.2%	34.5%	35.8%	32.7%	31.9%	26.9%	56.2%	37.8%
40-59	37.2	37.4	42.6	38.1	36.0	31.8	62.7	53.9
60 +	36.6	37.1	37.8	40.7	42.3	41.8	55.5	42.5
Total	35.5	36.2	38.8	36.6	35.8	32.2	58.5	44.9
1999-2000								
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	27.8	27.3	28.1	28.9	33.4	30.1	49.7	39.7

sources: NHANES, Flegal et al (2012); totals are age-adjusted; non-H refers to non-Hispanic

As can be seen in looking at Tables 1 and 2, obesity prevalence for adults during the decade of the 2000s has been relatively stable but slightly increasing (from 27.8 percent to 33.5 percent for males and from 33.4 percent to 36.1 percent for females between the 1999-2000 NHANES and the 2011-12 NHANES). Due to nature of the samples involved in these nationally representative surveys, statistical variations may be at work so it is difficult to determine whether the actual values are stable or slightly increasing over the decade. Table 2 shows that the percentage of Asian Americans who are obese is much smaller than that of the other race/Hispanic origin groups shown; however, some studies have shown that Asians have more body fat than whites, especially at lower BMIs, and there are concerns that health risks begin at a lower BMI among Asians compared with others.

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Table 2 – 2011-2012 adult obesity as a percent of U.S. population
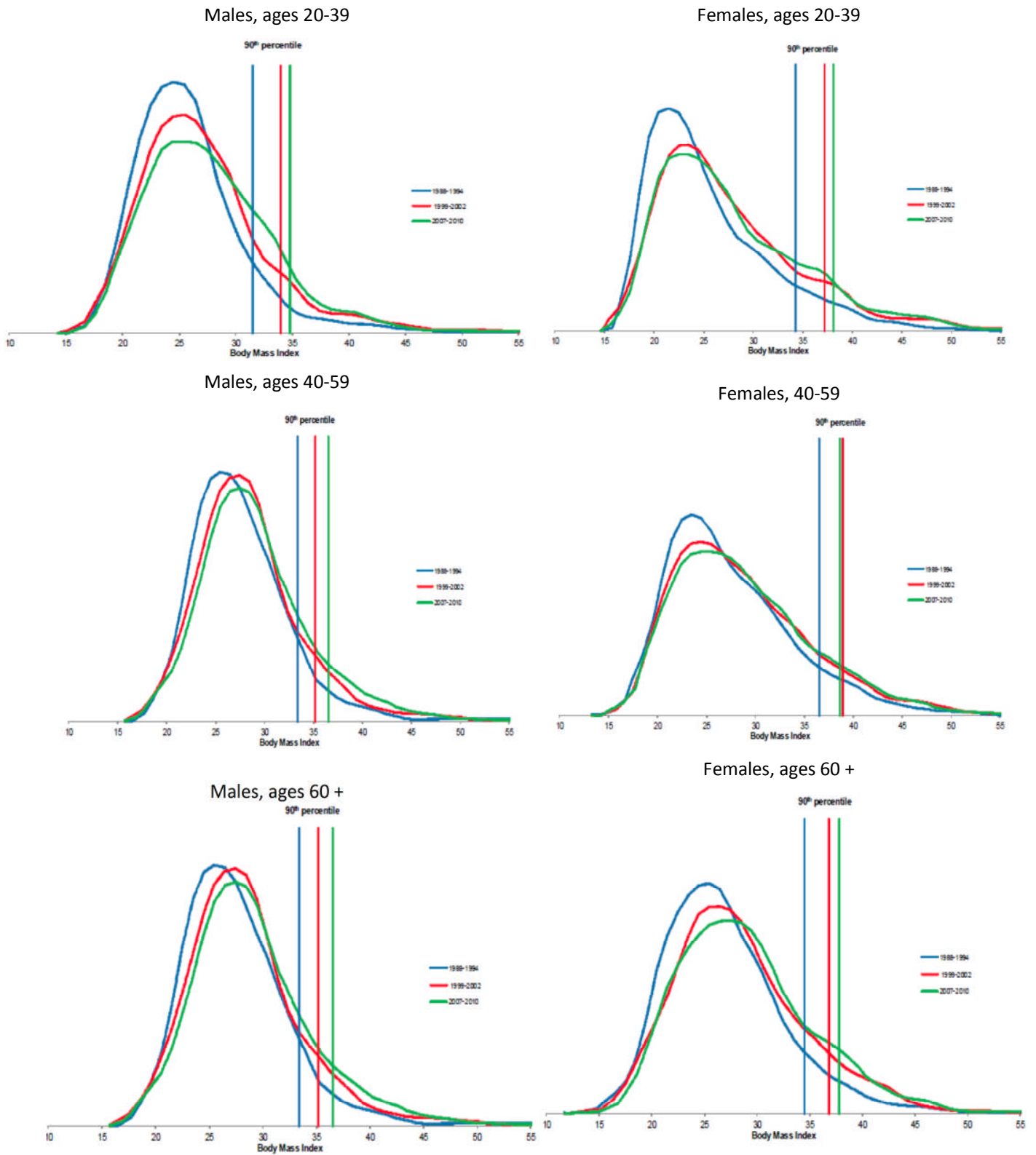### All obese, class 2+, and class 3+ by age and gender

Ages	Males					Females				
All obese	All	White (non-H)	Black (non-H)	Hispanic	Asian (non-H)	All	White (non-H)	Black (non-H)	Hispanic	Asian (non-H)
20-39	29.0%	24.6%	34.9%	42.0%	12.0%	31.8%	27.8%	55.8%	35.8%	10.9%
40-59	39.4	41.1	38.2	39.9	11.0	39.5	36.3	58.6	51.9	11.8
60 +	32.0	31.8	39.2	37.3	4.9	38.1	35.9	54.8	47.1	11.9
Total	33.5	32.4	37.1	40.1	10.0	36.1	32.8	56.6	44.4	11.4
Class 2+ obese										
20-39	11.9	10.0	18.0	14.9	3.8	15.4	13.7	30.6	15.1	1.1
40-59	12.2	12.8	15.7	8.7	0.0	19.1	16.9	30.4	25.5	4.6
60 +	11.2	10.9	12.8	11.9	0.8	16.3	15.5	25.0	20.5	3.6
Total	11.9	11.2	15.9	11.9	1.7	17.0	15.3	29.2	20.2	3.0
Class 3+ obese										
20-39	3.5	2.6	5.6	5.4	0.6	7.7	6.8	17.5	5.8	1.1
40-59	5.4	5.2	8.9	2.9	0.0	9.8	8.8	17.9	9.1	1.9
60 +	4.1	3.6	5.7	2.2	0.0	6.8	6.2	12.1	8.2	1.0
Total	4.4	3.8	6.9	3.7	0.2	8.3	7.4	16.4	7.6	3.7

sources: NHANES, Flegal et al (2014); totals are age-adjusted; non-H refers to non-Hispanic; certain Asian (non-H) categories have very low sample size

Figure 3, taken from Flegal et al (2012), illustrates the movement to the right in the distribution of BMI among most gender and age categories found in various NHANES. The figures also show the 90th percentile of each BMI distribution.

Figure 3 – Smoothed distributions of BMI by age and gender for 1988-1994, 1999-2002, and 2007-2010

Males, ages 20-39

Females, ages 20-39

Males, ages 40-59

Females, 40-59

Males, ages 60 +

Females, ages 60 +

source: Flegal et al (2012), NHANES

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Obesity can be the result of a complex interaction of forces and has many adverse consequences, directly contributing to conditions such as diabetes and hypertension, while indirectly contributing to others such as heart disease and stroke. It increases total blood volume and cardiac output and clogs arteries, resulting in a greater cardiac workload that can adversely affect hemodynamics and the cardiovascular structure and its function. Chronic diseases and conditions often associated with obesity include:

- Type 2 diabetes. Being highly related to diabetes, obesity is in turn associated with a doubling of the risk of heart disease and stroke, as well as being a leading cause of blindness, kidney failure, and non-traumatic amputations. Almost 90 percent of those with diabetes are overweight or obese.
- Cardiovascular and heart diseases. Obesity is associated with numerous cardiac complications, including coronary heart disease, congestive heart failure, angina, atrial fibrillation, and sudden death.
- Cardiovascular risk factors. These include hypertension, adverse cholesterol, and triglycerides.
- Cancers. The American Cancer Society has indicated that being overweight and obese is a cause of 14 percent to 20 percent of all cancer-related deaths, including cancers of the colon, esophagus, kidney, and pancreas.
- Kidney, liver, and gallbladder diseases.
- Psychological disorders. These include depression, anxiety, stress, bipolar disorder, schizophrenia, sex disorders, weight stigma, and dementia. In addition, fear of obesity can lead to avoidance techniques including eating disorders.
- Others, including musculoskeletal problems such as chronic back-pain, osteoarthritis, painful joints, arthritis, asthma, falls, and sleep apnea, as well as a general reduction in quality of life.

Although these associations generally relate to all classes of obesity, they particularly apply to the extremely obese. Compared with those in the "normal" BMI range (referred to in this paper as normal BMI, usually those with BMIs between 22.5 and 24.9, although sometimes extended to those with lower BMIs), extremely obese adults have been observed as having seven times the risk of diabetes, six times the risk of hypertension, four times the risk of arthritis, and three times the risk of asthma. Nevertheless, even with respect to diabetes, obesity may be an early symptom rather than an underlying cause. These conditions may be driven primarily by the underlying causes of obesity, such as poor nutrition and lack of physical fitness, rather than or in addition to being the result of obesity.

The trend in childhood obesity over the last several decades has also been an increasing one, with potentially serious long-term effects. Bethell et al (2013) indicated that the 2007 National Survey of Children's Health showed while the percentage of children ages 10-17 who are overweight remained relatively stable between 2003 and 2007, those who are obese increased significantly from 14.8 percent in 2003 to 16.4 percent in 2007.

Since 2007, obesity rates for children have experienced a modest decline in several cities; for example, a reduction of 5.5 percent in New York City kindergarten to eighth grade children (21.9 percent to 20.7 percent) between the 2006-07 and 2010-11 school years, with reductions in all ages, ethic-racial groups, and family income levels (although decreases were smaller among black and Hispanic children and students in schools in high-poverty school neighborhoods). Significant differences exist between population segments: obesity risk factors are greater and growing in children whose families are of lower income and whose parents have a lower amount of education, whose nutrition is less healthy, who are publicly insured, who are boys, who are black and Hispanic, who live in the southeast region of the U.S., and who live in poorly kept up neighborhoods. An August 2013 CDC study indicated a slight decline (2.8 percent) between 2008 and 2011 in obesity of 2 to 5 year old low-income American children in forty states, although there has so far been no consensus as to the primary contributing factors to this decline.

Table 3 shows American childhood obesity prevalence by race/Hispanic origin, age and gender categories from the 2011-2012 NHANES. Obesity is defined for children as the 95th percentile of the 2000 growth charts as published by the CDC. Note that several of the specific categories shown in this table are based on relatively small sample sizes, although estimated on a nationally representative basis. As a result, it is more important to follow trends in these values over a long period, rather than relying on the results of this biannual survey. Although there seems to be some decreases in these rates compared to the prior survey, the trend over the last several surveys does not indicate a significant change in obesity prevalence for

children in the U.S. For public policy implications, it will be important to follow these prevalence rates change in the near future.

Table 3 – 2011-2012 obesity in children as a percent of U.S. population, by gender, age, and race/Hispanic origin

Ages	Boys					Girls				
All obese	All	White (non-H)	Black (non-H)	Hispanic	Asian (non-H)	All	White (non-H)	Black (non-H)	Hispanic	Asian (non-H)
2-5	9.5%	6.3%	9.0%	18.0%	1.9%	7.2%	0.6%	13.9%	15.2%	4.7%
6-11	16.4	8.8	25.9	28.6	13.2	19.1	17.9	21.7	23.4	3.7
12-19	20.3	18.3	21.4	21.3	14.8	20.7	20.9	22.7	21.3	7.3
Total	16.7	12.6	19.9	24.1	11.5	17.2	15.6	20.5	20.6	5.6

source: 2011-2012 NHANES; obesity defined as those children with weight at least as great as the 95th percentile of CDC Growth Charts

Frederick et al (2013) indicated that, based on the 1988-2010 NHANES and the 2003-2011 National Survey of Children's Health, this overall reduction in obesity may have occurred due to the same forces that have resulted in a decrease in average energy intake in adolescents. The decrease from 2,487 kilograms in 1988-91 to 2,150 kilograms in 2009-10 in adolescents with college-educated parents was larger than the corresponding decrease in adolescents with high school educated parents from 2,271 kilograms to 2,105 kilograms. This differential appears to have been accompanied by a smaller percent of calories from carbohydrates and from fast food. In addition, 94.7 percent of adolescents of college-educated parents reported some physical activity in the previous 30 days, compared with 82.1 percent of those with only high school educated parents. Thus, it appears that, although on an overall basis obesity prevalence has stabilized or reduced somewhat in at least selected locations between 2003-2011, obesity prevalence in adolescents in high socioeconomic status and educated families have decreased, while obesity prevalence of those in lower-socioeconomic situations may have continued to increase, caused by differences in both caloric intake and physical activity.

Not only are obese children more likely to experience health and psychological problems during childhood, but they also likely to remain obese. In addition, they tend to be less likely engaged at school and more likely to repeat a grade.

The obesity epidemic has not been restricted to the U.S. It has been estimated that about 500 billion people are obese in the world with about an additional 1.0 trillion being overweight. Figure 4 and Table 4 show regional data taken from the United Nation's 2013 report entitled "The State of Food and Agriculture", which in turn were derived from Funicane et al (2011) and Stevens et al (2012). In part, the trends shown have been the result of the spread of some of the unhealthy nutritional habits currently embedded in Western culture.

Figures 4 and 5 provide perspectives on regional trends over the last several decades. Almost all regions have experienced significant growth in the number of those obese, albeit from considerably different starting points. Although the level of obesity may be leveling off in the U.S., that is not the case in all countries. For example, Midthjell et al (2013) indicated that, as found in the three waves of the HUNT study (90,000 participants), Norwegians' average BMI and the rightward shift in their BMI distribution curve continues, during which the obesity prevalence for males increased from 7.7 percent, to 14.4 percent, and to 22.1 percent, and for females from 13.3 percent, to 18.3 percent, and to 23.1 percent from the 1984-86 to 1995-97 and 2006-08 HUNT surveys, with the greatest increase in obesity occurring in the youngest adult age groups.

Figure 4
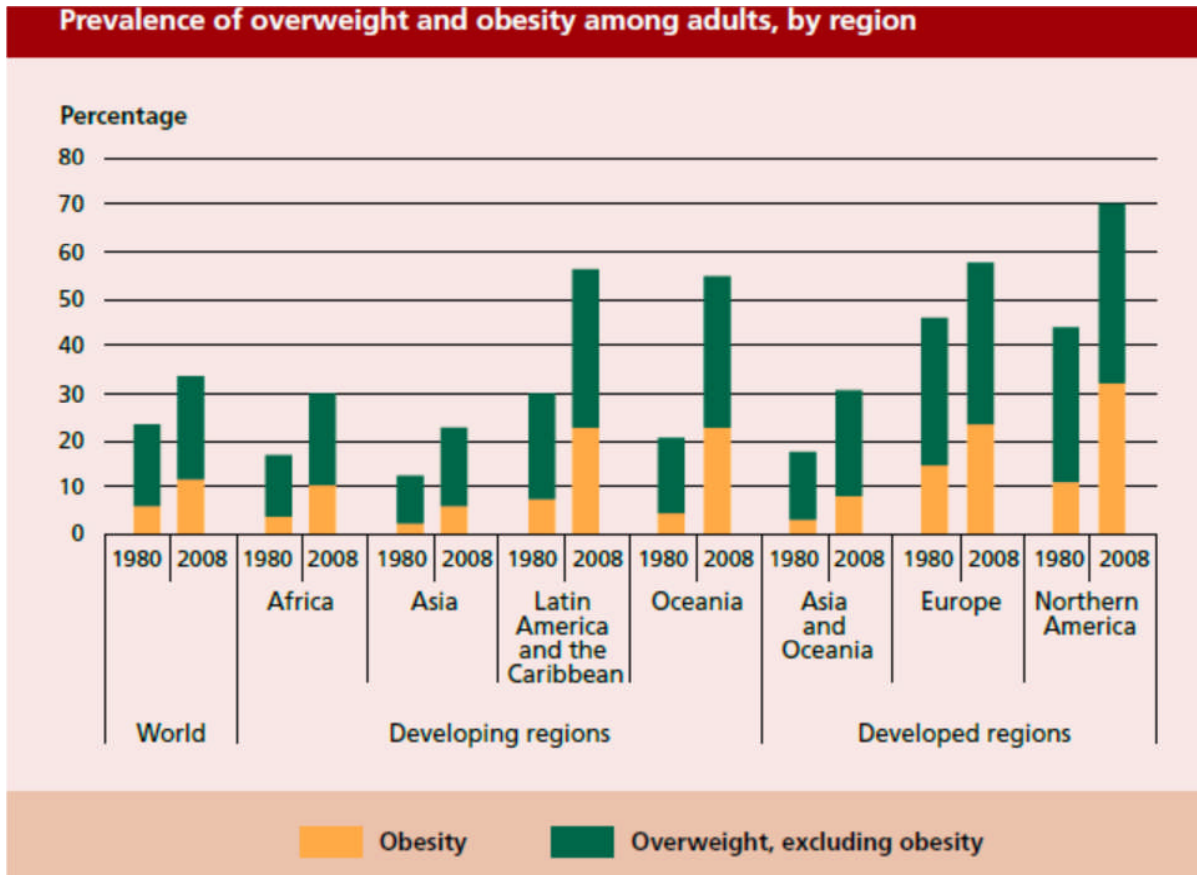
As indicated in the United Nations report, adult obesity in 2008 by developing/developed regions is shown in Table 4.

Table 4 – Obesity by geographic regions

|			
World	11.7%	Latin America and Caribbean	23.4%
Developing Countries	8.7	Caribbean	20.3
Africa	11.3	Central	30.4
Sub-Sahara	7.5	South	21.6
Middle	3.9	Oceania (excl Australia & NZ)	22.4
Northern	23.0	Developed Countries	22.2
Southern	31.3	Asia and Oceania	7.8
Western	6.6	Europe	21.4
Asia	6.0	Eastern	23.3
Central	18.4	Northern	22.9
Eastern	5.6	Southern	20.5
South-Eastern	5.3	Western	18.5
Southern	3.2	North America	31.0
Western	28.6		

Figure 5 – Obesity by geographic regions
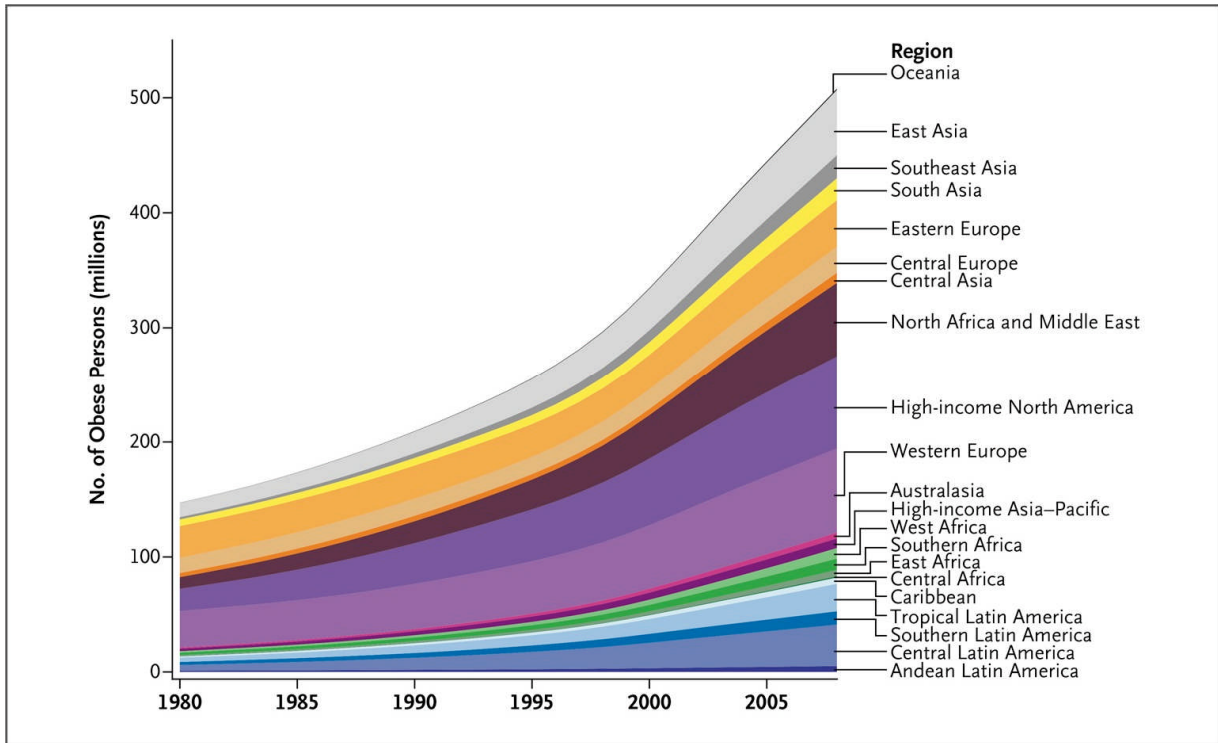
source: Ezzati and Riboli (2013)

Several comments regarding obesity in different countries follow. Obesity prevalence among adult Canadians doubled in most age / gender categories during this period. In some underdeveloped countries, subpopulations such as the underweight in South Asia have to date experienced greater health problems due to historical or current levels of malnutrition than to being overweight. However, in some cases these conditions may be changing. In addition, the lower rate of obesity in East Asia may indicate, due to genetic, ethnic, and cultural differences, as well as the proportion and distribution of adipose tissues in their body, a lower sensitivity to weight, measured by BMI, with respect to mortality, with greater diabetes prevalence and cardiovascular disease at lower BMIs (see below for a possible Korean exception) linked to proportionate increases in body fat. Figure 6 shows trends for selected countries.

Figure 6 – Trends in obesity in selected countries
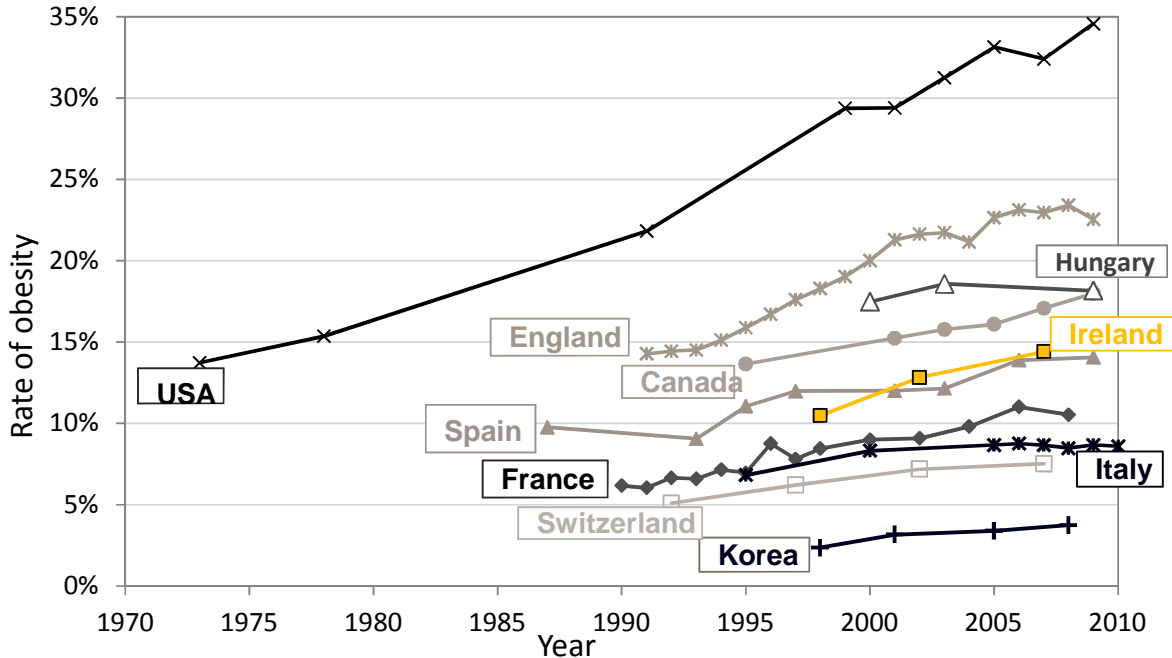
The case of obesity in Mexico is especially revealing. The United Nations (2013) indicated in mid-2013 that, measured on a self-reported basis, the percentage of those obese in the Mexican population is now greater than the corresponding percentage in the U.S. Until relatively recently Mexico's nutritional concerns were focused on malnutrition; now it is on excess weight, with the government becoming aggressively involved in attempting to reduce its citizen's obesity level by both "sin taxes", a peso on a liter of soda and an 8 percent tax on junk food (more than 275 calories per 100 grams) and a massive publicity campaign, including a ban on advertising to children in the afternoons and evenings and at the weekends. Its rate of childhood obesity may have already exceeded that of the U.S., and according to the United Nations is the world's highest. It is already leading the world in the prevalence rate of type 2 diabetes, which is also emerging at earlier ages than in the U.S. This has been, in part, the result of the recent surge in Mexican disposable income, especially in its Northern and Central regions, with many of its people facing the challenge of effectively managing the nutritional transition in becoming a wealthier nation. In reaction to what some believe their water as being unsafe to drink, an enormous amount of cola is drunk by Mexicans, along with a large amount of fried and fatty food is eaten, in part because more nutritious food such as fruits and vegetables that used to be food staples, are being priced out of the reach of many of lower income. An estimated 40 percent of the diet of rural Mexicans is now derived from packaged food, rather than from what they grow. Cash provided to rural families is now often spent on fried snacks and sodas. Carbohydrate and lard-loaded dishes are an everyday part of the family menu, instead of being eaten only on holidays. In addition, there are an increasing number of fast-food outlets and a great deal of non-nutritious foods served at schools.

In developed countries it is the well-off who have mostly managed to stay slim – or have at least gotten less fat – relative to those less well-off (e.g., American adolescents with college-educated parents are less than half as likely to become obese as those whose parents lack a high school degree). In contrast, in poor and middle-income countries, the more affluent are usually the first to put on excess weight. As their economies develop, they pursue the same habits and eat similar food as in the developed world, and their poor tend to become obese in disproportionately large numbers, as is the case in Mexico.

Many factors have been blamed as being a cause of the obesity epidemic (and disease, as designated by the American Medical Association in 2013). One candidate has been consumption of high fructose corn syrup (HFCS), whose use increased

by a factor of ten in the U.S. between 1970 and 1990, far exceeding the changes in intake of any other food or food group, representing 40 percent of caloric sweeteners added to food and beverages.

Sugar from sugar-sweetened beverages (including sodas, fruit drinks, sport drinks, low-calorie drinks, and sweetened teas) is the largest single caloric food source in the U.S., approaching 15 per cent of daily caloric intake for several population groups. It is nutrient poor and often is used in conjunction with salty foods and fast foods, which aren't especially healthy either. Among adolescents, sugar-sweetened beverages are currently the primary source of dietary fructose and total sugar.

Sucrose (table sugar) consists of roughly equal proportions of glucose and fructose. The most important natural source of fructose is whole fruit, which in contrast with HFCS (consisting of 55 percent fructose and 45 percent glucose) has been associated with reduced risk of obesity and related metabolic disorders. The favorable effect of whole fruit is likely to be the result of the slower digestion rate compared with that of sugar-sweetened beverages, producing portal fructose concentrations that do not exceed hepatic metabolic capacity. Thus, although fructose per se is not associated with adverse effects, excessive intake of refined sugar can play a significant role in determining the extent and level of the degree of obesity and related diseases, in part because in sufficiently large doses it can overwhelm hepatic biochemical pathways. Rapidly absorbed forms of glucose can also contribute to these diseases, especially considering their much greater caloric contribution to typical diets than fructose (and not every country that has experienced a surge in obesity uses HFCS to as great an extent as the U.S.).

The metabolic effects of HFCS are similar to that of sucrose. Fructose, a 6-carbon sugar, is more than twice as sweet as its isomer glucose. It has been associated with excess caloric intake, increased risk of diabetes, and cardiovascular diseases through an increase in body weight. Feeding studies indicate fructose increases food intake and body weight as it stimulates hunger, in contrast to glucose that tends to blunt hunger. While glucose stimulates insulin secretion, promoting glycogen synthesis in the liver and glucose uptake by tissues throughout the body, fructose does not directly stimulate insulin secretion and is taken up exclusively by the liver. It rapidly undergoes glycolysis, fueling de novo lipogensis under some conditions, which can cause fatty liver and hepatic insulin resistance or be exported, thus increasing serum triglycerides, systemic insulin resistance, and fat in adipose tissues, and may drive excessive uric acid production, thus possibly contributing to hypertension, high blood pressure, and endothelial dysfunction. In sum, it tends to increase total and visceral fat mass and accumulate ectopic fat in liver and skeletal muscles.

Basu et al (2013) conducted a study using cross-sectional data on diabetes and nutritional components of food from 175 countries using United Nations Food and Agricultural food supply data. They found that every 150 kcal/person/day increase in sugar availability (the equivalent of about one can of soda per day) was associated with an increased diabetes prevalence of 1.1 percent (in contrast, a 150 kcal/person/day increase in total calories adds 0.1 percent increase in diabetes prevalence). After controlling for obesity and other confounders (variables that are associated with mortality, but do not directly contribute to mortality by itself), no other food type was found to yield significant individual associations with diabetes. The effect of sugar was found to be independent of sedentary behavior, with its effect modified but not confounded by being obese or overweight. They also found that diabetes prevalence varied with the amount (both higher and lower) of sugar consumed. They therefore concluded that sugar included in processed food, in particular, monosaccharide fructose, may be the single most important cause of obesity and is associated with increased diabetes risk independently.

Consistent with the controversial nature of everything that has to do with obesity, some have expressed the view that studies of its adverse causes and effects often use unrealistically high amounts of fructose, resulting in limited convincing evidence indicating whether moderate amounts of fructose have adverse effects. These studies have also been criticized because fructose is usually consumed together with glucose. These critics have asked why a single component of diet should be singled out as representing "the" solution to obesity that they do not believe will work as advertised, as people will substitute something similar to if not worse than the outlawed or frowned upon food type. They claim it is better to look at overall caloric intake rather than to focus on a single food group and argue that focus should be better placed on reducing the intake of all highly processed carbohydrates, rather than solely focus on a particular food type such as refined sugar and sugar products. Note that, although this logic makes some sense, in some cases one has to start with something, especially if that something is a significant and easy-to-address item.

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In 2014 the WHO proposed that new guidelines that recommended that adults should limit themselves to no more than the equivalent of six teaspoons of sugar a day to avoid health risks, and changed its decade old recommendation that sugar should make up less than ten percent of an adult's daily caloric intake to five percent. This contrasts with the current average adult American intake of 16 percent, with the current top sources being carbonated drinks and energy and sports beverages.

All of these contributing factors inter-act. For example, even if sedentary behavior does not directly cause obesity, adults, and preschoolers planted in front of a screen tend to simultaneously eat sweet and salty foods rather than fruits and vegetables. In turn, HFCS use of reduces healthy nutrients by reducing milk consumption and increasing bone fracture risk and tooth decay. There is also strong evidence of significant interaction of sugar-sweetened beverages with genetic-predisposition for obesity, thus significantly affecting those people who are more susceptible to these adverse effects.

In summary, Mozaffarian et al (2011) found that in the health professional population studied over a four year period, the largest sources of the average increase in their weight of 3.35 pounds was due in part to (all measured in pounds): potato chips +1.69, potatoes +1.28, sugar sweetened drinks +1.00, meat +0.94; alcohol +0.41; new smokers +5.17, former smokers +0.14, sleep +0.31, and TV watching +0.31, with vegetables -0.22, fruits -0.49, yogurt -0.82, and physical activity -1.76.

2. MORTALITY

Despite the generally agreed-upon relationships between obesity and the adverse health conditions indicated above, the relationship between obesity and mortality (in contrast with the relationship between obesity and morbidity, discussed in "Morbidity" below) has been quite controversial. In fact at least at first blush, the increases in obesity over the last several decades seem inconsistent with the overall reductions in mortality rates that have occurred in almost all population segments worldwide during that same period. Although it can be claimed that without the increase in obesity, mortality improvement would have otherwise been even greater, it is by no means obvious what has been going on. The underlying mechanisms involved remain incompletely understood, with inconsistent findings with regard to the relationship between excess mortality and being overweight and moderate obese.

Many studies of obesity have found either a so-called J-curve (an example of which is shown in Figure 6) or U-curve relationship between BMI and mortality hazard ratios (usually with benchmark mortality rates based on experience of individuals in the normal BMI category). Both curve patterns indicate a greater mortality rate both for the underweight (with a BMI of less than 18.5 or sometimes less than 20.0) and those extremely obese, with a U-curve being flat at its bottom for a longer stretch, in those cases often representing relatively level mortality ratios across the normal and overweight categories, if not also the moderate obese level.

The values in these curves are usually hazard ratios[2]. Ratios greater than 1.0 represent the percentage of mortality rates in excess of the benchmark level. This relationship between BMI and mortality is sometimes more evident in those studies

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[2] In medical studies, association between two variables are commonly determined by Cox proportional hazards models, which assume that covariates are multiplicatively related to the hazard (in this case, mortality), based on the observation that if the hazard being evaluated is proportional to the risk, then the parameters (hazard ratios) can be estimated without consideration of the hazard function itself.

Non-parametric approaches have also been used, e.g., by Mehta and Chang (2009) and Zajacova and Burgard (2012). The latter, with a generalized non-parametric additive model using NHIS data between 1986 and 2004 for those between ages 50 and 80 with an average follow-up period of ten years, found that the optimal BMI was 28.1 for males and 27.2 for females. These are lower than the values that resulted from an application of a quadratic Cox model applied to the same survey population, with more of a V-shaped rather than U- or J-shaped curve (see Figure 6 for an example of the latter), especially for males. Zajacova and Burgard indicate that the parametric model usually used, especially those using broad BMI categories, may more often result in a U shape curve.

Wong et al (2011) used a data driven model to determine a non-linear and asymmetric functional form for mortality and BMI. With a logistic regression model reflecting a multivariable fractional polynomials procedure, Wong found that, using NHIS data between 1997 and 2000 resulted in a best fitting model containing the powers -1 and -2 for BMI (treating it as a continuous variable) with a J-shaped pattern for females and U-shaped pattern for males. They found the minimum point on the mortality-BMI curve by taking the first derivative with respect to BMI and setting it equal to 0. They avoided the proportional hazards model assumption that same pattern applies for everyone within a given BMI category (for example, by gender, age or a group of homogeneous risks within the normal BMI category), which as shown in this paper is not necessarily a good assumption. The resulting minimum mortality was at a BMI of 27 and 22 for never-smokers and ever-smokers, respectively, compared with 32 and

with a longer observation period, reflecting the long lag between onset of obesity and subsequent pre-mature death, especially with respect to adverse cardiovascular conditions. For example, the well-known Framingham Heart Study has found that being overweight can be an independent, long-term predictor of cardiovascular disease, associated with decreased life expectancy and increased premature mortality.

Studies of mortality (and morbidity) referred to in this paper can generally be categorized into two types: (1) cohort studies of a specified population, characteristics of participants determined at time of study entry, although some characteristics may be re-determined as time passes (e.g., weight that is re-measured or self-reported at periodic intervals), and (2) nationally representative studies, often consisting of smaller number of sampled participants. They both have advantages – cohort studies can study the effects of a variable in the same population over a long period of follow-up period, although their results can be difficult to extrapolate to a wider population, while nationally representative samples, although more easy to extrapolate results to a larger population, can suffer from relatively small sample size and changing population mix over time. In any case, the results of both types of well-designed studies need to be assessed based on their design and their ability to be relevant and comparable. This is the reason why well-designed meta-studies, which aggregate the results of individual study results, can be valuable if the individual study designs, metrics used, and objectives are similar.

Although still the dominant overall cause of death in most (even many developing) countries, deaths from cardiovascular disease have come to represent a diminishing proportion of total deaths for most developed countries (and an increasing cause of death for many developing countries) due to improvements in treatment of this disease and its risk factors. As a result, a change in the rate of mortality for these diseases has had a smaller effect on all-cause mortality over time, in part contributing to a diminished relationship between obesity and all-cause mortality.

Mehta and Chang (2011), in studying the effect of this secular change over the last several decades, compared non-overlapping periods of three long-standing sources of both all-cause and cause-specific U.S. mortality data – the Framingham Heart Study (original and offspring portion), the NHANES (I and III), and the National Health Interview Survey (NHIS, 1987-91 and 1997-2000 surveys) over a period from 1948 through 2006. Their overall conclusion was that the relationship between moderate levels of obesity and mortality has weakened over time, even while the size of the obese population segment has increased. In all three studies the interaction between class 1 obesity and mortality decreased over time, with hazard ratios declining from 1.27, 1.53, and 1.14 in the earlier period of each of these studies, respectively, to hazard ratios of approximately 1.0 in the later period. Although there was a significant decline in the association between mortality and class 2 and 3 obesity for NHIS (1.56 to 1.26), there was no similar reduction in hazard ratios for the more obese in the other two studies, nor in the relationships in the overweight (with hazard ratios of less than 1.0) or underweight categories. Relative mortality rates from cardiovascular disease for the class 1 obese was similar to that for all-cause mortality, with excess mortality decreasing by 89 percent in NHANES, 63 percent in NHIS, and by 47 percent in Framingham between the two sets of time. No significant trend in cancer or other cause mortality was indicated in this review of these studies.

Mehta and Chang (2011) indicated that these trends are the result of the obese also benefiting from declines in cardiovascular-related mortality even to a greater extent than persons with a normal BMI due to (1) increased effectiveness of treatment of cardiovascular diseases, (2) improvements in control of their risk factors, especially high blood pressure and total cholesterol, for example through increased use of lipid-lowering drugs (e.g., statins), (3) more aggressive treatment for risk-factor modifications for obese patients with diabetes relative to normal weight diabetes patients, and (4) changes in health behaviors. In addition, cohorts moving through the same attained ages have tended to be better educated and have

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23 for the same groups using a linear-quadratic proportional hazards model and the same data. They generally found the proportional hazards model to overestimate mortality in the upper part (obese) part of the BMI distribution, and for males they derived higher mortality estimates for subjects at low BMIs.

Chang et al (2013) used a mixed proportional hazard model, controlling for both observed and unobserved heterogeneity (e.g., through biases due to familial characteristics). Its use emphasized the importance and effect of co-morbidities (here including heart disease, hypertension, diabetes and stroke), which increased the chance of dying and was more pronounced for males, blacks, and the young. The effect of obesity-related diseases was, based on the NHIS, to shift the hazard ratio curve to the right, with the pattern being a U-shaped curve by BMI, with the effect reduced as age increases. The predicted life years lost was greater with larger BMI (for ages over 40, life years lost were either about the same or greater for those overweight than of normal BMI).

been subject to less significant work-related physical stress. Greenberg (2013b) confirmed that between NHANES I and III (about eighteen years between initial measurements), mortality for BMI-obese males improved to a greater extent than did mortality for BMI-normal males (hazard ratios of 2.22, 0.89 and 0.65 for NHANES I, NHANES II, and NHANES III, respectively).

The rapid inflow of people into the obese category over the past few decades has also resulted in a decline in the average duration of obesity for the average currently obese individual. To the extent that a long period or continued exposure to excess adiposity tissue (fat cells) lead to premature death, mortality results from recent studies of long-term risk to mortality for the obese may be understated so far, suggesting that the changes in relationships observed by Mehta and Chang may reverse in the future.

A possible conclusion that can be gleaned from a review of the results from many published papers and studies concerning the relationships between obesity and mortality, indicated below, is that seemingly inconsistent findings have been reached. An objective of the following discussion is to identify and discuss some of the methodological issues that may explain some of these differences.

2.1. Obesity paradox

A paradox is a set of results that seem on the surface to be illogical. The so-called "obesity-mortality paradox" (obesity paradox) represents a situation where mortality rates for the obese are better than those of normal BMI. Despite the role of obesity in the development of the chronic co-morbidities indicated above, once some of these conditions have manifested themselves, an inverse correlation of obesity with all-cause and even cardiovascular mortality has been observed (including in the case of a cardiovascular condition a need for repeat revascularization). If this is the case, having a BMI greater than "normal" may in some way provide some protective value. This situation has particularly been evident (1) in currently ill or recently operated-on patients and (2) in relatively recent studies in which mortality rates for those overweight, and in some cases the mildly obese, have been lower than those in the normal BMI category. It is likely that the origins of this paradox are multi-factorial, with its underlying mechanism(s) not yet completely understood.

A challenge in assessing the results of the multitude of studies has been the sometimes inconsistent findings relative to a specific medical or physical condition or population segment studied, even if the findings in these studies were seemingly statistically credible. However, some of this disparity may have been due to differences in study design and methodology that may be difficult to discern.

Early studies pointed to a significant association between obesity and adverse medical conditions, which usually tended to result in premature mortality. Thus, expectations developed that obesity, as well as those who were overweight, were associated with and probably represented a causative risk factor for premature mortality. In the 1980s obesity was linked to better mortality experience in kidney dialysis patients; in the 1990s patients with coronary artery disease undergoing percutaneous coronary intervention were found to experience a lower probability of death or complications compared with those in similar health circumstances of normal BMI. Subsequently, various studies of those suffering from conditions such as cardiovascular disease, myocardial infarction, stroke, and chronic kidney disease, have shown more favorable survival experience, at least in the short-term, for those in the overweight and at least in the moderate weight obese categories. For example, Lavie et al (2009) and Romero-Corral et al (2006)'s meta-analysis of forty studies indicated lower hazard ratios for both those who were overweight and class 1 obese (hazard ratios of 0.87 and 0.93, respectively), with increased mortality (a hazard ratio of 1.10 for all-cause mortality and 1.88 for cardiovascular mortality) for those of a BMI ≥ 35 compared with a benchmarked BMI of between 20 and 24.9.

Romero-Corral et al (2006) suggested that the cause of the observed obesity paradox was a lack of discriminatory power of BMI to differentiate all-cause and cardiovascular mortality between BMI and aerobic fitness levels, that is, between body fat and lean mass. Lavie et al (2009) indicated that the same paradox existed for those with both high and low body fat. It has also been shown that individuals with a combination of normal BMI and high body fat (or visceral or general adiposity) have greater mortality due to cardiovascular conditions and diabetes, especially among females and Asians/Asian Americans. This indicates that waist circumference (WC) or other measure of central (also referred to as abdominal or visceral) adiposity may represent a preferable metric by which to analyze mortality than solely relying on BMI as a metric. Note that, although

WC can be measured at minimal cost (thus being a useful proxy for more accurate imaging methods to assess abdominal fat), it also does not differentiate between subcutaneous and visceral adipose tissue.

Oreopoulos et al (2008) aggregated nine studies covering more than 28,000 patients with heart failure and found that those overweight had an all-cause mortality hazard ratio of 0.84 and all obese studied had a hazard ratio of 0.67. Other studies indicated in-hospital mortality significantly lower as BMI increases, even after adjustment for age, gender, blood pressure, and other potential risk-related factors.

McAuley and Blair (2011), in aggregating fourteen studies of 343,922 cardiovascular disease patients with a mean age of 63 with a follow-up period ranging from 2.0 to 8.4 years, suggested a U-shaped curve, with a steep reduction in mortality between those underweight (a BMI of less than 18.5) to about a BMI of 30.0, and level thereafter. In contrast, Wang (2013) reported greater mortality for those obese (a hazard ratio of 1.59) after aortic valve replacement with small prosthesis at both 30 days after surgery and periods up to eight years.

Uretsky et al (2010) followed 3,673 stress SPECT patients who were suspected of having coronary artery disease for a mean of 7.5 +/- 3.0 years. All-cause mortality hazard ratios (with a benchmark being experience for those BMI-normal) for those overweight were 0.54 and for obese was 0.49. These ratios were similarly low for all sub-categories studied, including those categorized by age, gender, and existence of chest pain. Other factors examined did not explain these low hazard ratios. For example, 47 percent of those in the normal BMI category had two or more coronary artery disease risk factors compared with 52 percent of the overweight and 68 percent of the obese, although on average those who were obese were four years younger and smoked less, but had more hypertension, family history of coronary artery disease, and diabetes. Note that it is also possible that those who were in the worst physical shape were not included because they were unable to participate in a stress test.

Recently, there have been similar mortality findings regarding those with diabetes, for which mortality was higher for those of normal BMI than those overweight or obese. An example is given in Carnethon et al (2012), who analyzed five longitudinal studies based on 2,625 people with newly diagnosed diabetes with hyperinsulinemia, insulin resistance, and hypertriglyceridemia and were predisposed to type 2 diabetes and premature coronary heart disease. Those with normal BMI had a higher body fat content and greater mortality (hazard ratios of 2.08, 1.52, and 2.32 for all-cause, cardiovascular, and non-cardiovascular mortality, respectively, with mortality for overweight and obese individuals as a benchmark—rather than those with normal BMI being the benchmark, as a large majority of those suffering from diabetes are overweight or obese) across minority groups and particularly among females.

Carnethon found no association between BMI and total mortality, but WC was significantly associated with adverse mortality. This appears to indicate that mortality may be better related to sarcopenic obesity (i.e., high body fat and reduced lean body mass), which is associated with low cardiorespiratory fitness and physical function that can lead to excess disability and premature death. In addition, the strength of association between normal BMI and total mortality became modestly stronger when WC was also considered in their models. Higher BMI could be the result of more lean muscle mass, which is more insulin-sensitive than adipose tissue and consequently metabolically favorable in those studied. They speculated that lower body weight in the presence of obesity-related metabolic disorders may reflect underlying illness that forms a predisposition to premature mortality. According to NHANES, 23.5 percent of American adults with normal BMI are metabolically abnormal. Carnethon also noted that overweight and obese patients with end-stage renal disease have better health outcomes than do leaner patients with the same disease.

A well-reported-on paper (Flegal et al (2013)) aggregated the results of almost a hundred studies that covered about 2.88 million individuals. Flegal found that those overweight experienced better mortality than those in the normal BMI category, with no adverse mortality experience reached until the higher classes of obesity (2 and greater). Flegal's results show an aggregate hazard ratio of 0.94 for those overweight compared to those in the normal BMI category, 0.95 for class 1 obese, 1.29 for class 2 and 3 obese and 1.18 for classes 1 through 3 obese combined (see Table 5). These relative findings remained substantively the same after adjusting for smoking status, pre-existing diseases, and type of weight and height reporting (self-reported compared with professionally measured). This illustrates that the use of a broad category (e.g., all obese) may produce incomplete and potentially misleading results if the population studied segments are too broad (e.g., all obese compared with separate class 1 and class 2+ obese) for a continuous variable (e.g., mortality across BMI).

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The findings of individual studies have in some cases shown quite different results. For example, Fontaine (2012) analyzed the results of five data sets, each studying at least five hundred Hispanic adults and at least a five year follow-up period adjusted for gender and smoking. Hazard ratios were found to be 0.38 and 0.84 for those overweight and 0.75 and 0.60 for obesity classes 1-3 for those ages 60 and younger and 70 and older, respectively, as well as an elevated hazard ratio for those underweight.

The meta-analysis in Flegal et al (2013) has been criticized by many experts. A discussion of the issues associated with the obesity paradox in general and their paper's findings in particular follows:

2.1.1 BMI is not a good measure of obesity

Several metrics have been used to identify those who are obese, but BMI, a simple function of weight and height, has been the most commonly (and currently almost universally) used, in large part due to its practicality. This is the case even though it may not be the best a surrogate and at best is an indirect estimate of a person's degree of adiposity and does not directly measure central adiposity. BMI's category cutoffs, which have been used to classify people as being underweight, normal, overweight or obese, have been endorsed by the World Health Organization (WHO). It is easy-to-measure and wide availability nature are valuable, both for study purposes and to communicate with individuals. In part as a result and for the purpose of aggregating the results of these studies, the scope of Flegal's paper only included studies assessing relative all-cause mortality based on overweight and individual classes of obesity as measured by BMI.

Obesity itself is fundamentally a concept that indicates that a person has a subjectively-determined specified level of overall excess fatness or adiposity in excess of a benchmark level at which energy is stored in fat adipose tissues. At a certain level this fatness or adiposity in turn secretes leptin (a 16kDa peptide) that can damage the cardiovascular system and resistin that can cause insulin resistance and type 2 diabetes.

The cut-points are determined by means of consensus, usually developed by a group of qualified health care professionals charged with its development. As indicated above, the cut-points currently in use with respect to BMI have been endorsed by WHO, although they have been challenged from time to time by some as being inappropriate for certain sub-populations, such as those from certain Asian countries or for certain age groups. For certain metrics and depending on the cut-off points used, some feel that its use can underestimate body fat.

The use of BMI in measuring the effect of obesity on mortality has been criticized for several reasons, similar to any metric solely based only on overall weight and height. These include:

- Adiposity/body fat. BMI doesn't measure and is not necessarily related to the amount of adiposity. It has been estimated that BMI fails to identify half the people with excess body fat. Cawley and Burkhauser (2006) concluded that obesity defined using BMI is only weakly correlated with obesity defined by percent body fat, with the differences resulting in substantial misclassification of individuals into weight/height categories, in part because BMI does not distinguish between fat and fat-free mass. However, Flegal et al (2009), in comparing body measures from NHANES 1999-2004, found that BMI, WC, and waist-to-hip (WtoH) ratio performed in a similar manner as an indicator of body fatness (using dual-energy X-ray absorptiometry (DEXA) to measure percent body fat and are more closely related to each other than with percentage body fat). They found that, although these measures may not be accurate surrogates for an individual, there was fairly good correspondence in the aggregate with percent body fat within gender-age groups and categories of percentage body fat. Flegal et al (2009) found that percent body fat tended to be more highly correlated with WC than with BMI in males, but had a significantly higher correlation with BMI than with WC among females (except in the older age category), while others have found that WC is somewhat more highly correlated with body fat among males than is BMI, but slightly less among females.

- Body composition. Body composition is more relevant to mortality than overall weight. The location of extra weight around the body's middle as indicated by central adiposity or proximity to certain internal

organs may be more important than total weight, as abdominal fat is usually more associated with greater health risks.

- Heterogeneity of any category. Those in each BMI category do not necessarily constitute a homogeneous group of people and body types. Individuals of any specific weight include those who are lean and those who are fat, who are never and current smokers, who recently gained or lost weight, and who have different percent of fat and muscle mass. Other factors should always be considered.

- Co-morbidities. BMI by itself is not as important as its combination with accompanying conditions (co-morbidities).

- Relation to age. The use of BMI may underestimate the adverse effects of excess adiposity tissues in older adults compared to these effects on younger adults, as there is a tendency to lose muscle and bone mass as fat tends to shift from peripheral to central body sites with age, with a resultant increase in WC and WtoH ratio but no reduction in BMI, which may result from inactivity, illness, or simply aging. Thus, weight and height history should be considered along with current measurements.

There is no current consensus as to which, if any, obesity measure represents the single best metric, or even a multiple set of measures that in combination is of sufficient practicality, accuracy and relevance to mortality and health. In addition to BMI, prime candidates include measures of body fat and central fat. The former is often measured by total body fat or percent body fat (total body fat divided by total weight), while the latter is commonly measured by means of WC or WtoH ratio, both of which can be difficult to measure or self-report in a consistent and accurate basis. However, in studies such as the fifty two country INTERHEART case-control study of myocardial infarctions, BMI was less relevant than measures of central obesity. In spite of its deficiencies, BMI is overall still the most commonly used metric since it is the most practical and consistent measure so far developed. In any event, since many studies had their initial measurement conducted on the basis of BMI, that metric will continue to be used for quite some time.

Other bases used as measures of obesity can also be available, e.g., Shah and Braverman (2012) favor the use of leptin as being more practical than, for example, using a DEXA, with obesity measured beginning at the 10 nanograms per milliliter level. The total volume of fat may determine the amount of leptin and resistin release, although percent body fat may be better where additional fat-free-mass dilutes the health effects of these secretions.

Using a DEXA full body scan (which measures body fat, muscle mass, and bone density) as a benchmark, Oreopoulos et al (2010) found that in a sample of patients who have suffered heart failure, BMI misclassified categories of body fat in 41 percent of those studied. Shah and Braverman (2012) found that, using the same type of scan on 9,088 patients in Manhattan, 46 percent and 74 percent of males and females, respectively were obese according to body fat measurement (in contrast to 27 and 26 percent of males and females, respectively, were found to be obese according to BMI). They also found that about 3 percent of males and 0 percent of females who were obese according to their BMI were not so classified according to DEXA findings. A total of 48 percent of females were misclassified as non-obese by BMI but were found to be obese by percent body fat, while in contrast 25 percent of males were misclassified as obese by BMI but were non-obese by percent body fat, consistent with a more muscular body composition. The obesity cut-off in terms of body fat percentage in this study used a currently accepted body fat percentage cut-points of 25 percent for males and 30 percent for females (in some studies 35 percent for females has been used)[3]. This body fat cut-off for obesity leads to a much greater percent of the population being classified as being obese than would result from accepted BMI-obesity categorization. This is not necessarily an indictment of that metric, as alternatively a higher threshold might prove to be more appropriate if desired.

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[3] In 1995, WHO defined obesity based on percent body fat to be ≥ 25 percent for males and ≥ 35 percent for females, while the most recent 2009 guidelines from the American Society of Bariatric Physicians (ASBP), an American Medical Association (AMA) specialty board used percent body fat ≥ 25 percent for males and ≥ 30 percent for females (which most studies comparing BMI with body fat percentage have used).

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When BMI is divided into fat and lean mass components, Oreopoulos et al (2010) concluded that a higher lean body mass and lower fat mass are independently associated with factors that are favorable with respect to chronic heart failure, and that, although BMI may not be a good indicator of adiposity, nevertheless it may be a better surrogate than others for lean body mass in many populations studied.

As Flegal et al (2013) identified, many studies have found either no significant association between BMI (for overweight and obese categories) and mortality or what this paper refers to as a BMI-obesity paradox. Some have found a more indicative relationship between mortality and central obesity measures, including WC and a WtoH ratio. Others have determined that percent body fat is more suggestive of a relationship, determined by means of, for example, skinfold thickness, body impedance analysis, or air displacement plethysmography, which are too cumbersome and time-consuming for use on a day-to-day basis. Computerized axial tomography (CAT), magnetic resonance imaging (MRI), and dual energy x-ray absorptiometry (DEXA) have also been used for this purpose, but the high cost of these tests makes them impractical for individuals to use, leading to what likely will be continued reliance on the BMI.

Examples of studies in which multiple obese metrics were assessed, generally indicating the superiority of metrics other than BMI in explaining mortality and body composition, include the following:

- Lahmann et al (2002), in the Malmö Diet and Cancer Study of 12,159 post-menopausal females with 5.7 years of follow-up, studied the effect of BMI, percent body fat, lean body mass, and the WtoH ratio, together with body composition derived from bioelectrical impedance analysis. Excess body fat was found to be associated with excess mortality in younger females, with a reduced association in older females. A weaker association was observed for BMI than for body fat percent (for males and females), with a stronger association with the WtoH ratio that was independent of overall body fat, and was a stronger predictor of mortality in females than in males (e.g., the hazard ratio for the third quintile of BMI was 0.85 for males between ages 46 and 59, and was 0.60 for males between ages 60 and 73, with corresponding ratios of 1.39 and 0.71, respectively, for females; corresponding values for the third quartile of WtoH ratio were 1.20, 1.09, 1.21, and 1.21, respectively). For both age and gender the effect of adiposity and WtoH ratio on mortality indicates the importance of considering more direct measures of adiposity, rather than BMI by itself, when describing obesity-related mortality risks.

- Tice et al (2006) compared hazard ratios for central adiposity (WtoH ratio) and BMI for 17,748 post-menstrual females, measured from 1990 to 1992 and followed up for nine years. The hazard ratio for those obese as measured by WtoH ratio was 1.3 in contrast with the hazard ratio for those obese as measured by BMI of 0.7.

- The European Prospective Investigation into Cancer and Nutrition (EPIC), a ten country study reported on by Pischon et al (2008), found that the use of WC enhanced the ability of BMI to predict all-cause and vascular mortality (the relative risks of the highest to lowest quintile of WC were 2.05 and 1.78 for males and females, respectively, and similarly, 1.68 and 1.51 for WtoH measures. Pischon noted that, after adjusting for BMI, the association of mortality and BMI was stronger among smokers than among non-smokers. They also observed that body mass is more closely related to the amount of visceral fat in males than in females, which may be one reason that the relative mortality risk among participants with a high BMI was higher for males than for females.

- Belloco et al (2010) found that BMI in males and WC in females better forecasted all-cause mortality. In this Swedish study of 41,000 adults with a ten year follow-up period, BMI, WC, and WtoH ratio were all positively related to mortality. In addition, no substantial effect modification between these three obesity measures and physical activity was found, as both sedentary behavior and the obesity measures seemed to increase all-cause mortality risk independently and additively. In addition, those who were obese and were sedentary had a hazard ratio of almost 2.0 compared with a benchmark of those of normal weight and a high level of physical activity, with weight here measured by the three metrics for males and by WC for

females. Belloco noted that the WtoH ratio may be more difficult to interpret because it could reflect the effect of either larger waist and/or smaller hip circumference, while WC is both more directly related to visceral adiposity and thus less complicated.

- Ioachimescus et al (2010), with a mean follow-up period of 5.3 years, compared results of WC and fasting triglycerides (TG) and BMI on Cleveland Clinic patients referred for primary and secondary prevention of coronary artery disease (high risk, but with no CAD at the time of reference). After adjusting for demographic factors, smoking, diabetes, blood pressure, and cholesterol, it was found that, although BMI was not associated with increased mortality (a hazard ratio of those BMI-obese of 1.06), a combination of WC and TG was independently associated with mortality with a hazard ratio of 1.46.

- Jacobs (2010) reported the results of the Cancer Prevention Study II Nutrition Cohort, a study of the association between WC and BMI and mortality of 14,648 American adults (with a minimum age of 50 and a median age of 69 in males and 67 in females in 1997) with about a ten year follow-up period and exclusion of those who had lost 10 or more pounds in the prior five years. A larger WC was associated with greater mortality, independent of BMI. After adjustment for BMI, a larger WC (and within each BMI category) was associated with greater mortality for both males and females. Those with very large WC (greater than 120 cm compared with those with a WC of less than 90 cm) had a hazard ratio of 2.02 for males and 2.36 for females whose WC was greater than 110 cm compared with those less than 75 cm). A 10 cm increase in WC was associated with a 1.16 hazard ratio for the normal BMI category, 1.18 for overweight, and 1.21 for obese, with similar hazard ratios for females. This result was explained by the fact that a small WC was associated with low BMI and low BMI was associated with increased mortality. Hazard ratios associated with WC were significantly lower for females with a history of a disease that could result in weight loss among females without those illnesses.

- In the linked 1998, 2003, and 2008 Scottish Health Surveys of 20,117 adults, Hotchkiss and Leyland (2011) found hazard ratios of 1.17 and 1.34 for higher values of WC and WtoH ratio, respectively, while mortality for BMI categories didn't reach excess levels until BMI reached 40. As in other studies, this study indicated significant adverse mortality experience for underweight BMIs, but not for lower values of WC or WtoH. Lavie et al (2013) indicates that patients with coronary heart disease with both low BMI and low body fat experience greater mortality, rather than either one alone.

- Petursson et al (2011) studied the relationship between mortality and participants' BMI, WC, hip circumference, WtoH, and waist-to-height ratios during a twelve year follow-up period of adult Norwegians in the Norwegian HUNT 2 Study. After adjusting for age, smoking, and physical activity, WtoH and waist-to-height ratios were found to be the best predictors of death, while hip circumference had a strong inverse association when adjusted for WC.

- Wormser et al (2011), summarizing 58 cohorts with at least one year of follow-up (in total 221,934 people without prior history of heart disease in seventeen developed countries, with 58 percent in Europe and 33 percent in North America) with BMI, WC, and WtoH ratio data, found that if systolic blood pressure, history of diabetes and lipid information are reflected, the addition of any of the three obesity measures does not significantly improve the prediction of cardiovascular disease risk (coronary heart disease, ischemic stroke, and cardiovascular disease). They observed, however, that obesity overall remains a key consideration, since significant cardiovascular disease risk factors tend to be adversely affected by being overweight. They found that each of the three measures of obesity had a similar strength of association with cardiovascular disease. However, in this study BMI was shown to be superior to WC or the WtoH ratio in terms of long-term reproducibility. Individuals younger than age 70 had three to four times the amount of excess risk than those at older ages.

- Coutinho et al (2013), combining the results of five studies from three continents of participants with cardiovascular disease, assessed the relationship between BMI and central adiposity measures (WC and

WtoH ratios). They found that, although there was significant interaction between BMI and WC or WHR, those individuals with the highest quintiles of central obesity had a greater mortality risk than other combinations. Conversely, those whose BMI who were overweight or obese and those in the lowest quintiles of central obesity, had lower mortality than other combinations. They found that, while increasing age attenuated the relationship between central obesity and mortality, the resulting relationships remained significant. The authors concluded that combining the effect of BMI and a central adiposity measure may provide a better discriminator with respect to mortality than either type of measure separately.

These studies suggest that BMI, although the most practical measure, may not always be the best one when relating obesity to mortality. In these studies, any adverse mortality experience at larger BMI, WC, or WtoH ratio values was generally found primarily related to cardiovascular disease and some cancer related causes. WC, for example, is certainly more highly related to abdominal adiposity than BMI. Although WC measurement involves only one factor, it also may be difficult to determine in a consistent manner. The WtoH ratio measures glutofenerol muscle, bone, and fat mass. Both of these alternatives have greater possibility of measurement error than BMI. The importance of their relationships with mortality has differed by study, although WC seems to have a somewhat better conceptual basis.

Nevertheless, even if BMI were a good measure of exposure to mortality risk, a single measure cannot provide a complete indication of the quality of a person's health. Consideration has to be given to a range of factors, such as body fat, medical condition, metabolic pattern, fitness, and cardiovascular risk factors, as well as BMI (or one of the central adiposity measures).

2.1.2 The benchmark

Studies of the relationship between mortality and obesity often use mortality experience of individuals in the normal BMI category as a benchmark from which mortality experience of other BMI categories is compared. The normal BMI category (sometimes referred to as the mortality nadir or ideal mortality point or range) has been defined somewhat differently among the studies, consisting in some studies of all participants with a BMI of less than 25, although other studies have included in their benchmark BMI category those in an 18.5-24.9, 20.0-24.9, or 23.5-24.9 range instead. Many if not most studies that include a separate underweight category have found excess mortality in the underweight category (e.g., less than the low point in the normal category range).

Since the relative size of the standard underweight class (a BMI of less than 18.5) in the adult U.S. population is currently between 1 and 2 percent (which has been shrinking), the experience for this population segment usually has limited effect on the benchmark used if their experience is included in the benchmark. For example, if a study's "normal" benchmark population includes everyone with a BMI under 25 and, say, 1.5 percent of the total population is underweight, say with a BMI under 18.5 with it a hazard ratio of 1.50, assigning those under a 25.0 BMI in the benchmark would result in a benchmark hazard ratio of 0.974 for the non-underweight normal BMI category.

Similarly, if the hazard ratio for an underweight category of those with BMIs of less than 23.5 consists of 50 percent of those with a BMI less than 25 was 1.30 compared with the total BMI category of less than 25.0, then a revised benchmark for the subgroup of BMIs (between 23.5 and 24.9) would be about 0.87 (derived from 1 / (1 + .30 x 50%)) rather than 1.00. The use of such a narrow normal BMI range suggests that the relative mortality experience of those overweight and obese might not represent a significant paradox after all, or at least it might be a reduced paradox, as otherwise indicated if the mortality for underweights included in the normal category is much higher than those in a normal category. In a real example, using the percentage distribution of BMI subpopulations (i.e., 18.5-20.9, 21.0-23.4, 23.5-24.9) and corresponding hazard ratios shown in Adams et al (2006), the hazard ratio of the narrow BMI range of 23.5-24.9 would be about ten percent lower than the mortality of the wider BMI-normal category. Thus, if these proportions and ratios are applicable in other studies, the narrower ideal mortality category might have a hazard ratio of about 90 percent of the broader category—in this case if the overweight hazard ratio

was above 90 percent, there would be no paradox in this case. This effect depends on the percent and degree of adverse mortality of the underweight, as well as the extent of the original paradox.

Flegal et al (2013) indicated that such a bias would have little effect – about a 0.005 hazard ratio for overweight (about 10 percent of the studies used in Flegal used a normal benchmark range covering all participants less than 25 and about three-quarters used a benchmark range of 18.5-24.9). Nevertheless, the effect of the choice of benchmark can differ significantly, depending on the current percent in the range with greater relative mortality from nil to something greater than 10 percent, on the distribution of study participant between BMIs up until 25.0, and the level of greater mortality at BMIs less than, say, 23.5.

Due to the shift of the BMI distribution to the right, the results for a combined obesity category can also be misleading. Flegal's study indicates that, although there is a significant amount of excess mortality for the entire obese category, when the disaggregated, the class 1 obese has experience at a level comparable to the normal BMI category, while the larger excess mortality at classes 2+ makes the entire obese category appear somewhat adverse. Since the relationships involved are continuous in nature, any conclusion based on results of the entire obese category will not apply to its subcategories. Thus, it is important to determine whether results apply equally to the entire BMI range being reviewed or differ within that range.

2.1.3 Reverse causation

Many mortality studies are influenced by associated or pre-existing risk factors, such as an existing smoking habit, illness, and recent weight loss indicative of existing (known or unknown) illness, any of which can contribute to biased relationships. Although most studies that have adjusted for this bias have found relatively small if any effects, inclusion of these factors might attenuate patterns. This effect is referred to as reverse causation. For example, being ill can involuntarily result in weight loss. Many studies have attempted to mitigate this effect by excluding those who (1) are current or previous smokers, (2) have current reported illnesses, (3) died during the first 1 to 5 years after initiation of the study, or (4) experienced a recent weight loss. Another approach is to focus on younger subjects who have fewer pre-existing conditions. This effect has been seen to be particularly significant at low BMI levels.

In a study of linked mortality from the U.S. NHIS with an average follow-up period of 16 years, Ma et al (2011) found that, including as participants only those who never smoked resulted in a hazard ratio of 1.07 for the overweight, 1.41 for the obese, and 2.46 for the extremely obese compared with those of normal weight in a young cohort (ages 18 through 39 at initial measurement). Corresponding hazard ratios for middle-aged or older populations were much smaller for those overweight or obese when not restricted to non-smokers. Hazard ratios were greater for cardiovascular disease (about two for those overweight, four for the obese, and eight for the extremely obese). Ma also found excess mortality due to cancer (including breast cancer), particularly in the extremely obese young adults who had never smoked.

Another example is given by Lawlor et al (2006) who aggregated the results of two Scottish studies with a 28 to 34 year follow-up period. Initially when smoking histories and other existing illnesses were not considered, Lawlor did not find an association between mortality and being overweight, and a weak to modest association between mortality and being obese. In contrast, when those included were only non-smokers and when the experience of the first five years of the study were not included, there was a significant increase in the association of BMI and all-cause mortality (hazard ratios between 1.12 and 1.38 for males and females, respectively) for those overweight at initial measurement and hazard ratios of about 2.00 and 1.56 for male and female obese, respectively. Relative to cardiovascular disease risk, both those who never smoked and current smokers, as well as those overweight and obese, were associated with significant increases. This illustrates both the effect of reverse causation and the potentially misleading effect of a short follow-up period.

Gelber et al (2007) studied the effect of eliminating contributors to reverse causation in the Male Physicians' Health Study covering about 100,000 physicians over a maximum of a 6.6 year period. Adjustments were made for age,

smoking, alcohol consumption, physical activity, pre-existing disease, and interactions between BMI and smoking. Current and prior smokers were analyzed separately and did not include experience over the two years after recruitment into the study. The result was a linear relationship between BMI and mortality using a BMI benchmark of 22.5-24.9, with hazard ratios of 0.88 for those with BMI of less than 20.0, 0.99 for those between 20.0 and 22.4, 1.20 for those overweight, 1.45 for class 1 obese, and 1.62 for class 2+ obese.

The adverse mortality effect of smoking can overwhelm the corresponding effect of obesity, so in some studies the relative effect of obesity can be masked, as the effect of obesity for smokers is less significant than the effect for non-smokers (this can be seen in Figure 6 by comparing its two curves). Flegal et al (2013) indicated they selected only those studies in which one or more techniques were applied to eliminate the effect of reverse causation.

2.1.4 Weight change

Not only can weight measured at a point in time be associated with future mortality, but changes in weight can be as well. If a lower weight is usually better for morbidity and in many cases for mortality, it would be reasonable to assume that weight loss (not caused by a specific adverse health condition) would correspondingly be accompanied by lower mortality risk. Nevertheless, especially but not exclusively at older ages, involuntary or non-purposeful weight loss may be the result of a respiratory or other illness (whether known or unknown) or even malnutrition. Some have expressed the view that unintentional weight loss, through the catabolic effects of heart failure, is the primary reason that low body weight is linked to poor health prognosis, as several studies have found that overall weight loss may be associated with a modest to strong increase in morbidity and mortality risk. In contrast, moderate weight gain has been shown in some studies to be protective against premature mortality. Some of these findings follow.

Janssen and Bacon (2008) reported results from the original Framingham Heart Study cohort (whose BMI were measured in their 50s and who lived to at least age 70). Compared with the rates of mortality of those who were non-obese at both ages 50 and 70, those who were obese at age 50 and non-obese at age 70 had a 55 percent greater risk of mortality, while those obese at age 70 had a 21 percent greater risk of mortality and those who were obese at both ages 50 and 70 had a greater mortality risk of 47 percent. Those who were obese at age 50 had a 56 percent greater risk of mortality compared to individuals who were non-obese at both midlife and old age. The weight loss of those who were in the obese group at age 50 was for the most part unintentional in nature, thus likely reflecting subclinical or clinical disease, which in turn increased the mortality in this group. In sum, in this long-term study, midlife (age 50) and current (at least age 70) BMI had independent effects on mortality risk. Although mortality risks were greater in older obese adults who were also obese at midlife, this was not the case for newly obese older adults. Conversely, non-obese older adults who were obese at midlife had an increased mortality risk. Based on this study, it is important to consider older adults' personal BMI history—not only may the increase in BMI-obesity over the last several decades have adverse effects when those currently obese in middle age reach older ages, whether they maintain that additional weight or lose it unintentionally.

Zheng et al (2013), based on findings of the U.S. Health and Retirement Survey, studied movements between BMI categories over time. Zheng indicated that trajectories of increasing obesity past age 51 result in excess deaths, with the effect generally differing by the magnitude of weight change. Weight loss, even a small weight loss among those in the normal BMI category at age 51, was also associated with significant additional mortality risk. Although other studies have produced mixed findings in this regard, a dynamic measure of weight status (incorporating weight change) may prove more predictive of mortality than a static measure.

Sun et al (2009) indicated that in the Nurses' Health Study, adiposity in mid-life was strongly related to a reduced probability of healthy survival (defined as having no history of eleven major chronic diseases at about age 50 and no substantial cognitive, physical or mental limitations at age 70) among female nurses who live to older ages. This was true not only for BMI, but also for measures of central adiposity (WC, hip circumference and WtoH ratio). Its findings emphasize the importance of maintaining a healthy weight from early adulthood. They also found that the more weight gained from age 18 until mid-life, the less likely that there would be healthy survival until at least age

70, with the lowest odds of healthy survival being among females who were BMI-overweight or obese and who gained more than 10 kg (a hazard ratio of 0.18 compared to females with a BMI between 18.5 and 22.9 and stable weight). Not only was every unit increase in BMI associated with a 12 percent reduction of the odds of healthy survival, but for every 1 Kg increase in weight since age 18 the odds of healthy survival decreased by 5%. Sun noted that although there were relatively few who lost weight between age 18 and mid-life, there was a small, non-significant increase in their odds of healthy survival.

The findings of several studies that assessed mortality based on body fat and lean mass rather than BMI have suggested that those who lose body fat rather than lean mass experience a lower mortality rate, while other studies have shown the opposite. Nevertheless, there are also possible adverse effects of weight loss as well. For example, Lavie et al (2009) noted that obese coronary heart disease patients experienced marked reductions in C-reactive protein levels while those of normal weight had small gains.

In addition, studies, such as a Mayo Clinic study reported on by Eilar-Adar (2005), have found that voluntary weight loss has often provided beneficial health results, both as to mortality and major cardiovascular events for each BMI category. Several meta-analyses have indicated that weight loss can, especially when accompanied by physical activity, provide beneficial health effects, especially related to cardiovascular risk factors. For example, one study indicated that for every 1 kg of weight lost, blood pressure dropped by 1.1mHg systolic and 0.9mmHg diastolic.

As physicians have obtained more experience with (bariatric) surgery, some patients have lost at least 50 percent of their excess weight (over normal BMI levels) and have experienced improvements in their cardiovascular risk factors, as well as better short and long-term mortality when compared with alternative treatments. However, at the same time, it has been observed that complications and re-admissions may occur.

It is common that attempts to quit smoking are accompanied by weight gain, up to 5kg for longer than four years after smoking cessation, and which often stays with the individual. Various studies have indicated that, although this gain can reduce the favorable effect of smoking cessation, the long-term effect of smoking cessation usually outweighs the effect of this weight gain. An example reported on by Claim et al (2013) is a Framingham Offspring Study with a 25 year follow-up period that addressed the interrelation between this weight gain and mortality experience reported by Clair et al (2013) on. The cardiovascular disease events (cardiovascular disease, cerebrovascular events, peripheral arterial disease, and congestive heart failure) recorded in this study were all more affected by smoking cessation than weight gain.

2.1.5 Heterogeneity of the normal BMI category

Those in the normal BMI category (often defined as covering those with a BMI of 18.5-24.9) can in turn be meaningfully segmented in several ways, for example, by degree of body fatness and visceral adiposity. Its accuracy in predicting obesity appears particularly limited in the intermediate ranges (as well as for males and in the elderly). This may result in greater relative mortality for those in the normal BMI category than if the normal population segment had been measured in terms of body fat or central adiposity.

Romero-Corral et al (2010), using data from NHANES III with a median follow-up period of 14.3 years, found that when segmenting those of normal BMI into tertiles of body fat (greater than 23.1 percent in males and 33.3 percent in females), the prevalence of metabolic syndrome was four times greater in the lowest body fat tertile than in the highest. There was also a higher prevalence of dyslipidaemia, hypertension in males, and cardiovascular disease in females. After adjustment, females in the highest tertile of body fat in the normal BMI category had 220 percent of the risk for cardiovascular mortality (there was no significant effect on cardiovascular mortality for males). Romero-Corral also conducted a similar analysis with WC as an obesity measure – although a larger WC was associated with cardiovascular disease risk, in normal BMI subjects WC was not related to greater cardiovascular mortality. Thus, at least for females, this study indicates that the obesity paradox might not exist if mortality experience of those with both normal BMI and normal body fat are used as a benchmark, rather than the total normal BMI category.

Sahakyan et al (2013), who also wrote about findings from NHANES III, focused on the effect of central adiposity of those with normal BMI. They found that only 47.8 percent of those with normal BMI were of normal WtoH ratio (less than 0.85 for females and less than 0.90 for males). The all-cause mortality hazard ratio was 2.08 for those obese according to WtoH ratio but normal BMI, using those in the normal WtoH ratio and normal BMI as a benchmark; the corresponding cardiovascular mortality hazard ratio was 2.75. This analysis was adjusted for age, gender, race, smoking, hypertension, diabetes, and dyslipidmia, with those suffering from chronic obstructive pulmonary disease and cancer excluded. In this case, no obesity paradigm was evident if central adiposity was used as the obesity measure. Sahakyan also found the same pattern both for those at least age 65 and those younger than age 65, although in most cases the hazard ratios were somewhat reduced.

In a cross-sectional study of 4,828 white subjects, Gómez-Ambrosi et al (2011) found a greater than expected number of normal BMI or normal WC subjects with pre-diabetes or type 2 diabetes who are considered obese according to the amount of their body fat as determined by air-displacement plethysmography when compared to those who were correspondingly overweight or obese BMI or overweight or obese WC. A higher body fat percentage was found in normal BMI females with pre-diabetes or type 2 diabetes than those with normoglycemia, and increased body fat percentage was found in normal BMI males with pre-diabetes or type 2 diabetes, with no corresponding differences in BMI or WC. They also found that in the BMI-obese, WC can be a significant factor, while in the BMI-normal, body fat may be a better classifier than WC. They speculated that WC may not be as good a surrogate of visceral fat in those individuals whose WC is more highly correlated to subcutaneous than to visceral adipose tissue, and that in normal-BMI individuals the amount of adipose tissue is a better indicator, especially in males, of increased obesity-associated risk than are indicators of adiposity distribution such as WC. Equivalent increases in body fat percentage had a smaller impact on insulin sensitivity in females than in males, possibly explaining significant differences in WC between groups observed in normal BMI-classified females, for whom a higher level of central obesity is needed to develop pre-diabetes.

Another differentiator in the normal-BMI category is the level of physical activity and fitness. Using NHANES III, Sahakyan et al (2012) found that those who were inactive and insufficiently active (measured in terms of METS/hour/month) had greater hazard ratios (compared with those who were active) of 1.47 and 1.60, respectively. Also for those in the normal BMI category, while strength training was independently associated with mortality with a hazard ratio of 0.63, alternative dietary patterns were not predictive for those in the normal BMI category.

2.1.6 Heterogeneity of individuals within any category

Each BMI category consists of a wide variety of individuals with different histories of medical conditions and treatment, including those with a range of past nutritional inputs, fitness, disability, and disease. In other words, any such category is not homogeneous with respect to mortality risk. It might be possible that body weight by itself is simply not a significant risk factor by itself for mortality, being a surrogate for other aspects of other lifestyle and genetic factors.

This suggests that the use of one body metric may be too simplistic to use as an effective indicator of mortality risk; rather, a combination of metrics may be better for this purpose, including blood pressure, cholesterol, and blood sugar. Greenberg et al (2007) referred to this as regression-dilution, in that people with higher body weight in a baseline survey tend to have lower weight before and after the survey and the opposite for persons with low indicated weight, thus possibly artificially decreasing the hazard ratios at high levels of baseline BMI. The Atherosclerosis Risk in Communities Study (ARIC) attempted to overcome this human reporting problem by averaging BMI at three consecutive visits to participants, which increased the resulting hazard ratio of obesity from 1.26 to 1.46.

As the normal BMI category can be split into segments to provide insight into the obesity paradox, so too can the BMI-obese category. For example, Neeland et al (2012) suggests that the healthier obese individuals have decreased fat deposits in their abdominal viscera, increased lower body subcutaneous fat storage, insulin

sensitivity, and adiponectin, with larger HDL and LDL particles. Distributions of BMI, total body fat, and abdominal and subcutaneous fat mass did not differ between obese categories. Thus, resistance to diabetes may be explained by the movement of excess fat from visceral and other ectopic sites to the lower body. Neeland concluded that excess visceral fat and insulin resistance, but not general adiposity, were independently associated with the incidence of pre-diabetes and type 2 diabetes in obese adults. In any case, looking at the trend between NHANES surveys as reported on by Mehta and Chang (2011) and Greenberg (2013b), a significant part of the obesity paradox may have been due to relative improvement over the last few decades of the mortality of the BMI-obese (according to Greenberg this trend has occurred in males but not females—the passage of time will show what types of individuals have benefited more.

A sizable percentage of those in the obese category have a normal cardiometabolic risk profile. When being healthy is defined as having none of six common risk factors (blood pressure, triglycerides, insulin resistance, diabetes, low HDL and high C-reactive protein), Wildman et al (2008) found that 35 percent of obese females and 29 percent of obese males should be considered healthy. However, BMI does influence the proportion in a high fitness condition–according to submaximal exercise testing in a secondary analysis of 1999-2002 NHANES, 30 percent of American adults in the BMI-normal category, 17 percent of those overweight and 9 percent of obese individuals were in the high fitness category.

The body's cumulative exposure to excess weight may be more important than current weight. The relatively recent current surge in the obese population comes from those previously overweight as they gained weight over time, while these overweight in turn are replaced by those who were in the normal BMI category. This large weight movement between BMI categories may have at the same time improved the average health in these higher BMI categories, as those previously not in weight-jeopardy would have previously been in a lower BMI category. It is particularly difficult to project mortality for the high percentage of those who have recently become obese, as adipose tissue has a cumulative effect.

2.1.7 Physical activity and fitness

Physical activity has sometimes been played second fiddle to obesity and nutrition in the assessment of health and mortality, in part because it is more difficult to measure in a consistent manner and diverse findings with regard to its independent effect. However, it should not be ignored. The important characteristics of physical fitness include lean mass, muscular fitness, and cardiorespiratory fitness. Based on a review of the literature, Swift et al (2013) concluded that higher levels of physical activity, participating in exercise training and higher overall cardiorespiratory fitness can provide considerable protection in the primary and secondary prevention of coronary heart disease, regardless of BMI or adiposity.

Lavie et al (2013) indicated that substantial evidence suggests that low cardiorespiratory fitness is, perhaps, the strongest cardiovascular risk factor. They also indicate that several studies have suggested that cardiorespiratory fitness markedly attenuates the relationship between adiposity and subsequent prognosis in patients with both coronary heart disease and heart failure. In a study of 2,000 such patients with higher levels of cardiorespiratory fitness, there was no evidence of an obesity paradox.

Even modest physical activity can protect against a range of conditions, from osteoporosis and diabetes to cardiovascular conditions. It not only improves the efficiency of the heart, but it also boosts the release of neurotransmitters and may stimulate a person's cell disposal structure that can alter how genes work in adiposity tissues. It can further stimulate epigenetic changes in muscle cells that can alter how muscles process sugar, reduce heart rates, lower blood pressure, and decrease cholesterol levels. Boström et al (2012) found that exercise has the capacity to turn on a phenol-type similar to that of brown fat that may be of clinical significance for metabolism, which may be protective against elements of the metabolic syndrome. Exercise and physical activity also stimulates the release of multiple myokines, manages the interplay of secreted and non-secreted proteins and has direct and indirect benefits on organs such as the brain and on the cardiovascular system.

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Adipose tissues are not just a passive store of energy, but can also be viewed as an organ in their own right, as they can serve as inflammatory agents and produce a range of biologically active chemicals that have many effects on the rest of the body. Eighteen genes are known to be associated with obesity and twenty one have been identified as being linked with adult-onset diabetes. Physical activity can contribute to making two of these genes more efficient at processing and allocating fat to appropriate parts of the body. Thus one reason exercise may be beneficial is that it improves the ability of fatty tissue to do its job more effectively. In addition, Li et al (2010) found in the European Prospective Investigation of Cancer – EPIC cohort with a follow-up period of 3.6 years, that a physically active lifestyle is associated with a 40 percent reduction in the genetic pre-disposition to obesity, as estimated by the number of risk alleles carried for any of the twelve recently Genome Wide Association Studies. Lipids also can become stored in a more appropriate place instead of settling elsewhere in the body where they can do harm. Since many chronic illnesses are metabolically demanding, this can serve as a metabolic reserve to deal with bodily needs as they arise.

In a study of the Mayo Clinic cardiac rehabilitation program (Goel et al (2011)) with a mean follow-up period of 9.7 years, fitness of study participants was categorized as being of low and high level. After adjusting for potential confounding factors, low fitness and central obesity (measured by a WtoH ratio) were independently associated with increased mortality. For example, a hazard ratio of 6.1 was observed for those in the low fitness / high WtoH ratio category, 4.2 for those of low fitness / low WtoH ratio, 9.6 for low fitness / normal weight, 6.8 for overweight / low fitness, 3.3 for high fitness / obese, and 3.2 high fitness / overweight (to a benchmark of normal weight / high level of fitness).

In a meta-analysis of 33 studies covering all-cause mortality, coronary heart disease, and cardiovascular heart disease (CHD/CVD), Kodama et al (2009) calculated an all-cause mortality hazard ratio of those with low cardiovascular fitness of 1.70 and a CHD/CVD hazard ratio of 1.56, based on a benchmark of mortality of those with high cardiorespiratory fitness. Compared with participants with intermediate cardiorespiratory fitness, those with low cardiorespiratory fitness had an all-cause mortality hazard ratio of 1.40 and for CHD/CVD events of 1.47. Thus, in the aggregate these studies found that better cardiorespiratory fitness was associated with a lower risk of all-cause mortality and CHD/CVD.

Crespo et al (2002) reported that findings from the Puerto Rico Heart Health program showed that independent of weight, being physically active was a significant factor in mortality, although the level of activity was not found to be significant. This is one more indication that it is more important that a person is physically active than the amount of the physical activity.

While sedentary behavior has been associated with adverse health outcomes in children for many years, Raynor et al (2012) confirmed that this association is independent of the time engaged in physical activity, as well as being associated with all-cause mortality, type 2 diabetes, cardiovascular disease, and weight gain, as well as obesity. Prolonged sitting and lack of contraction of lower limb muscles led to metabolic abnormalities via suppressed action of muscle lipoprotein lipase and insulin. In addition, this study confirmed that physical activity can moderate the desire for food, which can trigger the release of dopamine and endorphins that can supply a substitute reward different from eating good-tasting food. Although Ekelund et al (2012) found in a 14 study meta-analysis of the 20,871 children aged 4-18 from the International Children's Accelerometry Database that the mean time spent in moderate-to-vigorous-physical-activity (MVPA) was 30 minutes a day, while on average 354 minutes were spent being sedentary. With a 2.1 year follow-up period, neither MVPA nor time spent being sedentary were associated with WC at follow-up, although a large WC at baseline measurement was associated with higher amounts of sedentary time at follow-up. More MVPA time by children and adolescents was associated with better cardiometabolic risk factor values, regardless of the amount of sedentary time.

Belloco et al (2010) found in a Swedish study that obese sedentary males had a 98 percent increased risk of mortality compared to that of males with a normal BMI, WtoH, and WC with a high level of physical activity. Sedentary females with a WC of 88 cm or more had a hazard ratio of 1.97 compared to that of females with a WC of less than 80 cm. Based on the large (185,412 participants ages 51-72) National Institutes of Health-AARP Diet and

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Health Study, Koster et al (2009) indicated that each of being BMI-overweight, being BMI-obese, having a large WC, and experiencing low physical activity was an independent predictor of greater mortality. Compared with a benchmark of mortality of those who were normal weight and physically active (greater than 7 hours/week of moderate physical activity), those who were inactive and of normal weight had a hazard ratio of 1.62, those who were active class 2+ obese had a hazard ratio of 1.79, and those who were inactive class 2+ obese had a hazard ratio of 3.45. Similar results were observed for combinations of BMI and vigorous physical activity—those who were inactive with a large WC had a twice as large mortality risk and high physical activity, which attenuated but did not eliminate the increased mortality risk associated with obesity.

Findings of the Aerobics Center Longitudinal study (Sui et al (2007)) with a mean follow-up period of twelve years included that among those older than age 60, WC (superior to BMI by itself) was related to mortality after adjustment for BMI, but not after additional adjustment for fitness, which was associated with mortality risk even after further adjustment for smoking, base health, and either BMI, WC, or percent body fat. Sui concluded that fitness has a strong inverse association with mortality. Also, it was found that fitness expressed as skeletal muscle function (in terms of strength, power, and endurance) may contribute to improved physical functioning and longevity through biological pathways that are related to but independent of aerobic fitness.

In a subsequent paper covering findings of the Aerobics Center Longitudinal study with a mean 13.4 years of follow-up of almost 10,000 males with known or suspected coronary heart disease, McAuley et al (2012) indicated that males with low fitness (measured by a maximal treadmill exercise test) had greater all-cause mortality risks in the BMI-normal category with a hazard ratio of 1.60 compared with males in the normal BMI and high fitness benchmark group, while obese class 1 individuals had a hazard ratio of 1.38, obese class 2 and 3 had a hazard ratio of 2.43, and those in the BMI-overweight category had a hazard ratio of 1.09. A similar pattern was observed with measured WC and body fat. Among highly fit males there were no significant differences in cardiovascular disease and all-cause mortality across BMI, WC, and body fat categories. Only those severely obese (class 2+) and those younger than age 55 had a greater relative cardiovascular disease mortality risk—when data was stratified by age, greater cardiovascular disease mortality was only observed in males younger than 55. In sum, the findings indicated that standard measures of adiposity do not exert significant influence on survival in cardiovascular patients; rather, cardiorespiratory fitness was more closely related to mortality risk in populations exhibiting an obesity paradox.

Findings of a Cooper Institute study (Farrell 2010) involving females were similar. Based on a study of 11,335 females, low cardiorespiratory fitness was a significant independent predictor of all-cause mortality, while higher cardiorespiratory fitness was associated with lower mortality within each adiposity (BMI, WC, WtoH ratio and percent body fat) related category. Hazard ratios for all-cause mortality were 1.0, 0.60, and 0.54 for low, moderate, and high fit groups.

In a cohort of American male veterans, McAuley et al (2009) similarly indicated that exercise capacity and BMI were independently related to mortality risk. A larger BMI was associated with reduced mortality risk across fitness categories. When those who were highly fit were compared by BMI category, those who were overweight or obese experienced significant reductions in mortality risk (hazard ratios of 0.43 and 0.52, respectively). In a series of studies of American male veterans, those who were fit but overweight or obese experienced lower mortality than those of normal weight who were unfit.

Fasells et al (2012) reported on the interaction between fitness, fatness and mortality risk in hypertensive individuals (hazard ratio of 0.40 and 0.22 for BMI-overweight and obese, respectively) and Kokkinos et al (2012) reported on this relationship for those with diabetes (0.59 for obese), with an even lower hazard ratio for black males; fitness was a more significant differentiator in those overweight and obese in comparison with fitness of those of normal BMI.

Based on a 36 study meta-analysis, Fogelholm (2010) indicated that the risk for all-cause and cardiovascular mortality was lower in the class 1 BMI-obese and good aerobic fitness category compared with the risk for those with normal BMI and poor fitness. In contrast, those in the class 1 BMI-obese category, even with high physical

activity, were subject to a greater risk for diabetes incidence and prevalence for diabetes and cardiovascular risk factors than those of the normal BMI and low physical activity category.

Thus, some studies have indicated that obesity is not a risk factor for mortality in fit individuals, as mortality does not vary by BMI level. However, for those who are not highly fit, BMI does appear to have a strong relationship to mortality sometimes referred to as the "fat but not fit" paradox. Thus, as the percent of those who are fit in a BMI category changes, the overall mortality experience in that category will be affected.

Physical activity's lagged effects are similar to those of obesity – its future and cumulative health benefits are greater than its current effect, in some cases taking years to emerge, suggesting that continued effort is needed for it to provide a preventive effect on cardiovascular conditions, heart disease, and their risk factors. When assessing the impact of physical activity, not only should leisure exercise be considered, but also work-related activity and the level of sedentary time. In general, higher (but not extreme) levels of these activities have a greater effect than moderate levels.

Colman and Dave (2013) estimated, based on a study of NHANES interviewed between 1971 and 1974 with a follow-up spanning 1982-1984 that declines in high levels of recreational exercise and other physical activity can account for between 12 and 30 percent of the recent increase in obesity, hypertension, diabetes, and heart disease. They found that work activity can have a more significant effect on these health risk factors than the amount of leisure exercise, with reduction in work activity being a contributor to the increase in obesity. Colman and Dave also found that reductions in physical activity could explain about 3 to 10 percent of the increase in BMI and hypertension and consequently about 2 to 8 percent of the increase in diabetes and heart disease during the period studied.

Findings indicated in Hu et al (2005)'s study of about 47,000 middle-aged Finns that the physically active experienced lower age-adjusted mortality from cardiovascular, cancer, and all-cause mortality compared with those who were more sedentary. They concluded that regular physical activity and being of normal weight are both important indicators of decreased risk of mortality from all causes, cardiovascular disease, and cancer. Physical activity had a strong independent effect on mortality, in contrast to the effect of BMI, which was partly mediated through other obesity related risk factors.

No matter what level of obesity a person has, evidence indicates that the level of cardiovascular fitness over time is important for mortality and morbidity. It is clear that efforts to use more standardized metrics regarding levels of physical activity, fitness, and time spent in a sedentary condition would be useful to facilitate assessment of comparable results.

2.1.8 Treatment

Those who are overweight and obese have recently tended to seek medical help earlier and possibly to manage their health better than those in the normal BMI category. As a result of weight issues they or their physicians may be more sensitive to the treatment of other medical conditions, such as diabetes or cardiovascular problems, at an earlier point in their development. As a result, earlier and more aggressive treatment of those conditions once identified (e.g., being screened more often for chronic diseases stemming from their excess weight) may occur. Physicians have been trained to counsel patients who are overweight or obese to strongly consider strategies to control their weight and other health risks, while they may not have the same type of opportunity to counsel those with a normal BMI.

As an example, excess body weight may cause dyspnea for reasons unrelated to heart failure that then may lead to peripheral edema due to cardiovascular reasons, thus leading to a request for treatment at an earlier stage, increasing the chance of successful treatment and as a result, a person at a higher BMI level may have on average a less severe medical condition (even though there may be more of them) than would a person of normal BMI.

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Using data from the Medicare Beneficiary Survey (1994-2006) and recipients of care from the Veterans Health Administration, Chang et al (2010) reported that being obese or overweight was associated with a marginally higher rate of recommended care on several measures, including those based on lipid screening and HbA_{10} testing, care for diabetes, higher odds of vaccination and mammographies, and CRC and cervical cancer screening. In addition, As evidence, the use of lipid-lowering and antihypertensive medications has increased rapidly over the past two decades with, according to Gregg et al (2005), the largest increases being among the obese. These examples suggest that those overweight and obese, possibly with a higher sensitivity (of patient and medical practitioner) to medical treatment, may be diagnosed for a medical condition earlier, resulting in more favorable outcomes.

Steinberg et al (2007) suggested that in cases in which invasive treatments for coronary artery disease occur, lower hazard ratios for increasing BMI appear associated with better use of guideline-recommended medical treatment and invasive management (increased use of standard medical therapies and a greater likelihood of undergoing invasive procedures in the first place) of coronary artery disease. Others have speculated that obese patients who are subject to a study's medical condition (e.g., a cardiovascular condition) may on average be in better health than their non-obese counterparts in the studies.

Schenkeveld et al (2012), based on a 6.1 year follow-up period of patients treated with percutaneous coronary intervention, found that mortality was inversely related to BMI (hazard ratios of 0.75 and 0.72 for overweight and obese, respectively). However, after adjusting for suboptimal treatments (defined as two or fewer of the four types of medication known to improve prognosis in patients with coronary artery disease – aspirin, beta blockers, statins, and ACE inhibitors/angiotensin II receptor blockers) that patients in the normal BMI category may also have been subject to, mortality results did not indicate a reverse relation between mortality and obesity (hazard ratios of 0.90 and 1.07 for overweight and obesity, respectively). The authors suggested, as a result, that more aggressive lifestyle modification and optimization of medical treatment (including earlier and more complete action) may be a plausible reason for the enhanced survival in individuals who are obese at the time of coronary revascularization; therefore the normal and obese populations in such studies may not be comparable, i.e., all other things may not be equal.

Improvements in cardiovascular risk factors of the obese (more so than those of normal weight) have contributed to a decrease in relative mortality of the obese over the last few decades, resulting in somewhat lower or even an inverse association between BMI and mortality in relatively recent studies.

2.1.9 Age

Many studies have included older age individuals in an all-age adult study, or at least included data with respect to those at older attained ages because initial measurement was made at younger ages. Their inclusion may bias the aggregate results of these studies, as it has been observed that mortality patterns at middle ages are not consistent with those at older ages (see "Older ages" below), that is for the latter, mortality of those in the normal BMI category is often worse than mortality at higher BMI levels (especially but not exclusively in the overweight BMI category) at their current or a recent age.

These relativities among BMI categories may arise due to changes in body composition as people age, including weight loss due to health conditions, problems, or disease. With increasing age there is a loss of muscle mass, especially in females, leading to sarcopenic obesity (an increase in fat mass and a reduction in lean mass), which can exacerbate BMI misclassification, particularly important because most deaths occur at these ages. Flegal et al (2013) indicated that for those studies included in their study restricted to age 65 and younger hazard ratios for those who were overweight were 0.98 (rather than 0.94 for the total study) and 1.31 for all obese (compared with 1.19 for all ages). It can therefore be seen that if only studies of those who are middle age were included, the obesity paradox would be reduced. In addition to the age effect, differences exist among other population sub-categories (e.g., Asian and Asian-Americans tend to have a lower BMI and somewhat higher central adiposity than do American whites).

As the cumulative period while obese can affect mortality results, adverse mortality experience for the additions to adult- or older age-onset obesity (with its shorter accumulation period) over the last few decades may not yet have had an opportunity to fully emerge. For example, as indicated in Janssen and Bacon (2008), Framingham Heart Study participants who were both BMI-obese at age 50 and 70 had adverse mortality subsequent to age 70 compared with those participants who were BMI-normal at age 50 and obese at age 70. In addition, those who were obese at age 50 but normal BMI at age 70 also experienced increased mortality subsequent to age 70. This suggests that obesity at mid-life tends to lead to greater mortality in later life no matter what BMI becomes at older ages.

Hazard ratios have been found to differ by age in many studies, including Greenberg (2013a), who used data from NHANES I, II, and III with a follow-up period of 15 years relative to a benchmark BMI of 23.5-24.9. He reported hazard ratios of 0.92 for healthy overweight non-smokers for all ages, with hazard ratios of 1.33, 0.84, 0.92, and 0.63 for those of age less than age 55, 55-64, 65-75, and over 75, respectively. Corresponding healthy obese non-smoker hazard ratios were 1.84, 1.91, 1.52, and 0.89, respectively. For this study, the hazard ratio for all those overweight was 0.98 and for all those obese on an aggregate basis was 1.36, which in an attempt to reduce the effect of reverse causation, were studied simultaneously for smoking, pre-existing illness, and age.

Unlike at younger adult ages, some have suggested that a BMI somewhat greater than 25 may be a more appropriate upper cut-off point for a normal category for older adults. Others claim, as indicated above, that BMI is simply not a useful metric for older adults.

As some view with alarm, the increase in child/adolescent obesity onset may represent a greater long-term societal health issue.

2.1.10   Protective value

Body fat in some individuals seems to have a cardio-protective metabolic effect, constituting a higher metabolic or energy reserve that can, for example, result in lower systemic vascular pressure. This has been confirmed through evidence that those in a higher BMI level category experience can survive a medical emergency such as an infection or surgery (for example, if suffering from pneumonia, it may be useful to have more fat to lose). Earlier recognition of conditions referred to in "Treatment" above may result in better overall mortality results, although living longer in an unhealthy state can also lead to greater consequential health care costs and increased rates of disability. Additional fat might also neutralize the adverse effects of tumor necrosis factor-alpha, lower levels of circulating atrial natriuretic peptides, and attenuate the sympathetic nervous system and rennin-angiotensin responses.

Due to greater arterial pressure, those with a higher BMI may be able to tolerate higher levels of cardioprotective mediations (Lavie et al (2009)). It is not known yet whether this factor or a greater coronary artery size is a contributing cause of the obesity paradox. In any case, it may provide protective value once an adverse cardiovascular condition has arisen, reducing the ultimate mortality risk from such a condition (while at the same time treatment for such a condition and needed resulting care can increase health care and associated costs both currently and in future years).

As food is swallowed, it is broken down into fat, sugar, and protein. Most of the fat enters the bloodstream and moves to the liver, where it is converted into other substances such as cholesterol, with the remainder stored in fat cells. Schenkeveld et al (2012) suggested that peripheral adiposity confers cardiovascular benefits due to the secretion of adiponectin, a hormone that enhances the burning of both fat and sugar and has inflammatory, insulin-sensitizing, and anti-atherogenic effects. Overloaded fat tissues also generate fatty acids that enter organs where they don't ordinarily reside, especially in the liver. Inflammation and unhealthy fat storage may keep cells from responding properly to hormones, causing insulin-resistance. This in turn prompts the pancreas to generate high levels of insulin, which in turn leads the body to store excess sugar not used for energy as fat cells and tissues. Eventually blood sugar levels get out of control and result in diabetes, and as the body becomes obese, adiponectin

declines, hampering this additional check on weight gain. This then is likely to be followed by certain cancers, possibly because excess insulin supplies glucose to cancer cells, helping them to replicate.

Many of those who are obese have a lower total body fat content, which implies that subcutaneous body fat is relatively inert in metabolic terms. This body fat scavenges against unbound circulating lipopolysaccharides with a consequential anti-inflammatory response and improved long-term outcomes. Adipose tissues have been shown to produce tumor necrosis factor (TNF)-α receptors, which are positively correlated with the level of body fat. As a result, the overweight and obese may have a protective buffer from the negative effect of an increase in TNF-α by producing higher levels of these receptors compared with those with a normal BMI.

Although there is a great deal of speculation regarding the reasons that overweight and obese patients experience better mortality than those of normal BMI, possibilities include that many of the more severely ill patients have already lost weight on account of one or more related or unrelated illnesses, or more aggressive treatment of diseases such as diabetes or hypertension.

2.1.11 Representativeness

Even participants in so-called nationally representative surveys of mortality and obesity may not be fully reflective of the total population. For example, the exclusion of those in institutions (e.g., in hospitals or nursing homes) raise a potential bias in the findings of these studies, particularly surveys covering those of older ages (according to the 2000 census, about 1.1 percent of Americans reside in institutional settings; 3.6 percent of American older than age 65 as of 2011 are institutionalized). This can affect any category in which a heterogeneous population is included, which reminds us to recognize the limitations involved in any study.

Masters et al (2013a) suggested that older obese individuals, especially the morbidly obese, are less likely than their normal-weight peers to participate in health surveys because of obesity-related health complications, thus creating in sampling bias from underrepresentation of unhealthy sub-populations. As a result, they suggest that unlike most studies of older age mortality and obesity, based on their study of nineteen annual NHISs (1986-2004) and the NHIS Linked Mortality file, they found a strong effect on age-specific mortality risk for the older obese population. However, they found that excess deaths among those who were BMI-overweight was significant only for white females and excess mortality for those overweight was not significant for males (for whom only classes 2 and 3 obese had a significant adverse effect).

The studies included in the Flegal paper include several nationally representative surveys, which tend to consist of heterogeneous and more representative populations, but also may suffer because of smaller sample size and exclusion of the institutional population. In contrast, cohort studies, typically designed to study a particular population segment or include participants satisfying specified criteria, can be quite large and are more heavily represented in meta-studies, such as Berrington de Gonzalez et al (2010) discussed below. Both types of study can be valuable and equally revealing. However, the details of the methodology used by these studies should be assessed before a conclusion can be reached regarding which provides more useful results.

2.1.12 Study period

A study with inadequate follow-up (i.e., time between initial body characteristic measurement and average or end of the observation period) may not be able to reveal the effects of the long underlying lagged effect of a condition such as obesity. A limitation of most studies arises as it is impractical to know reliably or incorporate historical weight/height experience prior to initial measurement, as most studies measure the effect of physical condition as of a point in time. In any event, the use of a short follow-up period can introduce a bias if the effect of a condition or characteristic over a long period is of concern. Nevertheless, the obesity paradox has also been found in somewhat longer study periods and significant categories of the population, such as those older than age 65, for example, in Lavie et al (2009).

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Although the obesity paradox has been reported in many short-term studies (for example, those with a follow-up period of less than five years), in Flegal's meta-analysis about two-thirds of the studies had an average exposure period greater than ten years. Nevertheless, it is possible that the adverse effects of trends in obesity over the last thirty years have not yet had an opportunity to fully affect mortality experience in reported studies because of the long latency period between the onset of obesity and consequential disease and death, and because it is usually the cumulative effect of adiposity that is important, rather than the current or recent obese condition (however, note that an individual may have had obesity onset years if not decades before initial measurement in a particular study, so it is difficult to determine the true lag period that has been in effect).

Studies such as the Uppsala Longitudinal Study of Adult males of Sweden with a median follow-up period of 30 years (Ärnlöv et al (2009)) have indicated that even when other elements of the metabolic syndrome are present, BMI remains associated with mortality.

There have been mixed results for mortality after an acute myocardial infarction over a long term period. For example, Nigam et al (2006) reported on the results of a study of those admitted to the Mayo Clinic Coronary Care Unit over a six month period with hazard ratios for both those overweight and obese less than those of normal BMI. However, over a longer-term period the overweight and obese experienced mortality risks similar to that of those of normal weight, with a higher risk of recurrent acute myocardial infarction (a hazard ratio of 2.3 for those overweight and obese). They speculated that a younger age at time of initial infarction and fewer non-cardiovascular co-morbidities could help to explain the short-period obesity survival paradox.

The fact that certain more recent studies have indicated more of an obesity paradox may simply be an indication that the cumulative effect of obesity may not have yet had sufficient time to manifest itself in adverse mortality consequences in the study participants. As indicated below, it may be that adverse results from adiposity tissue exposure, especially for those at the high end of the obesity-mortality curve, can only be observed after a long-period of time, say ten or thirty years.

Because of the long lag period between exposure to conditions such as obesity, some public policy analysts believe it appropriate to consider the effects of and on obesity on public policy over time periods longer than the typical time horizon (for example, typical U.S. public policy analysis is limited to ten years).

Another aspect of a study period is differential experience by birth cohorts. The commonly used American approach is to either adjust the data for attained age experience, reflecting measurements made at the beginning of the study period or to segment experience by broad age groups. These have been incorporated into various projections, such as those developed by Reither et al (2011) and Preston et al (2012), as well as in analysis prepared by Yu (2012) and Masters et al (2013b). Masters indicated that ignoring birth cohorts can overlook substantial differences in the history of obesity and other factors, possibly resulting in biased estimates of the future effects of obesity, especially as a result of recent earlier onset of obesity, with corresponding adverse cumulative effect of excess adiposity. Masters found a substantially stronger association than found in earlier studies between obesity and mortality risk at older ages, with an increasing percentage of mortality attributable to obesity across birth cohorts. Yu's findings, based on NHANES III, indicated that including cohort effects and gender differences had a significant effect on study results. He found that early and extended exposure to excess BMI appeared to result in excess deaths among the moderately fat groups. Mortality differentials increased across cohorts, but did not decline with age or change over the study period (i.e., there were no significant period effects). Yu found that ignoring cohort differences led to a declining pattern of excess mortality by age.

2.1.13 Self-reporting bias

Some studies use self-reported, rather than professionally measured, weight and height values in calculating the BMI. Since people tend to under-estimate their weight (especially those who are heavier) and over-estimate their height, the results of studies using self-reported values can result in biased findings/relativities compared with those using professionally measured input values, with males tending to over-estimate their height more and

females tending to under-estimate their weight more, and with older individuals over-estimating both. Thus, biases can result when results are studied by gender or age. In addition, those who are underweight tend to over-report their weight, and those who are overweight tend to under-report their weight, thus resulting in underestimated extremes in weight or height-based indices like BMI. Based on NHANES III, estimates of the effect of reporting errors have been 2.5 percentage points for white females, 5 percentage points for black females, and 6 percentage points for Mexican-American females. Reporting errors in males have generally not been as significant, but while a slight increase in obesity for white and black American males may result, there would be a slight decrease for Mexican-American males.

Flegal studied the effect of this type of measurement bias by comparing studies of alternative measurement sources. For example, for the overweight the hazard ratio was 0.93 for those with measured values in contrast with 0.96 for those with self-reported values; for the obese, corresponding hazard ratios were 1.13 and 1.39. While in some studies this may not contribute significantly to any conclusions reached, NHANES researchers have found that every single unit increase in BMI can be associated with a two pound underestimation of weight. When segmented by gender, males on average were found to overestimate their weight by five pound and females to underestimate their weight by 1.8 pounds, and height was overestimated, especially by males. Those at older ages are especially prone to over-report their height, which can also lead to under-estimating their overweight or obese status, at least according to a BMI measure.

Thus, for example, the percent determined to be obese on a self-measured basis in the Behavioral Risk Factor Surveillance System (BFRSS) is significantly less than that determined by the professionally measured NHANES. This is the reason that the percentage of the population that is estimated as being obese has always been greater when reported by NHANES than by BFRSS. Overall, although the type of measurement may make a difference in obesity prevalence rates, resulting differences in mortality are usually not significant.

Self-reported levels of physical activity are also poorly estimated, with Van Poppel et al (2010) estimating that correlation with true levels are in the 0.3 to 0.5 range.

2.1.14   Inappropriate selection of studies

The selection criteria used to determine which studies to include in a meta-analysis can rule out many relevant high-quality studies (determined by one observer, the Flegal study did not include recent studies of more than six million people, several of which indicated that the lowest mortality is experienced by those in the BMI-normal category). It has been claimed that Flegal's selected studies included many participants who were chronically ill, current smokers, and older adults, all of which could have biased the findings of a meta-analysis study. In part in anticipation of this concern, the study assessed the effect of several of these larger cohort studies and indicated that their inclusion would not have changed its results significantly.

2.1.15   Prevalence of more extreme obesity

Experience of the aggregate class of obesity (i.e., BMI ≥ 30) in any study is influenced by the distribution of obese by class, especially the percent of those extremely obese included, in part because the effect operates in a continuous rather than a discrete (step-wise) manner for each particular category. For example, if the study is somewhat old and the percentage of extremely obese is relatively small compared to their current mix, the aggregate resulting hazard ratio will be understated relative to current mix. Thus, the study of obesity should segment various classes of obesity, at least separating class 1 obese from others.

2.1.16   Other factors influencing mortality

It is possible that in many studies the effect of obesity has been outweighed by other relevant factors. For example, several major risk factors for cardiovascular disease, including high blood pressure in males, smoking, and cholesterol levels have improved (including the positive effect of drugs) over the last several decades. In addition,

improved medical interventions linking obesity to cardiovascular disease and all-cause mortality, such as more intensive treatment of those obese (e.g., through statins and aspirin) and improved control of hypertension (although recent trends in the level of hypertension increased in the U.S. population from 25.8 percent in 2005 to 28.3 percent in 2009) may have reduced the effect of obese conditions.

2.1.17 Unintended consequences of paradigm discussions

A concern has been raised that publicizing obesity paradox results has the potential to send the wrong message to the public, that is, implying that being overweight or somewhat obese isn't that bad and may even have protective value. The fear is that such publicity will remove personal incentives to effectively manage weight, which can in any event be quite difficult. Although on the surface this argument makes sense, it presumes that the public and the media cannot recognize the overall physical (mortality and morbidity) and psychological issues involved, and limitations of the results and scope of the Flegal and similar studies discussed above.

To be fair, those who put forth this view are concerned that individuals and policymakers will take the obesity paradox results out of context, and will ignore limitations of the conclusions and alternative explanations, and ignore other related effects of obesity, including its adverse health and morbidity effects (see "Morbidity" below), as well as the significance of other obesity metrics and the uncertainty surrounding the long-term effects of obesity. Just because there may be limited evidence that you can't eat yourself to death as easily as some had thought, there are many other factors to consider, such as the implications of central obesity, degree of adiposity tissues and lack of fitness, as well as of the cost to society of excess health care cost and a lower personal quality of life.

Although it is unlikely that those who are BMI-obese or are at risk of becoming BMI-obese will be encouraged through information regarding the obesity paradox to significantly change their nutritional and physical activity decisions, it is desirable, where possible, to provide a more complete story regarding it. For example, if the paradox is at least in part due to earlier or more aggressive treatment or co-morbidities (for example, the other components of the metabolic syndrome—fasting blood glucose, blood pressure, triglycerides, high density lipoprotein cholesterol), this should lead to more appropriate attention and treatment for those who are BMI-normal. If what we are seeing is a lag in adverse mortality results, then current action is appropriate because avoidance of a significant weight gain is easier than taking weight off after the fact.

2.2 Mortality results

In contrast to the Flegal et al (2013) paper, in a meta-analysis of 19 studies of 1.46 million white American adults with a follow-up period of between five and 28 years, Berrington de Gonzalez et al (2010) found a J-shaped relationship (see Figure 6), reflecting all-cause mortality associations for those who never smoked) between BMI and mortality, with hazard ratios of 1.47 (BMI of 15.0 to 18.4), 1.14 (BMI of 18.5-19.9), 1.00 (BMI of 20.0 to 22.4), 1.13 (BMI of 25.0-29.9), 1.44 (BMI of 30.0-34.9), 1.88 (BMI of 35.0 to 39.9), and 2.51 (BMI of 40.0+), all in comparison to a 22.5-24.9 BMI benchmark. These results, as well as breakdowns of them, are shown in Figure 7 and Tables 5 and 6. Consistent with the results of other studies, hazard ratios were lower for ages older than 70 compared to those at ages less than 50, possibly indicating an additive rather than multiplicative nature of excess mortality that proportional hazard models indicate (and of course correspondingly greater hazard ratios at younger ages). With increasing follow-up periods, greater hazard ratios emerged for those whose BMI was less than 18.5. In addition, for those who are underweight, the effect of a higher level of physical activity reduced hazard ratios. For those overweight and obese the major source of additional mortality resulted from cardiovascular conditions and was lowest for cancers. The authors contrasted the results of their study with others that showed lower hazard ratios for those overweight and obese (e.g., those based on NHANES) and indicated that part of the difference could be due to the inclusion of current and previous smokers.

Whether or how to reflect experience of current or former smokers may depend upon the use of these results. For example, if the findings are to be used to extrapolate results to the entire population, then an inclusive basis that considers, for example, expected experience of those with current illnesses may be appropriate (note that assessment of each sub-population separately will result in a better projection only if reliable estimates of each of the sub-populations can be

developed). If, however, when solely examining the question of the effect of obesity, studying experience of non-smokers separately from current or past smokers would be desirable, thus reducing the effect of reverse causation – Berrington de Gonzalez took the latter approach, particularly as the effect of smoking and obesity have been negatively correlated. As the follow-up period was lengthened, as indicated in Table 5, the hazard ratios for the overweight and obese groups also increased, confirming the likely relevance of the lag time between obesity and consequential mortality. Flegal excluded studies that did not consider reverse causation.

Figure 7 illustrates a gap at low BMIs between the hazard ratio curves of never-smokers and all participants that reflects the likely effect of smoking and respiratory illnesses, while the reverse gap at higher BMI levels indicates the possible adverse effect of obesity on mortality due to cardiovascular co-morbidities. Berrington de Gonzalez viewed the uptick at the left side of the curves as possibly due to pre-existing undiagnosed diseases at the time of initial measurement, as this effect weakened after about a 15 year follow-up period and the relationship between mortality at low BMI for former smokers was stronger than for former smokers who had quit less than twenty years ago, likely reflective of the residual effects relating to smoking.

Figure 7 - Estimated hazard ratios for death from any cause according to BMI for all study participants and healthy subjects who never smoked
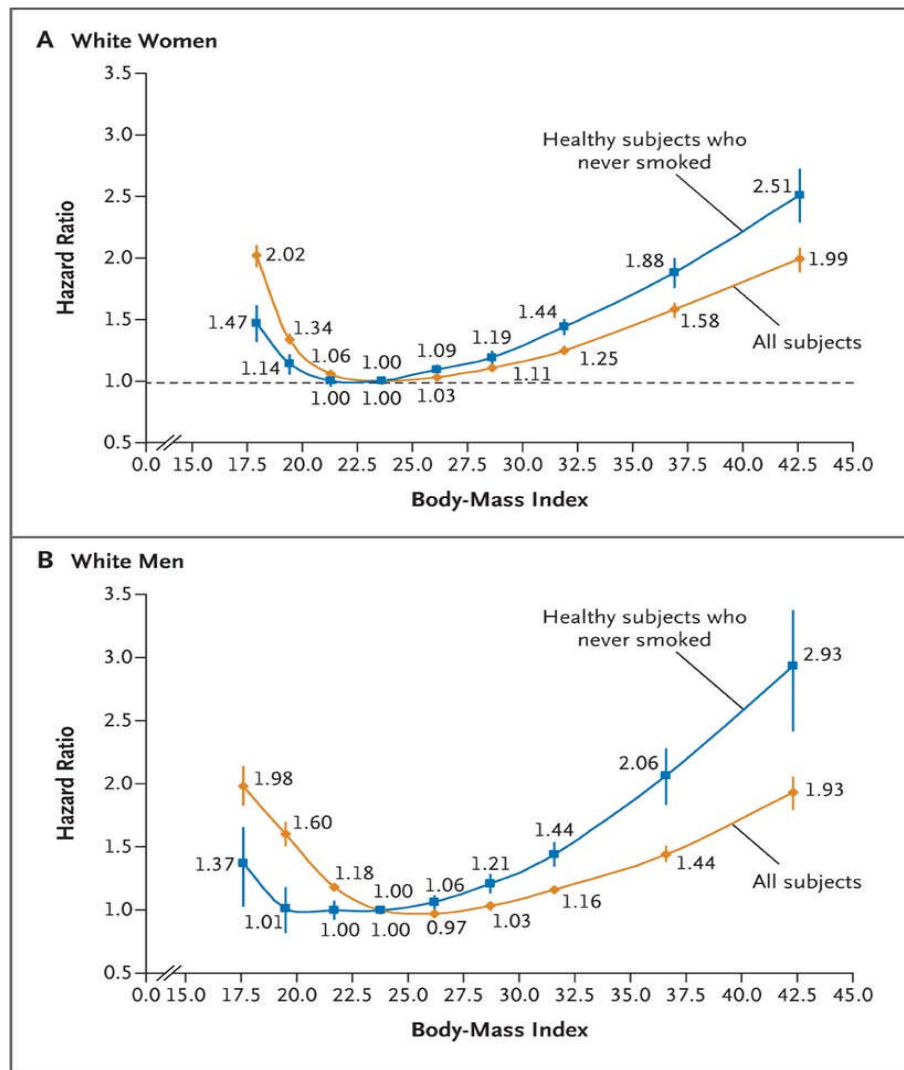
source: Berrington de Gonzalez et al (2010)

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Table 5 – Hazard ratios and extra deaths per 1,000 exposed lives by age, years of follow-up and death, for healthy lives, benchmark BMI of 22.5-24.9

Hazard ratios	BMI range							
	15.0-18.4	18.5-19.9	20.0-22.4	25.0-27.4	27.5-29.9	30.0-34.9	35.0-39.9	40.0-49.9
20-49	1.45	0.95	1.04	1.30	1.44	1.79	2.48	3.70
50-59	1.15	1.14	0.96	1.10	1.22	1.56	2.06	2.77
60-69	1.49	1.15	1.00	1.03	1.15	1.34	1.77	2.27
70-84	1.65	1.33	1.06	1.04	1.15	1.24	1.59	1.91
Extra deaths/1,000								
20-49	0.7	-0.1	0.1	0.5	0.7	1.3	2.4	4.3
50-59	0.6	0.5	-0.2	0.4	0.9	2.2	4.1	6.9
60-69	4.0	1.2	0.0	0.2	1.2	2.8	6.3	10.4
70-84	10.7	5.2	1.0	0.7	2.5	3.9	9.7	14.9
Years of follow-up								
< 5 years	1.73	1.19	1.04	1.07	1.19	1.44	1.67	2.28
5-9 years	1.51	1.23	1.03	1.07	1.20	1.42	1.93	2.39
10-14 years	1.24	1.18	1.03	1.04	1.19	1.47	1.97	2.65
≥ 15 years	1.21	0.92	0.92	1.12	1.19	1.41	2.04	3.11
Cause of death								
Cardiovascular disease	1.50	1.15	0.96	1.25	1.52	2.04	3.05	4.42
Cancer	1.09	0.96	0.95	1.06	1.13	1.37	1.55	1.85
Other	1.88	1.30	1.12	1.02	1.13	1.29	2.00	3.00

source: Berrington de Gonzalez et al (2010)

As suggested by the cause of death portion in Table 5, the pattern of the all-cause mortality-BMI curves in Figure 6 is not consistent with the pattern applicable to individual causes of death. For example, the curves for cancer and respiratory diseases are often higher at low BMI levels (especially for respiratory deaths for females) and not as high in the right tail of the mortality-BMI curve—the underweight BMI part of the curve in this case is relatively low because it excludes current and previous smokers. The curve due to diabetes can be steeper and is usually monotonically increasing as BMI increases. As the cause of death mix continues to change, the shape of the all-cause mortality curve would also be expected to change.

Table 6 compares hazard ratios from Flegal et al (2013) and Berrington de Gonzalez et al (2010), illustrating the inconsistent results (particularly with respect to those in overweight and class 1 obese categories).

Table 6 – Flegal and Berrington de Gonzalez BMI based hazard ratios

BMI Range	Flegal et al (2013)	Berrington de Gonzalez et al (2010)	
< 18.5	Excluded	1.47	1.37
18.5 – 19.9	1.00	1.14	1.01
20.0 – 24.9	1.00	1.00	1.00
25.0 – 29.9	0.94	1.13	1.13
30.0 – 34.9	0.95*	1.44	1.44
35.0 – 39.9	1.29*	1.88	2.06
40.0 – 44.9		2.51	2.93
≥ 45.0	Excluded		

* class 2-3 obese – 1.18

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Consistent with Berrington de Gonzalez et al (2010), Whitlock et al (2009), in the Prospective Studies Collaboration (with a primary focus on cardiovascular disease covering 61 studies and 900,000 individuals with most exposures in Europe and North America) found hazard ratios for individuals with a BMI greater than 25.0 increased on average by 30 percent for each increase in 5.0 of BMI from its benchmark of 22.5 to 24.9 (in which the lowest mortality rates were experienced). In the aggregate, Whitlock found a positive relationship between all-cause mortality and ischemic heart disease and stroke mortality at advanced ages (in the 80s of about 1.15, 1.30, and 1.10, respectively) for those overweight and obese. This large study indicated that being overweight or obese shortens life expectancy – for example, being in the class 1 obese category comes with a reduction in life expectancy of three years and obese class 3+ a reduction of about ten years. The main sources of this excess mortality were from heart disease and stroke, although it also showed increased mortality due to certain forms of cancer. In the U.K., the study found for those in their middle ages as many as one in four deaths due to heart attack or stroke, while one in sixteen cancer deaths was due to being overweight or obese.

Canoy et al (2013) studied coronary heart disease incidence and mortality in 1.2 million females in the nine year follow-up period (the first four years they excluded) of the Million Women Study, whose participants at the start of the study did not have heart disease, stroke or cancer. They found a J-shaped BMI relation in both 20 year incidence and mortality in smokers and non-smokers, physically active and inactive, and in upper and lower socioeconomic classes. With a benchmark BMI between 22.5 and 24.9, coronary heart disease incidence hazard ratios for those less than 20 BMI was 1.27, for overweight 1.23, class 1 obese 1.49, and for class 2+ obese 2.84, compared with mortality hazard ratios of 0.89, 1.20, 1.53, and 1.85, respectively, indicating that BMI's effect was more severe for incidence of coronary heart disease than for death, especially at the extremes of the BMI range. This suggests either more frequent but less severe cardiovascular disease episodes or preventive value from obesity once their more frequent episodes begin. Similarly, Tobias et al (2014) reported hazard ratios from the long follow-up period Nurses' Health Survey (U.S.) and the Health Professionals Follow-Up study of 1.29, 1.00, 1.12, 1.09, 1.24 and 1.33 for those who have never smoked and of 1.12, 1.00, 1.16, 1.21, 1.36, and 1.56 for those who are current or former smokers for BMI ranges 18.5-22.4, 24.9, 25.0-29.9, 30.0-34.9, and 35.0+, respectively).

Similar to findings in Flegal et al (2013), in a meta-analysis study of 26 studies (74 cohorts and 288,622 individuals in the Diverse Populations Collaboration), McGee (2005) found no excess mortality in those who were overweight (with a benchmark BMI of 18.5 to 24.9), but hazard ratios of 1.22, 1.57, 1.48, and 1.07 for all-cause, cancer, coronary heart disease, and cardiovascular disease, and cancer, respectively.

In contrast to McGee's findings, other studies have shown mortality hazard ratios greater than normal BMI in the overweight category, as well as for the obese. For example, the long running Nurses' Health Survey, with a follow-up period of 24 years beginning in 1980 as reported by van Dam et al (2008), showed that not only was the all-cause mortality hazard ratio of 1.18 for those overweight compared to a normal BMI category benchmark between 18.5 and 24.9, and 1.67 for obese, but hazard ratios for cardiovascular deaths were 1.46 and 2.81 and cancer deaths were 1.14 and 1.32 for the overweight and obese, respectively. Smoking, physical activity and a healthy diet were also associated with BMI-measured mortality. Also, mortality risk was higher for the overweight and the obese white adults in the Cancer Prevention Study II and for white female adults in the NIH-AARP Diet and Health Study.

Jee (2006), reporting on the large (1,213,829 participants aged 30 to 95) Korean Cancer Prevention Study with a follow-up period of twelve years through 2004, found a J-curve BMI relationship, with excess deaths beginning in the overweight category (for those who never smoked, a hazard ratio of 1.04 for overweight males and females and 1.71 for obese males and 1.20 for obese females, compared with a benchmark of those with a BMI between 23.0 and 24.9 who experienced the lowest mortality levels). This is inconsistent with other studies of East Asians that have indicated the lowest mortality was usually at somewhat lower BMI levels. As with many American and European studies, the high obesity hazard ratios declined with increasing age, beginning in this case after age 50, but especially for those ages 65 and older. This study excluded the first two years of its study period and adjusted for age, gender, physical activity level, and alcohol consumption. Patterns of mortality for smokers and non-smokers by cause of death were similar—cardiovascular disease mortality increased steadily after a BMI of 30.0, cancer increased after a BMI of 28.0, and respiratory deaths decreased throughout the BMI-mortality curve.

Further, Kenchaiah et al (2002), reporting on the 15th biennial Framingham and second offspring study with an average age of participants of 55 through 1983 with a mean follow-up period of 14 years, indicated that the obese experienced a hazard

ratio of 2.12 for females and 1.90 for males for the risk of heart failure, with corresponding hazard ratios for those overweight of 1.50 and 1.68, respectively. The results in this study did not differ significantly by age, gender, smoking status, alcohol consumption, or presence of valve disease or diabetes. However, hypertension and whether a myocardial infarction occurred were related, somewhat lowering these hazard ratios. The authors concluded that the relatively large ratios suggested that a conclusion could be that a causal relationship exists.

Bessonova et al (2013) reported on the results of the California Teachers Study, covering 133,433 teachers. After excluding those who died in the first year of the study period and those with a co-morbidity at baseline, all-cause mortality hazard ratios (relative to that of those between 18.5 and 24.9 BMI) were 1.33 for the underweight, 1.04 for the overweight, and 1.27 for the obese. After stratification for menopausal hormone therapy and smoking history, the study concluded that the obese had a greater risk of all-cause mortality and certain cancer and cardiovascular and respiratory diseases.

The following discussion covers results found from Canada, Mexico and large U.S. ethnic groups.

Four Canadian studies with relatively long follow-up periods provide additional perspective. A summary of their findings is given in Table 7, including hazard ratios at different BMI ranges to indicated benchmark BMIs. The nationally representative National Population Health Survey reported on in Orpana et al (2010) is the only study of these four that indicate an obesity paradox (note that it is the only study of the four that covered experience of all adult ages).

Table 7 – Canadian BMI studies

Authors	Katzmarzyk et al (2001)	Jain et al (2005)	Orpana et al (2010)	Katzmarzyk et al (2012)
Study	Canada Fitness Survey	Canada National Breast Screening Study	National Population Health Survey	Canada Heart Health Surveys
Participants	10,725	49,165	11,326	10,522
Initiation and follow-up	1981 for 13 years	1980-85 for 16.5 years	1994/5 for 12 years	1986/95 for 13.9 years
Ages at initial study	20-69	females 40-59	≥ 25	18-74
BMI < 18.5	1.63	1.12	1.73	1.25 (1.30CVD; 1.02C)
18.5 - 24.9	1.00	1.00 (18.5-21.9) 1.15 (22.0-24.9)	1.00	1.00 (1.00CVD; 1.00C)
25.0 - 29.9	1.16	1.28 (25.0-27.9 1.34 (28.0-29.9)	0.83	1.06 (1.57CVD; 1.14C)
30.0 – 34.9	1.25	1.30	0.95	1.27 (1.72CVD; 1.34C)
35.0 +	2.96	1.40	1.36	1.65 (2.09CVD; 1.82C)

Obesity is more highly concentrated among the poorest segment of the Mexican population than in other Latin American countries, with the young being especially adversely affected. In many regions, obesity has occurred alongside a relatively high prevalence of infectious and parasitic diseases in the same population (Monteverde et al (2010)). The country is also seeing a growth in obesity-driven deaths and adverse health care conditions, especially as a result of high levels of diabetes—the lag between growth in excess weight and consequential health deterioration may lead to dire consequences. Already, excess (greater than in the U.S.) mortality has been seen in the elderly, especially because once a chronic condition arises, the relative risk of dying from these diseases has been greater in Mexico than in the U.S. It is possible that obesity has a greater mortality effect in the elderly (even with a somewhat lower probability of suffering obesity-related chronic diseases) than in the U.S. because of (1) weaker self-reporting of health problems, (2) worse consequential mortality experience from these conditions and (3) a significant difference in educational level at those ages. However, as education converges in younger cohorts, this latter effect may reduce over time. This also may be due to better overall treatment and adherence to treatment protocols in the U.S. In addition, according to Monteverde, the higher level of excess mortality effect has to date been independent of age. The taxes introduced in late 2013 on drinks and food were implemented as a reaction to these adverse trends.

Many studies of mortality and obesity of American ethnic and racial groups have been conducted. A particular population segment of interest is black females, the population segment in which obesity is most heavily concentrated. Earlier studies of this group indicated that BMI did not have a significant relationship with mortality, except for very high levels of BMI. Boggs et al (2011), in the ongoing Black Women's Health Study (with a follow-up period of thirteen years for those between 21 and 69 at enrollment), found that all-cause mortality hazard ratios increased with an increasing BMI of 25.0 or higher. Using as reference BMIs between 22.5 and 24.9, mortality hazard ratios were 1.89, 1.36, 1.00, 1.12, 1.31, 1.27, 1.51, and 2.19, for BMIs of less than 18.5, 18.5-22.4, 22.5-24.9, 25.0-27.4, 27.5-29.9, 30.0-34.9, 35.0-39.9, and 40.0-49.9, respectively. Although they found that the association between BMI and mortality did not differ according to age or the extent of physical activity, there was a strong association with cardiovascular disease but no relationship with death from cancer, with a relationship with other causes only for the class 2+ obese. After adjusting for BMI in Boggs, WC was only associated with mortality for BMI of less than 30.0, with no such relationship among current smokers. This result is inconsistent with the results shown in Lakoski et al (2011) in the Reasons for Geographic and Racial Differences in Stroke (REGARDS) study of 11, 618 black participants with an average age of 64.9, in which neither BMI nor WC was associated with mortality risk in black males and only very high WC was associated with adverse mortality of black females. As with many studies of the total population, mortality in this study tended to show a weaker relationship with obesity among individuals older than age 60 compared with that among those younger than age 60.

Using percent body fat rather than BMI as an obesity metric, the black-white gap in obesity prevalence rate among females decreases by more than half, while the black males' obesity prevalence rate using percent body fat is 16 percentage points lower than white males compared to about the same prevalence rate using BMI to characterize obesity.

Krishnan et al (2007) reported earlier findings of the all-cause mortality hazard ratio in the Black Women's Health Study for black females in class 4+ obese were twenty three times that of those with a BMI of 23 or less, with central adiposity constituting a significant risk. Also the hazard ratio of those in the highest quintile of WtoH ratio relative to the lowest quintile was 2.3 (and the corresponding WC ratio was 2.4). This was after controlling for BMI, with diabetes being a strong risk factor, the prevalence of which is twice as high as for white females. A positive association between BMI and mortality was far stronger among black females with more years of education (consistent with the findings from the Cancer Prevention Study I and earlier studies).

Possibly the reason why the results of Krishan et al are inconsistent with those of prior studies of black females is that 97 percent of participants in this study had at least a high school education, which contrasts with Cohen et al (2012) reporting on the Southern Community Cohort study, largely of low-income participants in twelve southeastern states, in which obesity was not associated with the same level of excess mortality risk (1.13 and 0.87 hazard ratios for black males and females for obese class 3+, respectively). If this educational difference is a significant explanation for the difference in results, as the overall percent of more highly educated black females continues to increase, the effect of BMI on black female mortality may become more significant.

Based on eleven years of National Health Interview Survey data of about a third of a million Americans, Stommel and Schoenborn (2010), using narrow BMI groupings, studied mortality and obesity relationships for the four major American ethnic groups and found monotonically increasing mortality hazard ratios and chronic disease burdens (after adjustments for many socio-demographic characteristics and history of smoking, physical activity, and alcohol consumption) beginning at BMIs of 20-21. This study did not indicate any hazard ratio discontinuity across BMI values, suggesting that the use of broad BMI groupings may hide differing values within the groupings used (for example, the grouping of all those obese might have a hazard ratio of less than 1.0 for obese class 1 and greater than 1.0 for obese classes 2+). Stommel and Schoenborn found that, although racial and ethnic groups differ substantially in their overall risks for many chronic conditions (particularly for hypertension, diabetes, functionally limiting arthritis, cardiovascular diseases, and asthma (the latter at BMIs larger than 25)), the association between BMI and disease risk was broadly similar.

2.3. Older ages

On an overall basis, little, if any, additional mortality for the elderly (say, over age 75) has been observed in many studies for those overweight or at moderate obesity levels as measured by BMI. Nevertheless, because of the lengthy lags between

certain behaviors, physical condition, and consequential mortality, any negative effect on mortality levels from the increase in the prevalence of obesity may not yet have been observed and may have been overwhelmed by favorable changes over the last few decades that have resulted from the dramatic reductions in smoking and enhanced control of other cardiovascular risk factors.

Several observations are necessary to put obesity at older ages in the proper context, some of which are similar to the issues discussed in "Obesity paradox" above.

- Body composition changes. With age, musculoskeletal characteristics change as muscle mass and lean body mass tend to be replaced by fat mass. Body mass and skeletal as well as other structural changes, measured by means of height, weight or waist size, especially occur in the oldest old due to the aging process. This may result in a higher "ideal" BMI range and inappropriately designated BMI categories for those of older age. This could be addressed by using different minimum and maximum BMI values for each BMI category or using BMI quintiles or quartiles rather than fixed ranges of BMI.

- Weight loss. Unintentional weight loss and other confounders that are obscured or are more difficult to discern due to normal age-related changes in body composition. It is rare that a study can parse mortality experience between those who have lost weight and those who have had a stable weight history.

- Co-morbidities. There tends to be a weaker association with mortality risk at older ages because of the existence of more and more significant co-morbidities and cardiovascular risk factors, such as blood pressure and cholesterol. In addition, older persons may be more likely than those younger to have more undiagnosed as well as diagnosed medical conditions or diseases that may result in a lower BMI.

- Protective value. Current excess body fat may become of increasingly protective value with age, e.g., it reduces the likelihood of adverse consequences from falls (and may make it less likely that someone obese relative to someone who is frail is more likely to fall) and can provide metabolic and nutritional energy reserves in a period of caloric need during an acute illness. Overall, frailty or lack of excess weight may be an intermediating mechanism between weight loss after midlife and late life mortality.

- Inappropriate metric. Alternative measures, such as WC, may provide a better mortality risk metric than BMI or overall weight. In addition, since height as well as weight often decreases at older ages, corresponding BMI values may become inconsistent over time, even looking at the same individual. In studies of self-reported weights and heights, self-reporting errors of the elderly may be particularly significant. However, even waist measurement can prove difficult to obtain on a consistent basis as body composition and even the location of the waist can change.

- Inadequate follow-up periods. This particularly can prove problematic, as the obesity metric is often measured at an insufficiently young age. This becomes especially important to the extent that long-term exposure to adiposity affects mortality.

- Selective survivor effect. This is particularly relevant at older ages or in studies with a long follow-up period; that is, those obese who have prematurely died before reaching older ages may constitute a less healthy group than the obese who survive to older ages.

- Form of excess mortality. Even if the hazard ratio (a multiplicative relationship between the subject mortality rate and that of benchmark mortality at a normal BMI level) at higher BMIs is lower than that of younger ages, excess mortality may take the form of additive (expressed in terms of extra deaths per thousand of exposed lives) mortality, rather than taking a multiplicative form, due in part to the much larger rates of mortality at older ages.

Similar to studies of younger adults, studies of mortality of older adults have shown inconsistent results. For example, Janssen and Marks (2007) aggregated the results of 26 studies and found an average all-cause hazard ratio (compared with that in the normal BMI range) of 1.00 for those in the overweight category, while for that of moderate obesity was 1.10.

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The Cardiovascular Health Study, as described in Janssen and Marks with a follow-up period of up to nine years, experienced a mortality hazard ratio for those overweight 11 percent less than those in the normal BMI range. Nevertheless, a significantly higher rate of diabetes has emerged in recent years at ages older than 65 that might change those results.

Heiat (2001) evaluated 13 studies with follow-up periods of between three and 23 years in detail. A mixed relationship was found between BMI and mortality, although it was concluded that the optimal BMI was at least 27, rather than in the 22.5-25.0 range sometimes found at younger ages. Studies that measure BMI when participants are in their middle years and study mortality during later life can provide a better sense of the effect of exposure to excess weight over a longer period and its underlying relationship with mortality at older ages. Only two studies (the Framingham Heart Study and the American Cancer Society's Cancer Prevention Study) included in Heiat's meta-analysis showed a positive relation between BMI and mortality for the obese, with its other studies showing either no or a negative relationship. In these studies, a U-shaped BMI mortality curve for cardiovascular disease was evident, with the lowest mortality level not reached until a BMI of 31 or 32, with a less steep upward slope at older ages.

The Helsinki Businessmen study reported on by Strandberg et al (2009) compared the mortality of several weight trajectories between 2000 to 2006 of 1,114 males beginning at ages 25, 47, and 73 through a mean age of 73. From midlife to older age, the greatest coronary heart disease and all-cause mortality risk (a hazard ratio of 2.00) from the period after age 73 was for those who reduced their BMI, from the overweight or obese category to a normal BMI compared to those of stable weight between those ages. This suggests that a reason for lower mortality of those overweight and obese compared with normal BMI at older ages is that many in the BMI-overweight and obese categories at mid-life who lose significant weight may be relatively unhealthy, leaving healthier individuals in the overweight and obese categories at older ages, thus contributing to the obesity paradox at those ages.

Jacobs (2010) emphasized the importance of WC in older adults, regardless of BMI. Particularly in the elderly, a low BMI may reflect low lean body mass or the presence of diseases that can result in weight loss. Hazard ratios after adjustment for BMI were significantly lower in males younger than age 70 than in those 70 and older, with little difference by age among females (this result is different than in other studies where all hazard ratios were lower among the elderly). WC was associated with increased risk of mortality within each cause of death studied, more so for respiratory disease and all causes other than for cardiovascular disease and cancer.

A Finnish study of nonagenarians with a four year follow-up period presented in Lisko et al (2011) showed that males who are overweight have a hazard ratio of about a third that of those of normal BMI, while there was little difference when using WC (a 0.96 hazard ratio). In contrast, for females, WtoH ratios were positively associated with mortality (1.43 hazard ratio to those with a lower WtoH ratio), while BMI-overweight females with a relatively small WC experienced lower mortality than those of normal BMI (hazard ratio of 0.34).

Flicker et al (2010), reporting on the results of the Health in Men Study and Australian Longitudinal Study of Women's Health for those between ages 70 and 75 at the beginning of the study period with a follow-up period up to 10 years, found that the mortality of those overweight was lower than that for those of normal weight (a hazard ratio of 0.87 for males and females). Hazard ratios for the obese were similar, at 0.98. Females who were sedentary had hazard ratios of 2.08 and males who were sedentary had a hazard ratio of 1.28. This suggests that the protective effect of physical activity for females has a relatively greater effect than for males.

In the Leisure World Cohort Study of a self-reported BMI at age 21 with mortality experience followed from an average of age 73, with a subsequent follow-up period of 23 years, Corrada (2006) showed that those who were overweight or obese at age 21 had greater mortality (a hazard ratio of 1.17) after age 73 than those who were BMI-normal at age 21. Those who lost weight had larger mortality regardless of their BMI category at age 21 (1.93, 1.28, and 1.26 for those then underweight, overweight, and obese, respectively). This contrasts with those of normal BMI at age 21 who gained weight by age 73 who experienced lower hazard ratios. Those underweight at age 21 had the highest hazard ratios, while for the obese at age 21 excess mortality only occurred at ages under 73 and among those who never smoked or who were past smokers.

During 29 years of follow-up in the Adventist Health and Mortality Studies in California, Singh et al (2011) indicated that, with a BMI benchmark of less than 22.3 for males and between 22.3 and 27.3 for females, males between ages 75 and 99

who maintained stable weight experienced a hazard ratio of 1.88 for BMIs between 23.0 and 27.3 and 2.00 for BMIs greater than 27.3, while females experienced a hazard ratio of 1.12 for BMIs less than 20.6 and 1.41 for BMIs in excess of 27.3. In this study BMIs were re-measured several times during the study period, thus representing the relationship between mortality and current BMI values. Note that Seventh-Day Adventists are lifelong non-smokers who limit their alcohol consumption and were relatively active physically. Singh speculated that one reason for the lower relative mortality at higher BMIs was that body fat, being the primary source of estrogen in post-menopausal females, can protect against heart disease and hip fractures.

Although mortality hazard ratios for overweight and obese Swedes reported in Dahl et al (2013) were 20 percent lower than those in the normal/underweight categories, the mortality hazard ratio for those 70 and older using as a benchmark of those who were of stable weight was 1.65 for those who lost weight, while the hazard ratio was 1.53 for those who gained weight. Hazard ratios were lower for those older than 80, presumably because of their larger mortality rates, but were greater on an additive basis.

Augyeung et al (2010), using a DEXA with a 5.5 year follow-up period, measured adiposity in older Chinese adults. An inverse relationship was observed between general levels of adiposity measured by BMI, body fat index (BFI, body fat / height squared), and body muscle mass index (BMMI, body muscle mass / height squared), with a J-shaped relationship observed using central adiposity measures (WtoH ratio and trunk fat / total body fat).

In a study of the BMI level at which the effect of obesity reverses, using a twenty year follow-up period, Cohen-Mansfield and Perach (2011) found that obesity was significantly predictive of greater mortality for Israelis aged 75-84 (a 1.30 hazard ratio compared with those of a BMI between 22 and 30), but from age 85 and onwards obesity had a protective effect (a 0.94 hazard ratio, although not statistically significant compared with those of a BMI between 22 and 30). Those underweight (with a BMI less than 22) experienced a 1.28 hazard ratio at age 75-84 and a 1.47 hazard ratio at ages 85-94 (the often observed greater mortality rates of those underweight may be due to nutrient deficiency, frailty, reduced functional status, and underlying disease). The hazard ratios were about the same for former smokers compared with those who never smoked.

Physical activity and fitness are just as important for the elderly as for those younger. Lack of fitness has been found to be a significant indicator of greater mortality (for example, in Yates (2008) in a study of those over 65, the hazard ratio for the most lean quintile of fitness was about one third compared with that of the least-lean quintile), independent of overall or abdominal adiposity. In this study, the class 1 obese experienced about a 30 percent excess rate of mortality, while the class 2 and 3 obese experienced a 130 percent excess rate of mortality, with those with a WC of greater than 88 cm for females and 102 cm for males having about 30 percent excess rate of mortality. Supporting this result, McAuley (2010) found that, even though hazard ratios for 981 healthy male veterans aged at least 65 showed paradox characteristics (hazard ratios of 2.51, 0.66, 0.50, and 0.44 for underweight, overweight, class 1 obese, and class 2+ obese, respectively), those obese at a high fitness level had a hazard ratio of 0.26. Not smoking was also associated with significantly lower mortality risk.

In studying causes of death (or disability), it can be difficult to attribute premature mortality and health-related costs directly to obesity, particularly in the aged. This is due to the simultaneous effects of multiple co-morbidities. For example in 2002 a quarter of the obese had six or more adverse medical conditions. The difficulty in deriving obesity attribution is due in part to the complex nature of the causes of mortality and morbidity incidents including the inter-relationships among health processes, are exacerbated by the lag between cause and effect, affecting the findings of any study of the sources of excess mortality and health care costs.

2.4. Period Studied

The following further discusses two aspects of the time over which mortality is studied in obesity studies: (1) the extent that cumulative exposure to adiposity tissues has a significant effect on mortality and (2) results of more recent study periods compared with those of earlier studies.

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Cumulative exposure to extra weight and adiposity tissues has a greater influence on health and contributes to greater mortality rates than corresponding exposure over a short period, although each period of exposure can be measured at the beginning of each time period. As a result, age at onset of obesity can be an important indicator of current or expected future ill health – a significant reason for public policy to focus on childhood obesity. An example illustrates a reason for low observed correlation between current weight and additional deaths 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 occurring a year later. In this case, an epidemiological study of the relationship between BMI and mortality that only measures BMI at age 70 after the weight loss occurs would be unable to identify the original cause of the premature death and would contribute to an obesity paradox. As this type of weight loss can especially occur at older ages, one can only conjecture that this is more likely to affect the mortality-BMI relationship at older ages.

This example shows that the long lag between being obese and eventual death can lead to misleading results during a short follow-up period between body measurement and the end of the study period, as many interim adverse chronic conditions and diseases can lead to weight loss at older ages. Studies with longer follow-up periods and exclusions of pre-existing conditions (e.g., ever-smokers or who have cancer) can provide a better perspective on the relationship between obesity and mortality in the elderly than if measured concurrently. In fact, the cumulative effect of the obesity surge that began in the 1970s in the U.S. may not yet have been fully captured by long-duration mortality studies. In any event, to obtain more useful results, it is generally appropriate to segment experience by major age categories and duration since measurement. It may be impractical to determine the ideal follow-up period for such a study, as it differs by the situation including age, but some have suggested that it is about 15 years.

Reis et al (2013), using a twenty five year follow-up period, found that longer durations of overall and central obesity were associated with sub-clinic coronary heart disease and its progression through midlife, independent of the degree of adiposity. They found that each additional year with overall or central obesity beginning in early adulthood was associated with a hazard ratio of 1.02 to 1.04 for coronary artery calcification and its progression later in life. This suggests that both a longer exposure to excess adiposity and an earlier age at obesity onset may result in an increase in the rate of coronary atherosclerosis. Reis also found that a longer duration of obesity was associated with higher levels of systolic blood pressure, insulin, C-reactive protein, and triglycerides, greater use of antihypertensive and lipid-lowering medications, higher rates of diabetes, and lower levels of high-density lipoprotein cholesterol. They indicated that possible explanations include sustained expression and secretion of proinflammatory adipocytokines, extended impairment of the fibrinolytic system via increased markets of hypercoagulability and hypofibrinlysis, as well as impaired nitric oxidedependent endothelial function, oxidative stress and upregulation of vasoconstrictor proteins.

Heir (2011) in a Norwegian study found with a 25-27 year follow-up period that BMI-overweight was related to cardiovascular mortality (but not to non-cardiovascular mortality), but not until after fifteen years of follow-up. Ärnlöv et al (2009) similarly found that during a 30 year follow-up period, divergence of mortality from normal BMI levels (for those without other metabolic syndrome indications) did not appear until ten years after the inception of the study. Thus, if these long follow-up period results can be generalized, recent studies with shorter follow-up periods may not fully reveal the total effect of obesity.

Based on studies of mice (and observations of human behavior), it is extremely difficult to lose weight over time once one is overweight or obese. Sustained weight management remains a challenge for most people who are obese. Liu and Elmquist (2013) found that this may be the result of a single gene, proopiomelanocortin in the pituitary gland and hypothalamus. They indicated that it is conceivable that being chronically overweight or obese may alter the structure and function of brain circuits that mediate the central control of energy homeostasis, indicating that it is possible that obesity may induce maladaptive and irreversible changes that selectively affect neural circuits that regulate energy expenditure. This is consistent with previous findings that food intake and energy expenditure are regulated by distinct neural pathways downstream of hypothalamic neurons of this gene. As a result of this process it is important to avoid or at least delay the onset of significant weight gain.

Several recent studies have reported a reduced relationship between obesity and mortality, for example in Mehta and Chang (2011). This change has been attributed in part to the significant reduction in cardiovascular disease events and

deaths, which has remained the most significant cause of death, but it may also be due to recent improvements in the relative health status of the average BMI-obese individual. It should be noted that many recent studies have also had shorter follow-up periods. More of this will be discussed under "Projections".

2.5 Projections

The issue addressed here is not a simple one—what will be the effects on population mortality of carrying extra weight over the long-term. It should be easy to project mortality—applying a straight-line extrapolation to recent trends to project the future, continuing indefinitely. However, time after time, such projections have proved inaccurate—an example of a simple extrapolation that could have been performed in 2005 would have led to an impossibly high obesity prevalence in the U.S., as there had been an almost linear increase in obesity rates between 1975 and 2005. The results of any linear extrapolation are unlikely.

Some observers, including Reither et al (2011) and Preston et al (2012), have incorporated projections based on generational cohorts. Reither suggested that aggregate trends fail to incorporate the possibility that recently and yet-to-be-born generations may not see the same rates of mortality improvement in the future as the prior generation. Mehta and Chang (2011) found that a significant difference in mortality rate relativities over the past few decades have attenuated the previous relationships between BMI and mortality. Both Olshansky et al (2005) and Stewart et al (2009) relied on earlier NHANES' relativity data and derived relatively pessimistic mortality projections as a result of an assumption of earlier mortality studies of the relationship between mortality and BMI, while Preston (2012), using more recent NHANES' relativities derived a more optimistic overall projection. Stewart's forecast incorporated an assumption that adverse mortality due to obesity would exceed the favorable effects of reduced smoking during 2005-2020.

Using a later version of NHANES data by birth cohort, Preston et al (2014) developed mortality assumptions based on cohort-related smoking and obesity patterns, with changes in smoking and obesity being expected to have significant effects on future mortality. Preston projected that through 2040, the reductions in smoking by males will have a larger effect than their increases in obesity, with a combined effect being an increase in life expectancy at age 40 for males of 0.83 years, although for females, the effect of the two changes largely offset each other, with an expected gain in life expectancy at age 40 of 0.09 years by 2040; for changes in obesity alone the corresponding reduction in life expectancy was projected to be 0.73 years for males and 0.82 for females and for smoking alone the corresponding gain was projected to be 1.54 years for males and 0.85 years for females. Preston emphasized the importance of the time that individuals spend in an unhealthy status (that is, time while obese or while smoking). While, as noted above, there has been a significant increase in the number currently obese, this has also contributed to a decline in the average duration while obese from 17.6 years in 1990 to 14.3 years in 2004. Over the next decade the combined effect produces only a very small improvement, particularly because the heaviest smoking cohorts of American females are still in or approaching the ages of greatest vulnerability to death. Preston projected that the obesity effect will dominate the smoking effect at ages younger than 60, while above age 60 the effect of smoking will be dominant.

Preston and Stokes (2011) indicated that a significant portion of the overall difference in life expectancy between many developed countries and the U.S. is due to a higher level of obesity in the U.S. Note that the estimated difference in life expectancy is quite sensitive to the estimated attributable deaths due to obesity. However, in the sixteen country comparison made by Preston and Stokes, the portion of the difference due to obesity at age 50 (they adjusted mortality results for individuals with higher-than optimal BMI to the lowest-risk BMI for their age and gender using the results of the Prospective Studies Collaboration study) was 42 percent and 67 percent for males and females, respectively. They similarly calculated results based on alternative sets of BMI-mortality relationships (1) from Adams et al (2006), which resulted in corresponding percentages of 42 percent and 21 percent and (2) based of relations determined by Mehta and Chang (2009) with results fairly similar to (1). Differences between these alternative assumptions are due to a higher estimated rate of obesity at younger ages and a higher percent of severe obesity in the U.S. than in the countries compared. Whichever set of estimates turn out to be more accurate, in this study obesity contributes a significant portion of the difference in life expectancy between the U.S. and other developed countries. Other studies, including Preston et al (2012), have attributed more of this difference to smoking, at least during the immediate future.

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Reasons that have been put forth to support the proposition that mortality effects of prevalence, severity, and complications of the obesity epidemic will worsen, together with greater resultant mortality, include:

1. Current estimates are based on past trends when obesity prevalence was smaller;
2. Prevalence of obesity, especially among children, is likely to continue to increase adult levels and duration of obesity;
3. With the onset of obesity arising at younger ages, children and young adults will be obese for a longer period, and thus will tend to experience greater mortality rates as they become older;
4. The significant shift toward more extreme BMIs through all age ranges will continue;
5. Mortality rates from diabetes have risen steadily over the last twenty years as its incidence has increased and are expected to continue to increase as younger cohorts age; and
6. Treatment and management of obesity has by and large been largely unsuccessful.

Thus, due to the significant lags involved, it is possible that the surge in obesity hasn't had enough time for us to observe its cumulative effects in reported mortality studies. On the other hand, it may be that there will be an increasing percent of healthy lives in the overweight and obese BMI categories, which may offset the above trends.

In developing a projection, its use has to be kept in mind. Most of what is described above addresses the issue of the effect of obesity of otherwise healthy adults, thus not considering the portion of the population who are currently ill that is usually excluded from such studies and changes in the composition of the normal weight population segment. In a projection of a total population segment, it is necessary for completeness to consider all significant portions of the relevant population, including those otherwise excluded segments.

3. MORBIDITY

Although the primary objective of this paper is to analyze the effects of obesity on mortality, it would be remiss to not also provide some discussion regarding its relationship with morbidity, in part to emphasize the complex and inter-related nature of factors involved in determining the effects of the state of the human condition, it is important to assess it in a holistic manner, in this case, the health of the human body.

Morbidity can result in both human suffering and adverse financial consequences, including the cost of medical care services, loss of income and productivity, and needed assistance in performing activities of daily living (ADLs). Although the focus on obesity has often been on its adverse effects on mortality, a growing and proper concern is its contribution to direct and indirect health related costs. Although as discussed above, some studies indicate that being overweight may not constitute a significant mortality risk, being overweight is often a stage in becoming obese, and an independent health risk factor. In fact, over the last several decades twin factors of decreased mortality and increased health care costs have occurred simultaneously, in part as a the result of improvements in and aggressive treatment of cardiovascular disease risk factors and other obesity-related health effects. A decrease in expected mortality rates of the obese and the effect of a mortality obesity paradox is likely to be accompanied by and result in larger health care costs.

3.1. Health Care Costs

Increasing health care costs due to obesity is hardly new news, as the Surgeon General in 2001 indicated that morbidity due to obesity in the United States may be as great a problem as poverty, smoking, or drinking. Effective control of obesity is needed for there to be hope of controlling health care costs. Three factors contribute to health care costs associated with obesity: (1) the increase in the number of obese; (2) the aging of the obese population, including the effect of improved mortality experience that will increase co-morbidities of this population that on a combined basis will increase health care costs; (3) the increase in treatment costs for obesity-related conditions and (4) the more aggressive treatment of various medical conditions of the overweight and obese. Certain studies have asserted that health care costs for the obese may be equivalent to costs for those 20 to 25 years older of normal weight.

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The overall cost of obesity includes (1) direct health care costs, including preventive, diagnostic, and treatment services, bariatric surgery, and weight reduction and dieting products and services; (2) indirect costs that include wages and value of labor lost, decreased productivity, increased absenteeism, increased insurance premiums, non-medical support cost associated with the increase in the morbidly obese, and future lost earnings caused by premature death and disability; and (3) intangible costs, such as adverse effects on quality of life, psychological effects, and public costs. Indirect costs are also affected by the obesity and health of family members, for example as time off needed as a result of children, spouses/partners, and the elderly who are overweight and obese that demand incrementally greater personal support, care, and attention.

Most health care cost studies have attributed greater health care costs of the obese to the greater use of prescription drugs (several studies have indicated that the obese use between 70 percent and 80 percent more drug dispenses or costs, especially those relating to cardiovascular, asthma, ulcer, diabetes, thyroid, and analgesic drugs) and primary physician visits. Some studies have also found that the obese use more emergency department/outpatient clinic visits, specialty care clinics, inpatient visits, and diagnostic services.

Physical disabilities resulting from obesity that lead to increased health care costs include: (1) skeletal and joint problems such as orthopedic disorders and carpal tunnel syndrome; (2) respiratory problems such as sleep apnea, respiratory muscle inefficiency, and decreased functional reserve capacity; and (3) increased number of cesarean deliveries. In addition, these costs are associated with more intensive treatment of medical conditions and are consequentially associated with corresponding increases in risk-factors. To the extent that there is an obesity paradox, more people will be subject to the burden of these medical conditions for a longer period of time, resulting in greater lifetime health care costs and disability risks and costs because of a protracted life course.

Approaches taken to estimate costs attributable to obesity usually evaluate health care costs by either: (1) individual associated medical conditions, such as diabetes, cardiovascular disease, and cancer; (2) source of spending, such as by type of prescription drug; and (3) funding source, in the United States including Medicare, Medicaid, private health insurance and out-of-pocket expenditures. However, due to co-morbid conditions that often accompany obesity, it can be difficult to attribute most health care costs and disabilities specifically to obesity, thus making it more difficult to assess the amount of these costs than it is to identify obesity-attributable mortality.

Finkelstein and Brown (2008) showed that those who receive bariatric surgery incur annual medical costs that are $1,680 greater than those for obese who do not use this surgery. Chang et al (2012) indicated that total mean health care costs for those who had bariatric surgery were $9,326 pre-surgery, $13,400 during the first year after surgery, and $13,644 annually for at least six years after surgery, while Maciejewski and Arterburn (2013) in a study of health plan experience also found that total health care costs were greater for bariatric patients in the second and third years following surgery, leveling off thereafter, while those who underwent laparoscopic surgery had lower costs in the first few years after surgery but similar costs thereafter. Thus, even if bariatric surgery enhances mortality, mobility, and quality of life, it does not appear to decrease health care costs.

Certain recent studies have found a significant nonlinear relationship between health care expenditures and degree of obesity. Notably, Cawley and Meyerhoefer (2010), based on the 2000-05 wave of the Medical Expenditure Panel Survey (MEPS), found very heavy additional costs associated with the morbidly obese, the fastest-growing category of obesity, resulting from greater use of physician services, outpatient and inpatient visits, and prescription drugs. Note that since MEPS does not survey those who are institutionalized (e.g., in hospitals or nursing homes), their cost estimates may be somewhat low.

Obesity may have a greater effect on health care costs than does smoking, because of its effect on heart disease, hypertension, and diabetes, all of which tend to be chronic in nature with long-term drug and treatment regimens. In contrast, smoking has its strongest effects on lung cancer and respiratory disease that, while costly, generally have lower frequency and lead to death more quickly than do obesity-related conditions.

Unlike mortality, health care costs for the obese over age 65 (or those who were obese for a long period prior to age 65) are significantly higher than those of normal BMI of the same age. For example, Daviglus et al (2004) reported fee-for-service

Medicare charges for the severely obese were about 95 percent greater, for the obese class 1 50 percent greater, and for the overweight 20 percent greater than those who are BMI-normal.

Although earlier studies (in the 1990s) indicated that obesity-related health care expenditures were between 5 percent and 7 percent of annual U.S. health care expenditures, estimates based on two more recent studies are 9.1 percent (Finkelstein et al (2009)), especially as a result of pharmaceutical services, or 16.5 percent (Cawley and Meyerhoefer (2010) who attributed a significant portion to the extremely obese) of total health care costs that can be directly attributed to those overweight or obese. Finkelstein estimated that the obese had 42 percent greater per capita medical spending than those in the normal BMI category (45.5 percent increased inpatient costs, 26.9 percent more physician and emergency room visits, and 80.4 percent more prescription medicine costs). Given that 2013's total American health care expenditures were about $3.0 trillion, obesity is very costly whichever these additional percentages are accurate. It has also been estimated that up to a third of the increase in overall health care costs as a percent of U.S. gross domestic product (GDP) over the last two decades has been due to the increased prevalence of obesity.

According to Weir and Encinosa (2012), obesity accounted for 2 percent of hospital admissions of children 1-17 years old in 2009, with the rate of hospitalization doubling between 2000 and 2009, with a mean length of stay of 5.3 days compared with 3.8 days where obesity was not a co-existing condition. This means that stays where obesity is mentioned cost about 24 percent greater than stays where no obesity is mentioned, with the most common principal diagnosis being mood disorders. Weiss and Elixhauser (2012) indicated that in 2009 obese adults accounted for more than 9 percent of hospital stays with a primary or secondary diagnosis of obesity (up from 5.3 percent in 2004), for which 95 percent of stays being for obesity as a secondary diagnosis, with the most common primary diagnosis being osteoarthritis. The overwhelming percentage of stays for which the primary reason was identified as being due to obesity was for bariatric surgery.

This contrasts with a relatively small four percent of total health care costs in Canada being associated with obesity, estimated to be C$71 billion (in 2006C$) in Janssen et al (2009), which indicated that there were 14.7 percent and 18.2 percent greater physician costs for males and females, respectively, and were 5.3 percent greater for those aged 18-39, 7.0 percent greater for those 40-59 and 28.3 percent greater at ages 60 and older.

3.2. Disability

An important adverse effect of the increase in moderate obesity has been on disability-free life expectancy. Excess weight of a person increases that person's rate of disability and work limitations, as well as the activities of daily living for the elderly. It should be noted that disability is more complex than mortality to study because of different definitions of disability that have been used, the exclusion of those more seriously disabled in institutions or at home, co-morbidities, differing tolerances of adverse medical conditions, demand differentials by type of job, economic conditions, and inaccuracies of self-reported responses.

According to NHIS findings, obesity accounts for about half of the increased rate of disability for those aged 18–29, a quarter for ages 30–39, and a tenth for ages 40–49. Mehta and Chang (2011) indicated that improved survival among the obese may come at the cost of increasing levels of disability, with obesity's association with disability having been increased among older adults between 1988-94 and 1999-2004 because people are living longer with obesity, with resulting shortening of disability-free life expectancies. In addition, Mehta and Chang (2009) indicate that early onset of chronic illnesses and disability is increasingly evident. The fastest growing causes of disability for the non-elderly are diabetes and musculoskeletal problems, conditions often associated with obesity.

Armour et al (2012) indicated that of the 25 percent of adults who were found to be obese in the NHIS, 35.2 percent of obese males reported a disability, compared with 26.7 percent of BMI-normal males, while 46.9 percent of BMI-obese females reported a disability compared with 26.8 percent of BMI-normal females. Body movement difficulties were the most common type of disability among the male BMI-obese (32.9 percent of the BMI-obese compared with 24.1 percent of those of BMI-normal), with 45.0 percent and 25.1 percent for females, respectively. Movement difficulty was 1.5 times more frequent for BMI-obese males and twice as great for BMI-obese females. Among males, 13.4 percent of BMI-normal males

had a complex-activity limitation compared with 11.6 percent of BMI-normal, while the corresponding percentages were 11.6 percent and 23.0 percent for females, with work limitations being the most common example.

Tucker and Friedman (1998) found that obese employees are 1.74 times more likely to experience significantly more work-absences (defined as being seven or more absences due to illness in a six-month period) and 1.61 times more likely to experience a moderate level of more work-absences (three to six absences), respectively, than their leaner counterparts. Obesity work penalties can include job-related bias through weight-based discrimination, including a tendency to be less likely to be hired and if hired, to not receive equal wages or promotions, voluntary sorting into jobs with lower pay, lower self-esteem or depression, as well as poorer health. Work-related disability can also be manifested in a greater use of sick leave, early retirement, and disability pensions. While such a wage penalty can exist because of the stigma of being obese, it can also arise from real differences, such as from reduced productivity caused by ill-health.

Bhattacharya and Bundorf (2009) found that incremental health care and productivity costs associated with obesity are often passed through to obese workers in the form of lower cash wages. They estimated that obese males earn $1.21 an hour less than non-obese males, while obese females earn $1.66 less than non-obese females, rates that can increase with age. Differences arise by gender, race, and degree of obesity, with limited penalties for black or Hispanic females who are BMI-overweight and with inconsistent differences for males. In some occupations in which interpersonal skills involving human interaction (with customers, employers and fellow-workers) are important, a wage penalty exists, especially for females. In fact, how adolescents look in regard to body fat can influence their labor market success. The wage penalty in firms providing health insurance, one element of the cost of employment, was higher by an average of $2.64 an hour.

Majer et al (2011) reported results of the European Community Household Panel (an annual survey of nine countries with an average follow-up period between 2.5 and 2.9 years, with an average age of survey participants in their middle 40s) that also indicated a significant association between disability and BMI. This survey indicated that for those who had never smoked, hazard ratios of disability incidence for overweight males were 1.00, for overweight females 1.28, for total obese males 1.35, and total obese females 1.87, while for class 1 obese and class 2+ males and females were 1.60 and 1.91, respectively. Regarding recovery from a disability episode, the hazard ratio for overweight males was 1.04, for overweight females was 1.03, with 1.01 for total obese males and 0.92 total obese females (for which class 1 obese was 1.01 and for class 2+ was 0.79). They concluded that obesity is more highly associated with disability than with mortality, which contrasts with daily smoking, which is associated with mortality more than with disability.

Wada and Tekin (2010) found that greater body fat reduces wages, while greater fat-free mass (muscle) tends to increase wages, especially in males. Cawley and Burkhauser (2006) found that greater total body fat is associated with a higher risk of adverse disability outcomes, but greater fat-free mass is associated with a lower risk of such outcomes. Also, they found that muscle is more protective against work limitations for males than for females. Tomé (2010) indicated that being an underweight male might also result in a wage penalty in some occupations.

Lakdawalla et al (2004) found that the rate of more severe personal care limitations was 50 percent greater for the obese. They speculated that (1) the obese have co-morbidities that tend to contribute to more intense disabilities, (2) lifesaving medical techniques have resulted in a greater number of disabled who might otherwise have died, and (3) less-than-average wages and wage growth for the obese can provide them greater incentive to obtain disability insurance and present disability insurance claims.

After the U.S. Congress adopted amendments to the Americans with Disability Act (ADA) in 2008, the U.S. Equal Employment Opportunity Commission (EEOC) took the position that obesity, at least in its extreme form, is considered a disability. As a result, American employers are subject to enforcement of ADA employment rules by the EEOC, although to date enforcement appears to have been inconsistent.

Kunst et al (2009) found that, based on experience gathered in the Rotterdam Study (about 6,000 participants followed up for 15 years for mortality and six years for disability), although neither BMI nor WC was related to mortality at either overweight or obese BMIs, both of these measures were related to disability incidence and recovery. For example, the hazard ratio for disability incidence for the BMI-overweight was 1.33 and for the class 1 obese was 2.03. They were

correspondingly negatively related to disability recoveries. Both factors lead to an increase of healthy years lost due to disability.

Tommy et al (2004) studied 19,000 Finns between the ages of 20 and 92 for 15 years. They found significantly higher levels of disability for all categories of overweight and obesity, both for ages less than 65 and older than 65, and for each gender. This held true for work disability, coronary heart disease, need for long-term medication and all-cause mortality. For example, for those in the younger group, hazard ratios for work disability were 1.1 and 1.7 for overweight and obese, with corresponding hazard ratios of 1.2 and 1.6 for coronary heart disease, 1.5 and 2.5 for long-term medication needs, and 1.0 and 1.3 for all-cause mortality (1.0 and 1.6 for those aged 65 and older).

In a representative study of 7,678 Canadian workers, Janssen et al (2011) found that obese workers were more likely to report an occupational injury with a hazard ratio of 1.49 and a serious injury with a hazard ratio of 1.49. There was a more pronounced difference for sprains and strains (1.80), injuries to lower limbs (2.14) or torso (2.36), injuries due to falls (2.10), and overexertion (2.08), with obese workers who were female, over age 40 and in sedentary occupations especially prone to excess injuries. The BMI-overweight did not appear to experience excess injuries relative to those that were BMI-normal.

Seemingly in parallel to what may be a secular decline in relative mortality of the obese, Alley and Chang (2009) reported a corresponding increase in relative disability among the obese. The increase in obesity over the last several decades has and likely will for some time increase the rate of disability by 20 percent for those between ages 50 and 69 due to increasing ADL limitations, which may also lead to greater disability income and workers' compensation costs.

Several studies have found that dementia is associated with high BMI and visceral adiposity. However, Power et al (2011) found in a Perth Australia cohort of older males that the BMI-obese was not more likely to have dementia than those in the BMI-normal category, although those in the BMI-overweight category had a lower hazard ratio (0.82) and a J shape association was found. Those with a WtoH ratio greater than 0.9 had a higher hazard ratio with those with a WtoH ratio less than 0.9, with no obvious association found with WC.

According to Lang (2009), British males older than 65 (in the English Longitudinal Study of Aging) had an ADL difficulty hazard ratio of 1.99 and a functional impairment hazard ratio of 1.51, both inclusive and exclusive of those obese who recently lost weight, smoked, or self-reported poor health, as well as a mortality hazard ratio of 0.99. Similar results were observed in the American Asset and Health Dynamics Among the Oldest Old survey of males and females over 70. Backholer (2012) found an inverse relationship over a twelve study meta-analysis, indicating ADL limitation hazard ratios of 1.04 for overweight, 1.16 for the class 1 obese, and 1.76 for the class 2+ obese.

Studies differ as to what level of BMI that ADLs begin to deteriorate. For example, several studies have indicated a protective value for osteoporosis for those overweight and obese. Andrade et al (2013) found in a study of Brazilian older adults that not only did the obese experience more ADL limitations and lower rates of recovery, but those who gained weight experienced a greater incidence of ADL limitations, decreased physical functions, and role limitations. They also found that being overweight was not associated with a higher level of ADL incidence. Those who unintentionally lost weight faced higher rates of functional decline regardless of initial BMI level, while intentional weight loss was not associated with a functional decline. There was no mortality differential by BMI at all in this study of older adults; however, a large majority did not take part in any physical activity, being fearful of being robbed or of falling in part because of sidewalk defects.

There also appears to be an increased likelihood of falling and sustaining fall-related injuries and consequential ill health, as well as developing an increased loss of ability to perform ADLs, all increasing with an increase in obesity class.

4. WHAT CAN BE DONE

Because there are many contributors to today's high prevalence of obesity, a multi-faceted approach to the individual and community will be needed to effectively address this problem. Although its causes differ by individual, family, culture, and location, focus should not solely be on body weight—just as importantly it should include general health and fitness, by means of contributing behaviors, primarily nutrition and physical activity.

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Development, implementation and maintenance of effective and convenient long-term weight management programs, including a sustainable nutritional program, enhanced fitness, and tailored information campaigns across a wide range of the population will remain a challenge, as human behavior is quite resistant to required inconveniences. For example, in the U.S., some early results of expanded transparency in nutritional labeling and calorie counts in restaurants have not shown this requirement to have had a measurable effect on overall calorie reduction—those who tend to be most responsive to such information are those who are already healthy or those in a high-socioeconomic category who are calorie conscious in the first place. Obesity and physical activity are related in a vicious cycle, as the more weight one carries, the less physical activity is practical and as physical fitness declines, more weight is put on. Although the use of legal anti-obesity drugs has not seen a great deal of success, together with lifestyle interventions, Yanovski and Yanovski (2014) have shown that such comprehensive programs can have some success through a one year period.

Recently, edacity behavior has been explained in terms of addiction to food, meaning that the already obese tend to continue to over-eat, possibly as a result of reduced levels of dopamine, a signaling molecule with a role to generate pleasure and driving motivation, as usually the more pleasing the food, the more dopamine is produced (at least based upon mice experiments). Or a mechanism in the neostratium, a part of the brain that produces intense motivation to overeat tasty food; over time a glut of sweets seems to change the brain's reaction and people get conditioned just by the sight of food. The only conclusion that most studies of weight-reduction and diet effectiveness agree on is that adherence to an espoused diet over a long period is quite difficult, and that there is no single diet that works for everyone. They are rarely effective over the long-term, but it is rarely appropriate to simply blame obesity on a lack of personal self-control.

Energy-dense food and portion size (in some cases driven by economics and the psychological need to polish off what is served) contribute to consumption of excessive nutritional inputs. An example of the economics referred to is that many food-related entities aim to recover their per serving expenses in the price for their smaller serving, with relatively small price increments between serving sizes corresponding with the relatively small marginal cost for the increased size, providing perceived value to the consumer for moving "up" the serving size. In addition, putting on excess weight is often accompanied by fat cells that are also getting fatter, sometimes doubling in capacity and spreading, which, after significant time can become deadly. This is especially the case if enough adiposity tissue accumulates around the waist and also contains immune-system cells called macrophages that, although they normally work to remove pathogens and dead cells, when secreted by fat cells they can inflame arteries and trigger cancers, which can occur if overeating occurs over long periods of time and in large enough doses. Those who are obese appear to have fewer dopamine receptors, so get less pleasure from eating, which can then lead to even more eating. But these same mechanisms, some of which are driven by one's genes' propensity to get fat, also hinder the ability to lose weight – so a biological disposition of a certain size will trigger an effort to regain any pounds lost.

Firms in the food, drinks, and restaurant business have an inherent potential conflict of interest dealing with this issue, in part because healthier foods tend to have smaller margins than unhealthy ones, due to relative demand and costs. As long as their customers want certain types of food, it is difficult not to sell or to discourage their sale. Nevertheless, at the same time they want to be good citizens and to offer and promote a healthy diet. The best solution is to provide healthy food that is affordable and also tastes great – unfortunately, in some areas that is difficult to achieve.

The growth in obesity has taken place in spite of the fact that in general, obesity (and especially morbid obesity) is looked upon by large segments of society with disfavor. Unfortunately, as the percent of society that is obese has increased, its acceptability (at least for those not in the extreme category) has also increased. Once obesity is present, the cultural, behavioral and physiological feedback loops involved can make it extremely difficult to reverse a weight gain over the long-term.

Adults with greater adiposity have a smaller expected weight loss for the same change in energy intake – to reach a steady state normal weight thus takes longer than those with less body fat. Since it is so difficult to sustain a voluntary weight reduction, it may make more sense to focus efforts at reducing new onsets of obesity, whether in children or adults who have not reached that point in their personal development.

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Many of the trends that have contributed to the global growth in obesity are thus difficult to reverse. In fact the world has not seen many successful comprehensive strategies to reverse such trends (although note the recently reported declines in obesity in American children mentioned above, albeit still small compared to the significant increases in obesity in this population segment over the prior two decades). Because the underlying causes differ considerably between population groups, a one-size-fits-all solution cannot address the issues associated with all of groups.

Take cooking for example – even though a record number of cook-books and recipes are now bought and downloaded, very often cooking has reverted to heating cans of soup, micro-waving ready-to-eat meals and frozen pizzas, or opening a bag of mixed lettuce leaves. Over the last several decades fewer people have been cooking their evening meal (in the U.S., reportedly the most popular American meal has been a sandwich and a fizzy drink). Industrial and fast food almost always trade larger amounts of sugar, salt, and fat for quality ingredients – leading indicators of increased body weight. And even with increased calorie labeling and increased advertising of healthier menus by fast food restaurant firms, people have not appreciably changed their eating habits (and many of those who have changed behavior as a result of this data are probably those who were already lean and fit). Efforts will continue; for example, in early 2014 the U.S. Food and Drug Administration announced it will require disclosure of added sugars to current food labeling. "Traffic-light" labels, nutritional/physical activity incentives, reduced advertising to and expanded activity programs and effective nutritional programs for children (for example, in the U.S. the Healthy Hunger-Free Kids Act of 2010 that goes into effect in the fall of 2014 requires school vending machines, stores and a la carte lunch menus to provide only healthful foods), and even the targeted taxes in Mexico are approaches that might be taken, although each has its limitations.

While it is great to see a growth in health clubs, unfortunately most of those who take advantage of them are often the people who are already physically fit. Carrot or stick approaches, such as taxation, banning certain foods, or other penalties for "bad" or excess amount of food or lack of activity, facilitating leisure exercise, community or employer wellness programs, or insurance discounts have had a mixed track record, and can usually be subverted by substituting the wrong type of food or leisure at alternative locations or by ignoring the information provided. Although the U.S. Affordable Care Act does not permit refusal of insurance due to pre-existing conditions, it does permit employers to charge obese employees 30 to 50 percent more for health insurance unless the employee participates in a qualified wellness program of health promotion and disease prevention (although some research has indicated that a premium adjustment may be less effective than incentive programs with immediate and frequent feedback).

Different views exist regarding the basis for and role of public policy in this area that influence the degree and type of government involvement. One view would base anti-obesity policy on efforts to empower people who will help themselves, while another view claims that since much of the higher health care costs of the obese are absorbed by the non-obese population, contributions to obesity become a public good, justifying government intervention to change eating and activity habits. Government nudges have included attempts to protect children from making decisions that will harm them later in life.

Both government regulation and business self-regulation has become more active in recent years. These range from Michelle Obama's "Let's Move" program that includes recruitment of Sesame Street puppets to promote fruit and vegetables rather than sugary and fatty foods, introduction of restrictions and reductions in advertisement to children in many countries, to a reduction in the use of sugar by American cereal-makers that also have added whole grains based on consumer pressure. School food programs may help—for example, changes were made to the U.S. School Lunch Program during the 2012-2013 school year requiring students to select either a half cup of fruit or vegetables, increasing the use of whole grains, reducing serving sizes, and eliminating the availability of whole and 2 per cent milk, although its introduction has introduced more waste (one source indicates roughly 60 to 75 percent of vegetables and 40 percent of fruit are being discarded by students), greater food costs and difficulties in sourcing products. Debate about the substance and effectiveness of these programs will continue, but it is heartening that these efforts are being made.

The future effects on mortality, morbidity, and health care of those who are now and have been overweight and obese for a while, especially the growing population of those morbidly obese, are ignored at a person's own peril. However, in addition to its personal effects, the resulting health care and disability costs are shared by society. It has taken decades of intense government and private efforts to gain modest control over smoking prevalence. Until technology catches up with this growing worldwide phenomena (for instance, by being able to add the appropriate microbes to overcome the lack of

favorable gut bacteria), obesity will remain a significant public policy and personal issue. It will take a long time to win a war against obesity and sedentary living. In a society in which food is plentiful and affordable, and exercise is no longer necessary for immediate survival, only comprehensive long-term approaches and changes in attitude at both an individual and societal level will lead to effective solutions.

LIST OF FIGURES AND TABLES

REFERENCES

Adams, K.F., A. Schatzkin, et al. 2006. Overweight, obesity, and mortality in a large prospective cohort of persons 50 to 71 years old. *The New England Journal of Medicine* 2006; 355(8):763-778.

Alley, D.E. and V.W. Chang. 2007. The changing relationship of obesity and disability, 1988-2004. *Journal of the American Medical Association.* 298 (17): 2020-2027.

Andrade, F.C.D., A.I.N.M. Nazan, M.L. Lebrão, Y.A. de Oliveira Duarte. 2013. The impact of body mass index and weight changes on disability transitions and mortality in Brazilian older adults. *Journal of Aging Research.* 2013, article ID 905094.

Armour, B.S., E. Courtney-Long, V.A. Campbell, H.R. Wethington. Estimating disability prevalence among adults by body mass index: 2003-2009 National Health Interview Study. *Prev Chronic Dis. 2012; 9: 20136.*

Auyeung, T.W., J.S.W. Lee, J. Leung, T. Kwok, P.C. Leung, J. Woo. 2010. Survival in older men may benefit from being slightly overweight and centrally obese – A 5-Year follow-up study in 4,000 older adults using DXA. *Journal of Gerontology Series A: Biological Sciences and Medical Sciences.* (2010) 65A(1): 99-104.

Ärnlöv et al (2009), E. Ingelsson, J. Sundstöm, L. Lind. 2010. Impact of body mass index and the metabolic syndrome on the risk of cardiovascular disease and death in middle-aged men. *Circulation*. 20110;121:230-236.

Bar, J. 2010. The science of childhood obesity. *Health Affairs*. 29. 3(2010): 393-397.

Basu, S., P. Yoffe, N. Hills, R.H. Lustig. 2013. The relationship of sugar to population-level diabetes prevalence: an econometric analysis of repeated cross-sectional data. *PLOS One* Feb 2013: 8(2); 1-8. E57873.

Belloco, R., C. Jia, W. Ye, Y.T. Lagerros. 2010. Effects of physical activity, body mass index, wait-to-hip ratio and waist circumference on total mortality risk in the Swedish National March Cohort. *European Journal of Epidemiology (2010) 25:777-788.*

Berrington de Gonzalez, A., P. Hartge, J.R. Cerhan et al. 2010. Body-mass index and mortality among 1.46 million white adults. *The New England Journal of Medicine* 2010;363:2211-2219. December 2, 2010. Doi: 10.1056/NEJMoa1000367.

Bhattacharya, J., M.K. Bundorf. 2009. The incidence of the healthcare costs of obesity. *Journal of Health Economics.* 2009;28(3):649–658.

Bleich, S.N., et al. 2012. Impact of bariatric surgery on health care utilization and costs among patients with diabetes. *Medical Care.* 50, 58-65. 2012.

Boggs, D.A., L. Rosenberg, Y.C. Cozier, L.A. Wise, P.F. Coogan, E.A. Ruiz-Narvaez, J.R. Palmer. 2011. General and abdominal obesity and risk of death among black women. *The New England Journal of Medicine*. 365:10. 901-908.

Boström, P. J. Wu, M.P. Jedrychowski, et al. 2012. A PGC1-α-dependent myokine that drives brown-fat-like development of white fat and thermogenesis. *Nature*. 2012;481:463-8.

Canoy, D., B.J. Cairns, A. Balkwill, F.L. Wright, J. Green, G. Reeves. V. Bera. 2013. Body mass index and incident coronary heart disease in women: a population-based prospective study. *BMC Medicine*. 2013, 11;87.

Carnethon, M.R., P.J.D. De Chavez, M.L. Biggs, C.E. Lewis, J.S. Pankow, A.G. Bertoni, S.H. Golden, K. Liu, K.J. Mukamal, B. Campbell-Jenkins, A.R. Dyer. 2012. Association of weight status with mortality in adults with incident diabetes. *Journal of the American Medical Association* 2012;308(6): 581-590.

Cawley, J.H. (ed). 2011. The Oxford Handbook of the social science of obesity. Oxford University Press. New York City.

Cawley, J.H., R.V. Burkhauser. 2006. Beyond BMI: the value of more accurate measures of fatness and obesity in social science research. *National Bureau of Economic Research.* Working Paper 12291.

Cawley, J.H., C. Meyerhoefer. 2010. The medical care costs of obesity: an instrumental variables approach. *National Bureau of Economic Research*. Working Paper 16467.

Chang, S-H, L.M. Pollack, G.A. Colditz. 2013. Life years lost associated with obesity-related diseases for U.S. non-smoking adults. *PLOS One*. 8(6): e66550. Doi:10.1371/journal.pone.0066550.

Chang, V.W., D.A. Asch, R.M. Werner. 2010. Quality of care among obese patients. *Journal of the American Medical Association*. April 7, 2010; 303 (13):1274-1281.

Clair, C., N.A. Rigotti, B. Porncala, C.S. Fox, R.B. D'Agostino Sr, M.J. Pencina, J.B. Meigs. 2013. Association of smoking cessation and weight change with cardiovascular disease among adults with and without diabetes. *Journal of the American Medical Association*: 2013;309(10), 1014-1021.

Cohen, S.S., L.B. Signorello, E.L. Cope, J.K. McLaughlin, M.K. Hargreaves, W. Zheng, W.J. Blot. 2012. Obesity and all-cause mortality among black adults and white adults. *American Journal of Epidemiology:* 176 (5): 431-442.

Cohen-Mansfield, J., R. Prach. 2011. Is there a reversal in the effect of obesity on mortality in old age. *Journal of Aging Research*. 2011, article ID 765071.

Colman, G.J., D.M. Dave. 2013. Physical activity and health. *NBER Working Paper Series*. National Bureau of Economic Research. Working Paper 18858. February 2013.

Corrado, M.M., C.H. Kawas, F. Mozafter, A. Paganini-Hill. 2006. Association of body mass index and weight change with all-cause mortality in the elderly. *American Journal of Epidemiology. 163 (10): 938-949.*

Coutinho, T. et al. 2013. Combining body mass index with measures of central obesity in the assessment of mortality in subjects with coronary disease. *Journal of the American College of Cardiology.* V61, No. 5, 2013.

Crespo, C.J. et al. 2001. The relationship of physical activity and body weight with all-cause mortality. *Annals of Epidemiology.* 12 (8), 543-552. November 2002.

Dahl, A.K., E.B. Fauth, M. Ernsth-Bravell, L.B. Hassing, N. Ram, D. Gerstof. 2013. Body mass index, change in body mass index, and survival in old and very old persons. *Journal of the American Geriatrics Society*. 61 (4): 512-518. April 2013.

Daviglus, M.L., K. Liu, L.L. Yan, A. Pirzada, L. Manheim, W. Manning, D.B. Garside, R. Wang, A.R. Dyer, P. Greenland and J. Stamler. 2004. Relation of body mass index in young adulthood and middle age to medicare expenditures in older age. *Journal of the American Medical Association* 292: 2743–2749.

Economist. Special report – Obesity, the big picture. December 15, 2012. 1-16.

Ekelund, U., J. Luan, L.B. Sherar, D.W. Esliger, P. Griew, A. Cooper. 2012. Moderate to vigorous physical activity and sedentary time and cardiometabolic risk factors in children and adolescents. *Journal of the American Medical Association*: 2012;307(7), 704-712.

Farrell, S.W., S.J. Fitzgerald, P.A. McAuley, C.E. Barlow. 2010. Cardiovascular fitness, adiposity, and all-cause mortality in women. *Medical Science and Sports Exercise*. 2010 Nov; 42(11): 2006-2012. Doi: 10.1249/MSS.ObO13e3181df12bf.

Fasells, C., M. Doumas, D. Panagiotakos, R. Kheirbek, L. Korshak, A. Manolis, A. Pittaras, C. Tsioufis, V. Papademetriou, R. Fletcher, P. Kokkinos. 2012. Body mass index, exercise capacity, and mortality risk in male veterans with hypertension. *American Journal of Hypertension*. 2012 April; 25(4): 444-450. Doi: 10.1038/ajh.2011.242.

Finkelstein, E.A., D.S. Brown. 2008. Examining the economics of bariatric surgery. *The American Journal of Managed Care.* 14(9): 561-562.

Finkelstein, E.A., J.G. Trogdon, J.W. Cohen and W. Dietz. 2009. Annual medical spending attributable to obesity: payer- and service-specific estimates. *Health Affairs*. Doi: 10.1377/hlthaff.28.5.w822.

Finucane, M.M., G.A. Stevens, M.J. Cowan, G. Danaei, J.K. Lin, C.J. Paciorek, G.M. Singh, H.R. Gutierrez, Y. Lu, A.N. Bahalim, F. Farzadfar, L.M. Riley, M. Ezzati. 2011. National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. *Lancet*. 377; 9765; 557-567. February 12, 2011. Doi:10.1016/S01460-6736(10)62037-5.

Flegal, K.M., K. Brian, H. Orpana, B.I. Graubard. 2013. Association of all-cause mortality with overweight and obesity using standard body mass index categories, a systematic review and meta-analysis. *Journal of the American Medical Association*: 309(1):71-82. Doi:10.1001/jama.2012.113905. http://jama.jamanetwork.com/article.aspx?articleid=1555137.

Flegal, K.M., M.D. Carroll, B.K. Kit, C.L. Ogden. 2012. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. *Journal of the American Medical Association*. 2012;307(5):491-497. doi:10.1001/jama.2012.39.

Flegal, K.M., J.A. Shepherd, A.C. Looker, B.I. Graubard, L.G. Borrud, C.L. Ogden, T.B. Harris, J.E. Everhart, N. Schenker. 2009. Comparisons of percentage body fat, body mass index, waist circumference, and waist-stature ratio in adults. *American Journal of Clinical Nutrition*. 2009;89:500-508.

Flicker, L., K.A. McCaul, G.J. Hankey, K. Jamrozik, W.J. Brown, J.E. Byles, O.P. Almeida. 2011. Body mass index and survival in men and women aged 70 to 75. *Journal of American Geriatric Society*. 2010 Feb;58(2):234-41. Doi: 10.1111/j.1532-5415.2009.02677.x.

Fogelholm, M. 2010. Physical activity, fitness and fatness: relations to mortality, morbidity and disease risk factors. A systematic review. *Obesity Review*. 2010 Mar; 11(3): 202-221. Doi: 10.1111/j.1467-789X.2009.00653.x.

Fontaine, K.R., R. McCubrey, T. Mehta, N.M. Pajewski, S.W. Keith, S.S. Bangalore, C.J. Crespo, D.B. Allison. 2011. Body mass index and mortality rate among Hispanic adults: a pooled analysis of multiple epidemiologic data sets. *International Journal of Obesity* 36, 1121-1126 (August 2012).

Frederick, C.B., K. Snellman, R.D. Putnam. 2013. Increasing socioeconomic disparities in adolescent obesity. *PNAS.* Doi/10.1073/pnas.1321355110.

Gelber, R.P., T. Kurth, J.E. Manson, J.E. Buring, J.M. Gaziano. 2007. Body mass index and mortality in men: evaluating the shape of the association. *International Journal of Obesity* 31, 1240-1247 (2007).

Goel K. et al. 2011. Combined effect of cardiorespiratory fitness and adiposity on mortality patients with coronary artery disease. *American Heart Journal*. 2011 March, 161(3) 590-597. Doi: 10.1016/j.ahj.so10.12.012.

Gómez-Ambrosi, J., C. Silva, J.C. Galofré, J. Escalada, S. Santos, M.J. Gil, V. Valenti, F. Rotellar, B. Ramírez, J. Salvador, G. Frühbeck. 2011. Body adiposity and type 2 diabetes: increased risk with a high body fat percentage even having a normal BMI. *Obesity*. 19 (7); 1439-1444.

Greenberg J.A. 2013. Obesity and early mortality in the United States. *Obesity*. 2013 Feb; 21(2):405-12. Doi: 10.1002/oby.20023.

Greenberg J.A. 2013. The obesity paradox in the US population. *American Journal of Clinical Nutrition*. May 2013. Doi: 10.3945/ajcn.

Greenberg J.A., K. Fontaine, D.B. Allison. 2007. Putative biases in estimating mortality attributable to obesity in the U.S. population. *International Journal of Obesity.* 2007: 31, 1449-1455.

Gregg, E.W., Y.J. Cheng, B.L. Cadwell, et al. 2005. Secular trends in cardiovascular disease risk factors according to body mass index in US adults. *Journal of the American Medical Association*: 293(15):1868-1874.

Gutterman, S. 2011. Obesity—status and effects. *Living to 100 Symposium Monograph*. Society of Actuaries. http://www.soa.org/library/monographs/life/living-to-100/2011/2011-toc.aspx.

Heiat, A., V. Vaccarino, H.M Krumholz. 2001. An evidenced based assessment of federal guidelines for overweight and obesity as they apply to elderly persons. *Archives of Internal Medicine*. 161: 1194-1203.

Heir, T., J. Erikssen, L. Sandvik. Overweight as predictor of long-term mortality among healthy, middle-aged men: a prospective cohort study. *Preventive Medicine*. 52 (3-4):23-226.

Hotchkiss, J.W., Leyland, A.H. 2011. The relationship between body size and mortality in the linked Scottish Health Surveys: cross-sectional surveys with follow-up. *International Journal of Obesity*. 2011. 35, 838-851.

Hu, G. J. Tuomilehto, K. Silventoinen, N.C. Barengo, M. Peltonen, P. Jousilahti. 2009. The effects of physical activity and body mass index on cardiovascular, cancer and all-cause mortality among 47,212 Finnish men and women. *International Journal of Obesity*. 2005. 29, 894-902.

Ioachimescu, A.G., D.M. Brennan, B.M. Hoar, B.J. Hoogwerf. 2010. The lipid accumulation product and all-cause mortality in patients at high cardiovascular risk: a preCIS database study. *Obesity*. 18 (9): 1836-1844.

Jacobs, E.J., C.C. Newton, Y. Wang, A.V. Patel, M.L. McCullough, P.T. Campell, M.J. Thun, S.M. Gapstur. 2010. Waist circumference and all-cause mortality in a large US cohort. *Archives of Internal Medicine*. 170 (15), August 23, 2010.

Jain, M.G., A.B. Miller, T.E. Ronan et al. 2005. Body mass index and mortality in women, follow-up of the Canadian National Breast Screening Study cohort. *International Journal of Obesity.* 2005; 29: 792-797.

Janssen, I., E. Bacon. 2008. Effect of current and midlife obesity status on mortality risk in the elderly. *Obesity*. 16 (9) 2008.

Janssen, I., E. Bacon, W. Pickett. 2011. Obesity and its relationship with occupational injury in the Canadian workforce. *Journal of Obesity*. 2011:531403. Doi: 10.1155/2011/531403.

Janssen, I., A.E. Mark. 2007. Elevated body mass index and mortality risk in the elderly. *Obesity Reviews*. 8(1), 4a:59.

Jee. S.H., J.W. Sull, J. Park, S-Y Lee, H. Ohrr, E. Guallar, J.M. Samet. 2006. Body-mass index and mortality in Korean men and women. *The New England Journal of Medicine*. 2006;355:8:779-787.

Katzmarzyk, P.T., C.L. Craig, C. Bouchard. 2001. Underweight, overweight and obesity: relationship mortality in the 13-year follow-up of the Canada Fitness Survey. *Journal of Clinical Epidemiology 2001; 54: 916-920.*

Katzmarzyk, P.T., B.A. Reeder, S. Elliott, M.R. Joffres, P. Pahwa, K.D. Raine, S.A. Kirkland, G. Paradis. 2012. Body mass index and risk of cardiovascular disease, cancer and all-cause mortality. *Canadian Journal of Public Health*. 103(2): 147-151. Mar-Apr 2012.

Kenchaiah, S., et al. 2002. Obesity and the risk of heart failure. *The New England Journal of Medicine*. 347 (5) August 1, 2002;305-313.

Kodama, S., et al. 2009. Cardiorespiratory fitness as a quantitative predictor of all-cause mortality and cardiovascular events in healthy men and women. *Journal of the American Medical Association*. 2009;301(19):2024-2035.

Kokkinos, P., J. Myers, C. Faselis, M. Doumas, R. Kheirbek, E. Nylen. 2012. BMI-mortality paradox and fitness in African American and Caucasian men with type 2 diabetes. 2012 May; 35(5): 1021-2017. Doi 10.2337/dc11-24407.

Koster, A., T.B. Harris, S.C. Moore, A. Schatzkin, A.R. Hollenbeck, J.M. Van Eijk, M.F. Leitzmann. 2009. Obesity, exercise and mortality. *American Journal of Epidemiology.* 2009: 169(11):1344-1351.

Krishnan, S., L. Rosenberg, L. Djoussé, L.A. Cupples, J.R. Palmer. Overall and central obesity and risk of type 2 diabetes in U.S. black women. *Obesity*. 15(7) July 2007. 1860-1866.

Lackdawalla, D.N., J. Bhattacharya and D.P. Goldman. 2004. Are the young becoming more disabled? *Health Affairs* 23(1): 168-176.

Lahmann, P.H., L. Lissner, B. Gullberg, G. Bergland. 2002. A prospective study of adiposity and all-cause mortality: the Malmö Diet and Cancer Study. *Obesity Research*. 2002: 10 (5) May 2002: 361-369.

Lang, I.A., D.J. Llewellyn, K. Alexander, D. Melzer. 2008. Obesity, physical function and mortality in older adults. *Journal of the American Geriatric* Society. 56: 1474-1478.

Lavie, C.L., R.V. Milani, H.O. Ventura. 2009. Obesity and cardiovascular disease, risk factor, paradox and impact of weight loss. *Journal of the American College of Cardiology*. V53, No. 21, 2009.

Lavie, C.L., A. De Schutter, D.A. Patel, R.V. Milani. Does fitness completely explain the obesity paradox? *American Heart Journal*. 2013 July;166(1);1-3. Doi.10.1016/j.jhj.2013.03.016.

Li, S., J.H. Zhao, J. Luan, U. Ekelund, R.N. Luben, K-Y Khaw, N.J. Wareham, R.J.F. Loos. 2010. Physical activity attentuate the genetic predisposition to obesity in 20,000 men and women from EPIC-Norfolk Prospective Population Study. *PLoS Medicine*. 7(8) 1-9. E1000332.

Lisko, I., K. Tiainen, S. Stenholm, T. Luukkaala, A. Hervonen, M. Jylha. 2011. Body mass index, waist circumference, and waist-to-hip ratio as predictors of mortality in nonagenarians: the Vitality 90+ study. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*. 66A (1):1244-1256.

Liu, C., J.K. Elmquist. 2013. Tipping the scales early: probing the long-term effects of obesity. *The Journal of Clinical Investigation*. November 2012, vol 122, No. 11, 3840-3842.

Ma. J, W.D. Flanders, E.M. Ward, A. Jemal. 2011. Body mass index in young adulthood and premature death: analyses of the US National Health Interview Survey linked mortality files. *American Journal of Epidemiology.* 2011; 174 (8): 934-944.

Maciejewski, M.L. and D.E. Arterburn. 2013. Cost-effectiveness of bariatric surgery. *The JAMA Network: Clinical Review & Education.* V310(7):742-743.

Majer, I.M., W.J. Nusselder, J.P. Mackenback, A.E. Kunst. 2011. Life expectancy and life expectancy with disability of normal weight, overweight, and obese smokers and nonsmokers in Europe. *Obesity*. 19 (7) July 2011.

Masters, R.K., D.A. Powers, B.G. Link. 2013. Obesity and US mortality risk over the adult life course. *American Journal of Epidemiology*. March 1, 2013: 177(5):431-442.

Masters, R.K., E.N. Reither, D.A. Powers, C. Yang, A.E. Burger, B.G. Link. 2013. The impact of obesity on US mortality levels: the importance of age and cohort factors in population estimates. *American Journal of Public Health*. ePub August 15, 2013.

McAuley, P.A., E.G. Artero, X. Sui, et al. 2012. The obesity paradox, cardiorespiratory fitness, and coronary heart disease. *Mayo Clinic Proceedings*. 2012; 87: 443-451.

McAuley, P.A., S.N. Blair. 2011. Obesity paradoxes. *Journal of Sports Sciences*. May 2011; 29(8): 773-782.

McAuley, P.A., P.F. Kokkinos, R.B. Oliveira, B.T. Emerson, J.N. Myers. 2010. Obesity paradox and cardiorespiratory fitness in 12,417 male veterans aged 40 to 70 years. *Mayo Clinic Proceedings*. February 2010: 85(2):115-121.

McAuley, P.A., J. Pittsley, J. Myers, J. Abella, V.F. Froelicher. 2009. Fitness and fatness as mortality predictors in healthy older men: the Veterans Exercise Testing Study. *Journal of Gerontology. 2009 June; 64(6) 695-699. Doi: 10.1093/Gerona/gln039.*

McGee, D.L. 2005. Body mass index and mortality: a meta-analysis based on person-level data from twenty-six observational studies. *Annals of Epidemiology.* 2005 Feb; 15(2):87-97.

Mehta, N.K., V.W. Chang. 2009. Mortality attributable to obesity among middle-aged adults in the United States. *Demography*. 46(4), November 2009:851-872.

Mehta, N.K., V.W. Chang. 2011. Secular declines in the association between obesity and mortality in the United States. *Population Dev Rev. 2011 September; 37(3):435-451.*

Midthjell, K., C.M.Y. Lee, A. Langhammer, S. Krokstad, T.L. Holmen, K. Hveem, S. Colagiuri, J. Holmen. 2013. Trends in overweight and obesity over 22 years in a large adult population: the HUNT Study, Norway. *Clinical Obesity*. 2013 February; 3(1-2): 12-20.

Monteverde, M., K. Noronha, A. Palloni, B. Novak. 2010. Obesity and excess mortality among the elderly in the United States and Mexico. *Demography, 47: 1, February 2010: 79-96.*

Mozaffarian, D., T. Hao, E.B. Rimm, W.C. Willett, F.B. Hu. 2011. Changes in diet and lifestyle and long-term weight gain in women and men. *The New England Journal of Medicine* June 23, 2011. 364:2392-2404.

Neeland, I.J., A.T. Turer, C.R. Ayers, T.M. Powell-Wiley, G.L. Vega, R. Farzanch-Far, S.M. Grundy, A. Khera, D.K. McGuire, J.A. de Lemos. 2012. Dysfunctional adiposity and the risk of prediabetes and type 2 diabetes in obese adults. *Journal of the American Medical Association*. September 19, 2012-308(11);1150-1159.

Nigam, A., R.S. Wright, T.G. Allison, et al. 2006. Excess weight at time of presentation of myocardial infarction is associated with initial mortality risks but higher long-term risks including recurrent re-infarction and cardiac death. *International Journal of Cardiology*. 2006:110(2):153-159.

Ogden, C.L., M.D. Carroll, B.K. Kit, K.M. Flegal. 2013. Prevalence of obesity among adults: United States, 2011-2012. *NCHS Data Brief*, No. 131, October 2013.

Ogden, C.L., M.D. Carroll, B.K. Kit, K.M. Flegal. 2014. Prevalence of childhood and adult obesity in the United States, 2011-2012. *Journal of the American Medical Association*. 2014;311(8):806-814. Doi:10.1001/jama 2014.732.

Oreopoulos, A., J.A. Ezekowitz, F.A. McAlister, K. Kalantar-Zadeh, G.C. Fonarow, C.M. Norris, J.A. Johnson, R.S. Padwal. 2010. Association between measures of body composition and prognostic factors in chronic heart failure. *Mayo Clinic Proceedings*. July 2010 85(7):609-617. Doi:10.4065/mcp.2010.0103.

Oreopoulos, A., R. S. Padwal, K. Kalantar-Zadeh, G.C. Fonarow, C.M. Norris, F.A. McAlister. 2008. Body mass index and mortality in heart failure: a meta-analysis. *American Heart Journal*. 156(1), 13-22 (2008).

Orpana, H.M., J-M Berthelot, M.S. Kaplan, D.H. Feeny, B. McFarland, N.A. Ross. 2010. BMI and mortality: results from a national longitudinal study of Canadian adults. *Obesity*. 18 (1) January 2010.

Petursson, H., J.A. Sigurdsson, C. Bengtsson, T.I.L. Nilsen, L. Getz. 2011. Body configuration as a predictor of mortality: comparison of five anthropometric measures in a 12 year follow-up of the Norwegian HUNT 2 Study. *PLoS ONE* 6(10): e26621. Doi:10.1371/journal.pone.0026621.

Pischon, T. et al. 2008. General and abdominal adiposity and risk of death in Europe. *The New England Journal of Medicine* November 13, 2008.

Power, B.D., H. Alfonso, L. Flicker, G.J. Hankey, B.B. Yeap, O.P. Olmeida. 2011. Body adiposity in later life and the incidence of dementia: the Health in Men Study. *PLoS ONE* 6(3): e17902. Doi:10.1371/journal.pone.0017902.

Preston, S.H., A. Stokes. 2011. Contribution of obesity to international differences in life expectancy. *American Journal of Public Health*. 101(11):2137-2143. November 2011.

Preston, S.H., A. Stokes, N.K. Mehta, B. Cao. 2014. Projecting the effect of changes in smoking and obesity on future life expectancy in the United States. Demography (2014) 51:27-49. Doi:10.1007/s13524-013-0246-9.

Raynor, H.A., D.S. Bond, P.S. Freedson, S.B. Sisson. 2012. Sedentary behaviors, weight, and health and disease risks. *Journal of Obesity*. v2012. Doi:10.1155/2012/852743.

Reis, J.P., C.M. Loria, et al. 2013. Association between duration of overall and abdominal obesity beginning in young adulthood and coronary artery calcification in middle age. *Journal of the American Medical Association*. 2013;310(3): 280-288. Doi: 10.1001/jama.s013.7833.

Reither, E.N., Olshansky, S.J., Y. Yang. 2011. New forecasting methodology indicates more diseases and earlier mortality ahead for today's younger Americans. *Health Affairs*. Aug 30(8):1562-1568. Doi: 10.1377/hlthaff.2011.0092.

Romero-Corral, A., Montori, V.M., et al (2006). Association of bodyweight with total mortality and with cardiovascular events in coronary artery disease: a systematic review of cohort studies. *Lancet*. 2006; 368: 666-78.

Romero-Corral, A., V.K. Somers, J. Sierra-Johnson, Y. Korenfeld, S. Boarin, J. Korinek, M.D. Jensen, G. Parati, F Lopez-Jimenez. 2010. Normal weight obesity: a risk factor for cardiometabolic dysregulation and cardiovascular mortality. *European Heart Journal* (2010) 31, 737-746. Doi:10.1093/eurheartj/ehp487.

Sahakyan, K. V.K. Somers, P. Singh, T. Coutinho, M.D. Jensen, V.L. Rogers, O. Sochor, F. Lopez-Jimenez. 2013. Normal-weight central obesity and cardiovascular mortality risk in the US population. Presentation at European Society of Cardiology Congress. August 2012.

Sahakyan, K. V.K. Somers, J.P. Rodriguez-Escudo, R.W. Squires, T.G. Allison, R.J. Thomas, O. Sochor, P. Singh, F. Lopez-Jimenez. 2012. Physical activity, nutritional status and the risk of normal-weight obesity in the US population. *Circulation*. 2012;126:A9262.

Schenkeveld, L. M. Magro, R.M. Oemrawsingh, M. Lenzen, P. de Jaegere, R. van Geuns, P.W. Serruys, R.T. van Domburg. 2012. The influence of optimal medical treatment on the 'obesity paradox', body mass index and long-term mortality in patients treated with percutaneous coronary intervention: a prospective cohort study. *BMJ Open* 2012;2:e000535.

Shah, N.R., E.R. Braverman. 2012. Measuring adiposity in patients: the utility of body mass index, percent body fat and leptin. *PLoS ONE* (74):e33308. Doi10.1371/journal.pone.0033308.

Singh, P.N., E. Haddad, S. Tonstad, G.E. Fraser. 2011. Does excess body fat maintained after the seventh decade decrease life expectancy? *Journal of the American Geriatrics Society*. 59: 1003-1011.

Sternberg, B.A., et al. 2007. Medical therapies and invasive treatments for coronary artery disease by body mass: the "obesity paradox" in the Get With The Guidelines database. *The American Journal of Cardiology*. November 1, 2007. 100: 9: 1331-1335.

Stewart, S.T., Cutler, D.M., Rosen, A.B. 2009. Forecasting the effects of obesity and smoking on U.S. life expectancy. *The New England Journal of Medicine*. 361(23). 2252-60.

Stommel, M. and C.A. Schoenborn. 2010. Variations in BMI and prevalence of health risks in diverse racial and ethnic populations. *Obesity*. 18 (9) September 2010.

Strindberg, T.E., A.Y. Strandverg, V.V. Salomaa, K.H. Pitkälä, R.S. Tilvis, J. Sirola, T.A. Miettinen. 2009. Explaining the obesity paradox: cardiovascular risk, weight change, and mortality during long-term follow-up in men. *European Heart Journal* (2009) 30, 1720-1727. Doi.10.1093/eurheartj/ehp162.

Sui, X., M.J. LaMonte, J.N. Ladika, J.W. Hardin, N. Chase, S.P. Hooker, S.N. Blair. 2007. Cardiorespiratory fitness and adiposity as mortality predictors in older adults. *Journal of the American Medical Association*. 2007;298 (21): 2507-2516.

Sun, Q., M.K. Townsend, O.I. Okereke, O.H. Franco, F.B. Hu, F. Grodstein. 2009. Adiposity and weight change in mid-life in relation to healthy survival after age 70 in women: prospective cohort study. *BMJ*. 2009; 339;b3796, Doi:10.1136/bmj.b3796.

Swift, D.L., C.J. Lavie, N.M. Johannsen, R. Arena, C.P. Earnest, J.H. O'Keefe, R.V. Milani, S.N. Blair, T.S. Church. 2013. Physical activity, cardiorespiratory fitness, and exercise training in primary and secondary coronary prevention. *Journal of the Japanese Circulation Society*. 2013;77;281-292.

Tice, J.A., et al. 2006. Risk factors for mortality in middle-aged women. *Archives of Internal Medicine*. 2006;166:2469-2477.

Tobias, D.K., A. Pan, C.L. Jackson, E.J. O'Reilley, E.L. Ding, W.C. Willett, J.E. Manson, F.B Hu. Body-mass index and mortality among adults with incident type 2 diabetes. *The New England Journal of Medicine*. 2014; 370;3. 233-244.

Tomé, A.D. 2010. The dynamics of body fat and wages. Master Thesis CEMFI No. 1005. December 2010.

Tommy, L.S., A. Rissanen, J.C. Seidell, M. Heliövaara, P. Knekt, A. Reunanen, A. Aromaa. 2004. Obesity and unhealthy life-years in adult Finns. *Archives of Internal Medicine.* 2004;164:1413-1420.

Tucker, L. and G. Friedman. 1998. Obesity and Absenteeism: An epidemiologic study of 10,825 employed adults. *American Journal of Health Promotion*. 12(3): 202–207, Jan.–Feb.

United Nations. 2013. The state of food and agriculture, food systems for better nutrition. *Food and Agriculture Organization of the United Nations*, Rome, 2013.

Uretsky, S., et al. 2010. Impact of weight on long-term survival among patients without known coronary artery disease and a normal stress SPECT MPI. *Journal of Nuclear Cardiology 2010;17;390-397.*

van Dam, R.M., T. Li, D. Spiegelman, O.H. Franco, F.B. Hu. 2008. Combined impact of lifestyle factors on mortality: prospective cohort study in US women. *BMJ*. 937:a1440, 1-8.

Van Poppel, M.N.M., M.J.M. Chiapaw, L.B. Mokkink, W. van Mechelen, C.B. Terwee. 2010. Physical activity questionnaires for adults: a systematic review of measurement properties. *Sports Medicine*. 10, 565-600.

Wada, R., E. Tekin. 2010. Body Composition and wages. *Economics of Human Biology*. 2010 Ju;8(2):242-254.

Walls, H.L., K. Backholer, J. Proietto, J.J. McNeil. 2012. Obesity and trends in life expectancy. *Journal of Obesity*. 2012. Article ID 107989.

Wang, B., H. Yang, W. Zhu, X. Zhang, G. Cao, S Wu. 2013. Obesity is associated with higher long-term mortality after aortic valve replacement with small prosthesis. *Interactive CardioVascular Thoracic Surgery*. June 19, 2013.

Weiss, A.J. and A. Elixhauser. 2012. Obesity-related hospitalizations, 2004 versus 2009. *HCUP Statistical Brief #137*. July 2012. *Agency for Healthcare Research and Quality*, Rockville MD.

Wier, L.M., W. Encinosa. 2012. Obesity in children: hospitalizations from 2000 to 2009. *HCUP Statistical Brief #136*. July 2012. *Agency for Healthcare Research and Quality*, Rockville MD.

Whitlock, G, et al. 2009. Body-mass index and cause-specific mortality in 900,000 adults: collaborative analysis of 57 prospective studies. *The Lancet*. vol 373. March 28, 2009.

Wildman, R.P., P. Muntner, K. Reynolds, A.P McGinn, S. Rajpathak, J. Wylie-Rosett, et al. 2008. The obese without cardiometabolic risk factor clustering and the normal weight cardiometabolic risk clustering. *Archives of Internal Medicine.* 168, 1617-1624.

Wong, E.S., B.C.M. Wang, L.P. Garrison, R. Alfonso-Cristancho, D.R. Flum, D.E. Arterburn, S.D. Sullivan. Examining the BMI-mortality relationship using fractional polynomials. *BMC Medical Research methodology*. 2011, 11:175.

Wormser, D. et al. The Emerging Risk Factors Collaboration. 2011. Separate and combined associations of body-mass index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58 prospective studies. *The Lancet*. 377;1085-1095. March 26, 2011.

Yanovski, S.Z., J.A. Yanovski. 2014. Long-term drug treatment for obesity. *Journal of the American Medical Association*. 2014; 311(1); 74-86. Doi.10.1001/jama.2013.281361.

Yates, L.B., L. Djoussé, T. Kurth, J.E. Buring, J. M. Gaziano. Exceptional longevity in men. *2008. Archives of Internal Medicine.* 168 (3). 284-290.

Yu, Y. 2012. Reexamining the declining effect of age on mortality differentials associated with excess body mass: evidence of cohort distortions in the United States. *American Journal of Public Health*. May 2012. 102 (5). 915-922.

Zajacova, A., S.A. Burgard. 2012. Shape of the BMI-mortality association by cause of death, using generalized additive models: NHIS 1986-2006. *Journal of Aging and Health*. 24(2) 191-211. Doi:10.1177/0898264311406268.

Zheng, H., D. Tumin, Z. Qian. 2013. Obesity and mortality risk: new findings from body mass index trajectories. American Journal of Epidemiology. 2013: 178(178) 178(1): 1591-1959. Doi:10.1093/aje/kwt179.