Human Behavior:
An Impediment to Future Mortality Improvement

A Focus on Obesity and Related Matters

Sam Gutterman, FSA, FCAS, MAAA, HonFIA

Presented at the Living to 100 and Beyond Symposium

Orlando, Fla.

January 7-9, 2008

Copyright 2008 by the Society of Actuaries.
All rights reserved by the Society of Actuaries. Permission is granted to make brief excerpts for a published review. Permission is also granted to make limited numbers of copies of items in this monograph for personal, internal, classroom or other instructional use, on condition that the foregoing copyright notice is used so as to give reasonable notice of the Society's copyright. This consent for free limited copying without prior consent of the Society does not extend to making copies for general distribution, for advertising or promotional purposes, for inclusion in new collective works or for resale.

Table of Contents

Section	Page
1. **Executive summary**	2
2. **Issue background**	3
3. **Human behavior: impediment or opportunity?**	5
3.1. Measurement and causation	5
3.2. Smoking	6
3.3. Opportunity	12
4. **Introduction to obesity**	12
4.1. Measurement	15
4.2. What is overweight and obesity	16
5. **Obesity characteristics, trends and contributing factors**	17
5.1. Adults	19
5.1.1. Older adults	38
5.2. Children (through age 19)	38
5.3. Does childhood overweight lead to adult obesity?	50
5.4. Economic perspective	53
5.5. Contributing factors	58
5.5.1. Nutrition	58
5.5.2. Inadequate physical activity	64
5.5.3. Socio-economic factors	68
5.5.4. Other factors	71
5.6. Worldwide situation	72
5.7. Optimal weight	81
6. **Effect of obesity and related factors**	84
6.1. Adults	86
6.1.1. Aggregate mortality observations	108
6.1.2. Methodological issues	107
6.1.3. Follow-up period	110
6.1.4. Black females	112
6.1.5. CDC estimates of deaths due to overweight and obesity	115
6.1.6. Older adults	116
6.2. Children	122
6.3. Change in weight	127
6.4. Lack of physical activity	126
6.5. Underweight	129
6.6. Insurance experience	131
6.7. Morbidity, health care and total cost	134
6.8. Quality of life	145
7. **Prevention and management of obesity**	145
8. **Conclusion and implications for mortality projections**	169
9. **Glossary**	177
10. **Appendix -- Obesity measurement**	180
11. **References**	187
12. **List of figures and charts**	206

1. Executive Summary

The effects and consequences of human behavior on mortality and life expectancy have always been significant. Some of the recent significant trends in behavior have resulted in a significant and in some ways shocking increases in the prevalence of obesity in all sectors of the U.S. population (e.g., for all adults, the current reported obesity prevalence rate is 34.4 percent, based on a 2005-2006 nationally representative survey), as well as in almost all areas of the rest of the world. Since this increase has been gradual although relentless over the last 30 years, its existence has generally been accepted as a fact of life. Nevertheless, although this increase in weight and its general effect on the morbidity and mortality of those severely obese are uncontested, its overall effect on future mortality is still subject to debate and sometimes conflicting evidence.

The primary behavioral factors that have contributed to this trend involve the amount and mix of food intake and physical activity. Although it would be ideal if the specific short- and long-term effects of these factors could be identified, this is less useful than understanding the effect of these behavioral effects on a combined basis. In most cases, the level of obesity can serve as a reasonable surrogate for the combination of these factors.

This paper presents recent trends in obesity prevalence in significant population segments, including overall effects by age, gender and certain racial and ethnic groups, and discusses the ways in which obesity can influence mortality in each of these segments. It provides a synthesis of reported mortality experience relating to these behaviors by risk factors and diseases that will affect any mortality projection. Although usually measured in terms of Body Mass Index (BMI), alternative measures of adiposity, such as waist circumference, have shown that obesity, in one shape or another, can affect future mortality and morbidity levels.

These developments have significant implications for the individual and society as a whole. It is asserted that further data and analysis are needed, particularly in view of the long-term lags between the underlying behaviors and their mortality consequences. It also discusses the possible prevention and management of obesity. Although this paper focuses on the future obesity trends and their effect on mortality,

it describes the possibly more significant effects on morbidity, disability, health care costs and the quality of life, and the recent studies on health care costs and disability.

A key issue discussed is the adverse long-term health effects of adolescent obesity. It also indicates that the effects of obesity have to date been more than offset by significant risk mitigation and other developments, particularly treatments for high blood pressure and cholesterol levels and reductions in smoking. The uncertainty associated with mortality projections includes the extent that these sets of factors and future technological developments will offset each other in the future.

Whatever the ultimate effect of human behavior, understanding the contributions of its dynamic nature and effects on mortality, particularly of the adverse weight syndrome (obesity/fatness, excess and unhealthy food intake and sedentary lifestyle) is necessary to prepare soundly based mortality projections.

2. Issue Background

In projecting mortality rates, the effect of human behavior and potential changes in this behavior can be easily overlooked by assuming that past trends or current conditions continue indefinitely into the future. Historically, behavior has manifested itself in many ways in an uneven manner over time that can result in favorable or harmful effects on health and mortality. Examples include maintaining a healthy lifestyle and cigarette smoking, and their consequential effects. In addition to the activities by themselves, many behaviors are important because they can enhance or mitigate the positive or negative effects of other factors affecting mortality.

This paper focuses on the consequences of a set of adverse behavior, specifically those involving nutrition and physical activity, that has led to an unprecedented increase in obesity for a large part of the worldwide population, especially that of the United States. These trends will likely lead to higher mortality, morbidity and health care costs than if they had not occurred.

A so-called "obesity epidemic" has been underway for the last 30 years, with no end in sight. Interestingly, it was not predicted in advance and was not recognized until several years after it had begun. This trend has been particularly troubling,

because although it has occurred in all segments of the population, it has proven difficult to make a compelling case for any specific cause. Nevertheless, after a thorough review of the existing and emerging literature, it is clear that this is an extremely complicated issue, with the findings of numerous studies and analyses in some cases contradicting each other—some of which is due to inconsistent controls and methodology, with the resulting data being disappointingly ambiguous at times, although some of the disagreements are also due to the complex pathways among the factors involved.

The objective of this paper is to provide a synthesis of the results of recent studies regarding the prevalence and impacts of obesity and related factors on the future health of the population, with an emphasis on that of the United States.

These potential adverse effects, the extent to which have proven controversial, are in sharp contrast with what has been remarkably favorable reported overall mortality experience of developed countries, including the United States, resulting from advances in a wide range of areas. Indeed, a simple extrapolation of recent overall mortality trends and the application of knowledge gained from the rapid explosion of biological knowledge would lead to a projection of highly significant improvements in life expectancy in the future. The overall progress generated from the medical treatment of disease risk factors, reduction in heart disease and reduction in smoking (at least for males) has recently by and large more than offset the adverse effects of obesity and related risk factors. To how great an extent will our reaction to higher standards of living and technology allow this to continue?

After an introductory section on human behavior, the paper consists of five major sections:

- Section 4: an introduction to obesity
- Section 5: the characteristics, trends and contributing factors of obesity and related factors over the life-cycle, in both the United States and worldwide
- Section 6: the effect of obesity and related factors on mortality and health
- Section 7: a brief discussion of the prevention and management of obesity
- Section 8: a conclusion and implications for mortality projections.

The process followed to produce this paper was primarily based on a literature search, which seemed to proceed in many directions. As the pervasiveness of the problem has grown, so has attention paid to it. The literature is large and growing. Nevertheless, as a result of the inconclusiveness of some of the findings and difficulty in obtaining clear information, further fundamental research and insight are needed in almost all areas.

3. Human Behavior: Impediment or Opportunity?

There are many human behaviors that affect human health and longevity. They can be categorized into two types:

- Those of an individual nature. These include smoking, physical activity, diet and nutrition, drinking alcohol, driving skills and the extent of taking up a physician's prescriptions and advice.

- Those of an institutional nature. These include actions developed or regulated by institutions, including wars, laws, professional practice and commerce, such as a rule imposed dictating cleanliness in hospitals and modern sewage.

The focus of this paper is aimed at those behaviors that are the result of actions and decisions of individuals, although they clearly can be influenced by factors outside of their immediate control. An obvious example of these factors is smoking, a thorough discussion of which, although outside the scope of this paper, is briefly covered in Section 3.2, that nevertheless can serve as a useful background to the remainder of the paper.

Human behavior is usually quite difficult to change, although it seems that “bad” habits are more difficult to eliminate than “good” are to emerge. In some cases, legal, regulatory or catastrophic indications are necessary to change them.

3.1 Measurement and Causation

In many cases, it is difficult to measure and develop standardized metrics that can measure and compare these behaviors over time, especially those that can be

effectively used internationally. Once benchmarked, however, they can be used to set goals at a personal or national level.

An additional problem is the difficulty in distinguishing associations and causal relationships. The Advisory Committee to the Surgeon General in his report (1964) described the characteristics of a causal relationship as including consistency, strength of association and a dose-response relationship, temporal relationship and coherence and specificity of cause. Later another criterion was added, a requirement that as the purported cause decreases in intensity, the resulting effect decreases as well. I remember in the mid 1970s arguing with statisticians working within the tobacco industry about whether all of these characteristics had been satisfied in the relationship between cigarette smoking and mortality.

To demonstrate causation, as important as it is to focus efforts to change a particular behavior, identification of all of the potential causative factors may suffice. Thus, to a great extent, observed statistical relationships may be just as important as causal ones. Definitive proof of causal relationships can be complicated as well as controversial. As we will see later, these challenges arise in the area of obesity as well.

3.2 Smoking

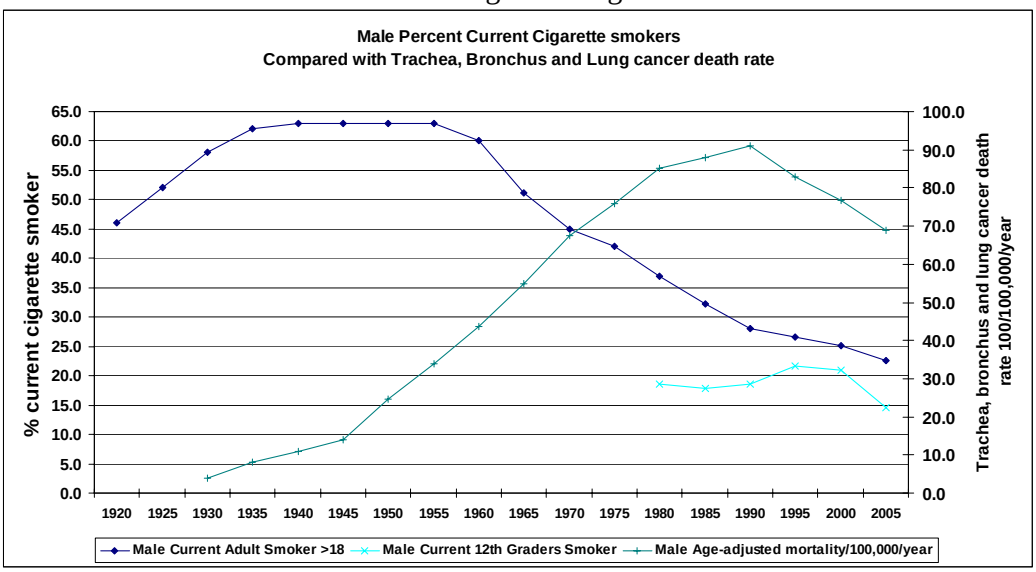
Over the last few decades the most favorable behavioral change has been the decrease in prevalence of cigarette smoking, even though smoking remains a serious problem today. In the early part of the 20th century, smoking prevalence increased as a result of product and technology changes (e.g., the introduction of blends, curing and mass production processes, invention of the safety match, distribution, mass media advertising and liberalization of women's roles and behavior). Smoking peaked for adult males in the 1940s and 1950s, while it peaked for adult females in the 1960s, as can be seen in Figures 1 and 2 (note that the sources of this data indicate that data prior to 1955 may not be as reliable as that since then).

Significant effort has been required, through public policy interventions, increased taxation and individual efforts, to help ensure that this trend continues, ranging from public policy rules (now widespread in many countries, in part due to increased understanding of the dangers to others of secondhand smoke), vigorous

anti-smoking information campaigns and medical encouragement. These constraints and adverse publicity, among other factors, may have contributed to the recent decreases in smoking in American 18-year-olds as well.

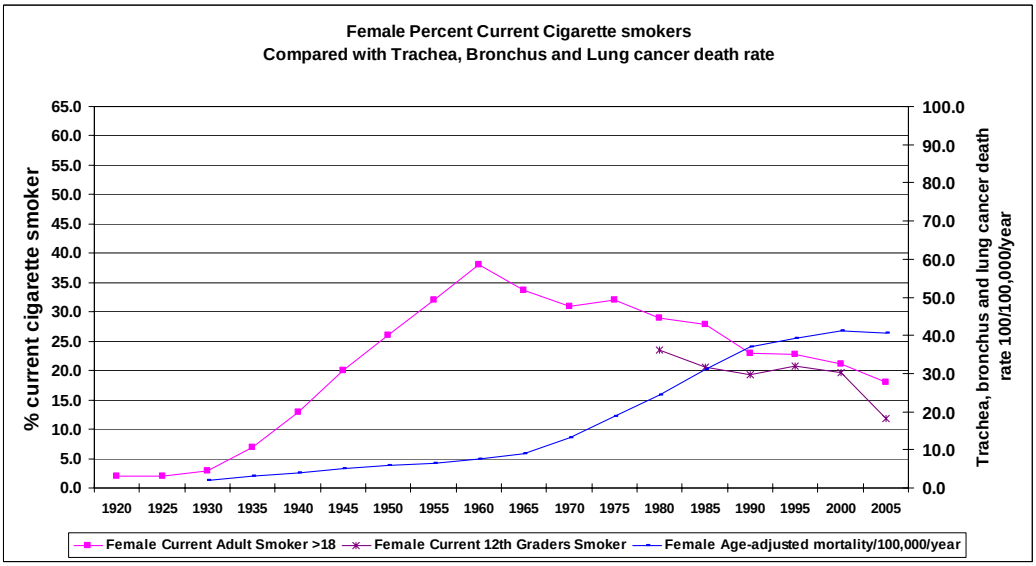
In contrast, in developing countries such as China and India, smoking prevalence rates continue to grow. In these countries, WHO has recommended that the quality of tobacco use data needs to be enhanced, sweeping smoking bans need to be imposed, marketing of smoking products should be banned, intensified efforts to induce and assist smokers to drop the habit are needed, and taxes on cigarettes need to be increased. But only nine countries offer all such programs now. The practical problem is that smokers face the familiar choice between short-term pleasure and long-term desire to quit, complicated by personal addiction to the habit. The tobacco industry is regrouping to focus on promising markets, and WHO has admitted that it is losing the battle in these countries. Only significant public policy efforts will likely change the tide, even in view of the clear health effects involved.

FIGURE 1
Male Smoking and Lung Cancer



In contrast, although the pattern in smoking and consequential mortality for females is similar, the peaks of both have occurred at different times, with the peak of mortality due to smoking for females just now reaching its maximum height.

FIGURE 2
Female Smoking and Lung Cancer



Sources: Weiss (1997), *Health, United States, 2006* and SEER Cancer Statistics Review 1975-2003

Nevertheless, it is estimated that in 2005 about 45 million U.S. adults still smoke cigarettes, about 80 percent of whom smoke every day, with a significantly higher percent of those with a lower educational and poverty level smoking. For example, 32.6 percent of those with 9-11 years of education and 29.9 percent of those

living below the poverty line smoke, compared with 20.6 percent of those living above that line.

These causes of death are not the only result of smoking, but are appropriate for illustrative purposes. For example, for adult males of all ages, the peak of deaths from these consequential diseases occurred about 1990 (for certain ages and causes the decline started in the 1980s), while for adult females, the peak has just been reached. This differential effect has been a significant contributing factor underlying the overall faster improvement in male mortality recently compared with that for females.

Although the time lag between cause and effect for smoking evident in Figures 1 and 2 may be extreme, the delayed adverse consequences of many adverse behaviors under modern conditions may not manifest themselves into chronic diseases for several years, if not decades. This lag has contributed significantly to the difficulty in changing long-term adverse behaviors.

The effects from trends in smoking have obscured some of the other important underlying trends affecting mortality. If the effects of changes in adverse behavior such as smoking (and more recently changes in lifestyle, nutrition and obesity) are controlled for, the overall mortality improvement can be more easily discerned. If the current prevalence of smoking in the United States continues or decreases, mortality rates will also continue to improve accordingly.

But even this overall favorable trend can have offsetting consequential adverse results, as smoking has served for many as a weight control device. According to Flegal et al. (1995), reporting on a study of the third National Health and Nutrition Examination Survey (NHANES, 1988-91), current smokers had the lowest age-adjusted prevalence of overweight and the lowest mean body mass index (BMI) of all groups studied. Those who quit smoking within the previous 10 years had the highest age-adjusted increases in BMI (4.4kg for men and 5.0kg for women, 7.4 and 11.0 pounds, respectively, although it should be noted that the rest of the population also experienced some increase during the same time), with a large increase in the prevalence of overweight.

In contrast, Flegal (2007) indicated that, based on the 1999-2002 NHANES, even relatively large changes in the prevalence of smoking are estimated to have little effect on obesity prevalence. She concluded by indicating that "Decreases in the prevalence of cigarette smoking probably had only a small effect (on obesity), often less than 1 percentage point, on increasing the prevalence of obesity and decreasing the prevalence of healthy weight in the population." This contrasts with results from NHANES III that found that smoking cessation results in an increase of from 10 to 11 pounds.

The consequential weight gain usually occurs shortly after cessation of smoking. It may be as a result of displacement of nervousness or the appetite suppressive quality of nicotine, by enhanced taste and smell perception after smoking cessation, and the substitution effect of nicotine and calories, all contributing to make food more desirable. Former smokers, including those of more than 10 years, were still more likely to be overweight than those who continued smoking. Among men, one-quarter of the increase in overweight for males and about one-sixth of the increase in females could be attributed to the cessation of smoking. Also, some smokers, particularly women, may not even attempt to stop smoking for fear of possible future weight gain. The U.S. Surgeon General in 1990 conducted a meta-analysis of 15 medical studies; the consensus indicated that between 58 percent and 87 percent of those who quit smoking gained on average four pounds more than those who continued during a median follow-up period of two years, with the additional weight retained for at least six years.

Chou et al. (2004) found that, using the results of the U.S. Behavioral Risk Factor Surveillance Survey (BRFSS) and applying a quadratic time trend, the real price of cigarettes (including taxes) had an elasticity with respect to BMI of 0.025, and an elasticity with respect to being obese of 0.445, indicating a significant effect of smoking on body weight. In contrast, Gruber (2006), using linear relationships between BMI and cigarette taxes, did not find a significant effect for ages under 65. Nevertheless, Gruber concluded that, although there is no evidence for a large weight effect from smoking cessation, he could not rule out a moderate-sized effect.

A simple comparison of the results from the BRFSS indicates that in 2000 the percentage of currently smoking adults who are obese was 16.3 percent compared with 22.7 percent for ex-smokers and 19.9 percent for those who had never smoked.

Obesity has sometimes been compared with smoking as an adverse behavior. Some of the similarities include having a relatively high prevalence in what has been referred to as an epidemic, often starting in childhood or adolescence, being relatively uncommon until the first (smoking) or second (obesity) half of the 20th century, both representing a major risk factor for chronic diseases, involving intensively marketed products, being more common in low socioeconomic classes, exhibiting regional prevalence with higher rates in southern and poorer states, currently carrying a social stigma and being very difficult to change.

In early 2008, three independent research teams identified a set of genetic variations that can increase the risk of lung cancer and make smokers more addicted to nicotine, although disagreements exist as to whether the risk is increased directly by triggering biological processes that lead to lung cancer or indirectly by making people more addicted to smoking. Similar research may find similar genetic predispositions to becoming fat.

Conversely, there are several differences, including the impossibility of stopping eating (although some smokers might indicate the same about their addiction) and the possibility of being misclassified as overweight or obese although healthy (e.g., those with large muscle mass or in certain population segments). A moderate amount of food intake is not hazardous, and does not cause injury to others as secondhand exposure analogy to obesity. The food industry is less concentrated than the tobacco industry and although sweet food manufacturers have been criticized due to its advertising to children, it hasn't been vilified like the tobacco companies have been. It took 40 years of hard work involving communicating what seemed to be obvious information to consumers, laws, taxes and ostracism to reduce the prevalence rate of smoking by half. Without this type of support, it may take longer to reduce the prevalence of obesity.

3.3 Opportunity

As can be seen by the smoking discussion in Section 3.2, significant opportunities for continued improvement in longevity remain. For example, Preston and Wang (2006) estimated that the probability of survival from age 50 to 85 would improve from 30.4 percent for men based on historical average smoking levels, to 38.4 percent at 2000 smoking levels, and to 46.4 percent with a complete cessation of smoking; corresponding percentages for women were 46.8 percent, 47.9 percent and 51.9 percent, respectively.

Although the possible longevity enhancement opportunities that came with the elimination of smoking may not be achievable with respect to the reduction in or elimination of other adverse behaviors, the possible impact of other lifestyle changes are nevertheless quite large.

4. Introduction to Obesity

Being overweight is not a behavior. So why has the author focused on it in a paper on the impact of human behavior? Primarily it is because obesity and changes in obesity are predominantly the result of the cumulative effect of the twin behaviors of physical activity and diet/nutrition. These individual behavioral processes can create an imbalance of energy inputs and energy expenditure. It is this combination of factors that is the focus of the remainder of this paper.

Why look at weight at all? There are several reasons:

1. At least one measure of it is an easy-to-determine metric.
2. It is, at least in theory, controllable.
3. Many chronic diseases are affected by weight.
4. It is by itself a factor in determining health status.

But it isn't sufficient to look just at weight, as the major determinants of weight can also play an independent or causative role in mortality. In fact, the determinants and effects of mortality are quite complex and have been subject to considerable disagreement.

Obesity has been widely viewed as being an epidemic or pandemic (see Flegal (2005) for a discussion of how obesity might satisfy the definition of an epidemic, usually characterized by high prevalence, rapid increase, unexpected nature and an initial reluctance to accept and acknowledge it). Although not necessarily a particularly good measure of public concern, in November 2007 there were 1.8 million Google entries when “obesity epidemic” or “obesity pandemic” was searched, 40 million entries for “obesity,” 13.6 million for “obese,” while there is 149 million entries for “diet,” 142 million entries for “nutrition,” 30 million for “physical fitness,” and 313 million for “exercise,” while the 574 million entries for “food” has overtaken the 439 million for “God”. Certainly this is evidence that the media has done an excellent job in communicating the existence of obesity, although some feel that since it has been so extensively written about it will take a lot to attract additional attention to it in the future. A public opinion survey conducted by Trust for America's Health in 2007 indicated that 85 percent of Americans believe that obesity is an epidemic. Nevertheless, even with the significant media attention over the past several years, the number of those who are obese continues to grow.

This increase in the extent of obesity over the last few decades reflects profound changes in society and in behavior patterns throughout the world. While genes may be important in determining a person's susceptibility to weight gain, man's genetic makeup cannot have changed that significantly during this short period, although genetic predispositions may have made certain aspects of genetics more important. More likely, personal energy balances are being affected by societal changes and the worldwide nutrition and activity transition. Economic growth, urbanization and globalization of food markets have and are having impacts on a worldwide basis, while in the United States significant shifts in the level of physical activity and eating habits head the list of contributing factors. Some view these fundamental forces as the inevitable result of our modern post-industrial and post-agricultural world.

However, as Helmchen and Henderson (2004) pointed out in their study of a randomly drawn group of Civil War Union veterans, this is not the first time that adverse trends in the prevalence of obesity have occurred. They indicated that "in relative terms, then, obesity was spreading at least as fast at the beginning of the 20th

century as at the end of the 20th century." For example, for a similar age 50-59 cohort, the rate of obese males increased from about 1 percent in 1880-84, to 3.5 percent in 1890-94 and 5.5 percent in 1900-04 status. The annual rate of growth of median BMI was about 0.3 percent per year between 1900 and 1976, but almost doubled to 0.5 percent per year between 1988 and 2000.

The fundamental formula of weight is:

Current weight = previous weight + energy inputs - energy outputs

This formulation is consistent with the first law of thermodynamics that states that the amount of stored energy equals the net energy intake and is applicable to all biologic systems. Of course, it isn't quite this simple, as people are affected differently, either through their genetic profile or their body, influenced by such factors as their metabolism and the environment.

For most people, being overweight or obese results primarily from a combination of excess calorie consumption and/or inadequate physical activity. Energy inputs include the amount and type of food intake, while energy expenditure or outputs are primarily influenced by basal metabolic rate, food-induced heat production and physical activity. The results are rather sensitive to the underlying factors, as an excess of intake over expenditure by just 2 percent daily for a year would result in an increase of about five pounds of adipose tissue.

Obesity results when body fat accumulates over time as a result of a chronic energy imbalance as evidenced by the above formula, through behavior and the process by which a person's body stores and converts caloric input into caloric expenditure. In the meantime, an increased amount of fat in the chest wall and abdomen can affect the total body oxygen consumption, increase in heart load and the breathing process.

However, a seemingly minor daily imbalance can lead to a gradual, but persistent weight gain over a considerable period as excess energy is stored as triacylglycerols in adipose tissue. Once established, physiological and mental

processes tend to maintain the new weight. In the long term, the fatness balance has to be regulated and managed to achieve energy and macronutrient balance—if not, obesity will inevitably result.

Energy is consumed (calories are burnt) through basal metabolism, processing food and physical activity. Cutler (2003) observed that in the aggregate in the United States, the number of calories expended has not changed significantly since 1980, while calories consumed have risen dramatically.

4.1 Measurement

Excess body fat stored in adipose tissue is usually a result of excess energy from food that is stored as fat. The amount and location of this stored fat varies considerably between populations, people and over the course of a person's lifetime. In addition, since fat is stored throughout the body, body fatness is difficult to measure directly and accurately.

The first common weight measure was developed in the 19th century when Dr. Paul Broca, based on a survey of soldiers, found that “normal” weight could be expressed in terms of height (measured in centimeters) - 100. In the 20th century, generally accepted measures of weight in the United States were based on surveys, conducted by the Metropolitan Life Insurance Company, that developed “ideal body weights” based on height, body frame and weight in 1942, “desirable body weights” in 1959 and “height and weight” tables in 1983.

At the end of the 20th century, the most common metric used is the “Body Mass Index” (BMI) that relates weight to height, with standard categories as recommended by the World Health Organization (WHO), and is now used worldwide. See the Appendix for further details and alternative measures. There is no unique perfect metric, and certain of the alternatives might be better for certain population segments and for certain conditions. BMI has been criticized for being an overall index, rather than more specific anthropometric measurement (e.g., circumferences, skinfolds, diameters) that can provide insight into certain body characteristics, such as fat mass or the distribution of adipose tissue. Nevertheless, BMI has been judged to be the most practical metric with a fairly high correlation (by some measures with an r =

0.6 to 0.8). Although it may not have a particularly high individual predictive value, it is quite useful for population studies.

In addition, this is only one dimension of being healthy, albeit an important one, as health represents a combination of many factors. In fact, in some cases, a person who is obese and physically fit might be in better overall health than someone else who is of normal weight but not physically fit. Nevertheless, it is usually considered better if you are fit and lean, rather than fit and fat; Hu et al. (2004) indicated that those who are active and obese are still at a 91 percent greater risk of dying than those who are active and lean.

4.2 What is Overweight and Obesity

Table 1 shows BMIs for some common weights and heights, particularly emphasizing those definitional cutoff points in the various overweight and obesity categories (obviously, this doesn't mean that all height and weight combinations within a category are truly homogeneous, as it is likely that a continuous curve of risks is applicable). This measure applies to both females and males.

TABLE 1
Relation between Body Mass Index (BMI) and Height (feet/inches) and Weight (pounds) for Adults

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

Although it is useful to look at averages, because of the non-uniform nature of mortality, prevalence by population segment is also important. Although some use average BMIs, it is more relevant to study the percentage of the population in excess of one or more benchmarks or cutoffs. Key ones often looked at are overweight and

obesity. Overweight for adults is defined as having BMIs of between 25.0 and 29.9, while obese adults have BMIs of 30.0 and greater. Because of recent gains in weight, the obese category is sometimes split into class 1 obese as 30.0-34.9, class 2 obese as 35.0-39.9, class 3 obese as 40.0 to 44.9, class 4 obese as 45.0 to 49.9 and class 5 obese as 50.0 and upwards. "Normal" weight is usually indicated by 18.5 to 24.9 BMI, with anyone at a BMI less than 18.5 being considered “underweight.”

For children, a different basis is commonly used. In the United States, the term “overweight” is used for a BMI at or above the 95th percentile for each gender and age. Note that since this was derived more than a decade ago, average weights have increased significantly; thus, the current values not longer represent the current 95th percentile. The term “at-risk of overweight” is used for BMIs between the 85th and 95th percentile of the out-of-date distribution of weights and heights. These percentages were developed looking at the corresponding adult values at age 18.

Being obese obviously represents more of a health risk than being overweight. Nevertheless, being overweight can still be a risk in many instances.

The incidence rates of BMI and obesity typically increase with age, generally peaking in the 50s, followed by a decrease beginning in the 60s. The typical American gains between 22 and 33 pounds between ages 20 and 50. Not only is it unlikely that caloric intake increases with age; nutrition surveys indicate that caloric input actually declines with age. Although an age-related decrease in resting metabolic rate may occur, a decline in physical activity and muscle mass may be equally important.

A weight gain of 22 pounds can be explained by an energy discrepancy of only about 300 calories per day. Thus, a reduction in caloric input combined with an increase in energy expenditure of 300 calories can eliminate 22 pounds excess in weight. Even someone very obese rarely consumes more than 500 calories per day above that needed to maintain their weight.

5. Obesity Characteristics, Trends and Contributing Factors

Evidence from all available statistics indicates that there has been a significant and rapid increase in individuals who are obese and who are overweight. In fact, in

the United States the entire BMI distribution has shifted to the right, with increases occurring in all age-gender-ethnic categories, suggesting that heavier people are getting heavier more quickly than others. But these changes are population-wide and not limited to just the upper portion of the distribution. More than 72 million adults are estimated to be obese in the United States, with about 140 million adults either obese or overweight.

In the ancient days when food was scarce, the ability to take advantage of rare periods of food abundance by storing energy efficiently was an evolutionary advantage. Individuals with greater adipose reserves were thus better equipped to survive subsequent famine. However, in societies in which adequate and stable food supplies exist, this is maladaptive. The human body aggressively responds to underweight by enhanced hunger and a decreased metabolic rate, yet it counteracts the accretion of excess weight poorly, possibly derived from the age-old struggle for survival, with the primary risk being death due to famine rather than being in danger from excess food.

In several cultures, obesity was associated with physical attractiveness, strength, fertility and in some cases a symbol of wealth and social status. In contrast, today obese body shapes are widely regarded as being unattractive.

In the 19th century, people were smaller, lighter and shorter-lived, and faced a heavier disease burden in old age. The average BMI of Union soldiers was 23 on average, compared to an average of 26 BMI today. In addition, there have been substantial changes in the human frame as men have become taller and heavier.

The average American adult female weighed 143 pounds in the early 1960s, while she weighs over 155 pounds now. The average American adult male weighs nearly 180 pounds now, compared to 168 then.

Today's fatness patterns suggest the prevalence of chronic conditions in past periods were greater than previously reported, where reliable causes of death information was limited or where people died from other causes prior to their old age. People are now generally healthier. For example, according to Miljkovic (2006), the

average onset of heart conditions in American males was 55.9 years of age about 1890, while it is 65.4 a century later, with the age distributions at onset also being quite different. Henderson (2005) concluded that the frontiers of overweight and obesity have been expanding, with health risk currently associated with higher levels of BMI than before. This is consistent with Flegal et al. (2005)'s conclusion that the mortality risk of overweight and obesity may have decreased over time.

5.1 Adults

Table 2 shows the current prevalence of obesity for U.S. adults (based on NHANES 2005-2006 made available in November 2007), broken down by age group, gender and certain racial/ethnic categories. Overall, it is estimated that 34.3 percent of adult Americans are obese (BMI>30), with a somewhat larger percent for females than for males. This relativity is largely but not exclusively driven by the wide disparity by gender for non-Hispanic blacks (see Section 6.1.4 for a further discussion of the high rates of obesity of non-Hispanic black females). Note that prevalence rates for Mexican-Americans are given as representative of Hispanics as they are the largest subgroup, although sub-populations of Hispanics differ. Neither the total prevalence rates by ethnic categories nor prevalence rates for those overweight were available at the time this paper was written, but given that the estimated percent obese has increased by two percentage points, it is likely that the percent overweight has correspondingly increased.

TABLE 2
Obesity Prevalence for U.S. Adults Aged 20 and Older
by Gender and Age: 2005-2006

Age	Non-Hispanic whites		Non-Hispanic blacks		Mexican Americans		Total	
	Men	Women	Men	Women	Men	Women	Men	Women
20-39	27%	28%	40%	49%	26%	37%	28%	30%
40-59	41	39	36	53	28	51	40	41
60 +	34	32	37	61	38	37	32	34
Total	--	--	--	--	--	--	33.3	35.3

Increasing age until the 60s is associated with an increase in obesity. This increase is not explained by increases in fat-free mass, as bone mass peaks around age 30 and muscle mass plateaus and later declines without strengthening exercises. The changes in body weight and body composition are attributable, in part, to the natural declines in certain hormones, as well as changes in metabolism.

Figure 3 shows the trend in adult obesity over the last several versions of NHANES, from 1999-2000 to 2005-2006. The upward trend in these rates continues, although that for women appeared to have flattened out in the early part of the decade. Although the biannual trends reported on are not significant (confidence intervals were not available at the time this paper was written), the upward continuing trend is clear.

FIGURE 3
Trends in Obesity Prevalence for U.S. Adults Aged 20 and Older by Gender between 1999-2000 and 2005-2006

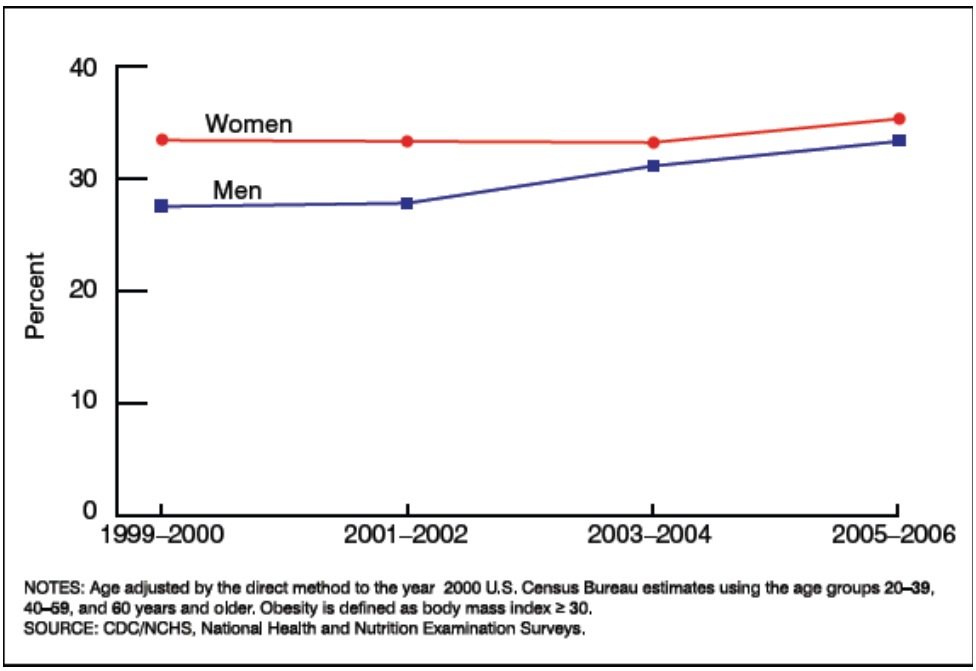
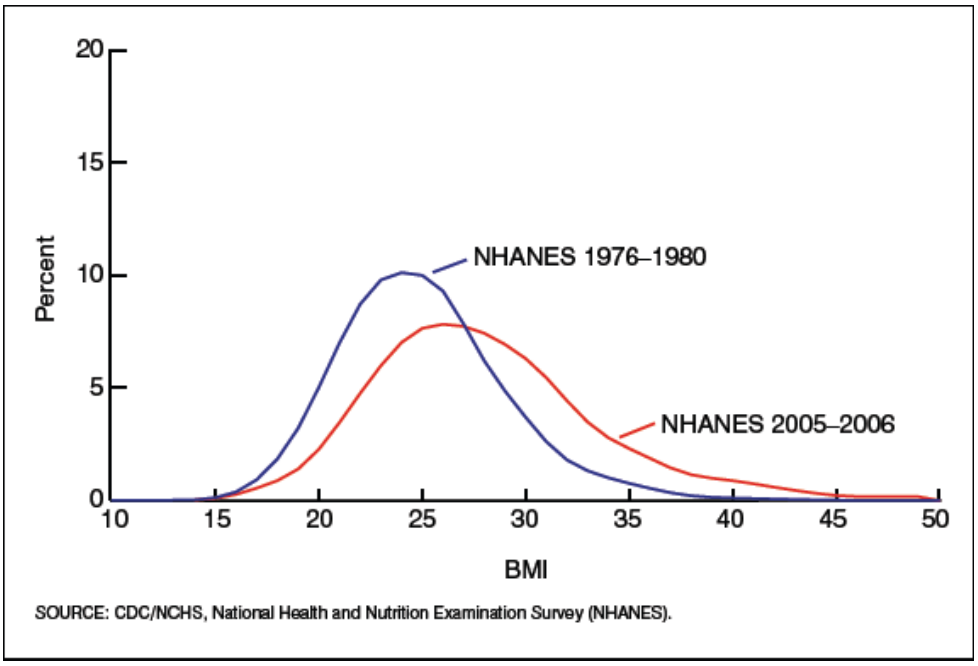


Figure 4 illustrates that the U.S. distribution of BMI has shifted dramatically to the right over the past 30 years, with a larger right-handed tail. Thus, not only have average BMIs shifted to the right, but the percent in excess of a given BMI level has also increased. In general, this implies that a large percentage of those formerly categorized as overweight are now in the obese category, while many of those previously considered “normal” are now overweight. Depending on the extent to which being overweight is an adverse risk factor, at the minimum most of those overweight might be considered to be “at risk” of becoming obese. The distribution has shifted to the right far more for males than for females.

FIGURE 4
Changes in the Distribution of BMI of U.S. Adults
between 1976-1980 and 2005-2006



Tables 3 and 4 provide additional comparisons of reported prevalence of adult obesity and overweight (including those obese) for various demographic characteristics based on NHANES, values of which were constructed based on professional measurement of weight and height for obese and overweight/obese, respectively.

Those married and widowed tend to have higher rates of BMI and obesity odds than those divorced, who in turn have higher rates than those never-married. For Mexican-Americans and non-Hispanic black men, average BMI increases with increasing income, while the opposite relationship is observed in other major population segments.

TABLE 3
1988-94 Compared with 2002-2004 Adult Obesity as a Percent of U.S. Population

Age	Males		Females	
	1988-94	**2001-04**	**1988-94**	**2001-04**
20-34	14.1%	23.2%	18.5%	28.6%
35-44	21.5	33.8	25.5	33.3
45-54	23.2	31.8	32.4	38.0
55-64	27.2	36.0	33.7	39.0
65-74	24.1	32.1	26.9	37.9
75+	13.2	19.9	19.2	23.2
Total age-adjusted	20.2	29.5	25.5	33.2
Ethnic Group				
Non-Hispanic white	20.3	30.2	22.9	30.7
Non-Hispanic black	20.9	30.8	38.3	51.1
Mexican	23.8	29.1	35.2	39.4
Income	**Males and females**			
	1988-94		**2001-04**	
< poverty line	28.1		33.7	
1-2x poverty line	26.1		33.6	
> 2x poverty line	21.1		30.0	
Total	22.9		31.4	

Source: NHANES III and continuous

TABLE 4
1988-94 Compared with 2002-2004 Adult Obesity and Overweight as a Percent of U.S. Population

Age	Males		Females	
	1988-94	**2001-04**	**1988-94**	**2001-04**
20-34	47.5%	59.0%	37.0%	51.6%
35-44	65.5	72.9	49.6	60.1
45-54	66.1	78.5	60.3	67.4
55-64	70.5	77.3	66.3	69.9
65-74	68.5	76.1	60.3	71.5
75+	56.5	68.8	52.3	63.7
Total - age-adjusted	60.9	70.5	51.4	61.6
Ethnic Group				
Non-Hispanic white	61.6	71.0	47.5	57.6
Non-Hispanic black	57.8	67.0	68.2	79.6
Mexican	68.9	74.6	68.9	73.0
Income	**Males and females**			
	1988-94		**2001-04**	
< poverty line	59.6		63.4	
1-2x poverty line	58.0		66.2	
> 2x poverty line	54.8		66.1	
Total	56.0		66.0	

Source: NHANES III and continuous

In contrast to statistics derived from the professionally measured weights and heights included in Tables 3 and 4, Table 5 provides age-adjusted average obesity prevalence based on BRFSS that uses self-reported BMI measures. These two sets of results demonstrate the potentially significant difference between professionally measured and self-reported weight and height measures. Although useful for relativities, BRFSS results that rely on self-reported weights and heights do not appear as reliable as those derived from more objective and consistent measurements as given in NHANES. Note that, although the annual change reported in BRFSS does not appear significant, the overall trend over the past decade certainly is.

TABLE 5
Prevalence of Obesity among Adults Ages 20 and Over on a Self-Reported Basis

Year	Age-adjusted	95% confidence interval
1997	19.4%	(18.9 - 19.9)
1998	20.6	(20.1 - 21.1)
1999	21.5	(20.9 - 22.1)
2000	21.8	(21.2 - 22.4)
2001	23.0	(22.4 - 23.6)
2002	23.9	(23.3 - 24.6)
2003	23.7	(23.1 - 24.3)
2004	24.5	(23.9 - 25.1)
2005	25.4	(24.8 - 26.1)
2006	26.4	(25.6 - 27.1)
Jan-Sept 2007	26.6	(25.5 - 27.6)

Source: National Health Interview Survey (self-reported weights and heights).
See Appendix for discussion of expected effect of self-reports

Table 6 shows data from NHANES from 1999-2002 for class 3 obesity (BMI>=40) by general age group and ethnic status, obtained by professional measurement. The most striking relativity that stands out is the very high prevalence of obesity in non-Hispanic black females, especially those with low levels of educational achievement, of more than twice that of other categories. Although stronger in females, an inverse relationship between income level and class 3 obesity has strengthened for men in the 1990s. Recent trends in the United States have seen a narrowing of the gap between those with higher income and those with lower income. In 1971-74, the prevalence was 9.7 percent for adults with annual income of more than $60,000 compared with 26.8 percent for those with less than $25,000 of annual income, while the corresponding percentages were 16.1 percent and 31.3 percent in 2001.

About 75 percent of adults with class 3 obesity also have at least one comorbid condition, such as high blood pressure or type 2 diabetes (unless specified otherwise, referred to in this paper as 'diabetes'). The prevalence of class 3 obesity quintupled between 1986 and 2000 (NHANES II to NHANES 1999-2000), while the prevalence of all those obese doubled. Thus, a disproportionate increase has occurred at the higher levels of obesity, with both class 2 and class 3 obese individuals increasing faster than those with normal BMIs, reflecting the shifting of the weight curve.

TABLE 6
Prevalence of Adult Class 3 Obesity (BMI>40)

Gender	Age	All	Non-Hispanic White	Non-Hispanic Black	Mexican American
Males	20 - 39	3.7%	3.5%	4.1%	2.0%
Males	40 - 59	3.9	4.1	2.9	4.4
Males	> 60	1.7	1.8	3.0	1.9
Males	All	3.3	3.3	3.4	2.9
Females	20 - 39	5.6	4.2	11.8	5.5
Females	40 - 59	7.8	7.0	15.1	6.6
Females	> 60	5.6	5.2	14.0	4.8
Females	All	6.4	5.5	13.5	5.7
Both	All	4.9	4.4	9.0	4.2

Source: NHANES 1999-2002

According to the self-reported BRFSS, the percent of adults who are class 3 and higher increased from 0.5 percent to 2.0 percent between 1986 to 2000, and increased again in 2005 by 50 percent, while the percentage in class 5 obesity increased by 75 percent. This increase in the number of severely obese will put increasing pressure in many areas in which “standard” equipment is used, be they in medical offices, wheelchairs or seats in airplanes.

Prevalence rates for overweight and obese men vary little by ethnic group, while non-Hispanic black women have, on average, significantly higher prevalence rates than non-Hispanic white women with Mexican American women in between. Asian men and women have significantly lower prevalence of overweight and obesity than that of other racial/ethnic groups.

In fact, evidence suggests significant ethnic and racial differences in both the prevalence of obesity and the susceptibility to obesity-related illnesses. On the other hand, overall disparities across social classes and various ethnic backgrounds appear

to have reduced over time, suggesting that there has been greater homogenization of living conditions, at least to the extent that they affect individuals' susceptibility to weight gain.

Most immigrants to the United States come from countries in which obesity is less prevalent than in the United States and are less sedentary than U.S.-born immigrants. Acculturation to U.S. norms, e.g., increased sedentary behavior and poor dietary patterns, with increased availability of calorically dense foods and higher reliance on labor-saving technologies, occurs fairly quickly, so that within 15 years their patterns are similar to those native-born (according to the 2000 Sample Module of the 2000 National Health Interview Survey (NHIS) as reported in Goel et al. (2004)).

The prevalence of obesity was about 40 percent greater for those disabled than for the non-disabled, although according to Weill et al. (2002), most of those disabled were just as likely to attempt weight loss or report exercise counseling as adults without disabilities. This was less common for those disabled with lower extremity mobility difficulties and more common for those with mental illness.

Among states participating in the BRFSS, in 1980, 10 states had a prevalence of obesity in adults less than 10 percent and no states had a prevalence rate equal to or greater than 15 percent. By 1998, no state had a prevalence rate less than 10 percent; seven states had a prevalence of obesity between 20-24 percent; and no state had a prevalence rate equal to or greater than 25 percent. In contrast, in 2006, only four states had a prevalence rate of obesity less than 20 percent; 22 had a prevalence rate equal or greater than 25 percent; while two of these states (Mississippi and West Virginia) had a prevalence rate of obesity equal to or greater than 30 percent. The large over-representation of obesity prevalence in Southern states is positively correlated with relatively high levels of diabetes and hypertension and low levels of physical activity in those states.

Some of the significant relationships between risk factors that can lead to a number of chronic diseases are given in the complicated Figure 5. In some cases these risk factors can affect one another, e.g., not only does a lack of physical activity

contribute to an increase in obesity and overweight, but once obese or overweight a person is then less likely to exercise. Some of these relationships are causative (e.g., physical activity and nutrition can contribute to overweight/obesity), while some are mitigating (e.g., medical treatment can reduce the effect of high blood pressure). The factors included in the "blood pressure, cholesterol level; chronic disease" box can interrelate with each other, e.g., diabetes interacts with cardiovascular conditions. The multiple inputs and outputs make any such single figure incomplete; it cannot provide insight to all of these and other relevant relationships, some of which are quite complex (e.g., socioeconomic factors, such as income and education, are not shown here). Each arrow can in fact be addressed in multiple papers.

A set of risk factors that in a combined manner contributes to diabetes and cardiovascular disease is called the metabolic syndrome (including obesity, insulin resistance and triglyceride fats in the blood), which, according to a 2002 study, about one-quarter of the U.S. population has and according to a 2003 study nearly a million American teenagers also have. Due to various types of measurement used in different studies, these percentages may not always be consistent by source. But in the last five years the percentage of adults with the metabolic syndrome increased by another 5 percentage points to about 31 percent. Prevalence of the metabolic syndrome by major demographic category, as taken from the NHANES 1999-2000, is shown in Table 7.

TABLE 7
Prevalence of Metabolic Syndrome in United States

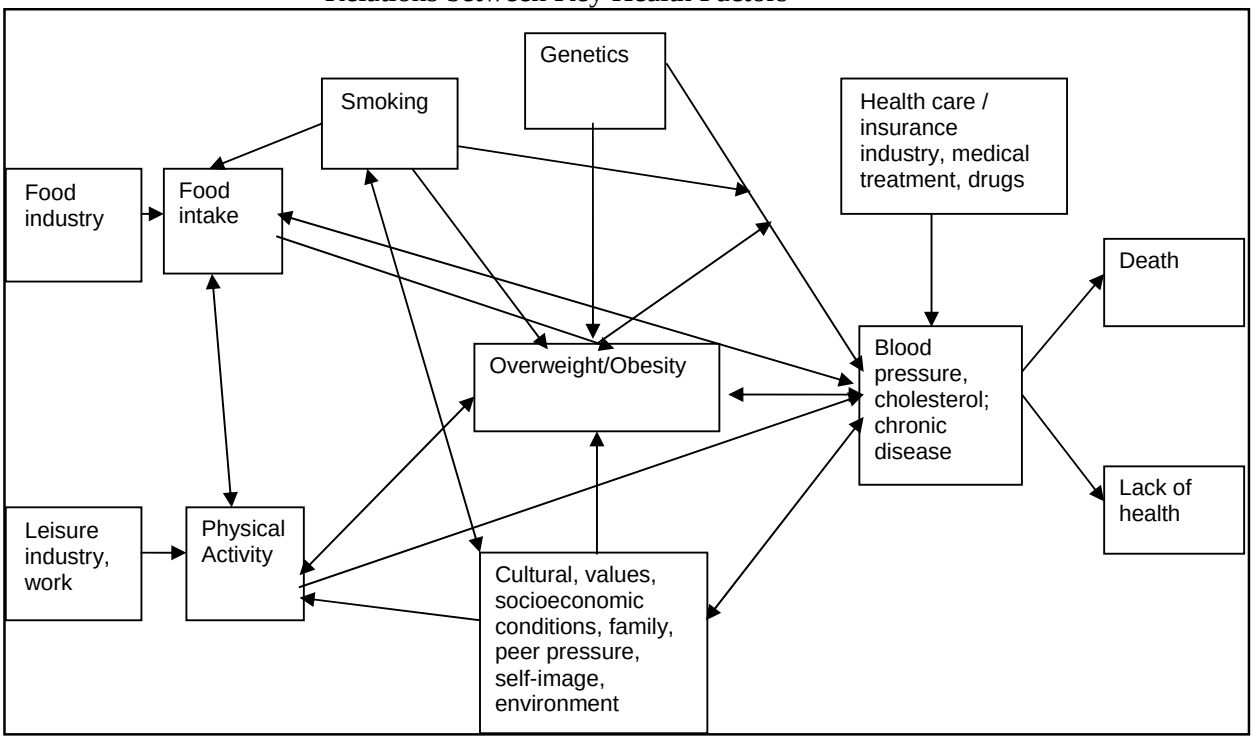
Demographic category	Women	Men
Non-Hispanic white	22.8%	24.8%
African-American	25.7	16.4
Mexican American	35.6	28.3
Other	19.9	20.9

Source: NHANES 1999-2000

Although the effect of the syndrome may not be much more than the total effect of the individual risk factors that constitute it, a speedier initiation of interventions can be taken if multiple risk factors are involved in a single individual, and a more holistic approach can be taken.

The contributors to the health of an individual at a given point in time that are reflected in Figure 5 can be assigned to five categories: (1) the starting point that is given to the individual by means of genetic transmission, including gender; (2) the external environment, including the home, the environment (e.g., medical practice and pollution) and the communities in which a person grows up and lives in; (3) the cumulative effect of individual behaviors and related mitigating factors, including nutrition, physical activity, smoking and medicine taken; and (4) the current individual risk profile, including weight, blood pressure, cholesterol level and socioeconomic characteristics, the latter of which is not included in Figure 5 for simplicity's sake only.

FIGURE 5
Relations between Key Health Factors



Barbara Hansen has argued that people don't become obese by choice; rather, they have "obesity propensity genes and physiology, of varying degrees." Over the past several decades, the environment and human behavior have facilitated weight gain, whether the result of the elimination of caloric restrictions or of reduced needs and desirability of physical activity. Certainly genetic factors exist that have recently been and will be found to predispose possessors of those genes to becoming obese or overweight. However, the rapid changes that have occurred over the last 30 years

point to a major role played by environmental, lifestyle and nutritional factors related to or caused by behavioral shifts. These two significant factors, the amount and mix of nutrition and extent and type of physical activity, possibly interacting with a given level of genetic susceptibility, can contribute not only directly to an individual's weight, but are themselves factors that can be direct risk factors of chronic disease.

Some have asked whether being obese or not being physically fit that contributes most to health problems. Although a growing number of researchers feel the latter may be more important, practically speaking, it really doesn't make much difference, as physical fitness is one of the significant tools that can be used to manage obesity and overweight in the first place, and thus is on its causal pathway. The use of weight, as expressed in terms of the BMI, has the advantage of being more easily quantifiable and comparable, as long as its limitations are dealt with appropriately.

Some individuals become overweight or obese because they have a genetic or biological predisposition to gain weight readily in an adverse environment. However, the fundamental causes may be rooted in nature of Western society, from an environment that provides incentives to live a significant portion of one's life in a sedentary manner and to consume a high fat, energy dense diet.

There are a number of theories that have been put forth to explain how the current obesity epidemic began and has continued. It can be viewed as a physiological disease with limited available cures and treatments, likely to have been caused by multiple factors, possibly interacting. Although there is clearly a baseline concern with obesity in and of itself, much of the current discussion has focused on the contributors and potential effects of the surge in obesity over the last 30 years. This suggests that behavioral and environmental influences dominate. A great deal of discussion has taken place about finding a single source or cause. For example, Jeffrey and Harnack (2007) indicated that, since all energy intake is behavioral, but less than half expenditure is behavioral, energy intake is conceptually a more appealing primary cause, although other experts disagree. Nevertheless, this argument is to a great extent misplaced, as there are undoubtedly multiple contributing elements for each particular individual.

Contributing obesity risk factors include:

- **Food intake**. In today's developed countries, food scarcities have mostly been eliminated and an attractive, diverse and energy-dense food supply has been created. There has been a marked increase in the amount of food intake in the United States over the last 30 years. In spite of substantial evidence that Americans want to lose weight, they continue to overeat. In part this is due to timing differences—food can provide instant gratification while the effect of it on health will always seem a long way off. This can be viewed as resulting from the use of a very high discount rate or simply due to a general lack of self control.

    - Insufficient use of healthy foods. Despite significant publicity, according to the 2005 BRFSS, only 22.6 percent of U.S. adults eat the recommended five or more servings of fruits and vegetables each day. What is often the high cost and inconvenience of healthy foods relative to unhealthy foods provide an inherent incentive toward an unhealthy diet.

    - Lower fat intake (e.g., 20-25 percent of energy) is needed to minimize energy imbalance and weight gain in those who are sedentary.

    - Technological factors have led to increased food consumption due to lower prices of food and less time involved in preparing it, through use of the microwave, food processing and improved packaging and storage. All of this has led to a shift in the division of labor in food production, a switch from individual food preparation within the home to mass food production at centralized shipping locations or restaurants. This in turn has led to a decrease in the time cost of food consumption and thus to an increase in the quantity and variety of food consumed. All of these factors have contributed to an increase in the amount and type of food input. The fact that many high energy foods taste good

(often caused by inputs such as fat and sugar, referred to as ”palatability”) helps increase their popularity as well.

- Portion size. Nielsen and Popkin (2003) reported on the results of a study based on the National Food Consumption Survey 1977 and the Continuing Survey of Food Intake for Individuals 1989 and 1996. Between 1977-78 and 1994-96 there has been a marked trend toward larger portion sizes in the United States both inside and outside the home. Portion sizes increased for salty snacks (58 percent), desserts, soft drinks (by 62 percent), fruit drinks (by 48 percent), french fries (by 57 percent), hamburgers/cheeseburgers and Mexican food. Of the food types studied, only the pizza decreased in size. In fact, the size of the increases is substantial, with the impact of increases ranging from 0.3 to 1.7 oz in weight per item. In many restaurants the use of “doggy-bags” is common, an indication of the demand for larger servings; eventually much of this additional food is eaten later as a snack or another meal.

  For example, in 1960 a portion of McDonald's french fries consisted of 200 calories, while in 2005 it consisted of 600 calories. The average energy intake and portion size of salty snacks increased from 132 to 225 kcal (1.0 to 1.6 oz).

  The size of burgers when fast food restaurants began to be popular is now no larger than many children's portions today. The size of some food items that were sold as “standard” (e.g., 6 or 8 ounce size of soft drinks) are no longer being sold at all.

  Couples in movie theaters in the 1950s shared three-cup bags of popcorn with 174 calories, while today moviegoers munch 21-cup bags smothered in butter and loaded with 1,700 calories. Full-service restaurant portion sizes were small when compared with fast-food restaurants and at home. Between 1994 and 1998, the largest portions

for most food portions were found at fast food restaurants. Interestingly, the location of the largest size of dessert was at home.

The average size chocolate bar has doubled. Just the other day in an American airport, I asked a convenience store clerk where the regular size candy bars were. The response I received was that they only carried the king size, because there was so little demand for the regular size anymore. It appears that supply has matched the demand of consumers.

Rozin et al. (2003) reported on the “French Paradox”—the French mortality rate from heart disease is lower than Americans and their average BMI is smaller (24.4 for French adults in 2002 compared with 26.6 for American adults, according to the NCHS), even though the French have a higher average blood cholesterol level and a diet containing more total and saturated fat and less of fat-reduced foods. They found that the French eat fewer total calories than Americans, as evidenced by smaller portion size in comparable restaurants, in the sizes of individual portions of foods in supermarkets, in portions specified in cookbooks, and in the prominence of “all you can eat” restaurants in dining guides, as well as snacking less. In comparing the portion size of McDonald's in Philadelphia to that of Paris in 2001, Rozin et al. (2003) reported that the ratio was 1.28:1.00, with a range of 1.0-1.94 in seven McDonald's. Interestingly, even though the French eat less, they appear to take a longer time eat their food.

According to the NHANES II, III and continuous, conducted in 1976-80, 1988-94, and 1999-2002, respectively, total energy intake for males on an age-adjusted basis increased from 2,439 to 2,666 to 2,634 kcals, respectively (about 9 percent increase between the first two surveys), and for females on an age-adjusted basis, increased from 1,522 to 1,798 to 1,874 kcals, respectively (about an 18 percent increase between the first two studies).

- Food industry pricing. Many fast food restaurants and snack foods include a large fixed price component, with the charge for larger sizes being quite small. This relatively small incremental price for a large additional quantity of food leads to an incentive to over-buy quantity relative to food needs. In addition, all-you-can-eat restaurants are always a test of one's self-control and ability to stop eating when full. These test one's willpower and desire to take advantage of apparent value.

- Increase in the number of sit-down and fast food restaurants, both in terms of the number of outlets and customers served. Increased urbanization and suburbanization have made such food distributor methods more commercially viable and easy to get to. Perceived need tends to drive availability, which in turn has increased usage. In addition, growing use of fast food among young adults in the United States increases the social acceptability of that type of food and restaurants. In part as a result, it has been found that urban residents consume smaller proportions of carbohydrates and larger proportions of protein and fat, particularly saturated fat.

- High-fructose corn sweeteners in soft drinks. These drinks currently contribute 7.1 percent of total energy intake of Americans and represent the largest single food source of calories in the U.S. diet. They were introduced about 1970, around the time that the average American began to gain weight. U.S. consumption of sweetened drinks increased by 135 percent between 1977 and 2001, and soft drink consumption increased by 61 percent in adults from 1977 and 1997. Meanwhile, milk consumption decreased by 38 percent.

  The Nurses' Health Study II (an eight-year follow-up study reported on by Weinstein et al. (2004)) indicated that women who increased their consumption of sugar-sweetened soft drinks during 1995-99 gained 17.6 pounds between 1991 and 1999, whereas women who decreased their consumption during this period only gained 6.2 pounds. Women

who increased their use of sugar-sweetened soft drinks also increased their reported total energy intake, while those who reduced their sugar-sweetened soft drink consumption also reduced their total energy consumption. Use of fruit punch had a similar effect (although fruit juice and diet soft drinks generally did not). Those who increased their use of sugar sweetened soft drinks also tended to be less physically active and to smoke more. The message is that those with healthy habits tend to have them across the board, and likewise for those with unhealthy habits.

- Widespread and effective food advertising.

(further discussion is in Section 5.5.1)

- **Lack of physical activity**. Modern living has facilitated a more sedentary lifestyle for many Americans. Contributing factors have included the increase in labor-saving devices at work, home, transit and play, while leisure time activities increasingly involve passive entertainment, such as watching television, playing video games and surfing the internet. Those who are already overweight or obese tend to be less active than those who aren't, both because of their overall lifestyle choices and the difficulty in being more active when obese. For a significant part of the population, particularly those who are of low income, work two jobs, live in an insecure area, have young children or are elderly and have never exercised before, there may be a lack of opportunity and motivation for regular exercise.

  Some relevant physical activity facts:

  - In 2006, Nielsen Media Research, Inc. indicated that a television was playing on average eight hours and 14 minutes daily in American households, while an average American watches four hours and 35 minutes every day. This is part of the overall trend to substitute sedentary leisure activities (TV, video games, the internet) for active activities.

- In 1999, 57 percent of American adolescents watched television more than two hours daily, with Hispanic and non-Hispanic blacks 50 percent more likely to watch TV at least this much.

- Black women report less frequent participation in exercise than white women. However, the relative importance of whether their higher level of obesity is in part due to this lack of exercise or whether their obesity contributes to this relative lack of exercise is uncertain.

- Although there has been a reduction in the level of physical intensity in the workplace, in most developed countries the more significant move away from manual work occurred decades ago, far before the rapid rise in obesity. It has also been pointed out that the recent increase in BMIs has been similarly experienced by children and older adults, both of whom do not work. Thus, although this may be a contributing factor over the long term, it does not appear to have been a significant driver toward the recent upsurge in obesity.

- Living conditions. Perceived safety concerns and urban/suburban sprawl with fewer sidewalks may influence walking behavior and physical activity outside the home. However, more housing clusters with workout rooms and walking paths, if they are used may offset this trend.

- Diminished use of dietary fats as fuel. More than half of U.S. adults do not meet the recommended amount of physical activity to provide the optimal amount of healthy benefits.

- Simply being “lazy” or not viewing physical activity to be sufficiently important to be worth the extra effort, e.g., by taking the elevator when a flight of stairs will work, or by driving into the town center which is only a three-block walk. Philipson (1999) indicated that some who work in offices often prefer e-mailing or instant-messaging to walking down the hall and talking with a fellow worker.

(Further discussion is in Section 5.5.2.)

- **Genetics**. Although population genetics change too slowly to be blamed for the recent rapid growth of fatness, genes can and do play a role in the process. A common genetic explanation for the increase in obesity is the mismatch between today's environment and the “energy-thrifty genes” that have multiplied under new environmental conditions.

  Henig (2007) indicated that there are 50 genes involved in developing obesity (e.g., in how fat is distributed and metabolized, the desire to eat and methods of using up calories). Two, FIT1 and FIT2, were found in December 2007 to be responsible for storing fat. Other scientists have found that differences in levels of genetically determined dopamine, a neurotransmitter that helps make activities and substances seem more rewarding, can provide motivation and rewards to eat and can motivate them to eat more.

  Obesity results from the interaction of genetic variation and changing environmental conditions through a series of predispositions and susceptibilities. Scientists simply do not yet know enough about these interactions to identify the silver bullet cause. But unless the factors that generate change have increased significantly in the last 30 years, genetics alone can't be blamed for the recent trends. Nevertheless, the differential effects of the contributing inputs to obesity indicate that there are people who are more susceptible to weight gain and the development of obesity than others, whatever the mechanisms that are involved.

  A number of studies of twins have evaluated the relative contribution of nature (genetics) versus nurture (environment) on weight, with relatively mixed results. According to Segal and Allison (2002), "Previous studies have estimated that genetic influences may explain approximately 25-75 percent of within-population variations in relative weight and BMI. Studies of twins reared apart and together have suggested that genetic effects explain 50-90 percent of the variation in BMI." They found in two separate studies conducted in four large U.S. cities of about 300 sets of

twins through age 9, including virtual twins (same-age unrelated siblings, with effects studied simultaneously), that genetic effects contributed about 60 percent of obesity which 40 percent arose from a shared environment. In a larger British study of more than 5,000 8- to 11-year-old twins (Wardle et al., 2008), a stronger genetic influence was suggested, contributing about 77 percent for BMI, about 10 percent from a shared environment and about 13 percent from non-shared environmental effects. Finally, on the basis of a study of 50 twin studies, Bouchard et al. (2003) concluded that genetic factors were likely to contribute 25 percent to 40 percent of the variations in the subjects' weight and fat mass. In summary, these studies all indicate a significant effect of both genetics and the environment.

- **Other biological factors**
  - Slow metabolism. The individual's metabolism is elastic up to a certain level of daily variation, but after a point the metabolism level can affect weight.
  - Lack of sleep. This can jolt the metabolic system into demanding doses of instant energy.
  - Gut bacteria. The existence of certain types of stomach bacteria can contribute to greater weight.
  - Increased stress and mental health problems, including depression and anxiety. These can result in eating more and being less active, although there may be at least a component of reverse causation at work to be certain of the severity of this relationship.
  - For women, menopause transition and hysterectomy.
  - Underlying illness, such as hypothyroidism.
  - Certain medications, e.g., atypical anti-psychotics.

- **Social factors**
  - The Agras and Mascola (2005) review found that parental overweight was the most potent risk factor in determining a child's overweight status.

- Two income households in which neither parent remains at home to look after the house, tends to increases the number of restaurants and take-out meals. This can also lead to a “TV dinner” approach to meals.
- The obese tend to marry other obese. This can amplify bad habits in the current and in future generations and may imply a certain level of genetic vulnerability over a long period of time.
- The obese are more likely to live where they can get around by car, thus reducing even further their physical activity.

- **Other behaviors**. Some examples include giving up smoking (see Section 3.2 for further discussion) and eating too fast that may not provide sufficient time for the full effect of satiety signals from the digestive system to work.

- **External environment**
  - Urbanization/suburbanization.
  - Industrial chemicals, persistent organic pollutants, are reported to be a disruptor of the endocrinology system that can accumulate in fatty tissue that in turn may contribute to diabetes.
  - Air conditioning.

- **Education.** Lack of awareness of the contributing factors to and dangers associated with obesity. (see Section 5.5.3 for further discussion of socioeconomic factors)

It is clear from these factors that obesity (and overweight) is a multifaceted and complex issue, particularly since these factors are so interrelated.

Rashad (2006)'s closed form model identified several significant contributors to obesity. It was found that the price of a restaurant meal, the change in number of cigarette smokers (especially for Hispanics), caloric intake (for females), education and income for married males and males with higher income were significant factors. Eating at fast food and related restaurants has contributed to increased caloric intake and is associated with being less active, less likely to prepare food at home and tend

not to travel further distances to obtain a healthy meal. Married men with higher incomes tend to have a higher BMI.

Even though the U.S. government has publicized a series of nutritional and exercise guidelines during the last 30 years and has a set of overall population goals for 2010, it is unlikely that many of these will be met. For example, in 1997 less than one-third of adults engaged in at least 30 minutes of moderate physical activity most days of the week, and 40 percent of adults engaged in no leisure-time physical activity at all. In addition, only 3 percent of all individuals meet four of the five U.S. Department of Agriculture recommendations for the intake of grains, fruit, vegetables, dairy products and meat.

5.1.1 Older adults

Flegal et al. (2002) indicated that between NHANES III (1988-94) and NHANES 1999-2000, the obesity prevalence rates of those aged 60-69 increased significantly to become the age group with the largest percentage of obese. During the 1990s, the distribution of weight in those in their 60s and 70s, especially non-Hispanics, shifted significantly to the right in a manner similar to what has occurred to those of younger ages, with a greater number of those at the higher obesity levels.

According to Michaud et al. (2007), based on the 2004 U.S. Health and Retirement Study, about 52 percent of men older than 50 hardly ever engaged in physical exercise, while 61 percent of females older than 50 hardly ever did. The prevalence of obesity in these inactive American males was about 66 percent. For females over age 50, this compares with 60 percent from Spain, 40 percent from northern Europe and 50 percent from elsewhere in Europe. In addition, American men over age 50 watched more television than European men (more than four hours per day compared with more than three hours per day), while U.S. females over age 50 spent about half the time at home cooking and had less physical activity than European females of similar age.

5.2 Children (Through Age 19)

In 1956, President Eisenhower established the President's Council on Youth Fitness, based on a 1953 paper by Dr. Hans Kraus, who found that U.S. children were less fit than those in Europe. He blamed U.S. lifestyles, e.g., the use of cars, school buses and elevators instead of walking or climbing stairs.

Today's youth are considered by some to be the most inactive generation in history. There are many contributing causes to this, but simply put there are fewer opportunities to take physical exercise. Factors include reductions in school physical education programs, parental security concerns due to seemingly unsafe community recreation facilities and neighborhoods and the substitution of sitting-on-a-chair activities for physically active leisure. In prior generations, children played games outside in the neighborhood in good weather. Now they are more likely to have play-dates in others' homes, watching TV or DVDs or playing video games.

It is common to place a certain amount of blame on soft drink manufacturers and fast food restaurants. Advertising budgets and the scale of use of unhealthy food and drink are indeed huge. For example, soft drink companies have aimed advertising campaigns at young children in order to develop lifetime brand loyalties and enhance market share at the same time. Many soft drinks and related drinks contain sugars and corn sweeteners with few essential nutrients. In part as a result, nearly 25 percent of adolescents drink more than 26 ounces of soft drinks per day, which provides at least 300 kcalories. Children who consume soft drinks take in fewer nutrients but more calories, resulting in a tendency to gain weight. At the same time, they become less likely to consume fruits, vegetables, juice and milk.

In addition, 60 percent of U.S. middle and high schools sell soft drinks in vending machines, although efforts continue to change this practice. In 2002, it was estimated that 240 U.S. school districts had entered "pouring rights" contracts with soft drink companies, giving the schools cash and other incentives in exchange for the rights to sell sodas in vending machines and to include public advertisements for their products.

Table 8 shows that the percentages of children and adolescents who are overweight have grown significantly over the past 30 years. During the 1960s and

1970s, these percentages were relatively stable, in the 4-6.5 percent range. But since then until at least the most recently published NHANES (2003-04), the percentages have increased, in some cases by three or more times, to the range of 13.9 percent to 18.8 percent, depending on age. Just in the short period between 1999-2000 and 2003-2004, the percentage overweight increased from 14.0 percent to 18.2 percent for boys and from 13.8 percent to 16.0 percent for girls; there is no reason to believe that this increasing trend has reversed itself since then. During the last period reported on, about 12.5 million adolescents were overweight.

TABLE 8
Trends in Prevalence in Overweight of U.S. Children and Adolescents between 1971-2004

Age (years)	NHANES I 1971-74	NHANES II 1976-80	NHANES III 1988-94	NHANES IV 1999-2000	NHANES 2001-2002	NHANES 2003-2004
2-5	5%	5%	7.2%	10.3%	10.6%	13.9%
6-11	4	6.5	11.3	15.1	16.3	18.8
12-19	6.1	5	10.5	14.8	16.7	17.4

Source: National Center for Health Statistics

The prevalence of overweight reported by the combined 2001-04 NHANES for children (ages 6-11) and adolescents (ages 12-19) is shown in Tables 9 and 10. In addition, according to the National Longitudinal Survey of Youth (NLSY) during 1986-98, overweight prevalence was greater for youths in an urban setting and in the U.S. South, with black and Hispanic youth experiencing significantly greater rates of being overweight than non-Hispanic whites, while those whose family income is less than 150 percent of the poverty line had a greater prevalence than those youth with greater family income. Other studies have found that, unlike adults, children in the poorest families are less likely to be overweight and have lower BMIs than others.

Among U.S. children, black girls and Mexican-American boys have the highest rates of obesity. Immigrants face both the social norms and habits of their new country and maintenance of their original culture, including those of diet and exercise. It has been noted, however, that over the years between 1985 and 2004, the gap in some areas has narrowed, e.g., between white and black boys, and between white and Mexican-American girls. Mexicans and Mexican-Americans face the same underlying trends in both Mexico and the United States, with (1) diets increasingly including more high-energy, less nutritious foods being chosen over fruits and vegetables as staples; (2) low physical activity in school; and (3) a trend toward more screen-

viewing activities. Differences by socioeconomic status do exist -- in Mexico, a higher socioeconomic status is more often associated with obesity, in part due to a perception that being plump may be viewed as being healthy, while in contrast in the United States those of lower socioeconomic status tend to be heavier.

TABLE 9
Trends in Prevalence of Overweight of U.S. Children ages 6-11 between 1976-1980 and 2001-2004

Gender	Ethnic group	NHANES 1976-80	NHANES 1988-94	NHANES 2001-04
Boys	Non-Hispanic whites	6.1%	10.7%	16.9%
	Non-Hispanic blacks	6.8	12.3	17.2
	Mexican-American	13.3	17.5	25.6
Girls	Non-Hispanic whites	5.2	9.8	15.8
	Non-Hispanic blacks	11.2	17.0	24.8
	Mexican-American	9.8	15.3	16.6
All	< 100% poverty line	--	11.4	20.0
	100%-199% poverty line	--	11.1	18.4
	> 200% poverty line	--	11.1	15.4
Boys	Total	6.6	11.6	18.7
Girls	Total	6.4	11.0	16.3
All	Total	6.5	11.3	17.5

TABLE 10
Trends in Prevalence in Overweight of U.S. Adolescents Ages 12-19 between 1976-80 and 2001-2004

Gender	Ethnic group	NHANES 1976-80	NHANES 1988-94	NHANES 2001-04
Boys	Non-Hispanic whites	3.8%	11.6%	17.9%
	Non-Hispanic blacks	6.1	10.7	17.7
	Mexican-American	7.7	14.1	20.0
Girls	Non-Hispanic whites	4.6	8.9	14.6
	Non-Hispanic blacks	10.7	16.3	23.8
	Mexican-American	8.8	13.4	17.1
All	< 100% poverty line	--	15.8	18.2
	100%-199% poverty line	--	11.2	17.0
	> 200% poverty line	--	7.9	16.3
Boys	Total	4.8	11.3	17.9
Girls	Total	5.3	9.7	16.0
All	Total	5.0	10.5	17.0

Source: NHANES

According to a review of trends in BMIs by birth cohorts of children and adolescents reported by NHES and NHANES between 1940 and the 1990s through 2006 conducted by Komlos et al. (2008), there has been an increase of about 5.6, 3.3, 2.4, and 1.5 BMI units, respectively, for non-Hispanic black girls, non-Hispanic black

boys, non-Hispanic white boys and non-Hispanic white girls. While the increase in BMI values began among the birth cohorts of the 1940s among black girls, the rate of increase accelerated among all four groups born in the mid-1950s to early-1960s. The rate of increase leveled off somewhat thereafter. There is some indication that among black boys and white girls born after about 1990, the level of adiposity has remained unchanged or perhaps even declined. Komlos inferred by their analysis of these trends that the incremental weight increases have been associated with the labor-saving technological developments of the twentieth century that brought about many significant cultural and nutritional shifts, including cars, TV and internet viewing. They ended their paper with "The obesity pandemic among US-born children and youth can be seen as the outcome of the combined incremental effects of the continuous and multifaceted technological, cultural and nutritional changes of the 20th century."

In analyzing NHANES III, Wang (2001) indicated that after income and urban-rural residence were adjusted for, the effect of ethnicity became insignificant, which may suggest that socioeconomic status differences across ethnic groups may be a more significant explanation for the difference in obesity prevalence across ethnic groups, especially among adolescents after age 10. Nevertheless, black and Mexican-American girls were more likely to have a higher BMI than white girls even when family income and urban/rural residence were controlled for.

As reported in 1976-80 and 1999-2000 in NHANES, the percentage of 6-23 month olds that are overweight increased from 7.2 percent in 1976-80, to 8.9 percent in 1988-94 and 11.6 percent in 1999-2000. Although it has not been established that weight at these very young ages will be carried forward until adulthood, this does confirm that the entire population has been affected by the overall population trends.

Some of the causes of the increasingly overweight in children and adolescents include:

- **Physical activity**.

- Physical education. Some schools have cut back on both recess and physical education, while many physical education requirements are limited in scope or not enforced. The CDC indicated that the percentage of high school children who take part in daily exercise dropped from 42 percent in 1991 to 28 percent in 2004. However, in 2007, a CDC survey indicated that more states and school districts are now insisting that elementary schools schedule recess and that physical education teachers have at least an undergraduate training.

  Be that as it may, several studies have found no detectable impact of physical education classes on BMI levels, even with a greater amount of physical activity. It appears that relatively little of the time spent in many physical education classes is spent exercising or playing sports. More than two hours per week of physical education is needed for students to self-report that they exercised vigorously for at least 20 minutes or did any strength-building exercise.

- School transportation. As distance to school has increased, fewer students are walking or biking to school. According to a telephone survey conducted in 2004, just 48 percent of children who even live within a mile of school walk or bike to get there, compared with 90 percent who did so in 1969. Another survey indicated that in 1969 42 percent of those who lived further than a mile walked or biked, while just 16 percent did so in 2001. This may be due in part because of safety concerns, lack of sidewalks, concern about peer pressure indicating that walking is not cool and a general lack of concern about the benefits of exercise. Children of well-educated parents are more likely to get a ride to school, in part because there are more cars in a family and a parent that doesn't work full-time. In addition, some children live so far away from school that by the time they get home after school-related activities, there isn't time to play and get exercise.

- Although 80 percent of children are regularly active in some way, rates of inactivity among adolescent girls, non-Hispanic blacks and Mexican Americans are greater than among non-Hispanic whites.

- The drive for more activities and higher exam results and grades has resulted in lower levels of physical activity and school sports. Between 1981 and 1997, free time has declined, as a result of a decrease in time spent in front of the TV, playing and other passive entertainment and an increase in time in school and daycare, studying, reading and activities after school.

- It has been estimated that it takes one to two hours of extremely vigorous activity to offset a single large-sized children's meal at a fast food restaurant. It is thus quite difficult to offset these convenience meals in any event.

- **Screen viewing (television, video-game playing and internet usage)**.

A 2004 meta-analysis found that time spent watching television was consistently linked with being overweight, although the association was relatively weak. Hancox and Poulton (2006) concluded that, although the relation between obesity and TV watching is somewhat stronger for teenage girls, it is weaker and often not significant among boys. It did not appear that TV viewing was a significant cause of obesity, although this may have been due to the fact that the children in the Hancox study were not particularly obese and by age 26, 41 percent of them were either overweight or obese.

Nevertheless, although the effect of TV watching does not appear to be large, it has altered what was previously considered to be normal childhood behavior, making it more difficult to play informal team sports after school if the neighbors are at home watching TV or on their computers and it appears to be a better predictor of BMI and either dietary intake or physical activity. Nevertheless, in studies such as Biddle et al. (2005) covering Scottish youth, the relationship between TV viewing and physically active behaviors were usually seen to be small, with the time spent after school being earlier in the evening than TV viewing. Because of the range of sedentary activities now available, a single intervention will not likely “solve” the problem. It may simply be better to encourage active activities and time spent outside.

Sedentary activities themselves are not the only contributor to increased weight, as viewing can also indirectly increase dietary energy intake by snacking while viewing or later as a result of food advertising. Anderson et al. (1998) found that, based on NHANES II (1988-91), leisure time activities, including TV viewing, video games (and if the study had been conducted more recently, internet use and instant messaging), have contributed to the increasing prevalence of overweight in America. This study also found a relationship between television watching time, physical activity and body composition, with the children who watched more TV being less likely to participate in vigorous activity and having higher BMIs.

NHANES III indicated that one-quarter of all U.S. children watched four or more hours of TV each day, as did 43 percent of non-Hispanic blacks. Forty-three percent of American students in grades 9 through 12 viewed television more than two hours per day. In 2007, the American Medical Association's Council on Science and Public Health estimated that 5 million American children may be addicted to video games and urged that their behavior be considered a psychiatric disease.

Active Healthy Kids Canada gave children an overall 2007 grade of D, as "children and youth report spending twice as much time in front of a screen as they do engaged in physical activity, decreases in physical activity and increases in screen (a TV, computer or video-game console) time are not only contributing to increases in overweight and obesity, but are now also associated with increasing reports of anxiety, depression and low self-esteem. (In addition), inactivity significantly worsens as children grow older, and teenagers, especially teen girls, are less active now than they ever have been."

- **Food intake**

  As one of the basic energy inputs, changes in food intake must be examined as a probable contributor to childhood obesity. The following are some of the key factors that might be involved.

  - School provided food. Only 17 states require that school meals and snacks meet the higher nutritional standards that the U.S. Department of Agriculture

requires, while 24 states do not limit where and when such foods can be sold on school property. In 2004, the U.S. Congress passed an act directing school districts that receive money from national school-lunch programs to create wellness policies by the start of the 2006-07 school year, setting standards for nutrition, physical activity and nutritional education. One year after the deadline, results were reported to be mixed at best. French fries and cookies were still often available alongside salads, juice and fresh food. Often students are not provided sufficient encouragement to eat well. However, schools offering fried potatoes have declined to 19 percent from 40 percent over the last seven years. In the fall of 2007, fewer than half of the schools had implemented the nutrition-education guidelines and enforced vending-machine rules, although 30 states have banned junk food from their vending machines, up from four in 2000, with 64 percent meeting physical education requirements. More could be done, with progress appearing good only because many schools started from such a low point.

- Soft drink consumption. Consumption by children and adolescents of soft-drinks more than doubled between 1977-78 and 1994-98 (Schulze et al., 2004). By 2001-02 soft drinks constituted about half of total beverage intake, a 58 percent increase since 1997. Evidence has suggested that there is an association with the intake of sugar-sweetened soft drinks and the risk of obesity in children.

- Milk consumption. Between 1970 and 1997, the U.S. Department of Agriculture surveys indicated an increase of 118 percent of per capita consumption of carbonated drinks and a decline of 23 percent of beverage milk. According to Dehghan et al. (2005), although soft drink intake has been associated with the epidemic of obesity and type 2 diabetes among children, "it cannot be concluded definitely that sugar containing soft drinks promote weight gain because they displace dairy products."

- Vegetable consumption. According to the U.S. Department of Agriculture, between 1997 and 2002 consumption by children of vegetables decreased by 42 percent and by adolescents 32 percent.

- Children may be particularly at risk if they still took a bottle to bed at age 3. A 2007 study indicated that 14 percent of Hispanic, 6 percent of white and 4 percent of black children did so. Low socioeconomic status may be a risk factor regarding overweight among children as young as age 3.

- Black and Hispanic children are more likely to eat a hot school lunch.

- The number of meals eaten outside the home has increased, although the nutritional value of those meals is uncertain.

- **Socioeconomic factors**

  More than one third of poor, inner-city toddlers are overweight or obese, according to a 2006 survey of 20 U.S. cities. These children are already disadvantaged because their families are poor, and by age 3 they can expect a lifetime of health problems. Thirty-five percent of all children in the study and 44 percent of Hispanics are overweight or obese. It is difficult to afford fruits and vegetables and to go outside and exercise if the neighborhoods aren't safe. Some Hispanic mothers believe that chubby children are healthier and are more likely to put their toddlers to bed with bottles and to pressure kids to eat even when they aren't hungry.

  Children from poorer families do not necessarily have a larger BMI than children from high income families. Children from families just under the poverty line have been the ones with the lowest probabilities of being overweight, with those just over the poverty line being most at risk of being overweight.

  Gunderson et al. (2008), based on a six-year study of the first wave of Welfare, Children, and Families: A Three-City Study of low-income households in Boston, Chicago and San Antonio starting in 1996, indicated that, even though half of the 1,031 children studied who were between 10 and 15 were overweight or obese, about 8 percent had food insecurity, that is, weren't getting enough to eat. This contrasts with a nationwide rate of food insecurity of about 35 percent when a child's household income is below the poverty line, or about 12 million families.

Food insecurity is typically associated with a range of medical problems, including diminished psychosocial functioning, frequent stomachaches and headaches, worse health outcomes, increased odds of hospitalization and seeing a psychologist, behavior problems and iron deficiency.

The authors could not explain why food insecurity and overweight coexists in low-income children, although they suggested several possible reasons, including over-consumption of less expensive energy-dense foods, overeating when food is more plentiful, metabolic changes to ensure a more efficient use of energy, different standards of what constitutes an adequate diet, and parents who overprotect their children by giving them more food than needed when food is available. Bottom-line, although food insecurity and overweight have recently been linked, there were no significant differences in this study between being overweight or at-risk of being overweight and being food insecure when measured for the same child. The authors indicated that prior studies had suggested that poor children weren't getting nutritious food and ate junk food instead.

It appears that the quantity of food purchased increases with income to a certain point, and in high income families more food expenditure most likely leads to higher quality rather than quantity of food. More frequent eating out also represents a greater risk of a higher BMI.

According to Anderson et al. (2007), the NHANESs since NHANES I indicated that there has been a trend for those children of a higher income-to-poverty family income to experience a higher relative increase in the rate of obesity.

- **Ethnic factors**

  The increase in prevalence of overweight between NHANES II and NHANES 1999-2000 was markedly greater for both children (ages 6 and 11) and adolescents (ages 12-19) who are non-Hispanic blacks and Mexican Americans. However, waist circumference values that roughly correspond to overweight and obesity BMI levels were substantially lower in non-Hispanic blacks and Hispanic Americans.

Whitaker et al. (2006) indicated that for selected racial groups of a similar socioeconomic status, 3-year-old Hispanics were at a 50 percent higher risk of obesity than non-Hispanic whites. It has long been suggested that these ethnic groups react differently to certain diets and are more prone to certain health conditions, including obesity and diabetes, but no one has identified the genes responsible for this difference yet. Disparity appears to develop early in life that is unlikely to be caused by the environment.

Whenever the results of studies are generalized to cover all Hispanics, it has to be remembered that the Hispanic category consists of several nationally or regional groups, each of which may have different characteristics. Recent immigrants have tended to be healthier, thinner and experience longer life expectancies than longer-term Hispanic residents who have acculturated with American habits such as the use of fast foods.

- **Sleep**

  A study conducted by Lumeng (2007) suggested that children who don't get enough sleep also may be at increased risk for being overweight. Boys seem to be more sensitive to this effect. This apparently works through disruption of hormones that regulate fat storage, appetite and glucose metabolism. It seems that instant messaging friends after midnight or playing massive competitive internet games may not be healthy after all.

- **Parental situation**

  One or both parents working outside the home and working longer hours can affect what, where and how children eat. Cawley and Liu (2007) found that, using the American Time Use Survey, employed mothers spend significantly less time cooking, eating and playing with their children and are more likely to purchase prepared foods. This was only partially offset by the efforts of their husbands or partners.

  Whether children's parents are obese can be a contributing cause, although the mother's weight has usually been shown to be more important. Anderson et al. (2007) found, based on NHANES, that a 10 percent increase in either mothers' or

fathers' BMI is correlated with about a 1.4 percent increase in child's BMI in NHANES I and II and about a 2.0 percent increase in NHANES III and 1999-2004. Although this might have occurred because of a common environment or genetics, based on the recently rapid increase in children who are overweight, it now appears to be more likely the former. The parents' BMI explains 37 percent of the increase in children's average BMI over the last 30 years.

Several studies have indicated that women with gestational diabetes have offspring with considerably greater birth weights and a greater risk of obesity and diabetes in later life.

- **Psychological reasons**
  - It is common for children, particularly adolescents, to gain weight when they are depressed, anxious, sad or lonely. Likewise, being obese can result in psychological problems as well. Peer pressure to eat unhealthy food and be physically inactive can be intense and difficult to overcome.

  - Even when it is easy to recognize the problem, it is quite another thing to develop sufficient motivation to translate that knowledge into action.

It should be kept in mind that you can't always generalize the results of studies of children to adults, and vice versa.

5.3 Does Childhood Overweight Lead to Adult Obesity?

Although it is not possible to determine which children will become obese as adults, being overweight when young appears to predispose a person to being overweight in adulthood. The correlation between BMIs in childhood and adulthood is not particularly strong—generally an r less than 0.5 from youth to age 35 and 50, although it is somewhat higher between adolescence and adulthood. Based on a review of eight prospective studies, one-third of obese preschool children and about one-half of obese grade school children became obese as adults; the more obese the child, the more likely he or she remained obese when an adult. In at least two earlier long-term studies, 80 percent of overweight children remained markedly overweight

when reexamined 20 or 30 years later. Koplan et al. (2004) indicated that nearly 50 percent of overweight children and adolescents will become obese adults.

Adolescent obesity might have relatively more harmful long-term effects in women than in men. Even when adult BMI was factored in, a Norwegian study of women who were obese as teenagers found that they were about 30 percent more likely than those with an average teenage BMI to die by middle-age.

A large scale study of more than one quarter of a million Danish schoolchildren was followed through early adulthood (Baker et al., 2007), initially measured between ages 7 and 13 for boys and ages 10 and 13 for girls, born between 1930 and 1976. They subsequently underwent mandatory annual physical examinations. This study covered all socioeconomic groups, although all were Caucasian due to the nature of the population of Denmark. Fatal and non-fatal coronary heart disease (CHD) events were studied. The rate of both event types increased monotonically by BMI level in childhood, that is, the risk was higher for higher BMIs at a given childhood age. Girls showed the same pattern as boys, but at a lower level (that is, constituted lower overall CHD risks). The association between BMI and CHD risks was greater for deaths. There was no association between event risk and birth weight, as there was little difference by birth cohort. The strength of the relationship was far stronger at BMIs measured at older adolescent ages. Each one unit increase in BMI resulted in a significant increase in the risk of a CHD event.

The authors of the Danish study speculated that a similar study conducted in Finland did not show this strong relationship because of its far smaller size. A somewhat comparable study of 2,499 British students showed a similar relationship for boys and girls combined, but did not do so when split by gender.

In contrast, the Aberdeen study (of 11,106 Scottish children measured at an average age of 4.9 with about a 24 year follow-up period) reported by Lawlor and Leon (2005) found no adverse CHD mortality associated with childhood BMI. They found that the 2.5% heaviest children did experience a hazard rate of 2.41 adjusted for gender, father's social class, number of siblings and birth weight of the corresponding mortality due to stroke of the average of all other children studied due to stroke.

Nevertheless, if the baseline measurement had been made at an adolescent age instead, a stronger relationship with CHD mortality might have been observed. Lawlor and Leon did indicate that most, but not all, previous studies had found a relationship between CHD mortality and BMI.

The Danish study indicated a linear relationship among BMI values, indicating that large BMI groupings do not capture the total effect of BMIs. The authors projected that the probability of a 13-year-old boy 24.6 pounds (11.2kg) greater than average of having a CHD event by age 60 was one-third more likely than one with an average BMI.

For children and adolescents with BMI above the 95th percentile at any age during childhood, the results of the Fels Longitudinal Study indicated that the probability of being obese at age 35 ranged from 15 percent to 99 percent. Whitaker (2006) indicated that three-quarters of teenagers who are overweight will grow up to be overweight or obese adults, although the effects of weight at very early ages (under age 3) are relatively weak. Early-onset obesity (after age 3), together with parental obesity (either due to their genes or a shared family environment), explain a disproportionate fraction of adult obesity, increasing with the severity of the obesity. For male adolescents, Monheit (2007) found that having a mother who is obese increases the chance of being overweight by 13.7 percent and a father who is obese by 7.4 percent, with similar percentages for females. With the recent increase in levels of obesity, this hand-me-down obesity does not bode well for the future.

Wright, et al. (2001), reporting on the results of long follow-up period Thousand Families Cohort Study from 1947 in Newcastle upon Tyne found that children in the top tenth BMI at age 13 were twice as likely as the remainder to be in the top quarter for adult percentage body fat, but children in the bottom quarter were equally likely to have either high or low body fat as adults. They found no evidence of the relationship of children at younger ages to adult obesity. They also found that most fat adults were not overweight as children.

The World Cancer Research Fund (2007) indicated that "early nutritional exposure is an important determinant of phenotypic expression during later life and is

likely to affect vulnerability to chronic diseases, including cancer." In addition, the habits gained while a child are often continued when an adult. The Harvard Growth Study also showed that being overweight in adolescence predicted a broad range of future adverse health effects that are independent of adult weight.

Bibbins-Domingo et al. (2007) projected future CHD deaths in 2035 using U.S. Census population data, NCHS mortality and Framingham Heart Study CHD incidence rates, together with CHD risk factor prevalence from the NHIS 2000. They projected excess deaths of 35-year-olds, projecting annual excess CHD deaths. Based on their projections, the rate of obesity for boys would be between 30 percent and 37 percent compared with 25 percent currently, and between 34 percent and 44 percent for girls compared with a current rate of 32 percent. The results indicate an excess of 33,000 CHD deaths in 2035 (a 14 percent increase from current levels), with a range of 14,000 and 45,000. This range is equivalent to a 5 percent to 16 percent increase in these deaths. The authors caution that their projections assume a continuation of current experience—if new treatments are found the projection could prove excessive, but if current adolescent obesity trends continue (i.e., no change in the upward trend in the prevalence of obesity), it might be conservative, resulting in a shorter life expectancy at birth of between two and five years.

Unhealthy behavior does not just emerge in adulthood. It usually starts in one's youth and is more likely to continue if it occurs as an adolescent, shaped by family, friends, peer groups, schools and the broader social environment. However, in summary, unless trends are reversed or if mitigating factors do not intervene, serious long-term health consequences may result from the recent rapid increase in childhood obesity.

5.4 Economic View

Economic perspectives of the sources of the recent increase in weight are often based on an analysis of economic-based incentives and decisions that are affected by the net cost associated with rational decisions involving prices of factors contributing to changes in weight. These factors include changes in food prices, increases in mass preparation of food, increases in the efficiency of food production and increases in the availability of fast food restaurants, calorie-dense food and exercise outlets.

Philipson and Posner (1999) indicated that weight management involves "not information but incentives; everyone knows how to lose weight, either you eat less or exercise more, but few want to pay the price, in effort, expense, or forgone pleasure, of doing it."

For example, a food purchase decision is made at a fast food restaurant based on the economic cost involved, including its price, convenience, quality, taste and the net cost relative to the alternatives, including home cooked healthy meals. These decisions also consider home location, means of transportation, use of leisure time, and type and amount of food intake, among many other factors.

Some economists emphasize the effect of technological changes, the result of which has been a reduction in cost of calories in food, while physical activity involved in working and leisure has become relatively more expensive. Individuals tend to make decisions to enhance their limited budgets, which have usually resulted in higher BMIs.

For example, in many cases in which both members of couples work (gaining overall financial benefits, although accompanied by reduced time to prepare food at home), cost-justifiable dinners are obtained at fast food, drive-through restaurants or through microwaveable foods that are easy and quick to cook in terms of both time spent and their cost, even when the food has a high fat and caloric content. Between 1965 and 1995, the time involved in preparing foods at home in the United States has been reduced by half. Married women and women with exactly a full high school education traditionally had both the largest increases in average BMI and spent a lot of time preparing meals at home, for which they now spend less time.

When people work on a farm or in a factory, they are paid to exert calories, but in many service businesses they are not, and may instead have to pay to exercise during leisure time.

Cutler et al. (2003) pointed out that people's choices are made to enhance their happiness, whether their actions contribute to weight gain, even though there is a

potentially long-term cost to becoming obese. This basis for behavior assumes that people are both knowledgeable and that their decisions are unconstrained. Cutler indicated that calories consumed have risen markedly since 1980, while calories expended has remained relatively stable.

Changes in relative prices and the density of fast food restaurants have also been advanced as an explanation for society's weight gain (Chou et al. (2002)). The relative prices of food in fast food and full service restaurants and the price of food at home are considered by many every day. In fact, according to Bleich et al. (2007), average food prices in the developed world fell by 12 percent from 1980 to 2002, ranging from 26 percent in the United Kingdom to 0.2 percent in France, with the U.S. reduction being 8 percent (although the Consumer Price Index for food items increased only 3 percent slower than the CPI for non-food items). Nevertheless, Cutler et al. (2003) pointed out that it is not clear that eating out should be assumed to increase caloric intake, as if the demand arises restaurants can cook low-calorie food almost as easily as high-calorie food, and food prepared at home can be just as junky as that eaten out in a fast food restaurant.

Overall, real food prices have been in a decline for decades. According to the IMF's index of food prices, in real terms food prices fell between 1974 and 2005 by three-quarters. In the 1960s food (including meals out) accounted for one-quarter of the average American's spending; by 2005 the share was less than one-seventh. The price by type of food is also important.

Over the past several years, the cost of fresh food has been put under pressure and therefore increased, while prices for processed food have been able to be reduced, thus giving a greater incentive to eat energy-dense food that may not be as healthy as the more expensive fresh foods are. According to the U.S. Department of Agriculture’s ERS FoodReview v25(3), between 1985 and 2000, the real price of fresh fruit and vegetables has increased by about 39 percent, all fruits and vegetables by 20 percent and cereals and bakery goods by 10 percent, while the real price of soft drinks decreased by 24 percent, fats and oils by 15 percent and sugar and sweets by 8 percent. Therefore some of the least healthy foods and drinks decreased in real price, while some of the most healthy foods increased. The decline in overall prices may

have been one factor in society's increase in calories during this period, but the mix of food may have had the opposite effect.

In contrast with the trends over the last few decades described above, the International Food Policy Research Institute estimated that cereal prices will rise by between 10 percent and 20 percent between 2008 and 2015. These food price increases, due to such factors as the recent increase in oil prices, substantially increased use of grains for biofuels (2007 U.S. legislation requires a fivefold increase in biofuels production in the next 15 years), inadequate agricultural investment over the past two decades, trade restrictions, weather-related supply disruptions, inventory levels and demand from quickly growing developing countries will likely place a dampening influence on future weight gain, at least to the extent that the cost of unhealthy food increases at or faster than the cost of healthy food.

The effect of these factors was seen in the middle of March of 2008 when in a single week there was a worldwide wheat price increase of 25 percent. The prices of wheat, corn and other agricultural commodities increased since late 2006, with no end in sight when viewed in early 2008, while the price of rice doubled between January and March 2008, a staple for 2.5 billion people worldwide. This food price crisis will likely result in an increase in malnutrition in some locations, while in others less expensive energy-rich and potentially more fattening food may be used as a substitute for others that are not as fattening. In early 2008, food price pressure has forced many cash-strapped schools in the United States either to raise prices or to serve more economical dishes, which makes it more difficult to offer healthy, low-fat foods.

The differential trend may not continue as the underlying cost of food increases in the future. Due to the inability of retailers to pass on cost increases to consumers, many food and restaurant prices have so far been shielded from these increases. However, due to the size of the underlying cost increases, it is likely that this will result in consumer price food hikes around the world, and in resulting reductions in affordability of food assistance programs to poor areas and increased malnutrition; early 2008 has already seen an introduction of food rationing in some countries. Food utilization may shift, depending on the differential price changes. Nevertheless, the amount of food consumed may decrease over the next several years

as a result, although it is currently speculative as to what direction this will move the relative healthiness of the average food consumed. Possibly this end of cheap food prices may bring to a halt the recent increase in prevalence of overweight and obesity.

Chou et al. (2004) pointed out that in societies whose jobs are based on services that require light work rather than manual work (e.g., in agriculture and mining), whose jobs involve vigorous physical activity, workers sell more of their time to the labor market and have less disposable time for entertainment and other household activities, including food preparation. This shift in work has been quite gradual and largely predated the dramatic increase in weight gained. This in part explains the growth of fast-food restaurants. With its share of energy intake increased from 2 percent in 1970 to 10 percent in 1995, and expenditure in fast food restaurants increased from 20 percent in 1970 to 40 percent in 1995.

Chou observed that urbanization and the increase in the number and availability of both fast-food and full service restaurants (i.e., travel and waiting time decreased for out-of-home meals) may be the most important factor (65 percent) in explaining increased obesity by contributing to larger food intake. According to Rashad and Grossman (2004), up to two-thirds of the increase in adult obesity can be explained by this factor. Other factors include the relative change in prices in fast food and full-service restaurants and food prices at home, which contribute 12 percent in explaining increased obesity. In addition, the reduction in cigarette smoking contributed another 20 percent.

Chou found that a recent change has been the perceived increase in the value of time, especially that of women, as evidenced by the growth in their labor force participation and in their hours of work. The decrease in home time has been associated with an increase in the demand for convenience food and consumption of fast food, whether through meals or snacks. In addition, Chou points out that the real cost (monetary and risk information) of cigarette smoking tends to increase weight (due to the resultant increased metabolic rate and the appetite suppressant feature of nicotine). Not only do restaurant availability and restaurant prices matter, but socio-demographic factors, such as those experienced by the black and Hispanic population segments who are more likely to suffer from obesity, also matter.

Philipson and Posner (1999) and Lakdawalla and Philipson (2002) suggested that increases in BMI have been due to a lower use of calories as a result of reductions in the strenuousness of work and unemployment. Because more time is spent at work, there is less time and energy available for home and leisure activities such as food preparation and active leisure. They also noted that obesity growth is self-limiting. Ruhm (2007) showed that obesity increases, physical activity declines during business cycle expansions and it is difficult to lose weight once gained. Bleich et al. (2007) found that in developed countries, both female labor force participation and urbanization were positively and significantly associated with caloric supply.

5.5 Contributing Factors to Obesity and Weight Gain

Sections 5.5.1 and 5.5.2 discuss the primary contributing factors to obesity and weight gain that have generally been attributed to the amount and type of food eaten and the extent of physical activity, respectively. Lakdawalla and Philipson (2002) estimated that 40 percent of the total growth in weight in the United States may have been due to an expansion of calories and 60 percent may have been due to demand factors such as a decrease in physical activity. After these are discussed, factors relating to socioeconomic conditions and the educational base are addressed in Section 5.5.3.

5.5.1 Nutrition

Historically, the amount of caloric intake has influenced the weight and height of society's members. Improved amounts and types of nutrition have significantly contributed to overall life expectancy for many years. However, the human body does not appear to have evolved sufficiently to either cope with or easily resist foods such as fat or sugar, which as hunter-gatherers would have been beneficial and desirable in the quantities then available.

Urban/industrial food systems generate relatively energy-dense diets, fairly high in meat, milk and related products, fats, starches, sugars, salt, baked goods, soft and alcoholic drinks. Patterns of vegetables, fruits and fish vary significantly, depending on climate and geographical location. Worldwide, more dietary energy is derived from animal and related sources—for low-income countries, from 160 to 340

kcal/day, in contrast to high-income countries of about 960 kcal/day. Overall in low-income countries, fats, oils and animal protein have increased as socioeconomic factors have improved.

Improved farming practices, subsidies and innovations in the processing, packaging, preservation and refrigeration of food have resulted in an abundance of food that can be easily stored and transported. The food industry spends more than $25 billion annually in the United States, and the abundance of restaurants and fast food outlets has made its products widely available. About one-quarter of U.S. adults eat fast food every day, and these individuals eat about twice as many sugary soft drinks as those who don't eat there.

In fact, according to the U.S. Food and Drug Administration, the percentage spent in the United States outside the home was about 49 percent of total food expenditures in 2006, compared with about 38 percent in 1980 and 20 percent in 1970. On average the food eaten outside the home was less healthy than that eaten in the home. In addition, more convenience food and larger portions have been eaten lately. On the other hand, food has on average a lower-fat content now and the availability of reduced energy and reduced fat products has increased. Nevertheless, Harnack et al. (2000) indicated that some data suggest that, due to the total increase in calories consumed, total fat eaten has increased.

Different sources of information have shown inconsistent nutritional trends. NHANES surveys have indicated that between 1971 and 2000 energy intake has increased, while the trend in the National Food Consumption Survey between 1965 and 1977-78 and the Continuing Survey of Food Intake by Individual between 1989-91 and 1994-96 indicate a reduction. Calories provided by the U.S. food supply have increased from 3,300 calories per capita in 1970 to 3,800 in the late 1990s. This is consistent with the increase in Canada of 530 kcal between 1985 and 2002. Some of these differences appear to be due to the use of self-reported dietary information, which have notoriously been under-reported, especially by those overweight.

Cutler et al. (2003) proposed that in the 1960s the bulk of food preparation was done and was eaten at home, while more recently the technological changes of

food production have expanded the number of restaurants and fast-food opportunities, through mass-produced food and expansion of outlets.

Affordability and children's food and exercise decisions are more based on their short-term needs and a general inability to act in a healthy manner, whether due to a lack of self-control, peer pressure or addiction problems. In addition, due to the existence of government and private health care and disability programs, much of the longer-term costs associated with these actions are shared with others, thus reducing but not eliminating economic incentives for healthy behavior.

The reductions in the time cost of preparing meals have been noted by Cutler et al. (2003), who have argued that obesity has increased primarily due to the consumption of more calories, sufficient on a per capita basis to explain the increase in weight of the American population. To explain this important contributing cause of obesity, the main increase has resulted from large increases in snacks (about 200 calories per day between 1977-78 and 1994-96 according to the Continuing Survey of Food Intake, more for males than for females) outside the main meals, as Americans eat more frequently, even though their average caloric consumption at dinner has in fact decreased during this period.

In contrast to Cutler's findings, Field et al. (2004) found, in an ongoing study of over 14,000 children, that snack foods were not found to be an important independent determinant of weight gain among children and adolescents. After controlling for dieting status and maternal overweight status for girls (both of which were positively related to BMI), the association between the number of servings of snacks and overweight was not significant for boys or girls. Although they may have low nutritional value, snacks were not an important independent determinant of weight gain among children and adolescents.

In the evaluation of mortality, the amount and type of nutrition and energy consumption not only contribute to an increased amount of obesity, but are also mortality risk factors themselves. However, according to Rosenbaum et al. (1997), since both protein and carbohydrate can be metabolically converted to fat, there is no evidence that changing the relative proportions of protein, carbohydrate and fat in the

human diet without reducing caloric intake promote weight loss. Key takeaways from Table 11 are the overall increase in mean energy and macronutrient intake and slight decrease in saturated fat during the years reported in the NHANES.

TABLE 11
Energy Intake in kcals and Percentage from Selected Sources

Gender	Ages	1971-1974	1976-1980	1988-1994	2001-2001
		Energy intake in kcals			
Male	Total	2,461	2,459	2,692	2,697
Male	20-39	2,784	2,753	2,964	2,949
Male	40-59	2,303	2,315	2,567	2.649
Male	60-74	1,918	1,906	2,104	2,117
Female	Total	1,540	1,525	1,804	1,884
Female	20-39	1,652	1,643	1,956	2,032
Female	40-59	1,510	1,473	1,734	1,836
Female	60-74	1,325	1,322	1,520	1,622
		Percent kcals from carbohydrates			
Male	Total	42.4%	42.7%	48.3%	48.2%
Female	Total	45.5%	46.1%	50.7%	50.6%
		Percent kcals from total fat			
Male	Total	36.9%	36.7%	33.9%	33.4%
Female	Total	36.1%	35.9%	33.3%	33.8%
		Percent kcals from saturated fat			
Male	Total	13.5%	13.2%	11.4%	10.8%
Female	Total	12.9%	12.5%	11.2%	10.9%

Source: NHANES

Some nutritionists indicate the largest concern is the amount of sugar or sugar substitutes being eaten. In the 1970s, food technology perfected a reliable way of turning corn starch into syrup sweet enough to become a sugar substitute. After 1982 when the American government imposed import quotas on foreign cane and beet sugar, the American food industry switched to inexpensive high fructose corn syrup—this can trick the brain into thinking that you are hungrier than you really are. It also causes the liver to provide more fat into the bloodstream.

But it appears that only an excessive amount is bad for you. In the United States where high fructose corn syrup use is common, about 31 percent of Americans suffer from the metabolic syndrome, while in Europe where this corn syrup can't compete with cane and beet sugar, only 15 percent of the adult population suffers from obesity. Vertanian et al. (2007), in a meta-analysis of 88 studies, found clear associations of soft drink intake with increased energy intake and body weight. They

also found that soft drink intake was associated with lower intakes of milk, calcium and other nutrients. They observed that studies funded by the food industry reported significantly smaller effects than did non-industry-funded studies.

According to Bray et al. (2004), there is a clear distinction between fructose and glucose, including the digestive and absorptive process for the two. Fructose was 73 percent sweeter than sucrose and contains more than double the sweetness of glucose. In addition, the significantly larger portion size of these beverages may have also contributed to weight gain in America.

In contrast, others have found that the link between the use of soft drinks and childhood obesity is weak, at best. Forshee et al. (2007) indicated that several of the major ecological, epidemiologic and randomized controlled trials addressing sugar-flavored soft drinks are either inconclusive or unreliable. They claim that it is unclear why high fructose corn syrup would affect satiety or absorption and metabolism differently than sucrose, and a recent expert panel convened by The Center for Food, Nutrition and Agriculture Policy concluded that this corn syrup does not appear to contribute to overweight and obesity any differently than do other energy sources. They did indicate that further study is warranted and that many other factors contribute to the obesity epidemic.

According to U.S. Food Supply Series data, there has been an increase in both high-energy-containing and low-energy-containing sweeteners. According to Harnack (2000), this suggests that, although the percentage of low-energy sweeteners has increased, the low-energy containing sweeteners are perhaps being consumed in addition to rather than in place of high-energy-containing sweeteners.

Interestingly, several recent studies have indicated that diet soft drinks might be contributing to weight gain. A study of rats conducted by Swithers and Davidson (2008) suggested that consumption of products containing artificial sweeteners resulted in increased caloric intake, increased body weight and increased adiposity. The weight gain appears to have resulted from interference with fundamental homeostatic, physiological processes. It seems that a sweet taste may cause animals to anticipate the caloric content of food; artificial sweeteners with limited calories may

undermine this connection, leading to an energy imbalance by increasing food intake or reducing energy expenditure. The experiments were conducted using saccharin, but the authors suggest that other artificial sweeteners probably have a similar effect. The authors cautioned that this has yet to be demonstrated on humans and on sweeteners other than saccharine and it may not be appropriate to generalize the results.

Some disagreement has arisen regarding the relative significance of key dietary factors in high fat, energy dense food, and carbohydrate rich food with high sugar content. Needless to say, the overall diet of Americans has not gotten significantly healthier over time.

Various studies have shown that a significant percentage of those dieting are unsuccessful and will terminate their dieting programs before successful results are achieved. In fact, dieting may result in weight gain through possible erratic delivery of nutrients that might trigger physiological responses conducive to gaining weight, rather than losing weight. Extreme outcomes of dieting have included eating disorders, dangerous weight loss behaviors, nutritional deficiencies, low birthweight babies, fear of weight prejudice and suicide. In turn, an underlying tendency to overeat may increase during periodic weight control efforts that may ultimately result in obesity in spite of good original intentions.

According to Hu et al. (2006), "analyses from prospective studies have confirmed that healthy diets are effective and safe ways to prevent type 2 diabetes and the metabolic syndrome." Aspects of nutrition that need to be avoided include an overemphasis on short-term and immediate weight loss, as the difficulty of maintaining such a loss is quite difficult and may in turn contribute to weight recycling, which several studies have indicated will provide adverse health results.

A great deal of sound nutritional advice is available and has been well publicized. It is outside the scope of this paper to discuss them, but certainly proper use of fruits and vegetables and fiber should be emphasized, with avoidance of such foods as red meat, saturated fats and sweetened soft drinks. U.S. Guidelines (2005) suggested nutrient-dense foods and beverages within and among the basic food groups while choosing foods that limit the intake of saturated and trans fats,

cholesterol, added sugars, sodium and alcohol, including nine servings of fruits and vegetables a day (a serving size is modest, e.g., a half-cup) and three or more ounce-equivalents of whole-grain products per day.

5.5.2 Inadequate Physical Activity

Just as for nutrition, inadequate physical activity does not just contribute to obesity, but also is an independent health risk factor. When men and women were hunters and farmers, physical activity was a fact of life; in fact it was life. But now most Americans not only do not rely on physical exercise at work and have not for several decades, many also lead a relatively sedentary lifestyle outside of work as well. Technology has been harnessed to make life easier through such gadgets as remote controls, microwaves, garage door openers, electric lawn mowers or lawn services, but as an intended or unintended consequence has decreased the total amount of personal resources spent in physical activity.

A larger amount of physical activity enhances caloric expenditure, promotes dietary compliance and specifically influences abdominal obesity that is the area of fat that is of most concern. As a result, it can serve as a valuable supplement to a personal program that also includes healthy nutrition and may be more successful than either one alone.

Physical inactivity appears to be both a cause and a consequence of obesity. Being sedentary has been found to increase the incidence of coronary heart disease, stroke, hypertension, obesity, diabetes, osteoporosis, breast and colon cancer and depression. Sedentary behaviors and obesity are independent risk factors for diabetes and the metabolic syndrome. Cardio-respiratory fitness resulting from regular physical activity may play a stronger role in attenuating age-related weight gain than in promoting long-term weight loss. Benefits derived include increased fitness, decreased fatness, enhanced metabolism and possible strengthening of the immune system.

Table 12 shows the percentage of adults who engage in regular leisure physical activity in the United States. Except for the middle ages, males exercise on

average more than females do. Interestingly, the decrease in this physical activity is similar to the increase in obesity at these same ages, except for ages over 75.

TABLE 12
Percentage of U.S. Adults Aged 18 and Over Who Engage in Regular Leisure Physical Activity

Ages	Total	Males	Females
18-24	37.2%	41.1%	33.3%
25-64	32.3	33.3	31.5
65-74	23.7	26.5	21.4
75 +	17.9	22.7	14.9
Total	31.1	33.0	29.3

Source: January-September 2007 NHIS

Historically, physical activity and fitness were integral aspects of daily life as a means of transportation, occupation and home maintenance. However, during the 20th century most physical activity was engineered out of daily living by various labor-saving devices and the automobile. For some people, by the beginning of this century physical activity is restricted to yard work and workouts at the gym.

The energy use of any particular activity varies depending on a person's basal energy expenditure and her or his demographic characteristics, e.g., age, gender, size and level of physical fitness. Even though it is now recognized that physical activity is important to the maintenance of good health, a large number of people do not have the ability, means, spare time nor energy to make it an integral part of their lives.

Modern transportation, whether through private cars or public transportation, has affected the amount of walking and cycling for work, chores and activities. Oil and gas have substituted for physical exercise as the key energy source. Public spending has tended to improve their ease of use, efficiency and overall desirability, thus placing popular public decisions in opposition to healthy habits. However, much of this was in place decades ago; e.g., according to the U.S. Department of Commerce, in 1980, 84 percent drove to work, 6 percent walked and 6 percent used public transportation, while in 2000, the corresponding percentages were 87 percent, 3 percent and 5 percent. Communities' physical infrastructure and characteristics, such as streetlights, the availability of playground and sports venues, have also affected to children's physical activity such as walking and biking to school. Overall, there

appear to have been limited changes in overall energy expenditures in recreational physical activity over the last 30 years in the United States.

Several attitudes have led to a reduced level of physical activity, including lack of motivation and lack of self-confidence and self-consciousness. Those adolescents already obese may perceive that there are more barriers to physical activity than actually exist. In addition, the level of parental and peer support for physical activity is related to the level of physical activity achieved or attempted.

Although a healthy weight or BMI is desirable, even those overweight who exercise sufficiently can experience lower premature mortality rates. Various studies support that 30 minutes of exercise per day is just as effective as 60 minutes per day. However, current U.S. Guidelines (2005) indicate that a minimum of 30 minutes of exercise daily is recommended, with 60 minutes a day of moderate to vigorous exercise (e.g., walking, bicycling, hiking and gardening) constituting the amount to keep from gaining weight. To take off pounds, the guidelines suggest up to 90 minutes a day.

Hu et al. (2003) estimated that about 30 percent of new cases of obesity could be prevented by adopting a relatively active lifestyle, including at least 30 minutes of brisk walking daily and less than 10 hours of watching TV per week. Both aerobic capacity and the ability to perform physical activities may be hindered by obesity, particularly those severely obese.

According to the NHIS, in 2004 59 percent of American adults do no vigorous physical activity in their leisure time, with only 26 percent engaging in vigorous leisure-time physical activity for at least 10 minutes three or more times per week. The highest prevalence of inactivity is for those over age 75 (about 57 percent of males and 66 percent of females), with 52 percent of both Mexican-Americans and non-Hispanic blacks being inactive. With respect to education, 29 percent of those with at least some college education are inactive, while 49 percent of those with at least high school completed and 64 percent of those without a full high school education are inactive.

This contrasts with the results of a 2006 survey by the Pew Research Center that indicate that 86 percent of adults believe exercising for fitness improves the odds of a long and healthy life, but only 28 percent say they personally exercise as much as they should. A common reason for not exercising is that it isn't fun, as many simply don't enjoy exercising, and thus avoid it.

About 25 percent of those between ages 12 and 21 participate in light-to-moderate activity nearly every day, with 50 percent regularly engaging in vigorous activity, while 14 percent report no recent physical activity of any kind.

In sharp contrast to this glum news regarding physical activity is the so-called jogging/gym revolution. In many U.S. cities there seem to be an overwhelming number of physical fitness clubs, and the popularity of marathoning is at an all-time high. Health clubs, walking and running paths and bike trails are abundant; and work-site and hotel fitness centers have increased dramatically. How can these diverse observations be reconciled? It seems that the population may be split into two groups—those who are lean and physically fit and those who are obese and sedentary.

However, people cannot be neatly categorized into simple traditional demographic categories. In fact, according to Bleich et al. (2007), estimated correlations between caloric intake and individual physical activity measures, although positive, are relatively weak (r's of moderately active work of 0.1, housework of 0.09 and commuting of 0.03).

Nevertheless, on an overall basis, technological advances have caused a marked reduction in the average daily expenditure of energy. There is a lot less need to expend calories to earn a living and obtain food, water and shelter, and for transportation and personal chores. The development of an effective segmentation of time remains that likely varies by individual, is left for another paper.

Many studies that include physical activity as a variable include only one variable that measures some form of aerobic exercise. It should be noted that there is some evidence that resistance training also reduces body fat and increases fitness. Both may be effective in producing changes in body composition and fitness. Note

that there is no recognized standard measure of physical activity. The analysis of future studies would benefit from such a measure.

5.5.3 Socioeconomic Factors

Many studies have shown that individuals of low income, wealth or education die earlier than those more fortunate. These socioeconomic factors constitute a bundle of characteristics that represent an individual's relative standing in society. Two of these related factors, income and education, have been shown to be related with mortality, with most analyses showing that differences in mortality have increased over the last half of the 20th century. However, recently the richer and higher educated have been “catching up” with the rest of the population and in many cases share the same weight issues.

Rogers et al. (1999) indicated that more highly educated individuals are more likely to engage in healthy behaviors, such as eating healthy foods and exercising, regularly visiting their doctor for preventive care, better understanding doctors' recommendations and adhering to recommended treatment. Other socioeconomic factors that may also be related to mortality include increased urbanization and suburbanization and increased female labor force participation.

Baum and Ruhm (2007)'s analysis, based on the National Longitudinal Survey of Youth in the United States, indicated that those growing up in disadvantaged families weigh more at all observed points of a person's lifecycle and disparities increase with age. Nevertheless, on an overall basis obesity has affected those in all income levels.

The analysis found that future body weight appears primarily transmitted through education and only to a lesser extent through race/ethnicity. Little evidence was found that socioeconomic gradients in body weight are related to income or health behaviors controlled for (drinking, smoking, exercise and job-related demands). Overall, a strong inverse relationship between socioeconomic status and obesity has been found among women in developed societies, with the relationship being mixed for men and children.

In contrast, Gruber and Frakes (2006) found that BRFSS (1984-2002) results indicate the existence of a negative and statistically significant relationship between real household income and BMI, although the estimated effect is quite small, with an implied elasticity of 0.2. However, it seems that there is an implicit income elasticity of 0.18 with the probability of being obese. In this study too, a strong and negative effect of years of education exists with both BMI and the probability of obesity.

Based on the 1995-99 BRFSS, although overall both the U.S. standard of living and education have strongly increased, people who reported concern about food security were those whose annual household income was less than $20,000. Just as in certain underdeveloped nations, overweight has replaced malnutrition as the most prevalent nutritional problem among the poor, although both obesity and food insecurity are increasing in the United States. It has been hypothesized that one explains the other because of both under and over food consumption, physiologic adaptation of increased body fat in response to food shortage episodes and higher consumption of cheaper foods that are higher in fat and sugar.

Those with no college education or who are female, black, American Indian, Hispanic or older than 45 are more likely to be obese than others. Although research has not concluded that food insecurity causes obesity, they certainly are related. For these households, the lack of money contributes to both hunger and obesity, for without money reliance has to be placed on cheaper, high calorie foods to cope, which inevitably constitutes a less healthy diet. In addition, areas with a concentration of the poor have fewer supermarkets that could provide greater choice of fruit and vegetables at a more affordable price and instead have more convenience stores with a narrower range of available food choices.

Although being overweight is often considered a problem of overeating rather than hunger and scarcity, low-income adults and children have gained the most weight in recent decades. Rates of obesity and overweight among the poor are increasing, while the number of poor Americans experiencing food insecurity remains high, estimated to be about 30 million in 1999 and 37 million in 2004. Recent and expected future increases in food prices may further exacerbate the problem.

Women of lower socioeconomic status (less than 130 percent of the poverty line) are 50 percent more likely to be obese than those with higher status, although men are equally likely to be obese on either side of the line. Overall, average BMI, obesity and class 3 obesity are more common and have increased faster for those disadvantaged. In addition, various studies have associated low socioeconomic status and physical inactivity. NHANES have shown that obesity and income are inversely related (Mokdad et al., 2001).

In some cases, mothers restrict their food intake during periods of food insufficiency to protect their children from hunger, which contributes to ups and down in their food intake and which inevitably contributes to weight cycling, which in turn can lead to obesity and poorer health in low-income women.

It seems that past deprivation, especially in the case of Hispanic mothers, may negatively affect their relationships to their children with respect to food—causing them to feel that their children should “clean their plates” and/or be indulged when there are “food treats,” thus in turn increasing the next generations' obesity. Sixty percent of Mexican-American low-income mothers who experience food insecurity are overweight. Food insecurity as children also influences them as they become adults.

Possible scenarios include ones in which high-fat and high-sugar foods are the cheapest source of calories for low-income parents to buy. Another is that when low-income parents have money, they tend to snack more and eat at fast-food restaurants, as well as experience unhealthy weight cycles. In many poor areas, fresh food is not easily available. Another theory is that low socioeconomic status leads to psychosocial stress, promoting increased fat deposits in the abdominal area through psychoneuroendocrinological pathways.

It is clear, based on the studies reviewed during the course of preparation of this paper, that analysis of data with demographic characteristics should be reviewed separately by gender, as patterns can be quite different.

5.5.4 Other Factors

One of the frustrating aspects of the study of obesity is the often seemingly contradictory and illogical findings. For example, there is often a high correlation between factors, but limited cause-and-effect proof. In studying social patterns and relationships, details of the methods applied are very important considerations, as different conclusions have been reached by experts, even using the same data base. Particularly in an area such as this one for which associations are easy to find but causes are not, it is therefore important to examine what may seem to be alternative views and factors that, at first blush, may not be significant contributors.

An example is an article (Keith et al., 2006) that presented 10 additional possible causative factors that may have contributed to the recent increase in obesity prevalence. A significant point made in the paper is that it is unlikely that there is a single factor that by itself caused the worldwide structural trend in obesity. The following is a brief description of the ten factors raised:

1. Sleep debt. It is generally recognized that the number of hours of sleep is inversely related to BMI levels. Data sources differ, but data collected from surveys conducted annually by the National Sleep Foundation, have found a continuing decrease in sleep time over the last several decades. In contrast, time-use surveys collected by the U.S. Census Bureau indicate that average sleep for adults, after being relatively stable for a number of years, may in fact have increased in the early 2000s, with this most recent trend being consistent with data gathered from Canada. Other sources, including
2. Endocrine disruptors. These industrially produced substances that can affect the body's endocrine function have increased in the food chain.
3. Reduction in variability in ambient temperature. The use of air conditioning has increased over the last 30 years and may have an affect on body fat.
4. Decreased smoking. The rate of cigarette smoking has been associated with weight gain, although no one has argued that the net health effect of smoking cessation is bad.
5. Certain pharmaceuticals increase weight. Weight gain is a consequential result of the use of several drugs, including antidepressants, antidiabetics, beta

blockers and protease inhibitors. The use of these drugs has increased substantially.

6. Changes in distribution of ethnicity and age. Most of the aging process, at least until old age is associated with increasing weight, so as the population has gotten older, the average per capita weight should be expected to increase.
7. Increasing gravida age. Some studies have shown that there is a correlation between maternal age at birth and fat. Globally, the average age at first birth has increased over the last several decades.
8. Intrauterine and intergenerational effects. It has been suggested that maternal obesity and resulting diabetes in utero, during gestation and lactation may promote the same conditions in subsequent generations.
9. Greater reproductive fitness yielding selection for obesity-predisposing genotypes. Those who are obese may have a greater tendency to have more children.
10. Assortative mating. To the extent that there is a genetic basis for obesity, an increase in births to those who are obese might lead to more future obesity in the next generation.

5.6 Worldwide Situation

The World Health Organization (WHO) currently estimates that there are about 1.6 billion adults (older than age 15) who are either overweight or obese worldwide, with at least 400 million obese and about 20 million children under 5 who were overweight in 2005. WHO currently estimates that in 2015 there will be 2.3 billion adults either overweight or obese and 700 million obese. They have also estimated that obesity accounts for 2 percent to 6 percent of total health care costs in several developed countries, with some estimates as high as 7 percent; but WHO has indicated that even this may be understated.

Due to the sudden worldwide increase in obesity, WHO has referred to it as a “globesity,” with a threefold increase since 1980 in parts of Eastern Europe, the Middle East, the Pacific Islands and China. This overall adverse trend has been due in part to the consequences of massive social, economic, cultural and technological upheavals that have and are continuing to occur throughout the developing and in parts of the developed world as well.

Although the United States has a very high percent of the population that is obese, other regions of the world have similar if not worse profiles, e.g., some of the Pacific islands (the extreme is the reported rate of obesity on Rarotonga island, the most economically developed of the Cook Islands, in which 100 percent of males under age 30 in 1996 were obese) and certain Middle East countries. And it is increasing fast in many other regions, e.g., Central Europe. Nowhere is it retreating, although overweight and obesity remains relatively low in Japan, whose citizens still have a diet relatively rich in fish and vegetables, but even there concerns have been expressed regarding recent adverse trends. Nevertheless, it is difficult to generalize about anything when characterizing national and sub-national characteristics.

Cutler et al. (2003) pointed out that time devoted to food preparation varies widely by country, with reduced time predominating, primarily due to the use of household appliances such as microwave ovens. There appears, at least in developed countries, a relation between the use of these time-saving devices and weight. For example, over 80 percent of U.S. households have microwaves, while in Italian households where obesity is much less common only 14 percent have one. Italian and French adults spend about 19 minutes more per day cooking than Americans, with correspondingly better nutritional content. In the United Kingdom, where adults spend almost the same time as Americans in food preparation and where 66 percent of households have microwaves, the obesity level is closer to that of the United States. This suggests that as other countries' food-related habits get closer to the United States, obesity problems will likely continue to spread.

Bleich et al. (2007) observed that similarities in the speed at which obesity prevalence has spread across all developed countries suggest a worldwide time-related systemic phenomenon, rather than a country-specific trend or an isolated event. Inserm (207) pointed out that, although a similar increase has occurred in Europe, in contrast to the United States whose significant increase in weight began in the 1980s, its corresponding increase only emerged in the 1990s. Inserm has found that this was not likely to have been caused by changes in work-related or leisure time activity, as changes in these factors have been occurring on a gradual basis prior to the time of

rapid increases in weight and there have not been sufficient changes in overall activity levels to have caused such large structural shifts.

The International Obesity Taskforce (2002) asserted that the causes of the obesity epidemic are twofold: an abundance of energy-dense foods and drinks, leading to a pervasive “passive over-consumption” of energy; and an environment that limits opportunities for physical activity, leading to an almost universal sedentary state.

Significant calorie growth has also been experienced in such countries as the Netherlands, New Zealand and Spain. In contrast, calorie control has maintained in Japan, which during the last 30 years has experienced the lowest growth of calories in the OECD, accompanied by one of the lowest average weight. Nevertheless, Australia has also experienced a moderate growth in caloric input, yet the percentage of obese individuals grew by 23.4 percent. Bleich et al. (2007) found that the portion of obesity due to increased caloric intake among the 15 developed countries studied was 62 percent, while if Australia and Finland, the outliers, had been excluded it would be 82 percent.

Between 1980 and 1995, for example, obesity rates increased from about 14 percent to 20 percent in Australia, from about 14 percent to 16 percent in the United Kingdom, and from about 7 percent to 12 percent in Brazil. In India, a survey of 83,000 women indicated that, while 33 percent were malnourished, 12 percent were overweight or obese. According to one study, three-quarters of Russian households contain at least one obese person. Mexicans are now the second heaviest nation among the 30 mostly rich countries of the Organization for Economic Co-operation and Development (OECD) after the United States, have the highest rate of diabetics and coincidentally are among the biggest drinkers of fizzy drinks in the world. In urban areas, a higher level of schooling is associated with a lower prevalence of obesity, while the reverse holds for educational level, income and wealth in less urban areas. And one-fifth of Chinese children between 7 and 17 who live in its cities are now obese. Recent obese prevalence rates for selected OECD countries are given in Table 13.

Table 13
Percent of Obese in Selected Countries

Country	% with BMI>30	
	2005	1995
Australia	21.7% (1999)	19.8%
Canada	18.0	12.1
Finland	14.1	10.4
France	9.5	7.0
Italy	9.9	7.0
Japan	3.0	2.6
Korea	3.5	2.2
Mexico	30.2	24.2(2000)
Netherlands	10.7	6.9
New Zealand	20.9	17.0
Spain	13.1	10.3
Sweden	10.7	7.9
Switzerland	7.7	6.8
United Kingdom	23.0	16.0
United States	32.2	22.9(1991)

Source: 2007 OECD health statistics.
Note that prevalence rates for Australia, Canada, New Zealand, the United Kingdom and the United States are measured, while for other countries they are based on self-reports, which should result in rates lower than if they were measured.

Although it has generally been accepted that obesity is inversely related to income, this relativity does not appear to currently apply to cross-country relationships. This relationship may be affected by relative access to food, especially meat and poultry, but also energy dense foods.

Obesity is no longer confined to developed countries. In fact, the rise in overweight and obesity has been much faster in lower-income countries since 1975 than in higher-income countries, albeit from a much lower level. With increasing levels of affluence in many countries, it is likely that obesity will become an increasing problem worldwide in the nutrition transition currently occurring from traditional diets low in fat and high in fiber to high-energy Western-style diets that are high in fat and low in fiber. As food in these countries increasingly take on Western-style characteristics, combined with an increased ability to buy high-fat and energy dense food resulting from rapid urbanization, industrialization, and development, expanded food availability and diversity may lead to unhealthy eating habits in all age groups and income levels.

Naturally, nutrition isn't the only factor involved, as increasing urbanization has generally resulted in increasingly sedentary lifestyles that have led to an increase in obesity. For example, the percentage obese in cities in China and Indonesia is twice that of those in the countryside, with a corresponding factor of six in the Congo. In contrast, obesity is more prevalent in rural areas in Russia.

If not arrested, this transition process will likely continue. In several low-income countries, high levels of body fatness exist side-by-side with malnutrition (possibly 30 percent of worldwide humanity), even in the same household. It is proving increasingly difficult for certain countries to deal with food insecurity and undernourishment at the same time now that chronic diseases are emerging as a major epidemic. Proper food distribution and mix will remain a continuing challenge.

The current rapidity of the evolution and convergence of culture can be seen in the larger weight of children in some of the immigrant communities in the United States. Those from Guatemala are 26 pounds heavier than those of corresponding age living in their home country. However, it is also clear (Loureiro and Nayga, 2004) that different socioeconomic and cultural factors are at work in different countries and segments of certain countries. One of the differences is a change in caloric intake—in the United States, the daily level grew by 716 calories between 1973 and 1999. As reported by Margarey et al. (2001), between the mid-1980s and mid-1990s, Australian children's fitness level has declined, as time spent in sedentary activities has increased and daily energy intake for ages 4-15 has increased.

In countries such as the United Kingdom, differences in obesity by social class (similar in nature to differences in educational attainment in the United States) have appeared, with more significant observed differences in BMI in females. The ObEpi survey of Europe in 2003 found that obesity was half as frequent in managers and the liberal professions (8.5 percent) than in craftsmen and tradesmen (16.1 percent), with 20 percent of adults with primary education only compared with 6 percent with higher education who are obese. These significant differences contrast with those of the United States, where such difference have in some cases observed to have been decreasing somewhat in size recently. Similar to the United States, the frequency of obesity has seemingly been increasing in all demographic categories everywhere

recently. In many underdeveloped or developing countries, including China and Russia according to Wang (2001), it is the wealthier and more educated who have tended to be heaviest. The problems noted for less developed countries also apply in certain population segments and areas in almost all developed countries as well.

To make a better inter-country comparison, Michaud et al. (2007) adjusted self-reported weights and heights for certain countries using the method described in Cawley and Burkhauser (2006). Table 14 shows the results of the United States (the Health and Retirement Study) and selected European countries (from the Survey of Health, Aging and Retirement) in 2004. Overall, the results indicated that there is a higher percentage of underweight, overweight and obese status for females than for males. The most significant difference is the higher percentage of severely obese (BMIs of 35+) in the United States than in any of the European countries shown for both females and males. It also shows that there is a significant difference in level between European countries.

TABLE 14
Percentage of Individuals Age 50+ by Corrected Self-Reported BMI

Country	**< 18.5**	**18.5-24.9**	**25.0 - 29.9**	**30.0-34.9**	**35.0+**
	Females				
U.S.	2.03%	29.45%	30.66%	20.06%	17.81%
Total Europe	2.04	35.30	38.46	17.48	6.72
France	4.09	44.65	30.98	14.83	5.46
Germany	1.15	35.63	40.34	15.87	7.01
Greece	0.88	23.80	44.16	22.70	8.46
Italy	2.58	33.36	40.70	17.69	5.66
Netherlands	1.45	34.50	40.83	17.19	6.03
Spain	0.75	25.70	39.94	23.66	9.96
Sweden	1.68	39.78	37.00	16.75	4.80
	Males				
U.S.	0.52	24.08	44.71	20.97	9.72
Total Europe	0.75	30.63	51.01	14.28	3.33
France	0.85	35.17	47.74	13.64	2.60
Germany	0.48	28.71	52.21	14.62	3.98
Greece	0.21	24.43	56.19	16.21	2.97
Italy	1.05	29.62	53.69	12.50	3.15
Netherlands	0.30	33.73	50.7	12.89	2.38
Spain	1.17	29.78	48.23	17.13	3.69
Sweden	0.74	34.28	49.18	12.89	2.91

Source: Michaud et al. (2007)

There has been a remarkable increase in the intake of dietary fats over the last three decades everywhere in the world except Africa, although the highest rate of consumption has been in parts of North America and Europe. The per capita supply of fat from animal foods has increased by 14 and 4 grams in developing and developed countries, respectively, while there has been a decrease of 9 grams per capita in pre-transition countries, most of which are in Africa. In lower-income countries, an equally large shift has occurred from added sugar.

Only in parts of North America and Europe is the intake of saturated fat at or above 10 percent of total energy intake. In less affluent regions, the proportion of dietary energy that comes from saturated fatty acids is lower, between 5 percent to 8 percent, and has not changed much recently. Recommending more fish in people's diets can be problematic in many locations, for example, where there are simultaneous concerns about sustainability of this food source. In some countries there may be a low level of consumption of fruits and vegetables, possibly due to lack of availability or affordability.

Culture and the environment can affect an individual's weight in many weighs. As indicated in Philipson et al. (1999) in a cultural and economic comparison of Americans and Europeans, differences in weight arise that at least in part are due to less expensive food and gasoline in the United States. These differences and an increase in suburbanization have resulted in a smaller incentive to economize on food purchase, eat away from home more often and reduce the amount of walking and biking exercise. Higher land prices have tended to lead Europeans to live closer to each other and to work and shop at distances where walking and biking may be more efficient than driving. Americans also watch more television, play more video games and surf the internet more than Europeans, in part due to a wider range of availability and earlier adoption of these technologies. A greater diversity in culture can also result in different responses to the same incentives.

There is currently no global standard regarding an acceptable level of physical activity, although WHO in 2002 recommended a minimum of 30 minutes of moderate physical activity most days. However, it has been estimated that about 60 percent of

the worldwide population does not currently meet that benchmark. In fact, any type of physical activity can be beneficial, except for extreme amounts.

In addition, there has been a worldwide reduction in the vigorous nature of work, transportation and leisure physical activity, all of which affect not only obesity, but body structure, fitness and in turn chronic diseases and disabilities as well. Historically, in poor and many developing countries, the obese have tended to be relatively wealthier, in contrast to experience in most relatively wealthy countries where the obese tend to be relatively poorer. This may change in the future.

Changes in the extent of sedentary work, leisure life, food and urbanization in some countries have taken decades, while in the West these changes took centuries. Cultural and socio-demographic changes have been and are also occurring now, although at very different rates. Although in theory an improvement in wealth should have a positive effect on health and a reduction in obesity, in fact it is having the opposite effect, as expanding middle classes are demanding the benefits of technological change and increases in an unhealthy food diet. There has been a decreased acceptance of health-promoting behaviors among the poorest sections of society, at the same time that more “desirable” food and living standards are coming within their reach; this makes efforts at prevention that much more difficult.

According to Hossain et al. (2007), the high risk of both diabetes and cardiovascular disease that are associated with obesity in Asians may be due to a predisposition to abdominal obesity, which can lead to the metabolic syndrome and impaired glucose tolerance. About 200 million people worldwide now have impaired glucose tolerance, the number of whom is expected to increase to 420 million by 2025. Wilde has estimated that worldwide, the number suffering from diabetes was 171 million in 2000 and will be 366 million in 2030, with the most noticeable increase occurring in developing countries. Meanwhile, in large part because of the growth in overweight and obesity, one billion people had hypertension in 2000, projected to be 1.56 billion in 2025.

The rich world's chronic diseases, typically associated with Western living standards, have become in less than a generation the middle-income and low-income

world's greatest health problem. The World Bank has stated that these illnesses will be the leading causes of death in low-income countries within the decade and the poor will be affected by them at a younger age than in the West. WHO (2003) indicated that by 2020 chronic diseases will account for almost three-quarters of all worldwide deaths, and that 71 percent of deaths due to ischemic heart disease, 75 percent of deaths due to stroke and 70 percent of deaths due to diabetes will occur in developing countries. The number of new cases of type 2 diabetes in China and India already exceeds corresponding new cases in the rest of the world.

In most countries (other than in certain Western European countries), obesity is more prevalent in women than in men. In 138 of 194 countries for which the WHO reports, women were more than 50 percent more likely to be obese than men. In South Africa, for example, the rate of obesity of black women was five times higher than of black men. Case and Menendez (2007) found that this was primarily due to women who were nutritionally deprived as children who are significantly more likely to be obese as adults, while men are not. In addition, and Case and Menendez indicates this to be more speculative, adult black women's perceptions of an ideal body in some countries may be heavier than men's perceptions of the ideal male body. When household resources are limited, women tend to choose not to eat to guarantee more to be provided for their children that contributes to weight cycling and resulting in adverse mortality experience. Although men report eating larger meals and drinking more soft drinks than women, they also exercise more and eat less sugar with their coffee or tea. This is an example of where cultural differences and attitudes can affect obesity, nutrition and physical activity, in turn affecting mortality and morbidity.

In summary, most of the issues that the developed world is facing are also now facing or will be facing the rest of the world, where resources and incentives to address them are much smaller. In addition, the residual effects from a long period of malnutrition and food insecurity in a period of adverse socioeconomic conditions still have to be dealt with. Although internationally more emphasis has been recently placed on the prevalence of obesity and lack of physical activity and their adverse effects on mortality, health, health care costs and on health care delivery systems around the world, more will be needed.

At April 2008, it would be pure speculation regarding the ultimate effect of the recent increase in food prices on the level of future obesity, especially if it reverses the trend over the last 30 years of reducing food prices. It is and will be impacting not only those most poor around the world, but also many in the middle class in developing countries, as it will affect the amount of the income and wealth that can be spent on food. It has been noted that the poor of El Salvador are eating only half as much food as they were a year ago, while Afghans are now spending half of their income on food, up from a tenth in 2006. (*Economist, 2008)* For the truly poor, it could increase the percentage of population suffering from malnutrition rather than obesity, but at the same time, it could push many others toward energy-rich less expensive foods than might favor an increase in obesity.

5.7 Optimal Weight

We all would like to weigh in at the point at which we are most healthy. But what is that weight? The answer to this question is important, not only as a personal goal (according to the Pew Research Center 2006 Survey, American adults would like to be, on average, 16 pounds lighter, with women's and men's goals being 18 and 14 pounds lighter, respectively), but also to communicate to society as a whole for public policy purposes. It would also be helpful to measure any differential mortality or morbidity from the best healthy weight.

Unfortunately this weight is not easy to determine, and well might vary depending on the question asked, e.g., optimal with respect to future mortality or morbidity risks or for quality of life, and with respect to what condition, population or sub-population.

The “best” BMI level has often been associated with BMIs between 18.5 and 24.9 or between 23.0 and 24.9. These ranges have evolved as a consequence of U.S. mortality investigations of life insurers conducted since the turn of the 20th century and subsequently when in 1942 Metropolitan Life Insurance Company developed tables of “ideal weights” for men and women.

However, as can be seen in the literature, a single best BMI might not exist and be applicable in all cases. What weight is best for one group or individual may not be the best for another.

- For children, no worldwide standard has been accepted; even in the United States, the cutoff point is based on old information.
- The “best” range may vary by age even, for adults.
- For those of older age, BMIs may not be the optimal measure in the first place, even though it is relatively simple to measure.
- For certain racial or ethnic population segments, what is “best” might be considerably higher than the currently accepted standard, such as for black American females, or lower than the currently accepted standard, such as for Asians.

Statistical problems exist in such a determination, from confounding variables to reverse causation (e.g., as a result of the effect of past smoking histories, multiple impairments and frailty in old age). Or for that matter, the optimal weight may vary by age, gender and to what chronic disease or objective (e.g., mortality or morbidity) is being addressed. In addition, because there has been a wide range of methodologies applied, based on a wide range of populations, the conclusions that have been reached have differed.

Whether the pattern of rates of mortality and weight is U-shaped, J-shaped or monotonically increasing (with mortality rates on the vertical axis and BMI on the horizontal one) may be less important on an individual level than recognizing the points at which an intervention is suggested or of the essence. In addition, although the focus of this paper is on the right tail of the distribution of BMIs, extremely low weight is also of concern, especially for the old and frail. Nevertheless, the largest public policy health issues relate to those on the far right part of the probability distribution.

In most instances, the healthy weight guideline has been expressed as a range, many times between 18.5 and 25 BMI. The American Cancer Society Study found such a range for men to be between 23.5-24.9 and for women to be 22.0 to 23.4.

Several studies indicate that the optimal weight for non-Hispanic black females is likely to take on a wider range, possibly at a higher level and Asians at a lower level. However, some studies, such as the Nurses' Health Study that do not include older adults, have found that, after adjustment for smoking and other factors, the lower your weight the better off you are. In contrast, NHANES has found that in some cases, the optimal weight may lie in what is currently considered to be the overweight category. Fontaine et al. (2003) found that the optimal BMI is approximately 23 to 25 for whites and 23 to 30 for blacks.

The optimal threshold over which adverse health can be expected may not even exist, as it may vary too much by individual or category of individuals. It may be that the current threshold level at which overweight is now considered in adults may be too low. In any case, benchmarks have been developed that, although not perfect, have proven helpful.

I am sure that this issue will continue to be pursued in the future.

6. Effect of Obesity and Related Factors

The underlying contention of this paper is that a proper understanding of the sources of change in mortality is important in developing any estimate of future mortality (see Gutterman and Vanderhoof, 1997). This section explores the ways in which obesity and related risk factors have and are expected to influence the mortality of various population segments.

It is difficult to attribute a given level of mortality to cause, in part because of the multiple causative factors often involved and the complex nature of underlying mortality. For example, it is common that only the proximate cause of death is assigned to a death record, even when the causal pathway is complicated and involves multiple sources. For example, a single case might involve “old age,” coronary heart disease, diabetes, obesity, high blood pressure, a deficient gene and being physically unfit. Which is coded on the death record can influence the attributions studied. As suggested by Section 3.1 and Figure 5, these factors both interrelate and are affected by each other. In addition, they can in turn be partially mitigated by another set of factors.

Not only are those overweight or obese more likely to suffer from chronic diseases, but their physical condition (e.g., weight) in turn can further contribute to poor nutrition and exercise, in a vicious circle. In addition, cultural, environmental and technological factors, as well as genetic susceptibility, can also have significant effects on the amount and type of energy input, physical activity and weight. Their individual effect is difficult to determine, complicated to record and subject to model over-specification and confounding even when all the variables are known. The most that can usually be said is that there is a strong relationship or association and that a practical attribution can be performed, although in many cases a single proximate cause cannot be identified with certainty.

The effects of overweight, obesity and changes in weight have been quite controversial, in part due to the stakeholders and methodological challenges involved, especially as a result of the difficulty in assigning causation, attribution and measurement. Common attribution methods where multiple causes may be involved include the development of a ratio of obesity's contribution to the additional deaths for

which there is a relationship between obesity and excess mortality, for example, Gronninger (2007) and Flegal et al. (2004). Gronninger reflected the relationship with socioeconomic factors while Flegal reflected the relationship with age, gender and inconsistent baseline measures, i.e., calculating excess ratios based on a healthy and younger baseline group while applying them to a mix of healthy and unhealthy and older lives. Some researchers have found evidence that overweight (measured in terms of BMI) has minimal or even preventative statistical effect on mortality, while other studies have found a great deal. The following discusses some of these findings.

Much of the controversy, in addition to the technical attribution formulas applied, is focused on whether those overweight (BMIs between 25.0 and 29.9) experience mortality greater than those of lower weight or rather are "protected" at these BMI levels. Also, the question as to the extent to which weight, rather than other characteristics or factors such as physical fitness or nutrition, contributes to the additional mortality observed is relevant. Constant relative mortality rates across a wide range of BMIs and their variation by certain demographic subgroups included in some studies have raised some doubt regarding the level of mortality that should be assigned to the BMI. In several, but by far not all studies where both levels of obesity and physical activity are available, obesity expressed in terms of BMI does not appear to be a significant variable. In other cases, alternative measures of adiposity, such as waist circumference, have shown that obesity, in one shape or another, can affect future mortality levels.

Since attribution of deaths to a single cause in many cases is difficult, it is often more important to look at related factors on a combined basis. But in explaining the relationships found, one has to be careful to properly express the combination of the factors studied.

Fontaine et al. (2003) estimated that, based on NHANES III, the life expectancy for those severely obese is on average reduced by 5 to 20 years compared that of those of normal weight. Olshansky et al. (2005) estimated, using the 2000 U.S. Life Table as a base, the expected effect of elimination of obesity (he assumed that that population segment experienced mortality at a level similar to mortality of a BMI of 24) as an increase in life expectancy at birth of one-third to three-quarters of a year.

The Olshansky paper has been criticized on technical grounds by some who have pointed out that since those who are obese also have other risk factors, it is inappropriate to assume that the total difference in life expectancy indicated by this single relationship can be attributed solely to obesity. On the other hand, since the time that Olshansky prepared his estimate, the obesity prevalence rate reported by NHANES increased by about 10 percent for males, offset by a reduction in mortality rates from cardiovascular disease, a principal source of excess mortality of the obese.

Significant mitigating factors can influence the extent that risk factors such as obesity affect health. In particular, even though, as seen in Section 5, the prevalence of obesity has skyrocketed over the last 30 years, the level of mortality rates has moved in the other direction. Two significant reasons for this apparent inconsistency appear to be that the reduction in smoking and the effect of mitigating factors on cardiovascular diseases, such as medical treatment of such risk factors as blood pressure and cholesterol, simply have more than offset by the negative influence of fat. A key question in assessing future mortality is the extent to which these potentially offsetting contributions to mortality will continue.

Although there are many consequences of being overweight or obese, many of these consequences may be reversible, even though some consequences may have a long-term effect—therefore, the importance of mitigating factors (see Section 7 for further discussion).

The effects of higher BMIs vary by age. As a result, although some observations relate to all ages, the following discussion is split into the three major age group categories (adults, older adults and children). Overall, obesity, particularly at a higher level, is clearly associated with a higher level of mortality at all ages, although the primary ages it affects appear to be non-elderly adults, while evidence has it affects certain population segments in different ways.

6.1 Adults

It is “common knowledge” that overall mortality has improved significantly over the last 30 years at the same time that Americans on average have gotten much fatter. At first view, these trends seem inconsistent. It seems that the decline in

mortality may better reflect increasing expenditures on efforts to treat, rather than prevent chronic disease. For example, Williamson (1999) pointed out that between 1987 and 1994, mortality from coronary heart disease decreased during the same period that the incidence of myocardial infarction remained constant among whites and increased among blacks, especially black women. The prevalence of another obesity-related disease, diabetes, has actually increased significantly over the past two decades. As effectiveness in treating heart disease has improved, the effect on the overall occurrence of diseases related to obesity may have declined. Thus although prevention of risk factors such as obesity, nutrition and physical activity will remain important, as other mitigating factors can offset the effect of the proximate cause of mortality, the ultimate effect of the risk factor can be reduced.

Some of the health hazards associated with being overweight or obese may be more strongly related to the pattern of body fat distribution or possibly to fluctuations in weight than to the excess weight per se. Those with an excess accumulation of abdominal adipose tissue are at increased risk for several medical conditions, in part because fat stored in different locations can have different characteristics. Metabolic changes can result from an increase in fat stores, in which the fat cells themselves enlarge and produce chemicals that increase the risk for several diseases. In addition, increased mass itself can cause disabling conditions and injury.

In addition to obesity and overweight being risk factors, significant change in weight can be a risk factor as well. Such a change will likely involve a change in body fat, as changes in weight is less likely to be due to a change in frame size or lean mass. Note that common obesity metrics will incorporate a change in weight.

However, some increase in weight as part of the aging process is to be expected and non-health threatening—interestingly, this expected age-related increase in weight occurs at the same time that there is an increase in mortality rates through the 50s, although they are not necessarily related. Beyond the 50s, muscle mass tends to be replaced by fat, much of which is in the abdomen, manifested by increasing waist circumference.

Unexplained or unintentional decreases in weight also can affect mortality.

Diseases and conditions often associated with obesity have been found to include:

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

This is one of the most costly and burdensome chronic diseases and one of the fastest growing public health problems globally. Its existence is associated with a doubling of the risk of heart disease and stroke and is the leading cause of blindness, kidney failure and non-traumatic amputations. In addition, a sufferer is twice as likely to become depressed as a non-sufferer. According to the CDC, in 2006, 7.8 percent of American adults had diabetes, up from 7.4 percent in 2005, 5.1 percent in 1997 and 3 percent in 1980. The corresponding 2006 incidence rates for Hispanics and non-Hispanic blacks are 10.4 percent and 11.7 percent, respectively. Its incidence generally increases with advance in age.

The burden of diabetes and cardiovascular disease falls disproportionately on racial and ethnic minority groups, as can be seen in prevalence rates, especially blacks and Hispanics, even after socioeconomic status and conventional heart disease risk factors are considered. Although it is tempting to ascribe these differences to genetic sources, other factors may also be involved, e.g., under- or overnutrition, including breast-feeding at critical stages of fetal development that can induce permanent changes in metabolism or body composition that result in insulin resistance.

Wilde has projected the number of diabetics in the world will be 366 million by 2030 compared with 171 million in 2000.

The significant increase in diabetes has been linked with the concurrent rise in obesity. Obesity, especially when centrally distributed, predisposes an individual to diabetes by means of increased portal delivery of fatty acids to

the liver from adipose tissue. This process induces both hepatic insulin resistance and reduced insulin clearance.

Because waist measurements may be even more indicative of susceptibility to diabetes, because of the time lag involved (about 12 years according to the Framingham study), a focus on current levels of BMI only may underestimate the future risks associated with the increase in obesity and the future trend in this disease.

The North American Association for the Study of Obesity found that 85 percent of those with diabetes are type 2 and of those almost 90 percent are overweight or obese. Corresponding percentages found in NHANES (1999-2002) were 86.3 percent. One of the most significant changes in diabetes incidence rates has been the result of a gradual improvement in its diagnosis, particularly in those obese, as they are so highly related. Thus, of the total diabetic sufferers, an increasing percentage is now being diagnosed.

The Nurses' Health Study indicates that the relative risk to women of developing diabetes increases from a low point of BMI of 22. Note that since women tend to underreport weight, its self-reported findings may underestimate the effect. The relative risks of diabetes were 2.7 for those of normal weight (23-24.9 BMI), 7.6 for those overweight, 20.1 for class 1 obese and 39 for those heavier than class 1. This study found that being overweight or obese is the single most important predictor of diabetes.

Similar results were found by Weinstein et al. (2004) in the Womens' Health Study. This study also studied the relationship of weight and physical activity. Weinstein observed that, although weight had a greater influence on diabetes incidence than the level of physical activity, the latter was seen to modestly reduce the risk of diabetes, similar to the findings of the Nurses' Health Study. However, for those of a certain level of BMI, physical activity had a more significant effect; Weinstein speculated that "Although they are viewed as independent variables, they may be influencing each other and contributing to the same causal pathway. Obesity is known to increase peripheral insulin

resistance and reduces beta cell sensitivity to glucose. Although physical activity among other things, increases insulin sensitivity and has complex effects that can improve glucose metabolism, it may not fully reverse the effects of obesity. Weight loss may therefore be a key mechanism to reduce the secretion of these factors by decreasing adipose tissue volume and subsequently reducing the risk of diabetes."

Hu et al. (2006) observed similar results in the Health Professionals' Follow-up Study. This study indicated that waist circumference was a better predictor of diabetes than BMI. At least one additional study confirmed that, although both BMI and waist circumference were related to the incidence of diabetes, waist circumference was a somewhat better metric to use for this purpose. The Nurses' and Professionals' studies also showed progressive reductions in the multivariate-adjusted relative risk of diabetes with increases in physical activity.

The results of NHANES III indicated that those who are obese are twice as likely as those of normal weight to develop diabetes, while those who are obese had three times the risk. Diabetes in turn can also lead to hypertension and high blood pressure, as well as contributing to severe disability and a reduced quality of life.

Hu et al. (2006) indicated that in a study of Finnish men and women aged 35-64 over a 12-year follow-up period, the effect on diabetes incidence from obesity, physical activity and glucose levels, as are shown in Table 15. Clearly these factors are strongly associated with diabetes.

TABLE 15
Relative Risk of Type 2 Diabetes by Level of Physical Activity, BMI and Glucose Levels

Level of physical activity	Glucose level	Less than 30 BMI	More than 30 BMI
Low	Normal	1.1	13.2
Low	Impaired glucose regulation	15.5	30.2
Medium	Normal	2.2	7.3
Medium	Impaired glucose regulation	12.7	30.1
High	Normal	1.0	3.8
High	Impaired glucose regulation	5.5	19.0

Source: Hu et al. (2006)

Hu et al. (2006) concluded that "the best long-term results may be achieved when physical activity produces an energy expenditure of at least 2,500 kcal per week. The optimal approach in weight reduction programs appears to be a combination of regular physical activity and caloric restriction. A minimum of 60 minutes, but most likely 80-90 minutes of moderate-intensity physical activity per day may be needed to avoid or limit weight regain in formerly overweight or obese individuals. Regular moderate-intensity physical activity, a healthy diet and avoiding unhealthy weight gain are effective and safe ways to prevent and treat type 2 diabetes, as well as cardiovascular disease, and to reduce premature mortality in all population groups."

Although total fat intake has not been directly associated with the increased incidence of diabetes, increased levels of saturated fats have been associated with higher impaired glucose levels, while vegetable fats and polyunsaturated fat intake has been associated with a lower level. Overall, more fruits and vegetables in one's diet have been associated with a reduced risk of diabetes, while a higher intake of typical Western-style diets has had the opposite relationship.

The Nurses' Health Study II found that an increase in sugar-sweetened beverages may be associated with an increased risk of diabetes, possibly by providing excessive calories and large amounts of rapidly absorbable sugars.

Overall mortality rates of male diabetics have decreased over the last few decades by 43 percent between NHANES I and NHANES III. In contrast, mortality rates increased even more for female diabetics (hazard rates of increased from 1.31 to 2.84). This different trend by gender was so large that the overall diabetic prevalence rate for males and females is virtually identical in the more recent survey. Note that this comparison, while somewhat startling, has to be viewed with caution because of the self-reported nature of diabetic status and as NHANES can only report on diagnosed cases (since females visit the doctor more often than males, some of this differential may be due to a higher diabetes diagnosis rate) and because of the relatively small size of its gender-specific study.

In summary, although scientists have linked several genetic mutations to an increased risk of developing diabetes, most people can forestall the onset of diabetes by keeping their weight down, eating the right foods and exercising. However, if they don't, we may see an increase in coronary heart disease, even in their 30s and 40s.

2. Cardiovascular and heart disease

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

Obesity is an independent risk factor for cardiovascular disease, in addition to contributing to several of its risk factors, e.g., through the metabolic syndrome.

In addition, central adiposity has been found to be independent of relative body weight.

Coronary heart failure, for which obesity is a significant risk factor, is the only common cardiovascular condition that is currently increasing in incidence, prevalence and resulting mortality rates, with the overall five-year survival rate being no better than 50 percent.

The Framingham Heart Study reported that for the obese during its 14-year follow-up period, the relative risk of heart failure doubled after correction for other known risk factors. As reported by Kenchaiah et al. (2002), the increase in relative risk of heart failure was 5 percent for men and 7 percent for women for each increase in body weight equivalent to a 1 BMI unit across the entire range of BMIs, with no minimum threshold. Increments of BMI had a smaller effect on the risk of heart failure for those with hypertension.

According to Kenchaiah, this is probably was due to a decreased contribution of obesity to the risk of heart failure in the presence of this major risk factor. It does suggest that approximately 11 percent of heart failure cases among men and 14 percent among women were attributable to obesity alone.

NHANES III indicated that those who are overweight were 40 percent more likely to develop heart disease, class 1 obese were twice as likely, and class 2 and greater obese had a risk nearly 70 percent higher than those of normal weight.

The Chicago Heart Association Detection Project in Industry indicated that, adjusted for systolic blood pressure and total cholesterol level, the hazard rates for those obese compared with those of normal BMI was 1.43 for low cardiovascular risk factors and 2.0 for moderate cardiovascular risk factors. With its long follow-up period, the Chicago study indicated that those with three or more elevated risk factors in middle age had a median survival period more than nine years shorter than men with none or one risk factor, with a corresponding ratio for women of a seven-year shorter survival period.

In a large 15-year Finnish follow-up study (Jousilahti et al., 1996), starting at a BMI of 22, each increase in body weight equivalent to a 1 BMI unit was related to a 4-5 percent increase in cardiovascular mortality. Some studies have indicated that cardiovascular disease as related to BMI may only show up after a long follow-up period. As a result, Jousilahti indicates that without sufficiently long follow-up periods, studies may not observe the full aspect of the relationship.

The Framingham Heart Study, as indicated in Eng (2003), has suggested the following relationships:

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

Nevertheless, based on the Framingham study, it appears that reduction in the primary causes of deaths (e.g., cardiovascular disease) together with the trend in several of the risk factors (see below, e.g., cholesterol and blood pressure levels) have decreased the overall risk of being overweight as well. However, the higher prevalence of obesity, particularly severe obesity, will keep weight as being a serious health condition that will adversely affect future mortality levels.

Nemetz et al. (2008) found in a study of autopsy results (the gold standard in assessing causes of death) during 1981-2004 analyzing the non-natural deaths of Olmsted County, Minnesota residents who were ages 16 through 64, that declines in the grade of coronary disease ended after 1995 and possibly has reversed after 2000. Any increased prevalence resulting from improved survival from coronary disease was offset by reductions in disease incidence. These findings suggest that the declines in coronary disease prevalence may

have ended. If this is the case, it is a signal that the significant reductions in cardiovascular disease experienced over the last 40 years may be ending—a significant finding for future possible trends in overall mortality levels as well. But note that the authors end their paper by indicating that "the extent to which recent trends are attributable to the epidemics of obesity and diabetes mellitus awaits further investigation." This study's limitations are its sample size and limitation to the study of a single county in the United States primarily consisting of non-Hispanic whites.

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

Supporting this possible trend, Ford and Capewell (2007) indicated that in the 1980s, mortality rates from coronary heart disease annually fell by 6.2 percent for men aged 35 and 54, 2.3 percent in the 1990s and 0.5 percent between 2000 and 2002, while for females mortality rates decreased by 5.4 percent in the 1980s and 1.2 percent annually in the 1990s, but increased by an average of 1.5 percent between 2000 and 2002.

3. Cardiovascular risk factors

In spite of the reported relationships between various risk factors and cardiovascular disease, the rate of ischemic heart disease as well as key cardiovascular disease risk factors have declined during the last 30 years. According to various NHANES (as reported by Gregg et al., 2005), there have also been large reductions (33-52 percent reductions in the last 30 to 40 years) in the prevalence of high cholesterol level, high blood pressure and smoking in the overall population that has been offset somewhat by an increase in

diagnosed and undiagnosed diabetes of 55 percent. According to Brown (2000), reporting on results from NHANES III, "the importance of increasing BMI as a determinant of these conditions (positive relationships with systolic and diastolic blood pressures, total cholesterol, as well as the negative relationship with HDL-C) is clear."

As gathered by NHANES 1999-2000, among the obese the prevalence of high cholesterol, high blood pressure and smoking was 21 percent, 18 percent and 12 percent less than that of corresponding obese individuals surveyed in NHANES I, while its prevalence declined between 12 percent and 14 percent for those in the normal BMI category.

Gregg thus found that, over the last 40-year period, with the exception of diabetes, the prevalence of the major risk factors has declined over recent decades among all BMI groups. This is consistent with the trend in risk mitigation (drug) treatment of these factors. Nevertheless, according to Ezzati et al. (2007), based on NHANES and BRFSS, this trend appears to have changed, the prevalence of uncontrolled hypertension throughout the 1990s among U.S. men has begun to stagnate or decline at a slower rate (19 percent to 17 percent between the early 1990s and early 2000s), while they have actually increased for females (17 percent to 22 percent). Unless this recent trend reverses itself, the rate of decline in cardiovascular disease may also begin to decelerate, consistent with indications of Nemetz et al. (2008).

While obesity remains associated with elevated levels of several risk factors relative to those of lean persons, it is often overlooked that the level of risk factors has now diminished such that some of them are lower than those of lean individuals of 30 years ago. BMI, one among several determinants of cardiovascular disease risk, may have been treated in a more preventive way recently, reducing its ultimate effect on this risk.

During the last part of the 20th century, overall mortality trends have been quite favorable. In large part, this has been due to the significant (by more than 40 percent between 1980 and 2000) reduction in cardiovascular heart disease.

Table 16 shows an attribution of the sources of this change developed by Ford et al. (2007).

TABLE 16
Attribution of Sources of Mortality Improvement Between 1980 and 2000

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

Source: Ford (2007)

Despite the observed relationship between obesity and hypertension and high levels of blood cholesterol, the effect of treatment to mitigate these risk factors has shown significant mortality improvement over this period in spite of the increase in those who are obese and overweight.

Bogers et al. (2007) conducted a 28 study meta-analysis of the relationship between coronary heart disease and overweight and obesity for which all 21 studies consisting of about 300,000 subjects with follow-up periods between 4.8 and 35 years that included multiple adjustments for age, sex, physical activity and smoking, with simultaneous adjustment for blood pressure and cholesterol levels. It found that even for moderate overweight there is a significant increased risk of coronary heart disease, independent of traditional risk factors. However, adjusting for blood pressure and cholesterol level lowered the excess risk by about 45 percent to a hazard rate of 1.16 for those overweight (before adjustment it was 1.32), although the authors noted that some of this may be due to confounding factors such as nutrition. The corresponding hazard rate for those obese after blood pressure and cholesterol level were adjusted for was 1.39 (before adjustment it was 1.69). In addition, the study indicated that overweight is associated with increased risk of diabetes.

A discussion of each of these major factors follows:

- Hypertension or high blood pressure. Obesity may blunt certain actions of insulin that open blood vessels and may cause structural changes in the kidney and abnormal handling of sodium. Because those overweight generally consume more calories, they also are likely to take in more sodium. It is also associated with alterations in the systems that regulate blood flow and cardiac output. Must et al. (1999) found that NHANES II indicated that the obese had high blood pressure twice as often as those of normal weight.

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

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

U.S. non-Hispanic adult blacks have a significantly higher prevalence of hypertension than non-Hispanic adult whites or Mexican Americans (rates for males of 41.5 percent compared with 29.3 percent and 26.1 percent, respectively, and for females of 44.3 percent compared with 29.0 percent and 29.7 percent, respectively, at 2001-2004, according to Health, United States, 2007, for those 20 years and over, on an age-adjusted basis). The prevalence of hypertension of women has been increasing while the trend for men has been decreasing.

A Finnish study (Jousilahti et al., 1996) found that with long-term intervention, voluntary weight loss has been shown to be effective in the prevention of hypertension. Since obesity is the strongest determinant of hypertension, weight control could be the most effective means to prevent hypertension. Estimation of the effect of weight control on coronary heart disease mortality among hypertensive subjects should be based on the simultaneous effect of BMI and blood pressure levels. This study indicates that even if obesity is not an independent risk factor and works through other factors, it should not matter, as it is the combination of risk factors, including weight, that is important in controlling cardiovascular disease.

Hypertension can lead to premature atherosclerosis, coronary artery disease, heart attacks, abnormally large hearts and strokes.

- High levels of cholesterol. In addition to the higher frequency of high levels of cholesterol, the pattern of fat distribution affects the level of cholesterol on an independent basis, particularly relative to those with predominant abdominal obesity. This can also lead to coronary artery disease and heart attacks and strokes.

Obesity has been shown to be associated with high levels of cholesterol. Although cholesterol has been treated effectively with medicine, reduced weight, proper nutrition and physical exercise have also been shown to have a favorable effect. During the 1980s and most of the 1990s significant reductions in cholesterol levels occurred, with smaller reductions in the late 1990s, even with an increase in the use of cholesterol-lowering drugs. Although it is unclear whether this trend will continue, the continued growth in prevalence in obesity will certainly not help.

- Triglyceride (fat) levels in adipose tissues represent the cumulative effect over time of differences between energy intake and expenditures. This is a key risk factor for heart disease that is usually high in the obese.

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

4. Cancer

Overweight and obesity, as well as physical activity, have been associated with and in some cases are direct causal factors for certain types of cancers. According to the American Cancer Society, "except for quitting smoking, the best way to cut your risk of cancer is to achieve and maintain a healthy weight, to be physically active on a regular basis, and to make healthy food choices." The American Cancer Society estimated in 2002 that obesity among women was linked to 51 percent of all new cancer cases and 28 percent of cancer deaths in 2002; the corresponding percentages were 14 percent and 13 percent for males.

Calle et al. (2003), reporting on the results of the Cancer Prevention Study II, showed that the proportion of deaths from all forms of cancer in the United States due to being overweight or obese was between 4.2 percent and 14.2 percent for men and from 14.3 percent to 19.8 percent among females, with the lower percentages reflecting those who had never before smoked. NHANES through 2004 indicated that men and women who were class 3+

obese experienced mortality 52 percent and 62 percent, respectively, of that in the normal BMI category. For female class 3 obese who had never smoked, the relative risk was 88 percent higher. BMI was shown to be significantly associated with higher rates of death due to cancer of the esophagus, colon and rectum, liver, gallbladder, pancreas and kidney, non-Hodgkin's lymphoma and multiple myeloma. In addition, significant premature deaths were observed in men of cancers of the stomach and prostate and in deaths of women from cancers of the breast, uterus, cervix and ovary.

The Nurses' Health Study showed that women who gained more than 20 pounds from age 18 to midlife doubled their risk of breast cancer compared with those with stable weight. Calle et al. (1999) found that the risk of cancer was monotonically upward sloping relative to increasing BMI levels.

The international panel convened by the World Cancer Research Fund (2007) performed a six-year evaluation of a wide range of original studies from around the world and observed that cancer patterns are primarily determined by environmental factors and not genetics, and in principle are preventable. The theme of the report was that "correlations between changes in patterns of diet, physical activity, body composition and changes in patterns of cancer provide evidence that these factors are important modifiers of cancer risk" and influence fundamental bodily processes that may promote or inhibit cancer development and progression. The chance of initiation, prevention and progression of cancer can be modified by many factors, including the amount and mix of food and nutrition. The panel emphasized that "the risk of cancer is modified, not only by obesity, as usually defined, but by overweight as well, and even by degrees of body fatness generally regarded as healthy." Possibly the most surprising finding was the degree to which being even a bit overweight is a risk for certain cancers.

Table 17 shows the level of confidence expressed by its expert panel, incorporating the results of its intense six-year information search regarding the causes of various types of cancer. The evidence cited in the third column indicates whether, in the panel's view, there was a plausible biological

connection and causal relationship between overweight and obesity and the particular type of cancer indicated. The extent of a dose-response indicates that there is a significant positive correlation between weight and the type of cancer indicated. Note that the findings also indicate that greater body fatness probably protects against pre-menopausal breast cancer.

TABLE 17
World Cancer Research Fund (2007) Assessment of the Relationship between Body Fatness and Various Cancers

Type of Cancer	Consistency Dose-response relationship	Plausible mechanisms	Cause
Post-menopausal breast	Abundant and consistent epidemiological evidence; clear dose-response	Robust evidence	Convincing
Endometrial	Abundant consistent epidemiological evidence with a clear dose-response	Robust evidence	Convincing
Oesophageal adenocarcinoma	Epidemiology is consistent, with evidence of dose-response relationship	Evidence	Convincing
Pancreas	Ample epidemiological evidence, generally consistent, there is a dose-response relationship	Evidence	Convincing
Colorectal	Abundant and consistent epidemiological evidence with a clear dose-response	Evidence	Convincing
Kidney	Abundant and consistent epidemiological evidence with a dose-response relationship	Evidence	Convincing
Gallbladder	Substantial amount of generally epidemiological evidence with some evidence of a dose-response	Probable cause of gallbladder cancer directly and indirectly through formation of gallstones	Probable
Pre-menopausal breast	Substantial amount of consistent epidemiological evidence, with a dose-response	Speculative	Probable
Liver	Epidemiological evidence shows some inconsistencies	Limited evidence	Speculative
Lung	Inverse relationship, could be caused by cigarette smoking or reverse causation due to weight loss	Limited evidence	Limited evidence

Source: World Cancer Research Fund (2007)

In addition, the World Cancer Research Fund's panel found that regular sustained physical activity protects against cancers of some sites, including

colon cancer (at a convincing level) and female hormone-related cancers (probably relating to post-menopausal breast cancer) independently of other factors such as body fatness. The panel was impressed by the overall consistency of the evidence and concluded that relatively high (but not extreme) levels of physical activity protect or may protect against cancers of the colon, post-menopausal breast cancer and endometrium. In addition, since physical activity can protect against overweight, obesity and weight gain, it also indirectly protects against the cancers indicated in Table 17 with the same likelihood. Conversely, it indicated that a sedentary lifestyle may increase these risks. The report notes that the panel is aware that average physical activity levels are continuing to decrease throughout the world. The panel found evidence of relations between physical activity and certain other cancers, but either could not identify mechanisms that might explain those relationships or that the evidence was not sufficient to reach a specific conclusion.

5. Kidney disease

Although obesity is the number one preventable risk factor for chronic kidney disease, obesity appears associated with improved survival in patients with end-stage renal disease. Obesity may result in an increased risk of chronic kidney disease, especially when additional adverse factors are present, such as diabetes or lipid abnormalities. Structural damage of the kidneys may further increase blood pressure and predispose an individual to cardiovascular events.

6. Gallbladder disease

The relationship between weight and gallstone formation is strong, with overweight women 2.5-3.0 times more likely to develop gallstones, while similar trends have been observed in men.

7. Dementia

Whitmer et al. (2008) in a long term (average 36 years) follow-up study conducted of 6,583 members of Kaiser Permanente of Northern California who had their sagittal abdominal diameter (SAD) measured in the period 1964 to 1973 when aged 40 to 45, with a diagnosis of dementia from medical records, from 1994 to mid-2006.

Their previous research demonstrated that overall, BMI is a strong predictor of dementia, including Alzheimer's, with a 75 percent increased risk for those with the highest BMI. 15.9 percent of the participants of this study were diagnosed with dementia, with hazard rates adjusted for age, gender, race, education, marital status, diabetes, hypertension, hyperlipidemia, stroke, heart disease, and medical utilization. Compared with those in the lowest quintile of SAD, those in the highest quintile had a hazard ratio of 2.72, the effect of which was only reduced to 1.92 after adding BMI to the model. Those with high SAD (>25 cm) and normal BMI had an increased risk (hazard ratio of 1.89) compared with those with low SAD (<25 cm) and normal BMI, while those who were both obesity and had a high SAD had the highest risk of dementia, a hazard rate of 3.60. This strongly suggests that obesity, especially a centralized distribution of adiposity, is a significant risk factor for dementia.

8. Other

In addition to the previously discussed conditions, other conditions that are associated with obesity include arthritis, muscular-skeletal problems caused by stress on joints, osteoarthritis, complications from hospital stays and mental conditions ending in suicide.

Possibly three-quarters of those obese suffer from fatty liver (also known as steatohepatitis). Although whether obesity causes fatty liver is not yet known, it might lead to more serious liver disease.

Cawley (2004), based on a study of the National Longitudinal Survey of Youth 1979, observed a consistent negative relationship between obesity and wages for women in the United States, whether white, Hispanic or black, although after controlling for other variables, he found that this relationship only significantly affected white females. Cawley cited sociological literature, suggesting that this is caused by the adverse psychological effect of excess weight on, or possibly discrimination against, white females, while for black and Hispanic females, heavier weight may be associated with greater self-perceived stability and power.

Klarenbach et al. (2006) studied the relationship between obesity and employment in Canada. They found that, using the Canadian Community Health Survey 2000 to 2001, the prevalence and class of obesity were associated with workforce participation and absenteeism. The study, although not performed separately for males and females, found that the odds ratios, adjusted for demographic, socioeconomic and health characteristic covariates for those in obesity class 1, 2 and 3 compared with those in the normal BMI category, were 1.06, 1.18 and 1.52, respectively. With respect to absenteeism, those in obesity class 1, 2 and 3 experienced 1.15, 1.15 and 1.24 times those in the normal BMI category, respectively. Thus, for both employment-related variables, the larger the BMI, the lower the labor force participation and higher the rate of absenteeism. The authors observed that "obesity per se may also impact worker productivity independently of the development of associated disease states," although this may be mediated by the type of jobs these workers have. The authors observed that the results may be underestimated, as they relied on self-reported weights and heights and did not take into account any premature mortality of the obese. A large 15-year follow-up Finnish study confirmed that those obese had significantly more years of work disability.

6.1.1 Aggregate Mortality Observations

Many of these diseases and conditions are related to those overweight as well as the obese, although Flegal et al. (2005) reported that, based on NHANES I, II, III and 1999-2002, those overweight were associated with about 85,000 fewer deaths than those of normal (BMI of 18.5-24.9) weight, although the CDC warns against being overweight. In contrast, underweight was associated by Flegal with about 35,000 deaths in excess of the normal weight range.

However, others such as Manson et al. (2007) have criticized the use of the NHANES experience because (1) the number of participants is not sufficiently large for the purpose used for; (2) the follow-up period is too short; (3) due to reverse causation, those with chronic diseases have not been excluded, although secondary analyses excluded smokers and those with recent weight loss were studied

simultaneously; (4) use of 18.5-24.9 rather than 23-24.9 as the referent group which would have increased the number of excess deaths by 45,000; and (5) a large percent of reported deaths occurred among those older than 70 at the time of BMI assessment resulting in the possible existence of illness-induced weight loss and loss of muscle mass. Responding to this criticism, Flegal et al. (2007) indicated that that the effects noted did not introduce a significant bias, but did agree that the health effects of overweight and obesity are complex and multifaceted.

Flegal et al. (2007), based on NHANES through 2002, estimated the number of cause-specific excess deaths in the United States in 2004. They found that 23,455 excess deaths occurred among those underweight, primarily due to non-cancer and non-CVD causes. Overweight was associated with significantly decreased mortality from cancer and non-CVD causes (-69,299 excess deaths) while obesity was associated with CVD morality. They concluded that excess mortality varied by cause, with the suggestion that there has been a decrease in the association of obesity with CVD mortality over time.

An extensive literature review prepared by the National Heart Foundation of Australia for the Australian Institute of Health and Welfare and the National Heart Foundation of Australia (2004) concluded that evidence connecting excess body weight and major risk factors for cardiovascular disease existed for: atherosclerosis, high blood pressure, particularly in children and adolescents, high total cholesterol, high LDL cholesterol, low HDL cholesterol, diabetes in adults with central adiposity, cardiovascular disease arising in young to middle-aged adults and coronary heart disease in adults.

Calle et al. (1999) found that, in general, the relative excess risk relating to obesity expressed in terms of percentage of population at older ages was greater than at younger ages, but when expressed as a hazard rate, that is, as a multiple of the benchmark mortality rate, at say BMIs between 23.5 and 24.9, it decreased. In general, the hazard rates found took the shape of a J-curve, with the lowest hazard rates at BMIs found in the range of 23.5 and 24.9, although at some age groups the lowest hazard rate was at lower BMIs. For class 2+ obese, the hazard rate varied

between 1.86 and 2.75 for those of ages less than 75, depending on age group and gender, and between 1.41 and 1.53 for ages 75 and older.

McTigue et al. (2007) makes the point that any study that finds additional mortality (or morbidity) associated with the obese, if the obese is split into its several classes, the lower classes will usually experience lower hazard rates relative to the more severe obese classes.

Ezzati et al. (2008) indicated that, based on cross-county U.S. mortality statistics, there appears to have arisen increasing mortality inequality. In fact, life expectancy declined in 11 counties for men and 180 counties for women, corresponding to 4 percent and 19 percent of the male and female U.S. population, respectively. It was concentrated in the Deep South, along the Mississippi River and in Appalachia, extending to the southern part of the Midwest and into Texas. This did not occur in the prior two decades. The decrease was due to an increase in cancers, diabetes, chronic obstructive pulmonary disease and diabetes. This trend was consistent with the geographic patterns and trends in smoking, high blood pressure and obesity.

In several types of situations it has been noted that some excess body fat (overweight) may be somewhat protective in the following cases, noting that these contribute only a very small amount of total mortality rates.

- The incidence of pre-menopausal breast cancer and the rates of hip fractures.
- In older women, some excess fat may produce extra estrogen that helps slow down bone loss and insulate bones from fall-related injuries that affect about 9 percent annually of those over age 75. When they lose weight, the additional weight may protect them from extreme frailty.
- Conditioned athletes may be overweight due to being fit and having dense muscle tissue. Being fit and overweight in these cases can be protective.
- Some ethnic groups, possibly blacks, may have a “best” weight somewhere higher than the current normal category.

- Children may have higher normal fat levels during growth spurts and around puberty.

6.1.2 Methodological Issues

Various studies of adult mortality by cause generally have made five different types of exclusions or adjustments:

1. Exclude current or former smokers. Where this information is available, it is used to avoid reverse causation effects, i.e., because smoking can cause higher mortality with a long lag period.
2. Exclude deaths in the first several years of a study, to eliminate those with undiagnosed illnesses at the baseline.
3. Exclude certain reported health conditions at the baseline.
4. Exclude those with recent weight changes, as these changes may be due to recent illness.
5. Adjust for self-reported weights or heights, as these self-reported values tend to be misstated, e.g., men tend to overestimate their own height while women tend to underestimate their own weight.

The effect of such exclusions or adjustments can be significant. For example, Stevens et al. (1998), using the results of the Cancer Prevention Study I (begun in 1960 sponsored by the American Cancer Society with a follow-up period of 12 years), simply eliminated current and previous smokers from the study. Calle et al. (1999), using the results of the Cancer Prevention Study II (begun in 1982 with a follow-up period of 14 years), went further and demonstrated that by eliminating current and previous smokers from a study, those who never smoked who were leaner than the smokers had more favorable mortality compared with current and previous smokers. As a result, Calle's resulting mortality rates had an upward sloping pattern (i.e., non-U shaped) with increasing BMI. Note that for both males and females there was a slight J-curve (somewhat greater mortality for those underweight than at normal weight), with this greater relative mortality for females. Lawlor et al. (2006) found that taking into account smoking and removing deaths during the first five years changed the findings of two different large prospective studies from not showing any relation between BMI and mortality to showing significant relationships between BMIs and mortality due to cardiovascular disease.

Adams et al. (2006) found that in the large NIH-AARP study of a cohort who were age 50 to 71 at enrollment with a follow-up period up to 10 years through 2005, there was an increased risk of death for the highest and lowest BMI categories for both men and women. These relationships were made stronger when only those who had never smoked were studied, with the risk being between 20 percent and 40 percent among those overweight and two to at least three times among those obese.

Adams' results, shown in Table 18, are an example of the similar results found in several other studies, with slowly rising hazard ratios, increasing rapidly as severity of obesity increases, sometimes more quickly for males than females, and certainly more steeply for cardiovascular disease and diabetes. At the same time, the risk of preventive death was reduced among those underweight when only smokers were studied. Although relatively few deaths were recorded for blacks and Hispanics, the relative flatness of the overweight and class 1 and 2 obese categories is noticeable, not increasing substantially until BMI levels are over 40. Note that since this study was based on self-reported weights and heights, the reported hazard ratios may have been somewhat understated as a result.

TABLE 18
Age-Adjusted Multivariate Mortality Hazard Ratios by BMI Based on the NIH-AARP Study

Characteristic		**<18.5**	**18.5-23.4**	**23.5-24.9**	**25.0-29.9**	**30-34.9**	**35.0-39.9**	**>40.0**
Men	All	1.97	1.21	1.00	0.97	1.10	1.35	1.83
	Never smoked	1.67	1.20	1.00	1.10	1.39	1.91	2.59
	White	1.99	1.23	1.00	0.96	1.08	1.32	1.82
	Black	1.90	1.35	1.00	1.02	1.16	1.44	1.68
	Hispanic	2.31	1.31	1.00	1.08	1.17	1.79	2.42
Women	All	2.03	1.15	1.00	1.04	1.18	1.49	1.94
	Never smoked	1.70	1.11	1.00	1.20	1.38	1.82	2.52
	White	2.04	1.15	1.00	1.04	1.19	1.54	1.95
	Black	2.16	1.31	1.00	0.88	1.03	1.06	1.70
	Hispanic	--	1.49	1.00	1.98	2.14	1.44	4.12

Source: Adams et al. (2006)
Note that experience for blacks and Hispanics had relatively few deaths, so their results should be viewed with caution.

Socioeconomic confounders can be significant. For example, Rogers et al. (2003) indicated that controlling for covariates and reverse causation such as

socioeconomic status and social class indicators can weaken the association between obesity and mortality, even to the point of insignificance. For example, since obesity can decrease income (wages), possible reverse causation indicates that estimates of obesity's effect should not be adjusted for income; conversely, being in poverty can contribute to obesity, suggesting that such a socioeconomic factor should be incorporated into survival models—there is no complete solution to this problem.

The Nurses' Health Study (Manson et al. (1995)) with a 16-year follow-up period found that eliminating current or former smokers suggested a monotonically increasing curve, rather than a J-curve, while including these smokers resulted in a J-curve result. A separate study of current and previous smokers resulted in hazard ratios that were relatively flat, with only a slight increase at BMIs greater than 32, with a J-curve effect at low BMIs. The increase in hazard ratios at higher BMIs was primarily due to deaths from cardiovascular diseases, although there was also a sizable increase due to cancer deaths as well. Those who gained weight (more than 10 kg, or 22 pounds) since age 18 experienced increased mortality in middle adulthood, while those who lost weight or gained only a small amount had no significant change in mortality.

It should be noted that, although it can be important to eliminate the effect of confounders or pre-existing conditions to study relative mortality risks of risk factors or conditions, the resulting hazard ratios cannot simply be applied to the entire population in projecting future deaths without further adjustment, as deaths also have to be projected for those with the pre-existing conditions or early deaths that had been deleted from a study and a projection/attribution of future deaths is never easy in this case.

6.1.3 Follow-Up Period

In some cases, it is the cumulative exposure to additional weight and fat, rather than their status at a particular point in time that more significantly contributes to higher mortality rates. If for example, someone was obese at age 40, became diabetic at age 50, had a heart attack at age 60, then lost a lot of weight and died at age 65, this person's BMI at 40 would be more significant than that at 62. The long latency period raises concerns with respect to a proper analysis of the effects of the

recent increase in obesity prevalence in that recent reported studies may not have had a sufficiently long follow-up experience period to fully recognize the effect of the overweight and obesity surge over the last 30 years.

It has been speculated that it may take 10 to 20 years or more for obesity to have its full impact on cardiovascular mortality, although Allison et al. (1999) found that in the six studies they reviewed there did not appear to be such a relationship. Dyer et al. (2004), reporting on the results of the Chicago Heart Association Detection Project in Industry study in which deaths from the first 15 years were excluded in a 25-year follow-up study, found a positive association in all age-gender sub-cohorts between BMI and mortality of those overweight and obese, indicating the importance of that follow-up period for cardiovascular disease.

According to Lew and Gajewski (1990), based on insurance experience the adverse effects on mortality of being overweight appeared to be delayed, sometimes for 10 years or longer. This finding was emphasized in an earlier investigation conducted by Provident Mutual Life that demonstrated that as the follow-up periods increased in duration, the mortality rates of overweight men rose over a 35-year period after insurance policy issuance.

Interestingly, Adams et al. (2006) split studied experience into its first five years and more than five years follow-up period. For men during the less-than-five-year follow-up period, the hazard ratios were 0.93, 0.99, 1.17 and 1.54 for those who were overweight, obese class 1, obese class 2 and obese class 3+, respectively, while for those with more than a five-year follow-up, the ratios were 0.99, 1.19, 1.52 and 2.11, respectively. For females, the corresponding ratios were 1.03, 1.10, 1.30 and 1.65, compared with 1.06, 1.25, 1.66 and 2.20. This may imply that a long lag period exists between a given categorization of BMI and resultant mortality.

Observations from the Framingham Study indicated that "overweight can be an independent, long-term predictor of cardiovascular disease." Also a long latent period was found in the Manitoba Study of Canadian Air Force recruits examined in World War II which suggested that this period might extend for as long as 16 years.

The relative level of mortality for those overweight (BMIs between 25 and 30) in Flegal et al. (2005) based on NHANES through 2000 indicated that being overweight was not associated with excess mortality. In addition, this was true even with a follow-up period up to 10 years. Flegal speculated that this might be due to improved medical management of obesity-related chronic diseases, especially cardiovascular disease (except for diabetes), while others have suggested that the results were, at least in part, due to inadequate control for the combined effect of smoking and chronic illnesses.

A time lag certainly exists between obesity and development of chronic health problems or death. To contrast obesity with smoking, the largest declines in smoking occurred between 1960 and 1980, whereas the largest increase in obesity rates occurred since 1980—we haven't seen the increase in obesity's full long-run consequences yet.

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

6.1.4 Black Females

A population segment with seemingly unique obesity and mortality characteristics consists of black females in the United States. This ethnic/gender segment has by far the largest percent overweight and obesity, and experiences greater postpartum weight retention than do white females. At the same time, their mortality has been relatively flat relative to corresponding BMI levels. The following is a discussion of this and related experience.

Calle et al. (1999) found that BMI was less of a mortality factor for black women than for white women. In contrast, they found little difference between white and black men, although this latter conclusion was not statistically significant because so few black men were included in the study. There was a relatively weak relation between weight and mortality among blacks relative to whites (1.35 and 1.21, as a multiple of

age-adjusted hazard ratios of 2.58 and 2.00 for whites) compared with those with a BMI in the range of 23.5 to 24.9, which was not statistically different from 1.0.

This relatively flat curve with respect to BMIs seems inconsistent with the observation that black women generally suffer from higher mortality than white women from diabetes and heart disease (a reasonable result given the higher average BMI of black women). However, Folsom et al. (1998) found that the relation between BMI and the incidence of coronary heart disease is broadly similar among blacks and whites. Meanwhile, Calle et al. (1999) observed that the central disposition of fat in many black women may have a weaker effect on risk factors such as levels of cholesterol, triglycerides and sex hormone-binding globulin and degree of peripheral insulin resistance.

A possible explanation presented by Williamson (1999) is that many blacks have less access or do not use regular sources of health care as frequently as their white counterparts and may on average have less adequate communication with physicians than whites. As a result, blacks may underreport current disease, resulting in an incomplete control for its confounding effects in this or similar studies. However, further research is needed to confirm this hypothesis.

Although several studies, such as Calle et al. (1999) have found that the expected association of BMI and mortality (based on studies of whites) for blacks, in NHANES and NHIS, the shape of the curve is similar. The use of a higher BMI cutoff point for determining what is obese for black females may be appropriate. Certainly when different subpopulations, such as those based on racial/ethnic group are compared, separate analysis of black females should be considered, as both their relative weight distribution and mortality results are quite different—with results for ethnic male groups often relatively similar and females being quite dissimilar.

Sutocky (2005) reported on a 2000-02 California study that indicated that black adults had an age-adjusted obesity related death rate (19.9 per 100,000) that was significantly greater than for all other racial and ethnic populations (e.g., whites had 10.5 per 100,000 rate and Asians/Pacific Islanders had a 1.2 rate), although this study did not adjust for confounding factors. As a direct or contributing cause of death,

obesity was responsible for a monotonically increasing mortality rate between 1990 and 1998. Stevens et al, (1998), describing the results of American Cancer Society I with 12 years of follow-up, found no significant association between BMI and mortality for black women with less than a high school education, while for high school-educated black women there was a significant association. A similar result was found in a study of members of the Kaiser Foundation Health Plan followed up for 15 years. Nevertheless, BMI was a less significant factor in black women compared with white woman of the same educational background in the study.

McTigue et al., in the Women's Health Initiative Observational Study with a follow-up period of 7 years, found somewhat higher all-cause mortality than black women compared with that for white women, with hazard rates adjusted for smoking, education, region and physical activity of 1.13, 1.35, 1.99 and 1.55 for overweight, class 1 obese, class 2 obese and class 3+ obese, compared with 0.99, 1.12, 1.37, and 1.86 for the same BMI class for whites, respectively. Interestingly, the hazard rates for coronary heart disease mortality were all less for black women than for white women, although the hazard rates for coronary heart disease incidence were all high for each obese class.

Fontaine et al. (2003), reporting on experience of NHANES I, II and III, indicated that reasons for the different pattern between black and white females included (1) the causal effect of BMI may differ between blacks and whites; (2) blacks and whites may be exposed to different competing mortality risks, e.g., the leading cause of death for blacks aged 15 to 34 is homicide that should be independent of BMI; (3) there may be different distributions of confounding variables; and (4) the critical variable may be central adiposity and not BMI, with the relationship between metabolism, BMI and central adiposity possibly differing by race. Cossrow and Falkner (2004) indicated that greater visceral adipose tissue has been observed in whites compared with blacks despite the greater total fat in black women. According to NHANES III, waist circumference values that corresponded to both overweight and obesity were substantially lower in blacks (and Hispanics) than whites, which existed in childhood as well. Note that Fontaine assumed an individual's BMI remains constant by age.

Kumanyika (2005) concluded that comparisons of relative risks between ethnic groups with grossly different mortality risk profiles might be misleading in the first place. The significant difference between white and black female mortality experience is in the lower range of BMIs, thus contributing to the flatness of the BMI relationship with mortality. She suggests that the selection of proper indicators is not as straightforward as using the BMI measure and may be significantly influenced by the rates in the lean reference population, and "it is difficult to conclude that obesity and, presumably, obesity-related morbidities in black women are making no contribution to their mortality."

6.1.5 CDC Estimates of Deaths Due to Overweight and Obesity

The current official CDC estimate of U.S. deaths caused by overweight and obesity is 112,000. However, the CDC notes that "this does not include deaths related to poor nutrition and physical inactivity. For example, people with a normal weight can die of heart disease caused, at least in part, by poor diet and/or lack of physical activity."

The reduction from the CDC's estimate of 365,000 prior to 2005 reflected the effect of mitigation against some of the key risk factors for heart disease, such as better drug management of high blood pressure and cholesterol, and life-saving interventions such as catheterization. This resulted in an estimate of fewer deaths due to obesity.

Such attribution studies to individual causes are usually difficult and as a result can be controversial. Some early studies applied the additional expected mortality rates to the total obese population to derive an attribution estimate to a single cause. Since there are often other contributing causes, the entire additional mortality should have not been included in the single cause attribution, so that methodology was flawed. That the proper percentage for attribution purposes is difficult to estimate should not hide the fact that there should be a reduction. Conducting the analysis separately by age where possible, as is currently the case, is reasonable.

The rapid change in the percentage of the population who are overweight or obese will tend to increase this attribution for the purpose of future mortality projections, although this may be offset at least to some extent due to more effective utilization of intervention methods. Due to the uncertainties regarding the future, this estimation will remain difficult and controversial.

6.1.6 Older Adults

Many studies that have analyzed experience of older ages have indicated that the relationship between BMI and mortality weakens (at least on a multiplicative basis) at advanced ages. BMI, a surrogate or indirect estimate of adiposity, may underestimate the fatness in older adults whose BMI level is similar to younger adults. In addition, those most sensitive to adverse health effects of obesity may have died before reaching older ages, resulting in older adult cohorts being more resistant to the effects of obesity. This is in part due to the loss in muscle and bone mass that can be the result of inactivity or illness. As a result, the interpretation of the effect of measures based on weight and height is complicated. Note that Calle et al. (1999) found that the absolute (not proportional) additional risk of death associated with adiposity was highest at the oldest ages.

Methodologically, due to the likely lag time between the time of obesity measurement and future physical conditions and death, as well as the cumulative effect of prior excess weight on internal organs, it may be more relevant to study obesity metrics of those who are younger for a longer follow-up period than it is to study mortality of the elderly based on current weight on a more concurrent basis.

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

The Cardiovascular Health Study followed 4,968 older than 65s for a follow-up period of up to nine years and was reported on by Janssen (2007). In it, the all-cause mortality risk for those overweight (defined in terms of BMIs for those greater than age 65) were 11 percent lower, but was moderately greater for arthritis and was greater for physical disability with diabetes by 78 percent, adjusted for relevant covariates. Janssen concluded by raising the possibility that a BMI of 25 may not be an appropriate cut-off point for use in studies of those over 65.

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

Crimmons and Saito (2005) found that in a study of a group of 7,000 over 70s, their remaining life expectancy was quite similar, 12.3 years for those non-obese males compared with 12.4 years for the obese males. However, a significant number of non-disabled (measured by activities of daily living) years was only 9.8 years for the non-obese, compared with 8.4 years for the obese, indicating a significant disability risk for the aged overweight or obese. Corresponding life expectancy values for females were 15.3 years compared with 15.4 years, and 8.1 and 7.4 non-disabled years.

In a follow-up study of up to nine years (the Cardiovascular Health Study of 4,968 men and women over age 65), Janssen (2007) found that overall mortality for

those overweight and obese was more favorable than for those in the normal category (BMI between 20.0 and 24.9). The results for overall mortality and morbidity conditions are shown in Table 19 for which information was provided. The hazard ratio results (as a ratio to the normal BMI category) shown were adjusted for age, gender, race, socioeconomic status and prior condition (for myocardial infarction, stroke and cancer), as applicable. It indicates that, based on the study subjects, mortality was favorable for those overweight or obese at the beginning of the study, while several of the morbidity outcomes were less favorable than those of normal BMI, particularly for diabetes and arthritis, for both of which weight is a significant risk or causative factor. In fact, the higher rates for diabetes are of concern, due to the significant increase in diabetes prevalence at older ages in recent years.

Similar to other such studies, the following limitations to the interpretation of the conclusions reached by Janssen apply: (1) waist circumference may be a more significant metric for those older than 65 than BMI; (2) height, weight and many of the outcome measures were based on self-reported values; (3) the study was composed primarily of Caucasians and thus may not be relevant to all racial/ethnic groups; (4) it may not have controlled adequately for all confounding variables; and (5) it was based on observational cohorts and not randomized trials.

TABLE 19
Hazard Ratios Relative to Standard BMI (20.0-24.9) for Older Adults (age-specific values shown where significantly different than all ages)

	Overweight			**Obese**		
	All ages	**65-74**	**75+**	**All ages**	**65-74**	**75+**
Mortality	0.89			0.83		
Morbidity						
Myocardial infarction	1.16	1.39	0.99	1.16	1.38	0.91
Stroke	1.05	0.90	1.00	1.11	1.21	1.16
Cancer	0.94	1.03	0.86	1.19	1.39	0.76
Diabetes	1.78			4.15		
Arthritis (hip/knee)	1.49			1.92		
Sleep apnea	1.10			1.48		
Physical disability	1.11			1.51		
Osteoporosis	0.88			0.74		

Source: Janssen (2007)

Takata et al. (2007) reported that a study of 80-year-olds conducted in Japan with a follow-up of four years indicates that mortality of those overweight in that elderly age category may be more favorable than that of normal BMI due to better

than normal cardiovascular experience. Meanwhile, mortality for those who are underweight (BMIs less than 18.5) was greater than normal due primarily to cancer risks. Note that experience of Japanese lives may not apply to American experience.

Patterson et al. (2004) noted that over a broad range of diseases and health complaints in the Vitamins and Lifestyle cohort study of western Washington State that studied 73,003 men and women in obese classes 2 and 3 between ages 50 and 76, there were significantly increased risks of diabetes, knee replacement and hypertension.

Lozonczy et al. (1995) found in the Established Populations for Epidemiologic Studies of the Elderly of 6,387 whites age 70 and over during the 1980s, that the reduction in hazard ratios between the 50s and 70s was explained, in part, by weight change. Compared with persons with stable weight, those who lost 10 percent or more of their weight, after adjustment for health status, eliminated the higher risk of death associated with low weight. The inverse association of weight and mortality in old age appeared to reflect illness-related weight loss from heavier weight in middle age.

Krueger et al. (2004) found, using NHIS data for U.S. adults aged 60 and over, that obese individuals have higher risks of overall, circulatory disease, and diabetes mortality. Their smoking status tended to suppress the relationships between obesity and overall, circulatory disease, and cancer mortality.

Reverse causation can cause significant problems in interpreting the results of studies of mortality of the older segment of the population, in that many chronic diseases lead to weight loss at those ages, particularly when relatively short follow-up periods are involved.

As has been described, studies of older age experience have found less correlation between BMI and mortality than that for younger ages. An example can illustrate the reasons for that if insufficient follow-up periods are provided for—an individual might be obese in his 40s that results in an onset of diabetes in his 50s, which in turn might lead to a myocardial infarction in his 60s, a heart failure and weight loss in his early 70s and death shortly thereafter. In this case, an

epidemiological study that only measures BMI at age 70 would not be able to identify the original cause of the premature death without a very long follow-up period. This causal pathway problem can lead to misleading results. Studies with longer follow-up periods and exclusions of pre-existing conditions (e.g., those who smoke) can provide a better perspective on the problem being assessed as a result (e.g., the Nurses' Health Study and the National Institutes of Health-AARP Diet and Health Study).

Rimm et al. (1995) and others have observed that BMI may be a less useful indicator of adiposity among those at advanced ages, who tend to have a shift of fat from peripheral to central sites with a resultant increase in their waist-to-hip ratio but no increase in BMI. Although Rimm found that men less than age 65 had a relative risk of 1.72 for BMIs between 25 and 29, 2.61 for BMIs between 29 and 33 and 3.44 for those greater than 33 BMIs, results for the larger BMIs for those older than age 65 were much weaker. In this case, the use of the waist-to-hip ratio metric provided a much stronger predictor of risk than did BMIs (a 2.76 hazard ratio between the highest and lowest quintile of the waist-to-hip distribution).

The effect of income, education and occupation also appears weaker for those at older ages, although income has been shown to have a larger effect among females. In addition, the flattening of the BMI/mortality curve at these ages may be in part the result of the “selective survivor” effect—that many at higher risk may have died at younger ages, with only the healthier obese individuals living at these ages.

Thinness or being underweight can be more significant for the older old (particularly for those age 85 and older), due to frailty that may be both a result of one or more diseases or exposures and may provide limited protection against others. The larger the number or severity of diseases, the more difficult is the assessment. The mitigating effect of fitness may be even more important for older adults than for those at younger ages.

Other studies relating to older age individuals include:

- Folsom et al. (2000), reporting on the Iowa Women's Health Study of 55- to 69–year-olds, found that an abdominal metric was superior to the BMI in predicting mortality over its five-year follow-up period, e.g., a

multivariable-adjusted relative risk ratio for the highest quantile relative to the lowest one of 1.2, compared with 1.1 using waist circumference and 0.91 for BMI. All three metrics were strongly correlated with the incidence of diabetes and hypertension, with waist-hip measure being less consistent with the other two with respect to cancer incidence.

- McTigue et al. (2006) reported on a study of 90,185 participants in the Women's Health Initiative Observational Study with an average follow-up period of 7 years. It demonstrated that at older ages, the hazard rate decreased by age, but increased by degree of obesity. For example, for white women, without adjustment for smoking, education, region and physical activity, the hazard rates are 1.17, 1.30, 2.05 and 3.34 for ages 50-59, compared with 1.15, 1.34, 1.62 and 2.35 for ages 60-69 and with 0.88, 1.04, 1.28 and 1.42 for ages 70-79. After adjustments, all of the values were reduced somewhat.
- Wannamethee et al. (2007) reported on a British study of older men with an average follow-up period of six years of underweight men who had exceptionally high mortality rates, and that the three metrics studied on women showed little relation to mortality after lifestyle characteristics were reflected. However, after adjustment for those underweight and muscle mass (measured by midarm muscle circumference, MAMC)), both high waist circumference (greater than 102 cm) and waist-to-hip ratio (looking at the top quartile), were related to increased mortality. It found that a superior metric was a combination of high waist circumference and low MAMC, with a risk ratio of 1.55.
- Manini et al. (2006) studied a randomly selected group of 70- to 82-year-old Medicare recipients in the Health, Aging and Body Composition Study with an eight-year follow-up period whose energy expenditure was measured over a two-week period. Those who reported low physical activity levels experienced elevated mortality, in that the highest tertile of activity energy expenditure was about half of that in the lowest tertile.
- Similarly, Sui et al. (2007), studying the 12-year follow-up results of those over age 65 enrolled in the Aerobics Center Longitudinal Study, found that fitness was a significant mortality predictor (a mortality ratio for the first quintile of about 300 percent of that of the fifth quintile), independent of

overall or abdominal adiposity. Those with class 1 obesity experienced about 30 percent higher and with class 2 or 3 obesity about 130 percent higher mortality rates, with those with a waist circumference of greater than 88 cm for women and 102 cm for men having about 30 percent higher mortality rates.

- Jenkins (2004), using data from the Asset and Health Dynamic Among the Oldest Old Survey, found that being overweight or obese makes individuals more likely to experience the onset of function impairments. Obesity had an independent effect on the onset of strength impairment, lower body mobility and activities of daily living problems. Separately, based on the increase in dementia found by Whitmer et al. (2008) associated with BMI and visceral adiposity, the use of long-term care facilities will increase in the future as those with greater weight reach older ages.
- Lackdawalla et al. (2005) indicated that, although obese 70-year-olds live about as long as those of normal weight, they will spend $39,000 more on health care, have fewer disability-free life years and have higher rates of diabetes, hypertension and heart disease. The additional health care costs are not expected to be offset by health care savings due to higher mortality rates.

6.2 Children

Several obesity-related chronic diseases are becoming more common in children, some with immediate adverse effects and others with a delayed effect expected to become more serious healthwise as they age. These longer-term effects might include a change in hormonal pathways, fat cells and eventually the brain, which in turn could increase subsequent appetite and adversely affect metabolism. Without effective intervention, these trends, together with the effects of adult obesity discussed in Section 5.3, also have the potential to increase future mortality, worker productivity and disability.

For example,

1. Type 2 diabetes. Once believed to affect only adults, type 2 diabetes is now being diagnosed in children. In some areas, almost half of the pediatric

diabetes cases has been type 2, when historically the rate during childhood was close to zero. This increased prevalence raises concerns regarding the effects of the long time exposure to this disease and potential early onset of diabetes-related comorbidities. Although still relatively rare, obese and overweight children with this disease will likely be at risk of suffering the serious complications of diabetes as adults. Note that this prevalence is still low, in 1999-2001 being less than 1 percent.

A 2007 *Financial Times* investigation carried out by Medco Health Solutions, Inc. (a drug benefits manager) between 2001 and 2005, found that the number of U.S. children covered by insurance who were prescribed medicine for type 2 diabetes increased by 115 percent in four years, to about 2.7 girls and 0.8 boys per 1,000 in 2006. Those uninsured may have even higher diabetic rates. The study also found that those children who were taking medicine for diabetes also faced other serious problems—17 percent of boys and 13 percent of girls currently on drugs for high blood pressure; 5 percent were taking cholesterol-reducing drugs and nearly 20 percent were also taking narcotic pain relievers and drugs for respiratory, asthmatic conditions and anti-depressants.

2. Cardiovascular disease and related risk factors. According to the CDC, 61 percent of overweight 5- to 10-year-olds have at least one risk factor for future heart disease and 26 percent have two or more risk factors. Hypertension and early symptoms of hardening of the arteries have become more common in youngsters—2.5 to 3.7 times greater for overweight children, depending on their ethnicity/race and gender. Liu et al. (2007) indicated that overweight adolescents have high levels of various insulin resistance syndrome factors, including an abnormal lipid profile during adolescence that may lead to correspondingly high levels of LDL cholesterol when they become adults. This is an example of additional future mortality risk for those currently obese adolescents.

Overall, obesity can harm the cardiovascular system and can accelerate the development of heart disease. It has been estimated that 4 percent overall and

30 percent of those obese have the metabolic syndrome that is associated with increased risk of cardiovascular disease and diabetes. Since the processes that lead to a heart attack or stroke can take decades to progress to the point of overt disease or attack, if they start in childhood, they are more likely to result in mortality at earlier adult ages.

3. Fatty liver. One in three obese children has a fatty liver (also known as steatohepatitis). Both obesity and insulin resistance probably play a strong role in this disease process. It might eventually lead to liver disease, such as cirrhosis of the liver.

4. Educational achievement. Although Kaestner and Grossman (2008), based on the 1979 cohort of the National Longitudinal Survey of Youth (NLSY), did not find evidence that in general children who are overweight or obese have achievement test scores significantly different than children of average weight, Sabia (2007) found that, based on the 1994-96 academic school years as reported by the National Longitudinal Study of Adolescent Health, there was strong evidence of a negative relationship between body weight and academic performance of white girls. Although the same negative relationship was found for non-white girls, they indicated that unobserved heterogeneity may explain this association. There was no corresponding relationship for males. It has been hypothesized that this relationship may be the result of a lack of self-esteem or even school discrimination.

5. Psychosocial. Social and psychological problems may arise as a result of obesity, including low self-esteem, negative body image, eating disorders and inappropriate weight loss attempts, depression, anxiety, social isolation, obesity stigmatization and avoidance of health services. The extreme result would be attempted suicide. A significant result may be increased periodic attempts at dieting, which is rarely effective in practice. Those obese are less likely to complete college and more likely to live in poverty. In fact, if body size is made too much of an issue, psychological issues associated with low self-worth and self-image may arise simply as a result of focusing on finding solutions to their overweight state.

6. Other. Other adverse effects can include dyslipidemia, musculoskeletal, orthopedic complications, learning disabilities, asthma, memory defects and sleep apnea. In addition, there is also a consequential increased future risk of obesity in their offspring.

Not only is there a minor risk of premature death while being a child, but as discussed in Section 5.3, adolescent obesity can significantly increase the odds of being obese when an adult. This in turn can contribute to premature death over the person's entire expected lifetime, although the precise pattern of additional premature deaths over the long term is not yet certain.

The large Nurses' Health Study II found (van Dam et al., 2006) future mortality experience based on self-reported recall (the author points out that this was a potential weakness of the study) of weight at age 18 in 1989 reflected on 12 years of subsequent follow-up. Even moderately higher adiposity at age 18 led to future higher mortality risk. Using as a base those with a BMI of 18.5-21.9, the hazard ratios were 1.18 for those with BMI of 22.0-24.9, 1.66 for those with a BMI of 25.0-29.9 and 2.79 for a BMI of 30 or more. This study was able to eliminate those with a history of significant cancer and smokers or previous smokers. In fact, adjustment for adult waist and hip circumference rather than adult BMI did not weaken the relationship between age 18 BMI and mortality. Van Dam et al. (2006) indicated that these findings were consistent with those of several other studies, although inconsistent with the findings of a Swedish study and the Harvard Growth Study. In a Norwegian study, the relationship between BMIs and adult men explained the association, but not for women.

6.3 Change in weight

Hu et al. (2006) showed that weight gain is independent of initial body weight as a risk factor. Weight gains in participants in the NHANES Epidemiologic Follow-up Study of 5-7.9, 8-10.9, 11-19.9, and 20+ kg, were associated with a 2.1-, 1.1-, 2.6- and 3.9-fold increased rate of diabetes when compared with those with relatively stable weight. In the Nurses' Health Study, the corresponding rates of increased diabetes for these same weight loss categories were 1.9, 2.7, 5.5 and 12.3, respectively.

In contrast, women who lost more than 5 kg (11 pounds) in the latter study reduced their risk of diabetes by at least 50 percent.

Weight cycling (or yo-yoing) is the result of repeated loss of weight by dieting, followed by weight regain. In fact, most attempts to diet ultimately fail, with participants typically resuming something close to their previous eating habits after some period of time. In fact, weight regain often results in a redistribution of weight, from the lower body where it may have a protective effect to the abdomen where it has been shown to be a health risk factor. Weight cycling has been shown in some cases to have just as unhealthy consequences as being at a somewhat higher but more stable weight level.

Gaesser (2004) suggested that weight cycling not only can significantly contribute to the adverse effect of obesity but can also act independent of obesity. Its effect can be especially significant for women. It can elevate blood pressure, reduce HDL cholesterol levels, deplete body reserves of omega-3 fatty acids and increase risk for gall bladder disease, kidney cancer and breast cancer. Gaesser indicated that the Framingham Heart Study revealed that virtually all of the excess cardiovascular disease mortality in obese men and women could be explained by lifetime weight fluctuations.

“Generic” weight loss is often associated with higher mortality. The frequency of unintentional weight loss is greater at older ages, often associated with a person's overall poor health status, recent hospitalization, treatment with certain medications or smoking habits. This contrasts with effective intentional weight loss that can result in lower blood pressure, lower blood sugar levels and improved lipid levels. Using results from the Framingham Heart Study, Higgins et al. (1993) found that weight loss was associated with improvements in blood pressure and cholesterol levels, as well as with continued smoking, and higher cardiovascular disease, diabetes and death rates (age- and risk-factor-adjusted ratios of 1.33 for males and 1.28 for females). Those with the greatest weight loss had larger excess mortality ratios. Being at a standard weight with stable weight maintenance were shown to be beneficial risk factors. Weight gain was associated with higher mortality for females, but with lower mortality for males. It also found that those suffering from hypertension were

particularly prone to weight gain. It was observed that it was not uncommon to lose weight shortly prior to death.

Extreme weight loss can be as a result of a serious eating disorder, such as binge eating, anorexia nervosa and bulimia, but is very case-specific.

Overall, both recent weight gain and unintentional weight loss have been shown to be risk factors. However, intentional weight loss accompanied by fat loss may be beneficial, particularly in those who are obese to begin with.

6.4 Lack of Physical Activity

Some have argued that for those not severely obese, an unhealthy diet and a lack of physical activity can cause more harm than being overweight or obese. It is accepted practice that physical fitness plays an integral role in a successful weight management program. Paffenbarger et al. (1993), commenting on the findings of the Harvard Growth Study, found that "most active men had half the risk of death of the least active." In that study, those men who walked less than nine miles per week had a 16 percent higher risk of death than those who had walked more than that amount, and those who climbed fewer than 20 flights of stairs per week had a 23 percent higher risk than those who climbed more than that amount. In addition, those who took up sports after college had mortality experience similar to those who had been vigorously active all along. The benefits of regular physical activity were found to include protection against coronary heart disease, diabetes and colon cancer.

Physical inactivity has a direct and biologically plausible relationship to the same health outcomes as those for obesity. Cardiovascular fitness can influence both fitness and health through related biological factors, and is more significant than self-reported physical activity as a predictor of many health outcomes. Wei et al. (1999), based on the Aerobics Center Longitudinal Study conducted in Texas between 1970 and 1993, studied 25,714 men for at least one year, found that obese individuals with at least moderate cardio-respiratory fitness have lower rates of cardiovascular disease and all-cause mortality (about half) than normal weight but unfit peers. This finding applied to men and women, those with diabetes, and to those at all BMI levels. Moderate intensity activity of 150 minutes a week was found to be sufficient to avoid

the effects of being in the low-fitness category. Wei observed that cardio-respiratory fitness and regular physical activity are the major determinants of fitness.

Similar results were seen in the Women's Ischemia Syndrome Evaluation conducted by the National Heart, Lung and Blood Institute and reported by Wessell et al. (2004) that studied those with chest discomfort and suspected myocardial ischemia. It found that women with a Duke Activity Status Index of 25 or greater (indicating greater fitness) had the lowest proportion of adverse event occurrences during follow-up and that physical activity and functional capacity are more important than weight status. And, despite its association with numerous cardiovascular risk factors, it found that BMI was a relatively poor predictor of both baseline coronary artery disease and future adverse events. When compared with BMI, indices of abdominal adiposity were all stronger predictors of coronary artery disease and the risk of future adverse events. Since excess weight is associated with reduced physical activity and functional capacity, the cardiovascular risk associated with obesity may be explained in part by the adverse effects of low fitness. It can be seen that both weight loss and exercise are therefore important to reduce these risks.

The results of the Nurses' Health Study (Hu et al., 2004) indicated that both lower BMI and moderate physical activity were related to lower mortality rates. Higher levels of physical activity appear to be beneficial at all levels of adiposity but did not eliminate the higher risk of death associated with obesity. Obesity and a lack of physical activity (less than 3.5 hours of exercise per week) accounted for 31 percent of all premature deaths in the study, 59 percent of the deaths from cardiovascular disease and 21 percent of the deaths from cancer among non-smoking women. A modest weight gain was also associated with a higher risk of death.

Other studies (e.g., Costa-Font and Gil (2005)) have indicated that having a sedentary job is often associated with a higher level of BMI.

A large Finnish study found that, on the basis of self-administered questionnaires, physically active subjects had significantly lower age-adjusted mortality from cardiovascular disease, cancer and all causes of death combined compared with those who are sedentary. Adjustments for smoking, blood pressure,

cholesterol, BMI, diabetes and education affected the results only slightly. It concluded that both physical activity and normal weight are important indicators of lower mortality. Physical activity had a strong independent effect on mortality, whereas the effect of BMI was partly mediated through other obesity-related factors.

Janssen (2006) conducted a meta-analysis of 26 studies that addressed the mortality effect of physical activity on those with coronary artery disease. The analysis found a significant beneficial effect, with a reduction in all-cause mortality of 27 percent. This effect was found to be consistent by age and gender. Favorable changes in physical activity were observed to have a significant effect on mortality.

From these studies, it can be concluded that a combination of physical fitness and a managed food program is a particularly powerful mitigation factor against many mortality risks.

6.5 Underweight

Anyone with a BMI of less than 18.5 is classified as being underweight (grade 1 is between 17.0 and 18.4, grade 2 is between 16.0 and 16.9 and grade 3 is less than 16.0). In many studies, excess (over the normal BMI grouping) mortality is usually reduced, but not always eliminated for those underweight, after adjustment for factors that can confound the relationship between factors, particularly current and previous smoking and pre-existing illness. For example, a large South Korean study (Jee et al., 2006) indicated that a "J-shaped” curve (with the left side of the curve being upturned at lower BMIs, although not nearly as high at obesity levels) always exists regardless of smoking history, with excess deaths being due to respiratory causes. In contrast, the Nurses' Health Study, for example, after elimination of current and prior smokers, showed no J-shape effect and with mortality increasing monotonically with BMI.

The sources of extra mortality for those who are underweight are primarily cerebrovascular disease, pneumonia and diseases of the central nervous system. Those underweight with hypertension appear to be at higher risk for heart attacks and stroke than those who are obese with high blood pressure. In addition, non-disease-caused frailty may be a factor by itself, although more often it will contribute to increased morbidity, especially in the elderly.

So much emphasis has been placed on problems of the overweight and obese that there is usually an under-emphasis on underweight problems by society and the media. Since the weight distribution has shifted so far to the right, the relatively few Americans left at the left tail of that distribution often get ignored. Certainly “ideal” underweight models are regularly paraded by the fashion industry. Those who are or want to be at those weights, especially adolescent girls, can suffer eating disorders such as anorexia nervosa and bulimia.

At the time of final submission of this paper, the French government appears to be so concerned with this problem that in April 2008 the French National Assembly passed a bill to make it illegal to "provoke a person to aspire to excessive thinness by encouraging prolonged food limitations" with a penalty of up to two years in prison and a fine of up to $47,000, although it had not yet passed the Senate. At this time it is uncertain how this law would be enforced on the fashion and advertising industries, it does mark the seriousness of concern regarding ultra-thinness in general and the large number suffering from conditions such as anorexia in particular. The French might be successful in their efforts—their fertility motivational programs have been successful in increasing their fertility rates.

Thinness in children and adolescents can also be a sign of malnutrition, as well as being a possible indicator of other diseases. Cole et al. (2007) has developed standardized BMI cutoffs for children and adolescents similar to the WHO categorization of underweight at age 18, although the authors caution that the tables need further testing.

There is concern with the elderly frail and with those who have experienced significant involuntary weight loss, who are subject to serious health risks. Another problem is associated with a lack of nutrition, whether in a relatively underdeveloped country or by those who are food insecure in developed countries. In 2008 Olle Ljungqvist, chair of the European Society of Clinical Nutrition and Metabolism, indicated that, according to the results of the annual NutritionDay study in Europe, 47 percent of patients who are hospitalized with signs of disease-related malnutrition, only 38 percent of patients eat all that they have been served and one in five eats less

than a quarter or nothing of what they are served. He indicated that a lack of adequate nutrition in the community, hospitals and in residential care inhibits patient recovery, lengthens hospital stays and increases mortality, placing unnecessary strains on the health care system.

Based on Heiat et al. (2001), those who are aged and underweight may experience even worse mortality than those who are aged and overweight or aged obese, with a U-shaped BMI mortality curve.

6.6 Insurance Experience

Before mortality statistics relating to build were first published in the United States in 1903, according to Brackenridge and Elder (1998), those underweight were thought to be much poorer insurance risks than those overweight, primarily due to the prevalence of tuberculosis. Those who were fat were thought to be well-nourished and healthy. But the gathering of life insurance industry mortality experience data changed that view.

In the Build and Blood Pressure Study of 1959, the lowest mortality ratios were those associated with those underweight, as those who would otherwise be in that category with certain diagnosed diseases were underwritten and assigned either higher premiums or declined for other reasons and therefore excluded from the study. Among those moderately overweight, the extra mortality experienced about 20 percent additional risk compared with that in the standard class for the first five years after policy issue, rising slightly in the next five years and continuing upwards over a period of another 10 years to about 35 percent additional risk, with those markedly overweight males rising to nearly 85 percent after 15 years had elapsed.

In the 1979 version of this study, the relative mortality rates for those 30-60 percent overweight improved by 10-15 percent compared with the 1959 study, while those underweight got 10 percent worse than the prior study, possibly correcting for the unexpected results of the 1959 study, although the general pattern of additional mortality was similar.

Insurance studies in the middle of the century had millions of lives traced for many years, although it included only scanty data on those extremely overweight, with experience not extending much beyond 60 percent of average weights. According to a mortality study of those overweight conducted by Cologne Reinsurance Company reported in 1969, mortality rates associated with overweight as the sole impairment remained relatively low for eight years and only then started to show a modest rise in comparison with those of standard weight. In an extension published in 1977, only very slight increments in mortality were shown with increasing durations among overweight insureds with no other complications.

In the 1979 Build and Blood Pressure Study (Brackenridge and Elder, 1998), men weighing 20 percent above average weight experienced mortality from all causes 20 percent greater than those of standard weight. These men experienced a 15 percent higher rate due to coronary disease, 150 percent higher due to diabetes and 20 percent higher due to digestive diseases. The effect of elevated weight increased significantly as weight increased. There was a slight J-curve effect at body weights for both men and women who were less than 20 percent of average weight of about 110 percent of mortality of those at average weight, although these included smokers. The results of the 1979 study have also been used to argue that optimal body weights increase at higher ages.

The extra mortality associated with being overweight when accompanied by another impairment has always been shown to be more than additive. As an individual's normal weight remains relatively constant over time, any significant deviation, particularly recent substantial deviation or rapid decreases or increases from an individual's norm has usually been considered to represent a signal of the existence or emergence of a serious disease.

It has been known by those in the life insurance industry that those with different builds (somatotypes) have different mortality characteristics. For example, the relative proportions of fat, muscle and bone making up any excess weight is important, but insurance studies have not generally taken these proportions into account, although some subjective adjustment for large abdominal girth is usually

taken into account. In general, those whose excess weight consists mainly of muscle or bone have usually considered to be better mortality risks.

Advantages of working with life insurance experience include its relatively homogeneous nature, the large number of lives studied, virtually complete follow-up (other than voluntary surrenders) over very long periods of time, accurate recording of deaths and the possibility of comparing the experience with and without other medical impairments. Limitations include the lack of those markedly underweight and obese, due to the likelihood that they would be declined coverage and that until the 1970s insurers did not distinguish between smokers and non-smokers therefore preventing the evaluation of the confounding effects of smoking.

Table 20 shows a simple (single impairment) underwriting treatment of BMI used by one life insurer.

TABLE 20
Underwriting Criteria by BMI Range Used by an Insurer

BMI	Loading (as % of standard mortality table)	Classification
<=15	decline	underweight
16	75	underweight
17	50	underweight
18-31	0	normal to overweight
32-36	25	Obese
37-39	50	Obese
40-41	75	Obese
42	100	Obese
43-44	125	Obese
45-46	150	Obese
>=47	Decline	Obese

Source: Converium standard acceptance criteria

According to Roudebush et al. (2006), based on insured experience for life insurance policies issued between 1989 and 2003 with an average duration of 2.5 years generated from the Impairment Study Capture System, standardized mortality ratios (SMRs) rose quite modestly as BMI increased up until reaching severe obesity levels. Nonsmoker insureds whose elevated build was the only reported impairment, experienced SMRs of 265 percent for BMIs less than 18.5, 130 percent at BMI 30-34.9, 160 percent at BMI 35-39.9 and 239 percent at BMI greater than 40, although

given the short follow-up period with this relatively new insurance database it is unlikely that all the adverse effects of overweight and obesity have had time to reveal themselves. Mortality ratios were greatest in policy durations 1 and 2, especially for those underweight and the extremely obese, possibly due to anti-selection.

Insurance experience regarding bariatric surgery has generally found that intestinal bypass operations have often resulted in weight stabilization in two to three years, at an average of a 30 percent reduction of initial weight. However, a relatively high rate of associated metabolic disorders follows surgery, with what is generally thought to be poor operative risks. In contrast, weight loss of 44 to 110 pounds in the first six months has been reported by gastric partition and bypass. Operative mortality rates have been 2-3 percent, with limited long-term metabolic complications reported to date.

6.7 Morbidity, Health Care and Total Cost

The primary focus of this paper and of most studies on the effects of obesity has been on mortality, even though obesity may have even a greater effect on morbidity and health care costs.

Morbidity is a measure of poor health, which can result in such adverse consequences and includes the possible risk of loss of income or an inability to perform certain activities of daily living (ADLs). Morbidity due to obesity might result from serious chronic diseases such as diabetes, heart disease, some cancers, respiratory difficulties, arthritis, osteoarthritis, chronic musculoskeletal problems, heat injuries and heat disorders, skin problems, reduced quality of life, pregnancy-related complications, infertility, psychological effects, weight stigma and medical costs for treatment of these and related ailments. Some of these conditions can also result in death, while some only contribute to what in some cases can be long-term morbidity.

Health care costs have increased in part due to more aggressive treatment of cardiovascular disease risk factors. Although these efforts may have resulted in a decrease in mortality, it has had the effect of increasing health care costs at the same time.

Physical disabilities resulting from obesity can include skeletal and joint problems such as orthopedic disorders and carpal tunnel syndrome, which are not usually associated with mortality, as well as respiratory problems, including sleep apnea, as those who are obese tend to have an increased demand for ventilation and breathing workload, respiratory muscle inefficiency, decreased functional reserve capacity and expiratory reserve volume and closure of peripheral lung units.

As an example of how difficult it is to attribute the costs associated with obesity, 25 percent of those who are obese had six or more adverse medical conditions during 2002. This complexity affects the study of the sources of both medical costs and mortality.

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

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

In a study that attempts to quantify the additional costs at current cost levels or present value, the determination of the proper discount rates and health cost inflation to be used can be significant. In addition, in certain techniques for which the value

assigned to a person's life and healthy life are attempted to be included in costs, the assignment of this values, which is subjective at best, can be important.

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

Roux and Donaldson (2003) compared the advantages of using a cost-of-illness approach to a solutions-based approach. The cost-of-illness approach aims to quantify all direct and indirect costs attributable to a disease. It includes opportunity costs associated with the allocation of resources to the interventions aimed at managing and alleviating the conditions. The solutions-based approach (used for decision-making and priority setting) is driven by a comparison of incremental costs and incremental benefits of interventions aimed at controlling the illnesses and their consequences. Opportunity costs in the latter approach include lost earnings and differences in productivity costs between the populations compared.

The approach taken to estimate the cost of obesity (or any other condition) depends on the problem for which models are developed and it is important to be clear the basis for the quantifications performed. The development of an independent estimate of the cost associated with obesity is outside the scope of this paper.

Many cost analyses have been conducted. The results of several of them follow:

- Colditz (1992). This study was conducted by evaluating the major consequential diseases associated with obesity using the results of other studies as input. First he estimated the additional cost associated with obesity by condition (diabetes, gall bladder, CVD excluding hypertension, hypertension and cancer) using a disease approach to be about 5.5 percent of the total health care costs. To supplement this, he added an assumed percentage of costs for certain other more minor conditions. In addition, he estimated the costs attributable to severe obesity (class 3 and 4) for a 34-year-old man and a 34-year-old woman, in part to estimate the total cost of bariatric surgery, including the cost of the surgery and savings due to consequential improved health effects, which in total he thought to be minor in comparison. He then added to this half the costs associated with muscular-skeletal disorders to derive a total of about 7.8 percent. In a separate estimate, Colditz estimated that $33 billion was spent in 1992 on weight related reduction products and services (including low-calorie food and diet sodas), although this did not include their possible adverse side effects.

- Wolf and Colditz (1998) estimated a direct cost of $51.6 billion and indirect cost of $47.6 billion in the United States in 1995 (equivalent to 5.7 percent of U.S. national health spending in that year).

- Quesenberry et al. (1998) reported, based on a study of Kaiser Northern California (a health maintenance organization) members, a 44 percent increase among class 2 and heavier obese and a 25 percent increase among class 1 obese compared with the health care costs of individuals whose BMIs were in the normal BMI range.

- Chu (2008) reported that in a study of 13,442 pregnancies between 2000 and 2004 covered by Kaiser Permanente Northwest, the use of health care services for overweight and obese pregnant women was significantly greater than that of those of normal weight. This was due to greater use of inpatient and outpatient health care services, including increased length of

stay during hospitalization, and greater use of physician services and less use of nurse practitioners, associated with increased use of cesarean deliveries, preexisting and gestational diabetes and hypertensive disorders. This was especially true for severely obese women.

- Allison et al. (1999). Using a prevalence-based approach, per capita health costs by age were estimated, reflecting the additional mortality for those who are obese that reduces longer-term costs, because there aren't as many alive in the later years of the projection. They compared the expected future cost of individuals aged 20 through 85. Two scenarios were presented, with a constant relative cost ratio for each (health care costs of the obese to that of the non-obese) of 2.15 and 1.35, corresponding to the assumed ratio at BMIs between 29.0 and 31.9, and 25.0 to 26.9, respectively. Their result was an estimate of a reduction of about 5.7 percent and 4.3 percent for the two scenarios compared to what the costs would be had there been no obese in the population.

- Sturm (2002) found that being obese has roughly the same association with chronic health conditions as does someone of normal BMI 20 years older, the effect of which greatly exceeds the association of either smoking or problem drinking. Obesity was associated with a 36 percent increase in inpatient and outpatient spending and a 77 percent increase in the cost of medications compared with corresponding spending in the normal weight range, and a 21 percent and 28 percent increase in spending in these two health care cost categories, respectively, in comparison with that of current and prior smokers. He associated various conditions with the following per year increases in cost:

  — obese -- $395
  — overweight -- $125
  — current or ever smoker -- $230
  — problem drinking -- $150
  — aging -- $225.

  The reason that obesity was assumed to have a larger effect than smoking is that obesity has a more significant effect on heart disease, hypertension and diabetes, all of which tend to have long-term drug regimens and are

chronic conditions, while smoking has its strongest effects on cancer and lung disease that although costly, tend to be less common and lead to death more quickly than diabetes or hypertension.

- Finkelstein et al. (2003) separately estimated the effect of obesity on Medicare, Medicaid, private and uninsured costs, the aggregate overweight and obesity-attributable medical spending for the United States and for selected private health care payers. It was estimated to account for 5.3 percent of total annual medical expenditures and 9.1 percent of the total increase in U.S. medical care costs in 1998, and as high as $92.6 billion of health care costs measured in 2002 dollars, with obesity accounting for an almost equal amount of indirect costs (mostly due to reduced productivity resulting from obesity-related morbidity). Since the percentage of overweight and obese in the population is much greater in 2007, a current estimate of this percentage would presumably be significantly larger than those estimates.

- Thorpe et al. (2004) estimated that the increase in the prevalence and corresponding health care spending of the obese relative to those of normal weight accounted for 27 percent of the increase in real per capita spending between 1987 and 2001, while the corresponding percent increase for hyperlipidemia was 22 percent, diabetes 38 percent, and heart disease 41 percent. They estimated that the increase in obesity prevalence alone accounted for 12 percent of the growth in health spending between those years, primarily due to increases in the cost of treatment for diabetes and hypertension. Others have estimated that obesity accounted for 34 percent of the growth in health care spending over the last 20 years. It is a shame that more political discussions haven't focused on the importance of prevention in any cost containment initiative, both with respect to obesity and the entire metabolic syndrome.

- The Chicago Heart Association Detection Project in Industry (Daviglus et al., 2004) that had a long follow-up period indicated that, adjusting for age and race, severely obese (BMI>35) men were 84.4 percent and women

were 81.8 percent more expensive than those who were non-overweight men based on fee-for-service Medicare charges for those age 65 through 83. The corresponding differences were 13.2 percent and 38.4 percent for male who were overweight and class 1 obese, respectively, and 9.6 percent and 35.0 percent for females, respectively. The differences in costs were due to both increased number of visits and higher costs per visit. There were also additional health care costs for those younger than age 65.

Daviglus also reported that in a Japanese study of health care costs of those overweight and obese there were 9.8 percent and 22.3 percent greater additional health care costs over a four-year period, respectively, for those aged 40-79.

- Anderson et al. (2005) estimated the combined effect of obesity, overweight and physical inactivity on health care charges of a medium size Minnesota health insurer during 1996-99 to be about 23.5 percent of total health care costs (a 95th percent confidence interval based a bootstrap measure was between 10 percent and 34 percent) of those over age 40. By adjusting this estimate to reflect more nationally representative health care charges, they derived an estimate of 27 percent on a national basis, higher than the 23.5 percent because of the different expected demographic mix. Although they estimated that the charges associated with these risk factors were greatest for the oldest group (age 65+), nearly half of the total charges were from the 40 to 64 age group without chronic diseases present. This suggests that a broad, population-wide approach to address physical inactivity and obesity may be a better strategy than a more focused one. It emphasizes the importance of primary prevention and effective management of the risk factors as a health care cost containment strategy. They were disproportionately higher for those who are old, male and have either heart disease, diabetes or both.

  This study is interesting, in part, due to its approach in studying the combined effect of obesity and physical activity because they are so behaviorally and physiologically interrelated. Either they should be

evaluated together or their interaction should be evaluated separately if possible. The limitations of this study is that it only involved a single health plan that was predominantly white and over age 40, it excluded pharmacy charges, the data used was derived from survey data and automated administrative databases, and they only included relatively simple metrics that were adjusted for self-response errors.

- Sutocky (2005) indicated that annual direct health care costs in the State of California associated with obesity were estimated by the California Department of Health Services to be about $4.11 billion in 2000, with indirect associated costs exceeding $2.25 billion.

- Hart et al. (2006), reporting on the results of the Renfrew/Paisley study in Scotland whose follow-up period was quite long (between 28 and 32 years), indicated that men who were underweight and normal weight experienced lower than expected rates of hospital admission, while obese men experienced higher than expected bed day rates. In contrast, women experienced a U-shaped admission rate relationship, with a rate of admission greater than expected for both those underweight and those obese, with normal weight women having the lowest admission rate.

- Monheit et al. (2007) reported that an econometric analysis based on data from the 2001-03 Medical Expenditure Panel Survey, found that adolescent bodyweight and the likelihood of being overweight are strongly associated with parental bodyweight, parental education, parental smoking behavior and neighborhood attributes such as the availability of fresh food markets and convenience/snack food outlets and neighborhood safety and material deprivation.

  Overweight females were estimated to have annual health care expenditures that exceeded those of normal weight by $622, while for those at risk of becoming overweight there was only a $68 difference in annual health care expenditures, with part of this difference due to

differences in mental health expenditures. Corresponding differences were not found for male adolescents.

- Van Baal et al. (2008) estimated the annual and lifetime medical costs in a Dutch study attributable to the obese, the smoker and those who weren't obese or a smoker. They used a simulation model based on relative medical costs per condition affected. Their assumptions led to the finding that until age 56 the obese have higher health care costs, while after age 56 smokers incur higher costs. However, due to differences in life expectancy, lifetime health costs were highest among non-obese and non-smoker (healthy) people and lowest for smokers; the cost for those who are obese was between the two. The conclusion was that it costs less for health care over an average lifetime for an individual who is obese or a smoker, due to their higher expected mortality rates. The assumption used was that on average smokers live about 77 years; those obese live about 80 years; and the healthy live about 84 years. The cost of care for the obese was $371,000 compared with $326,000 for smokers. They also determined the break-even discount rate that would equate the costs for the three population segments, to be used in determining whether potential intervention cost that would be justifiable on a pure cost basis. The discount rates that would equate the costs were 4.7 percent to equate the cost of the healthy and obese, and 5.7 percent to equate the cost of the healthy and smoker.

- According to the CDC, there was direct health care cost of $75 billion for those overweight and obese in 2003. It reported that annual health care costs had more than tripled over the previous two decades.

As Ken Thorpe has said, "These (obese) are very expensive patients."

Rates of disability, particularly those of the elderly, have generally improved over time. Nevertheless, there is a risk that this trend may turn around. According to several reports in Madrian et al. (2007), aged 51-56 baby boomers in the Health and Retirement study in 2004 were not in better health than those a decade earlier, with a

higher proportion reporting being in poorer health and having more difficulty in performing daily tasks.

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

According to the NHIS of 1984 through 2000 as reported by Lakdawalla et al. (2004), the rate of the more severe personal care-limitations had increased by 50 percent. Lakdawalla reasoned that the deterioration in health could be due to: (1) the tendency of the obese to have more disabilities, together with the significant increase in the percent obese; (2) lifesaving medical techniques may result in a higher percentage disabled who might otherwise have died; and (3) the less than average wage growth for less-skilled workers that results in a greater incentive to claim for disability insurance coverage. According to the NHIS, obesity accounts for about one-half of the increased rates of disability among those aged 18-29, one-quarter for those aged 30-39 and one-tenth for those aged 40-49. The two most important causes of disability among the non-elderly are musculoskeletal problems and mental illness, and together with the small but growing contribution of diabetes, suggest the increasing contribution of obesity to the disability rolls.

Among the obese aged 60 and older, as reported in Alley et al. (2007), between the period of NHANES III and NHANES 1999-2004, the prevalence of functional impairments increased 5.4 percent (from 36.8 percent to 42.2 percent),

although ADL impairments did not change. In NHANES III the odds ratio for obese compared to normal weight individuals was 1.78, but that increased to 2.75 in NHANES 1999-2004. With respect to ADL impairments, the odds ratio between obese and normal increased from 1.31 to 2.05 between these two surveys because although the rate of impairment did not change for those obese, they decreased by 34 percent for the non-obese. Alley concluded that over the 10-year period between the two surveys, (1) the obese were more likely to report functional impairment and (2) reductions in ADL impairment for non-obese older individuals did not occur in those who were obese.

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

In a 1990 U.K. study, obesity was a strong predictor of early work-related disability. The relative risks for this disability were 200 percent for women and 150 percent for men, respectively, primarily related to the effects of cardiovascular and musculoskeletal disease. Overall, females have higher relative morbidity when compared with that of men, in contrast to the opposite relativities for mortality.

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

6.8 Quality of Life

Less quantifiable is the effect that overweight, obesity and weight gain has on a person's quality of life. It has both psychological, social and physical aspects, including:

1. Negative personal perception and self-image regarding weight and health, particularly important for young adult and adolescent females, both of whom are currently more socio-psychologically burdened because of the general societal ideal of thinness.
2. Physical functioning, by restricted mobility and various activities of daily living.
3. Role limitations, including performance at work and leisure activities.
4. Pain.
5. Actual and perceived lack of energy and fatigue.
6. Social functioning and relative lack of friends, in part leading to obesity stigma that can result in even more eating.
7. Stress from being hounded to lose weight, making many miserable.
8. Social, academic and job (wage) discrimination, possibly being characterized as lazy, ugly or stupid. Wada (2007), using NHANES III for responders from the National Longitudinal Survey of Youth 1979, found that increased body fat was related to lower wages and that fat-free body mass was consistently associated with higher wages. Both of these measures are derived from bioelectrical impedance analysis. Wada indicated that this relationship contrasts with several earlier studies relating wages and BMI. This wage-penalty has been observed by others as well.
9. Mental health, including suicide, depression, nervousness and role limitations due to emotional problems.

To varying degrees, these quality factors can affect all segments of the population.

7. Prevention and Management of Obesity

The epidemic of obesity continues unabated as every couple of years seems to be accompanied with another increase in the number of Americans estimated to be

obese and overweight. Authoritative pronouncements, official public policy goals and specific guidelines and rules to increase physical exercise and improve nutritional content come and go with only a few media headlines to accompany them. They are generally ignored in daily personal decision-making and are subsequently forgotten, just like annual New Years' resolutions. It is difficult for people to change and make the “right” decisions on a daily basis.

How many people increased the amount of time they devote to physical activity when authoritative recommendations were announced to increase physical activity to an hour a day every day? In part, this is due to the belief of a large majority of Americans that their current body weight isn't a serious health concern to them or their parents in the case of children, as confirmed by public survey results. In contrast, the 2006 Pew Research Center Survey indicated that 42 percent (51 percent of women and 32 percent of men) worry about their weight. But it is very difficult to provide sufficient motivation by making people feel sufficiently unhappy to do something about it, as it can prove to be a self-fulfilling prophesy..

Overall, attempts to decrease the level of obesity in society that rely on individual behavioral change have been pretty much ineffective. In part this is due to the value placed on the immediate gratification provided by food and the inconvenience of physical activity relative to the longer-term positive effects of better health. It is quite difficult to motivate those overweight by making them feel bad enough about their body to take immediate action. This can be expressed in terms of a large discount rate or insufficient value attributable to long-term health in these individual decision-making processes or simply to a lack of self-control. In any event, this type of behavior change is quite difficult to carry out.

For such behavioral change to be effective, potentially different action needs to be taken with respect to the key audiences, including: (1) at-risk individuals or groups of individuals, (2) individuals or groups of individuals not yet at-risk, (3) national, community and employer pressure points, and (4) the media. Although a different level or type of attention may be appropriate to those who are at-risk and those currently considered to be “normal”, the underlying message is the same—only the urgency to action might differ. More attention should be given to the underlying

causes and related behaviors that develop obesity and weight gain, particularly the amount and mix of food eaten and physical activity, rather than the weight itself. An example of the type of action needed is that in any individualized anti-smoking program some attention should also be included on how to prevent obese-inviting incentives at the same time.

Ultimately, it is the individual who is responsible for his or her decisions. Nevertheless, there are many stakeholders and external players who have or could have a role to play in this process. Each person has his or her own set of motivations and incentives that might be exploited to promote healthy behaviors, but in most cases, without sufficient will power and control over cognitive and sensory factors, personal goals may not be able to be achieved. Possibly in the long-term future, an approach that might be taken is to use genetic information to identify specific interventions that may be successful on a personal level.

In some respects, obesity is similar to an epidemic. But as it has become more common to be overweight or obese, it is becoming more socially acceptable. As a result, according to Christakis and Fowler (2007) based on the Framingham Heart Study, if a friend becomes obese, an individual has a 57 percent increased chance of becoming obese; if an adult sibling becomes obese, it increases the chance by 40 percent (for a spouse this is 37 percent) that the individual will become obese as well. This growing acceptance and spread will make reducing its incidence that much more difficult to prevent and control.

According to Bloom (2007), the following are the four general medical approaches to reducing adiposity tissue, including some examples:

- Reduce caloric input (eat less, low-calorie food, drugs to impair absorption, environmental change)
- Reduce appetite (behavior training, drugs and environmental change)
- Increase energy expenditure (exercise)
- Remove fat or impair its metabolism (bariatric surgery, liposuction).

There does not appear to exist a single method or set of motivations to prevent the onset of obesity of becoming overweight. Similarly, there is no single best

approach to take to manage obesity, overweight and weight gain once the weight has been added. Also, due to the number of contributing factors and the uniqueness of each individual, relying on only one approach will usually be unsuccessful. A program is more likely to be successful if it emphasizes a slow and staged approach using multiple methods, rather than a short-term single directional roadmap to skinny nirvana.

As was pointed out in several places above, we saw that for some conditions and population segments, visceral adipose tissue (indicated by body circumference or abdominal measures) may be at least as predictive of excess mortality as overall obesity measured by BMI. It has been noted that, although in some cases more active and toxic, abdominal fat may respond well to diet and physical exercise, possibly better than other types or locations of body fat.

Nevertheless, similar methods can be effective in the prevention and management of obesity, however the adipose tissue is manifested. The following starts with a discussion of the big two approaches (diet and exercise), but it then describes other specific approaches, as well as potential overall strategies and stakeholders that may be usefully involved in the prevention and control processes.

- **Diet**. There has been a wide variety of diets and weight reduction techniques. Although many consist simply of reducing caloric intake, others involve the use of a biased mix of specific nutrition, such as being low in fat or carbohydrates. But diets themselves rarely “cure” obesity, can be quite costly, and can sometimes be dangerous in and of themselves, with the latest magical cure rarely magical and rarely a cure.

  Huge expectations are raised by many weight loss programs, as a skinny nirvana is usually promised, sometimes in a very short timeframe. When they only succeed in improving health a little bit, enthusiasm is lost and long-term sustainable goals are not reached.

  In many animal tests, simply eating less makes animals live longer by lowering the animal's metabolic rate. Although the obvious response to

these somewhat startling results would be to replicate this success in humans, that is, prevent obesity by restricting caloric intake—in today's world, that is not so easy to do. When weight loss reaches a plateau, as it must when neurochemical compensatory mechanisms come into play, many dieters become frustrated, particularly if they have not lost much weight, at which time they conclude that the diet has failed as they didn't reach their goal, which leads to discontinuance of the diet, and the common relapse or regain of the weight temporarily lost.

Even those who do lose 5 to 10 percent of their body weight can usually only keep that amount off for, maybe, two years, with earlier studies indicating that 90 to 95 percent of all patients who underwent weight reduction treatment regain their lost weight within seven years, with some estimates being only 2 percent to 5 percent of dieters actually lose weight and keep it off. It may be more effective therefore to set relatively modest and achievable short-term goals to reinforce the resolve to continue the effort over a longer time period. Nevertheless, according to several studies, it only takes weight loss in the range of 5-10 percent to prevent or delay the development of diabetes among high-risk adults.

In 1990, the NHIS found that 53 percent of overweight adults were trying to lose weight. Based on the 1996 BRFSS, Serdula et al. (1999) found that about 29 percent of all men and 44 percent of all women are attempting to lose weight at any given time, while about 20 percent were both eating fewer calories and exercising more than 150 minutes per week.

Interestingly, among women, about 29 percent of those at normal BMIs (between 18.5 and 24.9 BMI) are attempting to lose weight as well. The odds of trying to lose weight, compared to doing nothing about one's weight, increases with education and are about 40 percent lower among current smokers and 30 percent higher among former smokers when compared with those who have never smoked. Males and females seem to pursue weight control by similar methods.

Some buy low-calorie or special food/drinks or even pseudo-starve themselves, binge and purge (although dangerous anorexia and bulimia may result), drink diet sodas rather than fully sugared ones, join weight-loss clubs, visit fat-farms, take diet pills, have liposuction or bariatric surgery, or exercise obsessively. In 1998, a study by Cleland indicated that Americans spent $33 billion annually for weight loss products and services. But the level of overweight and obesity might be even worse than it now is without any of these massive efforts. Often when there is an overemphasis on cutting back of consumption of one type of food, those on a diet make up for it by overdoing another type of food. Simple techniques, like eating a sufficient amount of fruits and vegetables and avoiding an excess amount of red meat might be just as if not more successful.

The size of the weight loss and diet control markets are very large. Marketdata Enterprises, Inc., a market research publisher, has estimated that revenues of the U.S. weight loss industry were about $55 billion in 2006, projected to increase to $68.7 billion in 2010. John LaRosa, its research director, indicated that there are about “72 million dieters in the United States—about 70 percent of whom try to lose weight by themselves, are fickle and shift from fad to fad diet." Major components of this market include $19 billion of sales of diet soft drinks that have a 29.5 percent share of the soft drink market; $4.4 billion for bariatric surgeries, although more obese Americans are now having these procedures done in Latin America where the price is substantially less; $3.4 billion for the top six market leader weight control firms, including Weight Watchers with $1.2 billion and $0.6 billion for NutriSystem; $0.8 billion for diet food home delivery; and $0.5 billion for prescription drugs, not including diet books/videos, health clubs, and weight loss camps. This is an interesting growth industry, with most of its customers fail.

Making healthy eating more socially acceptable and tasty would help. Although women eating salads, fruits or fish for dinner have long been almost a badge of honor in some communities, it has yet to achieve the same status for many men, whether at home or a fast food or sit-down

restaurant. In some cases, it just takes one member of a couple to make such food consumption a regular habit. Conversely, a reduction of the social acceptability of over-eating or eating too much unhealthy food would also help, although in some population segments currently hoping for success in this area may be somewhat naïve.

In part to avoid regulatory oversight, some restaurants have voluntarily eliminated their use of trans-fats. Other related voluntary approaches are needed, such as simply including on the menu a healthier food and drink mix, including vegetables, fruits, grains and cereals, including dietary fiber.

A simple conclusion is that instead of concentrating on a single factor, i.e., a particular diet program, a well rounded approach to nutrition may be the most effective and likely to succeed approach to weight-related health issues. Overemphasis on short-term weight loss may in fact lead to unhealthy weight recycling or food disorders. Although weight or BMI may be a useful surrogate and metric from which to measure success, all three legs are needed with a long-term commitment: diet, physical activity and proper nutrition.

- **Physical activity**. A regular, moderate and sustainable physical activity program can help both weight management and improved overall health and fitness. An increase in physical activity, although not usually as effective by itself as decreased caloric intake, can be very helpful to prevent weight regain, particularly when combined with proper nutrition. Taking personal responsibility for maintaining such a program is required, whether it means giving up a couple of hours of sleep, climbing stairs even when an elevator is available or doing some home chores. Such a course of action may even reduce overall stress at the same time.

  In fact, some have taken up exercise and fitness with some fanaticism, resulting in an expansion of the supply of health clubs, fitness centers and personal trainers. Readily available and reasonably inexpensive home exercise equipment could also be used effectively, but in many cases the

users of such services and equipment are those who are already fit. The number of members at those clubs who show up are limited to the few regulars except during the first week in January; the home equipment sometimes help spiders grow their webs due to their lack of use.

Regular moderate exercise at all ages that keep arteries elastic is what is needed, a marathon/body building level is not. Nevertheless, voluntary action may not be sufficient—this is the reason that, if it can be afforded, a regular date with a personal trainer may be useful. The Cooper Center has conducted several studies indicating that aerobic fitness at all ages can be a significant factor to enhance longevity. In fact, some studies suggest that it may be better to be fat and fit, rather than lean and out-of-shape.

If society's priorities put obesity management higher than all other objectives, we might consider eliminating some of the technological conveniences our society has adapted, e.g., eliminate elevators and escalators so people will have to walk up stairs and eliminate microwaves. Although it is certainly impossible to turn back the clock in this way, it may be worth the effort to facilitate access in everyday living to the use of physical activity instead or as a supplement to the convenient way.

Some believe that, even though regular moderate exercise contributes only mildly to weight loss, it is important for weight maintenance to avoid the vicious cycle of inactivity, where inactivity contributes to obesity, obesity exacerbates disability, which in turn impedes exercise. Others believe that, given the lack of success of so many diets, an increased level of physical fitness through moderate physical activity may be more attainable.

Even some videogames can help, at least those that require physical activity, e.g., video dance or boxing games. For children and adolescents, sports, exercise and simply time outside running around can make a difference, requiring a mix of initiatives, ranging from structured activities to environmental modifications based on safety, accessibility and

attractiveness, recognizing that potential individual motivations differ considerably among individuals.

Although exactly the opposite of a physical activity, various studies have indicated that additional sleep will reduce the chance of becoming obese. Snell (2007) found that an extra hour of sleep reduced the risk of being overweight from 36 percent to 30 percent for young children and from 34 percent to 30 percent for older children. These percentage reductions don't look like much of a decrease, but given the other benefits of additional sleep, this may be worthwhile anyway.

- **Bariatric surgery**. The National Institutes of Health (1998) has recommended bariatric surgery for those with a BMI of 40 or more or BMI 35 or higher with co-morbid conditions. Such a procedure involves reducing the size of the stomach (gastric banding) or bypassing part of the intestines (gastric bypass). But even though this may not be recommended except in extreme cases, the number of such procedures has increased significantly over the last 10 years, from about 10,000 to 100,000 between 1996-98 and 2002-04. They cost upwards of $25,000 per operation.

  Two large scale studies (Sweden and the United States of 4,000 and 8,000 lives, respectively) with a follow-up period of 18 and seven years, respectively, found improved overall mortality of between 20 percent and 56 percent for heart disease, 92 percent for diabetes, and 60 percent for cancer. In addition, the weight taken off generally stayed off when compared with a control group which experienced minor weight reductions during the study period. This surgery is increasingly seen as a treatment for diabetics as well.

  Dixon et al. (2008) reported on an Australian randomized trial of 60 obese diabetic patients (between 30 and 40 BMI) who had laparoscopic adjustable gastric banding plus conventional medical/behavioral therapy compared with a control group that only had conventional therapy. The group who had surgery had a 73 percent two-year remission rate, while the

control group had a 13 percent remission rate, with a 20.7 percent and 1.7 percent weight loss, respectively, after two years for the two groups. There were no serious complications in either group.

Other studies have indicated that there is a reported mortality rate of about 1-2 percent in the year following the procedure, with about 10 percent of hospitalizations having a complication during the hospital stay, with one-fifth being subsequently hospitalized in the following year. This type of surgery can decrease resting oxygen consumption and cardiac output that is proportional to the amount of weight loss. Heart stroke volume can fall in parallel to the decrease in blood volume and heart volume.

Although not as severe a medical procedure as bariatric surgery, liposuction has been frequently attempted to remove body fat, by vacuuming fat from under the skin. In 1998 there were 400,000 liposuction procedures in the United States. Results have been mixed, with some indication that it does not do too much good, as the fat removed is from the surface, rather than the “worst” fat that is abdominal and inherent in many internal organs that this procedure does not affect.

- **Education**. Although articles in the consumer media have often highlighted the obesity epidemic, relatively limited conveniently accessible and useful information is available for consumers at the time of relevant personal decision-making. This includes easily accessible calorie information in restaurants, although a federal judge in New York recently struck down such an attempt through a government requirement, although on a technicality, or at supermarkets. Hopefully this will include more creative approaches to provide relevant and timely information to individuals regarding the health effects of being overweight and obese.

  Social marketing, with a focus on voluntary change, is a well-used technique. Unfortunately, limited funds have been provided for this type of effort. It can be important to have respected spokesmen speak out against society's infatuation with unhealthy habits, whether to promote exercise or

to discourage unhealthy snacks and large food portions. However, there are limits to the effectiveness of what might to some sound paternalistic and preachy, unless it is done in a creative manner.

It has to be remembered in any educational program that communication has to be tailored to the audience. For example, educational efforts geared at promoting cessation of cigarette smoking in the United States did not sufficiently benefit those of limited education and lower incomes, as evidenced by their relatively high current level of smoking. In developing an effective educational program aimed at promoting other healthy behaviors, these differences should be taken into account.

Pricing effects have often been found to be more effective than educational interventions, particularly because they are more often in clear view at the time of individual decision-making. Hopefully, during this time of food price inflation, retail food price relativities will become more favorable toward healthy food, although this result is doubtful at this time. Bottom line, it is often how the currently available information can be better used than to add more such information.

Can physical fitness and healthy food be sold like commodities? More physical activity and healthy foods can be a tough sell. A related question that needs to be answered is: who will pay? Although the knee-jerk response is to get government involved, but because there may be many powerful and persuasive stakeholders who would oppose a significant public policy or rule change, alternative approaches need to be developed. Tools include marketing, education and ultimately the law. It has to be kept in mind that although in general the media can increase awareness, it is far less effective in changing behavior.

- **Pharmacotherapy**. Products currently available can be placed into two major categories: those that act on the central nervous system to influence eating behavior and appetite and those that target the gastrointestinal system and inhibit absorption or enhance a feeling of fullness. Effective

weight-management drugs might be of help, and someday a magic bullet may be found, possibly from an idea currently under development, as basic scientific investigations have recently revealed and will certainly continue to reveal new knowledge regarding the relationships between health and obesity, nutrition and physical activity. Hopefully, as knowledge of the complex molecular, genetic and biological mechanisms of obesity expands and is applied, human behavior will not offset or more than offset these advances.

It is sometimes claimed that current drug treatments combined with lifestyle and diet therapies, can reduce weight by 10 percent. But so far their use has been plagued by high attrition rates, and many trials typically experience at least a 40 percent drop-out rate so tests can only be on those who are motivated in the first place. Often current treatments are for a limited duration, possibly 12 weeks or less, while managing one's weight is a much longer-term issue.

In one study in which participants continued with a comprehensive and rigorous program over a longer period of time, a weight-loss medication combined with group lifestyle modification (diet, exercise and behavior therapy) resulted in more weight loss than either medication or lifestyle modification alone.

More scientific progress concerning obesity and health may provide some help. For example, a British study in 2007 found that there was a 70 percent higher chance of being obese if one has a particular variant in the FTO gene. Treatment developed for this variant or even personal knowledge of its existence might provide the incentive for someone with it to live a healthier lifestyle.

In December 2007, scientists at the Albert Einstein College of Medicine of Yeshiva University indicated that they found how fat is stored in cells, through FIT1 and FIT2, genes crucial for packaging fat into lipid droplets. Dr. Silver at Einstein observed that "it should be possible to develop drugs

that can regulate their expression or activity…not only for treating the main result of excess lipid droplet accumulation, obesity, but for alleviating the serious disorders that arise from obesity including type 2 diabetes and heart disease."

However, "The medical community should be skeptical of quick pharmacologic fixes for obesity and should continue to support the view that alterations in diet and physical activity—though extremely difficult to implement and sustain—will always be central components of prevention and treatment." (Rosenbaum et al., 1997)

Because of the huge potential market, continued efforts by a large number of pharmaceutical companies will continue to develop enhanced weight-loss drugs, both in the near and longer-term future. According to Espicom Healthcare Intelligence, a drug industry market information provider, $600 million will be spent on these worldwide in 2005, with $2 billion expected in 2010. As found by Bhattacharya and Packalen (2008), medical research responds to changes in disease incidence and research opportunities and pharmaceutical innovation tends to respond to aging- and obesity-induced changes in potential market size. Their efforts in this area should be expected to continue to expand in the future.

- **Nutrition incentives**. Several approaches might be adopted to encourage a healthy diet—a carrot, stick or more information. However, other than enhanced educational information at point of purchase, successful programs will be difficult to adopt due to the stakeholders involved and potential consequential problems.

  Selective taxes for non-nutritious food and drinks are being tried, e.g., for sugar-flavored drinks (in 2007 the mayor of San Francisco proposed that retailers pay a fee for selling certain sugar-laden drinks to fund an initiative to encourage healthy eating and exercise), snack food and larger servings. Although good in theory, the so-called “twinkie-tax” can also be viewed as being regressive in nature, as it is likely to result in taxing the

poor more than the rich. The equivalent of such taxes could be implicitly included in higher prices for unhealthier foods sold in vending machines or cafeterias.

Unintended consequences or even worse substitution effects can be caused by local or isolated changes in tax and other government sponsored rules, even from what looks on the surface as a positive incentive. For example, local tax and rule enforcement may not be totally effective in solving certain behavioral issues, as found recently in a study of local smoking bans that have resulted in increased drunk-driving and in fatal accidents where smokers drove farther to locations where smoking in bars was allowed.

Subsidies to increase the incentives for distribution of healthier food or tax-deductibility of certain health club membership fees might be used. But these are often problematic as, given the choice, most people will go toward the more immediately desirable food types or avoid activities that are not pleasurable rather than doing what is “good” for them. Recognition of obesity as a disease or disability could lead to changes in the tax treatment of weight control or prevention activities and expansion in insurance coverage. It should be noted that the politics involved could become fierce and it may be difficult to craft a consensus approach.

Mandating higher quality food offered in schools and providing incentives to have more farmers' markets might assist, but supply problems and secondary consequences might limit the success of such programs. California implemented a limit on fat and sugar content and portion size of all food sold on public school campuses in mid-2007, while also requiring that half of the beverages sold to high school students be fruit-based, vegetable-based, water, milk products or electrolyte replacement drinks that contain no more than 42 grams of added sweetener per 20 ounce serving. Beginning in 2008 in U.K. schools, at least two portions of fruit and vegetables are required, deep-fried foods are restricted, healthier

ingredients will be subsidized and secondary school pupils will be offered cooking lessons.

Nevertheless, due to recent food price increases, budgetary pressure to move in the opposite direction may intensify.

Restrictions in trans-fatty acids have been discussed, although Willett et al. (2005) suggested that, although there may be 50,000 premature deaths caused by the use of trans-fatty acids, if they are phased out too quickly, restaurants will likely substitute saturated fats or other equally bad ingredients that may be just as bad. McDonald's eliminated supersized foods and drinks after very adverse publicity in the early 2000s, but it has recently introduced the 'Hugo' in some markets, a 42 ounce drink containing 410 calories.

Restrictions could also be applied in public programs, e.g., to food stamp recipients or to childhood food programs. In addition, improved access through subsidized prices of fruits and vegetables in low income areas or similar programs might be pilot-tested.

The seemingly simple approach might be adopted of making healthy food more attractive, whether in more appealing salad bars in major food stores or making this food look and smell better or less expensive.

- **External sources** that are currently and likely to become more involved in this process include:

    - Schools and communities. Much has been made of the possible contribution to lowering obesity in children and adolescents through actions taken in or for the public school system, whether through increased physical activities or the amount and type of available nutrition. For example, about 25 million students currently use the National School Lunch Program and 7 million use the National School Breakfast Program. Implementing the minimum requirements of these

programs for all school meals might help, as would better rules and enforcement of what can be offered in vending machine food and beverages. Although studies such as Forshee et al. (2005) indicated that, based on findings from the Continuing Survey of Food Intake by Individuals (1994-6, 1998), NHANES 1999-2000 and the National Family Opinion WorldGroup Share of Intake Panel, there would be no significant impact on BMI from removing regular carbonated soft drinks from schools.

Peer pressure, public acceptability, personal body image and the fashion industry are all sources of psychological inputs that are often more important than other inputs. Where practical, these need to be steered in healthy directions.

Nutrition education should continue to emphasize food mix—for example, fat comprises an average of 35 percent of total caloric intake for youths aged 2 through 19 and almost two-thirds did not eat the currently recommended daily amount of fruit and vegetables.

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

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

- The family. For children, the availability of positive parental role models and an overall healthy family lifestyle would likely promote

healthier choices. Parents have to better recognize a weight problem (improper nutrition or inadequate exercise) in their children and take a sufficiently active role in addressing it. Nevertheless, as indicated in the report card discussion above, care is needed to avoid unwanted side effects, including body hatred, inappropriate weight loss attempts, eating disorders and weight stigma while avoiding oppositional reactions that may produce actions the opposite those intended.

Exclusive breastfeeding for the first four to six months has been recommended based on several studies finding its inherent protective effects, possibly due to an enhanced learning of satiety by the infant, the composition of breast-milk and less insulin secretion post-breast-feeding. But in contrast, Lawlor and Chaturvedi (2006) indicated that "while mean BMI in later life was lower among breast-fed subjects the difference was small and likely to have been strongly influenced by publication bias and confounding factors. ... evidence to date does not support infancy as a critical period during which interventions might have long-term effects on the risk of obesity and its associated diseases."

- The workplace. Although not every place of work invites or requires physical activity, many employers, particularly the larger ones, can facilitate wellness programs and increase of awareness of good health habits, especially as they increasingly recognize that not only is it good for their employees, but it can at the same time reduce health care and disability costs for them.

- Insurance and health care services industry businesses. Taking increased action could help, e.g., by treating obesity as a chronic health disease or sponsoring wellness programs and health awareness programs, with more effective and active counseling. They can provide parents better information and understanding of the health risks and possible techniques to address them.

Pay for performance through wellness rebates or premium reductions, frequent health points, gym membership discounts or high deductible health insurance plans can be effective in some instances.

Additional ideas, none of which are original, include: enhanced counseling and more effective monitoring of patients' actions is needed, and improved training of health care professionals in best practices in the prevention and management of healthy weight and lifestyles.

- Food industry. Various elements of the food industry could develop improved product and packaging innovations that might help consumers make healthy choices. Enhancement of product development, promotion and advertising of healthy rather than unhealthy foods and snacks could prove beneficial as well. People can and do respond to simple innovations, such as smaller servings of snack food—in the week before this paper was submitted, I found that one of the most popular class of foods at my local convenience store was an area of mini-snack foods at the check-out counter. If made convenient and used as a substitute rather than a supplement for larger size snack food, some people will benefit.

  Certainly the food industry already has significant incentives to provide food that people want to buy, and certainly enough people would buy healthy foods if they tasted good, were affordable and had a good public image. It simply has to provide and promote those types of food. Healthy “junk food” and snacks might help somewhat, although marketing them may prove challenging. The food industry can either produce or prepare new or modified food products, or simply change the ingredients to make them healthier without making them less desirable to their consumers. An example is the bottled water industry, whose product has gained significant acceptance and may be healthier than what it in part has substituted for, e.g., soft drinks.

Product development might take the form of a new generation of genetically modified organisms (GMOs) that taste better and are healthier. For example, they might produce no trans-fat during the cooking process and reduce the impact on level of obesity at the same time. But of course, in some jurisdictions they will have to overcome consumer and regulatory concerns at the same time. If food better addresses consumers' needs and concerns, currently adverse opinions might change. Promotion of quality rather than quantity at an affordable price would be beneficial as well.

Fast food and full service restaurants could expand their healthier food options and provide more and transparent nutritional information. More effective marketing of smaller portion size might help. For example, at a local ice cream store, a single scoop is now often referred to as a 'kiddie' size, not particularly psychologically conducive to adult males ordering it. And of course, even stricter rules for advertising to children could be adopted.

Although the food industry has made significant amounts of relevant information available on companies' websites, it is not often used by the average consumer, and unfortunately little of that type of information is available at the time of food selection decision time. Although it does promote healthy lifestyles and healthy food choices, there is clearly a limit to the information consumers will pay attention to. Nevertheless, more creative delivery information is needed.

A radical approach would consist of lawsuits against food-related companies. However, not only would such an effort not be as likely to succeed as tobacco litigation, but the desirable result would be more effective consumer education, labeling and availability of food choices, rather than the monetary compensation that is usually the objective of such litigation.

- Leisure industry. This industry has inbred incentives to promote physical fitness and age-specific physical activities. It has to increase its reach into certain segments of the population.

- Behavior therapy, weight loss psychological reinforcement through support groups such as Weight Watchers. Recently, television shows glorifying and supporting large weight loss have emerged. Goal-setting, self-monitoring, frequent contact, feedback and continuous motivation and support are important components of any such program.

  To study the effectiveness of intensity of weight-loss interventions, Levine et al. (2007) studied three groups of healthy women between the ages of 25 and 45 who, during a three-year period, received different levels of interventions: a more active, clinic-based group that met bi-monthly and a group who received instruction through a correspondence course, both compared with a group only provided an information booklet about weight management. Neither of the groups with intervention was better at preventing weight gain than the control group; both gained at least some weight, with about 60 percent gaining at least two pounds during the period. Those on a diet prior to the commencement of the study period were less likely to be successful in controlling their weight, possibly indicating the difficulty that certain women have in controlling their weight. However, Levine indicated that based on information gained during the course of the study that for high-risk groups, intensive, structured interventions can be successful in preventing weight gain.

- Government. Although many resent a “nanny state,” various proposals have been put forth that would have government dictate action, particularly by the food industry. Advocates of increased government rules point out that government has a role when markets don't work, e.g., supply or affordability (to at least some segment of the population) of healthy food, portion serving size, sufficient information to make informed food choices, vulnerable individuals

including the poor, ill and the very young, and time-inconsistent preferences.

If obesity affected others, as in the case of second-hand smoke, its significant increase might lead to a demand by government for people to take more personal responsibility to change their lifestyle or risk paying for the results by themselves. Because this is not the case, other approaches have to be used.

For example, the European Commission in early 2008 proposed that energy, fat, saturated fat and carbohydrate contents be displayed clearly on the front of food packaging. Some, including the U.K. Food Standards Agency, instead favor a simple traffic light system (red means fat or sugar levels are high) so that consumers will have easily accessible information available at the time of food purchase decisions; its chairman, Dame Deirdre Hutton, has said that the intention of the proposal is the get food manufacturers to change their products.

Another approach is to enhance government guidelines, information and promotion policy. It has been suggested that some current U.S. promotion programs (known as “checkoff” programs, for which there are 35 now, according to Wilde, 2005) promote some of the wrong types of food, e.g., beef, pork and dairy products, and certain energy dense foods, while it would represent better public policy to be more consistent with the government's *Dietary Guidelines for Americans* that promotes healthier foods, such as fruit, vegetables, fish and whole grains.

Tax incentives, such as certain currently offered local programs that provide property or other tax relief if food establishments meet certain minimum “healthy” guidelines or national programs that provide agricultural subsidies to redistribute crops to a healthier food mix might prove effective.

In what some might view as being an extreme measure, the Japanese government in 2006 dictated that beginning in April 2008 everyone between age 40 and 74 has to have a health check, at which men with a waistline over 34 inches and women over 36 inches will be asked to see a doctor, go on a weight-loss program and start a diet. Entities that provide medical services (e.g., government for small companies, health insurance societies for large companies, mutual aid associations and municipalities for the self-employed and farmers) for the national health insurance program will begin to report on the success of this program in 2012, in terms of the number who went for a health check-up, how many actually saw a doctor and how many were judged to have gotten rid of or made progress with respect to their metabolic syndrome, although penalties for not achieving the goals established have not yet been specified. This rule was a reaction to a male population 10% and a female population 6.4% heavier than a decade earlier, with 27 million suffering from the metabolic syndrome and increasing health care costs.

- Technology. Technology should be emphasized in carrying out any public health program. It has been suggested that devices, possibly implantable, that would be less severe than bariatric surgery, but may prove more effective than diets and drugs that might become popular in the future.

- Continued research on issues related to behavioral choices, weight management and biological and incentive advancement will likely always be needed.

The key is not whether weight in the obese can be lost, but whether it can stay lost. The development of effective weight loss and management programs over the long term will remain a challenge.

It is useful to repeat the recommendations of the World Cancer Research Fund (2007) panel to individuals:

1. Be as lean as possible within the normal range of body weight.

2. Be physically active as part of everyday life.
3. Limit consumption of energy-dense foods and avoid sugary drinks.
4. Eat mostly foods of plant origin.
5. Limit intake of red meat and avoid processed meat.
6. Limit alcoholic drinks.
7. Limit consumption of salt and avoid moldy cereals (grains) or pulses (legumes).
8. Aim to meet nutritional needs through diet alone (i.e., don't rely on dietary supplements).

The goal should not be for everyone to reach the elusive American ideal of being slim and fit and remaining young forever. Nevertheless, maintaining as healthy a body as the individual's genetic situation practically allows is a worthwhile goal, although because the causes of obesity are so heterogeneous, a program to achieve a healthy body has to be tailored to the individual. It should focus on not only weight per se, but probably more importantly the contributing behaviors, primarily food input and physical activity, although BMI and other weight or related measures remain reasonable metrics by which to assess progress.

In a study of about 1,000 adults, Dunn et al. (2006) found that there was no preference for diet or physical activity change as a weight loss strategy. The two behaviors were synergistic rather than compensatory, although restricting fat intake was more effective than increasing exercise for weight loss. While fat restrictions alone contributed to weight loss for both men and women, exercise alone provided weight loss benefits for men only. The cumulative effect of weight loss behaviors varied by gender: for women, an interaction of the two techniques was observed, while in men there was no interaction. Increases in exercise helped to offset weight gain or provided small weight loss benefits at all levels of dietary fat input change. Note that that this study was conducted in a managed care environment with mostly middle class whites over a two-year period.

Since negative effects of weight-cycling can be even more harmful in certain cases than having a higher but stable amount of weight, long-term programs have to be emphasized. In a society in which food is plentiful and affordable and the need for

exercise is no longer necessary but just desirable for health, we will likely see a lot of fat people trying to get thin for a long time to come.

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

In any case, sufficient motivation or incentives are needed for any approach to work over a long period of time. With modern societal incentives and built-in mechanisms to satisfy short-term desires and preferences, it is difficult for the individual to meet healthy objectives at the same time. Too heavy a focus on weight may demonize and demoralize those who are currently obese that can create a significant psychological hurdle to good health and long life. Intervention for children and adolescents is likely to fail without active intervention and the assistance of schools and parents, but without starting young, in the long-term it will likely get worse before it gets better.

A major cultural/social shift may be needed, both on a national and local level, as opinions must be embedded more deeply, so as to influence individuals' decision-making processes through an environment that discourages overeating and encourages more physical activity. As individual efforts have not succeeded in the past, the need for population-level prevention strategies, possibly including communities, governments, the media and the food industry, as well as the individual may be needed to prevent avoidable premature deaths. Adolescents will have to become involved in helping themselves; imposed solutions will certainly not work. It is unlikely that there will be a magic bullet.

According to National Institutes of Health (1998): "Strong evidence exists that weight loss reduces blood pressure in both overweight hypertensive and non-hypertensive individuals; reduces serum triglycerides and increases high-density lipoprotein (HDL)-cholesterol; and generally produces some reduction in total serum cholesterol and low-density lipoprotein (LDL)-cholesterol . Weight loss reduces blood glucose levels in overweight and obese persons without diabetes; and weight loss also reduces blood glucose levels and HbA in some patients with type 2 diabetes."

Since it is difficult to reverse weight gain and obesity, in most cases weight management may be more important than weight reduction. In contrast with the millions who undertake diets, few want to put on weight. Elimination of fast-food and vending machine soft-drinks, although possible on school property, would not work on an overall basis. Although home health equipment has the potential to enhance physical fitness, it is not uncommon that such equipment purchased is not used. Although noble in thought, it is clear that general public health recommendations for weight reduction have not proven to be an effective approach to a solution. In addition, prevention rather than treatment may hold the highest potential in reversing the trend toward increased obesity and its unwanted costs in the United States and worldwide. The reason why so much public emphasis has been placed on preventing childhood obesity is the difficulty of curing obesity in adults and the many long-term adverse effects of childhood obesity.

8. Conclusion and Implications for Mortality Projections

The large and growing prevalence of obesity (>30 BMI for adults) is a significant and growing concern to society. Not only has average weight increased, whatever measurement basis used, but the percentage in excess of any given level has increased for all age categories, reflecting a change in the prevalence distribution itself. This trend has been shown to be not just an American phenomenon, where more than one-third of the adult population is now obese, but is one that is occurring in almost all countries, although the severely obese is where Americans stand out.

Similar increases have been experienced by all population segments, both involving those obese and overweight (>25 BMI for adults), including children and adolescents. Although the health implications of being obese, particularly those

extremely obese, are significant, studies of the mortality implications of those overweight have produced inconsistent findings. Nevertheless, whether the huge overweight segment is looked at as (1) being at-risk of becoming obese; (2) experiencing adverse mortality prospects itself; or (3) only having significant morbidity and health care risks, indicates that this segment is of societal concern as well.

When two-thirds of the adult population and a growing percentage of the pre-adult population have a certain condition, it has to warrant serious attention of anyone involved in mortality projections. Limits to growth of prevalence will certainly be reached at some point, but the concern will remain. In contrast, it is unlikely that we will see significant decreases in this population, as it is quite difficult to lose weight over a long period of time.

Although mortality experience for these population segments has been observed in numerous recent studies, relatively few have been able to provide sufficiently long-term follow-up information to provide a complete picture of their associated ultimate implications. Particularly given the rapid rise in weight reported over the last 30 years, this lack of long-term follow-up information is troubling for those involved in long-term mortality projections. Although the obesity risk may have a shorter average duration until its effects on morbidity and mortality than cigarette smoking, the lack of information results in significant uncertainty in the estimation of future patterns of mortality.

An example of the source for this concern involves the long-term effects of the rise in obesity of children and adolescents. Evidence has emerged that obese adolescents are likely to develop into obese adults that suggests that the current adult weight problem will, if anything, get worse before it gets better. The recently published large-scale Danish study that followed children and adolescents over a long time period indicates that significant premature mortality is likely, increasing with increasing BMI, with no J-curve relationship affecting both those who are overweight and obese.

Section 6 describes some of the adverse effects of greater adiposity, including diabetes, cardiovascular and related diseases and certain cancers. It was noted that since one of the significant results, most cardiovascular diseases, has experienced significant reduction in mortality over the last several decades, the effect of obesity may have been reduced. Nevertheless, the extreme optimism generated by a view of recent mortality gains may be misplaced, as at least a few early warning signals have been observed that such significant mortality improvements may not persist for long, with a reversal trend possibly the result of the increase in obesity and related behaviors and consequential disease conditions such as diabetes.

However, mitigating factors regarding future premature mortality have and will continue to affect the impact of these trends. Over the last several decades these have taken the form of effective medication to control adverse levels of blood pressure and cholesterol, as well as the favorable overall demographic effects of the reduction but not elimination of cigarette smoking as a risk factor. In the future they may take the form of less expensive and less risky bariatric surgery or medical breakthroughs such as finding a cure for diabetes or an effective "fat" pill. Section 7 describes some of the prevention and management techniques that are available to mitigate the existence and effect of obesity. Nevertheless, these are likely to remain imperfect and difficult to implement, particularly on an individual level, without an immediate crisis to enhance incentives and motivation sufficiently.

Several obesity-related factors point to an increasing level of premature mortality risks, including:

- The prevalence of diabetes continues to increase. Not only is it dangerous independent of other diseases, but it remains a major mortality risk factor for the more significant cardiovascular diseases.

- Hypertension and other health risk factors remain more prevalent among those obese.

- In view of the growing percent of adolescents who are overweight, the relatively high correlation between childhood (especially adolescent)

obesity and adult obesity indicates that the long-term additional adult mortality risks should be expected to continue to grow.

- The continued shift of the distribution of population BMI to the right.

- Given the latency period of many of the conditions affected by obesity, the recent increase in obesity prevalence suggests that we have not yet observed its full effect in reported experience. This lag, which varies by condition, is not yet fully understood.

- Several of the cancers which obesity appears to influence remain significant health risks.

- The effect on morbidity, health care costs, disability and quality life is large and unlikely to decrease, regardless of the effect of the mortality mitigating factors discussed.

The following obesity-related factors indicate that future experience may be better than otherwise indicated:

- One of the chronic diseases for which these conditions have the potential to most significantly affect is cardiovascular and related diseases. Although still the major overall cause of death, advances in medical therapies and drugs have narrowed the health gap between obese and non-obese populations. This family of diseases has shown the largest decrease over the last several decades. The difference in prevalence of high cholesterol levels between obese and those of lower BMI have been reduced.

- Evidence in several studies indicates that the effect of being overweight (as opposed to being obese) may have been exaggerated and in some cases can provide some protective health value.

The uncertainties associated with the projection of the effects of large and increasing weight include:

- Much of the experience has been gathered when the level of obesity was much less than current and projected levels. Thus, some of the observed experience may not apply to the future.

- Behavioral factors are particularly difficult to predict due to intervening and mitigating factors. Although diets have rarely worked in individual cases, the ultimate effect of the aggregate of all of the individual and societal efforts underway remain uncertain.

- It is possible that behavioral factors with a negative effect will continue to be overwhelmed by other factors, including effective medical treatments and new pharmaceutical products.

- The effect on mortality for older age (e.g., those older than 70 or 75) adults has not so far been significant, in part possibly due to confounding effects of simultaneously affecting risk factors. However, to the extent that the recent overweight trends continue, at the minimum toward current cohorts that will be approaching older ages in the future, any effect may become more apparent in the future. In addition, its effect may be more significant in the areas of functional limitations and healthy life expectancy.

A good example of a recent population mortality projection reflecting the effect of obesity and selected mitigating factors is provided in Cutler et al. (2007) in which they described a mortality projection over the next 10 years. It incorporated estimated changes in smoking, education, drinking, hypertension, cholesterol and obesity, developing two alternative sets of assumptions: (1) current levels of medication continue (NHANES 1999-2002 indicates that 60 percent of those with hypertension take anti-hypertensive medication and 35 percent of people with high cholesterol take cholesterol-lowering medication) and (2) all those with the adverse blood pressure and cholesterol levels take medication and people are 75 percent

effective with respect to this treatment (percentage diagnosed and percent taking medication as directed).

Cutler noted the favorable effect of the continuing reduction in smoking on mortality, both due to the reduction in smoking and the long lag-time between smoking cessation and improved mortality rates. Using the first set of assumptions, the effect of projected continued increases in obesity is expected to more than offset the continued favorable effect of the reductions in smoking and by itself is expected to result in a 13 percent increase in age-adjusted mortality. In contrast, the second set of assumptions in which the mitigating factors are far more effective than today, the net effect of the two offsetting factors almost totally offset each other. Cutler indicated that the magnitude of the effect of obesity was reflected through the use of the non-linear relationships between BMI and weight increase, and between BMI and health risks.

This projection example demonstrates both the magnitude of the potential effect of the growing level of obesity and the potentially offsetting effect of several key mitigating factors. This combination leads to a great deal of uncertainty associated with long-term mortality projections. Since it is unlikely that the noted mitigating factors will be as effective as is assumed in the second set of assumptions, continued increases in weight and consequential effects on mortality is certainly a possibility. However, the extent of this effect remains uncertain at this time.

The likely future mortality levels of the huge percent of the population that is now overweight and obese should not be ignored. It will remain a significant challenge to the actuarial profession in all practice areas and will benefit from the expected continued output of research results from outside the profession. It took decades of intensive government effort to gain a modest control over smoking; it will take at least as long to obtain improvements in the fight against obesity and sedentary living. Only long-term solutions will likely be effective in contributing to the solutions to the current trends.

Health care costs and rates of disability may be affected by the increasing obesity levels even more than mortality. Those involved in health-related projections should factor the effect of these trends in their projections as well.

The level of obesity by country will continue to differ, at least to the extent of cultural and eating habits, some as they move through their nutrition and sedentary transition, although there will likely be some convergence in most countries over the long term. In addition, the populations from which trends are observed and to which projections apply will differ. For example, for many life insurers whose higher income policyholders are intensely underwritten, different trends may apply. Nevertheless, the seemingly unstoppable spread of Western-style diets and convenience generating technology, unless significant food product development and activity changes occur, will ultimately lead to negative effects.

Mortality is affected both by human behavior, the human condition and the treatment of the underlying diseases and access to health care. The challenge of making future mortality projections involves quantification of the uncertainty associated with these factors.

The enhancements in available information regarding these human behaviors will continue to evolve and be enhanced. Nevertheless, as can be seen through the examination of the many studies currently available on this subject, some of which are highlighted in this paper, conflicting findings will likely continue to cloud the picture for quite some time yet. Significant research efforts are currently underway and should be encouraged, especially those involving long-term follow-up studies, as the effects of personal decisions are both short and long term in nature.

Metrics in this area need to be further refined. The BMI benchmark used to measure weight internationally for the very young and old should be reviewed. Based on available research, alternative obesity metrics, e.g., waist circumference measurement, may be superior either alone or as supplementary information in indicating mortality risks due to certain conditions or population segments. Although BMI might not indicate that obesity is a key mortality factor in some areas, e.g., for those older than age 70, weight/fat increases in certain body areas may lead to future

negative trends as well. Just because BMI does not appear to significantly affect mortality in a population sub-segment or for a condition does not mean that the combination of weight, nutrition and physical activity is not important to future mortality and morbidity in that area. In addition, more standard practical measurement benchmarks are needed to measure physical activity levels as well—supplemental measures may be needed, particularly with respect to the effective measurement of the contribution of fatness gain and physical fitness, which may be just as, if not more important, than weight gain.

As indicated by Grundy (1997), "Increased availability of food and a reduction in physical activity will combine to make obesity the number one health problem worldwide in the 21st century."

Glossary

Adipose tissue. Body fat.

Adiposity. The quality or state of being fat.

BRFSS (Behavioral Risk Factor Surveillance Survey). A large, random telephone survey conducted by the CDC, that tracks health conditions and risk behaviors.

Body mass index (BMI). A measure of an adult's weight in relation to his or her height, specifically the adult's weight in kilograms divided by the square of his or her height in meters. (See Table 1 for representative values in terms of inches and pounds.)

Cancer. A group of more than 100 diseases characterized by uncontrolled cellular growth as a result of changes in the genetic information of cells. "It is abundantly clear that the incidence of all the common cancers in humans is determined by various potentially controllable external factors." (National Research Council. *Diet, Nutrition and Cancer*. National Academy of Sciences, 1982)

Cardiovascular disease. A group of diseases that involve the heart and/or blood vessels. It is also used to refer to those related to atherosclerosis.

CDC (Centers for Disease Control and Prevention). It is the public health agency of the U.S. government whose objective is to achieve better health for Americans. It is responsible for gathering health-related information to further that objective.

Chronic disease. A disease that develops or persists over a long period of time, does not resolve spontaneously and is rarely cured completely. It includes non-communicable diseases such as cancer, cardiovascular disease and diabetes.

Confounder. A variable, within a specific study, that is associated with an exposure, which is also a risk factor for the disease, and is not in the causal pathway from the exposure to the disease. If not adjusted for, this factor may distort the apparent exposure-disease relationship.

Diabetes mellitus (referred to as *diabetes*). An insulin resistance condition, a metabolic disorder involving impaired metabolism of glucose due either to failure of secretion of hormone insulin (type 1) or to impaired responses of tissues to insulin (type 2). Type 2 traditionally had onset during adulthood.

Food insecurity. The inability to meet basic food needs because of a lack of resources to buy food.

Hazard ratio (also referred to as *prevalence ratio*, *risk ratio* or *relative ratio*). The ratio of mortality of one population subgroup to that of another. In this paper, references to hazard ratios are the ratio of a population with a range of BMIs to a benchmark, often the most favorable, of population with a range of BMIs, such as between 18.5 and 24.9.

High fructose corn syrup. A form of corn syrup that has undergone enzymatic processing to increase its fructose content.

Hyperlipidemia. An elevated level of lipids, e.g., cholesterol and triglycerides, in the bloodstream that can speed the hardening of the arteries.

Hypertension. Having elevated blood pressure and/or taking antihypertensive medication.

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

Mitigating factor. A variable shown to prospectively reduce the probability of onset of a condition or reduce the severity of an existing condition.

NHANES (National Health and Nutrition Examination Survey). A series of U.S. population surveys conducted by the Centers for Disease Control (CDC). Their results are often used in studies of weight, as measurement is done in a mobile examination center or in a limited examination at home by health care professionals. Its results are determined on a nationally representative basis. NHANES I was conducted during 1971-1974, NHANES II during 1976-80, NHANES III during 1988-1994, and continuous updates have been conducted thereafter, so far during 1999-2000, 2001-2002, 2003-2004 and 2005-2006.

NHIS (National Health Interview Survey). A 50 year old survey that is the principal source of information on the health of the civilian non-institutionalized population of the United States, conducted by the CDC.

Nutrition transition. A period during which a traditional diet that is low in fat and high in fiber transitions to a high-energy Western-style diet that is high in fat and low in fiber.

Obesity. Having a very high amount of body fat in relation to lean body mass (for adults, often characterized in terms of BMI of 30 or more). Class 1 obese is an adult with a BMI between 30.0-34.9; class 2 with a BMI of 35.0-39.9; class 3 with a BMI of 40.0-44.9; class 4 with a BMI of 45.0-49.9; and class 5 with a BMI of 50.0 or more.

Overweight (WHO refers to this as *pre-obese*). A condition in which a person weighs more than useful for the optimal functioning of the body. Often considered to be an adult with a BMI between 25.0 and 29.9. In the United States, for children over age 2 and adolescents, those weighing more than the 95th percentile of the gender-specific 2000 CDC BMI-for-age-growth charts; "at-risk for overweight" is considered to be the 85th percentile of these charts (some commentators refer to those above the 95th and between the 85th and 95th percentiles as being obese and overweight, respectively).

Preventative factor. A mitigating factor that reduces the probability of onset of a condition.

Physical activity. Any form of movement using skeletal muscles.

Reverse causation. A situation where an abnormal level of an exposure is caused by a particular disease or its treatment, rather than or in addition to the other way around. For example, if an undiagnosed case of cancer causes weight loss, then the finding that low BMI is associated with increased risk associated with the cancer rather than reflecting that low weight causes cancer.

Risk factor. A variable shown to prospectively predict onset of an adverse outcome among individuals who are otherwise free of the condition.

Sedentary. A lifestyle involving limited noticeable effort, with heart and breathing rates not raised perceptibly above resting levels.

Social marketing. The application of commercial marketing techniques to the analysis, planning and execution of programs designed to influence voluntary behavior to improve personal welfare and that of society.

Socioeconomic factors. These are a bundle of characteristics that represent an individual's relative standing in society, such as income, wealth and education.

Visceral adipose tissue. Internal body fat, specifically that within the chest or abdomen.

Weight cycling (also referred to as *weight fluctuation* or *yo-yoing*). Repeated weight loss followed by weight regain.

Weight stigma. Negative attitudes that can affect interpersonal interactions and activities in a detrimental manner.

Western-style diet. A mixture of food that is relatively high in fat and low in fiber and has a relatively high percentage of high-energy foods.

WHO. The World Health Organization.

Appendix—Obesity Measurement

The objective underlying the study of human weight usually focuses on the measurement of excess body fat, mainly composed of adipose tissues, the main stores of which are subcutaneous and intra-abdominal, although particularly in older adults it can also reside in muscles. However, it is not easy to measure body fat on a direct basis.

The best approach to its measurement is often considered to be weighed underwater, based on the principle that fat tissue is less dense than muscle and bone. However, the use of this approach is limited to labs with specialized equipment. An alternative approach recently used is dual-energy x-ray absorptiometry, due to its greater precision and simplicity. However, neither of these two approaches is available for routine measurement.

In most studies, body fat is measured by a combination of weight and height, due to their high degree of positive correlation. The advantage of this approach is that their measurement is usually reasonably accurate, even if self-reported, and easy to determine. Its primary limitation is that it does not distinguish between fat mass from lean mass or muscle.

The measurement approaches to overweight and obesity usually used are described below.

Body Mass Index (BMI)

The BMI, sometimes referred to as the Quetelet index, named for its 19th century Belgian originator, is a measure of an adult's weight in relation to his or her height, specifically the adult's weight in kilograms divided by the square of his or her height in meters. (See Table 1 for representative values in terms of inches and pounds. It is also equal to weight measured in pounds times 704.5, divided by the square of the subject's height in inches)

The BMI has been shown to be strongly correlated (r has been estimated to be about 0.9 for men and women) with fat mass for middle-aged adults, although it

appears to be a less valid measure of body fatness in older adults (many of whom have lost muscle mass, with resultant normal BMI corresponding to reduced nutritional reserves), children, certain ethnic groups, those with certain disabilities and extreme athletes. It also is an indirect or surrogate measure of fatness, as it does not distinguish between type and percent of muscle, nor does it focus on the location of fat, a deficiency as it has been shown that visceral fat deposition is more of a risk than were it elsewhere. It also does not distinguish between body fat and fat-free body mass, but can provide a measure of total body weight that is an independent risk factor for several adverse health conditions.

BMI standards for adults were established by the World Health Organization (WHO) in 1997 and published by WHO (2000). It has attempted to establish similar internationally accepted standards for children and adolescents, probably varying by age on standard age curves, although it is recognized that there are international differences in personal development, e.g., puberty. It would be worthwhile to revisit whether current BMI levels are appropriate for older ages.

The Childhood Obesity Working Group of the International Obesity Task Force proposed a set of standards for children in 2000, consisting of an average of the median growth curves for six countries (Brazil, Hong Kong, the Netherlands, Singapore, the United Kingdom and the United States) that vary by age, fitted to the currently used cutoffs for overweight and obesity for young adults. However, the recommendations (Cole et al., 2000), which appear less arbitrary and more international than previous measures, have not yet become generally accepted. Obtaining agreement regarding such a common standard has been difficult.

An adult is considered obese if he or she has a very high amount of body fat in relation to lean body mass (often measured in terms of BMI of 30 or more). Class 1 obesity has been referred to as an adult with a BMI between 30.0-34.9; class 2 with a BMI of 35.0-39.9; class 3 with a BMI of 40.0-44.9, class 4 with a BMI of 45.0-49.9 and class 5 with a BMI of 50.0 or more.

An adult is considered to be overweight with a BMI between 25.0 and 29.9. Overweight children and adolescents in the United States are considered to be those

weighing greater than the 95th percentile of the 2000 CDC gender-specific BMI-for-age-growth charts, while those between the 85th and 95th percentile are considered to be at-risk of becoming overweight. The 97.7th percentile of a reference population of British children has sometimes been used to define obesity in the United Kingdom.

A summary of the currently accepted categories of BMIs is given in Table 21.

Table 21
Weight categories

BMI Category	BMI Range
Grade 3 Underweight	Less than 16.0
Grade 2 Underweight	16.0 - 16.9
Grade 1 Underweight	17.0 - 18.4
Normal	18.5-24.9
Overweight	25.0 - 29.9
Class 1 Obese	30.0 - 34.9
Class 2 Obese	35.0 - 39.9
Class 3 Obese	40.0 - 44.9
Class 4 Obese	45.0 - 49.9
Class 4 Obese	50.0 and higher

These ranges of BMI are not perfect proxies for being obese or overweight. However, both cutoff points are commonly used and according to Beich et al. (2007), there are high correlations between obesity prevalence and BMI (e.g., the United States (0.99), the United Kingdom (0.95) and Japan (0.93)). In any event, the distribution of BMI, and certainly each of the tails of its distribution are important in the study of obesity.

The BMI z score is a statistic sometimes used in the study of children's obesity, often based in the United States from the CDC 2000 Growth Chart, at say, the 95th percentile confidence interval to combine BMIS of different ages and genders. It is equal to the difference between the mean value of the BMI of a population less an individual's BMI, divided by the standard deviation of the BMI distribution of that population.

Differences in degree of body fat composition and BMI exist between population segments. For example, it has been shown in several studies that Caucasians and Asians with the same body fat composition differ by two to three BMI units. A WHO Expert Consultation in Singapore in 2005 recommended that, based on local studies, the BMI cutoff standards should differ from international standards by a similar amount, i.e., overweight would be considered as those with a BMI of 23.0-27.4, while obese would be considered to be taken as being 27.5+. In addition, there is a wide range of body weight, nutritional habits and physical activity within demographic sub-categories of "Asians," just as there is a wide variation within the Hispanic population. For example, within the United States, Vietnamese tend to be far less active than other ethnic groups; 10 percent of Vietnamese and Japanese adults could be classified as being underweight, a higher percent of Filipino adults are obese, but even they on average have a far lower level of obesity than other American ethnic groups. On average, Cuban-Americans have lower BMIs than Mexican- or Puerto Rican-Americans.

Other Measures

Various other measures have been used, including:

- Body circumference, usually measured at the waist or hip. This has been shown to be positively correlated with abdominal fat content. It has in some cases been used as an independent risk factor for certain chronic diseases and morbidity. Abdominal fat is usually associated with excess fat and can be more relevant than BMI, especially for older aged individuals. Abdominal fat contrasts with so-called "peripheral" fat, which is not around the trunk and is not as often linked to bad health results. This measure is somewhat more subjective, as its measurement can depend on the measurer. Waist measurements of more than 40 inches for men and 35 inches for women are commonly thought as being associated with increased mortality risk.

- Ratio of waist circumference to hip circumference. Normally this is usually less than 0.8, with ratios of 0.9 for women and 1.0 for men sometimes considered to represent a higher mortality risk.

- Sagittal abdominal diameter (SAD or supine abdominal height). This is a measure of visceral adiposity, or the size of the belly, measured from the back to the upper abdomen. It has been found in some studies to be a useful metric to measure a particular location of fat tissues.

- Skinfold thickness. Usually this provides a reasonable assessment of body fat, especially if taken on multiple sites, such as the triceps, and can provide information on the location of the fat. The accuracy and consistency of measurement depends upon the skill of the examiner.

- Bioelectrical impedance analysis. This is based on the measurement of the resistance to a weak electric current applied across a person's extremities that reflects the total amount of water in the body. Its use is often combined with height and weight in an empirically derived formula. It is relatively simple to determine, although the subject should have fasted for the prior four hours. It has not been shown to measure fat or to predict biologic outcomes more accurately than the use of weight and height alone, although it may also be able to reflect insulin resistance. In addition, it may not be useful for severely obese individuals. However, Wada (2007) has developed body composition measures of body fat and fat-free mass that measure components of fat.

- Fasting levels of insulin and triglycerides and levels of high-density lipoprotein cholesterol.

Each of these measures is imperfect in some respects. Most researchers today use BMI, the simplest, easiest and possibly most objective approach, sometimes supplemented by other measurements, such as waist circumference.

Various studies have indicated that alternative measures can provide better indication of mortality risk than BMI. For example, Dagenais et al. (2005), based on the results of the Heart Outcomes Prevention Evaluation of 8,802 adults with a mean age of 66 and a follow-up period of 4.5 years, suggested that indices of abdominal adiposity, such as waist-to-hip ratio (WHR) and waist circumference (WC) predict coronary heart disease (CHD) and stroke better than BMI. Silventoinen et al. (2003) indicated that in a study of 11,510 Finnish adults aged 25 to 64 with a 5-11 year follow-up period abdominal fat had an effect on coronary heart disease independent of BMI; and that all three obesity indicators explained some part of the variation of the CHD independently of the other two indicators, with WHR being the best indicator.

Although standard cut-offs for other measures have not been universally agreed upon, a small-scale study by Colombo et al. (2008) of 63 Italian adults (age 20-65) indicated that "the use of BMI alone, as opposed to an assessment on body composition, to identify individuals needing lifestyle intervention may lead to unfortunate results." This study compared bioimpedance analysis, dual-energy X-ray absorptiometry, and WC, as alternatives to BMI to measure abdominal fat, metabolic syndrome risk and percentage body fat. Based on the cutoff values used (e.g., 88 cm for women and 102 cm for men for WC), BMI obese values identified a lower percentage of subjects for whom treatment would be recommended, with the largest triggers relating to the metabolic syndrome risk. Colombo concluded that although BMI may be useful on a group basis, it was not necessarily reliable for individual clinical assessments, and that it may be appropriate to either replace or supplement it with other measure(s).

The Melbourne Collaborative Cohort Study evaluated the effectiveness of most of the above measures as predictive metrics. It found that measures of central adiposity, such as waist and hip circumferences, were better predictors of mortality than overall adiposity using such measures as BMI. However, at least so far, BMI has proved easier to apply and obtain self-reports on. In the larger and more important studies, multiple measures should be used where practical.

Surveys and studies can differ in the approach taken to measure height and weight. Overweight self-reporters tend to underestimate their weight and to

overestimate their height. Women tend to self-report their weight too low and males tend to self-report their height too high. In either case, this tends to underestimate their BMIs. For example, the prevalence estimates of obesity from NHANES exceed those from the BRFSS and NHIS by a significant amount, the former being in-person measured (during 1999-2002 of 30.4 percent), while the latter two surveys were self-reported by telephone (BRFSS in 2001 of 20.0 percent and NHIS of 22.5 percent). Chou et al. (2004) indicated that adjusting for underreporting weight, using procedures developed by Cawley, resulted in a 27 percent increase in estimated percent obese.

References

Adams, K.F., Schatzkin, A., Harris, T.B., Kipnis, V., Mouw, T., Ballard-Barbash, R. Hollenbeck, A., and Leitzmann, M.F. 2006. "Overweight, Obesity and Mortality in a Large Prospective Cohort of Persons 50 to 71 Years Old." The New England Journal of Medicine 355: 763-778.

Agras, W., and Mascola, A. 2005. "Risk Factors for Childhood Overweight." Current Opinions in Pediatrics 17: 648-652.

Albert Einstein College of Medicine of Yeshiva University. 2007. "Fundamental Discovery by Einstein Researchers Reveals How Fat is Stored in Cells." News Release.

Alley, D.E., and Chang, V.W. 2007. "The Changing Relationship of Obesity and Disability, 1988-2004." Journal of the American Medical Association 298: 2020-2027.

Allison, D.B., Fontaine, K.R., Manson, J.E., Stevens, J., and VanItallie, T.B. 1999. "Annual Deaths Attributable to Obesity in the United States." *JAMA* 282: 1530-1538.

Allison, D.B., Zannolli, R., Venkat Narayan, K.M. 1999. "The Direct Health Care Costs of Obesity in the United States." American Journal of Public Health 89(8): August.

American Obesity Association. 2007. *AOA fact sheets*. www.obesity.org.

Anderson, L.H., Martinson, B.C., Crain, A.L. Pronk, N.P., Whitebird, R.R., Fine, L.J., and O'Connor, P.J. 2005. "Health Care Charges Associated with Physical Inactivity, Overweight, and Obesity." Preventing Chronic Disease [serial online] October.

Anderson, P.M., Butcher, K.F., Schanzenbach, D.W. 2007. "Childhood Disadvantage and Obesity: Is Nurture Trumping Nature?" NBER Working Paper No. 13479. National Bureau of Economic Research, Cambridge, MA.

Anderson, R.E., Crespo, C.J., Bartlett, S.J., Cheskin, L.J., and Pratt, M. 1998. "Relationship of Physical Activity and Television Watching with Body Weight and Level of Fatness among Children." *JAMA* 279: 938-942.

Anonymous. 1999. "Achievements in Public Health: Tobacco Use—United States, 1900-1999. Morbidity and Mortality Weekly Report 48:986-993.

Australian Institute of Health and Welfare, National Heart Foundation of Australia 2004. "The Relationship between Overweight, Obesity and Cardiovascular Disease." AIHW Cat. No. CVD 29. Australia Institute of Health and Welfare, Canberra.

Bhattacharya, J., Packalen, M. (2008). "Is Medicine an Ivory Tower? Induced Innovation, Technological Opportunity, and For-Profit vs. Non-Profit Innovation." NBER Working Paper No. 13862. National Bureau of Economic Research, Cambridge, MA.

Baker, J.L., Olson, L.W., and Sorensen, T.I.A. 2007. “Childhood Body-Mass Index and the Risk of Coronary Disease in Adulthood.” The New England Journal of Medicine 357: 2329-2337.

Baum, C.L. II, and Ruhm, C.J. 2007. “Age, Socioeconomic Status and Obesity Growth.” NBER Working Paper No. 13289. National Bureau of Economic Research, Cambridge, MA.

Bibbins-Domingo, K., Coxson, P., Pletcher, M.J., Lightwood, J., and Goldman, L. 2007. “Adolescent Overweight and Future Adult Coronary Heart Disease.” The New England Journal of Medicine 357: 2371-2379.

Biddle, S., Marshall, S., Gorely, T., Cameron, N., Murdey, I., Mundy, C., Vince, A., Whitehead, S. 2005. “Sedentary Behaviour among Scottish Youth: Prevalence and Determinants.” NHS Health Scotland: Edinburgh.

Blair, S.N., and LaMonte, M.J. 2005. “Commentary: Current Perspectives on Obesity and Health: Black and White, or Shades of Grey?” International Journal of Epidemiology. Doi: 10.1098/ije/dyi259. 1-3.

Bleich, S., Cutler, D.M., Murray, C., and Adams, A. 2007. “Why is the Developed World Obese?” NBER Working Paper No. 12954. National Bureau of Economic Research, Cambridge, MA.

Bloom, S. 2007. “Hormonal Regulation of Appetite.” Obesity Reviews 8(Suppl. 1): 63-65.

Bogers, R.P., Bemelmans, J.E., Hoogenveen, R.T., Boshuizen, H.C., Woodward, M., Kneckt, P., van Dam, R.M., Hu, F.B., Visscher, T.L.S., Menotti, A., Thorpe Jr., R.J., Jamrozik, K., Calling, S., Strand, B.H., Shipley, M.J. 2007. "Association of Overweight With Increased Risk of Coronary Heart Disease Partly Independent of Blood Pressure and Cholesterol Levels." Archives of Internal Medicine 167:16: 1720-1728.

Bouchard C., Pérusse, L., Rice, T., and Rao, D.C. 2003. “Genetics of Human Obesity.” In: G.A. Bray and C. Bouchard. *Handbook of Obesity*. New York: Marcel Dekker.

Brackenridge, R.D.C., and Elder, W.J. 1998. *Medical Selection of Life Risks*. New York: Stockton Press.

Bray, G.A., Nielsen, S.J., and Popkin, B.M. 2004. “Consumption of High-Fructose Corn Syrup in Beverages May Play a Role in the Epidemic of Obesity.” American Journal of Clinical Nutrition 79: 537-543.

Brown, C.D, Higgins, M., Donato, K.A., Rohde, F.C., Garrison, R., Obarzanek, E., Ernst, N.D., and Horan, M. 2000. “Body Mass Index and the Prevalence of Hypertension and Dyslipidemia.” Obesity Research 8(9): 605-619.

Brown, W.J., Williams, L., Ford, J.H., Ball, K., and Dobson, A.J. 2005. Identifying the Energy Gap: Magnitude and Determinants of 5-year Weight Gain in Midage Women." Obesity Research 13(8): August.

Burkhauser, R.V., and Cawley, J. 2004. "Obesity, Disability, and Movement onto the Disability Insurance Rolls." University of Michigan Retirement Research Center Working Paper, WP 2004-089.

Calle, E.E., Thun, M.J., Petrelli, J.M., Rodriguez, C., and Heath, Jr., C.W. 1999. "Body-Mass Index and Mortality in a Prospective Cohort of U.S. Adults." The New England Journal of Medicine 341: 1097-1105.

Calle, E.E., Rodriguez, C., Walker-Thurmond, K., and Thun, M.J. 2003. "Overweight, Obesity, and Mortality from Cancer in a Prospectively Studied Cohort of U.S. Adults." The New England Journal of Medicine 348: 1625-1638.

Case, A., and Menendez, A. 2007. "Sex Differences in Obesity Rates in Poor Countries: Evidence from South Africa." NBER Working Paper No. 13541. National Bureau of Economic Research, Cambridge, MA.

Cawley, J. 2004. "The Impacts of Obesity on Wages." Journal of Human Resources 39: 451-474.

Cawley, J., and Burkhauser, R. 2006. "Beyond BMI: The Value of More Accurate Measures of Fatness and Obesity in Social Science Research." NBER Working Paper No. 12291. National Bureau of Economic Research, Cambridge, MA.

Cawley, J., and Liu, F. 2007. "Maternal Employment and Childhood Obesity: A Search for Mechanisms in Time Use Data." NBER Working Paper No. 13600. National Bureau of Economic Research, Cambridge, MA.

Chang, V.W. 2005. "U.S. Obesity, Weight Gain, and Socioeconomic Status." Center for Health Equity Research and Promotion vIII(I): Fall: 1-4.

Chou, S.-Y., Grossman, M., and Saffer, H. 2004. "An Economic Analysis of Adult Obesity: Results from the Behavioral Risk Factor Surveillance System." Journal of Health Economics 23: 565-587.

Christakis, N.A., and Fowler, J.H. 2007. "The Spread of Obesity in a Large Social Network over 32 Years." New England Journal of Medicine 357: 370-379.

Chu, S.Y., Bachman, D.J., Callaghan, W.M., Whitlock, E.P., Dietz, P.M., Berg, C.J., O'Keeffe-Rosetti, M., Bruce, F.C., Hornbrook, M.C. 2008. "Association between Obesity during Pegnancy and Incrased Use of Health Care." New England Journal of Medicine 358: 1444-1453.

Colditz, G.A. 1992. "Economic Costs of Obesity." American Journal of Clinical Nutrition 55: 503S-507S.

Cole, T.J., Bellizzi, M.C., Flegal, K.M., and Dietz, W.H. 2000. BMJ 320: 1240, 1-6.

Cole, T.J., Flegal, K.M., Nicholls, D., and Jackson, A.A. 2007. “Body Mass Index Cutoffs to Define Thinness in Children and Adolescents: International Survey.” BMJ 335: 194-197.

Comptroller and Auditor General. 2001. “Tackling Obesity in England, Report by the Comptroller and Auditor General.” London: National Audit Office.

Cohen, D.A., and Sturm, R. 2008. “Body Mass Index is Increasing Faster Among Taller Persons.” American Journal of Clinical Nutrition 87(2): 445-448.

Colombo, O., Villani, S., Pinelli, G., Trentani, C., Baldi, M., Tomarchio, O., and Tagliabue, A. 2008. “To Treat or not to Treat: Comparison of Different Criteria Used to Determine Whether Weight Loss is to be Recommended.” Nutrition Journal 7:5.

Cope, M.B., and Allison, D.B. 2006. “Obesity: Person and Population.” Obesity 14(Suppl.) July: 156S-159S.

Cossrow, N., and Falkner, B. 2008. “Race/Ethnic Issues in Obesity and Obesity-Related Comorbidities.” The Journal of Clinical Endocrinology & Metabolism 89(6): 2590-2594.

Costa-Font, J., and Gil, J. 2005. “Obesity and the Incidence of Chronic Diseases: A Seemingly Unrelated Probit Approach.” Working Papers on Economics 137. Universitat de Barcelona, Espai de Recerca en Economia.

Crawford, P.B., Townsend, M.S., Metz, D.L., Smith, D., Espinosa-Hall, G., Donohue, S.S., Olivares, A., and Kaiser, L.L. 2004. “How Can Californians be Overweight and Hungry?” California Agriculture 58(1): 12-17.

Cutler, D.M., Deaton, A.S., and Lleras-Muney, A. 2006. “The Determinants of Mortality.” NBER Working Paper No. 11963. National Bureau of Economic Research, Cambridge, MA.

Cutler, D. M., Glaeser, E. L., and Rosen, A.B. 2007. “Is the U.S. Population Behaving Healthier?” NBER Working Paper No. 13013. National Bureau of Economic Research, Cambridge, MA.

Cutler, D.M., Glaeser, E.L., and Shapiro, J.M. 2003. “Why Have Americans Become More Obese?” Journal of Economic Perspectives 17: 93-118.

Dagenais, G.R., Yi, Q., Mann, J.F.E., Bosch, J., Pogue, J., and Yusuf, S. 2005. “Prognostic Impact of Body Weight and Abdominal Obesity in Women and Men with Cardiovascular Disease.” American Heart Journal. January. 149(1): 54-60.

Daniels, S.R. 2006. “The Consequences of Childhood Overweight and Obesity.” Future of Children 16(1): 47-67.

Daviglus, M.L., Liu, K., Yan, L.L., Pirzada, A., Manheim, L., Manning, W., Garside, D.B., Schiffer, L., Dyer, A.R., Grennland, P., and Stamler, J. 2004. “Body Mass Index in Middle Age and Health-Related Quality of Life in Older Age.” Archives of Internal Medicine 2003 163: 2448-2455.

Daviglus, M.L., Liu, K., Yan, L.L., Pirzada, A., Manheim, L., Manning, W., Garside, D.B., Wang, R., Dyer, A.R., Grennland, P., and Stamler, J. 2004. “Relation of Body Mass Index in Young Adulthood and Middle Age to Medicare Expenditures in Older Age.” JAMA 292: 2743-2749.

Dehghan, M., Akhtar-Danesh, N., and Merchant, A.T. 2005. “Childhood Obesity, Prevalence and Prevention.” Nutrition Journal 4(24): 1-8.

Dixon, J.B, O'Brience, P.E., Playfair, J., Chapman, L., Schachter, L.M., Skinner, S., Proietto, J., Bailey, M., and Anderson, M. 2008. “Adjustable Gastric Banding and Conventional Therapy for Type 2 Diabetes.” JAMA 299(3): 316-323.

Dyer, A.R., Stamler, J., Garside, D.B., and Greenland, P. 2004. “Long-Term Consequences of Body Mass Index for Cardiovascular Mortality: The Chicago Heart Association Detection Project in Industry Study.” Annals of Epidemiology 14: 101-108.

Dunn, C.L., Hannan, P.J., Jeffrey, R.W., Sherwood, N.E., Pronk, N.P., Boyle, R. 2006. “The Comparative and Cumulative Effects of a Dietary Restriction and Exercise on Weight Loss.” International Journal of Obesity 30: 112-121.

Economist. 2007. “Briefing: Food Prices, Cheap No More.” Economist: Dec. 8-14; 81-83.

_____. 2008. "The New Face of Hunger." Economist: April 19-25; 32-34.

Elo, I.T., and Preston, S.H. 1996. “Educational Differentials in Mortality: United States, 1979-85. Social Science and Medicine 42: 47-57.

Eng, E. 2004. *Too Big to Ignore: The Impact of Obesity on Mortality Trends.* Swiss Reinsurance Company. Zurich.

Eschtrut, A.D. 2005. “Projecting Mortality: Key Findings from a Forum of Experts.” Issue Brief, Center for Retirement Research at Boston College.

Ezzati, M., Oza, S., Danaei, G., and Murray, C.J. 2008. “Trends and Cardiovascular Mortality Effects of State-Level Blood Pressure and Uncontrolled Hypertension in the United States.” Circulation 117: 905-914.

Field, A.E., Austin, S.B., Gillman, M.W., Rosner, B., Rockett, H.R., Colditz, G.A. 2004. “Snack Food Intake Does Not Predict Weight Change among Children and Adolescents.” International Journal of Obesity 28: 1210-1216.

Finkelstein, E.A., Fiebelkorn, I.C., and Wang, G. 2003. “National Medical Spending Attributable to Overweight and Obesity: How Much, and Who's Paying?” Health Affairs W3-219-226.

Flegal, K.M. 2006. "Commentary: The Epidemic of Obesity--What's in a Name?" International Journal of Epidemiology. 35: 72-74.

Flegal, K.M. 2007. “The Effects of Changes in Smoking Prevalence on Obesity Prevalence in the United States.” American Journal of Public Health 1510-1514.

Flegal, K.M., Carroll, M.D., Ogden, C.L., and Johnson, C. L. 2002. “Prevalence and Trends in Obesity among U.S. Adults, 1999-2000.” JAMA 288: 1723-7.

Flegal, K.M., Graubard, B.I., and Williamson, D.F. 2004. “Methods of Calculating Deaths Attributable to Obesity. 2004. American Journal of Epidemiology 160: 331-338.

Flegal, K.M., Graubard, B.I., Williamson, D.F., and Gail, M.H. 2005. “Excess Deaths Associated with Underweight, Overweight, and Obesity.” JAMA 293: 1861-1867.

Flegal, K.M., Graubard, B.I., Williamson, D.F., Gail, M.H. 2007. “Cause-Specific Excess Deaths Associated with Underweight, Overweight, and Obesity.” JAMA 298(17): 2028-2037.

Flegal, K.M., Graubard, B.I., Williamson, D.F., and Gail, M.H. 2007. “Weight-Associated Deaths in the United States, Letter to the Editor.” Journal of Women's Health 16 (Nov. 9): 1368-1370.

Flegal, K.M., Ogden, C.L., Carroll, M.D. 2004. “Prevalence and Trends in Overweight in Mexican-American Adults and Children.” Nutritional Review 62(7, pt 2): S144-148.

Flegal, K.M., Troiano, R.P., Pamuk, E.R., Kuczmarski, R.J., and Campbell, S.M. 1995. “The Influence of Smoking Cessation on the Prevalence of Overweight in the United States.” The New England Journal of Medicine 333: 1165-1170.

Folsom, A.R., Kushi, L.H., Anderson, K.E., Mink, P.J., Olson, J.E., Hong, C.-P., Sellers, T.A., Lazovich, D., and Prineas, R.J. 2000. Associations of General and Abdominal Obesity with Multiple Health Outcomes in Older Women, the Iowa Women's Health Study.” Archives of Internal Medicine 160 (July 24): 2117-2128.

Folsom, A.R., Stevens, J., Schreiner, P.J., and McGovern, P.G. 1998. “Body Mass Index, Waist/Hip Ratio, and Coronary Heart Disease Incidence in African Americans and Whites.” American Journal of Epidemiology 148(12): 1187-1194.

Fontaine, K.R., Redden, D.T., Wang, C., Westfall, A.O., and Allison, D.B. 2003. Years of Life Lost Due to Obesity.” JAMA 289: 187-193.

Food Research and Action Center. 2004. *Proceedings of the Roundtable on Understanding the Paradox of Hunger and Obesity*. Nov. 22, 2004.

Ford, E.S., Ajani, U.A., Croft, J.B., Critchley, J.A., Labarthe, D.R., Kottke, T.E., Giles, W.H., and Capewell, S. 2007. “Explaining the Decrease in U.S. Deaths from Coronary Disease, 1980-2000.” The New England Journal of Medicine 356: 2388-2398.

Ford, E.S., Capewell, S. 2007. "Coronary Heart Disease Mortality Among Young Adults in the U.S. from 1980 through 2002: Concealed Leveling of Mortality Rates." Journal of American College of Cardiology. 50: 2128-2132.

Forshee, R.A., Storey, M.L., Allison, D.B., Glinsmann, W.H., Hein, G.L., Lineback, D.R., Miller, S.A., Nicklas, T.A., Weaver, G.A., and White, J.S. 2007. A Critical Examination of the Evidence Relating High Fructose Corn Syrup and Weight Gain." Critical Reviews in Food Science and Nutrition 47: 561-582.

Forshee, R.A., Storey, M.L., and Ginevan, M.E. 2005. "A Risk Analysis Model of the Relationship between Beverage Consumption from School Vending Machines and Risk of Adolescent Overweight." Risk Analysis 25(5): 1121-1135, October.

Fox, C.S., Coady, S., Sorlie, P.D., D'Agostino, R.B., Pencina, M.J., Vasan, R.S., Meigs, J.B., Levy, D., and Savage, P.J.. 2007. "Increasing Cardiovascular Disease Burden Due to Diabetes Mellitus—The Framingham Heart Study." Circulation 115(12): 1544-1550.

Frayling, T.M., et al. 2007. "A Common Variant in the FTO Gene is Associated with Body Mass Index and Predisposes to Childhood and Adult Obesity." Science 316: 889-894.

Freedman, D.S., Khan, L.K., Serdula, M.K., Galuska, D.A., and Dietz, W.H. 2002. "Trends and Correlates of Class 3 Obesity in the United States from 1990 through 2000." JAMA 288: 1758-1761.

Gaesser, G.A. 2004. "Weight Loss for the Obese: Panacea or Pound-Foolish?" Quest 56: 12-27.

Goel, M.S., McCarty, E.P., Phillips, R.S., and Wee, C.C. 2004. "Obesity among U.S. Immigrant Subgroups by Duration of Residence." JAMA 292(23): 2860-2867.

Gregg, E.W., Gu, Q., Cheng, Y.J. Venkat Narayan, K.M., and Cowie, C.C. 2007. "Mortality Trends in Men and Women, 1971-2000." Annals of Internal Medicine 1-11.

Gregg E.W., Yiling, J., Cheng, B.L., Cadwell, L., et al. 2005. "Secular Trends in Cardiovascular Disease Risk Factors According to Body Mass Index in U.S. Adults." JAMA 293: 1868-74.

Gronniger, J.T. 2005. "Familial Obesity as a Proxy for Omitted Variables in the Obesity-Mortality Relationship." Demography 42: 719-735.

Gruber, J., and Frakes, M. 2006. "Does Falling Smoking Lead to Rising Obesity?" Journal of Health Economics 25: 183-197.

Grundy, S.M. 1997. "Primary Prevention of Coronary Heart Disease: Role of Cholesterol Control in the United States." Journal of Internal Medicine 241: 295-306.

Gunderson, C., Lohman, B.J., Eisenmann, J.C., Garasky, S., and Stewart, S.D. 2008. "Child-specific Food Insecurity and Overweight Are Not Associated in a Sample of 10- to 15-year-old Low-income Youth." Journal of Nutrition 138: 371-378.

Gutterman, S., and Vanderhoof, I. 1998. "Forecasting Changes in Mortality: A Search for a Law of Causes and Effects." North American Actuarial Journal 135-138.

Hancox, R.J., and Poulton, R. 2006. "Watching Television is Associated with Childhood Obesity: But is it Clinically Important?" International Journal of Obesity 30: 171-175.

Harnack, L.J., Jeffery, R.W., and Boutelle, K.N. 2000. "Temporal Trends in Energy Intake in the United States: An Ecologic Perspective." American Journal of Clinical Nutrition 71: 1478-84.

Hart, C.L., Hole, D.J., Lawlor, D.A., and Smith, G.D. 2006. "Obesity and Use of Acute Hospital Services in Participants of the Renfrew/Paisley Study." Journal of Public Health 10.1093, 1-4.

Harvey, D., Hook, E., Kozyniak, J., and Selvanathan, M. 2002. "Building the Case for the Prevention of Chronic Disease." *Alliance for the Prevention of Chronic Disease*. DIR-31.

Hedley, A.A., Ogden, C.L., Johnson, C.L., Carroll, M.D., Curtin, L.R., and Flegal, K.M. 2004. "Prevalence of Overweight and Obesity among U.S. Children, Adolescents, and Adults, 1999-2002." JAMA 291: 2847-2850.

Heiat, A., Vaccarino, V., and Krumholz, H.M. 2001. "An Evidence-Based Assessment of Federal Guidelines for Overweight and Obesity as They Apply to Elderly Persons." Archives of Internal Medicine 161: 1194-1203.

Helmchen, L.A., and Henderson, R.M. "Changes in the Distribution of Body Mass Index of White U.S. Men, 1890-2000." Annals of Human Biology Mar-Apr; 31(2): 174-181.

Henderson, R.M. 2005. "The Bigger the Healthier: Are the Limits of BMI Risk Changing Over Time?" *Economics & Human Biology* 3(3) (December): 339-366.

Henig, R.M. 2006. "Fat Factors." New York Times Magazine. Aug. 13, 26-57.

Hernandez, D.J., and Macartney, S.E. 2008. "Racial-ethnic Inequality in Child Well-being from 1985-2004: Gaps Narrowing, but Persist." FCD Policy Brief, Child Well-Being Index, No. 9.

Higgins, M., D'Agostino, R., Kannel, W., and Cobb, J. 1993. "Benefits and Adverse Effects of Weight Loss: Observations from the Framingham Study." Annals of Internal Medicine 199 7(2): 758-63.

Hoerger, T.J. 2006. "Controversies in Obesity Mortality: A Tale of Two Studies." *RTI-UNC Center of Excellence in Health Promotion Economics*, v1, 1. 1-4.

Hossain, P., Kawar, B., and El Nahas, M. 2007. “Obesity and Diabetes in the Developing World—A Growing Challenge.” The New England Journal of Medicine 356: 213-215.

Hu, F.B., Manson, J.E., Stampfer, M.J., Colditz, G., Liu, S., Solomon, C.G., and Willett, W.C. 2001. “Diet, Lifestyle, and the Risk of Type 2 Diabetes Mellitus in Women.” The New England Journal of Medicine 345: 790-797.

Hu, F.B., Willett, W.C., Li, T., Stampfer, M.J., Colditz, G.A., and Manson, J.E. 2004. “Adiposity as Compared with Physical Activity in Predicting Mortality among Women.” New England Journal of Medicine 351: 2694-2703.

Hu, G., Lakka, T.A., Lakka, H-M., and Tuomilehto, J. 2006. “Lifestyle Management in the Metabolic Syndrome.” Liebertonline 4: 270-286.

Hu, G., Tuomilehton, J., Silventoinen, K., Barengo, N.C., Peltonen, M., and Jousilahti, P. 2005. “The Effects of Physical Activity and Body Mass Index on Cardiovascular, Cancer and All-cause Mortality among 47212 Middle-Aged Finnish Men and Women.” International Journal of Obesity 29: 894-902.

Inserm Collective Expert Review Center. 2007. “Obesity, Assessment and the Evaluation of Obesity Prevention and Management Programs.” French National Institute of Health and Medical Research.

International Obesity TaskForce and European Association for the Study of Obesity. 2002. “Obesity in Europe. The Case for Action.” London: International Obesity TaskForce and European Association for the Study of Obesity.

Janssen, I. 2006. “Morbidity and Mortality Risk Associated with an Overweight BMI in Older Men and Women.” Obesity 15(7): 1827-1840.

Janssen, I., and Jolliffe, C.J. 2006. “Influence of Physical Activity on Mortality in Elderly with Coronary Artery Disease.” Medicine and Science in Sports and Exercise 38(3): 418-423.

Janssen, I., Mark A.E. 2007. “Elevated Body Mass Index and Mortality Risk in the Elderly.” Obesity Reviews 8(1): 41-59.

Jee, S.H., Sull, J.W., Park, J., Lee, S-Y., Ohrr, H., Guallar, E., and Samet, J.M. 2006. “Body-Mass Index and Mortality in Korean Men and Women.” The New England Journal of Medicine 355: 779-387.

Jenkins, K.R. 2004. “Obesity's Effects on the Onset of Functional Impairment Among Older Adults." *The Gerontologist*. 44:206-216.

Jeffrey, R.W., and Harnack, L.J. 2007. “Evidence Implicating Eating as a Primary Driver for the Obesity Epidemic.” Diabetes 56: 2673-2676.

Jeffery, R.W., and Utter, J. 2003. “The Changing Environment and Population Obesity in the United States.” Obesity Research 11(Suppl.), October: 12S-22S.

Jousilahti, P., Tuomilehto, J., Vartiainen, E., Pekkanen, J., and Puska, P. 1996. “Body Weight, Cardiovascular Risk Factors, and Coronary Mortality.” Circulation 93: 1372-1379.

Kaestner, R., and Grossman, M. 2008. “Effects of Weight on Children's Educational Achievement.” NBER Working Paper No. 13764. National Bureau of Economic Research, Cambridge, MA.

Katzmarzyk, P.T., Church, T.S., Janssen, I., Ross, R., and Blair, S.N. 2005. “Metabolic Syndrome, Obesity, and Mortality: Impact of Cardiorespiratory Fitness.” Diabetes Care 28: 391-397.

Kenschaiah, S., Evans, J. C., Levy, D., Wilson, P.W.F., Benjamin, E. J., Larson, M.G., Kannel, W.B., and Vasan, R.S. 2002. “Obesity and the Risk of Heart Failure.” The New England Journal of Medicine 347: 305-313.

Klarenbach, S., Raj, P., Anderson, C., and Philip, J. 2006. “Population-based Analysis of Obesity and Workforce Participation.” Obesity 14(5): 920-927.

Komlos, J., Breitfelder, A., Sunder, M. 2008. "The Transition to Post-industrial BMI Values Among U.S. Children." *NBER Working Paper No. 13898.* National Bureau of Economic Research, Cambridge, MA.

Koplan, J.P., Liverman, C.T., and Kraak, V.A. (eds.) 2005. *Preventing Childhood Obesity: Health in the Balance*. National Academy of Sciences. Committee on Prevention of Obesity in Childhood and Youth.

Krueger, P.M., Rogers, R.G., Hummer, R.A., Boardman, J.D. 2004. "Body Mass, Smoking, and Overall and Cause-Specific Mortality Among Older U.S. Adults." Research on Aging, v 26(1), 82-107.

Kuchler, F., and Golan, E. 2004. “Is There a Role for Government in Reducing the Prevalence of Overweight and Obesity?” Choice 2004-3.

Kuczmarski, R.J., Flegal, K.M., Campbell, S.M., and Johnson, C.L. 1994. “Increasing Prevalence of Overweight among U.S. Adults.” JAMA 272: 205-211.

Kumanyika, S. 2005. “Obesity, Health Disparities, and Prevention Paradigms: Hard Questions and Hard Choices.” *Preventing Chronic Diseases.* 2005 serial online www.cdc.gov.

Lackdawala, D.N., Bhattacharya, J., and Goldman, D.P. 2004. “Are the Young Becoming More Disabled?” Health Affairs, January/February, 23(1): 168-176.

Lackdawala, D.N., Goldman, D.P., and Shang, B. 2007. “The Health and Cost Consequences of Obesity among the Future Elderly.” Health Affairs. Sept. 27, 2005. w5.r30.

Lackdawala, D.N., and Philipson, T. 2002. “The Growth of Obesity and Technological Change: A Theoretical and Empirical Examination.” NBER Working Paper No. 8946. National Bureau of Economic Research, Cambridge, MA.

Lawlor, D.A., Chaturvedi, N. 2006. "Treatment and Prevention of Obesity--Are There Critical Periods for Intervention?" International Journal of Epidemiology 35: 3-9.

Lawlor, D.A., Hart, C.L., Hole, D.J., and Davey Smith, G. 2006. "Reverse Causality and Confounding and the Association of Overweight and Obesity with Mortality." Obesity 14: 2294-2304.

Lawlor, D.A., Leon, D.L. 2005. "Association of Body Mass Index and Obesity Measured in Early Childhood with Risk of Coronary Heart Disease and Stroke: in Middle Age: Findings of the Aberdeen Children of the 1950s Prospective Cohort Study." Circulation. 111: 1891-96.

Levine, M.D., Klem, M.L., Kalarchian, M.A., Wing, R.R., Weissfeld, L., Qin, L., and Marcus, M.D. 2007. “Weight Gain Prevention among Women.” Obesity 15(5): May 1267-1277.

Lew, A.M., and Gajewski, J. (eds.) 1990. *Medical Risks: Trends in Mortality by Age and Time Elapsed*. New York, Westport, London: Praeger.

Liu, J., Wade, T.J., and Tan, H. 2007. “Cardiovascular Risk Factors and Anthropometric Measurements of Adolescent Body Composition: A Cross-sectional Analysis of the Third National Health and Nutrition Examination Survey." International Journal of Obesity 31: 59-64.

Loureiro, M.L. 2004. “Obesity: Economic Dimensions of a "Super Size." Choices. Agricultural Economics Association. Third quarter.

Loureiro, M.L, and Nayga, Jr., R.M. 2005. “Obesity Rates in OECD Countries: An International Perspective.” Presentation at EAAE XI Congress-Copenhagen.

Lumeng, J.C., Somashekar, D., Appugiese, D., Kaciroti, N., Corwyn, R.F., and Bradley, R.H. 2007. “Shorter Sleep Duration is Associated with Increased Risk of Being Overweight at Ages 9 to 12.” Pediatrics 120(5), Nov. 1020-1029.

Ludwig, D.S., Pereira, M.A., Kroenke, C.H., Hilner, J.E., Van Horn, L., Slattery, M.L., and Jacobs, Jr., D.R. 1999. “Dietary Fiber, Weight Gain, and Cardiovascular Disease Risk Factors in Young Adults.” JAMA 282: 1539-1546.

Lutsey, P.L., Steffen, L.M., and Stevens, J. 2008. “Dietary Intake and the Development of the Metabolic Syndrome, the Atherosclerosis Risk in Communities Study.” Circulation 117: 754-761.

Madrian, B., Mitchell, O.S., and Soldo, B. (eds.) 2007. *Redefining Retirement: How Will Boomers Fare?* New York: Oxford University Press.

Manini, T.M., Everhart, J.E., Patel, K.V., Schoeller, D.A., Colbert, L.H., Visser, M., Tylavsky, F., Bauer, D.C., Goodpaster, B.H., and Harris, T.B. 2006. “Daily Activity Energy Expenditure and Mortality among Older Adults.” JAMA 296: 171-179.

Manson, J.E., Bassuk, S.S., Hu, F.B., Stampfer, M.J., Colditz, G.A., and Willett, W.C. 2007. “Estimating the Number of Deaths Due to Obesity: Can the Divergent Findings be Reconciled?” Journal of Women's Health 16: 168-176.

Manson, J.E., Willett, W.C., Stempfer, M.J., Colditz, G.A., Hunter, D.J., Hankinson, S.E., Hennekens, C.H., Speizer, F.E. 1995. “Body Weight and Mortality among Women.” The New England Journal of Medicine 333: 677-685.

Margarey, A.M., Daniels, L.A., Boulton, T.J.C. 2001. “Prevalence of Overweight and Obesity in Australian Children and Adolescents: Reassessment of 1985 and 1995 Data against New Standard International Definitions.” Medical Journal Australia 561-565.

McDaid, J., and Brown, R.L. 2003. “Factors Affecting Retirement Mortality.” North American Actuarial Journal 7(2): 24-43.

McTigue, K., Larson, J.C., Valoski, A., Burke, G., Kotchen, J., Lewis, C.E., Stefanick, M.L., Van Horn, L., Kuller, L. 2006. "Mortality and Cardiac and Vascular Outcomes in Extremely Obese Women." JAMA 296: 79-86.

Metropolitan Life Insurance Company. 1983. “1983 Metropolitan Height and Weight Tables for Men and Women.” Statistical Bulletin: Jan.-June: 2-9.

Michaud, P.-C., van Soest, A., and Andreyeva, T. 2007. “Cross-Country Variation in Obesity Patterns among Older Americans and Europeans.” Social and Economic Dimensions of an Aging Populations (SEDAP) Research Paper No. 185.

Miljkovic, D. 2006. “Obesity: Health and Food Policy Dilemma.” Choices. 1st Quarter 2006, 21(1): 37-40.

Mokdad, A.H., Bowman, B.A., Ford, E.S., Vinicor, F., Marks, J.S., and Koplan, J.P. 2001. “The Continuing Epidemics of Obesity and Diabetes in the United States.” JAMA 286: 1195-1200.

Mokdad, A.H., Ford, E.S., Bowman, B.A., Dietz, W.H., Vinicor, F., Bales, V.S., and Marks, J.S. 2003. “Prevalence of Obesity, Diabetes, and Obesity-related Health Risk Factors." JAMA 289: 76-79.

Mokdad, A.H., Serdula, M.K., Dietz, W.H., Bowman, B.A., Marks, J.S., and Koplan, J.P. 1999. “The Spread of the Obesity Epidemic in the United States, 1991-1998.” JAMA 282: 1519-1522.

Monheit, A.C., Rogowski, J.A., and Vistnes, J.P. 2007. “Overweight in Adolescents: Implications for Health Expenditures.” NBER Working Paper No. 13488. National Bureau of Economic Research, Cambridge, MA.

MsJAMA. 2002. "Societal Change to Prevent Obesity." JAMA 288: 2175-2181.

Must, A., Spadano, J., Coakley, E.H., Field, A.E., Colditz, G., and Dietz, W.H. 1999. “The Disease Burden Associated with Overweight and Obesity.” JAMA 282: 1523-1529.

Narkiewicz, K. 2006. “Obesity and Hypertension—The Issue is More Complex than We Thought.” Nephrology Dialysis Transplant 21: 264-267.

Nemetz, P.N., Roger, V.L., Ransom, J.E., Bailey, K.R., Edwards, W.D., and Leibson, C.L. 2008. “Recent Trends in the Prevalence of Coronary Disease, A Population-based Autopsy of Nonnatural Deaths.” Archives of Internal Medicine 168(3), Feb. 11: 264-270.

Obesity Reviews. 2007. Foresight Programme of the Office of Science and Innovation, Department of Trade and Industry. 8 (Suppl. 1), 1-205.

Ogden, C.L., Carroll, M.D., McDowell, M.A., and Flegal, K.M. 2007. “Obesity among Adults in the United States—No Change since 2003-2004.” NCHS Data Brief. November 2007: 1.

National Institutes of Health. 1998. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults—The Evidence Report. Obesity Research 6 (Suppl. 2): 51S-209S.

Nielsen, S.J., and Popkin, B.M. 2003. “Patterns and Trends in Food Portion Sizes, 1977-1998.” JAMA 289: 450-453.

An Epidemic of Obesity Myths Web site (2007). as of June 4, 2007.

Ogden, C.L., Carroll, M.D., Curtin, L.R., McDowell, M.A., Tabak, C.J., and Flegal, K.M. 2006. “Prevalence of Overweight and Obesity in the United States, 1999-2004.” JAMA 295: 1549-1555.

Ogden, C.L., Flegal, K.M., Carroll, M.D., and Johnson, C.L. 2002. “Prevalence and Trends in Overweight among U.S. Children and Adolescents, 1999-2000.” JAMA 288: 1728-1732.

Olshansky, S.J., Passaro, D.J., Hershow, R.C., Layden, J., Carnes, B.A., Brody, J., Hayflick, L., Butler, R.N., Allison, D.B., and Ludwig, D.S. 2005. “A Potential Decline in Life Expectancy in the United States in the 21st Century.” New England Journal of Medicine 352: 1138-45.

Olshansky, S.J., and Persky, V. 2008. “The Canary in the Coal Mine of Coronary Artery Disease.” Archives of Internal Medicine 168(3), Feb. 11: 261.

Paffenbarger, R.S., Hyde, R.T., Wing, A.L., Lee, I-M., Jung, D.L., Kampert, J.B. 1993. “The Association of Changes in Physical Activity Level and Other Lifestyle Characteristics with Morality among Men.” New England Journal of Medicine 328: 538-545.

Patterson, R.E., Frank, L.L., Kristal, A.R., and White, E. 2004. “A Comprehensive Examination of Health Conditions Associated with Obesity in Older Adults.” American Journal of Preventive Medicine 27(5), Dec.: 385-390.

Philipson, T.J., and Posner, R.A. 1999. “The Long-run Growth in Obesity as a Function of Technological Change. NBER Working Paper No. 7423. National Bureau of Economic Research, Cambridge, MA.

Poirier, P., Giles, T.D., Bray, G.A., Hong, Y., Stern, J.S., Pi-Sunyer, F.X., and Eckel, R.H. 2006. “Obesity and Cardiovascular Disease: Pathophysiology, Evaluation, and Effect of Weight Loss.” Circulation 113: 898-918.

Polednak, A.P. 1990. “Mortality from Diabetes Mellitus, Ischemic Heart Disease and Cerebrovascular Disease among Blacks in a Higher Income Area.” Public Health Reports, July-August, 105(4): 393-399.

Preston, S.H. 2005. “Deadweight? The Influence of Obesity on Longevity.” New England Journal of Medicine 352: 1135-7.

Preston, S.H., and Wang, H. 2006. “Sex Mortality Differences in the United States: The Role of Cohort Smoking Patterns.” Demography 43(4) 631-646.

Quessenberry, Jr., C., Caan, B., and Jacobson, A. 1998. “Obesity, Health Services Use, and Health Care Costs among Members of a Health Maintenance Organization.” Archives of Internal Medicine 158(5): 466-472.

Racette, S.B., Deusinger, S.S., Deusinger, R.H. 2003. “Obesity: Overview of Prevalence, Etiology, and Treatment.” Physical Therapy 83(3), March.

RAND. 2007. “Obesity and Disability, the Shape of Things to Come.” RAND Health Research Highlights 1-4.

Rashad, I. 2006. “Structural Estimation of Caloric Intake, Exercise, Smoking and Obesity.” NBER Working Paper No. 11957. National Bureau of Economic Research, Cambridge, MA.

Rashad, I, and Grossman, M. 2004. “The Economics of Obesity.” The Public Interest. Summer, 156: 104-112.

Reynolds, S.L., and Himes, C.L. 2007. “Cohort Differences in Adult Obesity in the United States: 1982--2002.” Journal of Aging and Health 19(5): 831-850.

Rimm, E.B., Stampfer, M.J., Giovannucci, E., Ascherio, A., Spiegelman, D., Colditz, G.A., and Willett, W.C. 1995. “Body Size and Fat Distribution as Predictors of Coronary Heart Disease among Middle-Aged and Older U.S. Men.” American Journal of Epidemiology 141(12): 1117-1127.

Robinson, T.N. 1999. “Reducing Children's Television Viewing to Prevent Obesity, a Randomized Controlled Trial." JAMA 282: 1561-1567.

Rogers, R.G., Hummer, R.A., Nam, C.B. 1999. *Living and Dying in the USA: Behavioral, Health, and Social Differences of Adult Mortality.* Academic Press.

Rosenbaum, M., Leibel, R.L., and Hirsch, J. 1997. “Obesity.” New England Journal of Medicine 337: 396-407.

Roudebush, B., Ashley, T., and Titcomb, Jr., C.P. 2006. “Mortality and Morbidity Liaison Committee—ISCS Body Mass Index Study.” Journal of Insurance Medicine 38: 167-180.

Roux, L., and Donaldson, C. 2004. “Economics and Obesity: Costing the Problem or Evaluating Solutions?” Obesity Research 12(2), February: 173-179.

Rozin, P., Kabnick, K., Pete, E., Fischler, C., and Shields, C. 2003. “The Ecology of Eating: Smaller Portion Sizes in France than in the United States Help Explain the French Paradox.” Psychological Science 14 (5), September: 450-454.

Ruhm, C.J. 2007. “Current and Future Prevalence of Obesity and Severe Obesity in the United States.” NBER Working Paper No. 13181. National Bureau of Economic Research, Cambridge, MA.

Sabia, J.J. 2007. “The Effect of Body Weight on Adolescent Academic Performance.” Southern Economic Journal 73(4): 871-900.

Salk Institute. 2007. Salk researchers discover first gene that specifically links calorie restriction to long life. Press Release (Web site).

Schulze, M.B., Manson, J.E., Ludwig, D.S., Colditz, G.A., Stampfer, M.J., Willett, W.C., and Hu, F.B. 2004. “Sugar-Sweetened Beverages, Weight Gain, and Incidence of Type 2 Diabetes in Young and Middle-Aged Women.” JAMA 292: 927-934.

Segal, N.L, and Allison, D.B. 2002. “Twins and Virtual Twins: Bases of Relative Body Weight Revisited.” International Journal of Obesity 26: 437-441.

Serdula, M.K., Mokdad, A.H., Williamson, D.F., Galuska, D.A., Mendlein, J.M., and Heath, G.W. 1999. “Prevalence of Attempting Weight Loss and Strategies for Controlling Weight.” JAMA 282: 1353-1358.

Silventoinen, K., Jousilahti, P., Vartiainen, E., and Tuomilehto, J. 2003. “Appropriateness of Anthropometric Obesity Indicators in Assessment of Coronary Heart Disease Risk among Finnish Men and Women.” Scandinavian Journal of Public Health 31: 283-290.

Simpson, J.A., MacInnis, R.J., Peeters, A., Hopper, J.L., Giles, G.G., and English, D.R. 2007. “A Comparison of Adiposity Measures as Predictors of All-Cause Mortality: The Melbourne Collaborative Cohort Study.” Obesity 15(4): 994-1003.

Snell, E.K., Adam, E.K., and Duncan, G.J. 2007. “Sleep and the Body Mass Index and Overweight Status of Children and Adolescents.” Child Development Jan./Feb. 78 (7).

Society of Actuaries. 1959. *Build and Blood Pressure Study 1959*. Chicago: Society of Actuaries.

Society of Actuaries. 1980. *Build Study 1979*. New York: Society of Actuaries/Association of Life Insurance Medical Directors of America.

Solomon, C.G., and Manson, J.E. 1997. “Obesity and Mortality: A Review of the Epidemiologic Data.” American Journal of Clinical Nutrition 66: 1044S-1050S.

Stein, C.J., and Colditz, G.A. 2004. “The Epidemic of Obesity.” The Journal of Clinical Endocrinology & Metabolism 89(6): 2522-2525.

Stevens, J., Cai, J., Pamuk, E.R., Williamson, D.F., Thun, M.J., and Wood, J.L. 1998. “The Effect of Age on the Association between Body-Mass Index and Mortality.” New England Journal of Medicine 338: 1-7.

Stevens, J., Plankey, M.W., Williamson, D.F., Thun, M.J., Rust, P.F., Palesch, Y., and O'Neil, P.K.M. 1998. “The Body Mass Index-Mortality Relationship in White and African American Women.” Obesity Research 6: 268-277.

Strauss, R.S., and Pollack, H.A. 2001. “Epidemic Increase in Childhood Overweight, 1986-1998.” JAMA 286: 2845-2848.

Sturm, R. 2003. “Increases in Clinically Severe Obesity in the United States, 1986-2000.” Archives of Internal Medicine 163, Oct. 13: 2146-2148.

Sturm, R. 2002. “The Effects of Obesity, Smoking, and Drinking on Medical Problems and Costs.” Health Affairs 21(2): 245-253.

Sturm, R., Ringel, J.S., and Andreyeva, T. 2004. “Increasing Obesity Rates and Disability Trends.” Health Affairs 23(2): 199-205.

Sui, X., LaMonte, M.J., Laditka, J.N., Hardin, J.W., Chase, N., Hooker, S.P., and Blair, S.N. 2007. “Cardiorespiratory Fitness and Adiposity as Mortality Predictors in Older Adults.” JAMA 298(21): 2507-2516.

Sutocky, J. 2002. “Obesity-related Morbidity and Mortality, California, 2000-2--1.” Center for Health Statistics, State of California. Data Summary No. DS05-0800.

Swithers, S.E., and Davidson, T.L. 2008. “A Role for Sweet Taste: Calorie Predictive Relations in Energy Regulation by Rats.” Behavioral Neuroscience 122(1): 161-173.

Takata, Y., Ansai, T., Akifusa, S., Soh, I., Yoshitake, Y., Kimura, Y., Sonoki,, K., Fujisawa, K., Awano, S., Kagiyama, S., Hamaski, T., Nakamichi, I., Yoshida, A., and Takehara, T. 2007. “Physical Fitness and 4-year Mortality in an 80-Year-Old Population.” The Journal of American Geriatrics Society 55(6): 913-917.

Takata, Y., Ansai, T., Soh, I., Akifusa, S., Sonoki, K., Fujisawa, K., Awano, S., Kagiyama, S., Hamaski, T., Nakamichi, I., Yoshida, A., and Takehara, T. 2005. “Physical Fitness and 4-year Mortality in an 80-Year-Old Population.” The Journals of Gerontology Series A: Biological Sciences and Medical Sciences 62: 851-858.

Thorpe, K.E., Florence, C. S., Howard, D.H., and Joski, P. 2004. “The Impact of Obesity on Rising Medical Spending.” Health Affairs W4-480-486.

Thorpe, K.E., Florence, C.S., Joski, P. 2004. “Which Medical Conditions Account for the Rise in Health Care Spending?” Health Affairs W4-437-445.

Thorpe, K.E., Katz, D.L., Scandlen, G. 2007. “Obesity Driving U.S. Health Costs.” Health Affairs online. Oct. 2, 2007.

U.S. Department of Health and Human Services. 2006. *Chartbook on Trends in the Health of Americans*. 2007. Washington, D.C.: U.S. Government Printing Office.

_____. 2000. *Healthy People 2010: National Health Promotion and Disease Objectives*. Washington, D.C.: U.S. Government Printing Office.

_____. 2006. *Healthy People 2010: Midcourse Review*. Washington, D.C.: U.S. Government Printing Office.

_____. 2001. *The Surgeon General’s Call to Action to Prevent and Decrease Overweight and Obesity*. Washington, D.C.: U.S. Government Printing Office.

van Baal, P.H.M., Polder, J.J., Ardine de Wit, G., Hoogenveen, R.T., Feenstra, T.L., Bloshuizen, H.C., Engelfriet, P.M., and Brouwer, W.B.F. 2008. "Lifetime Medical Costs of Obesity: Prevention No Cure for Increasing Health Expenditure." PLoS Med 5(2): e29.0242-0249.

van Dam, R.M., Willett, W.C., Manson, J.E., and Hu, F.B. 2006. “The Relationship between Overweight in Adolescence and Premature Death in Women.” Annals of Internal Medicine 145: 91-97.

Vartanian, L.R., Schwartz, M.B., and Brownell, K.D. 2007. “Effects of Soft Drink Consumption on Nutrition and Health: A Systematic Review and Meta-analysis.” American Journal of Public Health. 97(4), April: 667-675.

Wada, R., and Tekin, E. 2007. “Body Composition and Wages.” NBER Working Paper No. 13595. National Bureau of Economic Research, Cambridge, MA.

Wang, Y. 2001. “Cross-national Comparison of Childhood Obesity: The Epidemic and the Relationship between Obesity and Socioeconomic Status.” International Epidemiological Association 30: 1129-1136.

Wardle, J., Carnell, S., Haworth, C., and Plomin, R. 2008. “Evidence for a Strong Genetic Influence on Childhood Adiposity Despite the Force of the Obseogenic Environment.” American Journal of Clinical Nutrition. 87 (2), Feb.: 398-404.

Wannamethee, S.G., Shaper, A.G., Lennon, L., Whincup, P.H. 2007. “Decreased Muscle Mass and Increased Central Adiposity are Independently Related to Mortality in Older Men.” American Journal of Clinical Nutrition 86(5), Nov.: 1339-1346.

Wei, M., Kampert, J.B., Barlow, C.E., Nichaman, M.Z., Gibbons, L.W., Paffenbarger, R.S., and Blair, S.N. 1999. “Relationship between Low Cardiorespiratory Fitness and Mortality in Normal-Weight, Overweight, and Obese Men.” JAMA 282: 1547-1553.

Weill, E., Wachterman, M., McCarthy, E.P., Davis, R.B., O'Day, B., Iezzoni, L.I., and Wee, C.C. 2002. “Obesity among Adults with Disabling Conditions.” JAMA 288: 1265-1268.

Weinstein, A.R., Sesso, H.D., Lee, I.M., Cook, N.R., Manson, J.E., Buring, J.E., and Gaziano, J.M. 2004. “Relationship of Physical Activity vs. Body Mass Index with Type 2 Diabetes in Women." JAMA 292: 1188-1194.

Weiss, W. 1997. “Cigarette Smoking and Lung Cancer Trends.” Chest 111: 1414-1416.

Wessel, T.R., Arant, C.B., Olson, M.B., Johnson, B.D., Reis, S.E., Sharaf, B.L., Shaw, L.J., Handberg, E., Spoko, G., Kelsey, S.F., Pepine, C.J., and Merz, C.N.B. 2004. "Relationship of Physical Fitness vs. Body Mass Index with Coronary Artery Disease and Cardiovascular Events in Women.” JAMA 292: 1179-1187.

Whitaker, R.C., Wright, J.A., Pepe, M.S., Seidel, K.D., Dietz, W.H. 2006. “Predicting Obesity in Young Adulthood from Childhood and Parental Obesity.” New England Journal of Medicine 337: 869-873.

Whitmer, R.A., Gustafson, D.R., Barrett-Connor, E., Haan, M.N., Gunderson, E.P., Yaffe, K. 2008. "Central Obesity and Increased Risk of Dementia More than Three Decades Later." Neurology. March 26, doi:10.1212/01.wnl.0000306313.89165.ef.

Wilde, P.E. 2004. “Message under Revision: USDA Speaks about Beef, Cheese, and Obesity.” Choices 2004-3.

Wilde, P.E. 2006. “Federal Communication about Obesity in the *Dietary Guidelines* and Checkoff Programs.” Obesity 14(6), June: 967-973.

Willett, W.C., Dietz, W.H., and Dolditz, G.A. 1999. “Guidelines for Healthy Weight.” New England Journal of Medicine 341: 427-434.

Willett, W.C., Hu, F.B., Colditz, G.A., and Manson, J. E. 2005. “Underweight, Overweight, Obesity, and Excess Deaths.” JAMA 294: 551.

Williamson, D.F. 1999. “The Prevention of Obesity.” New England Journal of Medicine 341(6): 1140-1141.

Wilson, P.W.F., D'Agostino, R.B., Levy, D., Belanger, A.M., Silbershatz, H., and Kannel, W.B. 1998. “Prediction of Coronary Heart Disease Using Risk Factor Categories.” Circulation 97: 1837-47.

Wolf, A., and Colditz, G. 1998. “Current Estimates of the Economic Cost of Obesity in the United States.” Obesity Research 6: 97-106.

World Cancer Research Fund/American Institute for Cancer Research. 2007. “Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective.” Washington, D.C.: AICR.

World Health Organization. 2000. *Obesity: Preventing and Managing the Global Epidemic, Report of a WHO Consultation*. Geneva, Switzerland. World Health Organization: WHO Technical Report Series, 894.

_____. 2003. *Diet, Nutrition and the Prevention of Chronic Diseases.* Geneva, Switzerland. World Health Organization: WHO Technical Report Series, 916.

Wright, C.M., Parket, L., Lamong, D., Craft, A.W. 2001. "Implications of Childhood Obesity for Adult Health: Findings from Thousand Families Cohort Study." British Medical Journal. BMJ 323; 1280-1284.

Yan, L.L., Daviglus, M.L., Liu, K., Stamler, J., Wang, R., Pirzada, A., Garside, D.B., Dyer, A.R., Van Horn, L., Liao, Y., Fries, J.F., Greenland, P. 2006. "Midlife Body Mass Index and Hospitalization and Mortality in Older Ages." JAMA 295: 190-198.

Yun, S., Zhu, R.-P., Black, W., and Brownson, R.C. 2006. "A Comparison of National Estimates of Obesity Prevalence from the Behavioral Risk Factor Surveillance System and the National Health and Nutrition Examination Survey." International Journal of Obesity 30: 164-170.

Zablotsky, D., and Mack, K.A. 2004. "Changes in Obesity Prevalence among Women Aged 50 years and Older." Research on Aging 26(1): 13-30.

12. List of Figures and Charts

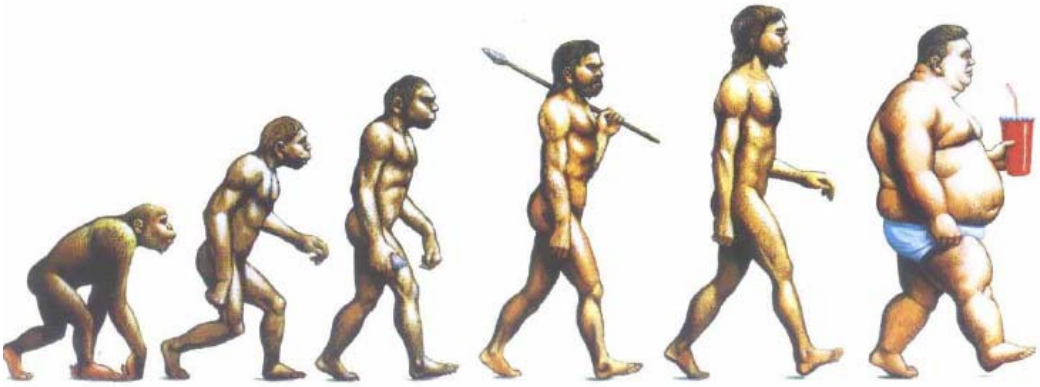
Figures **Page**

1	Male smoking and cancer	8
2	Female smoking and cancer	8
3	Trends in obesity prevalence for U.S. adults aged 20 and older by gender between 1999-2000 and 2005-2006	20
4	Changes in the distribution of BMI of U.S. adults between 1976-1980 and 2005-2006	21
5	Relations between key health factors	27

Tables **Page**

1	Relation between body mass index (BMI) and height (feet/inches) and weight (pounds) for adults	16
2	Obesity prevalence for U.S. adults aged 20 and older by gender and age: 2005-2006	19
3	1988-94 compared with 2002-2004 adult obesity as a percent of U.S. population	22
4	1988-1994 compared with 2002-2004 adult obesity as a percent of U.S. population	22
5	Prevalence of obesity among adults ages 20 and over on a self-reported basis	23
6	Prevalence of adult class 3 obesity (BMI>40)	24
7	Prevalence of metabolic syndrome in U.S.	26
8	Trends in prevalence in overweight of U.S. children and adolescents between 1971-2004	40
9	Trends in prevalence of overweight of U.S. children ages 6-11 between 1976-1980 and 2001-2004	41
10	Trends in prevalence in overweight of U.S. adolescents ages 12-19 between 1976-1980 and 2001-2004	41
11	Energy intake in kcals and percentage from selected sources	61
12	Percentage of U.S. adults aged 18 and over who engage in regular leisure physical activity	65
13	Percent of obese in selected countries	75
14	Percentage of individuals 50+ by corrected self-reported BMI	77
15	Relative risk of type 2 diabetes by level of physical activity, BMI and glucose level	91
16	Attribution of Sources of Mortality Improvement Between 1980 and 2000	97
17	World Cancer Research Fund (2007) assessment of the relationship between body fatness and various cancers	102
18	Age-adjusted multivariate mortality hazard ratios by BMI based on the NIH-AARP study	109
19	Hazard ratios relative to standard BMI (20.0-24.9) for older adults	118
20	Underwriting criteria by BMI range used by an insurer	133
21	Weight categories	182

Where We Have Come From; Where We Are Going?



From the cover of the *Economist*, Dec. 13-19, 2003