Quantifying Obesity in Economic Research: How Misleading is the Body Mass Index?

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Selected Paper prepared for presentation at the Agricultural & Applied Economics Association 2010 AAEA, CAES, & WAEA Joint Annual Meeting, Denver, Colorado, July 25-27, 2010.

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This draft: July 7, 2010

ABSTRACT

Obesity encapsulates the increased risk of disease and premature death associated with excess fat, but it is usually measured using a simple function of weight and height known as the Body Mass Index (BMI). Economists use the BMI to determine the prevalence of obesity within the population, to estimate the "disease burden" created by obesity, to establish the additional medical spending attributable to obesity, and to measure the current and potential effects of government policy on obesity. Estimates will be biased if the measurement error from using BMI to proxy for obesity is correlated with other covariates. In this paper we use a flexible function of percent body fat and several metabolic factors to construct an obesity index to predict death and disease. We use this index to show that using BMI leads researchers to misattribute obesity-related health outcomes to other factors such as aging, family health history, alcohol consumption, and household income. Moreover, we show how these biases necessarily generate the opposite implications for studies on the causes of obesity. Specifically, using BMI leads researchers to underestimate the effects of aging, family history of diabetes, alcohol consumption, and household income on obesity. We find no significant evidence of measurement error bias associated with smoking or insomnia. We also show that, compared with our obesity index, BMI generates substantially more false negative predictions of diabetes.

JEL Codes: C52, I10

Key Words: Obesity, percent body fat (%BF), body mass index (BMI), economic costs, measurement error.

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This project was supported by the National Research Initiative of the Cooperative State Research, Education and Extension Service, USDA, Grant # 2006-55215-16720 and by a cooperative grant from the USDA Economic Research Service, Agreement # 58-3000-8-013. We also gratefully acknowledge financial and indirect support from the University of California Agricultural Issues Center and the Giannini Foundation of Agricultural Economics.

I. Introduction

The medical community has broadly recognized that the body mass index (BMI) poorly measures an individual's fat content (adiposity) (Garn, Leonard, and Hawthorne, 1986; Smalley *et al.*, 1990; Gallagher *et al*., 1996; Deurenberg, 2001; Frankenfield *et al.*, 2001; Prentice and Jebb, 2001). But BMI is easy to measure: it equals the ratio of weight in kilograms to the square of height in meters.[1] This simplicity makes BMI an appealing and popular gauge of adiposity and overall health "even though it is as much a measure of Lean Body Mass as it is a measure of fatness or obesity" (Garn, Leonard, and Hawthorne, 1986, p. 997). BMI is the predominant instrument that both medical clinicians and researchers use for classifying individuals into obesity categories, to determine the prevalence of obesity within the population, and to estimate the "disease burden" created by obesity.[2]

For similar reasons, BMI has also become the main measure of obesity and related health outcomes used by economists. Several authors have attempted to estimate the effects on BMI resulting from participation in the Food Stamp Program (Gibson, 2003; Chen, Yen, and Eastwood, 2005; Meyerhoefer and Pylypchuk, 2008). Others have used BMI in studies of the relationships between obesity and factors such as the number of fast-food restaurants, time spent preparing meals, cigarette taxes, second-hand smoke laws, and U.S. farm policy (Carter, Glaeser, and Shapiro, 2003; Rashad, Grossman, and Chou, 2005; Alston, Sumner, and Vosti, 2008; Dunn, 2008; Anderson and Matsa, 2009). Also, BMI-defined obesity categories are used to estimate the direct and indirect costs attributable to obesity, the difference in medical

[1] In the studies mentioned above and throughout this paper, we use the term BMI to refer to this most commonly used formula. Smalley *et al*. (1990) discuss several other weight-to-height BMI formulas that have been proposed as alternatives.

[2] Throughout this paper we use the terms "obesity" and "obese" generically, to refer to both the overweight and the obese, as well as specifically, in context, to distinguish between "obese" and "overweight" as different categories of obesity.

expenditures for obese and overweight people compared with normal individuals, and the share of the growth in medical spending attributable to an increased prevalence of obesity.

In this paper we quantify the implications of using BMI to estimate the causes and effects of obesity. Standard econometric theory implies that measurement error generates bias depending on the correlation between the measurement error and the explanatory variables in a regression model (Fuller, 1987; Bound and Kreuger, 1991); estimates of the causes and effects of obesity could be quite erroneous when this correlation is high. For example, if BMI has a greater measurement error for the average heavy drinker of alcohol than for the rest of the population, a finding that heavy drinking predicts lower BMI may merely reflect prediction of the measurement error.

We build on a few recent papers that have addressed the drawbacks of BMI in economic research. Burkhauser and Cawley (2008) pointed out the substantial differences between BMI and percent body fat (%BF) in measuring obesity. They also found a negative correlation between employment and total body fat, but no significant correlation between fat-free mass and employment. This difference was obscured in previous papers that measured obesity using BMI. Similarly, Wada and Tekin (2010) found differences in how body fat and fat-free mass correlate to wages, and Johansson *et al*. (2009) concluded that labor market underperformance associated with obesity may be measured with bias.

To understand measurement error and its implications, we first have to quantify obesity. In this paper, we define obesity conventionally, as excess fat that increases the risk of developing diseases such as type 2 diabetes or of dying prematurely. We estimate the risk of such diseases or death and calculate an implied obesity index that depends on a person's sex and race. We find

evidence of significant measurement error bias in models that use BMI as a proxy for obesity in determining disease and mortality risk.

II. Quantifying Obesity: Background

An obese individual has a relatively high risk of disease and premature death attributable to excess fat (Allison *et al.*, 1999; US DHHS, 2001; Flegal *et al.*, 2007; Prospective Studies Collaboration, 2009). Quantifying excess fat is difficult, but medical researchers have begun to uncover the fundamental mechanisms behind the development of obesity and the underlying changes that link obesity to specific diseases. We begin by summarizing these mechanisms to provide context for our analysis.

A. Mechanisms Linking Obesity, Morbidity, and Mortality

Obesity exacerbates or contributes to numerous co-morbidities and diseases including stroke, cardiovascular disease, peripheral artery disease, colon cancer, postmenopausal breast cancer, various musculoskeletal conditions (e.g., osteoarthritis), gallbladder disease, and type 2 diabetes, among others (Must *et al.*, 1999; US DHHS, 2001; Flegal *et al.*, 2007). [3] Adipocytes (i.e., fat cells), are an "organ" of the endocrine system, secreting "a number of bioactive mediators that influence not only body weight homeostasis but also insulin resistance . . . lipids, blood pressure, coagulation, fibrinolysis and inflammation . . ." (Van Gaal, Mertens, and De Block, 2006).[4] These bioactive mediators, collectively known as adipokines, also respond to

[3] A co-morbidity is "A medical condition that exists in addition to, and is caused or worsened by, obesity or any other primary disease being studied or treated," from: http://health.ucsd.edu/specialties/lapband/about/glossary.htm.

[4] Fibrinolysis is the natural process that breaks down blood clots and keeps blockages and other problems from occurring. From: http://www.nlm.nih.gov/medlineplus/ency/article/000577.htm

signals from the central nervous and hormone systems (Kershaw and Flier, 2004; National Cancer Institute, March 2004; Poirier *et al*., 2006).

The metabolic and systemic abnormities associated with obesity lead to conditions such as the metabolic syndrome, which precedes the development of type 2 diabetes, cardiovascular disease (CVD), and death (American Heart Association, 2004; Kershaw and Flier, 2004). In obese persons, when lipids (fats) collect in the major organs rather than in fat cells, the ensuing "lipotoxicity" can lead to death of the organ tissues. Similarly, when fat accumulates around organs it impairs their function by secreting compounds onto them, compressing them into less space, or both (Poirier *et al*., 2006). Figure 1 illustrates the biological mechanisms that underlie the links between obesity, vascular disease, type 2 diabetes, and some cancers.

[Figure 1: Mechanics of Excess Adipose Tissue and Disease Risk]

Medical research has shown that the amount of fat and its distribution throughout the body affect health outcomes (Després *et al.,* 1990). In particular, it makes a significant difference whether fat cells are evenly dispersed throughout the body or have accumulated in and around the abdomen. Abdominal fat has an especially detrimental effect, increasing the risk of developing insulin resistance and several types of cancer (Kannel, D'Agostino, and Cobb, 1996; Okosun *et al.,* 2004).[5] Several biological mechanisms account for the role abdominal fat plays in raising the risk of CVD, type 2 diabetes, and cancer. In addition, abdominal fat independently increases the risk of high blood pressure and low high-density-lipoprotien (HDL) or "good"

[5] Central adiposity or obesity, trunk fat, belly fat, upper body fat, and android obesity are all synonyms for abdominal fat or adiposity. Central adiposity is defined as "the storage of adipose tissue preferentially in adipocytes on or within the trunk rather than the extremities" (Calle and Kaaks, 2004).

cholesterol.[6] Adipocytes also secrete sex hormones (e.g., testosterone and estrogens), which influence the body's preferred locations for storing fat and how lipoprotiens metabolize. Taken in combination, the metabolic effects of intra-abdominal adiposity independently increase the risk of CVD (Després *et al.*, 1990; Chan *et al.*, 2003; Okosun *et al.*, 2004). The elevated levels of estrogen in the blood stream associated with obesity significantly increase the risk of postmenopausal breast and endometrial cancers. Similarly, unable to metabolize the excess insulin and glucose building up circulating in their blood, those with central obesity have a higher risk of colon and endometrial cancer, and probably a higher risk of pancreatic and renal-cell (kidney) cancer as well. Insulin stimulates the production of IGF-1 and both hormones simultaneously encourage cell growth and impede cell death, having a tumor-producing effect (Calle and Kaaks, 2004). Smoking exacerbates the metabolic abnormalities that underlie the links between obesity, type 2 diabetes, CVD, and cancer, as evidenced by the shorter life expectancy of obese smokers (Van Gaal, Mertens, and De Block, 2006).

Maternal obesity, particularly in the second and third trimesters, also appears to affect predisposition of progeny to obesity and type 2 diabetes through various pathways (McGanity, Dawson, and Hook, 1998; King, 2006). Exposure in the womb to over-nutrition, type 2 diabetes, or gestational diabetes, exposure in early infancy to over-feeding, or both, can irreversibly "program the development of obesity and diabetes" by altering the biologic systems that regulate food intake, metabolism, and weight (Bouret *et al.*, 2004; Plagemann, 2008).

Aging also affects body composition and the distribution of body fat, even for those who maintain their body weight throughout old age. As people get older they lose muscle mass and

[6] An "independent risk factor" for a disease is a risk factor that is statistically significantly related to a health outcome, controlling for all other established risk factors. However, finding an independent risk factor is not equivalent to establishing causation (Brotman *et al.*, 2005).

height, gain fat, and have an increased predisposition to storing fat in the abdomen. Taken in combination, the changes in body composition, body fat distribution, and triglyceride levels associated with aging could explain much of the high incidence of the Metabolic Syndrome (TMS) among the old (Zamboni *et al.*, 2005; Miller, 2008).

B. Existing Measures of Obesity

The previous section describes how the accumulation of excess fat in the human body causes disease and premature death. Therefore, a first-order measure of obesity is the fraction of total body weight composed of only adipose tissue, expressed as the percent body fat. This measure quantifies overall body composition by distinguishing between the share of fat and non-fat tissues (i.e., bones, internal organs, and muscles) in total body weight: the greater the proportion of fat, the more obese is an individual. However, %BF is costly and difficult to measure accurately.[7]

The impracticality of large scale measurement and monitoring of %BF creates demand for simple and inexpensive measures of obesity. The most popular such measure is the BMI, defined as the ratio of weight in kilograms to the square of height in meters. The BMI had its genesis when the insurance industry recognized the increased mortality risk associated with obesity and the need for an index of "normal" body weight (Eknoyan, 2008). The "normal" range for BMI of 20-25 was defined because it correlates reasonably well with the minimum risk of death from life insurance tables (Pi-Sunyer, 1998). As a measure of relative weight-for-

[7] Body composition can be estimated using imaging techniques (e.g., Dual-Energy X-ray Absorption (DEXA), CAT scan, or MRI), body element assay methods (e.g., isotope ^{40}K counting), anthropometry (e.g., skinfold thickness or BMI), , and electrical conductivity techniques (bioelectrical impedance analysis (BIA)), among other techniques. The cost, accuracy, and ease of collecting the requisite measurements varies significantly among methods. Accounting for the influence of race, age, and sex, BIA accurately measures total body water (and from there, body fat) with a non-invasive (e.g., no radiation exposure or needle pricks), non-hazardous, and low cost device (Forbes, 1998).

height, epidemiologists started relying on BMI because it varies less with height or frame size than other anthropometric measures do, and correlates with body density (and thus body fat) at least as well as other indices do (Keys *et al.*, 1972).

Several authors have found that the common BMI inadequately predicts the amount of adipose tissue carried by an individual, and that general BMI cutoff points are unsuitable, especially for specific subpopulations (Burkhauser and Cawley, 2008; Garn, Leonard, and Hawthorne, 1986; Smalley *et al.*, 1990; Gallagher *et al*., 1996; Deurenberg, 2001; Frankenfield *et al.*, 2001; Prentice and Jebb, 2001). Frankenfield *et al.* (2001) showed that BMI does a poor job of predicting %BF for individuals who have a BMI below the obesity threshold (i.e., BMI < 30) and concluded that %BF is a better gauge of obesity than BMI in individuals having BMI < 30. Alternatives to BMI as measures of fatness include the waist-to-hip ratio (WHR), the waist-to-height (or waist-to-stature) ratio (WHtR), and the waist circumference (WC). These measures are all simple to calculate. Moreover, they bring information not just on the amount of excess fat accumulation, but also the distribution of body fat.

Based on the discussion in the previous section, the location of excess fat significantly affects the risk of obesity-related disease and premature death (Prentice and Jebb, 2001; Gelber *et al.*, 2008). Using data from the Physicians' Health Study (PHS) and Women's Health Study (WHS), Gelber *et al.* (2008) found that among anthropometric measures, WHtR best predicts cardiovascular disease (CVD).[8] Using the Third National Health and Nutrition Examination Survey (NHANES III) data that we use in this paper, Janssen, Katzmarzyk, and Ross (2004) confirmed that WC outperforms BMI at predicting health risks associated with obesity. They showed that WC (measured to the nearest 0.1 cm) significantly predicts obesity-related co-

[8] Gelber *et al.* (2008) appraised each anthropometric index based on model fit (determined by a likelihood ratio test) and the strength of the relationship between CVD and the index (as measured by the estimated relative risk ratio).

morbidity whereas BMI (also measured as a continuous variable) does not. That is, BMI does not add any information about co-morbidity risk when WC is known.

Similarly, using longitudinal data from the Health Professionals Follow-Up Study, Wang *et al.* (2005) showed that WC (as a gauge of abdominal obesity) relative to BMI (as a measure of overall obesity) better represents the risk of type 2 diabetes in adult men. As hypothesized, Chan *et al.* (2003) found that compared with BMI, WC better predicts visceral adipose tissue contained within and behind the abdominal cavity (i.e., the intraperitoneal and retroperitoneal regions). Chan *et al.* (2003) concluded that "in men WC is the anthropometric index that most uniformly predicts the distribution of adipose tissue among several fat compartments in the abdominal region, there apparently being little value in measuring WHR or BMI."

Several authors have noted that in *clinical work* BMI can serve as a useful gauge of disease risk and overall health of individual patients. Litwin (2008) pointed out that, although several alternatives to BMI exist, "there is no clear consensus on how (or where) to measure these parameters or on defining the optimal cutoff values for normal and abnormal." A number of other obesity classification systems and cutoffs based on measures other than BMI exist, but are less-widely used. Table 1 displays the obesity categories and corresponding measurement ranges for several alternative measures of fatness. Table 2 shows the distribution of the adult population based on these alternative systems.

[TABLE 1. Alternative Obesity Classification Systems]

[TABLE 2. Adult Weight Distribution by Obesity Classification System]

Depending on the classification system, the prevalence of obesity in the NHANESIII survey ranges from 18 percent to 62.5 percent for adult men and from 24 percent to 75 percent for adult females, respectively. Because these systems all measure fatness differently (i.e., as the

ratio of weight to height², overall body fat, and abdominal fat) and have different criteria for establishing cut-off points, they paint drastically different pictures of the health status of American adults. Even the %BF classification system, whose cut-off points were set to coincide with the standard BMI cut-off points, puts the obesity prevalence among males at 1.75 times that of the BMI-based system (Gallagher *et al.*, 2000).[9] Using the NHANES III Burkhauser and Cawley (2008) demonstrated that the difference in obesity prevalence between black and white women falls from 12 percentage points to 5 percentage points when obesity is defined as having a %BF of 30 percent.

BMI and the obesity categories defined by it might not accurately reflect the current and potential loss of life and decline in quality of life associated with an ever-growing number of people carrying excess body fat. Individuals, social scientists, and policymakers would like a "fatness" index and classification system that reflects the risks to health and wellbeing associated with carrying excess fat. An obesity index that reflects these risks is needed especially for overweight children, who stand to lose the most in terms of quality and length of life (Freedman and Perry, 2000; Deckelbaum and Williams, 2001; Ebbeling, Pawlak, and Ludwig, 2002; Boney *et al.*, 2005; Speiser *et al.*, 2005; Weiss and Caprio, 2005; Daniels, 2006).

III. How BMI is Used to Quantify Obesity and Its Consequences

The vast majority of research on obesity uses BMI to categorize people into a weight category (e.g., normal-, over-, or underweight). If measurement error biases are significant, such

[9] Gallagher *et al.* (2000) collected data on %BF and BMI for whites, African Americans, and Japanese and estimated the equation $PBF = \beta_0 + \beta_1 BMI^{-1} + \beta_2 Sex + \beta_3 Age + \beta_4 Age * Sex + \beta_5 Sex * BMI^{-1}$ for whites and African Americans, and $PBF = \alpha_0 + \alpha_1 BMI^{-1} + \alpha_2 Age$ for Japanese men and women separately. From the estimated coefficients Gallagher *et al.* (2000) then predicted $\widehat{PBF}$ corresponding to BMI of 18.5, 25, and 30 for specific age, race, and gender combinations.

use can lead to a misleading portrayal of the extent of the obesity epidemic, and biased estimates of its causes and costs. The measurement error bias associated with using BMI as a proxy for obesity may pervade many studies.

A. Excess Morbidity and Mortality Associated with BMI

Many health complications and conditions are associated with high BMI. Using data from the Third National Health and Nutrition Examination Survey (NHANES III), Must *et al.* (1999) found that among co-morbidities associated with high BMI, high blood pressure (HBP) is the most prevalent among U.S. adults. Their results indicate that type 2 diabetes, gallbladder disease, coronary heart disease and osteoarthritis are all more prevalent among those classified by BMI as overweight or obese and that the prevalence increases with the degree of obesity. In addition, they found that the prevalence of having two or more co-morbidities increases with ascending BMI class. Among co-morbidities associated with obesity, only high blood cholesterol (HBC) did not increase in prevalence by BMI category; however individuals with BMI $\geq$ 25 did have a higher prevalence of HBC than those with BMI < 25.

Excess weight caused by sedentary lifestyles and unhealthy diets comes in second only to, and may soon surpass, cigarette smoking as the leading "actual cause of death" in the United States (Mokdad *et al.*, 2004). Published estimates indicate that individuals with a BMI $\geq$ 30 have a 50-100 percent greater risk of premature death than those within the healthy BMI range (25 $\geq$ BMI > 20) (US DHHS, 2001).[10] Allison *et al.* (1999) produced a conservative estimate of

[10] The most commonly used BMI cutoff points are 18.5, 25, and 30 for lower bounds on the normal, overweight and obese categories. A BMI of 20 is also commonly used as the lower bound on the normal category, but some medical studies have alternative BMI cutoff points based on the BMI distribution of the sample under investigation.

approximately 300,000 excess adult deaths per year attributable to obesity.[11] Similarly, Flegal *et al.* (2007) used hazard rates generated from pooling the NHANES I, II, and III data to estimate cause-specific excess deaths attributable to overweight and obesity as defined by BMI. They found excess deaths from cardiovascular disease and obesity-related cancers associated with BMI > 35, and excess deaths from diabetes and renal disease were linked to BMI > 25. Jia and Lubetkin (2010) found that the number of healthy days of life lost from obesity has more than doubled, rising from 7.5 to 16.9 days of healthy life lost per adult between 1993 and 2008.

Prospective Studies Collaboration (2009) compiled data from fifty-seven separate surveys and found significantly increased risk of mortality from cardiovascular disease, diabetes, and respiratory disease for those above the upper bound of the healthy BMI range (BMI > 25). Their main finding was that, for those with a BMI between 25 and 50, every 5 point increase in BMI over the healthy threshold implied a 30 percent increase in all-cause mortality. They also estimated a 40 percent higher mortality from stroke and ischaemic heart disease and a 116 percent higher mortality from type 2 diabetes for every 5 point increase in BMI for those who had BMI > 25 (Prospective Studies Collaboration, 2009). Using data from several sources, including NHANES III, Fontaine *et al.* (2003) conducted a similar analysis and found a 1-13 year decrease in the expected remaining years of life for 20-year-old white males with BMI>30 relative to those with a "normal" BMI of 24. They also found significant differences in the predicted years of remaining life lost between races and sexes.

[11] The estimated number of excess deaths attributed to obesity is defined as the number of people who died in a given year who would not have died if they had been in the healthy BMI range at the start of the year (Allison *et al.*, 1999). Allison *et al.* (1999) predicted excess deaths attributable to obesity in 1991 and Flegal *et al.* (2007) approximated the share of excess cause-specific deaths attributable to overweight and obesity in 2004.

B. Impact of Economic Variables on Obesity

Researchers interested in the interactions between obesity and economic variables like price, consumer preferences, and government policy also rely heavily on BMI or weight categories defined by BMI as their measure of obesity. Several authors have attempted to estimate the effects on BMI resulting from participation in the Food Stamp Program (FSP).[12] Meyerhoefer and Pylypchuk (2008) found that women who participated in the FSP had a 2.5 percent lower chance of having a normal or under-weight BMI (i.e., BMI < 25). Their result is of the same sign, yet much smaller in magnitude, than those of similar previous studies (i.e., Townsend *et al.*, 2001; Gibson, 2003; Chen, Yen and Eastwood, 2005).

Rashad, Grossman, and Chou (2005) found that banning television ads for fast-food restaurants would reduce the numbers of children (3-11 years) and adolescents (12-18 years) classified as overweight by 10 and 12 percent, respectively. Dunn (2008) found that a 10 percent increase from the mean in the number of fast-food eateries in a region would increase BMI by $0.33 kg/m^2$ and Currie *et al.* (2009) found that the presence of a fast-food establishment less than a tenth of mile from a high school leads to a 5.2 percent increase in the percent of freshman who fall into the obese weight category. However, using the presence of an interstate highway as a proxy for fast-food restaurant supply, Anderson and Matsa (2009) found no evidence that having a greater number of restaurants available in an area leads to higher obesity rates for adults. They showed that even though people consume more calories at restaurants, they make up for this increase by eating fewer calories at meals eaten in the home, and they concluded, that health policy aimed at reducing obesity should not target restaurants. Cutler, Glaeser, and Shapiro

[12] The US Department of Agriculture Food Stamp Program (FSP) was renamed the Supplemental Nutrition Assistance Program (SNAP) in October, 2008. The FSP supplements the food budget of low income households with monthly benefits that can only be used to purchase food at certified retail locations.

(2003) agreed with Anderson and Matsa (2009), noting that the growth in calorie consumption between the 1970s and 1990s came from the consumption of more snacks and not from higher-calorie dinners.

Others have used BMI in studies of the relationships between obesity and factors such as the number of fast-food restaurants, time spent preparing meals, cigarette taxes, second-hand smoke laws (Lakdawalla and Phillipson, 2002; Chou, Grossman, and Saffer, 2004; Nonnemaker *et al.*, 2009). Chou, Grossman, and Saffer (2004) explored the relationship between real food prices, food availability, the real price of cigarettes, and obesity. They found that the change in the per-capita number of restaurants and the real price of cigarettes accounted for 60 and 20 percent, respectively, of the growth in BMI and obesity prevalence from 1984 to 1999. Nonnemaker *et al.* (2009) expanded on Chou, Grossman, and Saffer, estimating the effect of real cigarette prices for never, former, and current smokers separately. Without time and state specific time trends Nonnemaker et al. (2009) found the puzzling result that real cigarette prices (with the state real excise tax rate as an instrument) had the largest effect on the BMI for people who have never smoked. However, when they included time and state-specific time trends, consistent with the findings of the medical literature, Nonnemaker *et al.* found the largest effect of real cigarette prices on the BMI of former smokers.

Lakdawalla and Philipson (2002) proposed that the decline in energy expenditure and increase in calorie intake made possible by technological change explained 60 percent and 40 percent, respectively, of the rise in the prevalence of obesity from 1982 to 1996. The authors modeled BMI as a function of the strenuousness of an occupation, the strength needed for a given occupation, wages, and other individual characteristics. They found a negative and significant relationship between job strenuousness and BMI.

C. Estimates of the Cost of Obesity

Many authors have attempted to approximate the direct and indirect costs of obesity. Using the "prevalence approach" Colditz (1992) estimated that the combined indirect and direct economic cost of obesity in 1986 was $39.3 billion. This translates to 5.5 percent of the total cost of illness in 1986. Colditz (1992) attributed the second-largest share of the total cost of obesity in 1986, $11.3 billion, to non-insulin-dependent diabetes mellitus (i.e., type 2 diabetes). Wolf and Colditz (1998) estimated that obesity accounted for $52 billion of the direct costs of healthcare. More recently using health expenditure data from the National Health Accounts (NHA) Finkelstein *et al.* (2009) estimated the direct economic cost of overweight and obesity as $78.5 billion (9 percent of U.S. medical expenditures) in 1998 and up to $147 billion in 2008. Tucker *et al.* (2006) demonstrated a positive relationship between overall medical spending and BMI, controlling for the effect of increased BMI on life expectancy, with differences in this relationship dependent on gender and race. These estimates do not reflect the indirect costs associated with lost wages and forgone earnings because of heightened morbidity and mortality, and therefore they may understate the total economic cost. Obesity and ancillary conditions impose a significant burden on publicly funded programs (i.e., Medicaid and Medicare), which pay nearly half of the medical expenditures attributed to obesity.

In 2008 the American Diabetes Association estimated that treating type 2 diabetes in the United States imposed a total cost of $174 billion ($116 direct and $58 billion indirect); they attributed $26.9 billion of the indirect cost to premature mortality. Conditional on the prevalence of high BMI staying relatively stable over the next quarter century, Huang *et al.* (2009) estimated that the number of individuals diagnosed with type 2 diabetes and the number of those with diabetes who are eligible for Medicare will approximately double over this time span. They

concluded that the number of diagnosed cases will go from about 20 to 40 million, and the number of individuals with the disease who are eligible for Medicare will increase from 6.5 to 14.1 million.

D. BMI as an Anthropometric Index

The World Health Organization (WHO) defined an anthropometric index as a "combination of measurements . . . (that) relate to body size and composition. Sometimes this is the only type of relationship that can be inferred; indices should then be referred to as . . . nutrition or health indicators" (WHO 1995, pp. 7-8). The WHO stressed that improper use of indices as indicators of nutrition or health can lead to ineffectual public health policy and program choices. Anthropometric indices have several uses including identifying at-risk populations, determining populations for intervention, and establishing population norms and standards. Overall "a good indicator is one that best reflects the issue of concern or predicts a particular outcome" (WHO 1995, pp. 10-12).

The same 1995 WHO report stipulated that although the BMI cut-offs are based on the relationship between BMI and mortality, they do not imply targets for intervention, and they only serve as a useful tool to determine prevalence. Furthermore, WHO (1995) recommended using BMI in combination with many other measures (e.g., related to diet, exercise, and smoking) to determine individual risk. The WHO also advised against significant weight fluctuations within or between the healthy and overweight categories, as "weight-cycling" also relates to increases in morbidity and mortality risk, suggesting prevention as the preferred way to reduce the prevalence of obesity. Nevertheless, WHO (1995) conceded that "the method used to establish BMI cut-off points has been largely arbitrary . . .. It may therefore be necessary to revise the classification of overweight in terms of BMI based on health risk" (WHO 1995, pp. 312-313).

IV. Characterizing the Measurement Error from Using BMI to Measure Obesity

Percentage body fat provides a first-order approximation to obesity. We begin this section by presenting a framework for understanding the implications of using BMI to measure obesity instead of %BF. Next, given that the medical community has yet to reach a consensus as to what constitutes a high or unhealthy amount of body fat, we propose a new obesity index. Our index reflects the increased probability of a particular health outcome attributable to excessive fatness. We then develop a framework for understanding the implications of using BMI in place of the obesity index in economic research.

A. Measurement Error Bias when BMI is a Proxy for %BF

Suppose an economist aims to model the *determinants of obesity* and would like to estimate the regression model

$$\%BF_i = \beta' X_i + \varepsilon_i \tag{1}$$

for some set of explanatory variables X_i. However, the economist does not observe %BF, so instead uses BMI as the dependent variable. Equation (2) describes the relationship between *BMI* and *X*,

$$BMI_i = \gamma' X_i + u_i \tag{2}$$

Measurement error bias produces differences between the coefficients of interest, β, and the coefficients in the estimated model, γ.

From textbook econometric theory, OLS regression of *BMI* on *X* produces consistent estimates of β if the measurement error is uncorrelated with *X*. However, measurement error biases arise when *X* contains more or different predictive information about %BF than it does about BMI. For instance, suppose BMI has a greater measurement error for the average heavy

alcohol drinker than for the rest of the population. In this setting, a finding that heavy drinking predicts lower BMI may reflect prediction of the measurement error rather than prediction of obesity.

Expressed in terms of regression equations, measurement error bias manifests in a nonzero value of δ in the following regression:

$$\%BF_i = \alpha BMI_i + \delta' X_i + \nu_i \tag{3}$$

To see the connection between equations (1), (2), and (3), substitute (2) into (3) to get

$$\begin{aligned} \%BF_i &= \alpha\left(\gamma' X_i - u_i\right) + \delta' X_i + \nu_i \\ &= (\gamma\alpha + \delta)' X_i + \nu_i - \alpha u_i \\ &= \beta' X_i + \varepsilon_i \end{aligned} \tag{4}$$

where $\beta = \gamma\alpha + \delta$. Because *%BF* and *BMI* are measured on different scales, the absence of measurement error bias implies $\delta = 0$ or $\beta = \gamma\alpha$, where α denotes a scale factor. Thus, δ represents the difference between the coefficient of interest, β, and the coefficient we expect to estimate if we use BMI in place of %BF.

In Section VI of the paper, we run the regression (3) to assess the extent of measurement error bias. Nonzero estimates of the δ coefficient on an *X* variable imply that regressions of BMI on that variable are subject to measurement error bias. We do not address systematic measurement errors, such as those induced by deliberate underreporting of weight, because our BMI data are measured rather than self-reported. Nonetheless, as demonstrated by Rowland (1990) and others, if a particular dataset relies on self-reported weight values that are systematically biased in a way that induces correlation with explanatory variables of interest, then regressions based on those data will be subject to similar measurement error biases.

Similar measurement error problems also arise if BMI is used in place of %BF as a right-hand-side variable. Specifically, suppose an economist wishes to estimate the equation

$$y_i = \beta_1' X_i + \beta_2 \left(\%BF_i\right) + \varepsilon_i \tag{5}$$

but instead uses BMI in place of %BF. The resulting estimates of β_1 and β_2 will be biased if $\delta \neq 0$ in the regression in (3). Such estimates could be correctly interpreted as measuring the relationship between BMI and *y*, but not the relationship between obesity and *y*. The difference between these two interpretations could be significant if the estimates were used as a basis for a policy aimed at reducing %BF rather than BMI.

B. The Obesity Index and Measurement Error Bias

The medical community has not reached a consensus as to what constitutes a high or unhealthy amount of body fat. Furthermore, for a given amount of body fat, health outcomes may vary by race, age, and sex, and by the proportion of body fat amassed in the abdomen. Therefore one way to define obesity (*OB*) is as the increased probability of a particular health outcome owing to excessive fatness. We define an indicator variable *D* to equal one in the event of a bad health outcome (e.g., death or disease) and zero otherwise. We define two sets of variables that predict health outcomes: *F* denotes measures of fatness such as %BF and waist circumference, and *X* denotes a set of covariates. The obesity index for a particular individual *i,* is the increased risk resulting from carrying *extra* fat, i.e.,

$$OB_i = \Pr\left(D_i = 1 \mid F_i, X_i\right) - \Pr\left(D_i = 1 \mid \bar{F}_i, X_i\right) \tag{6}$$

where $\bar{F}_i$ signifies a healthy amount of fat.

To implement our index, we specify the model

$$\Pr\left(D_i = 1 \mid F_i, X_i\right) = \phi_{0i} + \phi_{1i} F_i + \phi_{2i} F_i^2 + \theta' X_i \tag{7}$$

where the *i* subscripts on ϕ_{0i}, ϕ_{1i}, and ϕ_{2i}, indicate that coefficient values may vary depending on individual characteristics such as sex and race. Because fatness as measured by F_i may affect health outcomes differently for people of different race and sex, the healthy amount of fatness $(\bar{F}_i)$ may also vary by race and sex. For example, women have greater average %BF values than men, yet women live longer than men. This fact suggests that the safe body fat percentage for women exceeds that for men.

Given the model in (7), the obesity index is

$$OB_i = \phi_{1i}\left(F_i - \bar{F}_i\right) + \phi_{2i}\left(F_i^2 - \bar{F}_i^2\right) \tag{8}$$

This index depends specifically on the health outcome or set of outcomes defined by *D*, and the set of control variables, *X*. Measurement error from using *BMI* in place of *OB* comes from two sources. First, *BMI* may not represent well the fatness variables in *F*. Second, compared with *OB*, *BMI* may correlate differently with the covariates. Thus, even if *BMI* = *F*, we would still have measurement error, because a high *BMI* may have a greater health effect for people with some characteristics than others (i.e., the ϕ_i coefficients can differ across individuals). These measurement errors might have implications for models of the determinants of obesity and for models and measures of the consequences of obesity.

As in the previous section, measurement error biases arise when *X* contains more or different predictive information about *OB* than it does about *BMI*. Specifically, suppose an economist aims to estimate β in the regression

$$OB_i = \beta' X_i + \varepsilon_i \tag{9}$$

but uses *BMI* in place of *OB* as in (2). The analogue to the measurement error equation (3) is

$$OB_i = \phi_{1i}(F_i - \bar{F}_i) + \phi_{2i}(F_i^2 - \bar{F}_i^2) = \alpha BMI_i + \delta' X_i + \nu_i \tag{10}$$

which we re-write as

$$\phi_{1i}F_i + \phi_{2i}F_i^2 = \phi_{1i}\bar{F}_i + \phi_{2i}\bar{F}_i^2 + \alpha BMI_i + \delta' X_i + \nu_i \tag{11}$$

By substituting this equation into the model of disease risk in (7) we get

$$\Pr\left(D_i = 1 \middle| F_i, X_i\right) = \phi_{0i} + \phi_{1i}\bar{F}_i + \phi_{2i}\bar{F}_i^2 + \alpha BMI_i + (\delta + \theta)' X_i + \nu_i \tag{12}$$

Since $\bar{F}_i$ may vary by individual characteristics such as race and sex, the term $\phi_{1i}\bar{F}_i + \phi_{2i}\bar{F}_i^2$ is a function of indicators for these characteristics and is therefore redundant when we include ϕ_{0i} in the equation. Thus, we have the measurement error equation

$$\Pr\left(D_i = 1 \middle| F_i, X_i\right) = \phi_{0i} + \alpha BMI_i + (\delta + \theta)' X_i + \nu_i \tag{13}$$

Comparing (7) and (13), we see that measurement error bias, which is indicated by $\delta \neq 0$, implies that the coefficients on X_i differ, depending on whether *BMI* or the fatness variables are used to predict health outcomes. For example, suppose a variable measuring alcohol consumption has a θ coefficient equal to zero in (7), but a negative coefficient when BMI is used to predict health outcomes as in (13). Based on BMI, we may conclude that alcohol consumption causes disease directly, when in fact it is unrelated to disease once we control for obesity.

Similarly to the previous section, we can write δ as the difference between the coefficient of interest β in (9) and the expected scaled coefficient from a regression of *BMI* on *X*, i.e., $\delta = \beta - \gamma\alpha$. This expression follows directly from substituting equation (9) into (7) and equation (2) into (13). In (7) and (13), the variables X_i affect D_i in two ways, both indirectly through the obesity variables and directly. An overestimate of the direct effect necessarily implies an underestimate of the indirect effect and vice versa. To continue the example in the previous

paragraph, suppose further that moderate alcohol consumption is uncorrelated with BMI, but is associated with a lower *OB* and therefore a lower diabetes risk. Using BMI, moderate alcohol would appear to have a direct negative effect on diabetes risk but no such effect on obesity when in fact alcohol consumption relates directly to obesity but not to diabetes. Thus, if we observe $\delta < 0$, then it means that using *BMI* leads to downward bias in the direct effect of the associated variable on health outcomes and upward bias in the effect of that variable on obesity. Because the *OB* index is unobservable, we estimate δ as the difference between the coefficients on *X* in equations (7) and (13). Figure 2 illustrates the sign of the bias implied when we estimate $\delta > 0$.

[FIGURE 2 . Interpretation of Measurement Error Bias when $\delta > 0$]

V. Data

We use the publically available NHANES III data and the many variables therein to carry out our statistical analysis. These data contain detailed personal characteristics and medical information for a nationally representative sample of individuals in the United States. We control for the complex survey design and sampling procedures using the sample weights and error estimating procedures recommended in the NHANES III analytical guide. A detailed description of the data and the adjustments we make to control for the survey structure follows.

A. NHANES III Data

Our data are from NHANES III, a periodic survey carried out between 1988 and 1994 by the National Center for Health Statistics (NCHS) and the Centers for Disease Control and Prevention (CDC). NHANES III contains details on household and individual characteristics, dietary recall information, lab test results, and medical exam measurements for a nationally representative sample of the civilian non-institutionalized population for respondents over two

months of age at the time of the survey.[13] We restrict our analysis to survey respondents over eighteen years of age, since BMI is not the standard metric used to diagnose childhood obesity.[14]

The use of a mobile examination center in NHANES III allowed for the collection of very detailed physical exam data. The examiners conducted bioelectrical impedance analysis (BIA) for individuals 12 years and older, with the exception of pregnant women and individuals who had pacemakers, using the Valhalla 1990B Bio-Resistance Body Composition Analyzer. BIA measurements can be used to calculate fat-free mass (FFM) (i.e., the weight of everything in the body that is not fat tissue), and thus %BF, or the fraction of total weight composed solely of body fat (Kyle *et al*., 2004). Table 3 displays selected summary statistics for adults surveyed in NHANES III.

[TABLE 3. *NHANES III Summary Statistics on BMI, Percent Body Fat, and Type 2 diabetes*]

We also use the public-use NHANES III Linked Mortality Files to examine the relationship between BMI-based obesity categories, various alternative measures of fatness (including %BF and WC), and the numerous causes of death associated with excess fat. For those individuals 17 years of age or older at the time of the NHANES III interview, the file includes their individual mortality status (alive or deceased), the number of months since their interview, and (if deceased) the underlying cause of death (COD). The follow-up period ended on December 31, 2006. The Linked Mortality data set used the 10th revision of the International Statistical Classification of Diseases, Injuries, and Causes of Death (ICD-10) coding system to

[13] Detailed information on the survey design and methods used to conduct the NHANES III survey and the public use data files are available from the NCHS website: http://www.cdc.gov/nchs/nhanes/nhanes_questionnaires.htm. Race/ethnicity is self identified in the survey as white, black, other, or Mexican American of unknown race.

[14] Obesity status for children is only partially determined by BMI for age and gender percentiles. Further testing and examination of diet and physical activity is needed to determine if a child is in fact carrying an unhealthy amount of body fat (Chinn, 2006).

classify primary COD, and also indicates if diabetes, hip fracture, or hypertension was cited as a secondary COD.[15]

B. Calculation of Percent Body Fat

Chumlea *et al.* (2002) used equations developed by Sun *et al.* (2003) from the NHANES III data to calculate measures of body composition, and described the distribution of %BF among the U.S. population. We use the same body composition equations as Chumlea *et al*. (2002) to calculate %BF for all individuals in the survey for whom we had complete data on BIA and body weight. We use equations (14) and (15) used to calculate FFM for males and females, respectively.

$$FFM_{Male} = -10.678 + 0.262\,Weight + 0.652\left(Height^2 \Big/ \mathrm{Res}^{RJL}\right) + 0.015\,\mathrm{Res}^{RJL} \tag{14}$$

$$FFM_{Fem} = -9.529 + 0.168\,Weight + 0.696\left(Height^2 \Big/ \mathrm{Res}^{RJL}\right) + 0.016\,\mathrm{Res}^{RJL} \tag{15}$$

where *FFM* and *Weight* is measured in kilograms, *Height* is measured in centimeters, and Res^{RJL} is a measure of electrical resistance in ohms. We convert the BIA measurements obtained in the NHANES III physical exam from Valhalla resistance units to RJL resistance units for males and females using equations (16) and (17).[16]

$$Res^{RJL}_{Male} = 2.5 + 0.98\,Res^{Valhalla} \tag{16}$$

$$Res^{RJL}_{Fem} = 9.6 + 0.96\,Res^{Valhalla} \tag{17}$$

[15] The ICD-10 system includes 113 possible causes of death. We group the 113 CODs into broader categories like heart disease, cancer, or respiratory disease.

[16] RJL and Valhalla are manufacturers of BIA devices. Since the FFM equations in Chumlea *et al.* (2002) were estimated with RJL BIA resistance values, we had to convert the BIA measurements from NHANES III.

We then calculate %BF as: $\%BF = 100 * (Weight - FFM) / Weight$.

C. Sampling and Replicate-Weighting Methods

NHANES III used a complex sampling method and, for the estimates to represent the whole U.S. population, we must properly weight and adjust the data prior to any statistical analysis. We use balanced repeated replication (BRR) methods, with the replicate and sampling weights included in the NHANES III data set, to account for the stratified sampling procedure and oversampling of specific subpopulations of interest. We also use the "*svy*" commands in STATA to generate all the summary statistics and to estimate all regression equations.

Sampling weights supplied in NHANES III take into account non-response bias, unequal sampling probability, and previously measured demographic information from the U.S. Census Bureau to ensure that the sample represents the U.S. population as a whole. The sampling weight indicates the number of people in the U.S. population that a respondent represents. In our analysis we apply the "MEC+Home" sample weights because we use data from both the mobile examination center and Household Survey portions of the data (Westat Inc., 1996).

D. Calculating Weight History Variables

It takes multiple years of being obese to cause death or disease, but we observe only a snapshot in time. We observe weight and disease status on the survey date, whether an individual died over the ensuing 12 to 18 years (depending on when they were surveyed) and, when one had died, the cause of death.

To better capture the lagged effect of obesity on health outcomes, we use weight recall data. We exploit the broad range of questions asked in the survey and knowledge about factors

that influence the change of FFM within individuals over time to extend the follow-up period by ten years. One of the many questions respondents were asked was: "what was your weight ten years ago?" Individual FFM and individual gains in weight over a period of time are largely a function of gender, age, physical activity, menopausal status, and whether and for how long the individual had been using hormone replacement therapy drugs (Fukagawa, Bandini, and Young, 1990; Poehlman *et al.*, 1995; Guo *et al.*, 1999; Kyle *et al.*, 2001). All of these characteristics can be deduced from information in NHANES III. Using individual characteristics and self-reported weight ten years previously, we estimate (18) and calculated (19) and (20).[17]

$$FFM_{i,t} = \beta_0 + \beta_1 Age_{i,t} + \beta_2 Female_i + \beta_3 Black_i + \beta_4 OtherRace_i + \beta_5 Menopause_i + \beta_6 Estrogen_i + \varepsilon_i \tag{18}$$

$$FFM_{i,t-10} = \hat{\beta}_0 + \hat{\beta}_1 Age_{i,t-10} + \hat{\beta}_2 Female_i + \hat{\beta}_3 Black_i + \hat{\beta}_4 OtherRace_i + \hat{\beta}_5 Menopause_{i,t-10} + \hat{\beta}_6 Estrogen_{i,t-10} \tag{19}$$

$$PBF_{i,t-10} = 100 * \left(\left(WtKgs_{i,t-10} - FFM_{i,t-10} \right) \Big/ WtKgs_{i,t-10} \right) \tag{20}$$

These calculations allow us to classify individuals based on their current BMI and %BF, but also on the BMI and %BF ten years previously, assuming no change in height. In essence, we extend the follow-up period by ten years and better capture the cumulative impact of excess body weight on morbidity and mortality.

[17] The regression results for this model are available upon request.

E. Identifying Type 2 Diabetes and the Metabolic Syndrome

The NHANES III survey did not distinguish between type 1 and type 2 diabetes in the health questionnaire, therefore we employ the strategy used by Thompson *et al.* (1999) to identify individuals with type 2 diabetes. We identify respondents as having type 2 diabetes if: (i) they reported having diabetes at any time other than during pregnancy (known as gestational diabetes), and they reported having been diagnosed with diabetes after age 30, or (ii) they were diagnosed as having diabetes between the ages of 18 and 30 years, and they were not taking insulin or they did not begin using insulin within one year of being diagnosed.

We use the International Diabetes Federation (IDF) definition of the metabolic syndrome to identify survey respondents who had the condition. The IDF defines individuals as having the metabolic syndrome if they have a measured $WC > 94$ cm for men (> 80 cm for women) and at least two of the four following factors: (i) blood triglycerides ≥ 150 mg/dL or treatment for high triglycerides, (ii) high-density-lipoprotein (HDL or "good") cholesterol < 40 mg/dL for men (< 50 mg/dL for women) or treatment for low HDL, (iii) systolic blood pressure (BP) ≥ 130 mm Hg, diastolic BP ≥ 85 mm Hg, or treatment for high BP, and (iv) fasting plasma glucose ≥ 100 mg/dL (IDF, 2006). In our analysis, if a respondent was taking prescription medications to lower BP or cholesterol, then we define that respondent as having "treatment for" these conditions. The NHANES III Exam and Lab data files contain all the measurements and blood test results needed to identify the metabolic syndrome.

VI. Results

To examine the value of BMI as a measure of disease risk and obesity we first estimate the measurement error associated with using BMI to predict obesity as measured by percent body

fat. Next, we construct our obesity index by combining information on the amount and location of body fat, weight history, and the metabolic abnormalities associated with obesity. Then we assess the nature and extent of measurement error problems in models that use BMI instead of our index to predict the risk of death and disease.

A. Evaluation of BMI as a Proxy for Percent Body Fat as a Measure of Obesity

As outlined in section II, the elevated risk of disease and death associated with obesity largely stems from the metabolic complications that excess adipose tissue promotes. We present in Table 4 results from regressions of %BF and BMI on a set of explanatory variables. We also present results from the measurement error regression in (13). We use race, sex, age, smoking status, having reached menopause (for women), and years of hormone replacement therapy as explanatory variables. Also, we model men and women separately because women need more body fat to initiate puberty and maintain fertility (Tataranni *et al.*, 1997; Speakman, 2007). The dichotomy in %BF for men and women appears in early childhood and should be controlled for (Pi-Sunyer, 1998). A positive and significant coefficient on an explanatory variable (the *Xs*) in column (3), (6), or (9) implies that a model using BMI as a proxy for obesity would understate the effect of *X* on %BF.

[TABLE 4. %BF Measurement Error Regressions]

The first column of Table 4 shows that women on average have %BF 11.3 percentage points higher than men. However, the second column shows that women have very similar BMI levels. Thus, we have a coefficient of 11.45 on the female dummy variable in the measurement error equation. This result shows how using BMI in place of %BF would vastly understate differences in body fat. For both men and women %BF increases with age, but this effect goes

understated when using BMI as a proxy for %BF in women. Similarly, black women have significantly higher %BF and BMI than white women, but the difference in %BF is relatively larger, so using BMI would understate the effect of race on obesity.

The smoking variables correlate minimally with measurement error, especially for men. This result implies that smokers tend to have a lower %BF and a similarly lower BMI, and that regression models that relate smoking to obesity will generate similar results whether using BMI or %BF. Women who take hormone replacement therapy (HRT) for longer time periods tend to have a lower BMI, but not a significantly lower %BF, so a model that uses BMI as a proxy for obesity will understate the effect of HRT on obesity. Thus, the apparent effect on obesity of this variable and any other variables correlated with it, would be spurious if the object of measurement is %BF. We find little evidence of measurement error bias for family history of type 2 diabetes or insomnia.

BMI predicts %BF much better for women than men, a result also found by Burkhauser and Cawley (2008). The R^2 is 0.69 in the measurement error equation for women and only 0.39 for men. This difference implies that many men would be classified differently by BMI than by %BF. We illustrate this point in Figure 3, which shows a scatter plot of %BF against BMI, along with the standard BMI classification cutoffs and %BF cutoffs based on predicted %BF (see Gallagher, 2000). Figure 3 shows that almost 60 percent of men would be classified in a different obesity category if BMI were used in place of %BF. Moreover, about 8 percent of men would be classified by BMI a full two obesity categories away from their %BF classification. Classification differences are smaller for women, with 45 percent misclassified by one category and about two percent misclassified by two categories.

[Figure 3. %BF vs. BMI Obesity Classification Matrix]

B. Evaluation of BMI as a Measure of Obesity for Predicting Health Outcomes

We model the risk of type 2 diabetes, cardiovascular disease (CVD), death, and death from an obesity-related cause as a function of our obesity index or BMI. In addition to information on weight history and the relative amount and location of body fat, our obesity index includes the degree of metabolic dysfunction present in an individual. Including metabolic dysfunction is important because it is entirely possible for the metabolic and hormonal disruptions precipitated by excess body fat, especially visceral abdominal fat, to manifest in people with a normal BMI. The phenomenon, referred to as metabolically obese normal weight (MONW) and first recognized in the 1980s, refers to individuals with elevated blood insulin (hyperinsulinemia) and insulin resistance (i.e., when the body requires more than the normal amount of insulin to elicit a normal metabolic response) (Ruderman *et al*., 1998). Conversely, metabolically benign obesity or MBO (i.e., individuals with BMI ≥ 30 but who do not display insulin insensitivity) also exists (Stefan *et al.*, 2008). Thus, the health effects of obesity have multiple dimensions, and both the amount of body fat and the extent of metabolic dysfunction occurring in an individual play important roles in defining and describing obesity.

We include several measures of metabolic dysfunction. The variables TRG_i, HDL_i, and BP_i are binary indicators that equal one in the event that individual *i* meets the criteria for metabolic syndrome with respect to that indicator. For example, $TRG_i = 1$ if individual *i* had blood triglycerides ≥ 150 mg/dL or received treatment for high triglycerides. $AbOB_i$ is a binary indicator that equals one if individual *i* meets the IDF criteria for central obesity, i.e., *WC* > 94cm for men and WC > 80 cm for women. We also include four other variables: (i) the quantitative insulin sensitivity check index (*QUICKI*), (ii) an interaction between the binary

indicator for high blood pressure (*BP*) and abdominal obesity (*AbOB*), (iii) an interaction between the binary indicator for high blood pressure (hypertension) and *QUICKI*, and (iv) an interaction between *QUICKI* and abdominal obesity (Katz et al., 2000; Quon, 2001). We include the interactions because there is some evidence that insulin resistance and abdominal adiposity exacerbate hypertension and insulin resistance (Pi-Sunyer, 1998). We also allow for the possibility of a non-linear relationship between current and lagged percent body fat (*%BF* and $\%BF_{t\text{-}10}$).

The vector *X* contains measures of age, age squared, family history of type 2 diabetes, smoking status, alcohol intake, relative income, and sleep patterns. Alcohol affects metabolism and nutrition in different ways depending on diet and the amount of alcohol consumed (Feinman and Lieber, 1998). We include the percentage of daily calories (kcals) from alcohol and the percentage of daily calories from alcohol squared, as we expect the relationship between alcohol consumption and health risk to be J-shaped. Relative income is measured as the ratio of annual household income to the poverty threshold, controlling for household size. We identify an individual as having a family history of type 2 diabetes if the individual indicated that a mother, father, sibling, or a grandparent was ever told by a doctor that he or she had diabetes.

We use a linear probability model framework to estimate our two competing models, as shown in Equations (21) and (22), using our obesity index versus BMI, respectively. Equation (21) incorporates quadratic terms in %BF, $\%BF_{t\text{-}10}$, with interactions between %BF and race and sex.

$$\begin{aligned}\Pr\left(D_i=1\middle|X_i,F_i\right)=&\beta_0+\beta_1 PBF_i+\beta_2\left(PBF_i\right)^2+\beta_3 PBF_i^{t-10}+\beta_4\left(PBF_i^{t-10}\right)^2\\&+\beta_5 Female_i\times PBF_i+\beta_6 Female_i\times\left(PBF_i\right)^2\\&+\beta_7 Black_i\times PBF_i+\beta_8 Black_i\times\left(PBF_i\right)^2\\&+\beta_9 WC_i+\beta_{10}TRG_i+\beta_{11}HDL_i+\beta_{12}BP_i\\&+\beta_{13}QUICKI_i+\beta_{14}QUICKI_i\times AbOB_i\\&+\beta_{15}BP_i\times QUICKI_i+\beta_{16}BP_i\times AbOB_i+\theta' X_i\end{aligned}\tag{21}$$

The alternative model uses *BMI* in place of our obesity index, i.e.,

$$\Pr\left(D_i=1\middle|X_i,F_i\right)=\beta_0+\beta_1 BMI_i^{t-10}+\beta_2 Female_i+\beta_3 Black_i+\eta' X_i+\varepsilon_i\tag{22}$$

Equation (22) is a simple linear equation with no quadratic terms and no interactions between BMI and race and sex. In either case, as health outcome measures we use type 2 diabetes, CVD, death during the follow-up period, and (given the individual died) death from an obesity-related cause.

Table 5 displays the results for type 2 diabetes and CVD and Table 6 contains the results for death and death from an obesity-related cause. Columns 2 and 6 of Tables 5 and 6 display the results from the model described by (21), and columns 3 and 7 display the results for (22). Columns 4 and 8 contain the difference in the estimated coefficients, or δ, on the explanatory variables, with the standard error of the difference in parentheses. The asterisks in these columns indicate that the coefficients are statistically and significantly different from zero at the 1 (**) percent or 5 (*) percent level. A statistically significant and positive estimate of δ implies that using BMI to predict health outcomes will result in an upward-biased estimate of the effect of the explanatory variables on the likelihood of having the health outcome. To provide a benchmark, columns 1 and 5 contain the estimated effects of the explanatory variable on health outcomes independent from a measure of obesity. We discuss the measurement error bias implied by our

results before examining differences in classification of individuals between BMI and our obesity index.

B.1. Measurement Error Bias

Compared to BMI, our obesity index predicts a significantly larger share of the variation in type 2 diabetes: the fit of the model beyond the control variables improves 334 percent when we use our obesity index (the R^2 increases from 0.05 to 0.23). The Vuong (1989) test statistic for non-nested hypotheses also indicates the model that uses our obesity index is better specified than one that includes BMI as a proxy for obesity. The coefficients on the components of the obesity index take the expected signs and may give some insight into which components are relatively important for a particular health outcome. For instance, insulin resistance (indicated by a lower QUICKI score), weight history, blood pressure, and the interaction between abdominal obesity and insulin resistance, are significant predictors of type 2 diabetes. However cholesterol, triglycerides and weight history are relatively more important for predicting CVD. These insights are foregone in models that use BMI to predict health risk.

[Table 5. BMI vs. Obesity Index Effects on Morbidity Outcomes]

When we use BMI to proxy for obesity, we find significant measurement error bias in (a) the estimated effect of aging on all four health outcomes, (b) the estimated effect of having a family history of type 2 diabetes on all four health outcomes, (c) the estimated effect of the percent of daily calories consumed in alcohol on all health outcomes except death during the follow up period, and (d) the estimated effect of income, as measured by the poverty income ratio, on all health outcomes except cardiovascular disease.

We obtain positive values of δ for age and family history of diabetes, which implies that using BMI when modeling or predicting type 2 diabetes risk leads to an upward-biased estimate of the effect of these variables on type 2 diabetes risk and a corresponding downward-biased estimate of the effect of obesity on type 2 diabetes risk. Specifically, a model using BMI would overstate by 0.03 the impact of a family history of diabetes on the probability of getting diabetes and understate by 0.03 the impact of obesity on getting diabetes. The BMI model would falsely attribute more blame for the disease to family history and less to obesity than it should. Given that 10 percent of our sample has type 2 diabetes, this prediction error of 3 percentage points is substantial.

Similarly, a model using BMI will overestimate the effect of being age 60 rather than 30 on type 2 diabetes risk by 8 percentage points ($(60-30)*[0.003]-(60-30)^2[0.00001] = 0.08$). The model substantially overstates the effect of aging on the probability of getting type 2 diabetes and understates the effect of obesity as measured by our index. This difference arises because BMI does not capture changes in body chemistry that increasingly affect the health of obese people as they age. It implies that researchers should be careful in interpreting estimated effects on obesity of variables that are correlated with age.

Table 5 also shows that a model using BMI would find more negative the effect of household income and daily alcohol intake on diabetes risk and correspondingly find less negative the effect of these variables on obesity. For instance, a model using BMI to predict type 2 diabetes risk will overestimate by 2.5 percentage points the decrease in type 2 diabetes risk associated with increasing the share of daily calories from alcohol from 0 to 15 percent. This difference arises because alcohol consumption has a miniscule effect on diabetes risk in the obesity index model and a negative coefficient in the model containing BMI.

Similarly, household income has a negative coefficient in the model with BMI and a small insignificant coefficient in the obesity index model. The δ estimate of -0.003 on the Income-Poverty ratio implies that using BMI would lead to a researcher overstating by 1 percentage point the negative effect on diabetes risk of an income four times the poverty line compared to an income at the poverty line. Correspondingly, the researcher would understate the negative effect of larger income on obesity. To look at it another way, the negative relationship between income and diabetes risk in column (1) of Table 5 disappears when we use our obesity index, but not when we use BMI to measure obesity; the measurement error in BMI is correlated with household income in a small but statistically significant way. This result is consistent with a finding in Burkhauser and Cawley (2008) that body fat correlates more strongly with employment than does fat-free mass.

Relative to BMI, our obesity index produces a small improvement in fit for CVD, although the Vuong test indicates that the improvement is statistically significant. As with type 2 diabetes risk, models using BMI to predict CVD overstate the effect of age on health and therefore understate the effect of age on obesity. Using BMI also inflates the effect of being an ex-smoker on CVD risk and correspondingly deflates the effect of being an ex-smoker on obesity-related CVD risk. For instance, a model that predicts CVD using BMI will overstate the effect of being an ex-smoker by 0.7 percentage points. On the other hand, a model that predicts CVD using BMI will overestimate the negative effect of alcohol by 3 percentage points for a 25 percentage point increase in the share of calories from alcohol.

In our obesity index, variables representing weight history, high blood pressure, insulin resistance, and interaction between insulin resistance and high blood pressure components

significantly predict death during the follow up period.[18] Our obesity index predicts death during the follow-up period only marginally better than BMI, but the Vuong tests imply the models containing our obesity index are better specified than the models using BMI. We find significant measurement error bias associated with age, family history, and the income-poverty ratio for death and for death from an obesity-related cause. We do not find significant measurement error bias associated with being a current or former smoker in models that use BMI to predict mortality and obesity-related mortality risk. The results imply that predicting mortality and death from an obesity-related cause with BMI will overstate the effect of age on mortality risk. For example, a model that uses BMI to predict mortality risk will overstate the effect of a 20-year age difference by 3 percentage points. Similarly, a model that uses BMI to predict the risk of death from an obesity-related cause will understate the effect of a 20-year age difference by 10.3 percentage points.

[Table 6. BMI vs. Obesity Index Effects on Mortality Outcomes]

B.2. Classifying Individuals Using Our Obesity Index

Because obesity is the major cause of diabetes, but one of many causes of CVD and death, the differences between BMI and our obesity index are most stark in the case of diabetes. These differences produce errors in prediction of the prevalence of disease among those who have it and those who do not. In Table 7, Panel A shows the distribution of the predicted probability of having type 2 diabetes for all individuals in the sample from the model using our obesity index (i.e., calculated using the estimated coefficients in Equation (21)). Panel B shows the corresponding distribution of the predicted probability from the model using BMI instead

[18] The negative coefficient on QUICKI implies a decrease insulin resistance, or an increase in QUICKI results in a lower risk of death. Similarly, decreasing insulin resistance when one has high blood pressure results in a decreased risk of death.

(i.e., using the estimated coefficients in Equation (22)). We calculate these probabilities at the mean of the explanatory variables (X) so as to isolate the effect of the obesity variables. The first row in Panel A (or B) of Table 7 describes the share of all non-diabetic respondents (90 percent of all respondents) who had a predicted probability of type 2 diabetes less than 10 percent, between 10 and 25 percent, between 25 and 50, or more than 50 percent using our obesity index (or BMI). The second row of Panel A (or B) of Table 7 contains the distribution of predicted type 2 diabetes risk for diagnosed diabetics (the remaining 10 percent of respondents).

Of the 90 percent of individuals in the sample who were non-diabetic, in Panel A (using our obesity index), 65.1 percent had a predicted probability of type 2 diabetes of less than 0.1, and a further 30.2 percent had a predicted probability of type 2 diabetes of between 0.1 and 0.25. In contrast, in Panel B (using BMI), 72.9 percent had a predicted probability of type 2 diabetes of less than 0.1, and a further 27.0 percent had a predicted probability of type 2 diabetes of between 0.1 and 0.25. Thus, our obesity index predicts a less than 25 percent risk of type 2 diabetes in 95.3 percent of non-diabetics whereas BMI predicts a less than 25 percent risk in 100 percent of non-diabetics. In this sense, the model based on BMI has a slightly lower propensity for false positives.

False negatives possibly generate more concern than false positives. The model based on BMI predicts less than 10 percent risk of diabetes for 59 percent of diagnosed diabetics, whereas our obesity-index model predicts such low risk for just 9 percent of diabetics. For all diabetics, the BMI model predicts less than 25 percent risk, but only 45 percent of diabetics receive this low-risk prediction from the obesity-index model. Our obesity index produces a much lower propensity for false negatives.

The superior ability of our obesity index to predict a significant type 2 diabetes risk in the diabetic is further illustrated in Figure 4. The horizontal axis displays the predicted type 2 diabetes risk using BMI and the vertical axis displays the predicted risk using our obesity index. The open circles indicate diagnosed diabetics and the crosses indicate non-diabetics. If the model using BMI could predict perfectly, then all the crosses would be at the value 0 at the left edge of the figure and all the circles would be at the value 1 on the right. Similarly, if our obesity index could predict perfectly, then all the crosses would be at the value 0 at the bottom of the figure and all the circles would be at the value 1 on the top. The observations lying above the 45 degree line have a greater predicted type 2 diabetes risk from our obesity index than using BMI. The figure shows that almost all diabetics are reported above the 45 degree line, implying our obesity index predicts a higher type 2 diabetes risk for these individuals than does BMI.

[TABLE 7. Actual Diabetes Status and Predicted Diabetes Status]

[FIGURE 4. BMI vs. the Obesity Index Predicted Risk of Type 2 Diabetes]

VII. Conclusion

In this paper we review and evaluate the use of BMI to identify the causes and predict the consequences of obesity. We use a flexible function of percent body fat and several metabolic factors to construct an obesity index, which we use to evaluate BMI. We find that economic research that uses BMI to proxy for obesity may suffer from significant measurement error bias. Models that use BMI without controlling for age, family medical history, household income, and alcohol consumption may generate significantly biased estimates of the causes and effects of obesity. We find particularly large differences in how aging correlates with BMI versus our

obesity index. Ignoring these drawbacks of BMI could lead to misguided policy if researchers attribute the health and economic effects of obesity falsely to other variables.

Our results do not imply that BMI is irrelevant as an obesity measure. Most individuals with high BMI also have high levels of body fat and face obesity-related health risks (see Figure 3). BMI is also easy to calculate because it requires only weight and height measurements. However, our obesity index suggests ways to create more refined measures of obesity. In particular, we suggest further research into the use of measures of insulin resistance, blood pressure, and waist circumference in measuring obesity.

VIII. References

Allison, D. B., K. R. Fontaine, J. E. Manson and T. B. VanItallie (1999). "Annual Deaths Attibutable to Obeisty in the United States." Journal of the American Medical Association **282**(16): 1530-1538.

Alston, J. M., D. A. Sumner and S. A. Vosti (2008). "Farm Subsidies and Obesity in the United States: National Evidence and International Comparisons." Food Policy **33**: 470-479.

American Diabetes Association (2008). "Economic Costs of Diabetes in the U.S. in 2007." Diabetes Care **31**(3): 596-615.

American Heart Association (2004). "Metabolic Syndrome - Statistics." Statistical Fact Sheet - Risk Factors. Retrieved May 3, 2010, from: http://www.americanheart.org/downloadable/heart/1136819875357META06.pdf.

Anderson, M. and D. A. Matsa "Are Restaurants Really Supersizing America?" Department of Agricultural and Resource Economics, UC Berkeley. CUDARE Working Paper No.: **1056,** from: http://repositories.cdlib.org/cgi/viewcontent.cgi?article=1232&context=are_ucb.

Boney, C. M., A. Verma, R. Tucker and B. R. Vohr (2005). "Metabolic Syndrome in Childhood: Association with Birth Weight, Maternal Obesity, and Gestational Diabetes Mellitus." Pediatrics **115**: 290-296.

Bound, J. and A. B. Krueger (1991). "The Extent of Measurement Error in Longitudinal Earnings Data: Do Two Wrongs Make a Right? ." Journal of Labor Economics, **9**(1): 1-24.

Bouret, S. G., S. J. Draper and R. B. Simerly (2004). "Trophic Action of Leptin on Hypothalamic Neurons That Regulate Feeding." Science **304**: 108-110.

Brotman, D. J., E. Walker, M. S. Lauer and R. G. o'Brien (2005). "In Search of Fewer Independent Risk Factors." Archives of Internal Medicine **165**: 138-145.

Burkhauser, R. V., John Cawley (2008). "Beyond BMI: The Value of More Accurate Measures of Fatness and Obesity in Social Science Research " Journal of Health Economics **27**: 519-529.

Calle, E. and R. Kaaks (2004). "Overweight, Obesity and Cancer: Epidemiological Evidence and Proposed Mechanisms." Nature ReviewsCancer **4**: 579-591.

Cameron, A. C. and P. K. Trivedi (2005). Microeconometrics: Methods and Applications. New York, NY, Cambridge University Press.

Chan, D., G. Watts, P. Barrett and V. Burke (2003). "Waist Circumference, Waist-to-Hip Ratio and Body Mass Index as Predictors of Adipose Tissue Compartments in Men." Quarterly Journal of Medicine **96**: 441-447.

Chen, Z., S. T. Yen and D. B. Eastwood (2005). "Effects of Food Stamp Participation on Body Weight and Obesity." American Journal of Agricultural Economics **87**(5): 1167-1173.

Chinn, S. (2006). "Definitions of Childhood Obesity: Current Practice." European Journal of Clinical Nutrition **60**: 1189-1195.

Chou, S. Y., M. Grossman and H. Saffer (2004). "An Economic Analysis of Adult Obesity: Results from the Behavioral Risk Factor Surveillance System." Journal of Health Economics **23**: 565-587.

Chumlea, W. C., S. S. Guo, R. J. Kuczmarski, K. M. Flegal and C. L. Johnson (2002). "Body Composition Estimates from NHANES III Bioelectrical Impedance Data." International Journal of Obesity **26**: 1596-1609.

Colditz, G. A. (1992). "Economic Costs of Obesity." American Journal of Clinical Nutrition **55**: 503s-507s.

Currie, J., S. D. Vigna, E. Moretti and V. Pathina (April 2009). "The Effect of Fast Food Restaurants on Obesity." National Bureau of Economic Research. NBER Working Paper Series: **No. 14721**.

Cutler, D. M., Edward L. Glaeser, Jesse M. Shapiro (2003). "Why Have Americans Become More Obese?" Journal of Economic Perspectives **17**(3): 93-117.

Daniels, S. (2006). "The Consequences of Childhood Overweight and Obesity." The Future of Children, Childhood Obesity **16**(1): 47-67.

Deckelbaum, R. J. and C. L. Williams (2001). "Childhood Obesity: The Health Issue." Obesity Research **9**: 239s-243s.

Despres, J., S. Moorjani, P. Lupien, A. Tremblay, A. Nadeau and C. Bouchard (1990). "Regional Distribution of Body Fat, Plasma Lipoproteins, and Cardiovascular Disease." Arteriosclerosis, Thrombosis, and Vascular Biology **10**(4): 497-511.

Deurenberg, P. (2001). "Universal Cut-Off BMI Points for Obesity Are Not Appropriate." British Journal of Nutrition **85**: 136-136.

Dunn, R. A. (March 31, 2008). "Obesity and the Availability of of Fast-food: an instrumental variables approach." University of Wisconsin. from: http://ssrn.com/abstract=989363.

Ebbeling, C. B., D. B. Pawlak and D. S. Ludwig (2002). "Childhood Obesity: Public Health Crisis, Common Sense Cure." The Lancet **360**: 473-481.

Eknoyan, G. (2008). "Adolphe Quetelet (1796–1874)—The Average Man and Indices of Obesity." Nephrology Dialysis Transplantation **23**: 47-51.

Feinman, L. and C. S. Lieber (1998). Nutrition in Diet and Alcoholism. Modern Nutrition in Health and Disease. M. E. Shils, J. A. Olson, M. Shike and A. C. Ross. Baltimore, Maryland, Williams & Wilkins**:** 1395-1418.

Finkelstein, E. A., J. G. Trogdon, J. W. Cohen and W. Dietz (2009). "Annual Medical Spending Attributable to Obesity: Payer-and Service-Specific Estimates." Health Affairs: w822-w831.

Flegal, K. M., B. I. Graubard, D. F. WIlliamson and M. H. Gail (2007). "Cause-Specific Excess Deaths Associated with Underweight, Overweight, and Obesity " Journal of the American Medical Association **298**(17): 2028-2037.

Fontaine, K. R., D. T. Redden, C. Wang, A. O. Westfall and D. B. Allison (2003). "Years of Life Lost Due to Obesity." Journal of the American Medical Association **289**(2): 187-193.

Forbes, G. B. (1998). Body Composition: Influence of Nutrition, Physical Activity, Growth, and Aging. Modern Nutrition in Health and Disease. M. E. Shils, J. A. Olson, M. Shike and A. C. Ross. Baltimore, Maryland, Williams & Wilkins**:** 789-809.

Frankenfield, D. C., William A. Rowe, Robert N. Cooney, J. Stanley Smith, and Dolores Becker (2001). "Limits of Body mass Index to Detect Obesity and Predict Body Composition." Nutrition **17**: 26-29.

Freedman, D. S., Geraldine Perry (2000). "Body Composition and Health Status Among Children and Adolescents." Preventive Medicine **31**: s34-s52.

Fukagawa, N. K., L. G. Bandini and J. B. Young (1990). "Effect of Age on Body Composition and Resting Metabolic Rate." American Journal of Physiology Endocrinology and Metabolism **259**(2): E233-E238.

Fuller, W. (1987). Measurement Error Models. Wiley.

Gallagher, D., Steven B. Heymsfield, Moonseong Heo, Susan A. Jebb, Peter R. Murgatroyd, and Yoichi Sakamoto (2000). "Healthy Percentage BodyFat Ranges: An Approach for Developing Guidelines Based on Body Mass Index." American Journal of Clinical Nutrition **72**: 694-701.

Gallagher, D., M. Visser, D. Sepulveda, R. N. Pierson, T. Harris and S. B. Heymsfield (1996). "How Useful is Body Mass Index for Comparison of Body Fatness Across Age, Sex, and Ethnic Groups?" American Journal of Epidemiology **143**(3): 228-239.

Garn, S. M., W. R. Leonard and V. M. Hawthorne (1986). "Three Limitations of the Body Mass Index." American Journal of Clinical Nutrition **44**: 996-997.

Gelber, R. P., J. M. Gaziano, J. Orav, J. E. Manson, J. E. Buring and T. Kurth (2008). "Measures of Obesity and Cardiovascular Risk Among Men and Women." Journal of the American College of Cardiology **52**(8): 605-615.

Gibson, D. (2003). "Food Stamp Program Participation is Positively Related to Obesity in Low Income Women." Journal of Nutrition **133**: 2225-2231.

Guo, S. S., C. Zeller, W. C. Chumlea and R. M. Siervogel (1999). "Aging, Body Composition, and Lifestyle: the Fels Longitudinal Study." American Journal of Clinical Nutrition **70**: 405-411.

Harvard Health Newsletters (2006). "Abdominal Fat and What to do About it." Harvard Women's Health Watch. Retrieved April 23, 2009, from: https://www.health.harvard.edu/newsweek/Abdominal-fat-and-what-to-do-about-it.htm.

Huang, E. S., A. Basu, M. O'Grady and J. C. Capretta (2009). "Projecting the Future Diabetes Population Size and Related Costs for the U.S." Diabetes Care **32**(12): 2225-2229.

International Diabetes Federation "The IDF Concensus Worldwide Definition of the Metabolic Symdrome." from: http://www.idf.org/webdata/docs/IDF_Meta_def_final.pdf.

Janssen, I., P. T. Katzmarzyk and R. Ross (2004). "Waist Circumference and not Body Mass Index Explains Obesity-related Health Risk." American Journal of Clinical Nutrition **79**: 379-384.

Jia, H. and E. I. Lubetkin (2010). "Trends in Quality-Adjusted Life-Years Lost Contributed by Smoking and Obesity." American Journal of Preventive Medicine **38**(2): 138-144.

Johansson, E., P. Bökerman, U. Kiiskinen and M. Heliövaara (2009). "Obesity and Labor Market Success in Finland: The Difference Between Having a High BMI and Being Fat." Economics and Human Biology **7**: 36-45.

Kannel, W. B., R. B. D'Agostino and J. L. Cobb (1996). "Effect of Weight on Cardiovascular Disease." American Journal of Clinical Nutrition **63**: 419S-422S.

Katz, A., S. S. Nambi, K. Mather, A. D. Baron, D. A. Follman, G. Sullivan and M. J. Quon (2000). "Quantitative Insulin Sensitivity Check Index: A Simple, Accurate Method for Assessing Insulin Sensitivity In Humans." The Journal of Clinical Endocrinology & Metabolism **85**(7): 2402-2410.

Kershaw, E. E. and J. S. Flier (2004). "Adipose Tissue as an Endocrine Organ." The Journal of Clinical Endocrinology & Metabolism **89**(6): 2548-2556.

Keys, A., F. Fidanza, M. J. Karvonen, N. Kimura and H. L. Taylor (1972). "Indicaes of Relative Weight and Obesity." Journal of Chronic Disease **25**(6-7): 329-343.

King, J. C. (2006). "Maternal Obesity, Metabolism, and Pregnancy Health Outcomes " Annual Reviews Nutrition **26**: 271-291.

Kyle, U. G., I. Bosaeus, A. D. D. Lorenzo, P. Deurenberg, M. Elia and J. M. Gomez (2004). "Bioelectrical impedance analysisFpart I: review of principles and methods." Clinical Nutrition **23**(5): 1226-1243.

Kyle, U. G., L. Genton, D. Hans, L. Karsegard, D. Slosman and C. Prichard (2001). "Age-Related Differences in Fat-Free Mass, Skeletal Muscle, Body Cell Mass, and Fat Mass Between 18 and 94 Years." European Journal of Clinical Nutrition **55**: 663-672.

Lakdawalla, D. and T. Philipson (May 2002). "The Growth of Obesity and Technological Change: A Theoretical and Empirical Examination." NBER Working Paper Series. **No. 8946**.

Litwin, S. E. (2008). "Which Measures of Obesity Best Predict Cardiovascular Risk?" Journal of the American College of Cardiology **52**(8): 616-619.

McGanity, W. J., E. B. Dawson and J. W. V. Hook (1998). Maternal Nutrition. Modern Nutrition in Health and Disease. M. E. Shils, J. A. Olson, M. Shike and A. C. Ross. Baltimore, Maryland, Williams & Wilkins**:** 811-838.

Meyerhoefer, C. D. and Y. Pylypchuk (2008). "Does Participation in the Food Stamp Program Increase the Prevalence of Obesity and Health Care Spending? ." American Journal of Agricultural Economics **90**(2): 287-305.

Miller, S. L. and R. R. Wolfe (2008). "The Danger of Weight Loss in the Elderly." The Journal of Nutrition, Health, and Aging **12**(7): 487-491.

Mokdad, A., M. K. Serdula, W. H. Dietz, B. A. Bowman, J. S. MArks and J. P. Koplan (1999). "The Spread of the Obesity Epidemic in the United States, 1991-1998." Journal of the American Medical Association **282**(16): 1519-1522.

Must, A., J. Spadano, E. H. Coakley, A. E. Field, G. Colditz and W. H. Dietz (1999). "The Disease Burden Associated with Overweight and Obesity." Journal of the American Medical Association **282**(16): 1523-1529.

National Cancer Institute (March 2004). "Obesity and Cancer: Questions and Answers." Retrieved April 20, 2010, from: http://www.cancer.gov/images/documents/0ed3af8f-0d8d-41aa-8613-acf53a1070f3/Fs3_70.pdf.

National Diabetes Information Clearinghouse (2008). "Diagnosis of Diabetes." NIH Publication No. 09–4642. Retrieved May 24, 2010, from: http://diabetes.niddk.nih.gov/dm/pubs/diagnosis/#diagnosis.

Nonnemaker, J., E. Finkelstein, M. Engelen, T. Hoerger and M. Farrelly (2009). "Have Efforts to Reduce Smoking Really Contributed to the Obesity Epidemic?" Economic Inquiry **47**(2): 366-376.

Okosun, I. S., K. M. D. Chandra, J. M. Boltri and D. C. Parish (2004). "Abdominal Adiposity in U.S. Adults: Prevalence and Trends, 1960-2000." Preventive Medicine **39**: 197-206.

Pi-Sunyer, F. X. (1998). Obesity. Modern Nutrition in Health and Disease. M. E. Shils, J. A. Olson, M. Shike and A. C. Ross. Baltimore, Maryland, Williams & Wilkins**:** 1395-1418.

Plagemann, A. (2008). "A matter of Insulin: Developmental Programming of Body Weight Regulation." The Journal of Maternal-Fetal & Neonatal Medicine **21**(3): 143-148.

Poehlman, E. T., M. J. Toth and A. W. Gardner (1995). "Changes in Energy Balance and Body Composition at Menopause: A Controlled Longitudinal Study." Annals of Internal Medicine **123**(9): 673-675.

Poirier, P., T. D. Giles, G. A. Bray, Y. Hong, J. S. Stern, F. X. Pi-Sunyer and R. H. Eckel (2006). "Obesity and Cardiovascular Disease: Pathophysiology, Evaluation, and Effect of Weight Loss: An Update of the 1997 American Heart Association Scientific Statement on Obesity and Cardiovascular Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism." Circulation **113**: 898-918.

Prentice, A. M., S.A. Jebb (2001). "Beyond Body Mass Index." The International Association for the Study of Obesity, Obesity Reviews **2**: 141-146.

Prospective Studies Collaboration (2009). "Body-Mass Index and Cause Specific Mortality in 900,000 Adults: Collaborative Analyses of 57 Prospective Studies." The Lancet **373**(9669): 1083-1096.

Quon, M. J. (2001). "Editorial: Limitations of the Fasting Glucose to Insulin Ratio as an Index of Insulin Sensitivity." The Journal of Clinical Endocrinology & Metabolism **86**(10): 4615-4617.

Rashad, I., M. Grossman and S. Y. Chou (2005). "The Super Size of America: An Economic Estimation of Body Mass Index and Obesity in Adults." NBER Working Paper No. 11584.

Rowland, M. L. (1990). "Self-Reported Weight and Height." American Journal of Clinical Nutrition **52**: 1125-1133.

Ruderman, N., D Chisholm, X. Pi-Sunyer and S. Schneider (1998). "The Metabolically Obese, Normal-Weight Individual Revisited." Diabetes **47**: 699-713.

Smalley, K. J., A. N. Knerr, Z. V. Kendrick, J. A. Colliver and O. E. Owen (1990). "Reassessnt of Body Mass Indices." American Journal of Clinical Nutrition **52**: 405-408.

Speakman, J. R. (2007). "A Nonadaptive Scenario Explaining the Genetic Predisposition to Obesity: The ‘‘Predation Release’’ Hypothesis." Cell Metabolism **6**: 5-12.

Speiser, P. W., M. C. J. Rudolf, H. Anhalt, C. Camacho-Hubner, F. Chiarelli, A. Eliakim and M. Freemark (2005). "Consensus Statement: Childhood Obesity." The Journal of Clinical Endocrinology & Metabolism **90**(3): 1871-1887.

Stefan, N., K. Kantartzis, J. Machann, F. Schick and C. Thamer (2008). "Identification and Characterization of Metabolically Benign Obesity in Humans." Achives of Internal Medicine **168**(15): 1609-1616.

Sun, S. S., W. C. Chumlea, S. B. Heymsfield, H. C. Lukaski, D. Schoeller, K. Friedl, R. J. Kuczmarski, K. M. Flegal, C. L. Johnson and V. S. Hubbard (2003). "Development of Vioelectrical Impedance Analysis Prediction Equations for Body Composition With the Use of a Multicomponent Model for Use in Epidemiologic Surveys." American Journal of Clinical Nutrition **77**: 331-340.

Tataranni, P., M. Monroe, C. Dueck, S. Traub, M. Nicholson, M. Manore, K. Matt and E. Ravussin (1997). "Adiposity, Plasma Leptin Concentration and ReproductiveFunction in Active and Sedentary Females." International Journal of Obesity **21**: 818-821.

Thompson, D., J. Edelsberg, G. A. Colditz, A. P. Bird and G. Oster (1999). "Lifetime Health and Economic Consequences of Obesity." Achives of Internal Medicine **159**: 2177-2183.

Townsend, M. S., J. Peerson, B. Love and C. Achterberg (2001). "Food Insecurity is Positively Related to Overweight in Women." Journal of Nutrition **131**: 1738-1745.

U.S. Department of Health and Human Services (2001). The Surgeon General’s Call to Action to Prevent and Decrease Overweight and Obesity 2001. Rockville, MD, U.S. Department of Health and Human Services, U.S. Public Health Service, Office of the Surgeon General**:** 39.

VanGaal, L. F., I. L. Mertens and C. E. DeBlock (2006). "Mechanisms linking obesity with cardiovascular disease." Nature **444**: 875-880.

Vuong, Q.H. (1989). “Likelihood Ratio Tests for Model Selection and Non-Nested Hypotheses.” Econometrica, **57**: 307-333.

Wada, R. E. Tekin (2010). “Body Composition and Wages.” Economics & Human Biology In Press.

Wang, Y., E. B. Rimm, M. J. Stampfer, W. C. Willet and F. B. Hu (2005). "Comparison of Abdominal Adiposity and Overall Obesity in Predicting Risk of Type 2 Diabetes Among Men." American Journal of Clinical Nutrition **81**(3): 555-563.

Weiss, R. and S. Caprio (2005). "The Metabolic Consequences of Childhood Obesity." Best Practices & Research Clinical Endocrinology & Metabolism **19**(3): 405-419.

Westat Inc. (1996). National Health and Nutrition Examination Survey: Weighting and Estimation Methodology, CDC, National Center for Health Statistics**:** 35.

WHO (1995). Physical Status: The Use and Interpretation of Anthropometry. Geneva, Expert Committe on Physical Status, WHO**:** 450.

Wolf, A. M. and G. A. Colditz (1998). "Current Estimates of the Economic Cost of Obesity in the United States." Obesity Research **6**(2): 97-106.

Zamboni, Z., G. Mazzali, E. Zoico, T. B. Harris, J. B. Meigs, V. D. Francesco, F. Fantin, L. Bissoli and O. Bosello (2005). "Health Consequences of Obesity in the Elderly: A Review of Four Unresolved Questions." International Journal of Obesity **29**: 1011-1029.

Zhu, S., Z. Wang, S. Heshka, M. Heo, M. S. Faith and S. B. Heymsfield (2002). "Waist Circumference and Obesity-Associated Risk Factors Among Whites in the Third National Health and Nutrition Examination Survey: Clinical Action Thresholds." American Journal of Clinical Nutrition **76**(4): 743-749.

IX. Tables and Figures

Table1. Alternative Adult Weight Classification Systems and Cutoffs

	Underweight	Normal	Overweight	Obese
BMI (kg/m^2)	<18.5	18.5-25	25-30	≥30
%BF[a] (%)				
Male				
20-39 years	<8%	8-20%	20-25%	≥25%
40-59 years	<11%	11-22%	22-28%	≥28%
60-79 years	<13%	13-25%	25-30%	≥30%
Female				
20-39 years	<21%	21-33%	33-39%	≥39%
40-59 years	<23%	23-34%	34-40%	≥40%
60-79 years	<24%	24-36%	36-42%	≥42%

WC[b] (cm)	Not Overweight	Overweight	Obese
Male	<94 cm	94-102 cm	≥102 cm
Female	<80 cm	80-88 cm	≥88 cm

%BF[c]	Not Obese	Obese
Male	<25%	≥25%
Female	<30%	≥30%
WC[d] (cm)		
Male	<90 cm	≥90 cm
Female	<84 cm	≥84 cm

c: These are the NIH and NIDDK recommended %BF cutoffs.

a: These are the %BF cutoffs suggested in Gallagher et al. (2000), based on BMI cutoffs.

b: These are the WC cutoffs used by the American Heart Association.

d: These are the WC cutoffs suggested in Zhu et al. (2002), which only distinguish between obese and non-obese.

Table 2. Prevalence of Obesity by Weight Classification System (percentages)

	Percent			
	Underweight	Normal	Overweight	Obese
BMI				
Male	4.80	37.51	38.92	18.77
Female	11.54	38.95	25.52	24.00
%BF[b]				
Male	1.31	31.35	34.60	32.74
Female	4.47	36.30	26.36	32.87
WC[c]	Not Overweight		Overweight	Obese
Male	50.11		22.79	27.11
Female	34.39		20.24	45.36
%BF[a]	Not Obese			Obese
Male	55.09			44.91
Female	24.81			75.19
WC[d]				
Male	37.54			62.46
Female	41.63			58.37

1: These are based on the NIH and NIDDK recommended %BF cutoffs, which only distinguish between obese and non-obese.

2: The %BF cutoffs suggested in Gallagher et al. (2000), based on BMI cutoffs.

3: The WC cutoffs used by the American Heart Association.

4: The WC cutoffs suggested in Zhu et al. (2002), which only distinguish between two weight categories: obese and non-obese.

Notes: All frequencies were calculated using STATA "svy" command for individuals 18 years of age and older for whom a waist circumference measurement was taken. A total of 7,739 men and 8,709 women from the NHANES III adult survey data file satisfied these criteria.

Table 3. BMI, Percent Body Fat, and Type 2 Diabetes Summary Statistics

	Panel A: Women					
	White			*Black*		
	18-30 years	*30-60 years*	*Over 60 years*	*18-30 years*	*30-60 years*	*Over 60 years*
Type 2 Diabetes Prevalence (%)	0.2	3.2	10.7	0.4	5.1	21.3
Percent Body Fat (%)	31.4	35.2	36.3	35.0	38.8	38.7
BMI (kg/m^2)	23.8	26.7	26.8	25.8	29.5	29.2
No. of observations	1,111	2,402	1,854	657	1,249	472
	Panel B: Men					
	White			*Black*		
	18-30 years	*30-60 years*	*Over 60 years*	*18-30 years*	*30-60 years*	*Over 60 years*
Type 2 Diabetes Prevalence (%)	0.4	4.1	11.4	0.7	4.6	15.4
Percent Body Fat (%)	21.7	24.4	25.5	22.9	24.3	24.3
BMI (kg/m^2)	24.9	27.1	27.0	25.7	26.6	26.3
No. of observations	1,178	2,193	1,779	602	1,017	457

Note: Those with type 2 diabetes are identified using the method outlined in Thompson *et al.* (1999).
Source: NHANES III.

Table 4. Measurement Error Regressions: BMI as a Proxy for Percent Body Fat

	All Adults			*Women*			*Men*		
Dependent Variable:	%BF	BMI	%BF	%BF	BMI	%BF	%BF	BMI	%BF
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Regressors									
BMI			0.89**			0.94**			0.77**
			(0.02)			(0.03)			(0.03)
Female	11.28**	-0.19	11.45**						
	(0.19)	(0.16)	(0.16)						
Age	0.54**	0.36**	0.23**	0.55**	0.37**	0.21**	0.29**	0.24**	0.11
	(0.06)	(0.05)	(0.05)	(0.09)	(0.08)	(0.07)	(0.08)	(0.07)	(0.08)
Age^2	-0.0045**	-0.00**	-0.0017**	-0.0049**	-0.00**	-0.0017**	-0.0024**	-0.00**	-0.0006
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Black	2.06**	1.56**	0.67**	3.33**	2.69**	0.80**	0.30	-0.02	0.31
	(0.29)	(0.21)	(0.15)	(0.35)	(0.31)	(0.22)	(0.33)	(0.23)	(0.23)
Current Smoker	-1.43**	-1.15**	-0.41*	-1.82**	-1.33**	-0.57*	-1.21**	-1.03**	-0.41
	(0.24)	(0.16)	(0.21)	(0.34)	(0.23)	(0.26)	(0.32)	(0.27)	(0.32)
Ex-Smoker	0.68**	0.65**	0.11	0.78*	0.67	0.15	0.67**	0.74**	0.01
	(0.19)	(0.18)	(0.18)	(0.31)	(0.36)	(0.25)	(0.24)	(0.24)	(0.28)
Alcohol Calorie Share	-0.04	-0.09**	0.03	-0.19**	-0.18**	-0.01	-0.01	-0.06*	0.03
	(0.03)	(0.02)	(0.03)	(0.05)	(0.04)	(0.03)	(0.03)	(0.02)	(0.03)
$(\text{Alcohol Calorie Share})^2$	0.0003	0.00	-0.0009	0.0065**	0.01**	0.0015	-0.0007	0.00	-0.0012
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Menopause				2.28**	1.77**	0.63*			
				(0.42)	(0.37)	(0.31)			
< 5 Yrs HRT				-0.57	-1.00*	0.37			
				(0.43)	(0.39)	(0.27)			
> 5 Yrs HRT				-0.35	-1.35**	0.91**			
				(0.46)	(0.39)	(0.29)			
Family History of Type 2 Diabetes	1.11**	1.24**	0.01	1.43**	1.53**	-0.0074	0.63*	0.83**	-0.01
	(0.20)	(0.14)	(0.15)	(0.22)	(0.19)	(0.19)	(0.27)	(0.18)	(0.24)
Insomnia Spells	1.74*	1.19**	0.69	2.03	1.95**	0.20	0.85	-0.07	0.91
	(0.71)	(0.43)	(0.49)	(1.02)	(0.68)	(0.64)	(0.79)	(0.53)	(0.68)
Hypersomnia Spells	-0.88	-0.18	-0.72	-0.43	0.32	-0.73	-0.61	-0.98	0.14
	(1.12)	(0.83)	(0.73)	(1.38)	(1.02)	(0.82)	(0.99)	(1.25)	(1.22)
Constant	8.93**	17.32**	-6.41**	19.70**	16.36**	4.36*	16.39**	21.28**	0.03
	(1.83)	(1.54)	(1.45)	(2.72)	(2.19)	(2.08)	(2.28)	(2.17)	(2.20)
Observations	7,826	7,826	7,826	4,012	4,012	4,012	3,814	3,814	3,814
R^2	0.45	0.05	0.76	0.08	0.07	0.69	0.03	0.05	0.39
Null F-Stat			21.91			18.63			7.67
Null p-value			0.00			0.00			0.00

Notes: Standard errors in parentheses, **p<0.01, *p<0.05. HRT=hormone replacement therapy (estrogen).

Table 5. BMI vs. Obesity Index Effects on Type 2 Diabetes and CVD

	Type 2 Diabetes				*Cardiovascular Disease*			
	(1)	(2)	(3)	δ=(3)-(2)	(5)	(6)	(7)	δ=(7)-(6)
BMI			0.006**				0.003**	
			(0.001)				(0.001)	
%BF		-0.006				0.001		
		(0.004)				(0.011)		
$\%BF^2$		-0.00003				0.00001		
		(0.000)				(0.000)		
%BFt-10		0.001				-0.004*		
		(0.001)				(0.001)		
(%BFt-10)2		0.00007**				0.00006*		
		(0.000)				(0.000)		
Female*%BF		0.005				-0.002		
		(0.005)				(0.011)		
Female*$(\%BF)^2$		-0.00009				0.00000		
		(0.000)				(0.000)		
Black*%BF		-0.007				-0.007		
		(0.004)				(0.005)		
Black*$(\%BF)^2$		0.00011				0.00011		
		(0.000)				(0.000)		
Waist Circumference		0.001*				-0.001		
Criteria for Metabolic Syndrome Dummies:		(0.001)				(0.001)		
Triglycerides		-0.012*				0.026*		
		(0.005)				(0.010)		
HDL Cholesterol		0.001				0.046**		
		(0.007)				(0.006)		
Blood Pressure		0.625**				0.492**		
		(0.122)				(0.102)		
QUICKI		-5.450**				0.162		
		(0.485)				(0.454)		
(Blood Pressure)*(QUICKI)		-4.025**				-3.074**		
		(0.773)				(0.645)		
(Abdominal Obesity)*(QUICKI)		-0.143*				0.049		
		(0.062)				(0.093)		
(Blood Pressure)*(Abdominal Obesity)		-0.038**				-0.032		
		(0.012)				(0.019)		

Table 5 (continued). BMI vs. Obesity Index Effects on Type 2 Diabetes and CVD

	Type 2 Diabetes				*Cardiovascular Disease*			
	(1)	(2)	(3)	δ=(3)-(2)	(5)	(6)	(7)	δ=(7)-(6)
Female	-0.022**	-0.001	-0.021*	-0.020	-0.043**	0.005	-0.042**	-0.048
	(0.006)	(0.079)	(0.009)	(0.100)	(0.010)	(0.142)	(0.010)	(0.143)
Black	0.031**	0.095	0.023*	-0.072	-0.010	0.087	-0.014	-0.101
	(0.008)	(0.062)	(0.009)	(0.071)	(0.009)	(0.079)	(0.011)	(0.093)
Age	0.009**	0.004*	0.007**	0.003**	0.007**	0.004	0.006	0.002**
	(0.002)	(0.002)	(0.003)	(0.001)	(0.002)	(0.002)	(0.003)	(0.001)
Age^2	-0.00006**	-0.00002	-0.00003	-0.00001	-0.00002	0.00001	-0.00001	-0.00002**
	(0.000)	(0.000)	(0.000)	(0.00000)	(0.000)	(0.000)	(0.000)	(0.00001)
Current Smoker	-0.009	-0.004	-0.002	0.002	0.041**	0.040**	0.045**	0.005
	(0.008)	(0.008)	(0.011)	(0.005)	(0.009)	(0.008)	(0.013)	(0.003)
Former Smoker	0.018*	0.001	0.014	0.006	0.040**	0.031**	0.038**	0.007**
	(0.007)	(0.007)	(0.010)	(0.004)	(0.009)	(0.009)	(0.012)	(0.002)
Alcohol Calorie Share	-0.002**	-0.000	-0.002*	-0.002**	-0.001	-0.000	-0.001	-0.001**
	(0.001)	(0.001)	(0.001)	(0.000)	(0.001)	(0.001)	(0.001)	(0.000)
$(\text{Alcohol Calorie Share})^2$	0.00003*	0.00001	0.00002	-0.00167	-0.00001	-0.00002	-0.00001	-0.00100
	(0.000)	(0.000)	(0.000)	(0.00042)	(0.000)	(0.000)	(0.000)	(0.00031)
Family History Type 2 Diabetes	0.078**	0.048**	0.071**	0.023**	0.034**	0.021*	0.030**	0.009**
	(0.010)	(0.008)	(0.008)	(0.004)	(0.009)	(0.009)	(0.011)	(0.002)
Income-Poverty Ratio	-0.004**	0.00001	-0.003	-0.003**	-0.015**	-0.014**	-0.015**	-0.001
	(0.001)	(0.002)	(0.002)	(0.001)	(0.002)	(0.002)	(0.003)	(0.000)
Insomnia Spells	0.056**	0.050**	0.050	0.000	0.058	0.051	0.055	0.004
	(0.020)	(0.017)	(0.040)	--	(0.029)	(0.029)	(0.037)	(0.005)
Hypersomnia Spells	-0.007	-0.007	-0.006	0.002	0.071	0.073	0.071	-0.002
	(0.019)	(0.023)	(0.028)	(0.016)	(0.050)	(0.048)	(0.059)	(0.005)
Constant	-0.277**	0.684**	-0.381**		-0.166**	-0.083	-0.219*	
	(0.052)	(0.125)	(0.075)		(0.059)	(0.175)	(0.086)	
Observations	7,826	7,826	7,826		7,826	7,826	7,826	
R-squared	0.053	0.230	0.069		0.058	0.080	0.060	
Ho: Equivalence of Xs in Obesity Index and BMI								
F-Stat				9.595				4.051
p-value				0.000				0.000
Ho: Obesity Index model better specified than BMI model								
Vuong test statistics			16.601				5.859	
p-value			0.000				0.000	

Notes: Standard errors in parentheses, ** p<0.01, * p<0.05

Table 6. BMI vs. Obesity Index Effects on Death and Death from Obesity-Related Cause

	Death				*Death from an Obesity-Related COD*			
	(1)	(2)	(3)	δ=(3)-(2)	(5)	(6)	(7)	δ=(7)-(6)
BMI			0.002*				0.009**	
			(0.001)				(0.003)	
%BF		-0.004				0.016		
		(0.007)				(0.019)		
$\%BF^2$		0.00010				-0.00029		
		(0.000)				(0.000)		
%BFt-10		-0.001				-0.003		
		(0.001)				(0.003)		
(%BFt-10)2		0.00006**				0.00007		
		(0.000)				(0.000)		
Female*%BF		-0.006				-0.005		
		(0.009)				(0.023)		
Female*$(\%BF)^2$		0.00003				0.00013		
		(0.000)				(0.000)		
Black*%BF		-0.009				0.001		
		(0.006)				(0.012)		
Black*$(\%BF)^2$		0.00010				0.00004		
		(0.000)				(0.000)		
Waist Circumference		0.000				0.003		
Criteria for Metabolic Syndrome Dummies:		(0.001)				(0.002)		
Triglycerides		-0.016				0.04724		
		(0.009)				(0.031)		
HDL Cholesterol		-0.004				0.069*		
		(0.011)				(0.026)		
Blood Pressure		0.371**				0.321		
		(0.123)				(0.295)		
QUICKI		-0.731*				0.704		
		(0.360)				(1.427)		
(Blood Pressure)*(QUICKI)		-2.148**				-1.174		
		(0.754)				(1.973)		
(Abdominal Obesity)*(QUICKI)		-0.176				0.099		
		(0.090)				(0.405)		
(Blood Pressure)*(Abdominal Obesity)		-0.010				-0.107		
		(0.022)				(0.058)		

Table 6 (cont). BMI vs. Obesity Index Effects on Death and Death from Obesity-Related Cause

	Death				*Death from an Obesity-Related COD*			
	(1)	(2)	(3)	δ=(3)-(2)	(5)	(6)	(7)	δ=(7)-(6)
Female	-0.053**	0.090	-0.052**	-0.142	0.041	0.093	0.038	-0.054
	(0.009)	(0.123)	(0.010)	(0.149)	(0.030)	(0.339)	(0.030)	(0.122)
Black	0.034**	0.205*	0.031**	-0.174	0.078**	0.030	0.077*	0.047
	(0.009)	(0.091)	(0.011)	(0.103)	(0.027)	(0.190)	(0.030)	(0.117)
Age	-0.030**	-0.032**	-0.031**	0.001*	-0.003	-0.010	-0.005	0.005**
	(0.002)	(0.003)	(0.003)	(0.001)	(0.008)	(0.008)	(0.011)	(0.002)
Age^2	0.00041**	0.00042**	0.00042**	0.00000	0.00005	0.00010	0.00007	-0.00003
	(0.000)	(0.000)	(0.000)	(0.00000)	(0.000)	(0.000)	(0.000)	(0.00001)
Current Smoker	0.134**	0.135**	0.137**	0.001	-0.119**	-0.109**	-0.096*	0.012
	(0.011)	(0.010)	(0.014)	(0.003)	(0.036)	(0.034)	(0.041)	(0.007)
Former Smoker	0.045**	0.042**	0.044**	0.002	-0.05422	-0.066*	-0.055	0.011
	(0.011)	(0.011)	(0.011)	(0.002)	(0.031)	(0.031)	(0.034)	(0.007)
Alcohol Calorie Share	-0.001	-0.000	-0.001	0.000	-0.006	-0.005	-0.006	-0.001*
	(0.001)	(0.001)	(0.001)	(0.000)	(0.004)	(0.003)	(0.005)	(0.000)
$(\text{Alcohol Calorie Share})^2$	0.00001	-0.00000	0.00000	0.00000	0.00018	0.00016	0.00018	-0.00100
	(0.000)	(0.000)	(0.000)	(0.00026)	(0.000)	(0.000)	(0.000)	(0.00050)
Family History Type 2 Diabetes	0.007	-0.003	0.004	0.007**	0.063*	0.043	0.054	0.011**
	(0.008)	(0.009)	(0.010)	(0.002)	(0.025)	(0.024)	(0.029)	(0.003)
Income-Poverty Ratio	-0.01991**	-0.01776**	-0.01942**	-0.002**	0.007	0.009	0.008	-0.001**
	(0.002)	(0.002)	(0.003)	(0.000)	(0.006)	(0.006)	(0.008)	(0.000)
Insomnia Spells	0.009	0.006	0.006	0.000	-0.337**	-0.309**	-0.322**	-0.015
	(0.015)	(0.016)	(0.019)	--	(0.074)	(0.091)	(0.085)	(0.033)
Hypersomnia Spells	-0.018	-0.019	-0.018	0.001	0.021	0.002	0.015	0.013
	(0.012)	(0.013)	(0.016)	(0.005)	(0.218)	(0.182)	(0.217)	(0.015)
Constant	0.606**	0.796**	0.570**		0.479	0.018	0.262	
	(0.064)	(0.111)	(0.080)		(0.262)	(0.451)	(0.359)	
Observations	7,826	7,826	7,826		2,536	2,536	2,536	
R-squared	0.357	0.369	0.357		0.036	0.060	0.044	
Ho: Equivalence of Xs in Obesity Index and BMI								
F-Stat				3.423				1.369
p-value				0.000				0.173
Ho: PSA Obesity Index model better specified than BMI model								
Vuong test statistics			6.168				3.127	
p-value			0.000				0.002	

Standard errors in parentheses, ** p<0.01, * p<0.05

Table 7. Diabetes Status and Predicted Diabetes Status: Obesity Index versus BMI

	Predicted Probability				
	$p < 0.1$	$0.1 \leq p < 0.25$	$0.25 \leq p < 0.5$	$p \geq 0.5$	
	Panel A: Our Obesity Index				
					Row Total
Non-Diabetic	0.651	0.302	0.047	0.001	0.900
Diabetic	0.090	0.360	0.480	0.070	0.100
		Panel B: Body Mass Index			Row Total
Non-Diabetic	0.729	0.270	0.000	0.000	0.900
Diabetic	0.590	0.410	0.000	0.000	0.100

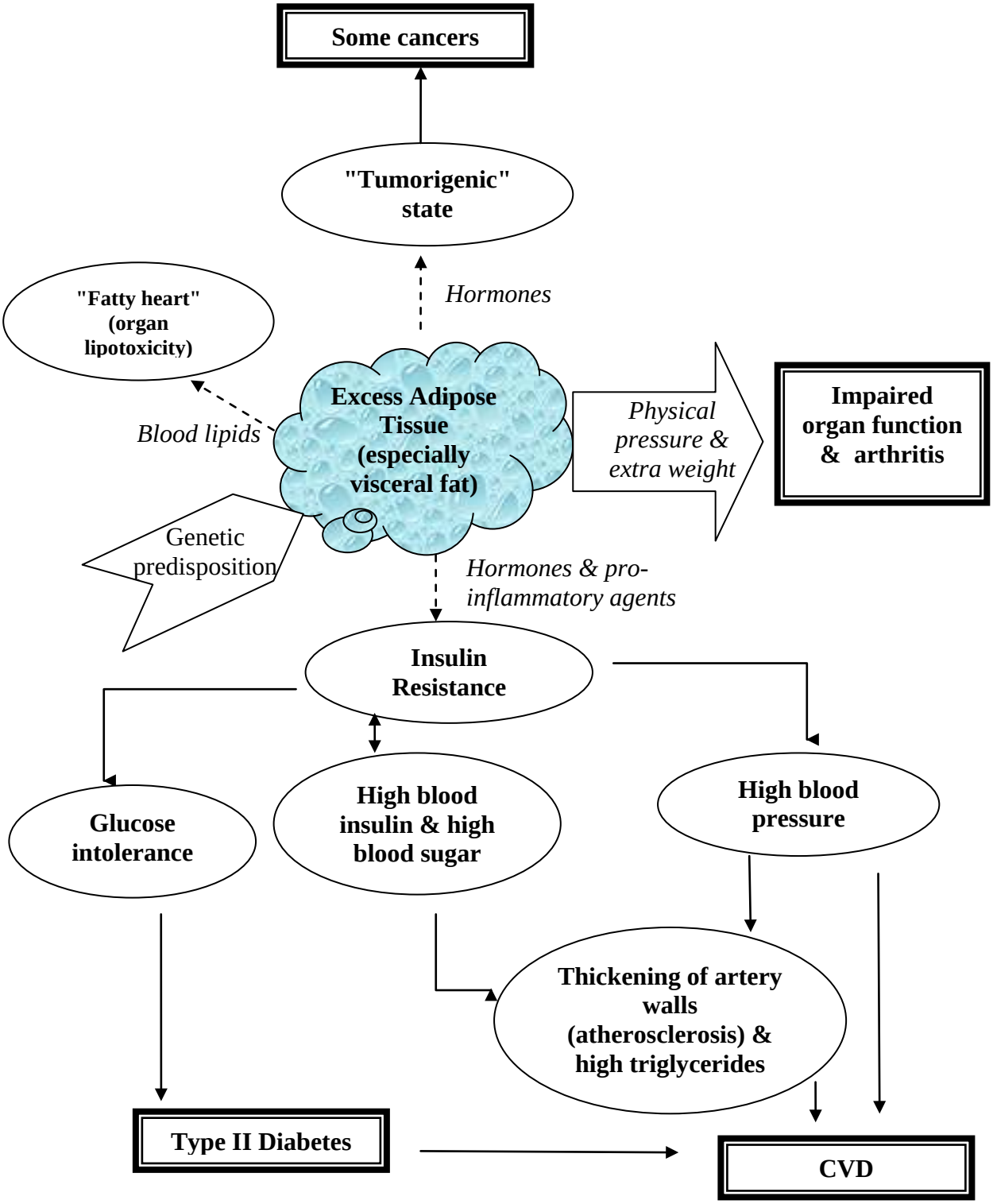

Figure 1: Mechanics of Excess Adipose Tissue and Disease Risk.
Adapted from figures and results in Calle and Kaaks (2004), Van Gaal, Mertens, and De Block (2006), and Plagemann (2008).

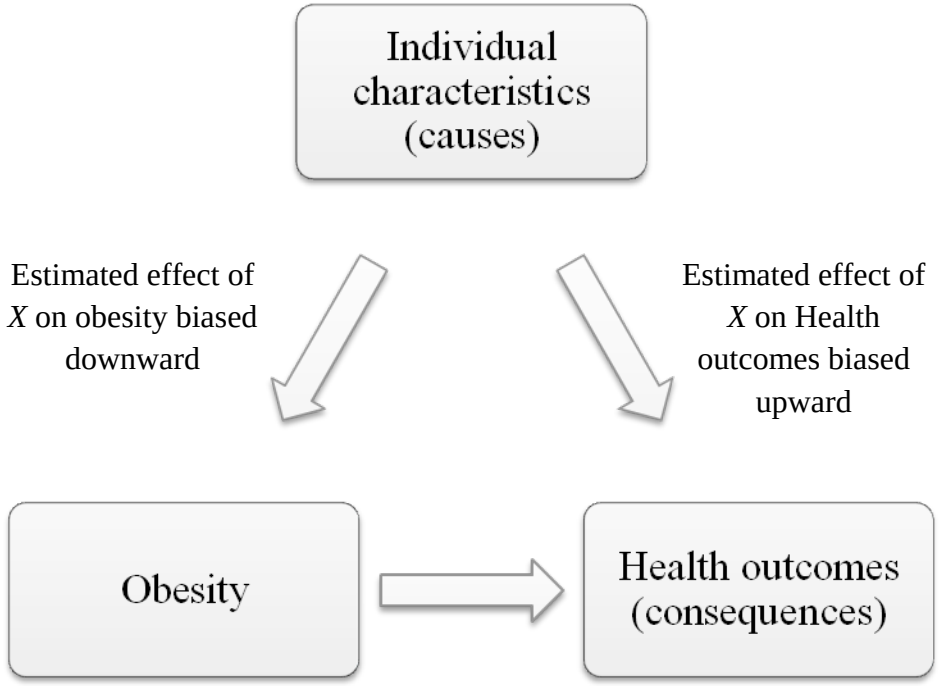

Figure 2: Interpretation of Measurement Error Bias $\delta > 0$.

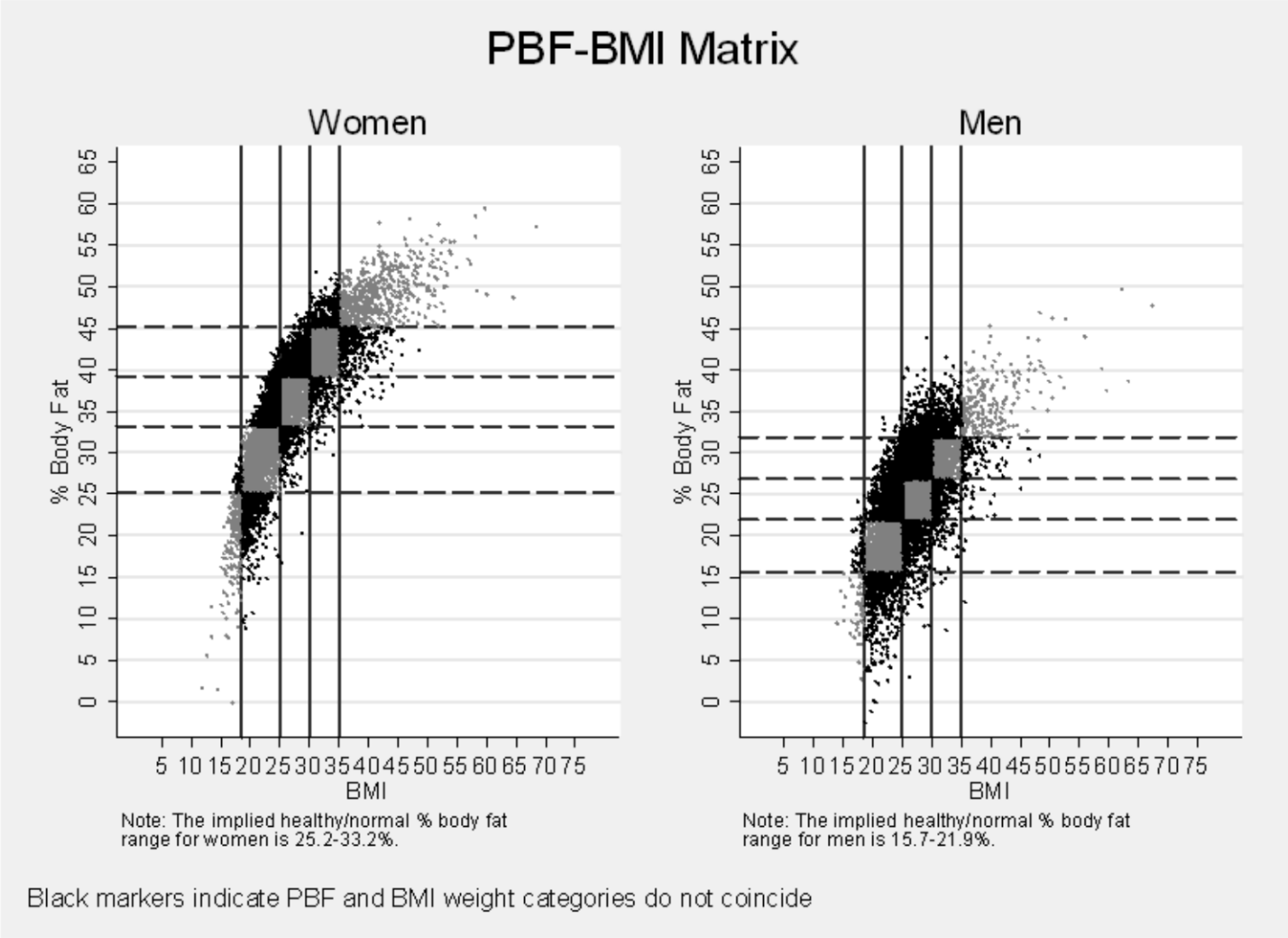

Figure 3. *%BF vs. BMI Obesity Classification Matrix*

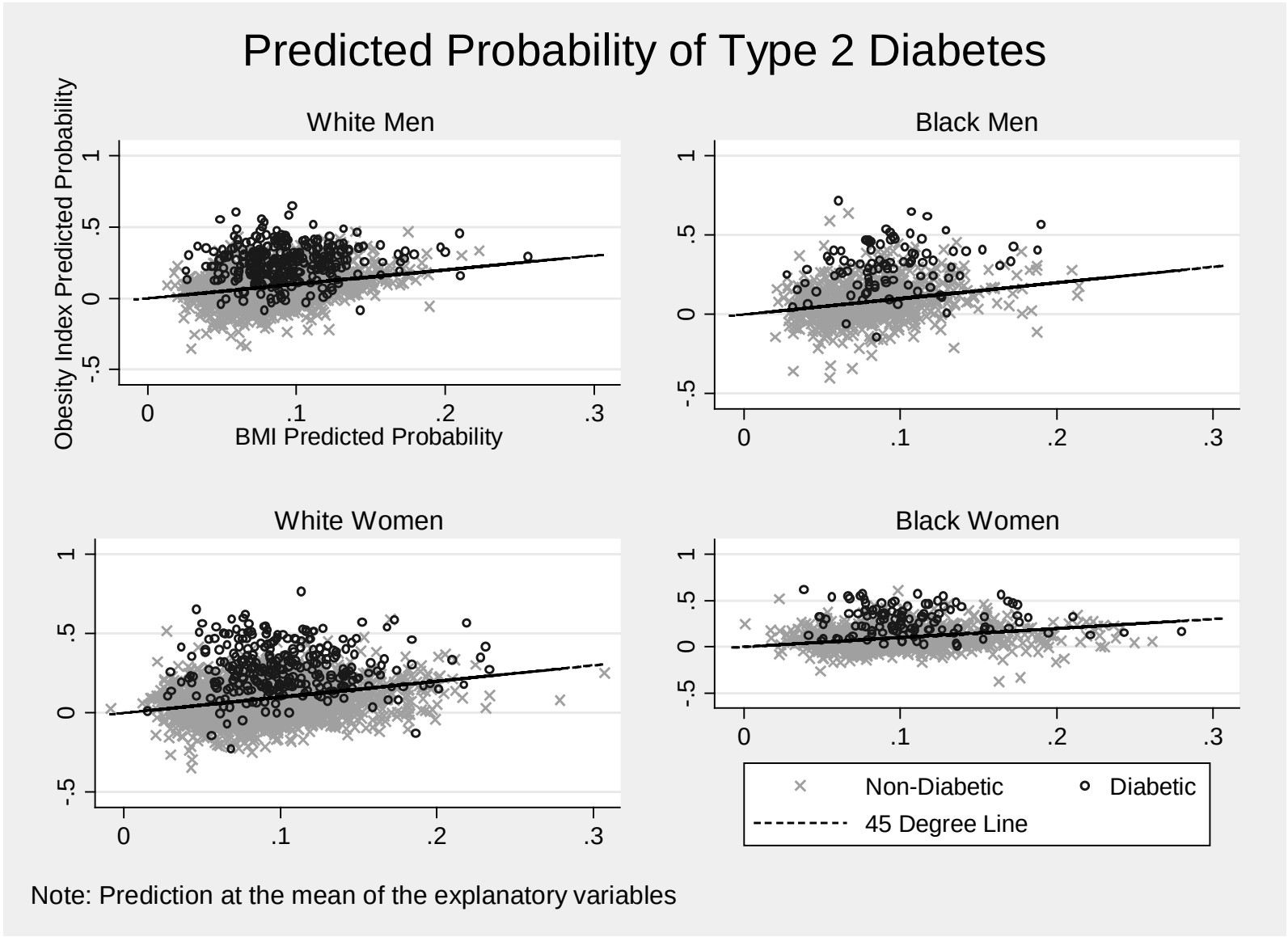

Figure 4. BMI vs. Our Obesity Index Predicted Probability of Type 2 Diabetes