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

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ABSTRACT

The body mass index (BMI) is a single statistic used to proxy for the complex way in which excess body fat increases the risk of disease and premature death. Although BMI is useful as a coarse approximation and as a clinical tool, we urge caution in using it in econometric models to assess the causes and consequences of obesity. We show that regression analysis using BMI to proxy for obesity would overstate the effects of low-alcohol consumption, high income, and old age on the risk of death and disease.

JEL Codes: C52, I10

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

Obesity is widely understood to be excess body fat that increases the risk of disease and premature death. Some of the complexities associated with concepts and measures of obesity and their implications are less widely appreciated. Obesity itself is multidimensional because an excess of body fat can contribute to many different diseases, and to different extents, depending on the location of the fat within the body and the other characteristics of the individual. However, for good reasons, medical researchers, economists, and others interested in measuring and modeling obesity and evaluating its costs, and public policy options for reducing them, naturally want to use a simple one-dimensional index to represent obesity; preferably a measure that is cheap and easy to apply such that data will be affordable and abundant.¹

The body mass index (BMI) meets these requirements. BMI has been so widely adopted as an index of obesity that it is sometimes treated as a synonym for, or a definition of, obesity.² 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. 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,

¹ 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.

² Throughout this paper, we use the term BMI to refer to the most commonly used formula, given by weight (in kilograms) divided by the square of height (in meters squared). Smalley et al. (1990) discuss several other weight-to-height BMI formulas that have been proposed as alternatives.
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). BMI-defined obesity categories are also used to estimate the direct and indirect costs attributable to obesity, the difference in medical 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 article, we argue that the most important and interesting economic questions about the causes and consequences of obesity should be framed in terms of excess body fat that increases the risk of disease and premature death, even if the researcher is constrained to use the body mass index (BMI) as a proxy for obesity. This advice is useful only if we understand how BMI relates to obesity, broadly defined, and to the causal and consequential variables under study. Using a comprehensive dataset on individual health, and information on the biological pathways through which excess fat affects health, we assemble a set of variables that capture the amount and location of body fat and the predisposition of individuals to develop obesity-related diseases. We then assess the effect of omitting these obesity variables from regression models aimed at understanding the causes and consequences of obesity. We find that:

1. Disease risk as measured by our obesity model tends to be lower for high-alcohol users than for low-alcohol users. A model based only on BMI would conclude that this lower risk was attributable to high alcohol consumption rather than lower obesity.

2. Disease risk as measured by our obesity model tends to be lower for high-income households than for low-income households. A model based only on BMI would conclude that this lower risk was attributable to higher income rather than lower obesity.
3. Disease risk as measured by the obesity variables tends to be greater for older people than younger people. For example, an older person is likely to have a larger waist circumference than a younger person with the same BMI. A model based on BMI alone would conclude that this higher risk was attributable to having lived longer rather than obesity.

We also find that smoking affects the probability of disease or premature death similarly whether we do or do not control for obesity either with BMI or our obesity measure. A model based on BMI would imply the same conclusions about the connection between smoking and obesity as a model using the rich set of obesity variables. Most individuals with high BMI also have high percent body fat percent and face obesity-related health risks, so BMI is clearly a useful proxy for obesity. However, we urge caution when using BMI to assess causes and consequences of obesity, in particular in regression models. In some cases, large biases can arise from using BMI as a proxy for a more complete measure of obesity, and the size and direction of the bias cannot be known with confidence until a more complete measure is tried.

2. Background on Obesity and Measures of Obesity

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). But the linkages are complex and manifold, and to make informed choices about measures of obesity, it is necessary to understand the relationships between body fat and human health risks, as discussed next.

Obesity and Health
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). The metabolic and systemic abnormalities associated with obesity lead to conditions such as “metabolic syndrome,” which precedes the development of type 2 diabetes, cardiovascular disease (CVD), and death (Metabolic Syndrome - Statistics, 2004; Kershaw and Flier, 2004). More details on these relationships can be found in Appendix A.

Medical research has shown that both 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). 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). 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 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

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 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).
explain much of the high incidence of metabolic syndrome among the old (Zamboni et al., 2005; Miller, 2008).

Alternative Measures of Obesity

A first-order measure of obesity is the fraction of total body weight composed of only adipose tissue, expressed as the percent body fat (%BF). 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.5 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 standard BMI.

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.6 Appendix B provides more detail on the use of BMI in economic research. 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

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5 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 40K counting), anthropometry (e.g., skin fold 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 (i.e., no radiation exposure or needle pricks), non-hazardous, and low cost device (Forbes, 1998).

6 Examples include Burkhauser and Cawley (2008), Garn, Leonard, and Hawthorne (1986), Smalley et al. (1990), Gallagher et al. (1996), Deurenberg (2001), Frankenfield et al. (2001), and 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.
the amount of excess fat accumulation, but also the distribution of body fat. 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).\(^7\) 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. 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. 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.

\(^{[TABLE 1. Alternative Obesity Classification Systems ]}\)

\(^{[TABLE 2. Adult Weight Distribution by Obesity Classification System]}\)

\(^7\) 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).
Using data from the NHANES III, we calculate the prevalence of obesity under the alternative measures. Table 2 reports the results. Depending on the classification system used, the prevalence of obesity (BMI ≥ 30) ranges from 19 percent to 63 percent for adult men and from 24 percent to 75 percent for adult females, respectively. Because these systems all measure fatness differently (e.g., as the ratio of weight to height-squared, 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, for which the 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). 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 ≥ 30 percent rather having a BMI ≥ 30.

3. Characterizing the Error from Using BMI to Measure Obesity

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. 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. Here, we discuss the nature of the biases that can arise when
BMI is used as a proxy for a multi-dimensional measure of obesity that reflects the increased probability of a particular health outcome attributable to excessive fatness.\(^8\)

*A Model of Obesity and Omitted Variables 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. An alternative 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: \(X\) denotes a set of covariates (including a constant), and \(F\) denotes a vector of measures of fatness such as the amount and location of body fat, weight history, and the metabolic abnormalities associated with obesity.

We conceptualize obesity for a particular individual \(i\) as the increased risk resulting from carrying *extra* fat, i.e.,

\[
OB_i = \Pr(D_i = 1 \mid F_i, X_i) - \Pr(D_i = 1 \mid \bar{F}, X_i)
\]

where \(\bar{F}\) signifies the healthy amount of each of the measures of fatness. The vector \(F_i\) has dimension \(M\) and the vector \(X_i\) has dimension \(K\). For brevity, this formulation restricts both the effect of \(F_i\) on the health outcome and the value \(\bar{F}\) to be constant across individuals. Later, we allow variation across race and sex in these elements.

We specify a linear model for disease risk

\(^8\) 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.
Given the model in (1), the measure of obesity is

\[ OB_i = \phi' \left( F_i - F_i \right) \quad (3) \]

This measure depends specifically on the health outcome or set of outcomes defined by \( D \), and the set of control variables, \( X \). 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 thereby causing omitted variables bias in models of the determinants of obesity and for models and measures of the consequences of obesity.

Omitted variables bias arises when \( X \) contains more or different predictive information about \( OB \) than it does about \( BMI \). This bias affects regression estimates of the causes of obesity, and it also affects estimates of the effect of \( X \) on health outcomes. Moreover, these two consequences are directly related; they are opposite sides of the same coin, which we show next.

Suppose a researcher aims to estimate the effect of \( X \) on health outcomes by estimating by ordinary least squares the model

\[ D_i = \beta' \cdot BMI_i + \theta'' X_i + u_i \quad (4) \]

where we use asterisks on the coefficients and error term because these terms may differ from their counterparts in the correctly specified model in (2). This model uses \( BMI \) to control for obesity and includes dummy variables for race and sex, but omits the relevant fatness variables \( F_i \). Standard formulas (e.g., Wooldridge 2002, p. 64) imply that the omitted variables bias \((\theta^* - \theta)\) equals the dot product of coefficients in (2) on the variables omitted from (4) with the coefficients on \( X \) in auxiliary regressions of each omitted variable on the included variables.

Mathematically, the omitted variables bias is

\[ \theta^* - \theta = d\phi \quad (5) \]
where $d$ denotes the $K \times M$ matrix of coefficients on $X$ in regressions of the fatness variables on the variables included in (4), i.e.,

$$F_i = b BMI_i + d'X_i + w_i$$

(6)

Thus, when estimating the effect of $X$ on health outcome $D$, the bias induced by controlling for obesity using $BMI$ depends on the relationship between $X$ and the full vector of fatness measures. For example, suppose moderate alcohol consumption is uncorrelated with BMI, but is associated with a lower $OB$ and therefore a lower diabetes risk (i.e., it has a negative $d$ coefficient in (6)). The true effect of alcohol consumption on diabetes risk is zero ($\theta=0$). However, the regression in (4) would produce a negative estimate ($\theta^* < 0$) because that regression does not control adequately for obesity.

To see how omitted variables bias distorts estimates of the causes of obesity, suppose a researcher would like to estimate $\gamma$ in the regression

$$OB_i = \gamma'X_i + \epsilon_i$$

(7)

but uses $BMI$ in place of $OB$ and estimates

$$BMI_i = \gamma''X_i + \epsilon_i^*$$

(8)

If $X$ has the same effect on $BMI$ as it has on $OB$, then it means that $X$ has no incremental effect on $OB$ after controlling for $BMI$, i.e., the vector of coefficients, $\delta$ would all equal zero in the following regression:

$$OB_i = b^*BMI_i + \delta'X_i + \nu_i$$

(9)

Substituting (8) into (9) reveals how $\gamma$ relates to $\gamma^*$, i.e.,
Thus, we have

$$\gamma^* b^* - \gamma = -\delta$$

(10)

After scaling by $b^*$ to account for the fact that $OB$ and BMI are measured on different scales, the bias in the estimated effect of $X$ on obesity equals $-\delta$.

Finally, we note that we can write (9) as

$$\phi^t_i F_i = \phi^t \bar{F} + b^* \text{BMI}_i + \delta^t X_i + \nu_i$$

(11)

Apart from a constant, this regression is identical to equation (6) multiplied by $\phi^t$, i.e., $\delta = d\phi$. Thus, the parameter vector $\delta$ measures the bias in the estimated effect of $X$ on health outcomes and $-\delta$ measures the bias in the effect of $X$ on obesity. We introduced an example above in which moderate alcohol consumption is uncorrelated with BMI, but is associated with a lower $OB$ and therefore a lower diabetes risk. In this example, the relevant element of $\delta$ is negative. Measuring obesity using BMI would cause us to believe that moderate alcohol consumption lowers disease risk and has no effect on obesity, whereas the truth is that moderate alcohol consumption is negatively associated with obesity and has no effect on disease risk.

Figure 1 illustrates this phenomenon for a case in which $\delta > 0$. The $X$ variables affect disease risk 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. In our empirical analysis, we estimate $\delta$ as the difference between the coefficients on
$X$ in equations (2) and (4). Before proceeding to the empirical analysis, we show that the above results generalize directly to a more general setting in which the effect of $F_i$ on the health outcome and the healthy value of the fatness variables vary across individuals of different race and sex.

**Generalizing the Model to Allow Dependence on Race and Sex**

We develop the arguments in a parallel fashion to the previous section. We allow $F_i$ to affect the health outcome differently depending on race and sex, by generalizing (2):

$$D_i = \sum_{j=1}^{J} (\alpha_j + \theta_j F_i) C_{ij} + \theta'X_i + u_i$$

(12)

where $C_{ij}$ is a dummy variable indicating that individual $i$ is a member of race-sex group $j$. Thus, the parameters $\alpha_j$ and in the vector $\phi_j$ may vary by sex and race. Because fatness as measured by the variables $F_i$ 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. We distinguish between variables that affect the interaction between obesity and health (e.g., race and sex) and variables that affect disease risk directly. Our focus is on how the measurement of obesity distorts the estimated effects of some $X$ variables while accounting for the characteristics defined by $C_{ij}$.

Given the model in (12), the measure of obesity is

$$OB_i = \sum_{j=1}^{J} \phi'_j (F_i - \bar{F}_j) C_{ij}.$$  

(13)

---

9 We index $F_j$ by $j$ rather than $i$ to reflect our specification that this variable is constant across individuals within each race-sex group.
Suppose a researcher aims to estimate the effect of $X$ on health outcomes by estimating by ordinary least squares the model

$$
D_i = \sum_{j=1}^{J} \alpha_j^* C_{ij} + \beta^* BMI_i + \theta^* X_i + u_i^*,
$$

(14)

where we use asterisks on the coefficients because these parameters may differ from their counterparts in the correctly specified model in (12). This model uses $BMI$ to control for obesity and includes dummy variables for race and sex, but omits the relevant fatness variables $F_i C_{ij}$.

The omitted variables bias is

$$
\theta^* - \theta = \sum_{j=1}^{J} d_j \phi_j,
$$

(15)

where $d_j$ are coefficients on $X$ in the regressions

$$
F_i C_{ij} = \sum_{k=1}^{J} a_{jk} C_{ik} + b_j BMI_i + d_j X_i + w_{ij} \quad j = 1, 2, \ldots, J.
$$

(16)

This equation represents a separate regression for each race-sex group ($j$). Next, we take a weighted sum of across race-sex groups

$$
\sum_{j=1}^{J} \phi_j' F_i C_{ij} = \sum_{j=1}^{J} \left( \sum_{k=1}^{J} \phi_j' a_{jk} C_{ik} + \phi_j' b_j BMI_i + \phi_j' d_j X_i + \phi_j' w_{ij} \right).
$$

(17)

Recognizing that, apart from race-sex specific means, the left-hand-side of this equation equals the $OB$ in (13), we can re-write as a generalized version of (11):

$$
OB_i = \sum_{j=1}^{J} a_j^* C_{ij} + \beta^* BMI_i + \delta X_i + v_i.
$$

(18)

Thus, as above, the parameter $\delta$ measures the bias in the estimated effect of $X$ on health outcomes and $-\delta$ measures the bias in the effect of $X$ on obesity.
4. Data

We use the publicly 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.

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. We restrict our analysis to survey respondents over eighteen years of age, since BMI is not the standard metric used to diagnose childhood obesity.10

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.

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10 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).
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. 11

[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 classify primary COD, and also indicates if diabetes, hip fracture, or hypertension was cited as a secondary COD. 12

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.

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11 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](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.

12 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.
and oversampling of specific subpopulations of interest. We also use the “\texttt{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).

5. **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 BMI or our alternative measure of obesity. We specify our measure of obesity—which is intended to represent the increase in probability of a particular health outcome attributable to excess body fat—as a flexible function of a large array of measures suggested in the medical literature. In addition to information on weight history and the relative amount and location of body fat, our measure of obesity includes the degree of metabolic dysfunction present in an individual. It is important to include metabolic dysfunction, 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
Thus, the health effects of obesity have multiple dimensions, and the amount of body fat and the extent of metabolic dysfunction both play important roles in defining and describing obesity as it occurs in an individual.

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 $\geq 150$ 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., $WC > 94$ cm for men and $WC > 80$ cm for women. In the models of CVD and mortality 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). The complex and interdependent relationship between type 2 diabetes and insulin resistance suggests that $QUICKI$ may not belong in the model of type 2 diabetes, so we use it only in the CVD and death models. We also allow for the possibility of a non-linear relationship between current and lagged percent body fat ($%BF$ and $%BF_{t-10}$).

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 the two models defined in (12) and (14). The first model allows the coefficients on $\%BF$ to vary by race and sex, whereas the second model contains no such interaction terms. In some senses it may seem unfair to expect BMI as a measure of obesity to compete well with our more general and flexible model in which we estimate the parameters for its elements jointly with the rest of the model, with interaction terms. It might seem fairer to allow BMI to enter more flexibly, too, and would be so if we were comparing the relative performance of BMI and $\%BF$ as alternative measures of obesity. However, our purpose here is to characterize how BMI can produce misleading regression results when used to measure obesity, rather than to investigate how to improve the performance of BMI by transforming it in various ways. To achieve this purpose it is appropriate to introduce BMI in the models in a typical fashion. 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 4 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 4 and 5 display the results from the model described by (12), and columns 3 and 7 display the results for (14). Columns 4 and 8 contain the difference in the estimated coefficients, or $\delta$, on the explanatory variables, with the standard error of the difference in parentheses. The asterisks in these columns indicate that the coefficients are statistically significantly different from zero at the 1 (**%) percent
or 5 (*) percent level. A statistically significant and positive estimate of $\delta$ 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 measure of obesity.

Omitted Variable Bias

Compared to BMI, the full set of obesity variables predicts a significantly larger share of the variation in type 2 diabetes: the fit of the model beyond the control variables improves nearly 100 percent when we use our obesity measure (the $R^2$ increases from 0.05 to 0.10). The Vuong (1989) test statistic for non-nested hypotheses also indicates that the model including the full set of obesity measures is better specified than one that uses BMI as a proxy for obesity. The coefficients on the components of the obesity measure take the expected signs and may give some insight into which components are relatively important for a particular health outcome. For instance, weight history, blood pressure, family history and triglycerides 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 4. BMI vs. Obesity Measure 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 $\delta$ for age, smoking, 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 these variables on obesity. Specifically, a model using BMI would overstate by 0.005 the impact of a family history of diabetes on the probability of getting diabetes and understate by 0.005 the impact on obesity. The BMI model would falsely attribute more blame for the disease to family history and less to obesity than it should. Similarly, a model using BMI will overestimate the effect of being age 60 rather than 30 on type 2 diabetes risk by 6 percentage points ($(60–30)\times[0.002]–(60–30)^2[0.000] = 0.06$). The BMI model substantially overstates the effect of aging on the probability of getting type 2 diabetes and understates the effect of obesity as represented by the full set of obesity variables. 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. The BMI model also overstates the effect of smoking on type 2 diabetes risk relative to the model containing the full set of obesity measures.

Table 4 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 1.3 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 model containing the full set of obesity variables, and a negative coefficient in the model containing BMI instead.

Similarly, household income has a small, insignificant coefficient in the model that uses our measure of obesity, and a negative coefficient in the model with BMI. The estimate of $\delta = -0.001$ on the variable, Income-Poverty ratio implies that using BMI would lead a researcher to overstate by 0.03 percentage points the negative effect on diabetes risk of an income four times the poverty line compared with 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 4 disappears when we use the full set of obesity variables, 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, the full set of obesity variables produce 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 measured effect of being an ex-smoker on CVD risk and correspondingly deflates the effect of being an ex-smoker on obesity. 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.

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.\textsuperscript{13} The full set of obesity variables predict death during the follow-up period only marginally better than BMI, but the Vuong tests imply the models containing our measure of obesity are better specified than the models using BMI. We find significant omitted variable 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 omitted variable 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 a model that predicts mortality and death from an obesity-related cause using 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 percentage points.

\textit{[Table 5. BMI vs. Obesity Measure Effects on Mortality Outcomes]}

6. 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 a measure of obesity—its self a function of several measures of fatness and

\textsuperscript{13} The negative coefficient on QUICKI implies that a decrease in insulin resistance, or an increase in QUICKI results in a lower risk of death. Similarly, reducing insulin resistance when one has high blood pressure results in a reduced risk of death.
obesity indicators—, 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 measure of obesity. 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 percent 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 analysis and results suggest ways to create more refined measures of obesity for use in econometric models. In particular, we suggest further research into the use of measures of insulin resistance, blood pressure, and waist circumference to represent obesity.
7. Main Text References


Zhu, ShanKuan, ZiMian Wang, Stanley Heshka, Moonseong Heo, Myles S. Faith and Steven B. Heymsfield. 2002. "Waist Circumference and Obesity-Associated Risk Factors Among
8. Tables and Figures

Table 1. Alternative Adult Weight Classification Systems and Cutoffs

<table>
<thead>
<tr>
<th></th>
<th>Underweight</th>
<th>Normal</th>
<th>Overweight</th>
<th>Obese</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BMI (kg/m²)</strong></td>
<td>&lt;18.5</td>
<td>18.5-25</td>
<td>25-30</td>
<td>≥30</td>
</tr>
<tr>
<td><strong>%BF° (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Male</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-39 years</td>
<td>&lt;8%</td>
<td>8-20%</td>
<td>20-25%</td>
<td>≥25%</td>
</tr>
<tr>
<td>40-59 years</td>
<td>&lt;11%</td>
<td>11-22%</td>
<td>22-28%</td>
<td>≥28%</td>
</tr>
<tr>
<td>60-79 years</td>
<td>&lt;13%</td>
<td>13-25%</td>
<td>25-30%</td>
<td>≥30%</td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20-39 years</td>
<td>&lt;21%</td>
<td>21-33%</td>
<td>33-39%</td>
<td>≥39%</td>
</tr>
<tr>
<td>40-59 years</td>
<td>&lt;23%</td>
<td>23-34%</td>
<td>34-40%</td>
<td>≥40%</td>
</tr>
<tr>
<td>60-79 years</td>
<td>&lt;24%</td>
<td>24-36%</td>
<td>36-42%</td>
<td>≥42%</td>
</tr>
<tr>
<td><strong>WCb (cm)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Male</strong></td>
<td>&lt;94 cm</td>
<td>94-102 cm</td>
<td>≥102 cm</td>
<td></td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td>&lt;80 cm</td>
<td>80-88 cm</td>
<td>≥88 cm</td>
<td></td>
</tr>
<tr>
<td><strong>%BFc (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Male</strong></td>
<td>&lt;25%</td>
<td></td>
<td></td>
<td>≥25%</td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td>&lt;30%</td>
<td></td>
<td></td>
<td>≥30%</td>
</tr>
<tr>
<td><strong>WCd (cm)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Male</strong></td>
<td>&lt;90 cm</td>
<td></td>
<td></td>
<td>≥90 cm</td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td>&lt;84 cm</td>
<td></td>
<td></td>
<td>≥84 cm</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th></th>
<th>Percent</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Underweight</td>
<td>Normal</td>
<td>Overweight</td>
<td>Obese</td>
</tr>
<tr>
<td><strong>BMI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>4.80</td>
<td>37.51</td>
<td>38.92</td>
<td>18.77</td>
</tr>
<tr>
<td>Female</td>
<td>11.54</td>
<td>38.95</td>
<td>25.52</td>
<td>24.00</td>
</tr>
<tr>
<td><strong>%BF(^b)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1.31</td>
<td>31.35</td>
<td>34.60</td>
<td>32.74</td>
</tr>
<tr>
<td>Female</td>
<td>4.47</td>
<td>36.30</td>
<td>26.36</td>
<td>32.87</td>
</tr>
<tr>
<td><strong>WC(^c)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>50.11</td>
<td>22.79</td>
<td>27.11</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>34.39</td>
<td>20.24</td>
<td>45.36</td>
<td></td>
</tr>
<tr>
<td><strong>%BF(^a)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>55.09</td>
<td></td>
<td>44.91</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>24.81</td>
<td></td>
<td>75.19</td>
<td></td>
</tr>
<tr>
<td><strong>WC(^d)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>37.54</td>
<td></td>
<td>62.46</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>41.63</td>
<td></td>
<td>58.37</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\): These are based on the NIH and NIDDK recommended %BF cutoffs, which only distinguish between obese and non-obese.

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

\(^c\): The WC cutoffs used by the American Heart Association.

\(^d\): 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>
<thead>
<tr>
<th></th>
<th>Panel A: Women</th>
<th>Panel B: Men</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>White</td>
<td>White</td>
</tr>
<tr>
<td>Type 2 Diabetes Prevalence (%)</td>
<td>18-30 years 30-60 years Over 60 years</td>
<td>18-30 years 30-60 years Over 60 years</td>
</tr>
<tr>
<td></td>
<td>White</td>
<td>White</td>
</tr>
<tr>
<td>Percent Body Fat (%)</td>
<td>0.2 3.2 10.7 0.4 5.1 21.3</td>
<td>0.4 4.1 11.4 0.7 4.6 15.4</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.8 26.7 26.8 25.8 29.5 29.2</td>
<td>24.9 25.5 26.8 24.3 24.3</td>
</tr>
<tr>
<td>Number of observations</td>
<td>1,111 2,402 1,854</td>
<td>1,178 2,193 1,779</td>
</tr>
</tbody>
</table>

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

**Source:** NHANES III.
<table>
<thead>
<tr>
<th>Measure</th>
<th>Type 2 Diabetes</th>
<th>Cardiovascular Disease</th>
<th>( \delta = (3)-(2) )</th>
<th>( \delta = (7)-(6) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>0.006**</td>
<td></td>
<td></td>
<td>0.003**</td>
</tr>
<tr>
<td>%BF</td>
<td>-0.00612</td>
<td>0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>%BF(^2)</td>
<td>0.00002</td>
<td>0.00001</td>
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<td></td>
</tr>
<tr>
<td>%BF(t-10)</td>
<td>-0.00055</td>
<td>-0.004*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(%BF(t-10))(^2)</td>
<td>0.00008**</td>
<td>0.00006*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female*%BF</td>
<td>0.00393</td>
<td>-0.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female*%BF(^2)</td>
<td>-0.00011</td>
<td>0.00000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black*%BF</td>
<td>-0.00242</td>
<td>-0.007</td>
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<td>Black*%BF(^2)</td>
<td>0.00004</td>
<td>0.00011</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waist Circumference</td>
<td>0.00447**</td>
<td>-0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Criteria for Metabolic Syndrome Dummies:</strong></td>
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<tr>
<td>Triglycerides</td>
<td>0.03530**</td>
<td>0.026*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL Cholesterol</td>
<td>0.00850</td>
<td>0.046**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>0.02240*</td>
<td>0.492**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>QUICKI</td>
<td>0.0162</td>
<td>0.454</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Blood Pressure)*(QUICKI)</td>
<td>-3.074**</td>
<td>(0.645)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Abdominal Obesity)*(QUICKI)</td>
<td>0.049</td>
<td>(0.093)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Blood Pressure)*(Abdominal Obesity)</td>
<td>-0.01186</td>
<td>-0.032</td>
<td></td>
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</tr>
</tbody>
</table>

Table 4. BMI vs. Obesity Measure Effects on Type 2 Diabetes and CVD
Table 4 (continued). BMI vs. Obesity Index Effects on Type 2 Diabetes and CVD

<table>
<thead>
<tr>
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<th>Type 2 Diabetes</th>
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<th>Cardiovascular Disease</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
<td>(5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(4)</td>
<td></td>
<td>(6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>δ=(3)-(2)</td>
<td>(7)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>δ=(7)-(6)</td>
</tr>
<tr>
<td>Female</td>
<td>-0.022**</td>
<td>0.04827</td>
<td>-0.021*</td>
<td>-0.069</td>
</tr>
<tr>
<td></td>
<td>(0.006)</td>
<td>(0.086)</td>
<td>(0.009)</td>
<td>(0.104)</td>
</tr>
<tr>
<td>Black</td>
<td>0.031**</td>
<td>0.07657</td>
<td>0.023*</td>
<td>-0.054</td>
</tr>
<tr>
<td></td>
<td>(0.008)</td>
<td>(0.054)</td>
<td>(0.009)</td>
<td>(0.071)</td>
</tr>
<tr>
<td>Age</td>
<td>0.009**</td>
<td>0.00488**</td>
<td>0.007**</td>
<td>0.002**</td>
</tr>
<tr>
<td></td>
<td>(0.002)</td>
<td>(0.002)</td>
<td>(0.003)</td>
<td>(0.001)</td>
</tr>
<tr>
<td>Age²</td>
<td>-0.00006**</td>
<td>-0.00003</td>
<td>-0.00003</td>
<td>0.00000</td>
</tr>
<tr>
<td>Current Smoker</td>
<td>-0.009</td>
<td>-0.01573</td>
<td>-0.002</td>
<td>0.014**</td>
</tr>
<tr>
<td></td>
<td>(0.008)</td>
<td>(0.009)</td>
<td>(0.011)</td>
<td>(0.003)</td>
</tr>
<tr>
<td>Former Smoker</td>
<td>0.018*</td>
<td>0.01436*</td>
<td>0.014</td>
<td>-0.001</td>
</tr>
<tr>
<td></td>
<td>(0.007)</td>
<td>(0.007)</td>
<td>(0.010)</td>
<td>(0.002)</td>
</tr>
<tr>
<td>Alcohol Calorie Share</td>
<td>-0.002**</td>
<td>-0.00130</td>
<td>-0.002*</td>
<td>-0.0007**</td>
</tr>
<tr>
<td></td>
<td>(0.001)</td>
<td>(0.001)</td>
<td>(0.001)</td>
<td>(0.000)</td>
</tr>
<tr>
<td>(Alcohol Calorie Share)²</td>
<td>0.00003*</td>
<td>0.00001</td>
<td>0.00002</td>
<td>0.00**</td>
</tr>
<tr>
<td>Family History Type 2 Diabetes</td>
<td>0.078**</td>
<td>0.06613**</td>
<td>0.071**</td>
<td>0.005**</td>
</tr>
<tr>
<td></td>
<td>(0.010)</td>
<td>(0.009)</td>
<td>(0.008)</td>
<td>(0.002)</td>
</tr>
<tr>
<td>Income-Poverty Ratio</td>
<td>-0.004**</td>
<td>-0.00209</td>
<td>-0.003</td>
<td>-0.001*</td>
</tr>
<tr>
<td></td>
<td>(0.001)</td>
<td>(0.002)</td>
<td>(0.002)</td>
<td>(0.001)</td>
</tr>
<tr>
<td>Insomnia Spells</td>
<td>0.056**</td>
<td>0.04855*</td>
<td>0.050</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>(0.020)</td>
<td>(0.019)</td>
<td>(0.040)</td>
<td>(0.006)</td>
</tr>
<tr>
<td>Hypersonnia Spells</td>
<td>-0.007</td>
<td>0.00090</td>
<td>-0.006</td>
<td>-0.007</td>
</tr>
<tr>
<td></td>
<td>(0.019)</td>
<td>(0.021)</td>
<td>(0.028)</td>
<td>(0.009)</td>
</tr>
<tr>
<td>Constant</td>
<td>-0.277**</td>
<td>-0.49157**</td>
<td>-0.381**</td>
<td>-0.166**</td>
</tr>
<tr>
<td></td>
<td>(0.052)</td>
<td>(0.089)</td>
<td>(0.075)</td>
<td>(0.059)</td>
</tr>
<tr>
<td>Observations</td>
<td>7,826</td>
<td>7,826</td>
<td>7,826</td>
<td>7,826</td>
</tr>
<tr>
<td>R-squared</td>
<td>0.053</td>
<td>0.101</td>
<td>0.069</td>
<td>0.058</td>
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</table>

Ho: Equivalence of Xs in Obesity Measure and BMI models

F-Stat: 6.134
p-value: 0.000

Ho: Obesity Measure model better specified than BMI model

Vuong test statistics: 8.63
p-value: 0.000

Notes: Standard errors in parentheses, ** p<0.01, * p<0.05
Table 5. BMI vs. Obesity Measure Effects on Death and Death from Obesity-Related Cause

<table>
<thead>
<tr>
<th></th>
<th>Death</th>
<th>Death from an Obesity-Related COD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
</tr>
<tr>
<td>BMI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>%BF</td>
<td>-0.004 (0.007)</td>
<td>0.016 (0.019)</td>
</tr>
<tr>
<td>%BF^2</td>
<td>0.00010 (0.000)</td>
<td>-0.00029 (0.000)</td>
</tr>
<tr>
<td>%BFt-10</td>
<td>-0.001 (0.001)</td>
<td>-0.003 (0.003)</td>
</tr>
<tr>
<td>(%BFt-10)^2</td>
<td>0.00006** (0.000)</td>
<td>0.00007 (0.000)</td>
</tr>
<tr>
<td>Female*%BF</td>
<td>-0.006 (0.009)</td>
<td>-0.005 (0.023)</td>
</tr>
<tr>
<td>Female*(%BF)^2</td>
<td>0.00003 (0.000)</td>
<td>0.00013 (0.000)</td>
</tr>
<tr>
<td>Black*%BF</td>
<td>-0.009 (0.006)</td>
<td>0.001 (0.012)</td>
</tr>
<tr>
<td>Black*(%BF)^2</td>
<td>0.00010 (0.000)</td>
<td>0.00004 (0.000)</td>
</tr>
<tr>
<td>Waist Circumference</td>
<td>0.000 (0.000)</td>
<td>0.003 (0.000)</td>
</tr>
<tr>
<td><strong>Criteria for Metabolic Syndrome Dummies:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triglycerides</td>
<td>-0.016 (0.009)</td>
<td>0.04724 (0.031)</td>
</tr>
<tr>
<td>HDL Cholesterol</td>
<td>-0.004 (0.011)</td>
<td>0.069* (0.026)</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>0.371** (0.123)</td>
<td>0.321 (0.295)</td>
</tr>
<tr>
<td>QUICKI</td>
<td>-0.731* (0.360)</td>
<td>0.704 (1.427)</td>
</tr>
<tr>
<td>(Blood Pressure)*(QUICKI)</td>
<td>-2.148** (0.754)</td>
<td>-1.174 (1.973)</td>
</tr>
<tr>
<td>(Abdominal Obesity)*(QUICKI)</td>
<td>-0.176 (0.090)</td>
<td>0.099 (0.405)</td>
</tr>
<tr>
<td>(Blood Pressure)*(Abdominal Obesity)</td>
<td>-0.010 (0.022)</td>
<td>-0.107 (0.058)</td>
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</table>
Table 5 (cont). BMI vs. Obesity Index Effects on Death and Death from Obesity-Related Cause

<table>
<thead>
<tr>
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<th>Death</th>
<th>Death from an Obesity-Related COD</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
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<td>(2)</td>
<td>(3)</td>
<td>(4)</td>
<td>(5)</td>
<td>(6)</td>
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<tr>
<td>Female</td>
<td>-0.053**</td>
<td>0.090</td>
<td>-0.052**</td>
<td>-0.142</td>
<td>0.041</td>
<td>0.093</td>
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<td></td>
<td>(0.009)</td>
<td>(0.123)</td>
<td>(0.010)</td>
<td>(0.149)</td>
<td>(0.030)</td>
<td>(0.339)</td>
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<tr>
<td>Black</td>
<td>0.034**</td>
<td>0.205*</td>
<td>0.031**</td>
<td>-0.174</td>
<td>0.078**</td>
<td>0.030</td>
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<tr>
<td></td>
<td>(0.009)</td>
<td>(0.091)</td>
<td>(0.011)</td>
<td>(0.103)</td>
<td>(0.027)</td>
<td>(0.190)</td>
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<tr>
<td>Age</td>
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<td>-0.032**</td>
<td>-0.031**</td>
<td>0.001*</td>
<td>-0.003</td>
<td>-0.010</td>
</tr>
<tr>
<td></td>
<td>(0.002)</td>
<td>(0.003)</td>
<td>(0.003)</td>
<td>(0.001)</td>
<td>(0.008)</td>
<td>(0.008)</td>
</tr>
<tr>
<td>Age²</td>
<td>0.00041**</td>
<td>0.00042**</td>
<td>0.00042**</td>
<td>0.00000</td>
<td>0.00005</td>
<td>0.00010</td>
</tr>
<tr>
<td></td>
<td>(0.000)</td>
<td>(0.000)</td>
<td>(0.000)</td>
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</tr>
<tr>
<td>Current Smoker</td>
<td>0.134**</td>
<td>0.135**</td>
<td>0.137**</td>
<td>0.001</td>
<td>-0.119**</td>
<td>-0.109**</td>
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<tr>
<td></td>
<td>(0.011)</td>
<td>(0.010)</td>
<td>(0.014)</td>
<td>(0.003)</td>
<td>(0.036)</td>
<td>(0.034)</td>
</tr>
<tr>
<td>Former Smoker</td>
<td>0.045**</td>
<td>0.042**</td>
<td>0.044**</td>
<td>0.002</td>
<td>-0.05422</td>
<td>-0.066*</td>
</tr>
<tr>
<td></td>
<td>(0.011)</td>
<td>(0.011)</td>
<td>(0.011)</td>
<td>(0.002)</td>
<td>(0.031)</td>
<td>(0.031)</td>
</tr>
<tr>
<td>Alcohol Calorie Share</td>
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<td>-0.000</td>
<td>-0.000</td>
<td>0.000</td>
<td>-0.006</td>
<td>-0.005</td>
</tr>
<tr>
<td></td>
<td>(0.001)</td>
<td>(0.001)</td>
<td>(0.001)</td>
<td>(0.000)</td>
<td>(0.004)</td>
<td>(0.003)</td>
</tr>
<tr>
<td>(Alcohol Calorie Share)²</td>
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<td>-0.00000</td>
<td>0.00000</td>
<td>0.00000</td>
<td>0.00018</td>
<td>0.00016</td>
</tr>
<tr>
<td></td>
<td>(0.000)</td>
<td>(0.000)</td>
<td>(0.000)</td>
<td>(0.000)</td>
<td>(0.000)</td>
<td>(0.000)</td>
</tr>
<tr>
<td>Family History Type 2 Diabetes</td>
<td>0.007</td>
<td>-0.003</td>
<td>0.004</td>
<td>0.007**</td>
<td>0.063*</td>
<td>0.043</td>
</tr>
<tr>
<td></td>
<td>(0.008)</td>
<td>(0.009)</td>
<td>(0.010)</td>
<td>(0.002)</td>
<td>(0.025)</td>
<td>(0.024)</td>
</tr>
<tr>
<td>Income-Poverty Ratio</td>
<td>-0.01991**</td>
<td>-0.01776**</td>
<td>-0.01942**</td>
<td>-0.002**</td>
<td>0.007</td>
<td>0.009</td>
</tr>
<tr>
<td></td>
<td>(0.002)</td>
<td>(0.002)</td>
<td>(0.003)</td>
<td>(0.000)</td>
<td>(0.006)</td>
<td>(0.006)</td>
</tr>
<tr>
<td>Insomnia Spells</td>
<td>0.009</td>
<td>0.006</td>
<td>0.006</td>
<td>0.000</td>
<td>-0.337**</td>
<td>-0.309**</td>
</tr>
<tr>
<td></td>
<td>(0.015)</td>
<td>(0.016)</td>
<td>(0.019)</td>
<td>--</td>
<td>(0.074)</td>
<td>(0.091)</td>
</tr>
<tr>
<td>Hypersonomnia Spells</td>
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<td>-0.019</td>
<td>-0.018</td>
<td>0.001</td>
<td>0.021</td>
<td>0.002</td>
</tr>
<tr>
<td></td>
<td>(0.012)</td>
<td>(0.013)</td>
<td>(0.016)</td>
<td>(0.005)</td>
<td>(0.218)</td>
<td>(0.182)</td>
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<td>Constant</td>
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<td>0.262</td>
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<tr>
<td></td>
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<td>(0.080)</td>
<td>(0.262)</td>
<td>(0.451)</td>
<td>(0.359)</td>
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<td>7,826</td>
<td>7,826</td>
<td>2,536</td>
<td>2,536</td>
</tr>
<tr>
<td>R-squared</td>
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<td>0.369</td>
<td>0.357</td>
<td>0.357</td>
<td>0.036</td>
<td>0.060</td>
</tr>
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Ho: Equivalence of Xs in Obesity Measure and BMI models

F-Stat: 3.423
p-value: 0.000

Ho: Obesity Measure model better specified than BMI model

Vuong test statistics: 6.168
p-value: 0.000

Standard errors in parentheses, ** p<0.01, * p<0.05
Estimated effect of $X$ on obesity biased downward

Estimated effect of $X$ on Health outcomes biased

**Figure 1:** Interpretation of Measurement Error Bias when $\delta > 0$. 
Appendix A. 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.

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). 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). These bioactive mediators, collectively known as adipokines, also respond to signals from the central nervous and hormone systems (Kershaw and Flier, 2004; Obesity and Cancer: Questions and Answers, March 2004; Poirier et al., 2006).

The metabolic and systemic abnormalities associated with obesity lead to conditions such as the metabolic syndrome, which precedes the development of type 2 diabetes, cardiovascular disease (CVD), and death (Metabolic Syndrome - Statistics, 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

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14 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.

15 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
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). 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-lipoprotein (HDL) or “good” cholesterol.

Adipocytes also secrete sex hormones (e.g., testosterone and estrogens), which influence the body’s preferred locations for storing fat and how lipoproteins 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, less able to metabolize the excess insulin and glucose as it builds up in their blood stream, 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 the 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 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 among the old (Zamboni et al., 2005; Miller, 2008).

Existing Measures of Obesity

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16 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).

17 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).
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.18

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-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

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18 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 40K counting), anthropometry (e.g., skin fold 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).
cardiovascular disease (CVD).\textsuperscript{19} 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-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.

\begin{table}[H]
\centering
\caption{Alternative Obesity Classification Systems}
\end{table}

\begin{table}[H]
\centering
\caption{Adult Weight Distribution by Obesity Classification System}
\end{table}

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 height\textsuperscript{2}, 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).\textsuperscript{20} Using the NHANES III Burkhauser and

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\textsuperscript{19} 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).

\textsuperscript{20} Gallagher et al. (2000) collected data on %BF and BMI for whites, African Americans, and Japanese and estimated the equation $\text{PBF} = \beta_0 + \beta_1 \text{BMI}^{-1} + \beta_2 \text{Sex} + \beta_3 \text{Age} + \beta_4 \text{Sex} \times \text{Age} \times \text{BMI}^{-1}$ for whites and African Americans, and $\text{PBF} = \alpha_0 + \alpha_1 \text{BMI}^{-1} + \alpha_2 \text{Age}$ for Japanese men and women separately. From the estimated
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).

coefficients Gallagher et al. (2000) then predicted PBF corresponding to BMI of 18.5, 25, and 30 for specific age, race, and gender combinations.
Appendix B. 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 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.

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 ≥ 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 ≥ 30 have a 50-100 percent greater risk of premature death than those within the healthy BMI range (25 ≥ BMI > 20) (US DHHS, 2001). Allison et al. (1999) produced a conservative estimate of approximately 300,000 excess adult deaths per year attributable to obesity. 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.

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21 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.

22 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.
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.

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). 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 0.33kg/m² 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 (2003) agreed with Anderson and Matsa (2009), noting that the growth in calorie consumption

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23 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.
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 Philipson, 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.

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 understimate 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.

**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).

**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 (19) and (20) used to calculate FFM for males and females, respectively.

\[
\text{FFM}_{\text{Male}} = -10.678 + 0.262 \text{Weight} + 0.652 \left( \frac{\text{Height}^2}{\text{Res}_{R,IL}} \right) + 0.015 \text{Res}_{R,IL}
\]  

(19)
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 (22) and (23).

\[
Res^{RJL}_{Male} = 2.5 + 0.98 Res^{Valhalla}
\]

\[
Res^{RJL}_{Fem} = 9.6 + 0.96 Res^{Valhalla}
\]

We then calculate %BF as:

\[
%BF = 100 \times \frac{(Weight - FFM)}{Weight}.
\]

### 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 to estimate past values of %BF and BMI for each individual. 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 estimated (23) and calculated (24) and (25).

\[
FFM_{i,t} = \hat{\beta}_0 + \hat{\beta}_1 Age_{i,t} + \hat{\beta}_2 Female_i + \hat{\beta}_3 Black_i + \hat{\beta}_4 OtherRace_i + \hat{\beta}_5 Menopause_i + \hat{\beta}_6 Estrogen_i + \hat{\epsilon}_i
\]

\[
FFM_{i,t-10} = \hat{\beta}_0 + \hat{\beta}_1 Age_{i,t-10} + \hat{\beta}_2 Female_i + \hat{\beta}_3 Black_i + \hat{\beta}_4 OtherRace_i + \hat{\beta}_5 Menopause_{i,t-10} + \hat{\beta}_6 Estrogen_{i,t-10} + \hat{\epsilon}_i
\]

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24 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.

25 The regression results for this model are available upon request.
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.

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 $\geq 150$ 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) $\geq 130$ mm Hg, diastolic BP $\geq 85$ mm Hg, or treatment for high BP, and (iv) fasting plasma glucose $\geq 100$ 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.
Appendix References


