

The Protective Effect of High Body Mass on Mortality Risk.  
Obesity Paradox or Data Artifact?

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## **ABSTRACT**

An “obesity paradox” is often reported in mortality analyses, wherein high body mass is found to confer a survival advantage at older ages. Some researchers have cautioned that obesity paradoxes should be met with skepticism because age-specific estimates of the obesity-mortality association are likely biased by (1) reverse causality and/or (2) age-related selection processes. We test the intuitions behind these claims by analyzing the age-specific associations between high body mass and mortality risk in two U.S. samples. The National Health and Nutrition Examination Survey (NHANES) 1988-1994, 1999-2004 and the National Health Interview Survey (NHIS) 1986-2004 were prospectively linked to individual mortality records at the National Death Index (NDI) through December 31, 2006. Respondents were aged 25 to 84 years at survey and survival was followed for up to 21 years between ages 26 and 95. We examined all-cause mortality risk by body mass index (BMI) in both data sets, BMI 10 years prior to survey in NHANES data, and waist-to-thigh ratio (WTR) in NHANES data. Cause-specific mortality risk (circulatory diseases and accidents) was also assessed by BMI in NHIS data. Results from all analyses indicate that “obesity paradoxes” are likely data artifacts that reflect biased estimates from age-related selection processes.

Obesity prevalence in the US adult population is said to have reached “epidemic” levels,<sup>1</sup> with obesity rates having risen from about 13% in the 1970s to almost 36% in 2009-2010.<sup>2</sup> The increases in obesity rates among US adults have raised alarm among medical practitioners<sup>3</sup> and have prompted targeted and large-scale responses from US policy makers.<sup>4,5</sup> Concerns over the health consequences of rising obesity prevalence stem from evidence indicating high body mass to be a risk factor for early onset of disability<sup>6</sup> as well a risk factor for many chronic diseases such as hypertension,<sup>7</sup> heart disease,<sup>8,9</sup> diabetes,<sup>9</sup> and several site-specific cancers.<sup>10</sup> Yet despite the strong associations between high body mass and degenerative disease, some evidence suggests high body mass to be only weakly associated with all-cause mortality risk.<sup>11-14</sup> A recent meta-analysis by Flegal et al. (2013) found that overweight (body mass index [BMI] 25.0-29.9) populations have about a five percent lower mortality risk than normal weight (BMI 18.5-24.9) populations. This same analysis also found that class 1 obese (BMI 30.0-34.9) populations and normal weight populations experience no significant difference in mortality risk. Moreover, numerous studies report a so-called “obesity paradox”, in which the excess mortality associated with an obese BMI is found to weaken across age, with most evidence indicating that a high BMI confers a survival advantage at older ages.<sup>12,14-20</sup> These findings have been explained in terms of the obese population’s frequent use of medical services,<sup>21</sup> adiposity providing metabolic reserves during times of illness,<sup>21-24</sup> and adiposity reducing the severity and health consequences of accidental falls.<sup>25-27</sup>

Other research has suggested that the paradoxical diminishing strength of the obesity-mortality association across age is a data artifact.<sup>28</sup> Some suggest that high rates of smoking and/or preexisting disease among the normal weight samples (i.e., reference groups) might confound

estimates of the obesity BMI-mortality association. Any apparent “obesity paradox,” therefore, might simply reflect reverse causation whereby the normal weight sample at time of survey is disproportionately composed of respondents who have experienced both rapid weight loss and elevated mortality risk from preexisting health conditions and/or behaviors.<sup>29-32</sup> When prospective data permits long durations of mortality follow-up, the normal weight sample is thought to become composed of fewer frail members, and the association between high BMI and mortality risk is argued to reappear. Other work contends that observational studies used to analyze the association between obesity and mortality risk are often biased from mortality selection and/or differential selection into surveys by obesity status at older ages.<sup>28,33</sup> Thus, “obesity paradoxes” might reflect age-related selection biases in sample data and researchers’ inattention to the sources of these biases.

Our aim in the present study is to adjudicate between these competing perspectives, involving: (1) those who affirm that *obesity paradoxes* represent the true association between high body mass and mortality risk in the US population, (2) those who believe the paradoxes stem from *reverse causality*, and (3) those who argue obesity paradoxes are artifacts of age-related *selection biases*. First, we replicate the analytic approach used by Flegal et al. (2005) to examine all-cause mortality differentials by BMI categories across three age groups (25-59; 60-69; 70-95).<sup>11</sup> The Flegal et al. (2005) analyses suggested that the substantive strength of the obesity-mortality association weakened with increasing age, and the association was nonsignificant among the oldest age group (70-95). We replicate this work in two US nationally representative datasets, the National Health Interview Survey (NHIS) Linked Mortality Files (LMF), 1986-2004, and the National Health and Nutrition Examination and Survey (NHANES) Linked Mortality Files,

1988-1994 and 1999-2004. These tests are meant to reproduce prior research showing that the U.S. obesity-mortality association paradoxically declines in age, and to determine if high BMI confers a survival advantage at older ages.

Next, we perform a series of tests to address the reverse causality perspective. First, we refit the models in the NHIS data but this time account for potential sources of reverse causality by limiting samples to respondents reporting very good or excellent health at time of survey who have never been smokers. We also refit models using NHANES respondents' reported BMI ten-years prior to the survey, minimizing the chance that health conditions at time of survey are affecting BMI. Finally, we investigate the plausibility of reverse causal processes in survey data by examining health-related compositional changes in the Health and Retirement Study (HRS) 1992 original cohort across time-in-study.

We then address the perspective that obesity paradoxes are artifacts of age-related *selection biases* by refitting survival models and adjusting for respondents' cohort and age-at-survey. If a sample is biased by mortality selection and/or healthy participant effects, a respondent's age-at-survey will be inversely associated with mortality risk. We fit these adjusted models on the entire NHIS and NHANES samples as well as on the samples accounting for sources of reverse causality.

Finally, we further investigate the plausibility of these competing explanations of the obesity paradox by conducting a series of sensitivity tests. We first test the sensitivity of the BMI-mortality results by refitting survival models using an alternative measure of obesity, waist-to-

thigh ratio (WTR). Next, we examine BMI differences in cause-specific mortality rates from circulatory diseases and accidents. We contrast results from the cause-specific models to determine which perspective – conventional, reverse causality, or selection – produces results most consistent with medical understanding of the known effects of obesity on circulatory diseases, and which perspective obliges the possible survival advantage conferred by high body mass from accidental falls and other external causes of death. Finally, we end by showing that failure to account for age-related selection biases in survival analyses can lead one to the implausible conclusion that a “smoking paradox” also exists in both the NHIS and NHANES data. On balance, results favor the perspective that obesity paradoxes are artifacts of age-related *selection biases*.

## **METHODS**

### **Study Samples**

Nineteen continual waves of the National Health Interview Survey (NHIS), 1986-2004, and the 1988-1994, 1999-2004 waves of the National Health and Nutrition Examination Survey (NHANES) were linked to the National Death Index to record respondents’ quarter-year mortality status from survey date to December 31, 2006. Respondents’ self-reported race/ethnicity (non-Hispanic black, non-Hispanic white, non-Hispanic other, Hispanic), sex (women, men), education (< high school, high school, some college, and  $\geq$  college), marital status (married, widowed, divorced/separated, never married), and height and weight were obtained at baseline in both surveys. Self-reported smoking status (smoker, former smoker, never a smoker) was also obtained in NHANES surveys and in NHIS adult subsamples for years 1987, 1990-1995, and 1997-2003.

For our analyses that test confounding from reverse causal processes, we created a “healthy” NHIS data set by restricting the adult subsample to respondents who never smoked and whose self-rated health at baseline (poor, fair, good, very good, excellent) was “very good” or “excellent.” An NHANES-LMF subsample was also created to analyze differential survival among respondents who reported their weight (lbs.) 10 years prior to the survey date. Further, to account for weight loss prior to survey, we created a dummy variable indicating whether or not an NHANES respondent moved to a lower BMI category during the ten years prior to survey (1=yes, 0=no). To directly observe reverse causal processes in prospective data, we compared compositional changes in the original Health and Retirement Study (HRS) cohort between 1992 and 2008 resulting from follow-up mortality (n=1,979) or attrition (n=863) in the normal weight, overweight, class 1 obese, and class 2/3 obese samples. HRS respondents in 1992 were aged 50-60 at time of interview, and subsequently interviewed every two years. The final sample of the HRS 1992 cohort was composed of 9,961 respondents at baseline, of whom 6,088 were interviewed in 2008.

Consistent with Flegal et al.’s (2013) standardized BMI categories,<sup>12</sup> respondents in all samples were coded as having a “normal” weight if their body mass index (BMI) was [18.5-25) at time of survey, “overweight” if BMI was [25-30), “class 1” obese if BMI [30-35), and “class 2/3” obese if BMI  $\geq 35$ . While other measures of obesity are more sensitive to adipose distribution on the body,<sup>34</sup> evidence suggests BMI adequately captures the obesity-mortality association.<sup>35</sup> We corrected discrepancies in self-reported measures of height and weight in the NHIS by (1) adjusting proxy-reported answers,<sup>36</sup> and (2) adjusting trends in NHIS BMI to match BMI levels in NHANES, which reports clinically measured height and weight. Respondents with BMI <18.5 were omitted from analyses because “underweight” BMI is often indicative of

terminal illness and the small cell sizes hindered stable model fit. NHIS and NHANES respondents older than 84 at time of survey were omitted to safeguard against bias induced by the open-ended 85+ coding in the NHIS. All cases with missing values on covariates were excluded.

### **Outcome Measures**

All-cause mortality risk was assessed using Cox regression with attained age as the underlying survival time metric. Left-censoring was accounted for by starting exposure at survey date, and the clustered sampling designs of the NHIS and NHANES were accounted for using Stata 12's *svy* command and sampling weights. Cause of death was classified “circulatory diseases” if the NCHS 113-underlying cause of death file was coded 55 to 75 (all diseases/disorders of the circulatory system) and “accidents” were coded from values 114 through 123. Due to small cell counts of cause-specific deaths in both the NHANES sample and the NHIS “healthy” subsample, cause-specific mortality analyses were performed only on the full NHIS sample. The full NHIS sample was composed of 784,888 respondents and 129,263 deaths (54,070 from circulatory diseases and 3,762 from accidents) and the NHIS “healthy” sample contained 159,557 non-smoking healthy respondents and 10,611 deaths. The full NHANES analytic sample was composed of 25,650 respondents and 4,325 deaths, and the NHANES sample using BMI 10 years prior to survey was composed of 18,693 respondents and 3,958 deaths.

We examined compositional changes in (1) the proportion of HRS respondents self-rating their health as either “poor” or “fair”, (2) the count of health conditions reported at baseline (0 to 7), (3) the proportion reporting heart problems at baseline, and (4) the proportion that reported having missed a substantial time of work due to health complications. Proponents of reverse



causality argue that frailer and sicker members of normal weight samples bias the obesity-mortality association by elevating the mortality risk among this weight group. If reverse causal processes are occurring in HRS data, we ought to observe more rapid compositional changes in the normal weight sample as a result of higher mortality among this group's subset of frailer respondents.<sup>37</sup> That is, we ought to observe the normal weight sample becoming "healthier" as mortality and attrition select out the frailer respondents.

### **Statistical Analyses**

We replicate the approach used by Flegal et al. (2005) to estimate BMI differentials in survival by age groups [25-60), [60-70), and [70-95) in the NHIS-LMF and NHANES-LMF samples.<sup>11</sup> We first fit a survival model that does not account for reverse causality or selection biases, but controlled for respondents' sex, race/ethnicity, educational attainment, and marital status.

We tested for "reverse causality" in three ways. First, we refit the survival models using the NHIS "healthy" subsample, which was restricted to never smoking respondents whose self-rated health at baseline was very good or excellent. Thus, we lessen the chance that the normal weight sample is composed of a subset of respondents who have both lost weight and increased mortality as a result of pre-existing illnesses. Second, we refit the survival models on the NHANES sample using BMI 10 years prior to survey because this measure is far less susceptible to respondents' health at time of survey. Third, we observed the HRS 1992 cohort across time-in-study till 2008 to contrast compositional changes in the health conditions of the normal weight and obese samples as a result of mortality or attrition.

We tested for age-related selection processes by refitting the survival models while controlling for (1) five-year birth cohort because recent findings in the NHANES data suggest the effect of obesity on mortality risk across age is confounded with cohort,<sup>38</sup> and (2) respondents' ages-at-survey.<sup>33</sup> If a sample is biased by either mortality selection and/or healthy participant effects, respondents' ages-at-survey will be significantly and negatively associated with mortality risk. That is, respondents who enter the sample at older ages are likely to have lower hazards of mortality risk than do equally-aged respondents who were sampled at younger ages. Two-way interactions between BMI and age-at-survey are included in the obesity-mortality investigations to test for differing degrees of selection bias by BMI.

## **RESULTS**

### **Obesity Paradox?**

#### **Table 1 about Here**

Descriptive statistics from the four data sources used in the survival analyses are presented in Table 1.

#### **Table 2 about Here**

Age-specific mortality hazard ratios for overweight, class 1 obese, and class 2/3 obese from conventional survival models are presented in the top panel of Table 2. Results in both the NHIS and NHANES data are consistent with previous findings showing mortality risk for high BMI weakens with age (note: age-specific HRs for each BMI level do not significantly differ between NHIS and NHANES). Also consistent with previous findings, results in each dataset suggest that an overweight BMI is protective at all age groups. Taken together, results corroborate the existence of an obesity paradox in these data sets, with the effect of obesity on mortality risk

diminishing with increasing age, and class 1 obesity conferring a survival advantage among the oldest age groups.

### **Reverse Causality?**

#### **Table 3 about Here**

#### **Table 4 about Here**

Table 3 contains baseline descriptive statistics of HRS respondents belonging to the original 1992 cohort. Changes in four negative health outcomes between 1992 and 1998 and between 1992 and 2008 among BMI-specific samples are presented in Table 4. All results indicate that the normal weight sample became “healthier” across time-in-study more slowly than did the high BMI samples. That is, reductions in (1) the percent of respondents reporting poor/fair health, (2) respondents’ average number of health conditions, (3) the percent of respondents reporting heart problems, and (4) the percent of respondents missing work as a consequence of health conditions are all faster among the high BMI samples of the HRS. This is true across the entire duration of time-in-study, as well as across the first six years of time-in-study – when reverse causal processes are more likely to be observed. Taken together, all health-related changes across time-in-study resulting from mortality and attrition are inconsistent with reverse causal processes. Further, the relative size of the obese sample in the HRS 1992 cohort decreases by 10.8% as a result of disparate mortality and attrition, whereas the relative size of the normal weight population remains unchanged. Thus, no evidence is found supporting the existence of reverse causal processes in these data. On the contrary, mortality and attrition in this sample behave in the opposite direction to what one would expect if reverse causal processes were occurring.

#### **Table 5 about Here**

Age-specific mortality hazard ratios for overweight, class 1 obese, and class 2/3 obese from conventional survival models fit to data that account for reverse causal processes are presented in the top panel of Table 5. The estimated overweight and obese HRs are significantly larger than the respective HRs estimated in the full samples (top panel of Table 2), but the results from models accounting for reverse causality continue to show a weakening effect of obesity on mortality risk across age.

### **Age-related Selection Biases?**

#### **Table 6 about Here**

Table 6 contains estimated coefficients of two-way interactions between age-at-survey and BMI level on all-cause mortality risk for each sample, as well as the coefficients for the main effect of age-at-survey in the NHIS respondents (estimated coefficients were not statistically significant in the NHANES samples).

Results show that the effect of age-at-survey on all-cause mortality risk in the three samples is negative and dose-responsive in BMI. At high BMI levels, respondents' ages-at-survey are found to be significantly and substantively associated with lower mortality risk. This age-at-survey result indicates that the "healthy participant effect" in these samples rises in BMI level.<sup>39</sup> Thus, insofar as one fails to account for age-at-survey in survival models, the age-related selection bias in estimates of mortality risk amplifies in BMI level.

Estimates of all-cause mortality HRs by BMI-level from models that account for age-related selection biases are presented across the bottom panels of Table 2 (full samples) and Table 5 (samples correcting for reverse causality). Results from survival models that account for age-related selection show that relative mortality risks associated with class 1 and class 2/3 remain significantly positive at all age groups. Evidence further shows the marginal effect of obesity on mortality risk to grow significantly stronger with age.

### **Figure 1 about Here**

Figure 1 plots estimates of all-cause mortality HRs and 95% CIs for overweight, class 1, and class 2/3 obesity by age group from both the conventional survival model and the selection survival model fit to the NHIS full sample in Table 2. Discrepancies between the two models' results are significant, with results from conventional models showing the association between high body mass and mortality risk to decrease with age. Conversely, models accounting for survey-selection behave oppositely, indicating the strength of the obesity-mortality association grows stronger with age. Taken together, evidence found in both the NHANES and NHIS data suggests that the decreasing age patterns of the obesity-mortality association are not robust to corrections for selection processes. Thus, the observed survival advantage for the obese population in older ages likely reflects age-related selection biases that disproportionately affect the obese samples.

### **Obesity Paradox using Waist-to-Thigh-Ratio?**

The BMI cut points used to define overweight and obesity statuses in these analyses have been criticized as being too crude to accurately assess the health consequences of obesity.<sup>34, 35</sup> While

both the U.S. federal government and the World Health Organization endorse the use of such cut points<sup>40,41</sup> – and these categories were recently used in a meta-analysis finding the “obesity paradox” to exist across multiple data sets<sup>12</sup> – they are known to imperfectly measure adipose distribution on the body.<sup>34,35</sup> To address the possibility of error-in-measurement, we reanalyzed NHANES data using waist-to-thigh ratio (WTR) as an indicator of high body mass. Models estimating survival differences between the bottom 50<sup>th</sup> percentile of WTR (reference group) and the top two quartile ranges of WTR produce results consistent with model estimates using BMI. Age-at-survey coefficients are strongly conditioned by quartile ranges of WTR, and age patterns of the WTR-mortality association are consistent with those of the BMI-mortality association.

### **Figure 2 about Here**

Figure 2 shows mortality HRs for the top quartile ranges of WTR in the conventional models to grow significantly weaker with increasing age, while mortality HRs from the models controlling for age-related selection biases to grow significantly larger with increasing age.

### **Cause-specific Obesity Paradoxes?**

#### **Table 7 about Here**

Table 7 provides estimates of the two-way interactions between BMI and age-at-survey for mortality risk from circulatory diseases and accidents. Consistent with results in Table 6, the results indicate that the models accounting for selection biases differentiate the association between age-at-survey and mortality risk by BMI level, but only for circulatory disease mortality. That is, age-at-survey coefficient sizes for circulatory disease mortality risk are dose-responsive by BMI, and the coefficients are significantly larger than those for all-cause mortality

risk reported in Table 6. These results are consistent with medical intuition, in that age-related survey selection among the obese samples ought to amplify for causes of death more strongly associated with high body mass such as atherosclerosis, cerebrovascular disease, and myocardial infarctions (the causes of death most represented in the circulatory disease category). Conversely, the two-way interactions between BMI and age-at-survey are substantively small and not significant for accident-related mortality risk.

### **Figure 3 about Here**

The impacts of these selection processes on BMI differentials in mortality risk are evident in Figure 3, which plots estimated cause-specific mortality HRs for overweight, class 1, and class 2/3 obesity by age group from both the conventional survival model and the survival model controlling for selection processes in the NHIS full sample. The estimated HRs from each model are nonsignificantly different for accident-related mortality, with both models showing overweight and class 1 obesity to be nonsignificantly associated with accident-related mortality risk across age groups. The models also both show a nonsignificant protective effect against accident-related mortality for the class 2/3 obese sample at older ages. For deaths from circulatory diseases we see a strikingly different pattern. The conventional survival model estimates the obesity-mortality association to weaken with increasing age. Moreover, the rate by which the BMI-mortality association weakens across age is faster for circulatory disease-related mortality than for all-cause mortality. In contrast, and more consistent with established medical understanding,<sup>8,9,13</sup> the model accounting for age-related survey-selection shows the association between high body mass and mortality risk from circulatory diseases to grow stronger with age, and, overall, the association between high body mass and mortality risk is estimated to be stronger for deaths from circulatory diseases than from all causes of death.

## **Smoking Paradox?**

To further test the intuition that age-related selection processes may account for the observation of an obesity paradox, we refitted conventional survival models and selection models for the less equivocal smoking-mortality association.<sup>42-49</sup> The estimated coefficients of age-at-survey on mortality risk are significantly negative for NHANES and NHIS respondents who self-report as “smokers” (reference group “nonsmokers”) (results not shown, but available upon request).

### **Figure 4 about Here**

Figure 4 graphs the estimated age patterns of the association between smoking and all-cause mortality risk in both NHANES and NHIS by model. Failure to account for selection processes leads one to the medically implausible conclusion that the strength of the smoking-mortality association declines across age. Such patterns are inconsistent with existing research pointing to the cumulative effect of years spent smoking on mortality risk.<sup>44-49</sup> Conversely, the model sensitive to selection errors correctly matches the consensus opinion of the cumulative effect of smoking on mortality risk by estimating an increasing effect of smoking on mortality risk across age. Taken together, these results are consistent with evidence suggesting the “smoking paradox” in mortality risk observed in these data likely stems from the biasing effects of selection processes among the smoking sample at older ages.<sup>48,49</sup>

## **DISCUSSION**

Several studies report a so-called “obesity paradox” in which high body mass confers a survival advantage at older ages. Researchers have recently cautioned that age-specific estimates of the



obesity-mortality association are likely biased by mortality selection and age-related survey selection.<sup>28,33</sup> Thus, before attempting to explain obesity paradoxes in terms of health-protective mechanisms from higher body mass in older adulthood (e.g., metabolic reserves), medical researchers ought to question the age patterns of the obesity-mortality association and test alternative explanations of the weakening obesity-mortality association across age. Indeed, Banack and Kaufman (2013:461) recently advised that “‘paradoxes’ should be met with skepticism” and “future analyses should correct for survivor selection” before interpreting estimates of the obesity-mortality association.<sup>28</sup> In this line, we found evidence consistent with the contention that age-related selection processes bias estimates of the obesity-mortality association in both the NHIS-LMF and NHANES-LMF samples. When accounting for these selection processes the obesity-mortality association was found to be substantively large at all ages. We also found very little support for the contention that reverse causal processes substantively affect the age patterning of the US obesity-mortality association. Such processes do not appear to be strong factors behind the “obesity paradox” observed in the NHIS-LMF and NHANES-LMF data. In general, controlling for “reverse causal” processes strengthened the overall obesity-mortality association, but did not alter the age patterning of the association.

Cause-specific analyses were especially informative in evaluating the competing explanations of the “obesity paradox.” The estimated age patterns of the obesity-mortality association for circulatory diseases were quite different between models. Conventional models suggested that the association weakened rapidly across age, whereas the models accounting for selection showed the association to grow substantively stronger with increasing age. Conversely, for accidental deaths the selection model estimated the obesity-mortality association to be statistically indistinguishable from that estimated by the conventional models. Taken together,

results from the selection model differentiate the age-specific patterns of obesity on cause-specific mortality risk in a medically plausible manner. In contrast, models inattentive to potential biases from selection processes produced age diminishing patterns of obesity on mortality risk for all causes of death examined. On balance, the evidence supports the contention that model results suggesting an “obesity paradox” likely reflect biases from age-related selection processes. In this line, similar evidence was found for the existence of a “smoking paradox” that likely reflects age-related selection biases in these data.

The current results challenge the prevailing thinking that the effect of high body mass on mortality risk declines with age, and that high BMI in older adulthood is protective against early death. While evidence supporting the existence of an obesity paradox has been reproduced in numerous datasets, time settings, and different populations,<sup>11</sup> it is based on a methodological approach to modeling survival that is inattentive to age-related selection processes. Present findings support the contention that the obesity paradox is likely an illusion reflecting selection biases in observational data. The findings further indicate that the strength of the obesity-mortality association likely grows stronger, not weaker, with increasing age.

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## FIGURE LEGENDS

Figure 1. All-cause Mortality Hazard Ratios by BMI and Age Group, Full NHIS Sample, 1986-2004.

Abbreviations: NHIS, National Health Interview Survey. BMI, Body Mass Index.

Note: Hazard ratios relative to normal weight BMI on the log scale y-axis.

Figure 2. All-cause Mortality Hazard Ratios by WTR and Age Group, NHANES Sample, 1988-1994, 1999-2004.

Abbreviations: NHANES, National Health and Nutrition Examination Survey. WTR, Waist-to-Thigh Ratio.

Note: Hazard ratios relative to 50<sup>th</sup> percentile WTR on the log scale y-axis.

Figure 3. Mortality Hazard Ratios from Circulatory Diseases and Accidents by BMI and Age Group, Full NHIS Sample 1986-2004.

Abbreviations: NHIS, National Health Interview Survey. BMI, Body Mass Index.

Note: Hazard ratios relative to normal weight BMI on the log scale y-axis.

Figure 4. All-cause Mortality Hazard Ratios by Smoking Status and Age Group, NHIS Adult Sample, 1986-2004, and NHANES Sample, 1988-1994, 1999-2004.

Abbreviations: NHIS, National Health Interview Survey. NHANES, National Health and Nutrition Examination Survey.

Note: Hazard ratios relative to non-smoking sample on the log scale y-axis.

**Table 1.** Samples Providing Mortality Data

	<b>NHIS 1986-2004</b>	<b>NHIS Healthy Sample</b>	<b>NHANES 1988-1994/1999-2004</b>	<b>NHANES BMI 10yr Prior</b>
Unweighted Sample Size	784,888	157,557	25,650	18,693
Mortality follow-up Year	2006	2006	2006	2006
Person-years of follow-up	1,083,010	196,964	32,780	25,526
25 to <60	580,548	125,960	16,711	10,174
60 to <70	270,640	38,845	7,701	7,472
70 to <95	231,822	32,869	8,368	8,140
Number of Deaths	129,263	10,611	4,325	3,958
Circulatory Deaths	54,070	5,096	2,404	2,184
Accidental Deaths	3,762	454	125	42
Prevalence of BMI Level, %				
<18.5	0.01	0.36	1.63	1.87
18.5 to <25	37.37	36.22	32.00	40.97
25 to <30	38.94	41.15	36.35	36.92
30 to <35	16.39	16.50	18.49	13.92
≥35	7.29	5.77	11.53	6.32

Abbreviation: NHIS, National Health Interview Survey. NHANES, National Health & Nutrition Examination Survey.

Note: Samples limited to mortality follow up >1 year



**Table 2.** Mortality Hazard Ratios by Age Group and BMI Level from NHIS and Combined NHANES Data Sets

	NHIS						NHANES					
	25-59		60-69		70-95		25-59		60-69		70-95	
	HR	SE	HR	SE	HR	SE	HR	SE	HR	SE	HR	SE
<b>Conventional</b>												
Overweight	0.84	(.019)	0.81	(.017)	0.84	(.009)	0.78	(.159)	0.79	(.170)	0.88	(.065)
Class 1 Obese	1.06	(.022)	0.96	(.022)	0.91	(.012)	1.15	(.203)	0.97	(.206)	0.96	(.069)
Class 2/3 Obese	1.57	(.027)	1.30	(.028)	1.13	(.016)	1.65	(.182)	1.27	(.225)	1.03	(.091)
<b>Selection Correction</b>												
Overweight	0.84	(.019)	0.86	(.024)	0.94	(.033)	0.71	(.158)	0.93	(.178)	1.34	(.166)
Class 1 Obese	1.11	(.023)	1.25	(.029)	1.43	(.041)	1.03	(.208)	1.21	(.229)	1.60	(.163)
Class 2/3 Obese	1.70	(.028)	2.02	(.043)	2.41	(.058)	1.44	(.180)	1.71	(.232)	2.01	(.217)

Abbreviations: NHIS National Health Interview Survey, NHANES National Health and Nutrition Examination Survey, HR Hazard Ratio, SE Standard Error, BMI Body Mass Index.

All models control for sex, race/ethnicity, educational attainment, and marital status.

Models correcting for selection further control for age-at-survey and BMI X age-at-survey.

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**Table 3.** Descriptive Statistics of Health and Retirement Study 1992 Cohort at Baseline Survey

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	<b>Normal Weight</b>	<b>Overweight</b>	<b>Class 1 Obese</b>	<b>Class 2/3 Obese</b>
Unweighted Sample Size	3,304	4,012	1,655	690
Died in follow-up	645	773	348	213
Poor/Fair Self-rated Health	.173	.205	.282	.428
Number of Health Conditions	.829	1.044	1.346	1.864
Heart Problems	.100	.129	.150	.212
Missed Work Due to Health	.175	.201	.241	.385

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**Table 4.** Percentage point change in health outcomes from baseline to 1998 and to 2008 in the 1992 HRS cohort resulting from mortality and attrition.

	Poor/Fair Health	<u>Negative Health Outcome</u>		
		# of Conditions	% Heart Problems	% Missing Work
1998				
Normal Weight	-3.91	-5.67	-0.52	-1.44
Overweight	-5.18	-7.17	-1.49	-1.78
Class 1 Obese	-4.41	-9.15	-0.52	-1.00
Class 2/3 Obese	-5.10	-11.28	-1.27	-1.84
2008				
Normal Weight	-23.2	-13.4	-1.6	-3.7
Overweight	-26.5	-15.4	-2.7	-4.4
Class 1 Obese	-21.8	-15.6	-2.5	-2.8
Class 2/3 Obese	-31.1	-25.6	-5.4	-7.8

**Table 5. Mortality Hazard Ratios by Age Group and BMI Level from "Reverse Causality" NHIS and NHANES Data Sets**

	NHIS Healthy						NHANES BMI 10 years Prior					
	25-59		60-69		70-95		25-59		60-69		70-95	
	HR	SE	HR	SE	HR	SE	HR	SE	HR	SE	HR	SE
<b>Conventional</b>												
Overweight	1.00	(.071)	1.07	(.085)	0.91	(.029)	1.02	(.157)	1.07	(.128)	1.08	(.052)
Class 1 Obese	1.40	(.076)	1.20	(.097)	0.97	(.038)	1.34	(.219)	1.27	(.183)	1.38	(.068)
Class 2/3 Obese	1.90	(.107)	1.72	(.127)	1.24	(.059)	2.15	(.228)	2.16	(.245)	1.49	(.086)
<b>Selection Correction</b>												
Overweight	1.03	(.067)	1.17	(.103)	1.04	(.129)	.89	(.170)	1.16	(.137)	1.37	(.163)
Class 1 Obese	1.50	(.080)	1.54	(.131)	1.41	(.163)	1.11	(.238)	1.39	(.214)	2.02	(.243)
Class 2/3 Obese	2.09	(.103)	2.74	(.179)	2.68	(.237)	1.83	(.245)	2.60	(.267)	2.63	(.326)

Abbreviations: NHIS National Health Interview Survey, NHANES National Health and Nutrition Examination Survey, HR Hazard Ratio, SE Standard Error, BMI Body Mass Index.

All models control for sex, race/ethnicity, educational attainment, and marital status. NHIS Healthy limited to nonsmoking respondents with self-rated health very good or excellent. NHANES controls for smoking status.

Models correcting for selection further control for age-at-survey and BMI X age-at-survey.

**Table 6. Effects of Age-at-Survey on All-Cause Mortality Risk**

	NHIS Full Sample		NHIS Healthy Sample		NHANES 1988-1994, 1999-2004		NHANES 10yrs Prior	
	b	SE	b	SE	b	SE	b	SE
Age-at-Survey	-.0007	(.002)	-.031	(.003)	NS		NS	
Age-at-Survey <sup>2</sup>	-.0001	(2.6E-5)	NS		NS		NS	
Overweight*Age-at-Survey	-.004	(.001)	-.004	(.004)	-.016	(.006)	-.012	(.006)
Class 1 Obese*Age-at-Survey	-.015	(.001)	-.011	(.005)	-.021	(.006)	-.021	(.009)
Class 2/3 Obese*Age-at-Survey	-.024	(.001)	-.023	(.007)	-.028	(.008)	-.028	(.014)

Abbreviation: NHIS, National Health Interview Survey. NHANES, National Health & Nutrition Examination Survey.

**Table 7.** Two-way Effects of Age-at-Survey X BMI on Mortality Risk from Circulatory Diseases and Accidents in Full NHIS Sample, 1986-2004

	<b>Circulatory Diseases</b>		<b>Accidents</b>	
	b	SE	b	SE
Age-at-Survey	.011	(.002)	-.027	(.007)
Overweight*Age-at-Survey	-.009	(.002)	-.003	(.006)
Class 1 Obese*Age-at-Survey	-.026	(.002)	-.007	(.008)
Class 2/3 Obese*Age-at-Survey	-.037	(.003)	-.007	(.011)

Figure 1

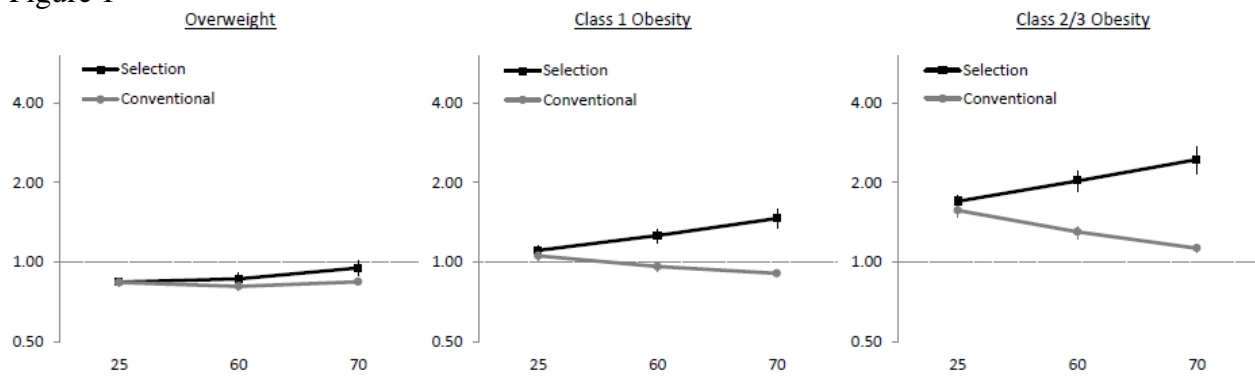


Figure 2

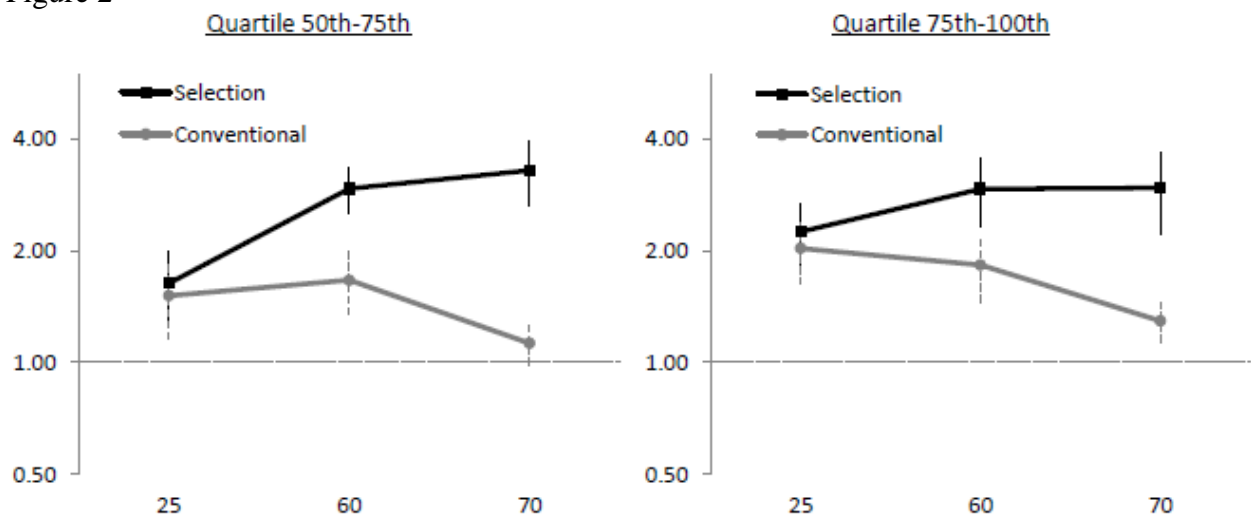




Figure 3

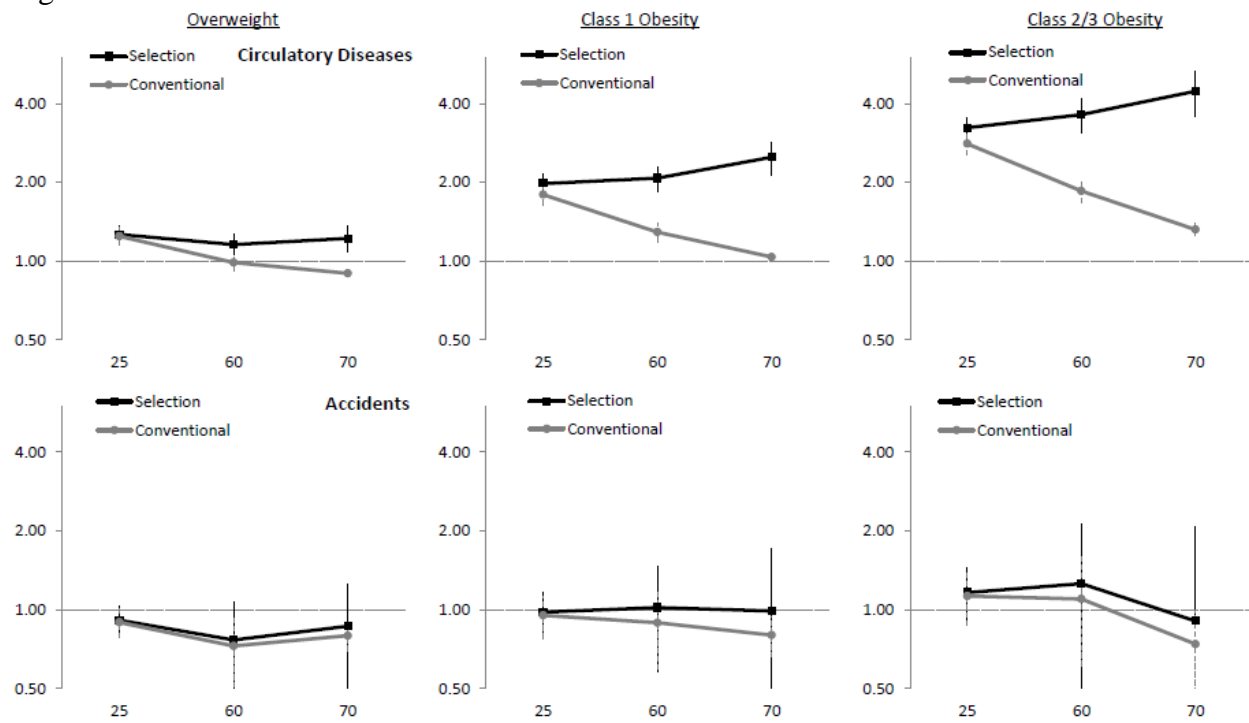


Figure 4

