

**Obesity and U.S. Adult Mortality:
A Bigger Picture of the Growing Threat***

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Context: The association between obesity and mortality risk is widely studied in the United States, but not fully understood. A key limitation of existing studies is that no analysis of the obesity-mortality relationship has fully accounted for the temporal dimensions of the U.S. obesity epidemic.

Objective: To estimate the percent of deaths in the U.S. black and white men's and women's adult populations between 1986 and 2006 associated with obesity by examining the combined effects of (1) age variation in the obesity-mortality relationship and (2) age-specific cohort trends in obesity prevalence.

Design, Setting, and Participants: Nineteen cross-sectional, nationally representative waves of the National Health Interview Surveys 1986-2004 linked to individual mortality records at the National Death Index (NDI) through 2006 were examined for age and cohort patterns in the link between obesity and adult mortality risk.

Main Outcome Measures: Percent of U.S. adult mortality attributable to obesity between years 1986 and 2006. Intermediate outcomes were individual-level all-cause mortality risk in the National Health Interview Survey-Linked Mortality Files (NHIS-LMF), 1986-2006. Additional outcomes were five-year cohorts' age-specific obesity prevalences between ages 35 and 85 during the time period 1986-2004.

Results: The percent of U.S. deaths between ages 35 and 85 associated with obesity between 1986 and 2006 was estimated to be 7.45 and 9.86 percent for the black and white male populations, respectively, and 20.13 and 16.64 percent for the black and white female populations, respectively. The percent of U.S. adult deaths associated with obesity will likely rise

in the future as older age groups become increasingly composed of cohorts with higher obesity prevalence.

Conclusions: The relationship between U.S. adult mortality and obesity is significantly conditioned by the temporal dimensions of the obesity epidemic. This fact has been overlooked in prior analyses, an omission which has misrepresented the impact of the obesity epidemic on U.S. mortality trends. Overall, obesity is associated with a large proportion of U.S. adult deaths, and this association is likely to rise in the future as cohorts with higher obesity prevalence age across their respective life courses.

INTRODUCTION

There is now ample evidence and widespread agreement that a massive epidemic of obesity has spread across the United States.¹ What is less clear and much more thoroughly debated are the public health consequences the epidemic confers. Concern over the epidemic is spurred by evidence of strong links between obesity and disability and between obesity and life threatening diseases such as diabetes, heart disease, stroke, and multiple cancers. In this view, the public health consequences are probably already large and only likely to grow as the prevalence of obesity expands and the duration of exposure accumulates across cohorts. But another strand of investigation has invigorated a different view of the epidemic's public health consequences. This approach takes a seemingly straightforward gauge of the epidemic's impact by assessing the link between obesity and U.S. adult mortality risk. The results of these inquiries are startling in that they appear to directly challenge claims that the epidemic has or will induce a toll on the public's health. The obesity mortality association is not as large as expected, often appearing to be robust only in the extremely obese ($BMI \geq 35.0$). Moreover, the association is estimated to diminish with age so as to suggest, for example, that for middle aged people (40-65) obesity is a significant risk factor for mortality, but for the older aged (65+) obesity is not significantly associated with increased mortality risk – or, as some evidence suggests, might even confer a survival advantage.²⁻⁶ For some investigators, the accumulated evidence concerning the obesity-mortality link reveals that stated public health concerns were hyped – more of a “moral panic” than a “public health crisis”.⁷

Indeed, above all factors driving the public health debate over obesity in the United States is the fact that the number of U.S. deaths associated with obesity is unknown.⁸ On one side

of this debate, obesity has been blamed for a large and growing share of premature deaths.⁹⁻¹³ On the other side, rising obesity prevalence is thought to pose little threat to secular trends in U.S. longevity because many investigations show the obesity-mortality link to be substantively weak.¹⁴⁻²⁰ The debate is largely fueled by widely divergent estimates of two important factors: (1) the individual-level association between obesity and mortality risk – that is, divergent estimates of hazard ratios (or the “effect” of obesity), and (2) varying estimates of U.S. obesity prevalence – that is, the population’s exposure to the “effect”. We argue that existing estimates of both factors are biased, but for different reasons. On the one hand, analyses estimating survival differences between the obese and normal weight populations have failed to account for confounding influences of cohort differences in mortality risk and age-related survey selection biases. On the other hand, existing estimates of population attributable fractions (PAFs) of obesity on U.S. adult mortality have used average obesity rates across age groups, thereby overlooking a great deal of heterogeneity in cohorts’ obesity rates. As a result, existing population attributable fractions (PAFs) of obesity on U.S. adult mortality rest on *questionable* hazard ratios combined with *average* prevalence rates of obesity.

Accurately estimating the population-level association between obesity and adult mortality risk is critical to understanding and addressing the health consequences of the U.S. obesity epidemic. It is certainly the case that policy makers need a more precise assessment of the aggregated toll obesity takes on U.S. mortality than the extensive bounds currently provided by existing research. In this paper we suggest that extant research on obesity-related mortality in the United States is fundamentally limited in both its thinking and analyses because it has not employed a cohort perspective to understand the link between the U.S. obesity epidemic and population-level mortality. We discuss this limitation and then estimate the percent of U.S. adult

mortality associated with obesity by accounting for (1) age variation in the effect of obesity on individual-level mortality risk, and (2) cohort variation in obesity prevalence.

The Association between Obesity and Individual-level Mortality Risk

We begin with the contention that the obesity-mortality association grows substantively stronger with increasing age. Surprisingly, the association between obesity and U.S. adult mortality risk has proven difficult to estimate, despite strong evidence linking obesity to a number of physical disabilities and diseases.²¹⁻³⁸ Data limitations remain a chief obstacle to good estimates of the U.S. obesity-mortality relationship. Because the U.S. National Vital Statistics System (NVSS) does not obtain information on the deceased's height and weight health researchers have overwhelmingly relied on survey data linked to official death records in order to investigate mortality differences between the obese and normal-weight populations. Consequently, various data sources, different measures of obesity, and inconsistent analytical methods have produced widely divergent estimates of the U.S. obesity-mortality association.⁹⁻²⁰ Indeed, the existing evidence linking obesity and mortality is quite mixed, with some findings indicating that only extreme obesity ($BMI \geq 35.0$) is significantly associated with increased mortality risk.^{17,18} And while much work has established the "J"-shaped functional form of BMI's relationship with mortality risk, age variation in this functional form is rarely explored. In studies that model age differences in the relationship, the substantive strength of obesity's association with mortality risk has been found to diminish with age.²⁻⁶ These findings are consistent with other bodies of evidence suggesting that relative risks associated with many risk factors (e.g., low levels of educational attainment) might decline with age as well.^{39,40} In fact,

some research has estimated that obesity status is associated with lower mortality risk at older ages, thereby awarding a survival advantage to the elderly obese population.⁶ Such findings have prompted some to argue that rapid increases in obesity rates have received underserved attention as a public health concern.⁷ Alternatively, others argue that while estimated mortality risks between the obese and normal weight populations are relatively different the absolute differences are substantively small, and thus have little impact on population health and longevity.^{18,41}

Yet, there are both empirical and methodological reasons to question findings that suggest the obesity-mortality association is either substantively small and/or diminishes with age. For one, the leading causes of death at younger ages in the United States – accidental injury, assault, intentional self-harm – are not positively associated with obesity status, whereas the leading causes of death at older ages – malignant neoplasms, diseases of the heart, chronic lower respiratory disease, cerebrovascular disease, diabetes mellitus – are significantly and substantively associated with obesity status.^{42,43} Further, obesity has been shown to be an important factor in the onset of frailty at older ages,⁴⁴ acting through multiple pathways such as inflammation,⁴⁵ insulin sensitivity,⁴⁶ oxidative stress, and the release of free fatty acids.⁴⁷⁻⁴⁹ Functional deterioration associated with comorbidity of these processes has been linked to increased risk of accelerated physical decline, disability, and death.^{49,50} In short, obesity is significantly associated with processes related to the leading causes of death at older ages, while unrelated to factors associated with the leading causes of death at younger ages. Thus, we might expect obesity status to be more strongly associated with mortality risk at ages wherein the proximate causes of such risk are more strongly associated with obesity. And yet this expectation is not supported by existing research.

However, the vast majority of evidence suggesting that the obesity-mortality relationship diminishes with age come from survival analyses that model the two-way interaction between age and obesity – or stratify analyses by age groups – without controlling for the confounding influences of respondents’ ages-at-survey and/or years of birth (i.e., cohort). These are critical omissions because using “attained age” as the time-metric in survival models captures variation in mortality risk associated with biological processes linked with *aging* (e.g., senescence⁴⁹), variation associated with *age-at-survey* (e.g., age-related survey selection biases, or “healthy participant effects”⁵¹), and variation associated with *cohort* membership (e.g., different “cohort morbidity phenotypes”⁵²).

To date, no analysis has accounted for these different time dimensions when modeling age patterns in the relationship between obesity and U.S. adult mortality risk. Yet there are reasons to believe that age-specific estimates of the obesity-mortality relationship may be affected by respondents’ ages-at-survey and cohorts, regardless if the survey data are cross-sectional or the product of a prospective cohort study. On the one hand, “healthy participant effects” have been shown to be strong in survey data, and we might expect confounding from survey selection to be especially strong when estimating age-varying health outcomes – particularly in data obtained from the non-institutionalized population such as the NHIS.⁵² That is, surveys’ exclusion criteria are strongly associated with respondents’ ages and health status, and thus are especially confounded with estimates of age patterns of health outcomes. And regarding the question at hand, exclusion criteria are likely even more pronounced in the U.S. obese population. For instance, the non-institutionalized older-aged obese population that is healthy enough to fully participate in the NHIS is likely composed of more robust members than the older-aged institutionalized obese population. We hypothesize that the confounding of

institutionalization and differential frailty on age-specific mortality risk is relatively greater in the obese population than in the normal-weight population, and, thus, survey selection bias in the NHIS will be stronger in the obese sample than in the normal-weight sample.

Regarding the importance of cohort confounding, recent research has shown temporal reductions in U.S. adult mortality rates to be overwhelmingly related to cohort, not period, phenomena.⁵³⁻⁵⁵ And because age and cohort are highly collinear the estimated age patterns of hazards observed in survey data may be partly due to differences between cohorts' mortality risks.⁵⁴ Thus, analyses of age patterns of mortality risk using survey data such as the NHIS must account for cohort variation in mortality risk, especially when exploring age variation in mortality.

In the current study, a series of three Cox survival models were estimated to investigate age patterns in the association between obesity and U.S. adult mortality risk. We first estimated Cox proportional hazard models, which assume no age variation in the obesity-mortality association. We then estimated Cox hazard models in which the obesity-mortality association varies by 10-year age groups, but did not account for age-at-survey or birth cohort. Lastly, we re-estimated the obesity-mortality association across the 10-year age groups accounting for variation in five-year birth cohorts as well as obesity differences in age-related selection bias. Several alternative models were estimated to explore age-variation in the relationship between obesity status and individual mortality hazards. For instance, we stratified Cox regression models by obesity status and obtained obesity-specific smoothed baseline hazards. The baseline hazards for the class 1 and class 2/3 obese samples were then compared with the normal/overweight baseline hazards to estimate obesity hazard ratios across age. Because these hazard ratios were found to increase with age, we proceeded to estimate a full model that accounted for age-

variation in the obesity-mortality association. Furthermore, we conducted several sensitivity analyses by limiting age-at-survey to single-year and three-year age-bands and re-estimated the Cox models. That is, we explored age-variation in the relationship between obesity status and mortality at select ranges of age-at-survey, effectively holding constant the ages at which respondents were surveyed. Specifically, we estimated the models only for respondents aged 55-57; 61-63; 67-69; and 73-75 at time of survey to guard against extrapolating the linear estimates of age-at-survey in the final model. Estimates showed that variation associated with obesity status in men's and women's mortality hazard increased with age for each specific sub-sample. We are confident, therefore, that we are not forcing or extrapolating the estimate of age-at-survey in our models. All analyses and results are available upon request.

Results from these preliminary analyses provide evidence suggesting the need to properly account for mortality risk variation in *age-at-survey*, variation in *cohort* membership, and variation in *aging* when estimating survival models. Failing to do so significantly biases estimates of age patterns in the obesity-mortality association in the United States. In fact, inconsistent with existing evidence the results suggest that the association between obesity status and individual-level mortality risk grows substantively stronger with age once obesity-related variation in age-at-survey and cohort variation in mortality are accounted for. As such, we proceeded to estimate Royston-Parmar survival models to contrast instantaneous age hazards of the normal/overweight, class 1 obese, and class 2/3 obese samples. The details of these models are later discussed in the methods section.

A Cohort Perspective of the Obesity Epidemic

Existing research suggests that the U.S. obesity epidemic is largely a period phenomenon, but evidence also shows that increases in obesity prevalence is heavily concentrated in more recent birth cohorts.^{56,57} It is also important to note that the cumulative person-years one spends obese ultimately determines the substantive effect that obesity has on individual-level mortality risk.⁵⁸ It therefore is essential to consider cohorts' disparate exposures to the obesity epidemic when estimating the effect of obesity on current and future population health. The relationship between obesity and mortality risk might vary across cohorts in at least two ways. One, rising obesity prevalence across cohorts will increase obesity's impact on aggregate U.S. mortality rates even if the effect of obesity on individual-level mortality risk remains constant across time and/or cohorts. That is, the simple fact that increasing proportions of U.S. cohorts are obese will increase the cumulative toll obesity is taking on aggregate mortality as cohorts with higher rates of obesity age across their respective life courses. Two, the effect of obesity on mortality risk might be changing across cohorts. For instance, the effect might be lessening across cohorts because the ability to manage obesity might improve with the use of new medical and pharmacological technologies and/or surgical procedures.^{14,19} More recent cohorts' greater exposure to such advances could lessen the deleterious effects of obesity on their health and mortality risk. Conversely, the relative effect of obesity on mortality risk might be increasing across cohorts due to the fact that duration of obesity is greater in recent cohorts than in older cohorts. That is, recent cohorts have endured higher rates of obesity across longer segments of their life courses, which could strengthen the association between obesity and mortality risk.⁵⁸ Overall then, obesity possibly affects a cohort's mortality risk through both its effect and exposure to this effect. In this paper we account for the former of these two cohort processes by combining cohorts' age-specific obesity rates with age-specific estimates of the obesity-mortality

association. That is, we calculate cohort-specific population-attributable risk fractions (PAFs) of obesity on U.S. adult mortality between 1986 and 2006 by accounting for: (1) age-variation in the effects of class 1 and class 2/3 obesity on mortality risk, and (2) cohort variation in age-specific obesity prevalence. Accounting for these temporal dimensions permits a more accurate estimate of the percent of U.S. deaths associated with obesity status than previous studies have afforded.

METHODS

Survey

We use data from 19 continual waves of the National Health Interview Surveys (NHIS), 1986 through 2004, linked to follow-up mortality information for all eligible respondents through December 31st, 2006. This linked dataset was made publicly available by the National Center for Health Statistics (NCHS), as well as the Minnesota Population Research Center in the form of the Integrated Health Interview Survey (IHIS). The NHIS uses a multistage probabilistic sampling design, and respondents of the NHIS were matched to the computerized mortality records of the National Death Index using a 14-item identification scheme.¹³ Respondents of the NHIS not eligible for matches to death records were dropped from the final sample, and accounting for the clustered sampling design of the NHIS and using analytic weights make results representative of the U.S. non-Hispanic black and non-Hispanic white non-institutionalized adult population.

Outcome and Measures

Survival times were measured by respondents' attained ages at either time of death or censoring on December 31st, 2006. Respondents' ages at time of survey were estimated using quarter-year of birth and quarter-year of survey, and respondents' survival times from time of survey were estimated in quarter-years as a function of either censoring time or quarter-year of death. Respondents younger than age 25 at time of survey were omitted to allow sufficient time to achieve all levels of educational attainment, and respondents aged over 84 years were dropped to safeguard against bias induced from open-interval age coding in the NHIS. Respondents with a BMI < 18.5 were also dropped from the sample because an "underweight" BMI is often indicative of preexisting illness, frailty, and/or increased mortality risk. Lastly, cases with missing values on height, weight, educational attainment, income level, marital status, or region of residence were omitted from analyses, and due to low frequencies attained ages greater than 99.9 were also deleted. Analytical samples were stratified by sex and race/ethnicity, resulting in sample sizes of 289,514 non-Hispanic white men; 40,169 non-Hispanic black men; 314,981 non-Hispanic white women; and 58,615 non-Hispanic black women (other race/ethnicity groups were omitted from analyses due to small sample sizes).

Educational attainment was categorized as less than highschool, highschool graduate, some college, and bachelor's degree or higher, and family income was categorized as earning less than the federal poverty line, greater than poverty but less than \$45,000; and \$45,000 or greater. Marital status was measured using the four categories married, divorced/separated, never married, and widowed, and region of residence was measured as Northeast, South, Midwest, and West. Respondents were coded as "obese" if their BMI was calculated to be greater than or equal

to 30.0, and the obesity category was further analyzed as “Class 1” (BMI 30.0-34.9) and “Class 2/3” obesity (BMI \geq 35.0). The reference category is composed of respondents with a “normal weight” BMI (18.5-24.9) or an overweight BMI (25.0-29.9) as it has been argued that being overweight is not a significant risk factor for increased mortality risk.¹⁸ NHIS respondents’ self-reported weight was adjusted in two stages to account for (1) underestimates of weight by respondent proxies and (2) to standardize age-specific annual weight to clinical measures reported in NHANES. We used procedures advanced by Reither and Utz (2009) to adjust for proxy-reported weight and standardizing procedures made available by Reither (2005) to adjust NHIS self-reported weight to measured weight in NHANES.^{59,60}

[Table 1 About Here]

Data Analysis and Statistical Methods

Hierarchical Age-Period-Cohort (HAPC) Cross-classified Fixed (CCFEM) and Random Effects models (CCREM) were used to estimate age, period, and cohorts effects on the likelihood of overweight, class 1 obesity status, and class 2/3 obesity status for the U.S. black and white male and female populations between 1986 and 2006.^{61,62} These models assume a binomial distribution for the likelihood of the outcome, with a logit transformation to make linear the binomial response mean as a generalized linear model. A quadratic functional form of age was modeled, and single-year dummy variables of survey year and five-year dummy variables of birth cohort were included to account for temporal dimensions of the obesity epidemic. These models were estimated using Stata 12’s *xtnlogit* and *logit* programs. Due to space limitations, tabulated results are not shown, but sex-specific graphical results of temporal

trends in age-specific obesity rates are presented in Figure 1. Fitted values of sex- and race/ethnic age-specific rates of class 1 obesity and class 2/3 obesity from sex- and race/ethnicity-specific samples were estimated across five-year cohorts. These cohorts' age-specific rates of overweight, class 1 obesity, and class 2/3 obesity were used to estimate population attributable fraction (PAFs) for obesity's effect on mortality of the U.S. non-Hispanic black and non-Hispanic white male and female populations between 1986 and 2006.

Royston-Parmar survival models were used to estimate obesity-specific hazards for each sex-race/ethnicity subsample between ages 25.0 and 99.9 across the time period 1986 through 2006. Attained age was used as the time metric and age-at-survey was included in all models to account for late-entry bias. Analyses were stratified by sex and race/ethnicity and models were estimated using Stata 12's *stcox* and *stpm2* programs.⁶³ Age-specific fitted hazard ratios for the class1 and class 2/3 obese populations were combined with cohorts' age-specific class 1 and class 2/3 obesity prevalences to calculate the amount of U.S. adult mortality between ages 35 and 85 associated with obesity across ages and cohorts. These products were then standardized by the age distribution of mortality in each race/ethnicity-sex sample. In short, to accurately estimate the population-attributable risk fraction of obesity on U.S. adult mortality, we build-in sex, race/ethnicity, age, and cohort effect modifiers in the PAF equation.⁶⁴ Specifically, we use the following formula to estimate population-attributable risk fraction (PAF) of obesity on mortality:

$$PAF_{ik} = \left[pd_{ik} \left(\frac{HR_{class1_{k-1}}}{HR_{class1_k}} \right) \right] + \left[pd_{ik} \left(\frac{HR_{class23_{k-1}}}{HR_{class23_k}} \right) \right] \quad (1)$$

where PAF_{ik} represents the PAF for the exposed population at k th age and i th five-year birth cohort; pd_{ik} is the fraction of total deaths that are exposed to the i th five-year birth cohort at age k , and HR_{class1_k} is the hazard ratio of class 1 obesity mortality risk to normal weight mortality risk at age k , and $HR_{class23_k}$ is the hazard ratio of class 2/3 obesity mortality risk to normal

weight mortality risk at age k . This PAF equation is separately applied to the non-Hispanic white and non-Hispanic black male and female samples of the NHIS-LMF 1986-2006 to generate sex- and race/ethnicity-specific PAFs across age and cohort.

RESULTS

Cohort-variation in Obesity Prevalence

Accounting for the combined effects of age, period, and cohort on the risk of obesity reveal significant cohort variation in the age patterns of U.S. obesity prevalence. Due to space limitations, tabulated results of HAPC-CCFEM and HAPC-CCREM analyses of obesity status are not shown but are available upon request. Here, in Figure 1, we present fitted probabilities of overall obesity ($BMI \geq 30.0$) across age and cohorts for U.S. men and women to illustrate the degree of cohort variation in U.S. adult obesity prevalence.

[Figure 1 About Here]

Substantial variation in age-specific patterns of obesity prevalence across birth cohorts is clearly evident. While the average prevalence of obesity in adult men and women in the NHIS-LMF 1986-2006 is 20.12 percent and 23.47 percent, respectively, we see a great deal of cohort heterogeneity in age-specific obesity rates (average prevalence indicated by blue horizontal line). Not accounting for such heterogeneity when calculating population attributable fractions for obesity as a cause of mortality biases estimates.⁶⁴ Most important for assessing immediate and long-term effects of obesity on adult survival are the substantial cohort differences in age patterns of obesity prevalence. These are not “subtle shifts” in BMI levels over time, but rather

are rapid cohort changes in obesity prevalence that indeed constitute an epidemic.⁷ For instance, U.S. Men's fitted obesity prevalence at age 61 for the 1920-1924 birth cohort is 17.3%, but the same age-specific prevalence is 24.2% and 33.3%, respectively, for the 1930-1934 and 1940-1944 birth cohorts. Researchers' use of average prevalence in calculating population-attributable risk fractions (PAFs) for obesity's effect on U.S. mortality fail to build in the much needed life course and cohort perspectives of the obesity epidemic, and consequently washes out the substantial differences in age-specific prevalences across cohorts.^{9-11,14,15} We must consider the composition of individual cohorts as a "third dimension" of the obesity epidemic in order to assess the life course effects of obesity on mortality risk.⁶⁰ Failing to do so by using average annual obesity prevalence underestimates the degree of obesity in the population. The U.S. obesity epidemic is indeed a real phenomenon, and it manifests itself most evidently across different cohorts' life courses.

Obesity and Individual-level Mortality Risk

Online Table S1 presents results from Royston-Parmer analyses of U.S. men's survival across ages 25 to 99.9 during the time period 1986 to 2006, and online Table S2 presents respective results for U.S. women. Estimated hazard ratios between the class 1 and class 2/3 obese populations and the normal weight population are presented from survival models that control for educational attainment, income level, marital status, region of residence, age-at-survey, and birth cohort. Results indicate that class 1 obesity and class 2/3 obesity are significantly associated with U.S. men's and women's adult mortality, and that the strength of the association varied substantially by age, sex, and race/ethnicity. Fitted age-specific hazard

ratios from the Royston-Parmar models are graphically depicted by race/ethnicity and sex in Figure 2.

[Figure 2 About Here]

Our results are inconsistent with the majority of existing findings, in that they estimate the association between obesity status and mortality risk to grow substantively stronger with increasing age. These age patterns became evident only after controlling for obesity variation in the effect of respondents' ages-at-survey on mortality risk, which suggests that age-related survey-selection biases the estimates of obesity's effect on age-specific mortality risk.

Percent of U.S. Deaths Associated with Obesity

Age-specific obesity hazard ratios between ages 35 and 85 were combined with cohorts' age-specific rates of class 1 and class 2/3 obesity and then standardized by the age distribution of deaths to estimate the percent of U.S. men's and women's mortality attributable to each class of obesity.

[Table 2 About Here]

Estimates of the percent of U.S. deaths between ages 35 and 85 associated with class 1 class 2/3, and overall obesity in the black and white male and female populations are presented in Table 2. We found that 7.45 [2.31,13.55] percent and 9.86 [7.32,12.36] percent of deaths occurring between ages 35 and 85 in the black and white male population between 1986 and 2006 were associated with obesity, respectively. Furthermore, 20.13 [10.75,30.29] percent of deaths in the black female population and 16.64 [12.94,20.62] percent of deaths in the white female population were associated with obesity across the respective age-range and time period.

Thus, our findings suggest that the amount of U.S. adult mortality related to obesity is higher than recent estimates,^{17,18} and also that they are inherently tied to the cohort dimensions of the obesity epidemic.⁶⁵ Indeed, cohort variation in PAF estimates is depicted in Figure 3. Patterns of age-specific PAFs for five-year cohorts born between 1905 and 1945 between ages 50 and 85 show that the percent of deaths from class 2/3 obesity at all ages is increasing across subsequent birth cohorts.

[Figure 3 About Here]

Given the increasing prevalence of obesity across successive birth cohorts, the percent of U.S. adult mortality attributable to obesity is likely to increase in the near future. That is, the U.S. population is increasingly composed of birth cohorts with relatively higher rates of obesity. If the association between obesity status and mortality risk remains at the age-specific levels observed across the 1980s, 1990s, and 2000s then the aggregate toll of the U.S. obesity epidemic will increase in size.

COMMENT

Recent work has shown the need to account for "three dimensions" of time when estimating population health patterns and forecasting health and mortality trends.⁶⁵ Specifically, we must be particularly mindful of significant cohort variation in health outcomes in addition to the more common accounting for variation across age and time periods. Here, we showed that while increases in U.S. obesity rates have occurred across time periods, the way by which obesity status affects age-specific mortality risk unfolds across cohorts in unique ways. Consequently, in order to accurately estimate population attributable fractions for obesity as a

cause of U.S. mortality requires one to consider these cohort dimensions of the U.S. obesity epidemic. As a result, we see that the effect of obesity on U.S. adult mortality is unfolding across cohorts in significantly different and substantive ways.

We also find evidence suggesting that the association between obesity status and individual mortality risk varies significantly with both age-at-survey and attained age, and also that cohort variation in mortality is significantly associated with estimates of age-specific mortality hazards. Consequently, survival models must account for all these temporal dimensions in order to accurately estimate age patterns in the association between obesity status and mortality risk. Heretofore research, however, has not accounted for these confounders and, as a result, the bulk of existing public health literature has suggested that the association between obesity status and mortality risk is either age-invariable or substantially weakens with increasing age. Our results show that both of these findings stem from survival models that incorrectly estimate the obesity-mortality relationship in the United States. After accounting for age-related survey selection bias and cohort differences in mortality risk, the associations between class 1 and class 2/3 obesity and U.S. men's and women's adult mortality risk grow substantively stronger with age.

Taken together, our findings add to the obesity-mortality literature in four substantial ways. One, we build on recent research by demonstrating the need to use cohort-based prevalence of obesity when estimating the percent of U.S. mortality attributable to obesity.⁶⁵ Two, we show significant age-, cohort-, race/ethnic-, and sex-based variation in the obesity-mortality relationship. Three, our findings support earlier contentions that obesity accounts for a large share of U.S. adult mortality, accounting for an estimated percent of U.S. men's and percent of U.S. women's deaths between ages 35 and 85 during the time period 1986 to 2006.

And four, the cumulative effect of the obesity epidemic on U.S. adult mortality will likely increase in the coming future, as younger cohorts with higher obesity prevalence age across their respective life courses.

As such, our results have three implications for future research. One, consistent with previous calls, we believe health researchers and demographers must factor cohort forces into assessments and projections of population health. Two, barring a revolutionary medical intervention that reduces obesity's effect on mortality risk, and/or highly improbable reductions in childhood and adolescent obesity, U.S. mortality attributable to obesity is likely to increase in the near future. And three, projections of U.S. life expectancy ought to heed prior calls to adjust estimates to account for the continued toll obesity will have on life expectancy.¹²

TABLES AND FIGURES

Table 1. Descriptive Statistics of non-Hispanic black and non-Hispanic white Male and Female NHIS-LMF Samples, 1986-2006

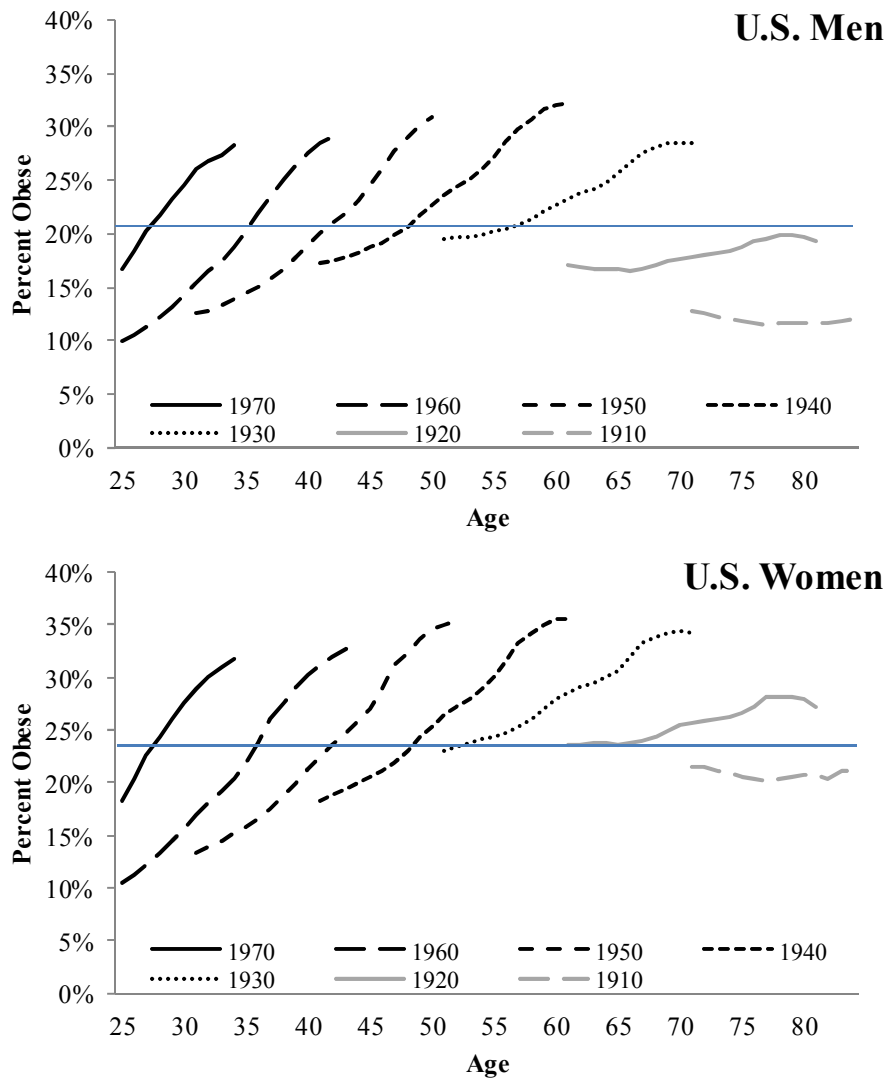
	Women				Men			
	<u>Black</u>		<u>White</u>		<u>Black</u>		<u>White</u>	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
Age at Survey	46.8	15.3	49.5	16.2	46.7	14.8	48.1	15.3
Age at Exit	58.9	14.7	62.1	15.3	58.5	13.9	60.5	14.2
Survey Year	1993.2	5.0	1992.9	4.8	1993.1	4.9	1992.8	4.7
Birth Year	1946.5	15.7	1943.4	16.4	1946.4	15.3	1944.7	15.6
Deceased	*15.2	35.9	15.2	35.9	21.0	40.7	18.0	38.4
<i>BMI</i>								
Normal/Overweight	60.7	48.8	76.4	42.4	78.3	41.2	80.9	39.3
Class I Obese	22.7	41.9	14.9	35.6	15.3	36.0	14.6	35.3
Class II/III Obese	16.6	37.2	8.7	28.1	6.4	24.5	4.5	20.7
<i>Region</i>								
Northeast	19.0	39.2	21.4	41.0	17.9	38.3	21.6	41.1
Midwest	20.2	40.1	26.6	44.2	19.6	39.7	28.1	44.9
South	51.8	50.0	34.5	47.5	52.2	50.0	31.1	46.3
West	9.1	28.7	17.4	37.9	10.2	30.3	19.2	39.4
<i>Income Level</i>								
Income<Poverty	31.1	46.3	10.6	30.8	19.1	39.3	7.4	26.3
Poverty<Income<\$45,000	50.2	50.0	53.6	49.9	55.5	49.7	51.7	50.0
Income>\$45,000	18.6	39.0	35.8	48.0	25.4	43.5	40.9	49.2
<i>Educational Attainment</i>								
< H.S.	27.8	44.8	15.4	36.1	29.2	45.5	15.6	36.3
H.S. Graduate	37.1	48.3	40.0	49.0	36.7	48.2	34.9	47.7
Some College	21.9	41.4	22.7	41.9	20.7	40.5	21.1	40.8
B.A.+	13.3	33.9	21.9	41.3	13.3	34.0	28.4	45.1
<i>Marital Status</i>								
Married	38.3	48.6	66.7	47.1	58.1	49.3	75.6	42.9
Never Married	23.5	42.4	8.4	27.8	20.9	40.6	12.2	32.7
Divorced/Separated	24.2	42.8	12.3	32.9	16.5	37.1	9.3	29.1
Widowed	14.1	34.8	12.5	33.1	4.5	20.8	2.9	16.8
N	58,615		314,981		40,169		289,514	

* All numbers listed below line are percentages.

Table 2. Percent of U.S. Adult Mortality between Ages 35 and 84 for Birth Cohorts 1905 to 1964, Associated with Class I and Class II/III Obesity, NHIS-LMF 1986-2006

	Class 1 Obesity		Class 2/3 Obesity		Total	
Black Women	6.02	(1.20,11.22)	14.12	(9.55,19.06)	20.13	(10.75,30.29)
White Women	8.97	(6.84,11.21)	7.67	(6.10,9.41)	16.64	(12.94,20.62)
Black Men	3.44	(0.31,7.05)	4.01	(2.00,6.50)	7.45	(2.31,13.55)
White Men	5.51	(3.93,7.18)	4.35	(3.38,5.45)	9.86	(7.32,12.63)

Figure 1: Obesity Prevalence of U.S. Men's and Women's Birth Cohorts Across Age, NHIS-LMF 1986-2006*



* Fitted Prevalence estimated using HAPC-CCFEM logit models

Figure 2. Class 1 and Class 2/3 Mortality Hazard Ratios, by Race/Ethnicity and Sex, NHIS-LMF 1986-2006

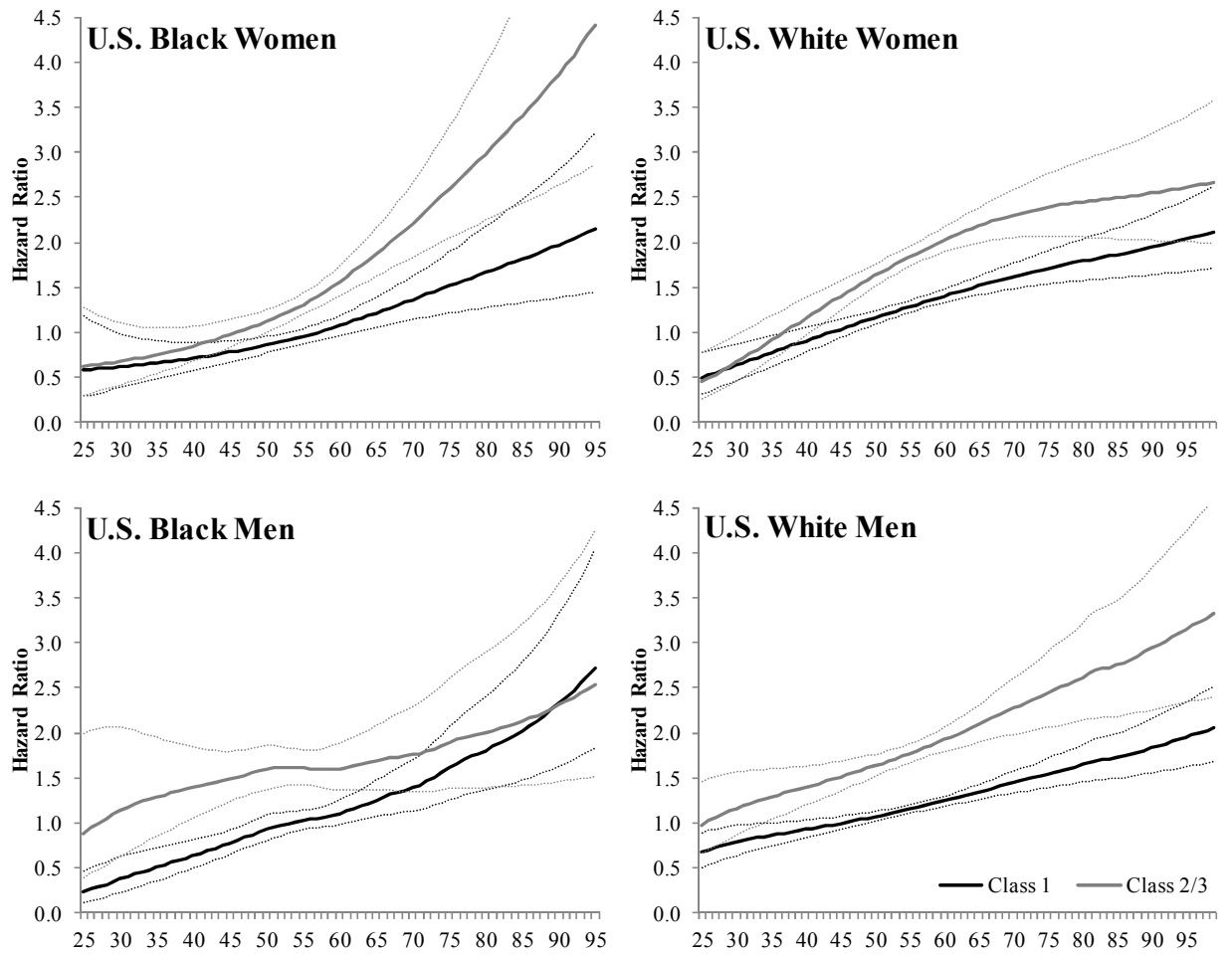
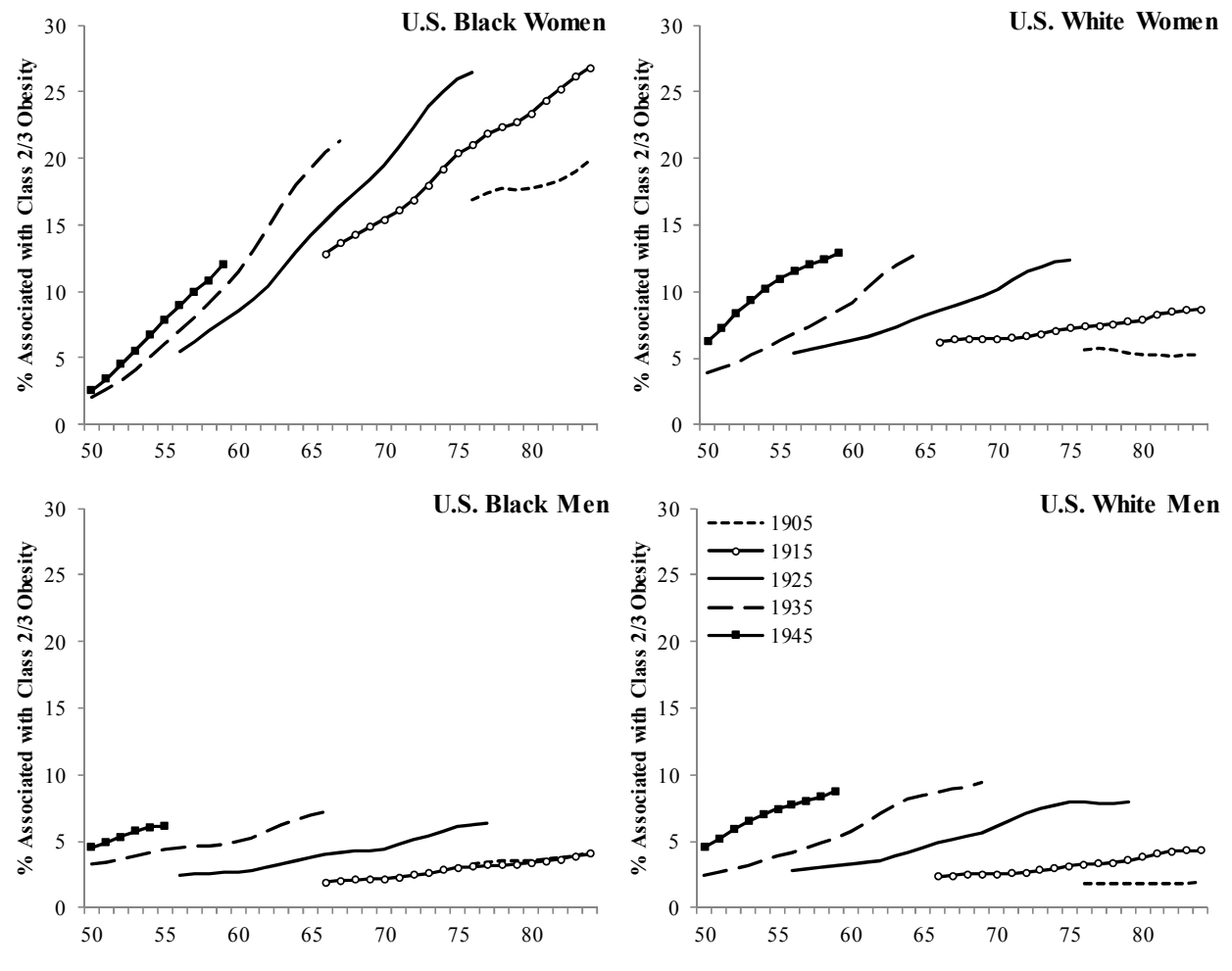
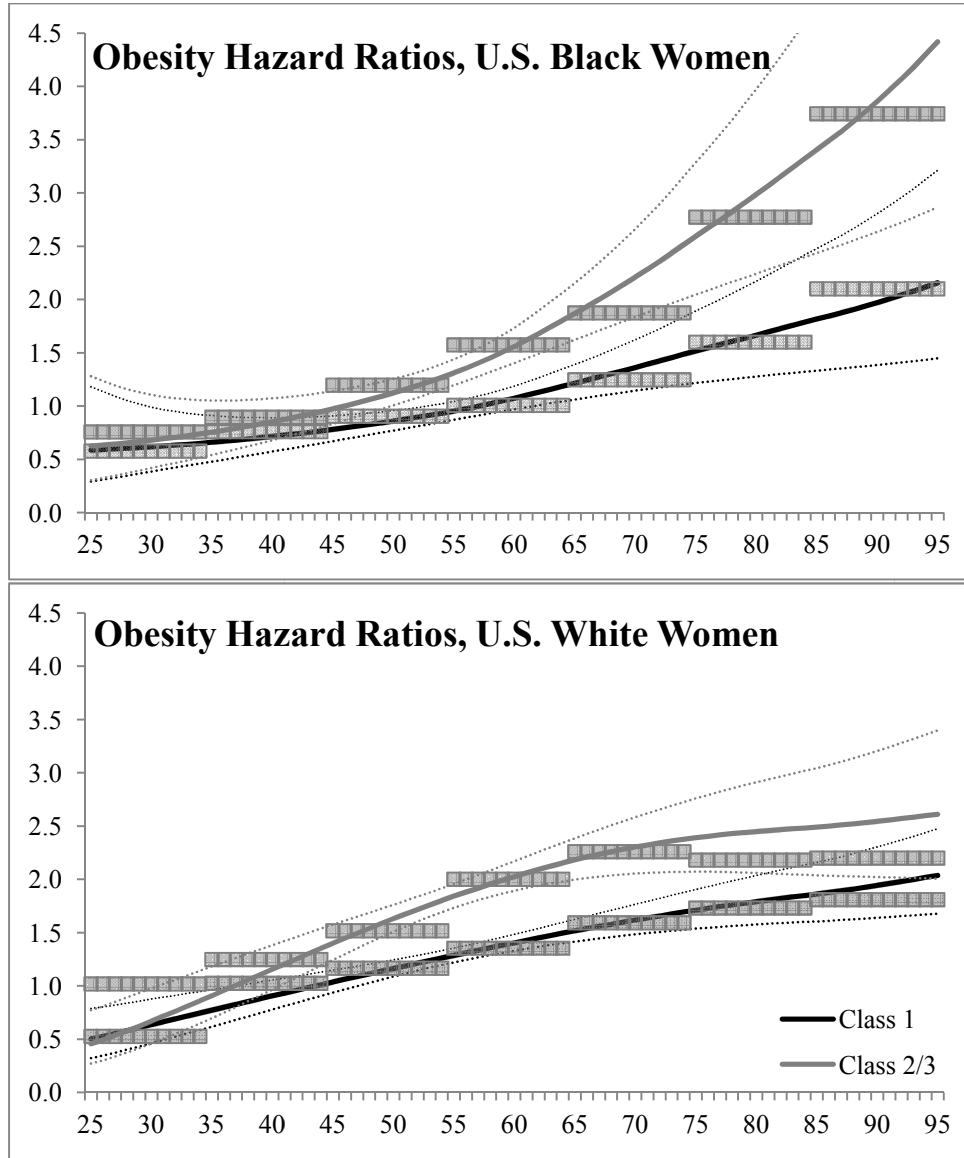


Figure 3. Class 2/3 Obesity PAFs across Age and Cohort, by Race/Ethnicity Sex, NHIS-LMF 1986-2006

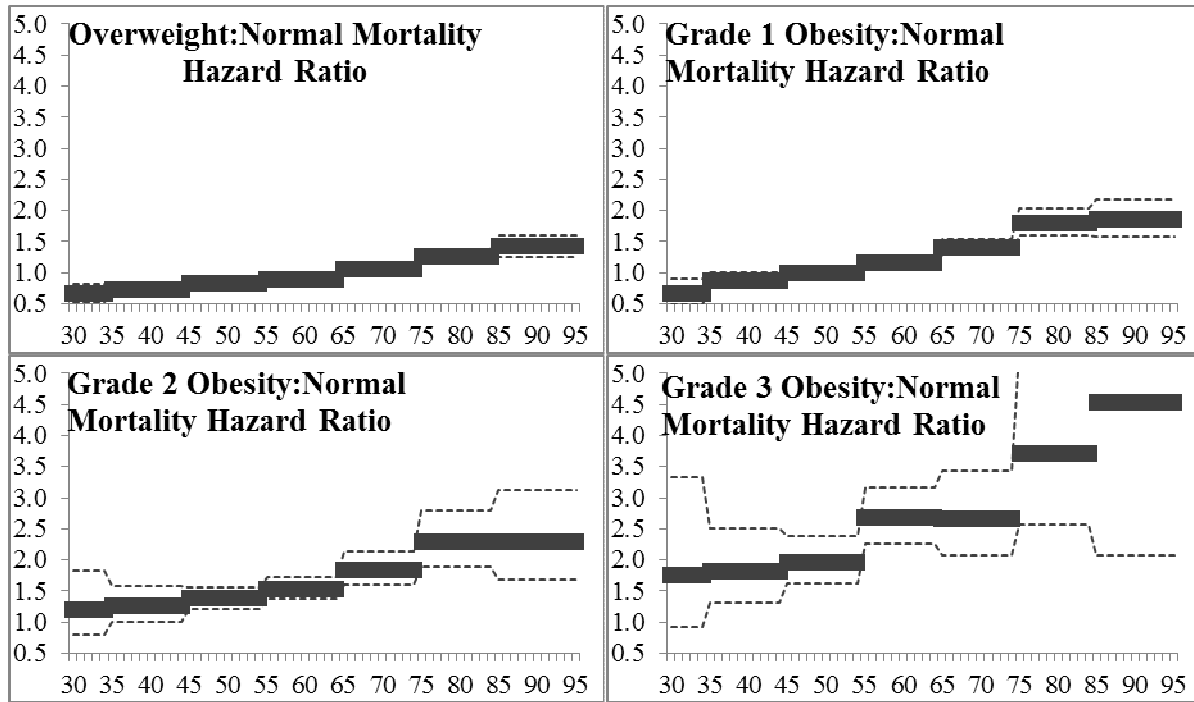


SUPPLEMENTAL MATERIAL

1. Sensitivity Analyses: Royston-Parmar Models' Estimates of Continuous Age Patterns of Obesity-Mortality Association Compared to Cox Regression using 10-year Categorical Age Groups



2. Sensitivity Analyses: Cox Regression using 10-year Categorical Age Groups with “normal weight” only as reference category (Men’s sample):



3. The three Cox regression models used to explore age-variation in the obesity-mortality relationship:

Model 1: Cox Proportional Hazard Model:

$$h_i(t) = h_0(t) \exp\left(\sum_k \beta_k O_{ik} + \mathbf{x}_i \beta_c\right), \quad (1)$$

where the hazard function for the i th individual, $h_i(t)$ is conditional on covariates \mathbf{x}_i and obesity class (O_k) and where β_k are the coefficients pertaining to the k th obesity class ($k=1,..,3$) and β_c is the vector of coefficients pertaining to \mathbf{x} .

Model 2: Cox Model with Grouped Age-dependency of Obesity:

$$h_i(t) = h_0(t) \exp\left\{\sum_j \sum_k \beta_{jk} A_{ij} \times O_{ik} + \mathbf{x}_i \beta_c\right\}, \quad (2)$$

The hazard function for the i th individual is conditional on covariates \mathbf{x}_i and the age x obesity interactions. β_{jk} , corresponds to the 21 possible combinations of the seven 10-year age groups $A_j = [25-35), \dots, [85+)$ and the 3 obesity classes $O_k=1, \dots, 3$, and β_c denotes the estimates of the remaining covariates (\mathbf{x}).

Model 3: Cox Model with Grouped Age-dependency of Obesity, Controlling for Age-at survey and Cohort:

$$h_i(t) = h_0(t) \exp \left\{ \sum_j \sum_k \beta_{jk} A_{ij} \times O_{ik} + \sum_\ell \beta_\ell C_{i\ell} + \sum_m \beta_m S_i \times O_{im} + \mathbf{x}_i \beta_c \right\}, \quad (3)$$

where β_{jk} is the vector of coefficients corresponding to the 21 interactions of age group and obesity class as specified in Model 2, β_l are the estimated coefficients of the 14 five-year birth cohort (C_l , $l = [1905-1909], \dots, [1970-1974]$), β_m are interactions between the centered linear age-at-survey term (S) (ranging from 25 to 84.99 and centered on mean values) and the obesity classes (O_m , $m = 1, \dots, 3$), and β_c corresponds to the coefficients of the remaining covariates (\mathbf{x}).

The time-metric in all survival models is attained age, and left truncation is accounted for by starting respondents' exposure at age-at-survey. The first two models are most often employed by researchers to investigate the association between obesity and adult mortality risk. Model (1) assumes the association between obesity status and mortality risk is proportional across age, while Model (2) assumes the association is conditional on one's age group. However, neither model accounts for variation in mortality risk by age-at-survey (i.e., age-related survey selection bias) or cohort membership. We estimate Model (3), which extends Model (2) to account for mortality variation in birth cohorts and obesity variation in the association between age-at-survey and mortality risk. The functional form of the relationship between age-at-survey and mortality risk is quadratic so we include both centered age-at-survey and centered age-at-survey squared.

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