"UNHEALTHY" RETURNS TO EDUCATION: VARIATION IN BMI-ASSOCIATED PREMATURE ADULT MORTALITY BY EDUCATIONAL ATTAINMENT

Iliya Gutin

A thesis submitted to the faculty at the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Masters of Arts in the sociology department in the school of liberal arts and sciences.

Chapel Hill
2016

Approved by:

Robert A. Hummer

Claire Yang

Ted Mouw

**ABSTRACT**

Iliya Gutin: "Unhealthy" Returns to Education: Variation in BMI-associated Premature Adult Mortality by Educational Attainment
(Under the direction of Robert A. Hummer)

While obesity continues to be a significant health issue, the relationship between body weight and mortality risk remains unclear. Research notes the strong association between obesity and higher mortality risk, along with the "protective" effect of higher weight for some groups. Few studies have examined this relationship when stratified by socioeconomic status, especially when considering *premature* mortality among working-aged adults. Using recent National Health Interview Survey data, this study examines variation in BMI-associated premature mortality risk across different levels of education. Results indicate overweight and class I obesity are associated with lowest mortality risk among the lower-educated. Conversely obesity is associated with increased mortality risk for individuals with a college education or greater, while overweight is not associated with reduced risk. Thus, obesity may pose a greater relative health risk in more advantaged groups, such as the highly educated, while other socio-behavioral factors account for premature mortality among lower-educated individuals.

# ACKNOWLEDGEMENTS

# TABLE OF CONTENTS

# LIST OF TABLES

## LIST OF FIGURES

# LIST OF ABBREVIATIONS

BMI   Body Mass Index

SES   Socioeconomic Status

HR   Hazard Ratio

RR   Relative Risk

SE   Standard Error

NCHS   National Center for Healt hStatistics

NDI   National Death Index

NHIS   National Health Interview Survey

LMF   Linked Mortality File

NHANES   National Health and Nutrition Examination Survey

# INTRODUCTION

Government agencies, medical organizations, and public health officials continue to stress the threat that the obesity and overweight epidemic poses to the United States population. At present, over 35% and 67% of US adults are classified as obese and overweight/obese, respectively (Ogden et al., 2014). The warnings are clearly substantiated by the considerable body of research reaffirming the association between individuals' bodyweight and many aspects of their health. However, what remains much less clear is the nature of the relationship between bodyweight (as often measured by Body Mass Index, or BMI) and mortality. For instance, the BMI range that the Centers for Disease Control and World Health Organization define as "normal" and "healthy" is not necessarily associated with the lowest risk of death.  Only recently have studies and national health statistics noted the existence of an "obesity paradox", whereby overweight and mildly obese individuals have significantly lower mortality risk as compared to their normal weight counterparts. Thus, a "J"- or "U"-shaped mortality risk curve is produced, with overweight individuals at the lowest point, or nadir, of this curve. While these findings do not prove that a higher BMI in the overweight range is favorable with respect to health, they certainly question some of the core assumptions about BMI and long-term health outcomes.

However, one limitation of much of the existing work on the obesity paradox and BMI-associated mortality is that it has largely focused on older and sick populations. Moreover, most of this work has controlled for sociodemographic characteristics rather than considered interactions and variation in BMI-mortality risk across groups. Mortality risk is much higher for

both the elderly and infirm, and thus establishing a link between individuals' BMI at these ages and health outcomes is important. It is equally important, though, to determine the association between BMI and mortality at younger ages in order to better understand how bodyweight does or does not relate to premature death. This is especially pertinent given that the rapidly growing prevalence of obesity and obesity-related diseases among young and middle-aged adults (Reither et al., 2009; WHO, 2011). In fact, research suggests that recent cohorts are already seeing the effects of obesity in childhood and adolescence on adult health (Reilly & Kelly, 2011; Park et al., 2012), including mortality (Reither et al., 2011).

Additionally, much research has noted the vast disparities in health attributable to individuals' socioeconomic status (SES). Having a lower education and/or income is consistently associated with both higher BMI (i.e. higher rates of overweight and obesity) and higher mortality rates. Thus, there is likely to be significant variation in the strength of the BMI-mortality relationship by socioeconomic status. Applying a *fundamental cause* perspective to this problem suggests that more socially advantaged groups – notably those with more education – draw upon these fundamental advantages in acquiring the kinds of "flexible resources" (such as knowledge, prestige, and power: Link & Phelan, 1995; Link et al., 2008) that allow them to more adeptly deal with the consequences of unhealthy weight. In fact, research finds larger education gradients in mortality risk for causes of death that are under greater human control than for less preventable causes of death (Phelan et al., 2004; Masters et al., 2015). Consequently, an extremely high (or low) BMI among highly educated individuals can potentially be "counteracted" through better access and ability to improve their health, thus reducing associated mortality. However, in the only US-based study examining socioeconomic variation in the BMI-mortality relationship, Zheng and Yang (2012) find that college-educated adults ages 30 and over

instead had higher obesity-associated all-cause mortality than their lower-educated counterparts. Given that no subsequent work has further examined this issue, it remains unclear as to how education and BMI interact in shaping mortality risk from non-external causes (i.e. excluding deaths attributable to injuries, poisoning, accidents/falls, self-harm and assault) among working-aged adults.

With the above considerations in mind, this paper builds upon existing BMI-mortality literature by focusing on the relationship between BMI and premature mortality among working-aged Americans (ages 30-64) and by examining variation in this relationship by educational attainment. I use a recent nationally-representative mortality data set to ask: W*hat is the current relationship between BMI and mortality risk across different levels of educational attainment?* This is an especially pertinent question in light of contemporary increases in the prevalence of overweight and obesity, as well as chronic BMI-related diseases and health conditions, within this age group. Per fundamental cause theory, more advantages with respect to such critical indicators of socioeconomic status as education are often manifest in better health outcomes, especially for preventable conditions. Given that obesity is poised to overtake smoking as the leading cause of preventable death in the U.S. (Ligibel et al., 2014), it is important to better understand variation in the strength of the BMI-mortality relationship for socially advantaged and disadvantaged groups. One hypothesis emerging from this conceptual framework suggests that (H1) *overweight and obesity are associated with increased mortality risk among those with lower educational attainment compared to college or higher*, perhaps stemming from inadequate resources to contend with the risks posed by excess body weight. Alternatively, based on research concluding that obesity and other unhealthy behaviors pose a greater relative risk for more highly educated individuals, an alternate hypothesis suggests that (H2) *a higher BMI is*

*associated with reduced mortality risk among low educated adults compared to those with greater educational attainment* due to the possibility of body mass itself becoming a health "resource" when access to food and health care may be limited, and/or because of the confounding effects of poor health attributable to other diseases and adverse social conditions, especially among low-SES individuals. In narrowing the focus of this study to contemporary working-aged adults dying from non-external causes, I simultaneously test both of the above hypotheses to better specify the dynamic effects of education on the BMI-mortality relationship.

# LITERATURE REVIEW

## Obesity Paradox and Age Variation

The recurring pattern noted by much of the literature examining the BMI-mortality relationship is the existence of a "J"- or "U"-shaped curve with respect to how mortality risk varies as individuals' BMI increases. In one of the most frequently cited works addressing this subject, Flegal et al., (2005) use National Health and Nutrition Examination Survey (NHANES) data to assess excess deaths associated with various weight conditions (based on BMI cutoffs defined by the National Institutes of Health, 2000: <18.5 is underweight; 18.5-24.9 is normal weight; 25.0-29.9 is overweight; 30.0-34.9 is obese class I; and 35.0+ is obese class II or III). The highest number of these deaths is attributable to the underweight and severely obese categories, while no such penalty exists among the overweight group. Similarly, examining years of life lost due to obesity, Fontaine et al. (2003) observe a nearly 22% reduction in life expectancy for severely obese men, while concluding that overweight status has no significant impact on mortality and may in fact be advantageous for some groups such as black men and women. In fact, confirmation of this BMI-mortality relationship is found in a variety of nationally-representative data sets, including the Health and Retirement Study (Reuser et al., 2008; Reuser et al., 2009), Longitudinal Study of Aging (Allison et al., 1997; Grabowski & Ellis, 2001), Framingham Heart Study (He et al., 2009), and Million Women Study (Reeves et al., 2007). Perhaps most definitively, in a 2005 meta-analysis of 26 observational studies encompassing a variety demographic groups, and even samples from other countries, McGee

(2005) found that the relative risk of all-cause mortality among the obese individuals was 1.22, while concluding that the use of "overweight" as a medical classification may not be fully appropriate given the lack of evidence substantiating any increased risk of mortality.

The discrepancy in how these and other studies characterize the BMI-mortality risk, as either "J"- or "U"-shaped, is largely a function of whether or not underweight individuals are included in the data (accounting for the left-hand portion of the "U" curve). For instance, many studies exclusively focus on the mortality risk posed by obesity and extremely high bodyweight. In a pooled analysis of 20 prospective studies, Kitahara et al. (2014) found that the highest level of obesity (Class III; 40+ BMI) was associated with mortality risks ranging from 2.25 to 5.91 times higher than the reference normal weight group. Furthermore, Zheng et al. (2013) use Health and Retirement Survey data to conclude that adults with an "upward" weight trajectory have the greatest risk for mortality, with ~7.2% of deaths attributable to weight gain among class I and class II obese individuals. Olshansky et al. (2005) go so far as to project future decline in US life expectancy due to the increased prevalence of obesity within the population.

Conversely, studies have also drawn attention to underweight status as equally, if not more strongly, associated with increased mortality risk. Comparing the relative risks associated with high and low BMIs (with normal weight as a reference), Seidell et al. (1996) find a significantly increased risk of death among underweight men (relative risk (RR), 2.6) as compared to obese men (RR, 1.5), possibly attributable to early death among smokers. Katzmarzyk et al. (2001) observe a similar elevated mortality risk for underweight individuals (RR, 1.69) as compared to normal and class I obese (RR, 1.25) individuals. In general, many studies have concluded that low BMI is associated with poor health outcomes and increased mortality risk among older adults (Ensrud et al., 2007; Corrada et al., 2006; Price et al., 2006;

Kaplan et al., 1987). These, and other, studies are careful to note that low BMI and underweight may be working in one of three ways in affecting mortality: (1) it is likely to be an indicator of preexisting illness and "frailty" among this subset of the population; (2) individuals who are underweight, and acquire an illness or other ailment (such as an injury or condition intensive care), are then at greater risk of death; and (3) that BMI may be interacting with other important health behaviors/indicators, such as smoking status (Krueger et al., 2004), to modify the effect of underweight on mortality.

The lack of a significant mortality risk among overweight and lower-grade obese individuals noted above has been increasingly referred to as the "Obesity Paradox". In fact, much of the current work on BMI-attributable mortality has observed this relationship between moderately elevated bodyweight and lower mortality risk across a number of health conditions, including: coronary artery disease (Romero-Corral et al., 2006), hypertension (Uretsky et al., 2007), lung cancer (Yang et al., 2011), and heart failure (Curtis et al., 2005). More pertinent to this study, research suggests that individuals' weight status may have varying effects on mortality at different ages. On the one hand, the majority of studies examining the obesity paradox consistently find lower mortality among overweight and class I obese individuals ages 65 and over (Donini et al., 2012; Oreopoulos et al., 2009; Janssen et al., 2005), and medical research has demonstrated that obesity can be a "healthy" weight status with potentially health-positive aspects that can be protective of certain infectious diseases or act to sustain a healthier metabolism at older ages (Hamer & Stamatakis 2012; Kramer et al., 2013).

On the contrary, studies extending their focus to younger and middle-aged adults (25-64) have observed that the obesity paradox and effects of elevated BMI and obesity on mortality risk are not age-invariant, though there is disagreement with respect to how this relationship changes

with age. Masters and colleagues (2013a; 2013b) and others (Yu, 2012) note that the mortality risk associated with obesity and overweight is relatively stable or increasing over the life course, once cohort and period variation is taken into consideration, and that these findings should be used as evidence against the "inappropriate denial of the [obesity] epidemic's consequences for US mortality" (Masters et al., 2013a; 439). However, a competing body of work contends that BMI-associated mortality is most pronounced among young and middle-aged adults, with a gradual decrease in the strength of this relationship after age 30 (Stevens et al., 1998; Bender et al., 1999; Kuk & Ardern, 2009; Zheng & Yang, 2012). Ultimately, while there is continued debate regarding whether this obesity paradox might be attributable to statistical bias or selection bias (Preston & Stokes, 2014; Robinson et al., 2014), and whether excess mortality attributable to obesity is particularly significant from a public health perspective (Mehta & Chang, 2009; Mehta & Chang, 2011), the mass of evidence speaking to varying mortality risk associated with elevated BMI suggests that this relationship warrants further examination, especially among working-aged adults.

**Fundamental Cause Theory and the Role of Educational Attainment**

While many of the above cited studies account for various sociodemographic factors within their models, few consider variation in the BMI-mortality relationship when stratifying on these variables. This is especially surprising given the substantial socioeconomic variation in obesity prevalence, notably the much higher rates for individuals with lower SES (Wang & Beydoun, 2007). As such, the moderating effects of education on the BMI-associated mortality have largely been unexamined, which seems to be a critical oversight in light of the well-studied association between educational attainment and mortality. Within the field of social demography and population health, a myriad of recent and prominent studies have hypothesized and tested

8

potential pathways through which we can help explain education's consistent relationship with many diverse health outcomes. Though a narrow, albeit highly representative, sample of the publications in this area, the work of Ross & Wu (1995), Hummer and Hernandez (2013), Rogers et al., (2013}, Montez et al., (2011), Zajacova (2006), Masters et al., (2012), Hummer and Lariscy (2011), Hayward et al., (2014), Ross et al., (2012), Lleras-Muney (2005), and Pampel et al., (2010) has considered the sociodemographic, geographic, temporal, and cause-specific variations in education-attributable mortality within the US adult population. While too numerous to be specified in their entirety, the authors consider a wide swath of causal mechanisms linking education to better health. For instance, higher educational attainment may lead to: improved cognitive and soft skills for troubleshooting health crises; an enhanced ability to navigate health care and modify personal health behaviors if/when necessary; increased economic and social resources alleviating the burden of adverse health events; reduced stress and an improved sense of control over one's life; and access to better neighborhoods and communities with more safe spaces, less disorder, and greater opportunities for both physical activity and social engagement.

Much of the work on education and mortality grounds its theoretical foundation in fundamental cause theory, as first described by Link & Phelan (1995), and elaborated upon over ensuing articles (Link & Phelan, 1996; Phelan et al., 2004; Phelan et al., 2010). As described by the authors, educational attainment is a *fundamental* cause due to its role in generating "flexible resources" that can be deployed by individuals in seeking to maintain or improve their health. In contemporary society, education leads to a "a wide range of serviceable resources, including money, knowledge, prestige, power and beneficial social conditions, that can be used to one's health advantage (Phelan et al., 2004: 267)." With respect to studying mortality associated with

individuals' weight status, adults with lower education may have fewer opportunities for effectively managing obesity-related disease, and thus an increased likelihood of seeing these conditions result in preventable death. For example, Link and Phelan (2010) stipulate that those with higher education are the first to benefit from new medical advances and knowledge, such as various technologies, techniques, and public health messaging that are so critical in preventing and managing chronic diseases. Though the pathways of action through which educational attainment works are diverse, Cutler & Lleras-Muney (2006) provide a comprehensive overview of the many potential mechanisms linking education to health and mortality through the lens of fundamental causality, including group differences in time-discounting and the effects of an individual's rank within society on their overall health and well-being.

Furthermore, Link and Phelan explicitly call for and welcome studies testing the appropriateness of fundamental cause theory in helping to explain existing health disparities and trends (2004). Of particular relevance to studying weight-related health and mortality, Chang & Lauderdale (2009) examine the use of and access to statins in the mid-1990s, finding that the gradual downward diffusion of medical technologies throughout the population helps to explain the reversal of the SES gradient for high cholesterol following the approval of statins for prescription use, with higher-income individuals being the first to see health improvements. Similarly, Miech et al. (2011) question who benefits most from new medical interventions, again concluding that the most advantaged individuals in society (i.e. those with the most education) experience the most immediate and substantial health benefits associated with medical innovations, as is the case for many other medical technologies (Glied & Lleras-Muney, 2008; Goldman & Lakdawalla, 2005). Given that education is likely to be a critical component of an individual's sociodemographic profile, the ability to exert control over the modification of one's

health behavior – such as upon receiving weight management-related health advice or information from a medical professional – is highly dependent upon their level of educational attainment (Mirowsky & Ross, 1998; Mirowsky & Ross, 2003).

Of particular relevance to this study, the work of Masters et al. (2015) uncovers larger education gradients in mortality for health conditions considered to be more "preventable" (or manageable through individual control), especially in the case of heart disease, a condition closely tied to obesity. Examining mortality trends from 1986-2006, the authors find overall reductions in heart disease mortality risk for both low and high educated individuals; yet, the evidence suggests a much steeper decline for those with a college education or greater as compared to those with less than a high school degree. This work speaks to the importance of educational disparities as most pronounced in preventable causes of death (Link & Phelan, 2005), and lends credence to the hypothesis that more highly educated individuals are less likely to see as strong of a relationship between increased BMI and elevated mortality as their lower educated counterparts.

Despite the vast body of work on education and mortality, a careful analysis of the literature assessing potential moderating effects of education uncovered only two studies using education as a key stratifying variable to better understand the relationship between BMI and mortality. Schnohr et al., (2004) use pooled population data from Copenhagen in order to examine how SES (proxied by education) might influence mortality of four health-related behaviors, one of which is BMI. Stratifying education into three categories (<8 years; 8-11 years; and >11 years), the authors observe fairly consistent "J"- or "U"-shaped curves at all three levels, for both men and women. They further find that low-educated men have the highest mortality risk at the lowest BMI level (~1.5 RR), and the second highest risk at the highest BMI level (also

11

~1.5 RR). Highly-educated men had the lowest risk at low BMI (RR <1.0), but the highest risk at high BMI (RR ~1.75). Among women, the low-educated individuals had the highest mortality at both the lowest and highest BMI groups. In a more exhaustive and US-based analysis of population heterogeneity in mortality associated with body weight, Zheng and Yang (2012) analyze NHANES data (1988-2006) to observe a similar pattern of elevated obesity-associated mortality risk for individuals with bachelor degree or higher (HR=2.49, p<0.01) as compared to those with less education (HR=1.34, p<0.01). Interestingly, these differences were only found in the most severe cases of obesity, classes II/III, while the two educational groups were comparable with respect to the mortality risk associated with overweight and class I obesity. In concluding, the authors suggest three important mechanisms underlying this variation. First, they hypothesized that the sickest individuals may be dying prior to the onset of the study and exaggerating the protective effects of certain weight statuses. Second, they suggest that excess bodyweight may become a critical health resource (e.g. a nutritional reserve) in the absence of traditional support mechanisms. Finally, they consider possible confounding attributable to competing causes of death and disease, possibly unrelated to obesity, for more disadvantaged groups.

While not framed as such, both Schnohr et al. and Zheng and Yang provide results consistent with the "Blaxter hypothesis". Based on empirical results suggesting that healthy lifestyles have less influence on lowering mortality among low-SES groups than high-SES groups, this theoretical model maintains that "unhealthy behaviour does not reinforce disadvantage to the same extent as healthy behaviour increases advantage" (Blaxter, 1990: 233). Due to the multitude of negative social circumstances likely to have a more persistent and cumulative impact on the health of low-SES individuals (such as unsafe housing, work, and

neighborhood environment (Kreuger & Chang, 2008)), it may be the case that unhealthy behaviors are especially detrimental to the health of higher SES individuals who might otherwise enjoy good health as a function of their social advantages. In fact, in a test of the Blaxter hypothesis focusing on racial/ethnic differences in adult mortality and the role of health behaviors, Kreuger et al. (2011) find that more socioeconomically disadvantaged groups experienced a weaker relationship between unhealthy behavior and mortality. This was especially pronounced in the case of reduced impact of smoking on mortality for Hispanics as compared to whites, and low or high levels of alcohol consumption, high levels of physical inactivity, and short or long sleep hours on mortality for blacks compared to whites.

Nonetheless, both of the above studies shed much needed light on the role of educational attainment in conditioning the effects of BMI and obesity on mortality, and the broader issue of the relationship between social advantages, health behaviors, and mortality. However, they are limited in a few critical areas which the present study builds upon. Likely owing to limitations in sample size and/or space constraints, neither study is able to provide a more comprehensive specification of educational attainment, to limit their focus to working-aged adults and death by non-external causes more closely related to obesity, or to formally test the differences in mortality risk by gender and across education groups. Here, I use the large and expansive data from the National Health Interview Survey to address some of these outstanding questions, and to build upon the analytic framework established by these insightful studies.

In sum, the substantial literature on the relationship between BMI and mortality – examined in such diverse fields as demography, public health, epidemiology, gerontology, and various medical subfields – continues to observe the elevated risk of death attributable to very high or very low BMI and the paradox of overweight and moderately obese individuals having

the lowest mortality, suggesting that some degree of excess weight may in fact be "protective". However, other aspects of the BMI-mortality relationship require further analysis, namely with respect to premature death and educational variation. Though studies suggest that underweight and severe obesity pose a comparable death risk for middle-aged adults, there appears to be no definitive answer as to the mortality risk associated with overweight and moderate obesity in this age group. Furthermore, this paper examines unaccounted-for variations in the BMI-mortality relationship among working-aged adults by addressing the limitations of previous work on the moderating effects of educational attainment. In doing so, I consider the role of fundamental causality and social advantages in identifying population subgroups for whom high or low BMI might pose a greater risk of premature adult mortality.

## DATA

The primary data for these analyses come from the National Health Interview Survey (NHIS), the principal source of information on the health of the civilian noninstitutionalized population of the United States (CDC 2015). With tens of thousands of participants in each wave, NHIS is one of the largest nationally-administered surveys, collecting data on a broad range of health topics continuously since 1957. While the NHIS is conducted annually, the Integrated Health Interview Series (IHIS) allows for the creation of pooled samples, combining respondent information across multiple waves/years of data collection. Furthermore, the National Center for Health Statistics (NCHS) has linked death records from the National Death Index (NDI) to NHIS records from 1985 to 2009 (referred to as the NHIS-Linked Mortality File, or NHIS-LMF), which includes mortality data up through December 31, 2011. For the purpose of this analysis I use the integrated series spanning 1997-2009 (including mortality follow-up through 2011), which is not only the most recent set of public-use data, but also uses a consistent sampling fame following the survey questionnaire redesign in 1997. This is an especially critical issue given the dynamic nature of educational attainment in both measurement and meaningfulness over time (Heath, 2009; Brock, 2010).

From within the larger sample of NHIS respondents, my data are initially limited to those individuals completing the "Sample Adult Questionnaire", given to a single adult in each sample household. These "sample adults" provide additional, detailed information on both sociodemographic and health items critical to my analyses, in addition to their having available

15

mortality data. Specifically, of the initial sample I select out those individuals for whom there are complete records for: Mortality status; BMI (calculated from height and weight); Age; Gender; Race; Foreign born status; Education; Alcohol use; and Smoking status. Of the 241,534 sample adults eligible for inclusion based on age and interview year, 211,452 (89%) have complete information on the variables noted above, with mortality data and BMI accounting for the majority of missingness, at 4% and 3% respectively each. Finally, weights have been applied (available within NHIS) to account for the sample adult characteristics as well as those individuals ineligible for mortality follow-up.

## MEASURES

The focal outcome variable of these analyses is death from non-external causes that are attributable to a number of health conditions previously found to be associated with body weight and BMI-related mortality. Based on an NHIS variable that groups individuals' specific cause of death into one of the ten leading causes, the BMI-related causes of death I use are diseases of heart (Hubert et al.,, 1983), malignant neoplasms (Calle & Thun, 2004), chronic lower respiratory diseases (Poulain et al.,, 2006), cerebrovascular diseases (Isozumi, 2004), Alzheimer's disease (Naderali et al.,, 2009), diabetes mellitus (Mokdad et al., 2003), influenza and pneumonia (Jain & Chaves, 2011), and nephritis/nephrotic syndrome (Bonnet et al., 2001). Though they represent a significant number of deaths among young and middle-aged adults, accidental and other external causes of death are excluded because the potential causal association of BMI with such deaths is unclear (Flegal et al., 2007). The integrated series data provide information on an individual's interview year, quarter, and week (of the quarter), as well as the year and quarter of death. Using these data, I constructed a measure of survival time, based on how many person-years a respondent contributed to the study window – in this case, defined as 1997 through the end of 2011, given the available linked-mortality data. As such, the analyses focus on individuals ages 30-64 at time of survey who are then statistically matched to mortality records and followed for survival status until: (1) their death, (2) their 65th birthday, or (3) the end of the year 2011.

The main predictor variable of interest is respondents' BMI, based on a ratio of individuals' self-reported weight to height ($mass_{kg}/height^2_m$). Misreporting of weight and height is a potential issue with the NHIS measure of BMI (Stommel & Schoenborn, 2009); however, previous work comparing BMI in NHIS to measured BMI in NHANES found a very high correlation among respondents participating in both surveys (Durazo-Arvizu et al., 1997). Rather than treat BMI as a continuous variable, I recoded BMI into categories of weight status: <18.5 is underweight; 18.5-24.9 is normal weight; 25.0-29.9 is overweight; 30.0-34.9 is obese class I; and 35.0+ is obese class II or III (NIH, 2000). This "grouping" of BMI into weight status follows the organization of most studies examining BMI-associated mortality and health, as is the grouping of obese class II and class III into one category (Masters et al., 2013b; Zheng & Yang, 2012).

Other variables include gender, race, foreign-born status, educational attainment, alcohol use, and smoking status, in an effort to choose controls that would remain relatively static over the follow-up period (a critical assumption for proportional hazards modeling), as well as helping to account for additional health behaviors that may be related to mortality. As mentioned earlier, age was limited to 30-64 which narrows the sample to a "working age" adult population with a consistent mortality hazard function (Flegal et al., 2005), and serves as an acceptable upper limit to the definition of "premature" mortality occurring prior to age 65 (Dranger & Remington, 1998). Gender is coded with a dummy variable for "Male" respondents. Race/ethnicity is coded as "white" (non-Hispanic), "black" (non-Hispanic), "Hispanic" (of any ethnicity), "Asian/Pacific Islander" (non-Hispanic), "Native American/American Indian", and "Other". Foreign-born status is a dummy variable derived from a reverse coding of the "US-born" variable in the NHIS. Education is based on an existing NHIS-provided variable of education categories, though new categories were created representing educational attainment of "Less than High School", "High

School", "Some College/Non Four-year College", and "Four-year College or Higher". Finally, alcohol use and smoking status are based on an existing NHIS variables, with categories for "Lifetime abstainer", "Former drinker", and "Current drinker" for alcohol consumption, and "Never Smoked", "Former Smoker", and "Current Smoker" (based on numbers of cigarettes smoked over a certain time period) for smoking status. While Zheng and Yang (2012) also use self-rated health as a critical covariate for individuals' health status, initial analyses using this measure significantly suppressed the effects of obesity on mortality (though the effect was reversed in the Zheng and Yang paper). The interaction of self-rated health and obesity appears to be a complex issue warranting further investigation (Altman et al., 2016; Imait et al., 2008; Okosun et al., 2011), but in an effort to more clearly discern the standalone effects of BMI on mortality, I exclude it from these analyses.

**METHODS**

Due to the structure of the NHIS integrated data, with respondents being interviewed in different years and contributing varying amounts of person-years to the designated study window (1997-2011), I use Cox proportional hazard models to construct premature mortality risk ratios based on BMI categories. Previous studies examining BMI-associated mortality consistently report risk in the form of such hazard ratios, frequently using "normal" weight (BMI = 18.5-24.9) as the reference group for BMI set at a default hazard ratio of 1.00 (Masters et al., 2013a; Masters et al., 2013b; Adams et al., 2006). Though some researchers exclude the "underweight" category from their analyses, due to small cell counts and justifiable concerns about individuals in this BMI category being sick or at a disproportionately higher risk of mortality, I choose to include this group in the interest of seeing potential subgroup variation in underweight hazard ratios.

Stata version 13 is used to run these analyses, applying the STSET command and defining the failure event as a working-aged death, study time as age (calculated as a function of their age upon entry and subsequent person-years contributed the study), and entry into the study defined by age at survey time. Models control for gender, race (as categorical), foreign-born status, education (as categorical), alcohol use (as categorical), and smoking status (as categorical). The focus of this study is to examine educational variation in BMI-associated mortality; thus, I run separate models by each level of educational attainment in order to examine whether the BMI-mortality relationship is consistent or not both within and across educational

groups. I also include gender-stratified models to examine any evidence of gender differences in the moderating effects of education. Finally, I compare the hazard ratio coefficients for BMI groups across these sociodemographic strata through the use of interaction models, to determine whether there are also statistically significant differences for BMI-associated mortality risk across different levels of education, and by gender. In these models I allow all covariates to vary by education (and gender), focusing on differences in the relative hazard ratios for the BMIxEducation interaction.

**RESULTS**

I begin by comparing the baseline characteristics of the individuals in the sample surviving through the full study to the subset of respondents experiencing a death between the ages of 30 and 64. As descriptive Table 1 shows, while the surviving and decedent subsets of individuals are relatively similar with respect to the percentage normal weight, overweight, or class I obese, respondents experiencing a premature death were more likely to be underweight (2.9% vs. 1.3%) or severely obese (14.3% vs. 10.1%). The mean age of the surviving sample is 45.3 consistent with previous work using the entire adult NHIS sample (Masters et al., 2013b), as this sample excludes both older and younger participants in roughly equal amounts. The decedents were approximately 5 years older on average, at 50.2, and had a higher proportion of men (54.8% vs. 45.6%). With respect to race and foreign born status: both subsets have approximately equal proportions of white respondents (~63%); there is a higher proportion of black respondents among those dying (21.5% vs. 14.4%) contrasted with a lower proportion of Hispanics (12.6% vs. 16.9%); and a lower proportion of foreign born individuals among those dying prematurely (11.6% vs. 17.9%).

Fairly large differences are observed in the educational composition of the two sub-samples. While comparable in the middle education categories – High School and Some College – the surviving sample has about three-fifths as many respondents with less than a high school education (16.2% vs. 25.7%) and almost double the percentage of respondents with a college degree or higher (27.4% vs. 15.1%). Finally, there are considerable disparities in smoking and

alcohol use among the two groups. Though there are no differences with respect to being a lifetime alcohol abstainer, a higher proportion of those who died were classified as former drinkers (25.8% vs. 14.9%), while 65.4% of those surviving are current drinkers as compared to 55.0% of those dying. Over half of the surviving sample has never smoked, at 53.8%, as compared to only 32.0% of the dying subset; and just under a quarter, or 24.8% of the overall sample are current smokers, as compared to 45.0% of those dying prior to age 65.

The first model, Table 2, begins by presenting BMI-associated premature mortality for this working-aged sample for the years 1997-2011, without any stratification by education. This model includes the hazard ratios for all of the covariates included in subsequent models, and serves as a preliminary check on the validity of the Cox models for this analysis. As seen in Table 2, when excluding the underweight category the overall "U-" or "J-shaped" pattern of BMI-associated mortality is relatively consistent with that observed in previous research. Individuals in the underweight category are at the highest risk of mortality relative to normal weight, at 2.61 (p<0.001), while severely obese individuals (class II/III), are at a higher risk of mortality relative to normal weight with a hazard ratio of 1.40 (p<0.001). These risks are comparable to coefficients obtained in previous studies (Masters et al., 2013a; Masters et al., 2013b; Zheng & Yang, 2012; Fontaine et al., 2003; Adams et al., 2006). Conversely, those in the overweight category have the lowest risk of mortality at 0.86 (p<0.01), which is similar to estimates obtained for older adults and studies imposing no restriction on age (Mokdad et al., 2003), but less pronounced when compared to other studies reporting on working-aged adults (Zheng & Yang, 2012). Class I obese individuals are not at a statistically significant different risk of mortality as compared to normal weight individuals, with a hazard ratio of 0.97. In sum, mortality risk for contemporary adults in this age group is only elevated at the most "unhealthy"

BMI levels, representing both extremes of body weight (i.e. underweight and class II/III obese). Overweight continues to be associated with reduced mortality risk, while the risk associated with class I obesity is not statistically different from that of normal weight.

Turning to the covariates in this model, gender, race/ethnicity, foreign-born status, education, alcohol use, and smoking are all significantly associated with working-aged mortality. All else equal, there is approximately a 33% (p<0.001) reduction in risk for females, and a 54% (p<0.001) increase for blacks as compared to whites. Foreign-born individuals exhibit lower mortality than US-born individuals (HR=0.80, p<0.01), and mortality risk decreases substantially as education increases, with individuals having a college education or higher having approximately half of the mortality risk relative to those with less than a high school education. Finally, being a current drinker greatly reduces the risk ratio compared with lifetime abstainers (HR=0.70, p<0.001). Former and current smokers have higher mortality risk than never smokers, with hazard ratios of 1.35 and 2.50 respectively (p<0.001 for both).

**Within-Group Educational Differences in BMI-Associated Mortality**

Addressing the primary research question, the next set of models considers how the shape of the BMI-mortality relationship varies when stratified by education, for both the overall sample and by gender. The different levels of educational attainment are treated separately and all control variables are allowed to vary. Though there are interesting and significant differences among these covariates when examining the separate models (available upon request), given the focus of this paper only the hazard ratios for the BMI categories are presented within Tables 3a and 3b.

Table 3a provides evidence of substantial differences in the mortality risk associated with BMI categories from one level of educational attainment to the next. While all education groups

have a significantly higher relative mortality risk for the underweight group (HR=2.47, p<0.001 for <HS; HR=1.86, p<0.01 for HS; HR=3.43, p<0.001 for Some College; HR=3.40, p<0.001 for College+), there are notable within- education group differences for the mortality risk associated with BMIs of 25.0 or greater. For instance, class II/III obesity is also associated with substantially elevated mortality risk relative to normal weight at all levels of education with the exception of those with less than a high school education. While individuals with a college education or greater have almost double the mortality risk associated with class II/III obesity (HR=1.97, p<0.001), the same level of obesity yields a hazard rate of 1.15 among the least educated adults, which is not statistically different from the baseline of normal weight. Similarly, class I obesity poses an elevated mortality risk only within the highest educated group (HR=1.37, p<0.05) and is instead associated with *decreased* mortality risk among individuals with less than a high school education (HR=0.80, p<0.05). Further, class I obesity is not associated with increased risk among those with high school or some college education. Finally, overweight status, which has been found to be associated with the lowest mortality risk for this age group as a whole, has no such "protective" effect among those with a college education or greater (HR=1.146) while being associated with an approximately 20% reduction in relative risk for those with some college education or less.

Extending the analyses of educational heterogeneity, Table 3b (below) considers the added dimension of gender as possible source of further variation in the effects of education on BMI-associated mortality. Given past research indicating gender differences in both BMI-associated mortality (Zheng & Yang, 2012; Fontaine et al., 2003) and education-associated mortality (Montez et al., 2011), there is reason to believe that the effects of excess body weight at a given level of education may be different for men and women.

Overall the pattern of BMI-associated mortality by educational attainment is relatively similar for both men and women, especially when comparing the two ends of the BMI spectrum. For men, underweight is associated with a substantially elevated mortality risk at all levels of education except college or more, though the insignificant hazard ratio of 2.07 at this education level suggests that statistical power is an issue for this combination of BMI, education, and gender. The same pattern holds for females, who experience elevated mortality risk associated with underweight across all education groups, with the highest risk among the college educated (HR=4.15, p<0.001). Class II/III obesity also mirrors the educational variation observed in the overall sample. The least educated men and women experience no significant risk associated with severe obesity, while the highest educated see significantly elevated mortality risk ranging from HR=1.64 (p<0.05) for females to HR=2.21 (p<0.001) for males.

There is evidence of compelling gender differences in the pattern of BMI-associated mortality by educational attainment when examining the overweight and class I obese weight categories. Among men, overweight is associated with a similar 20-30% reduction in mortality risk across all education levels except college or more, as was observed in the overall sample (Table 3a). However, for women overweight is associated with reduced mortality risk only in the less than high school category (HR=0.77, p<0.10). Further, class I obesity is associated with reduced mortality risk for men with less than a high school education and not women (HR=0.77, p<0.10). And while class I obesity is not associated with increased mortality risk for either high school or some college education for either gender, among college educated women class I obesity increases relative mortality risk by almost 88% (p<0.01) as compared to a lack of elevated risk for men. It is worth noting that further analyses testing whether gender differences are significant across BMI categories by educational attainment (not shown, but available upon

request) indicate that hazard rates are largely not statistically different from one another. However, the aforementioned difference in mortality risk associated with class I obesity does emerge as statistically different at the p<0.10 level, with women experiencing 1.6 times the relative mortality risk of men.

**Across-Group Educational Differences in BMI-Associated Mortality**

As evidenced by the tables above, there are clear differences in the within-group patterns of BMI-associated mortality when stratifying by educational attainment and gender, especially in the case of obesity-associated mortality risk for college-educated individuals, and the reduced mortality risk associated with overweight among lower-educated groups. While these findings are compelling in and of themselves, I advance the current literature on educational variation in BMI-mortality risk by conducting further analyses that test whether the differences *across* education groups might be significant as well. This line of inquiry examines whether or not the relative "unhealthiness", or risk factor, specific to a given weight status is significantly different across levels of educational attainment. Thus, I run separate models, for men and women combined as well as separate, allowing all covariates to vary through the use of interaction terms by education. I focus primarily on the interaction terms corresponding to the different BMI groups, allowing me to observe whether hazard ratios across groups are significantly different from one another. Only these interaction terms, representing the ratio of one level of education interacted by weight status relative to normal weight compared to another level of educational attainment (e.g. the mortality risk of being obese relative to normal weight for college-educated individuals as compared to the same risk for those with less than a high school education), are presented in Tables 4a and 4b below.

Consistent with the previously observed within-group variations in the mortality risk associated with different BMI categories, Table 4a shows statistically significant differences when comparing these hazard ratios across educational attainment groups for the overall sample. In these comparisons, a relative risk of 1.00 would indicate no difference in mortality risk associated with a given "unhealthy" weight status relative to normal weight; e.g. that the relative risk of being overweight as compared to normal weight is equal between two levels of educational attainment.

Mortality risk associated with underweight as compared to normal weight shows the least across-group variation, though there is some evidence of significant differences when comparing relative underweight mortality risk for those with some college education to high school (RR=1.84, p<0.05) as well as those with college or more education to high school (RR=1.81, p<0.10). However, these estimates should be treated with caution given there are very few deaths among underweight adults when further stratifying by educational attainment. However, examining the interactions of weight status and education at BMI values of 25.0 or greater also reveals significant differences in relative mortality risk, especially when comparing those with a college education to lower-educated groups. This highest level of educational attainment is consistently associated with higher relative mortality risk in the overweight, obese class I, and obese class II/III weight groups. Specifically, the risk of overweight-associated mortality among adults with a college degree or higher is almost 1.5 times (p<0.01) that of individuals with an education level of less than high school, 1.4 times (p<0.01) that of individuals with a high school education, and also 1.4 times that of individuals with some college education (p<.05). Similarly, when examining mortality associated with mild obesity, the relative mortality risk for those with a college education is also elevated as compared to those with lower educational attainment (RR

compared to: <HS = 1.73, p<0.01; HS = 1.48, p<0.05; Some College = 1.48, p<0.05) . This intriguing finding is also true with respect to severe obesity, as the analysis indicates that class II/III obesity poses 1.7 (p<.051) times the relative mortality risk for college or higher educated adults as compared to those with less than a high school education, and a similarly elevated relative risk when compared to individuals with a high school education (RR=1.37, p<0.10) and some college education (RR=1.56, p<0.05).

In light of previous results showing gender differences in the within-group pattern of BMI-associated mortality by educational attainment, I apply the same methodology as before to test whether across-education group differences are statistically significant when looking at men and women separately. As seen in Table 4b, underweight mortality risk is approximately 2.5 times higher for those with some college education as compared to a high school or less education for males, whereas the only significant difference in underweight mortality risk for females occurs when comparing those with a college education or more to high school (RR=2.49, p<0.05). Educational differences in mortality risk associated with overweight are also more pronounced for men than women. Most notably, college educated men experience approximately 1.5 times the overweight mortality risk of those with less education, whereas for women this difference is only statistically significant when comparing overweight risk for those with a college education to adults with a high school education or less. Finally, the most apparent gender differences across education groups are evident in the mortality risk associated with obesity. For both men and women, college-educated individuals have a significantly higher relative mortality risk than their less educated counterparts. However, these differences are only true of class II/III obese for men (RR for college or more compared to: <HS=2.01, p<0.01; HS=1.61, p<0.10; some college=1.88, p<0.05), with no significant differences across education

groups in class I obesity-associated mortality. The opposite relationship holds for women, wherein being college-educated is associated with approximately twice ($p<0.01$) the class I obesity-associated mortality risk as compared to those with less education.

## SENSITIVITY & ANCILLARY ANALYSES

Prior to moving on to a more full-fledged discussion of the results presented above, I seek to preempt potential critiques of these analysis related to the cross-sectional nature of the data as it relates to subsequent mortality, and the reliance on self-reported measures of height and weight in NHIS. Specifically, the choice of mortality follow-up time is a persistent issue in any analysis of cross-sectional data, with this study proving no exception. Namely, an assumption must be made about the time-invariance of BMI as measured at baseline. While an individual's BMI is subject to change over time, which itself has a distinct effect on mortality risk (Zheng et al., 2013; Zajacova & Ailshire, 2014; Myrskyla & Chang, 2009), there are no data on final BMI at a respondent's time of death. Similarly, the potential "mortality" or "healthy participant" selection effects of sick, dying, or otherwise unhealthy individuals into certain BMI groups at baseline cannot be fully ascertained (Flanders & Augestad, 2008), especially in trying to distinguish between the effect of unhealthy body weight on mortality as an acute or chronic and cumulative process[1].

Furthermore, much research has found evidence of discrepancies between self-reported and measured BMI, typically due to the underestimation of weight by respondents, and subsequent misclassification with respect to weight status. Though these results were found to be more true of older adults, who may also overestimate their height, these studies have important

---

[1] Though there is evidence from Zheng and Dirlam (2016) suggesting that neither of these selection effects fully explains away the BMI-mortality relationship. Specifically, using NHANES data from 1988-2011, they find that "even if the healthy participant effects were stronger among obese adults, they are not strong enough to produce a weakening association between obesity and morbidity at higher ages at the time of the survey."

implications for the estimates of prevalence rates of obesity in the population, as well as the

comorbidities and mortality associated with the disease, leading researchers to speculate that we

might actually be *under*estimating the burden of obesity on individual and population health

(Stommel & Schoenborn, 2009; Merrill & Richardson, 2009; Craig & Adams, 2009). Further,

there is continued debate over the external validity of BMI as an indicator of "unhealthy" weight,

given its inability to distinguish between lean and adipose tissues, and the imprecision in

describing the distribution of fat on an individual's body (Prentice & Jebb, 2001; Garn et al.,

1986; Nyholm et al., 2007; Frankenfield et al., 2001). The latter has especially important health

implications, given the scientific and medical consensus on "central adiposity" – or the

"accumulation of fat in the lower torso around the abdominal area" (Bacon, 2013) – as strong

indicator of metabolic syndrome and cardiovascular risk (Alberti et al., 2009; Després &

Lemieux, 2006; Alberti et al., 2006). Waist circumference, while a more infrequent measure in

surveys and other data collection efforts, provides a more direct measure of central adiposity and

has been found to be a comparable – if not superior – independent predictor of obesity-related

morbidity and mortality (Koster et al., 2008; Bigaard et al., 2003; Katzmarzyk, et al., 2006;

Sahakyan et al., 2015).

Given the above, I conduct a series of additional analyses seeking to evaluate these

limitations and better understand how the current set of findings compare to alternate measures

of BMI and central adiposity, as well as to different specifications of mortality follow-up. Table

5a (below) presents two alternate specifications of mortality follow-up time for the NHIS

sample. The first limits mortality to a 5-year window, in an effort to mitigate the potential of

respondents' BMIs drastically increasing or decreasing over time. In the second specification I

exclude the first two-years of mortality follow-up, as deaths occurring in this window of time

might be the result of pre-existing and/or severe medical conditions. This also helps to differentiate the role of BMI as an indicator or precursor for chronic, underlying conditions contributing to mortality (such as type-II diabetes, heart disease, or cancer), distinct from the possible immediate health threats associated with a very high body weight (such as sudden cardiac arrest or ischemic stroke).

Based on the results in Table 5a, my initial findings prove robust to both the five-year mortality follow-up, and the mortality follow-up excluding deaths occurring in the first two-years after the initial interview. In fact, there is a notable increase in the relative mortality risk attributable to severe obesity among the most highly educated individuals, with a hazard ratio of 2.44 (p<0.001) as compared to 1.97 (p<0.001) given unrestricted mortality follow-up. While there is a less noticeable education "gradient" in the increase of relative mortality risk associated with higher BMIs, the relative differences for mortality risk attributable to any grade of obesity are clearly much higher for individuals with a college education or greater as compared to those with less education. A similar pattern emerges when excluding the first two years of mortality follow-up results. There is less evidence of a clear education gradient, but the increased mortality risk associated with moderate and severe obesity continues to be more pronounced among the highly-educated respondents. Interestingly, the exclusion of these "early" deaths from analysis results in a reduced relative mortality risk for higher BMIs among this group (HR=1.67, p<0.01), while having the opposite effect for those with less education. The increased relative mortality risk associated with severe obesity for those with a high school educated is now 1.64 (p<0.001) and 1.39 (p<0.01) for those with some college education; compared to 1.45 (p<0.001) and 1.27 (p<0.05) in the unrestricted sample. Thus it would appear that the previously observed

educational differences in mortality risk associated with higher BMIs are consistent with respect to both short- and long-term mortality follow-up.

Next, in Table 5b, I create a series of comparable subsamples of NHANES respondents in order to assess any differences in the results obtained when using measured height and weight to calculate BMI (as NHANES participants undergo an in-person physical examination) to those from the self-reported measures in NHIS. I also re-run the analyses for these same NHANES subsamples using waist circumference rather than BMI as a predictor of mortality risk, examining differences by level of educational attainment.

Using the same set of covariates (with the exception of alcohol use), a comparable period of years for both survey and mortality follow-up, and Cox proportional hazard analysis, results from Table 5b largely support the education gradient in the BMI-mortality relationship observed for self-reported BMI in NHIS. This gradient is more clearly evident in the all-cause mortality and all-age NHANES subsample, as the relative mortality risk of severe obesity for respondents with a college education or greater (HR=1.831, p<0.05) is nearly double that of those with a less than high school education (HR=0.966). These relative mortality risks, and the ratio between them, also compare favorably to estimates using NHIS. Limiting the NHANES analyses to non-external mortality at all ages, and all-cause premature mortality (ages 30-64), results in a fairly large reduction in observed deaths and statistical power. While the observed mortality risks are not flagged as statistically significant (especially given the very large confidence intervals), we can still observe an increasing relative risk of mortality associated with severe obesity for those with higher educational attainment. When restricting the NHANES sample to only non-external premature mortality, as in the NHIS analyses, there is more than tenfold reduction in observed deaths, resulting in highly unstable estimates. Given the impending release of linked mortality

data through 2015 (NCHS, 2016), the potential increase in deaths in this age-range will hopefully allow for future re-estimation and better comparison to the NHIS results presented in this paper.

Also presented in Table 5b are analyses for these same NHANES samples with waist circumference as an indicator of unhealthy body composition in the place of measured BMI. According to the National Heart, Lung and Blood Institute (NHLBI) guidelines, "waist circumference provides an independent prediction of risk over and above that of BMI… [and] is particularly useful in patients who are categorized as normal or overweight on the BMI scale. At BMIs greater than or equal to 35, waist circumference has little added predictive power of disease risk beyond that of BMI." Further, the NHLBI defines a "high risk" waist circumference as greater than 102 cm (or 40 in) for men, and greater than 88 cm (or 35 in) for women. Using a gender-specific dummy variable for high-risk waist circumference, Table 5a does not provide evidence of an educational gradient in mortality risk comparable to that seen with BMI as an indicator of unhealthy weight. In fact, across most all of NHANES subsamples and levels of educational attainment, high-risk waist circumference does not emerge as statistically significant with respect to elevated mortality risk. However, caution should be taken in given this lack of significance too much attention, seeing as there are even fewer observed deaths among individuals with data on waist circumference, resulting in a further loss of statistical power, and high-risk waist circumference is significantly associated with increased mortality risk for the NHANES sample as a whole (HR=1.11, p<0.10; results not shown).

Though these data limitations prevent a more in-depth analysis and comparison of mortality risk associated with unhealthy BMI and high-risk waist circumference, as well as the educational gradient (or lack thereof) in this relationship, I nonetheless consider some of the implications of the observed discrepancy in the results. First and foremost, the hypotheses I

present regarding the role of education and social advantage in modifying the effects of unhealthy body weight or composition on mortality can and should be expanded to include waist circumference and other measures of health. While the present ancillary analysis prevents a clear answer with respect to waist circumference, further work can explore these questions in better understanding the degree to which an individuals' SES can serve to either mitigate or amplify the effect of existing health risks on later mortality. Further, it may very well be the case – as noted in much prior literature – that BMI is a potentially inconsistent and unreliable proxy for a genuinely "unhealthy" body, especially as it pertains to increased or decreased mortality risk. If waist circumference is a superior and more valid indicator of health (e.g. central adiposity), then it may stand to reason that the increased risk of mortality attributable to a high risk waist circumference does not exhibit any significant variation by educational attainment. Thus, compared to BMI, it may be a more "equal opportunity" risk factor, such that having more education (and, by extension, a higher SES) does not mitigate its strong effect on increased risk of death. BMI, on the other hand, having a possibly "weaker" association with mortality, might require a lower group-specific overall risk threshold in order to emerge as significant indicator of increased relative mortality risk. In other words, the already increased risk of mortality for lower-educated adults is not significantly affected by a higher BMI (especially if BMI is a poor indicator of health risk); while the relatively low overall mortality risk for higher-educated adults might be more sensitive to the additional risk imposed by an "unhealthy" BMI. This discussion of the "additive" effect of BMI-associated mortality risk is further elaborated in the ensuing section, especially as it pertains to the main findings of this paper.

**DISCUSSION & CONCLUSION**

Given the lack of consensus on BMI-associated mortality, as well as the relative paucity of research examining this relation when limited to premature mortality (<65 yrs.) and stratifying by socioeconomic indicators, this study compared the pattern of BMI-associated mortality risk across different levels of educational attainment. Based on the most recent sample of NHIS-LMF data, the results of Cox hazard models revealed variation in the relative mortality risk associated with "unhealthy" weight categories when comparing across education groups, as well as gender differences in this variation. While the overall, unstratified model exhibited a BMI-mortality relationship similar to the commonly observed "U" or "J" in previous studies among older adults, models stratified by educational attainment and gender demonstrated that among working-aged adults underweight BMIs are consistently associated with a high mortality risk ratio (~2.00-3.00), overweight is associated with anywhere from a 0-30% reduced mortality risk, class I obesity is typically not associated with an increased or decreased relative risk, and severe obesity is associated with a large range of increased mortality risks, from 30 to over 100%.

In contrast to prior work, the obesity paradox did not extend to the class I obesity weight group, as there was no significant difference in mortality risk as compared to normal weight with the exception of a few subgroups. Research has suggested that the burden of some grades of obesity on excess mortality may be declining in recent years (Mehta & Chang, 2011), and the aforementioned finding lends credence to this hypothesis, at least among working-aged adults. Furthermore, the reduced mortality risk associated with overweight was lower than previous

estimates studying individuals in or near this age group (Zheng & Yang, 2012; Mehta & Chang, 2009), though drawing exact comparisons is difficult given key differences in the choice of study samples and selection of covariates included in analytic models.

Turning to the primary research aim concerning BMI-associated mortality and its relationship to the social advantages that certain educational groups may have, results revealed that more highly educated individuals (those with a college education or greater) were at a substantial disadvantage for relative mortality risk associated with higher weight when compared to those with a lower level of educational attainment. In addition to the lack of any decreased risk associated with overweight, the increased obesity-associated mortality risk for highly-educated individuals tends to favor the hypothesis positing that increased body weight is less harmful among lower-educated adults (H2) due to its possible role as a vital health resource and/or the confounding effects of other adverse health conditions among low-educated individuals. That is to say, highly-educated individuals might represent an otherwise "healthy" group of adults for whom the mortality risk of elevated BMI poses a more significant issue amid a lack of extraneous threats to health (Zheng & Yang, 2012).

However, further variation in this relationship by gender suggests that different sources of social advantage/disadvantage may interact in unexpected ways. For instance, college-educated men experienced significantly increased class II/III obesity-associated mortality compared to other levels of education, whereas similarly-educated women saw no significant differences in mortality risk at this weight status. Mortality risk for college-educated women was instead most different from other levels of educational attainment when examining mortality associated with class I obesity. Similarly, among men, reduced mortality risk associated with overweight was found across all levels of education less than a college degree, as opposed to

only for women with less than a high school education.  This set of findings suggests that the threshold at which obesity poses a significant mortality risk may be *higher* for college-educated men than women, while the social disadvantage (i.e. education) threshold at which overweight reduces mortality risk may be *lower* for men. While the exact mechanisms underlying these associations are difficult to ascertain given current data, future research may seek to consider both the additive and multiplicative effects of different sociodemographic characteristics on the relationship between BMI and mortality[2].

This study also highlights a methodological consideration for future work in this area, namely that a dichotomized treatment of educational attainment (such as that in Zheng & Yang, 2012) may obscure potentially interesting nuances in the effect of education on BMI-associated mortality. For instance, the "protective" effects of overweight are true for all groups except those with at least a college education, while obesity is not associated with increased mortality risk only among individuals with a high school education or less. Thus, the functional form of education's moderating effect on the BMI-mortality relationship is highly variant depending on the weight status of interest. With reference to Table 3a, it is clear that overweight-associated mortality can be effectively summarized by comparing college-educated adults to those with less education. However, class I obesity-associated mortality necessitates the inclusion of less than high school, college or greater, and an intermediate level of educational attainment to best demonstrate variation in the relationship. Finally, class II/III obesity-associated mortality can once again be most parsimoniously explained with only two educational groups; except in this

---

[2] For instance, additional analyses (not shown) considered the role of race as a critical source of variation with respect to both education and the interaction of education and gender. While preliminary analyses suggest differences between black and white adults (as well as black and white males and females) at different levels of education, cell sizes were too small to yield stable estimates and are excluded from the results.

case the comparison would be made between those with a high school education or less and any greater level of educational attainment.

Prior to drawing further conclusions about the more far-reaching implications of these results, it is worth noting that there are limitations to the analytic approach used in this study. Though the self-reported measures of height and weight in NHIS are comparable to other studies, and similar results are obtained for modeling the BMI-mortality relationship when using objective height and weight in NHANES (Zheng & Yang, 2012; Masters, 2013; ancillary analyses in this paper), there is continued debate over the validity of BMI as an accurate measure of weight status and health (McAdams et al., 2007). While body fat percentage, skinfolds, or even adding waist circumference can help to provide a better measure of individuals' health relative to weight (Burkhauser & Cawley, 2008), BMI will likely remain the primary measure of weight status as it is relatively easy and low-cost with respect to data collection. However, results of my ancillary analysis using a measure of "high-risk" waist circumference from NHANES in lieu of BMI do not uncover significant differences in relative mortality risk by educational attainment, suggesting that further work is necessary in: (1) developing broader consensus on the validity of existing measures of body composition in population research, and the appropriateness of using these measures in analyses; and (2) integrating this knowledge towards better understanding socioeconomic differences in mortality associated with high body weight and/or obesity.

Furthermore, this BMI value is being assessed at only one point in time, and I assume that this value remains constant through respondents' time in the study window. Thus, despite robustness to different specifications of follow-up time (including a 5-year follow-up, during which BMI is relatively unlikely to exhibit significant variation (Heo et al., 2002), or to increase

marginally (Hopman et al., 2007)), my analysis cannot accommodate possible changes in weight, in terms of weight loss and weight gain, both of which have been found be associated with health and mortality outcomes in a number of longitudinal studies (Zajacova & Alishire, 2014; Myrskyla & Chang, 2009). Notably, Zheng et al. (2013) apply latent class trajectory models to adults aged 51-77 in the US Health and Retirement Study (HRS) and find that people who remained stably overweight had the highest survival rate, followed by those moving from overweight to obesity, normal weight upward, class I obese upward, normal weight downward, and class II/III obese upward trajectories. Most critically, their analyses suggested that "BMI trajectories were more predictive of mortality risk than was static BMI status", and that these later life increasing trajectories of obesity "pose a substantive threat to future gains in life expectancy" (Zheng et al., 2013: 1591). Thus, any bias induced by a static rather than time-variant measure of BMI is likely to be downward, suggesting that the results presented in this study may in fact be conservative estimates of relative mortality risk.

Statistical power also poses an issue for some of the sociodemographic groups used in these models. While the overall sample size is very large, extending the analysis of mortality risk among underweight participants to the larger population is difficult given the relatively small cell counts and high standard errors, especially when further stratifying by education and then gender. This is also true of severely obese individuals, who are also less frequently occurring in the data as compared to normal and overweight. Finally, the specification of only five BMI categories, while a commonly used approach, does not allow for more fine-grained analysis of variations in mortality risk within the various weight status levels. Individuals at the low end of the "normal" BMI range might have relatively high mortality risks, given the proximity to underweight, while the opposite may be true at the high end of normal, where the protective

effects of overweight may be evident. The current models are only capable of providing a summary mortality risk for a given BMI group, and any significant internal variation may be unaccounted for.

Despite these limitations, I believe that the present study helps to show that while there is still uncertainty in the overall relationship between BMI and mortality, sociodemographic variation in the magnitude and shape of this relationship is an important domain for further analysis. Lower-educated individuals have the highest premature mortality risk (Table 2), yet higher-educated groups experience significantly higher relative mortality risk associated with the overweight and obese categories. The central tenets of fundamental cause theory would maintain that low-educated adults might have higher mortality at unhealthy weights on account of their inability to access the resources (medical, financial, information/knowledge, and others) that could lead to improved health outcomes and potentially delay death. However, the results seem to instead suggest that a very high BMI is more closely associated with increased relative mortality risk among advantaged groups, such as the highly-educated.

Lack of confirmation for the first explanatory hypothesis (H1) does not imply that this study should be used as evidence to undermine fundamental cause theory. I instead propose an intermediary explanation that bridges fundamental cause theory with the explanatory arguments laid out in the second hypothesis, arguing that BMI – as a more "proximate" health risk – is likely to be more closely associated with mortality for highly-educated individuals than it is for lower educated. In other words, the causes and pathways of premature death for low-educated individuals, even when limiting analyses to non-external causes of death associated with obesity, may be less contingent upon weight and BMI than among highly-educated groups.

For instance, there are likely to be differences in mortality risk associated with the baseline reference for "normal" weight. In the case of education, Montez and Zajacoa (2013) use NHIS-LMF data to observe that nearly 47% of the variation in education-attributable mortality is due to smoking, as reflected in lung cancer and chronic lower respiratory disease. While these are two causes of death where BMI may play some role in shaping mortality outcomes (Yang et al., 2011), it may very well be the case that educational differences in smoking behavior also lead to differences in the *degree* to which the effects of smoking obscure any additional effect that obesity may have. That is to say, among lower-educated individuals mortality risk for the above conditions associated with normal weight is already significantly elevated, and thus being severely obese is unlikely to increase mortality risk any further. This important point is highlighted in Figure 1, presenting the cumulative mortality risks associated with educational attainment, BMI weight category, and the interacting of the two (using normal weight adults with 16+ years of educational attainment as the reference group). Despite the significantly elevated relative mortality risk associated with severe obesity for the highly educated respondents, it is still approximately 20% less than the *lowest* mortality risk for the least educated individuals (corresponding with overweight respondents). Most drastically, normal weight, college-educated adults have a *3.5 to 4 times lower relative mortality risk* than their severely obese, lower-educated counterparts. Overall we can observe a clear educational gradient across all weight categories, whereby the most highly educated respondents have a consistently at lower mortality risk regardless of their BMI, with the most notable discrepancies evident in the normal weight BMI range.

Furthermore, the main causes of death are similar for most Americans, such as conditions of the heart and malignant neoplasms (which, combined, account for 78% of the deaths in this

study). BMI is associated with both of these conditions (Eckel & Krauss 1998; Renehan et al., 2008), and studies have examined variation in cause-specific BMI-mortality risk for heart disease and many types of cancer (Flegal et al., 2007). However, these studies do not stratify by education, and it remains unclear whether the relationship between BMI and these diseases (as well as other leading causes of death) is similar for those with different education levels. Even in the case of heart disease, for which overweight and obesity pose significant risk factors, research has shown that a number of factors contribute to educational differences in coronary heart disease among adults. Unregulated blood pressure, due to negative psychosocial profiles and increased stress, the co-incidence of other hypertensive diseases, such as diabetes, and reduced social and emotional support and networks all act to increase the risk of heart disease prevalence for those with low education (Matthews et al., 1989).

Critically, the above causes of death are incredibly multi-factorial, and possibly not directly related to obesity among low-educated adults, thus helping to account for underlying differences in normal-weight mortality risk for obesity-related causes of death. It is possible that low-educated individuals experience mortality from causes *associated* with unhealthy weight but not *resulting* from unhealthy weight, and that instead may be attributable to adverse social circumstances. In this respect we can better appreciate the immense flexibility of fundamental cause theory, and the role of the Blaxter hypothesis as an *extension* of this theory. Given that Link and Phelan (1995) and others (Pampel et al., 2010) describe how the elimination of SES differences in health behaviors is unlikely to change the underlying relationship between SES and health, the Blaxter hypothesis and its applications demonstrate how the more "upstream", or fundamental, inequalities in SES may inhibit the effectiveness of efforts to mitigate more "downstream", or proximate, determinants of health such as how individuals' weight status

impacts mortality. As a comparable example, research into the effects of SES on the relationship between smoking and mortality has considered how curbing tobacco use among low-SES groups is unlikely to result in significant health or longevity benefits given the many competing sources of stress and danger in their lives (Bosma et al., 1999; Pampel & Rogers, 2004), and that population-wide public health initiatives aimed at reducing smoking behavior may only serve to exacerbate existing health inequalities by failing to address deeper-seed causes (Lawlor et al., 2003).

Consequently, the results of this paper should not be taken as evidence suggesting that future policies addressing the negative health consequences of high BMI and obesity should primarily target highly-educated individuals due to their elevated relative risk of mortality, while ignoring the issue among those with less education. Rather, this research highlights the importance of identifying and better specifying population-specific causes of mortality and adverse health, as well as the individual-level characteristics (e.g. BMI) that may increase or decrease relative risk within these populations. For instance, it is well-established that having an "unhealthy" BMI is not the only risk factor for being diagnosed with and/or dying from heart disease or certain types of cancer; and the present study suggests that these underlying risks appears to differ across socioeconomic groups. A better understanding of these differences may allow policymakers and public health advocates to avoid making sweeping generalizations about the immediate consequences of being overweight and even mildly obese, while ignoring the underlying structural inequalities contributing to divergent health outcomes.

More broadly, we can also take an opportunity to consider these findings as further evidence questioning the meaning and measures of BMI, or obesity, as a covariate in our models of morbidity and mortality. For instance, some measure of weight status is frequently used as a

proxy for unhealthy behavior or as a poor health outcome. This is a perfectly legitimate methodological decision, especially when considering the close link between obesity and certain chronic diseases and conditions. However, recent studies are beginning to more systematically examine many of the assertions we have about obesity, based on BMI, as a genuine indicator of unhealthiness in and of itself (Tomiyama et al., 2016), and much research has tested the degree to which BMI's accuracy and validity may vary based on certain sociodemographic factors (Gallagher et al., 1996; Deurenberg et al., 1998; Prentice & Jebb, 2001). This present study can be viewed as in conversation with this body of work, demonstrating the degree to which mortality outcomes fail to align with our expectations of unhealthy bodyweight among certain population groups. However, given the lack of any biomarker data or ability to "medically" gauge individuals' true cardiometabolic health, further research would be necessary to more rigorously test the underlying mechanisms explaining this variation in the relationship between BMI and mortality.

Despite this potential methodological pitfall of misclassifying certain BMI groups as physically unhealthy, I believe it is worthwhile to consider examining other potential "modifiers" of BMI-attributable mortality to better understanding why some groups fare better or worse than others depending on their weight status. Much of the work in this area has focused on age, gender, and race/ethnicity as key modifiers, but this study reaffirms the importance of education, and SES more broadly, as a potential source of variation. Continued research on this subject would benefit from improving measures of SES by integrating income and other wealth variables, as Zheng and Yang (2012) provide additional evidence of variation in BMI-associated mortality across income quartiles. Moreover, many of the social determinants of health studied in sociology and public health serve as likely candidates for future analysis as well. Family

background, social support, neighborhood characteristics, and other factors might further help to explain variation the BMI-mortality relationship, helping to identify those social circumstances putting individuals at higher risk. The above is equally true for the underweight category. While variation in underweight mortality risk was difficult to assess in this study, this is not a weight status group that should be excluded from analysis when examining BMI-associated mortality. Though the mortality risk may be high because of the presence of sick and dying individuals, attempting to parse out the effects of underweight among otherwise "healthy" individuals will allow researchers to more accurately gauge the health consequences (positive or negative) of having a low body weight.

Finally, I recommend continued work in examining spatial variation in the BMI-mortality relationship. Given the vast differences in international approaches to healthcare and medicine – as well as cultural variations with respect to diet and exercise – it would be very interesting to see how these socio-structural differences are manifest in BMI-attributable mortality. I might expect the overall shape of this risk curve to vary substantially, especially in nations with universal healthcare for whom the BMI-associated chronic disease might be better managed, thus reducing both overall and sociodemographically stratified BMI-associated mortality. These kind of comparisons can be done at the state level within the US, given the country's relatively heterogeneous composition with respect to both population and healthcare services. Current research on inter-"regional" and international comparisons is sparse (Visscher et al., 2000; Murray et al., 2006), while preliminary work can be conducted using this same NHIS data and computing relative risks by geographic region. Ultimately, combining these types of spatially-focused analyses with the existing and ongoing work in sociodemographic variation can help to

resolve outstanding questions and uncertainties about the relationship between BMI and

mortality.

# APPENDIX: TABLES & FIGURES

**Table 1: Baseline Characteristics of 1997-2011 NHIS-LMF(Ages 30-64)[a]: Survivors vs. Premature Mortality[b]**

| Variable | Survivivors | Premature Death |
|---|---|---|
| *BMI Group* | | |
| Underweight (<18.0) | 1.3% | 2.9% |
| Normal (18.0-24.9) | 35.0% | 32.0% |
| Overweight (25.0-29.9) | 36.6% | 33.3% |
| Obese, class I (30.0-34.9) | 16.9% | 17.5% |
| Obese, class II/III (35.0+) | 10.1% | 14.3% |
| *Age at Survey* | | |
| Mean (SD) | 45.3 (9.7) | 50.2 (7.5) |
| *Gender* | | |
| Female | 54.4% | 45.2% |
| Male | 45.6% | 54.8% |
| *Race/Ethnicity* | | |
| Non-HispanicWhite | 64.7% | 62.6% |
| Non-Hispanic Black | 14.4% | 21.5% |
| Hispanic | 16.9% | 12.6% |
| Non-Hispanic Asian/PI | 3.4% | 2.2% |
| Native/American Indian | 0.6% | 1.0% |
| Other | 0.1% | 0.1% |
| *Foreign born* | | |
| Yes | 17.9% | 11.6% |
| *Education* | | |
| Less than HS | 16.0% | 25.7% |
| HS | 28.0% | 32.7% |
| Some College | 28.6% | 26.5% |
| College + | 27.4% | 15.1% |
| *Alcohol Use* | | |
| Lifetime abstainer | 19.4% | 19.3% |
| Former drinker | 14.9% | 25.8% |
| Current drinker | 65.7% | 55.0% |
| *Smoker status* | | |
| Never smoked | 53.8% | 32.0% |
| Former smoker | 21.4% | 23.1% |
| Current smoker | 24.8% | 45.0% |
| | | |
| *Sample size* | 206,753 | 4,699 |

N=211,452

[a]All estimated based on unweighted values

[b]Premature mortality defined as death occuring between 30 and 65

**Table 2: Overall Model for BMI-associated Working-Aged Mortality (Ages 30-64)**

| Variable | Haz. Ratio | | 95% CI | |
|---|---|---|---|---|
| *(reference: "Normal weight")* | | | | |
| Underweight | 2.613 | *** | 2.116 | 3.225 |
| Overweight | 0.861 | ** | 0.791 | 0.937 |
| Obese, class I | 0.967 | | 0.874 | 1.069 |
| Obese, class II/III | 1.403 | *** | 1.253 | 1.570 |
| *Sociodemographic controls* | | | | |
| Female | 0.674 | *** | 0.628 | 0.724 |
| Race/Ethnicity (reference: "White") | | | | |
| Black | 1.535 | *** | 1.406 | 1.675 |
| Hispanic | 1.038 | | 0.908 | 1.186 |
| Asian/Pacific Islander | 1.295 | * | 1.013 | 1.655 |
| Native or Indian American | 1.076 | | 0.761 | 1.521 |
| Other | 1.276 | | 0.432 | 3.769 |
| Foreign born | 0.801 | ** | 0.692 | 0.926 |
| Education (reference: Less than High school) | | | | |
| High school | 0.759 | *** | 0.692 | 0.834 |
| Some college | 0.675 | *** | 0.611 | 0.746 |
| College or higher | 0.496 | *** | 0.440 | 0.558 |
| *Health Behaviors* | | | | |
| Alcohol use (reference: Lifetime Abstainer) | | | | |
| smoker | 1.139 | * | 1.023 | 1.268 |
| smoker | 0.701 | *** | 0.636 | 0.772 |
| Smoker status (reference: Never smoked) | | | | |
| smoker | 1.353 | *** | 1.229 | 1.489 |
| smoker | 2.502 | *** | 2.301 | 2.720 |

† for p<0.10; * for p<0.05; ** for p<0.01; *** for p<0.001

N = 211,452

## Table 3a: Multivariate Relative Risk of Working-Aged (Age 30-64) Mortality, in Relation to BMI[a]; By Educational Attainment

| | Less than HS | | HS | | Overall Some College | | College or More | |
|---|---|---|---|---|---|---|---|---|
| BMI Group[b] | Haz. Ratio | 95% CI | Haz. Ratio | 95% CI | Haz. Ratio | 95% CI | Haz. Ratio | 95% CI |
| Underweight (<18.5) | 2.470 *** | 1.674 3.645 | 1.864 ** | 1.268 2.740 | 3.427 *** | 2.314 5.075 | 3.397 *** | 1.932 5.972 |
| Overweight (25.0-29.9) | 0.786 ** | 0.661 0.935 | 0.809 ** | 0.698 0.939 | 0.813 * | 0.689 0.960 | 1.146 | 0.929 1.414 |
| Obese, Class I (30.0-34 | 0.800 * | 0.656 0.977 | 0.938 | 0.788 1.116 | 0.927 | 0.763 1.127 | 1.373 * | 1.050 1.795 |
| Obese, Class II/III (35.0 | 1.147 | 0.921 1.429 | 1.445 *** | 1.185 1.763 | 1.272 * | 1.030 1.571 | 1.970 *** | 1.456 2.666 |
| | N=34,285 | | N=59,351 | | N=60,422 | | N=57,394 | |

† for p<0.10; * for p<0.05; ** for p<0.01; *** for p<0.001

[a]Multivariate model used age as the underlying time metric and included the following combinations of covariates: gender, race/ethnicity, foreign born status, level of education, alcohol use, and smoking status.

51

**Table 3b: Multivariate Relative Risk of Working-Aged (Age 30-64) Mortality, in Relation to BMI[a]; By Educational Attainment and Gender**

### Male

| BMI Group[b] | Less than HS | | HS | | Some College | | College or More | |
|---|---|---|---|---|---|---|---|---|
| | Haz. Ratio | 95% CI | Haz. Ratio | 95% CI | Haz. Ratio | 95% CI | Haz. Ratio | 95% CI |
| *Underweight (<18.5)* | 2.548 ** | 1.357  4.785 | 2.418 ** | 1.320  4.428 | 6.006 *** | 3.154  11.436 | 2.071 | 0.600  7.148 |
| *Overweight (25.0-29.9)* | 0.800 † | 0.637  1.004 | 0.757 ** | 0.621  0.923 | 0.728 ** | 0.582  0.911 | 1.105 | 0.847  1.442 |
| *Obese, Class I (30.0-34.9)* | 0.774 † | 0.591  1.014 | 0.903 | 0.712  1.146 | 0.878 | 0.675  1.142 | 1.138 | 0.806  1.606 |
| *Obese, Class II/III (35.0* | 1.094 | 0.791  1.514 | 1.379 * | 1.045  1.820 | 1.181 | 0.861  1.621 | 2.213 *** | 1.481  3.309 |
| | N=15,678 | | N=27,284 | | N=26,337 | | N=27,561 | |

### Female

| BMI Group[b] | Less than HS | | HS | | Some College | | College or More | |
|---|---|---|---|---|---|---|---|---|
| | Haz. Ratio | 95% CI | Haz. Ratio | 95% CI | Haz. Ratio | 95% CI | Haz. Ratio | 95% CI |
| *Underweight (<18.5)* | 2.441 *** | 1.483  4.020 | 1.646 * | 1.000  2.711 | 2.714 *** | 1.669  4.414 | 4.147 *** | 2.189  7.855 |
| *Overweight (25.0-29.9)* | 0.770 † | 0.590  1.005 | 0.881 | 0.706  1.099 | 0.958 | 0.752  1.220 | 1.189 | 0.846  1.673 |
| *Obese, Class I (30.0-34.* | 0.832 | 0.618  1.121 | 0.957 | 0.744  1.232 | 0.970 | 0.724  1.299 | 1.875 ** | 1.247  2.820 |
| *Obese, Class II/III (35.0* | 1.168 | 0.857  1.593 | 1.488 ** | 1.117  1.983 | 1.355 * | 1.022  1.797 | 1.642 * | 1.032  2.613 |
| | N=18,607 | | N=32,067 | | N=34,085 | | N=29,833 | |

† for p<0.10; * for p<0.05; ** for p<0.01; *** for p<0.001

[a]Multivariate model used age as the underlying time metric and included the following combinations of covariates: gender, race/ethnicity, foreign born status, level of education, alcohol use, and smoking status.

[b]Significance level compares BMI category to reference group (Normal weight).

**Table 4a: Comparison of Relative Risk of Working-Aged (30-64) Mortality, in Relation to BMI[a]; Across Education**

| Ratio of Risk by Group[b] | Underweight (<18.5) | | Overweight (25.0-29.9) | | Obese, Class I (30.0-34.9) | | Obese, Class II/III (35.0+) | |
|---|---|---|---|---|---|---|---|---|
| | *RR* | *95% CI* | *RR* | *95% CI* | *RR* | *95% CI* | *RR* | *95% CI* |
| *HS/<HS* | 0.748 | 0.433 1.294 | 1.028 | 0.819 1.292 | 1.169 | 0.897 1.523 | 1.256 | 0.934 1.689 |
| *Some College/<HS* | 1.380 | 0.794 2.398 | 1.039 | 0.818 1.321 | 1.164 | 0.881 1.538 | 1.108 | 0.818 1.501 |
| *College+/<HS* | 1.353 | 0.682 2.685 | 1.464 ** | 1.116 1.921 | 1.726 ** | 1.236 2.409 | 1.724 ** | 1.188 2.502 |
| *Some College/HS* | 1.844 * | 1.063 3.197 | 1.011 | 0.809 1.262 | 0.996 | 0.767 1.293 | 0.882 | 0.660 1.178 |
| *College+/HS* | 1.808 † | 0.913 3.581 | 1.424 ** | 1.102 1.840 | 1.477 * | 1.073 2.032 | 1.372 † | 0.957 1.969 |
| *College+/Some Colleg* | 0.980 | 0.493 1.950 | 1.409 * | 1.079 1.840 | 1.482 * | 1.065 2.063 | 1.556 * | 1.078 2.247 |

† for p<0.10; * for p<0.05; ** for p<0.01; *** for p<0.001

[a]Multivariate model used age as the underlying time metric and included the following combinations of covariates: gender, race/ethnicity, foreign born status, level of education, alcohol use, and smoking status.

[b]

**Table 4b: Comparison of Relative Risk of Working-Aged (30-64) Mortality, in Relation to BMI[a]; Across Education**

**Male**

| Ratio of Risk by Group[b] | Underweight (<18.5) | | Overweight (25.0-29.9) | | Obese, Class I | | Obese, Class II/III (35.0+) | |
|---|---|---|---|---|---|---|---|---|
| | RR | 95% CI | RR | 95% CI | RR | 95% CI | RR | 95% CI |
| HS/<HS | 0.932 | 0.390 2.228 | 0.944 | 0.698 1.277 | 1.159 | 0.809 1.661 | 1.249 | 0.814 1.916 |
| Some College/<HS | 2.336 † | 0.950 5.744 | 0.912 | 0.662 1.254 | 1.135 | 0.779 1.653 | 1.069 | 0.679 1.683 |
| College+/<HS | 0.793 | 0.196 3.199 | 1.379 † | 0.972 1.957 | 1.464 † | 0.945 2.267 | 2.012 ** | 1.200 3.375 |
| Some College/HS | 2.505 * | 1.037 6.053 | 0.965 | 0.716 1.302 | 0.979 | 0.687 1.395 | 0.856 | 0.562 1.304 |
| College+/HS | 0.850 | 0.213 3.393 | 1.460 * | 1.049 2.034 | 1.263 | 0.831 1.920 | 1.611 † | 0.989 2.626 |
| College+/Some Colleg | 0.339 | 0.084 1.379 | 1.513 * | 1.069 2.141 | 1.290 | 0.837 1.988 | 1.881 * | 1.129 3.136 |

**Female**

| | Underweight (<18.5) | | Overweight (25.0-29.9) | | Obese, Class I | | Obese, Class II/III (35.0+) | |
|---|---|---|---|---|---|---|---|---|
| | RR | 95% CI | RR | 95% CI | RR | 95% CI | RR | 95% CI |
| HS/<HS | 0.673 | 0.332 1.364 | 1.141 | 0.808 1.612 | 1.153 | 0.780 1.703 | 1.275 | 0.836 1.945 |
| Some College/<HS | 1.117 | 0.556 2.247 | 1.253 | 0.875 1.795 | 1.177 | 0.776 1.785 | 1.166 | 0.767 1.771 |
| College+/<HS | 1.676 | 0.745 3.771 | 1.562 * | 1.016 2.402 | 2.289 ** | 1.381 3.793 | 1.414 | 0.811 2.465 |
| Some College/HS | 1.660 | 0.825 3.341 | 1.098 | 0.792 1.523 | 1.021 | 0.694 1.501 | 0.914 | 0.611 1.367 |
| College+/HS | 2.490 * | 1.106 5.605 | 1.369 | 0.914 2.050 | 1.985 ** | 1.229 3.207 | 1.109 | 0.643 1.911 |
| College+/Some Colleg | 1.500 | 0.671 3.354 | 1.246 | 0.823 1.889 | 1.945 ** | 1.178 3.210 | 1.213 | 0.706 2.083 |

† for p<0.10; * for p<0.05; ** for p<0.01; *** for p<0.001

[a]Multivariate model used age as the underlying time metric and included the following combinations of covariates: gender, race/ethnicity, foreign born status, level of education, alcohol use, and smoking status.

**Table 5a: NHIS Multivariate Relative Risk of Non-External Working-Aged (Age 30-64) Mortality, in Relation to BMI[a]; By Educational Attainment**

5-Year Mortality Follow-up: N(Deaths) = 2,451

| BMI Group[b] | Less than HS | | HS | | Some College | | College or More | |
|---|---|---|---|---|---|---|---|---|
| | HR | 95% CI | HR | 95% CI | HR | 95% CI | HR | 95% CI |
| Underweight (<18.5) | 3.787 *** | 2.372 6.046 | 2.140 ** | 1.273 3.597 | 4.459 *** | 2.750 7.231 | 4.757 *** | 2.356 9.605 |
| Overweight (25.0-29.9) | 0.891 | 0.703 1.128 | 0.678 *** | 0.548 0.839 | 0.751 * | 0.594 0.950 | 1.124 | 0.824 1.533 |
| Obese, Class I (30.0-34.9) | 0.700 * | 0.525 0.935 | 0.928 | 0.725 1.189 | 0.898 | 0.686 1.175 | 1.700 ** | 1.170 2.470 |
| Obese, Class II/III (35.0+) | 1.171 | 0.862 1.590 | 1.406 * | 1.053 1.878 | 1.071 | 0.787 1.457 | 2.440 *** | 1.621 3.673 |

Full Mortality Follow-up, Excluding First Two years: N(Deaths) = 3,655

| BMI Group | Less than HS | | HS | | Some College | | College or More | |
|---|---|---|---|---|---|---|---|---|
| | HR | 95% CI | HR | 95% CI | HR | 95% CI | HR | 95% CI |
| Underweight (<18.5) | 1.672 * | 1.040 2.687 | 1.700 * | 1.088 2.657 | 2.412 *** | 1.530 3.802 | 2.346 * | 1.167 4.719 |
| Overweight (25.0-29.9) | 0.786 * | 0.646 0.957 | 0.872 | 0.739 1.029 | 0.853 † | 0.708 1.028 | 1.195 | 0.950 1.504 |
| Obese, Class I (30.0-34.9) | 0.904 | 0.725 1.126 | 0.963 | 0.791 1.174 | 0.899 | 0.721 1.121 | 1.317 † | 0.973 1.781 |
| Obese, Class II/III (35.0+) | 1.199 | 0.941 1.528 | 1.641 *** | 1.317 2.046 | 1.394 ** | 1.108 1.753 | 1.674 ** | 1.187 2.360 |

† for p<0.10; * for p<0.05; ** for p<0.01; *** for p<0.001
[a]Multivariate model used age as the underlying time metric and included the following covariates: gender, race/ethnicity, foreign born status, and smoking status.

55

**Table 5b: NHANES (1999-2010) Multivariate Relative Risk of Mortality, in Relation to BMI[a] and Waist Circumference; By Educational Attainment**

All-Cause Mortality, All-Ages: N(Deaths) = 3,663

| BMI Group[b] | Less than HS | | | HS | | | Some College | | | College or More | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | HR | 95% CI | | HR | 95% CI | | HR | 95% CI | | HR | 95% CI | |
| Underweight (<18.5) | 1.517 | 0.898 | 2.562 | 1.253 | 0.325 | 4.825 | 1.169 | 0.469 | 2.916 | 2.114 | 0.860 | 5.199 |
| Overweight (25.0-29.9) | 0.802 * | 0.646 | 0.996 | 0.744 * | 0.569 | 0.974 | 0.822 | 0.618 | 1.093 | 0.826 | 0.565 | 1.208 |
| Obese, Class I (30.0-34.9) | 0.955 | 0.738 | 1.237 | 0.837 | 0.613 | 1.143 | 1.115 | 0.787 | 1.580 | 0.861 | 0.543 | 1.363 |
| Obese, Class II/III (35.0+) | 0.966 | 0.728 | 1.282 | 1.257 | 0.871 | 1.815 | 1.318 | 0.862 | 2.014 | 1.831 * | 1.011 | 3.316 |
| High-Risk Waist Circumference | 1.046 | 0.863 | 1.269 | 1.249 † | 0.977 | 1.597 | 1.090 | 0.850 | 1.399 | 1.020 | 0.727 | 1.432 |

Non-External Mortality, All-Ages: N(Deaths) = 2,396

| BMI Group[b] | Less than HS | | | HS | | | Some College | | | College or More | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | HR | 95% CI | | HR | 95% CI | | HR | 95% CI | | HR | 95% CI | |
| Underweight (<18.5) | 1.504 | 0.793 | 2.851 | 0.900 | 0.182 | 4.457 | 1.325 | 0.388 | 4.519 | 1.154 | 0.459 | 2.901 |
| Overweight (25.0-29.9) | 0.744 * | 0.568 | 0.975 | 0.717 * | 0.522 | 0.986 | 0.868 | 0.614 | 1.226 | 0.716 | 0.457 | 1.122 |
| Obese, Class I (30.0-34.9) | 0.970 | 0.704 | 1.336 | 0.809 | 0.554 | 1.182 | 1.334 | 0.884 | 2.014 | 0.642 | 0.375 | 1.098 |
| Obese, Class II/III (35.0+) | 1.013 | 0.704 | 1.457 | 1.144 | 0.715 | 1.832 | 1.143 | 0.685 | 1.906 | 1.451 | 0.677 | 3.109 |
| High-Risk Waist Circumference | 1.082 | 0.851 | 1.374 | 1.332 † | 0.989 | 1.792 | 1.179 | 0.876 | 1.588 | 0.780 | 0.530 | 1.148 |

All-Cause Mortality, Ages 30-64: N(Deaths) = 606

| BMI Group[b] | Less than HS | | | HS | | | Some College | | | College or More | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | HR | 95% CI | | HR | 95% CI | | HR | 95% CI | | HR | 95% CI | |
| Underweight (<18.5) | 2.169 | 0.767 | 6.138 | 3.565 | 0.799 | 15.909 | 0.568 | 0.075 | 4.311 | 2.744 | 0.353 | 21.361 |
| Overweight (25.0-29.9) | 0.981 | 0.601 | 1.602 | 0.577 † | 0.307 | 1.086 | 0.540 † | 0.292 | 1.000 | 0.444 | 0.149 | 1.322 |
| Obese, Class I (30.0-34.9) | 1.035 | 0.588 | 1.823 | 0.684 | 0.338 | 1.384 | 1.043 | 0.555 | 1.960 | 0.797 | 0.256 | 2.479 |
| Obese, Class II/III (35.0+) | 1.146 | 0.646 | 2.036 | 1.532 | 0.799 | 2.937 | 1.264 | 0.688 | 2.324 | 1.949 | 0.702 | 5.410 |
| High-Risk Waist Circumference | 1.334 | 0.876 | 2.031 | 1.243 | 0.756 | 2.044 | 1.114 | 0.709 | 1.752 | 1.334 | 0.615 | 2.893 |

Non-External Mortality, Ages 30-64: N(Deaths) = 359

| BMI Group[b] | Less than HS | | | HS | | | Some College | | | College or More | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | HR | 95% CI | | HR | 95% CI | | HR | 95% CI | | HR | 95% CI | |
| Underweight (<18.5) | 1.630 | 0.283 | 9.374 | 2.759 | 0.374 | 20.348 | 1.101 | 0.137 | 8.844 | 0.000 *[:] | 0.000 | 0.000 |
| Overweight (25.0-29.9) | 0.688 | 0.360 | 1.314 | 0.565 | 0.254 | 1.259 | 0.567 | 0.257 | 1.252 | 0.211 * | 0.051 | 0.867 |
| Obese, Class I (30.0-34.9) | 1.083 | 0.535 | 2.190 | 0.665 | 0.261 | 1.698 | 1.495 | 0.687 | 3.251 | 0.065 *[:] | 0.008 | 0.508 |
| Obese, Class II/III (35.0+) | 1.078 | 0.499 | 2.328 | 1.792 | 0.811 | 3.957 | 1.258 | 0.565 | 2.798 | 0.804 | 0.155 | 4.170 |
| High-Risk Waist Circumference | 1.568 | 0.873 | 2.818 | 1.373 | 0.723 | 2.606 | 1.321 | 0.724 | 2.408 | 0.376 † | 0.122 | 1.162 |

† for p<0.10; * for p<0.05; ** for p<0.01; *** for p<0.001

[a]Multivariate model used age as the underlying time metric and included the following covariates: gender, race/ethnicity, foreign born status, and smoking status.

Figure 1: NHIS 1997-2011: Relative Mortality Risk, Across BMI* and Education
(ref. 16+ Years of Education, Normal Weight)
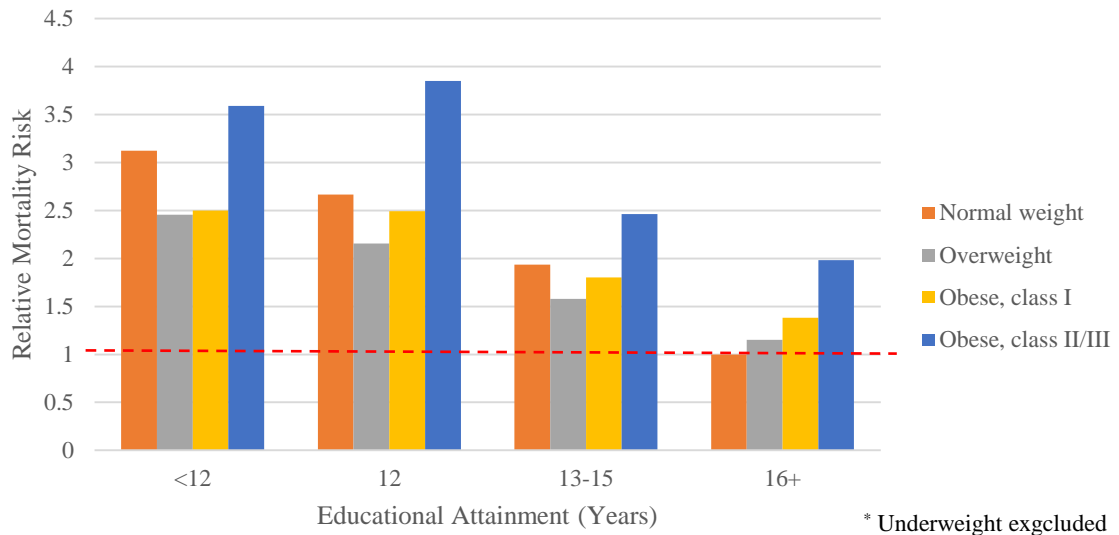
# REFERENCES

About the National Health Interview Survey. (2015). *Centers for Disease Control.* Retrieved from http://www.cdc.gov/nchs/nhis/about_nhis.htm

Adams, K. F., Schatzkin, A., Harris, T. B., Kipnis, V., Mouw, T., Ballard-Barbash, R., ... & Leitzmann, M. F. (2006). Overweight, obesity, and mortality in a large prospective cohort of persons 50 to 71 years old. *New England Journal of Medicine*, *355*(8), 763-778.

Allison, D. B., Gallagher, D., Heo, M., Pi-Sunyer, F. X., & Heymsfield, S. B. (1997). Body mass index and all-cause mortality among people age 70 and over: the Longitudinal Study of Aging. *International journal of obesity*, *21*(6), 424-431.

Altman, C. E., Van Hook, J., & Hillemeier, M. (2016). What Does Self-rated Health Mean? Changes and Variations in the Association of Obesity with Objective and Subjective Components Of Self-rated Health. *Journal of health and social behavior*, 0022146515626218.

Bender, R., Jöckel, K. H., Trautner, C., Spraul, M., & Berger, M. (1999). Effect of age on excess mortality in obesity. *Jama*, *281*(16), 1498-1504.

Blaxter, M. (1990). *Health and lifestyles*. Routledge.

Flanders, W. D., & Augestad, L. B. (2008). Adjusting for reverse causality in the relationship between obesity and mortality. *International Journal of Obesity*, *32*, S42-S46.

Bacon, S. (2013). Central adiposity. In *Encyclopedia of Behavioral Medicine* (pp. 368-369). Springer New York.

Bonnet, F., Deprele, C., Sassolas, A., Moulin, P., Berthezène, F., & Berthoux, F. (2001). Excessive body weight as a new independent risk factor for clinical and pathological progression in primary IgA nephritis. *American journal of kidney diseases*, *37*(4), 720-727.

Bosma, H., Schrijvers, C., & Mackenbach, J. P. (1999). Socioeconomic inequalities in mortality and importance of perceived control: cohort study. *BMJ*, *319*(7223), 1469-1470.

Brock, T. (2010). Young adults and higher education: Barriers and breakthroughs to success. *The Future of Children*, *20*(1), 109-132.

Burkhauser, R. V., & Cawley, J. (2008). Beyond BMI: the value of more accurate measures of fatness and obesity in social science research. *Journal of health economics*, *27*(2), 519-529.

Calle, E. E., & Thun, M. J. (2004). Obesity and cancer. *Oncogene*, *23*(38), 6365-6378.

Chang, V. W., & Lauderdale, D. S. (2009). Fundamental cause theory, technological innovation, and health disparities: the case of cholesterol in the era of statins. *Journal of Health and Social Behavior*, *50*(3), 245-260.

Corrada, M. M., Kawas, C. H., Mozaffar, F., & Paganini-Hill, A. (2006). Association of body mass index and weight change with all-cause mortality in the elderly. *American journal of epidemiology*, *163*(10), 938-949.

Curtis, J. P., Selter, J. G., Wang, Y., Rathore, S. S., Jovin, I. S., Jadbabaie, F., ... & Krumholz, H. M. (2005). The obesity paradox: body mass index and outcomes in patients with heart failure. *Archives of internal medicine*, *165*(1), 55-61.

Cutler, D. M., & Lleras-Muney, A. (2006). *Education and health: evaluating theories and evidence* (No. w12352). National Bureau of Economic Research.

Deurenberg, P., Yap, M., & Van Staveren, W. A. (1998). Body mass index and percent body fat: a meta analysis among different ethnic groups. *International journal of obesity*, *22*, 1164-1171.

Donini, L. M., Savina, C., Gennaro, E., De Felice, M. R., Rosano, A., Pandolfo, M. M., ... & Chumlea, W. C. (2012). A systematic review of the literature concerning the relationship between obesity and mortality in the elderly. *The journal of nutrition, health & aging*, *16*(1), 89-98.

Dranger, E., & Remington, P. (1998). U YPLL: A Summary Measure of Premature Mortality Used in Measuring the Health of Communities. *Planning*, *113*, 55-61.

Durazo-Arvizu, R., Cooper, R. S., Luke, A., Prewitt, T. E., Liao, Y., & McGee, D. L. (1997). Relative weight and mortality in US blacks and whites: findings from representative national population samples. *Annals of epidemiology*, *7*(6), 383-395.

Eckel, R. H., & Krauss, R. M. (1998). American Heart Association call to action: obesity as a major risk factor for coronary heart disease. *Circulation*, *97*(21), 2099-2100.

Ensrud, K. E., Ewing, S. K., Taylor, B. C., Fink, H. A., Stone, K. L., Cauley, J. A., ... & Study of Osteoporotic Fractures Research Group. (2007). Frailty and risk of falls, fracture, and mortality in older women: the study of osteoporotic fractures. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *62*(7), 744-751.

Bigaard, J., Tjønneland, A., Thomsen, B. L., Overvad, K., Heitmann, B. L., & Sørensen, T. I. (2003). Waist Circumference, BMI, Smoking, and Mortality in Middle-Aged Men and Women. *Obesity research*, *11*(7), 895-903.

Katzmarzyk, P. T., Janssen, I., Ross, R., Church, T. S., & Blair, S. N. (2006). The Importance of Waist Circumference in the Definition of Metabolic Syndrome Prospective analyses of mortality in men. *Diabetes care*, *29*(2), 404-409.

Koster, A., Leitzmann, M. F., Schatzkin, A., Mouw, T., Adams, K. F., van Eijk, J. T. M., ... & Harris, T. B. (2008). Waist circumference and mortality. *American journal of epidemiology*, *167*(12), 1465-1475.

Flegal, K. M., Graubard, B. I., Williamson, D. F., & Gail, M. H. (2005). Excess deaths associated with underweight, overweight, and obesity. *Jama*, *293*(15), 1861-1867.

Flegal, K. M., Graubard, B. I., Williamson, D. F., & Gail, M. H. (2007). Cause-specific excess deaths associated with underweight, overweight, and obesity.*Jama*, *298*(17), 2028-2037.

Fontaine, K. R., Redden, D. T., Wang, C., Westfall, A. O., & Allison, D. B. (2003). Years of life lost due to obesity. *Jama*, *289*(2), 187-193.

Gallagher, D., Visser, M., Sepulveda, D., Pierson, R. N., Harris, T., & Heymsfield, S. B. (1996). How useful is body mass index for comparison of body fatness across age, sex, and ethnic groups?. *American journal of epidemiology*, *143*(3), 228-239.

Glied, S., & Lleras-Muney, A. (2008). Technological innovation and inequality in health. *Demography*, *45*(3), 741-761.

Goldman, D. P., & Lakdawalla, D. N. (2005). A theory of health disparities and medical technology. *Contributions in Economic Analysis & Policy*, *4*(1).

Grabowski, D. C., & Ellis, J. E. (2001). High body mass index does not predict mortality in older people: analysis of the Longitudinal Study of Aging. *Journal of the American Geriatrics Society*, *49*(7), 968-979.

Hamer, M., & Stamatakis, E. (2012). Metabolically healthy obesity and risk of all-cause and cardiovascular disease mortality. *The Journal of Clinical Endocrinology & Metabolism*, *97*(7), 2482-2488.

Hayward, M. D., Hummer, R. A., & Sasson, I. (2014). Trends and group differences in the association between educational attainment and US adult mortality: Implications for understanding education's causal influence. *Social Science & Medicine*, *30*, 1e11.

He, J., McGee, D., Niu, X., & Choi, W. (2009). Examining the dynamic association of BMI and mortality in the Framingham Heart Study. *International journal of environmental research and public health*, *6*(12), 3115-3126.

Frankenfield, D. C., Rowe, W. A., Cooney, R. N., Smith, J. S., & Becker, D. (2001). Limits of body mass index to detect obesity and predict body composition. *Nutrition*, *17*(1), 26-30.

Heath, A. F. (2009). *Confusing credentials: The cross-nationally comparable measurement of educational attainment* (Doctoral dissertation, University of Oxford).

Hubert, H. B., Feinleib, M., McNamara, P. M., & Castelli, W. P. (1983). Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. *Circulation*, *67*(5), 968-977.

Hummer, R. A., & Hernandez, E. M. (2013). The Effect of Educational Attainment on Adult Mortality in the United States*. *Population bulletin*, *68*(1), 1.

Hummer, R. A., & Lariscy, J. T. (2011). Educational attainment and adult mortality. In *International handbook of adult mortality* (pp. 241-261). Springer Netherlands.

Imai, K., Gregg, E. W., Chen, Y. J., Zhang, P., Rekeneire, N., & Williamson, D. F. (2008). The Association of BMI With Functional Status and Self-rated Health in US Adults. *Obesity*, *16*(2), 402-408.

Isozumi, K. (2004). Obesity as a risk factor for cerebrovascular disease. *The Keio journal of medicine*, *53*(1), 7-11.

Jain, S., & Chaves, S. S. (2011). Obesity and influenza. *Clinical infectious diseases*, *53*(5), 422-424.

Janssen, I., Katzmarzyk, P. T., & Ross, R. (2005). Body mass index is inversely related to mortality in older people after adjustment for waist circumference. *Journal of the American Geriatrics Society*, *53*(12), 2112-2118.

Kaplan, G. A., Seeman, T. E., Cohen, R. D., Knudsen, L. P., & Guralnik, J. (1987). Mortality among the elderly in the Alameda County Study: behavioral and demographic risk factors. *American Journal of Public Health*, *77*(3), 307-312.

Katzmarzyk, P. T., Craig, C. L., & Bouchard, C. (2001). Original article underweight, overweight and obesity: relationships with mortality in the 13-year follow-up of the Canada Fitness Survey. *Journal of clinical epidemiology*, *54*(9), 916-920.

Kitahara, C. M., Flint, A. J., de Gonzalez, A. B., Bernstein, L., Brotzman, M., MacInnis, R. J., ... & Weiderpass, E. (2014). Association between class III obesity (BMI of 40–59 kg/m 2) and mortality: a pooled analysis of 20 prospective studies. *PLoS Med*, *11*(7), e1001673.

Kramer, C. K., Zinman, B., & Retnakaran, R. (2013). Are metabolically healthy overweight and obesity benign conditions?: A systematic review and meta-analysis. *Annals of internal medicine*, *159*(11), 758-769.

National Heart, Lung, and Blood Institute. (n.d.). Obesity Education Initiative Electronic Textbook--Treatment Guidelines. Retrieved from https://www.nhlbi.nih.gov/health-pro/guidelines/current/obesity-guidelines/e_textbook/txgd/4142.htm

Krueger, P. M., & Chang, V. W. (2008). Being poor and coping with stress: health behaviors and the risk of death. *American journal of public health*, *98*(5), 889-896.

Krueger, P. M., Rogers, R. G., Hummer, R. A., & Boardman, J. D. (2004). Body mass, smoking, and overall and cause-specific mortality among older US adults. *Research on Aging*, *26*(1), 82-107.

Krueger, P. M., Saint Onge, J. M., & Chang, V. W. (2011). Race/ethnic differences in adult mortality: the role of perceived stress and health behaviors. *Social science & medicine*, *73*(9), 1312-1322.

Kuk, J. L., & Ardern, C. I. (2009). Influence of age on the association between various measures of obesity and all-cause mortality. *Journal of the American Geriatrics Society*, *57*(11), 2077-2084.

Lawlor, D. A., Frankel, S., Shaw, M., Ebrahim, S., & Smith, G. D. (2003). Smoking and ill health: Does lay epidemiology explain the failure of smoking cessation programs among deprived populations?. *American Journal of Public Health*, *93*(2), 266-270.

Ligibel, J. A., Alfano, C. M., Courneya, K. S., Demark-Wahnefried, W., Burger, R. A., Chlebowski, R. T., ... & Jones, L. W. (2014). American Society of Clinical Oncology position statement on obesity and cancer. *Journal of clinical oncology*, *32*(31), 3568-3574.

Link, B. G., & Phelan, J. (1995). Social conditions as fundamental causes of disease. *Journal of health and social behavior*, 80-94.

Sahakyan, K. R., Somers, V. K., Rodriguez-Escudero, J. P., Hodge, D. O., Carter, R. E., Sochor, O., ... & Lopez-Jimenez, F. (2015). Normal-weight central obesity: implications for total and cardiovascular mortality. *Annals of internal medicine*, *163*(11), 827-835.

Link, B. G., & Phelan, J. C. (1996). Understanding sociodemographic differences in health--the role of fundamental social causes. *American journal of public health*, *86*(4), 471-473.

Link, B. G., Phelan, J. C., Miech, R., & Westin, E. L. (2008). The resources that matter: fundamental social causes of health disparities and the challenge of intelligence. *Journal of Health and Social Behavior*, *49*(1), 72-91.

Lleras-Muney, A. (2005). The relationship between education and adult mortality in the United States. *The Review of Economic Studies*, *72*(1), 189-221.

Masters, R. K., Hummer, R. A., & Powers, D. A. (2012). Educational differences in US adult mortality a cohort perspective. *American Sociological Review*, *77*(4), 548-572.

Masters, R. K., Link, B. G., & Phelan, J. C. (2015). Trends in education gradients of 'preventable' mortality: a test of fundamental cause theory. *Social Science & Medicine*, *127*, 19-28.

Masters, R. K., Powers, D. A., & Link, B. G. (2013). Obesity and US mortality risk over the adult life course. *American journal of epidemiology*, *177*(5), 431-442.

Masters, R. K., Reither, E. N., Powers, D. A., Yang, Y. C., Burger, A. E., & Link, B. G. (2013). The impact of obesity on US mortality levels: the importance of age and cohort factors in population estimates. *American journal of public health*, *103*(10), 1895-1901.

Matthews, K. A., Kelsey, S. F., Meilahn, E. N., Muller, L. H., & WING, R. R. (1989). Educational attainment and behavioral and biologic risk factors for coronary heart disease in middle-aged women. *American Journal of Epidemiology*, *129*(6), 1132-1144.

McAdams, M. A., Dam, R. M., & Hu, F. B. (2007). Comparison of Self-reported and Measured BMI as Correlates of Disease Markers in US Adults. *Obesity*, *15*(1), 188-188.

McGee, D. L., & Diverse Populations Collaboration. (2005). Body mass index and mortality: a meta-analysis based on person-level data from twenty-six observational studies. *Annals of epidemiology*, *15*(2), 87-97.

Mehta, N. K., & Chang, V. W. (2009). Mortality attributable to obesity among middle-aged adults in the United States. *Demography*, *46*(4), 851-872.

Mehta, N. K., & Chang, V. W. (2011). Secular declines in the association between obesity and mortality in the United States. *Population and development review*, *37*(3), 435-451.

Miech, R., Pampel, F., Kim, J., & Rogers, R. G. (2011). The enduring association between education and mortality the role of widening and narrowing disparities. *American Sociological Review*, *76*(6), 913-934.

Mirowsky, J., & Ross, C. E. (1998). Education, personal control, lifestyle and health A human capital hypothesis. *Research on Aging*, *20*(4), 415-449.

Mirowsky, J., & Ross, C. E. (2003). *Social causes of psychological distress*. Transaction Publishers.

Mokdad, A. H., Ford, E. S., Bowman, B. A., Dietz, W. H., Vinicor, F., Bales, V. S., & Marks, J. S. (2003). Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *Jama*, *289*(1), 76-79.

Montez, J. K., & Zajacova, A. (2013). Trends in mortality risk by education level and cause of death among US White women from 1986 to 2006.*American journal of public health*, *103*(3), 473-479.

Montez, J. K., Hummer, R. A., Hayward, M. D., Woo, H., & Rogers, R. G. (2011). Trends in the educational gradient of US adult mortality from 1986 through 2006 by race, gender, and age group. *Research on Aging*, *33*(2), 145-171.

Murray, C. J., Kulkarni, S. C., Michaud, C., Tomijima, N., Bulzacchelli, M. T., Iandiorio, T. J., & Ezzati, M. (2006). Eight Americas: investigating mortality disparities across races, counties, and race-counties in the United States.*PLoS Med*, *3*(9), e260.

Myrskylä, M., & Chang, V. W. (2009). Initial BMI, Weight Change, and Mortality among Middle-and Older-Aged Adults. *Epidemiology (Cambridge, Mass.)*, *20*(6), 840.

Naderali, E. K., Ratcliffe, S. H., & Dale, M. C. (2009). Review: obesity and Alzheimer's disease: a link between body weight and cognitive function in old age. *American journal of Alzheimer's disease and other dementias*, *24*(6), 445-449.

NIH & North American Association for the Study of Obesity. (2000). The Practical Guide Identification, Evaluation, and Treatment of Overweight and Obesity in Adults.". *NIH Publication Number DO-4084*, 35-38.

Merrill, R. M., & Richardson, J. S. (2009). Validity of self-reported height, weight, and body mass index: findings from the National Health and Nutrition Examination Survey, 2001–2006. *Prev Chronic Dis*, *6*(4), A121.

Craig, B. M., & Adams, A. K. (2009). Accuracy of body mass index categories based on self-reported height and weight among women in the United States. *Maternal and child health journal*, *13*(4), 489-496.

Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2014). Prevalence of childhood and adult obesity in the United States, 2011-2012. *Jama*, *311*(8), 806-814.

Okosun, I. S., Choi, S., Matamoros, T., & Dever, G. A. (2001). Obesity is associated with reduced self-rated general health status: evidence from a representative sample of white, black, and Hispanic Americans. *Preventive medicine*, *32*(5), 429-436.

Olshansky, S. J., Passaro, D. J., Hershow, R. C., Layden, J., Carnes, B. A., Brody, J., ... & Ludwig, D. S. (2005). A potential decline in life expectancy in the United States in the 21st century. *New England Journal of Medicine*,*352*(11), 1138-1145.

Oreopoulos, A., Kalantar-Zadeh, K., Sharma, A. M., & Fonarow, G. C. (2009). The obesity paradox in the elderly: potential mechanisms and clinical implications. *Clinics in geriatric medicine*, *25*(4), 643-659.

Pampel, F. C., & Rogers, R. G. (2004). Socioeconomic status, smoking, and health: a test of competing theories of cumulative advantage. *Journal of Health and Social Behavior*, *45*(3), 306-321.

Pampel, F. C., Krueger, P. M., & Denney, J. T. (2010). Socioeconomic disparities in health behaviors. *Annual review of sociology*, *36*, 349.

Park, M. H., Falconer, C., Viner, R. M., & Kinra, S. (2012). The impact of childhood obesity on morbidity and mortality in adulthood: a systematic review. *Obesity Reviews*, *13*(11), 985-1000.

Phelan, J. C., Link, B. G., & Tehranifar, P. (2010). Social conditions as fundamental causes of health inequalities theory, evidence, and policy implications. *Journal of health and social behavior*, *51*(1 suppl), S28-S40.

Phelan, J. C., Link, B. G., Diez-Roux, A., Kawachi, I., & Levin, B. (2004). "Fundamental causes" of social inequalities in mortality: a test of the theory. *Journal of health and social behavior*, *45*(3), 265-285.

Poulain, M., Doucet, M., Major, G. C., Drapeau, V., Sériès, F., Boulet, L. P., ... & Maltais, F. (2006). The effect of obesity on chronic respiratory diseases: pathophysiology and therapeutic strategies. *Canadian Medical Association Journal*, *174*(9), 1293-1299.

Prentice, A. M., & Jebb, S. A. (2001). Beyond body mass index. *Obesity reviews*, *2*(3), 141-147.

Preston, S. H., & Stokes, A. (2014). Obesity paradox: conditioning on disease enhances biases in estimating the mortality risks of obesity. *Epidemiology (Cambridge, Mass.)*, *25*(3), 454.

Price, G. M., Uauy, R., Breeze, E., Bulpitt, C. J., & Fletcher, A. E. (2006). Weight, shape, and mortality risk in older persons: elevated waist-hip ratio, not high body mass index, is associated with a greater risk of death. *The American journal of clinical nutrition*, *84*(2), 449-460.

Reeves, G. K., Pirie, K., Beral, V., Green, J., Spencer, E., & Bull, D. (2007). Cancer incidence and mortality in relation to body mass index in the Million Women Study: cohort study. *Bmj*, *335*(7630), 1134.

Reilly, J. J., & Kelly, J. (2011). Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. *International journal of obesity*, *35*(7), 891-898.

Reither, E. N., Hauser, R. M., & Yang, Y. (2009). Do birth cohorts matter? Age-period-cohort analyses of the obesity epidemic in the United States. *Social science & medicine*, *69*(10), 1439-1448.

Reither, E. N., Olshansky, S. J., & Yang, Y. (2011). New forecasting methodology indicates more disease and earlier mortality ahead for today's younger Americans. *Health Affairs*, 10-1377.

Renehan, A. G., Tyson, M., Egger, M., Heller, R. F., & Zwahlen, M. (2008). Body-mass index and incidence of cancer: a systematic review and meta-analysis of prospective observational studies. *The Lancet*, *371*(9612), 569-578.

Reuser, M., Bonneux, L. G., & Willekens, F. J. (2009). Smoking kills, obesity disables: a multistate approach of the US Health and Retirement Survey. *Obesity*, *17*(4), 783-789.

Reuser, M., Bonneux, L., & Willekens, F. (2008). The burden of mortality of obesity at middle and old age is small. A life table analysis of the US Health and Retirement Survey. *European journal of epidemiology*, *23*(9), 601-607.

Robinson, W. R., Furberg, H., & Banack, H. R. (2014). Selection bias: a missing factor in the obesity paradox debate. *Obesity*, *22*(3), 625-625.

Alberti, K. G. M. M., Eckel, R. H., Grundy, S. M., Zimmet, P. Z., Cleeman, J. I., Donato, K. A., ... & Smith, S. C. (2009). Harmonizing the metabolic syndrome a joint interim statement of the international diabetes federation task force on epidemiology and prevention; national heart, lung, and blood institute; American heart association; world heart federation; international atherosclerosis society; and international association for the study of obesity. *Circulation*, *120*(16), 1640-1645.

Després, J. P., & Lemieux, I. (2006). Abdominal obesity and metabolic syndrome. *Nature*, *444*(7121), 881-887.

Alberti, K. G. M. M., Zimmet, P., & Shaw, J. (2006). Metabolic syndrome—a new world-wide definition. A consensus statement from the international diabetes federation. *Diabetic medicine*, *23*(5), 469-480.

Rogers, R. G., Hummer, R. A., & Everett, B. G. (2013). Educational differentials in US adult mortality: An examination of mediating factors. *Social science research*, *42*(2), 465-481.

Romero-Corral, A., Montori, V. M., Somers, V. K., Korinek, J., Thomas, R. J., Allison, T. G., ... & Lopez-Jimenez, F. (2006). Association of bodyweight with total mortality and with cardiovascular events in coronary artery disease: a systematic review of cohort studies. *The Lancet*, *368*(9536), 666-678.

Ross, C. E., & Wu, C. L. (1995). The links between education and health. *American sociological review*, 719-745.

Ross, C. E., Masters, R. K., & Hummer, R. A. (2012). Education and the gender gaps in health and mortality. *Demography*, *49*(4), 1157-1183.

Schnohr, C., Højbjerre, L., Riegels, M., Ledet, L., Larsen, T., Schultz-Larsen, K., ... & Grønbæk, M. (2004). Does educational level influence the effects of smoking, alcohol, physical activity, and obesity on mortality? A prospective population study. *Scandinavian Journal of Public Health*, *32*(4), 250-256.

Seidell, J. C., Verschuren, W. M., van Leer, E. M., & Kromhout, D. (1996). Overweight, underweight, and mortality: a prospective study of 48287 men and women. *Archives of Internal Medicine*, *156*(9), 958-963.

Sørensen, T. I., Rissanen, A., Korkeila, M., & Kaprio, J. (2005). Intention to lose weight, weight changes, and 18-y mortality in overweight individuals without co-morbidities. *PLoS Med*, *2*(6), e171.

Stevens, J., Cai, J., Pamuk, E. R., Williamson, D. F., Thun, M. J., & Wood, J. L. (1998). The effect of age on the association between body-mass index and mortality. *New England Journal of Medicine*, *338*(1), 1-7.

Zheng, H., & Dirlam, J. (2016). The Body Mass Index-Mortality Link across the Life Course: Two Selection Biases and Their Effects. *PloS one*, *11*(2), e0148178.

Stommel, M., & Schoenborn, C. A. (2009). Accuracy and usefulness of BMI measures based on self-reported weight and height: findings from the NHANES & NHIS 2001-2006. *BMC public health*, *9*(1), 421.

Tomiyama, A. J., Hunger, J. M., Nguyen-Cuu, J., & Wells, C. (2016). Misclassification of cardiometabolic health when using body mass index categories in NHANES 2005–2012. *International Journal of Obesity*.

Uretsky, S., Messerli, F. H., Bangalore, S., Champion, A., Cooper-DeHoff, R. M., Zhou, Q., & Pepine, C. J. (2007). Obesity paradox in patients with hypertension and coronary artery disease. *The American journal of medicine*, *120*(10), 863-870.

Visscher, T. L. S., Seidell, J. C., Menotti, A., Blackburn, H., Nissinen, A., Feskens, E. J. M., ... & Seven Countries Study Research Group. (2000). Underweight and Overweight in Relation to Mortality Among Men Aged 40–59 and 50–69 Years The Seven Countries Study. *American journal of epidemiology*, *151*(7), 660-666.

NCHS Data Linkage. (2016). What's New. Retrieved from http://www.cdc.gov/nchs/data-linkage/new-linkage.htm

Wang, Y., & Beydoun, M. A. (2007). The obesity epidemic in the United States—gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. *Epidemiologic reviews*, *29*(1), 6-28.

Williamson, D. F., Pamuk, E., Thun, M., Flanders, D., Byers, T., & Heath, C. (1999). Prospective study of intentional weight loss and mortality in overweight white men aged 40–64 years. *American journal of epidemiology*, *149*(6), 491-503.

World Health Organization. (2011). Preventing chronic diseases: a vital investment. 2005.

Yang, R., Cheung, M. C., Pedroso, F. E., Byrne, M. M., Koniaris, L. G., & Zimmers, T. A. (2011). Obesity and weight loss at presentation of lung cancer are associated with opposite effects on survival. *Journal of Surgical Research*, *170*(1), e75-e83.

Yu, Y. (2012). Reexamining the declining effect of age on mortality differentials associated with excess body mass: evidence of cohort distortions in the United States. *American journal of public health*, *102*(5), 915-922.

Zajacova, A. (2006). Education, gender, and mortality: Does schooling have the same effect on mortality for men and women in the US?. *Social Science & Medicine*, *63*(8), 2176-2190.

Zajacova, A., & Ailshire, J. (2014). Body Mass Trajectories and Mortality Among Older Adults: A Joint Growth Mixture–Discrete-Time Survival Analysis. *The Gerontologist*, gns164.

Zajacova, A., & Hummer, R. A. (2009). Gender differences in education effects on all-cause mortality for white and black adults in the United States. *Social Science & Medicine*, *69*(4), 529-537.

Zheng, H., & Yang, Y. (2012). Population heterogeneity in the impact of body weight on mortality. *Social science & medicine*, *75*(6), 990-996.

Zheng, H., Tumin, D., & Qian, Z. (2013). Obesity and mortality risk: new findings from body mass index trajectories. *American journal of epidemiology*, *178*(11), 1591-1599.

Heo, M., Faith, M. S., & Pietrobelli, A. (2002). Resistance to change of adulthood body mass index. *International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity*, *26*(10), 1404-1405.

Hopman, W. M., Leroux, C., Berger, C., Joseph, L., Barr, S. I., Prior, J. C., ... & Goltzman, D. (2007). Changes in body mass index in Canadians over a five-year period: results of a prospective, population-based study. *BMC Public Health*, *7*(1), 1.

Masters, R. K. (2013). *The Protective Effect of High Body mass on mortality risk. Obesity paradox or data artifact*. IBS working paper. Available from: http://www. colorado. edu/ibs/pubs/pop/pop2013-0007. pdf.