Obesity Over the Life Course: A Study of How Obesity Produces Health Disadvantage and Excess Mortality in the United States

by

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Abstract

This dissertation explores the influence of obesity on U.S. population morbidity and mortality. Across three essays, I examine the relation of obesity to work disability, activity impairment, and mortality. Chapter 1 looks at how obesity in early adulthood affects work disability at young and middle ages. Using data from the National Longitudinal Study of Youth 1979, I employ logistic regression to assess whether an early onset of obesity affects the likelihood of developing a work disabling condition and use event history analysis to predict the time at which that work disability occurs. Results indicate that early obesity increases the likelihood that a person will develop a work disability and uniformly increases the relative hazard of the disability occurring. The association of obesity and work disability remains robust to the inclusion of covariates and modeling the process that selects a person to become obese. Chapter 2 shifts to looking at how mid-life obesity may alter the effect of occupational exposures on later life limitations in activities of daily living. Using data from the Health and Retirement Study and latent growth curve models, I find that the effects of past occupational exposures differ by whether or not a respondent is obese around retirement. For non-obese respondents, physically demanding occupations are associated with a lower risk of developing health impairments, but no such association is found for obese persons. Instead, although not statistically significant, physically demanding occupations are associated with a greater rate of accumulation in health limitations for obese persons. Chapter 3 moves to examine why it is that older obese adults appear more likely to get sick but less likely to die. Using data from the Health and Retirement Survey and a multi-state modelling framework, I find that the obesity-mortality paradox may be explained by obese adults having a higher risk of becoming ill, losing weight, and subsequently dying having a history of obesity. Together, these three chapters suggest important influences of obesity on the health and well-being of adults throughout the life course and highlight the importance of studying the obesity-health relationship in

a longitudinal framework.

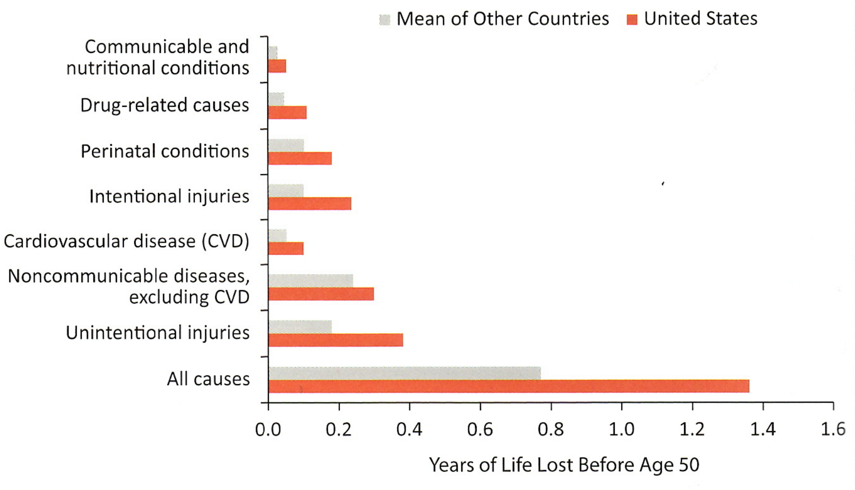
0.1 Introduction

Health has a profound effect on the subjective well-being of individuals and populations (Gakidou et al., 2000). Prolonging the period of good health, reducing the duration of chronic illness, and increasing certainty of healthy survivorship to old age are all markers of societal development (Gakidou et al., 2000; Riley and Riley, 1986). People are disadvantaged in societies where there is a high degree of variability, and consequently, uncertainty, surrounding the period of good health and survivorship (Wilmoth and Horiuchi, 1999). Individuals who are unsure about their demographic trajectories are unable to effectively plan for the future and may consequently have lower life time utility (Zureick, 2010). A person who overestimates their healthy lifespan may end life with excess resources while underestimates of longevity may leave one resource deprived at the oldest ages (Hamermesh, 1982; Bloom et al., 2007).

While much of demographic theory would suggest that the duration of life has and will continue to increase (Omran, 1971; Olshansky and Ault, 1986), notable inequalities in health and healthy longevity remain (Marmot, 2004; Salomon et al., 2013). Although innovations may improve health, new factors emerge which perpetuate health disadvantage for minority and economically disadvantaged populations (Link and Phelan, 1995; Olshansky et al., 2012). This dissertation explores how one such emerging trend, the rise in obesity, may act in conjunction with other sociodemographic and economic factors to produce health inequalities in the United States.

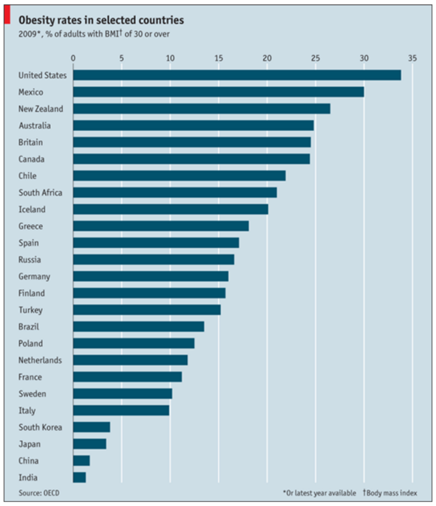
Despite the highest health expenditures of any country , the United States is ranked last on key indicators of population health relative to other high income countries (Woolf et al., 2013). The health disadvantage in the U.S. holds up to age 75 for all sexes and across social classes(Woolf et al., 2013). Disparities in U.S. life expectancy have been growing over the past thee decades and the U.S. has the greatest number of life years lost prior to age 50 (Woolf et al., 2013) compared to any other developed nation. Figure 0.1 shows that no single cause may be responsible for all U.S. health disparities.

Figure 0.1 Person Years Lost for Men in The United States (Woolf et al., 2013)



However, obesity and obesity induced chronic health conditions are a contributing factor to flagging U.S. health and reduced economic productivity (Crimmins et al., 2011) . As shown in Figure 0.2, the United States has one of the highest rates of obesity worldwide.

Figure 0.2 Obesity Rates Across Countries Compared (Economist, 2010)



Obesity has a demonstrable effect on population morbidity by increasing risk for developing hypertension, type 2 diabetes, kidney failure, osteoarthritis and mobility limitations (Must et al., 1999). The elevated disease risk occurs among young people as well as middle age and older adults. The prevalence of disability among young and middle-aged Americans have been rising with obesity related health conditions thought to be at least partially responsible for this recent uptick (Alley and Chang, 2007). Obesity also exacerbates health inequality as the prevalence of obesity is higher among those of low socioeconomic status and those belonging to a racial minority (Wang and Beydoun, 2007).

The health consequences of obesity have demonstrable financial consequences as well. Obesity related health conditions are estimated to increase annual U.S. medical expenditures by 147 billion dollars (Finkelstein et al., 2009). The health consequences of obesity are also projected to reduce the life time labor force productivity of recent cohorts by 998.5 billion dollars (Barkin et al., 2010). The total direct economic costs of obesity have been estimated at 215 billion dollars in the United States (Hammond and Levine, 2010), and projections suggest these costs will continue to rise substantially (Wang et al., 2011). In summary, the rising prevalence of obesity in the United States has detrimental health consequences for the population, economic consequences, and the potential to exacerbate inequalities in health.

Because of the widespread financial and health implications of obesity for the United States, many studies have sought to confirm the relationship between a person's obesity status and their risk of developing health impairments. Yet, little research has examined how the effects of obesity may unfold over a person's life course. Life course perspectives emphasize that the production of health is determined by changing genetic, biological, behavioral, social, and economic contexts, and a person's health may adapt to changing contexts. These exposures have a cumulative impact on health with the timing and sequence of events having individual and population influences. Adapting life course theories to the study of obesity, this study can enrich existing literature and answer questions such as:

how early exposure to obesity may affect the timing of work disability, if effects of obesity on later life health differ by past exposures and stressors, and how changes in weight may affect well-being and longevity.

This dissertation presents three empirical analyses which chronicle the effects of obesity from young adulthood through middle age and into retirement. Chapter 1 presents a survival analysis examining how the early onset of obesity may affect subsequent risk of developing a work disability. Chapter 2 uses longitudinal growth curve models to assess whether the effect of past occupational exposures differ by a person's obesity status around retirement age. Chapter 3 explores the relation of obesity to disease onset, weight loss and mortality risk in order to explain a paradox found in cross-sectional analyses: why it is that obesity appears to elevate the risk of developing chronic conditions but reduce mortality risk. Together, these three analyses promote a nuanced understanding of the implications of obesity for health over the life course.

1 DOES OBESITY CHANGE THE ONSET OF DISABILITY?

Abstract

A past body of work has established that obesity increases an individual's likelihood of becoming work disabled, unable to be formally employed in the labor market due to poor health. However, fewer studies have considered how obesity may affect the likelihood and wait time to the onset of a disabling condition in young and middle aged Americans by adjusting for factors that may select an individual to become obese and influence subsequent disability risk. Using data from the National Longitudinal Study of Youth 1979, this study employs propensity score adjusted logistic regression and event history analysis to predict the onset of a work disability between ages 30 and 50 controlling for whether an individual became obese before age 30. Findings suggest that obesity increases the likelihood that an individual will develop a work disability. The effect of obesity on work disability holds even after controlling for factors that may select an individual to become obese and the influence of confounders. Obesity also increases the relative hazard of developing a work disabling condition. Obesity had the greatest impact on the work disability risk for African American men, 85% of non-obese African Americans were expected to remain without a disability compared to 75 % of the obese. Overall, this study suggests that the likelihood a person becomes obese does not affect the obesity-disability association; obesity is an important factor influencing if and when a person is work disabled.

1.1 Introduction

The prevalence of obesity has dramatically risen in the United States, and obesity has become an increasingly common mechanism that may select people into forced unemployment due to health. Over a third of adults above age 20 are now obese (Flegal et al., 2010; Burkhauser and Cawley, 2004) and being overweight or obese has been shown to substantially increase an individual's risk of receiving a disability pension as a consequence of exiting the labor market due to poor health (Armour et al., 2013).

The first instance when a person becomes unemployed due to poor health marks a crucial time in the life course with enduring consequences for their wellbeing. A person may become unable to work because of cumulative health stressors ,and the unemployment spell may in turn trigger future health problems, stress, economic insecurity, and increased disease risk (Martikainen and Valkonen). The detrimental outcomes following a spell of unemployment may reflect a person's previous disease risks (Pollitt et al., 2005) but can also lead to harmful behavioral and biological responses. The negative effects of unemployment for health have been found consistently (Bambra and Eikemo, 2009; Cook et al., 1982; Bartley, 1994); the unemployed have: a 63% higher mortality risk, report greater levels of stress, and are more likely to be in fair or poor health (Roelfs et al., 2011).

Because of the increasing prevalence of obesity in the United States, the association of obesity to work disability, and the harmful effects of work disability for subsequent health and employment outcomes, it is extremely important to estimate how obesity affects the likelihood of an individual developing a work disability and the time at which disability occurs. Despite prior research suggesting that obesity has adverse consequences for health and that the obese are more likely to develop work disabilities (Armour et al., 2013), less work has explicitly examined whether in fact obesity is actually increasing risk for disability or if the factors that select an individual to become obese also are influencing their likelihood to develop a work-disability.

This paper explores whether and how an early onset of obesity, before age 30, may affect both the likelihood that an individual will develop a work disability and the time at which the disability occurs. It consists of four sections. Section one review studies evaluating the relation of obesity to disability and summarize which populations are at greatest risk for developing a work disabling condition related to obesity. Section two reviews NLSY data available to assess the obesity disability relationship. Section three describes the logistic regression and event history methods used and highlights some key findings. Sections four concludes by relating these findings to existing literature and summarizing obesity's effect on work disability.

1.2 Background

Relation of Obesity to Disability

An extensive literature has established that obesity increases the likelihood that an individual will develop a disability that limits labor force participation (Tunceli et al., 2006; Burkhauser and Cawley, 2004; Cawley, 2004) Recent estimates from the National Health and Interview Survey suggest that 41.7% of obese adults have a disability that limits their daily activities while only 26.7% of normal weight adults report a disability (Armour et al., 2013). Comparing overweight and obese adults to their normal weight counterparts, a recent meta-analysis puts the relative risk of collecting a disability pension at 1.1 for overweight persons and 1.5 for the obese (Robroek et al., 2013). In addition to establishing these associations, several causal mechanisms between obesity and work-disability have been established. Obesity increases pressure on joints leading to a greater risk of back pain, osteo-arthritis, and impaired daily functioning (Himes, 2000; Schulte et al., 2007). Moreover, the obese are more likely to suffer from sleep apnea and resulting fatigue makes them more likely to experience a work place accident (Schulte et al., 2007). . The obese are also more likely to suffer from sleep apnea, and the resulting fatigue makes them more likely to experience a workplace accident (Schulte et al., 2007). Finally, the obese may be more likely to have chronic conditions, particularly diabetes, a disease shown to increase work absenteeism (Howard and Potter, 2014). However, while associated diseases and injuries are clear mechanisms to suggest that obese persons will be more likely to develop health impairments and work disabilities, selection into obesity is non-random.

Individuals who become obese are also much more likely to be disadvantaged on many other social and demographic factors. Fundamental cause theory suggests that disadvantages in social position have enduring negative effects for health although the mechanisms linking position to health may change over time (Link and Phelan, 1995). By this theory, obesity may not be an independent predictor of disability but a mechanism through which social disadvantages like race and class may affect health. Many of the factors associated with obesity, including socioeconomic position, race, and genetic predisposition, are correlated with unfavorable economic outcomes, including work disability. In the mid-1980s, approximately the time when obesity is measured in this study, adults possessing less than a high school education and those living with a poverty income ratio below 350% were more likely to be obese (Ogden et al., 2010). [1]. Not only does low socioeconomic position increase obesity risk but it has also been linked to a greater risk for developing work disability, and more generally, poor health (Williams, 1990). A consistent gradient between socioeconomic position and health outcomes suggests that socioeconomic status is a significant moderating circumstance that may be related to the onset of obesity and subsequent morbidity. Similar relations are found between race, obesity and impairment. Perhaps related in part to their socioeconomic disadvantages, African American and Hispanic persons are more likely to be obese and face impairments (Hayward et al., 2014; Flegal et al., 2010). An important takeaway from this literature is that while there are reasons to suggest that obesity is associated with work disability, its effects may be overstated if the race and socioeconomic processes selecting an individual to become obese are not

---

[1] The relationship of ses to obesity has weakened in recent years. There are some disagreements about the cutpoint at which the poverty income ratio is a significant predictors of obesity and whether these associations are uniform for adults compared to adolescents. See (Skelton et al., 2009) (Ogden et al., 2010)

appropriately modeled.

A key limitation of this study is that it is unable to adjust for the genetic factors that may lead an individual to develop obesity and a disability but due to limited data and limited existing knowledge about the genetic components that explain obesity. However by controlling for more proximate measures of disadvantage including race and education, this study hopes to account for the major factors that may select an individual to become obese and affect the timing of disability. Although prior work has found that obese persons are more likely to face work disability, the social and demographic factors highlighted above may select an individual to become obese and develop a work-disability, few studies have examined the association of obesity to work disability accounting for the factors that may predispose an individual to become obese. More broadly, this study generates estimates of the effect of obesity in young adulthood for work disability risk at young and middle ages.

Objectives

The prevalence of obesity has dramatically increased and obesity has been linked to a greater risk of developing a work disability (Flegal et al., 2010; Burkhauser and Cawley, 2004), but few studies have explicitly controlled for the selection processes that may predispose an individual to become obese and develop a subsequent workplace disability. Given these limitations of the current literature, this study has three aims

1. To ascertain whether an early onset of obesity increases an individual's risk of developing a work disability adjusting for selection factors.
2. To examine if obesity changes the timing of a workplace disability.
3. To assess if the association of obesity to work place disability differs by a person's propensity to become obese.

To achieve these aims, this study first estimates an individual's propensity of becoming obese, then employs propensity score weighted logistic regression and event history analysis to determine whether obesity increases an individual's risk of becoming work disabled and the time at which the disability is expected to occur.

1.3 Data

This study uses data from the 1986-2010 waves of the National Longitudinal Survey of Youth 1979 (NLSY 1979). The NLSY-1979 is a nationally representative sample of adolescents who were between the ages of 14 and 22 in 1979. Adolescents sampled in the NLSY-1979 were surveyed annually from 1979 to 1992 and biennially from 1994 to 2010. NLSY 1979 respondents have been surveyed on their education, labor force participation and health across surveys. In this analysis, the sample and survey years used are truncated. Respondents are maintained in this analysis if they were male identified, between the ages of 24 and 29 at the 1985 survey round, and if they participated in the NLSY surveys between 1984 and 2000. Data is restricted to the male sub sample because men are more likely to consistently be in the labor force and cannot experience pregnancy, a condition which can affect obesity and work abilities for women. Ages of respondents are restricted to those between the ages of 24 and 29 at the 1985 survey round to limit age related heterogeneity. Finally, only respondents with complete data for these survey rounds are kept because no imputation is used in this analysis.

Measures of Disability and Obesity

The two key outcomes of this study are whether an individual develops a disability that limits labor force participation and the expected timing of that disability In the NLSY, a work disabling health condition is ascertained by a two question sequence, a filter question determining whether an individual is currently employed in the formal labor market, and a second question asked if a respondent is unemployed whether the respondent's health prevents them from working. When examining whether obesity increases an individual's risk for becoming work disabled differs by their obesity status, work disability is measured as a binary outcome, did the respondent develop a work disability between 1986 and 2010, when respondents were, roughly, between the ages of 30 and 55. In the event history analysis, the outcome of interest is the first survey year when the work disability was reported. [2]

Obesity is the key independent variable in this analysis and is here approximated by body mass index. Measures of height and weight are taken from 1985 NLSY survey round. The measure of obesity is taken at this survey round, when respondents were between the ages of 25 and 29 for a few key reasons. First, this is the last measure of height obtained in the NLSY and is taken at ages when respondents have attained their adult height. Second, measuring obesity at this young adult age means that obesity status is ascertained close to the time when these respondents began their occupational careers, thus obesity is likely not influenced by occupational environments.

According to the Center for Disease Control standards, individuals are classified as obese if they had a body mass index above 30 at the 1985 survey round. [3]

Covariates:

To avoid confounding effects of obesity with other factors, this analysis will examine a number of factors that may select a person to become obese. Among these are demographic factors, a respondent's race (black/white), ethnicity (hispanic/non-hispanic) and year of birth. Education, whether or not a person completed high school by the 1984 survey round is also controlled for as less educated individuals are thought to have a greater risk of becoming obese. An interesting contribution of this study is that it measures a correlate of educational attainment, cognitive ability. Cognitive ability is measured as the average score on the reading and arithmetic sections of the ASVAB administered to all respondents at the 1980 survey round. In addition to controlling for these factors which positively select

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[2] Between 1986 and 1993, surveys were conducted annually but between 1994 and 2010 the surveys were conducted biennially. To account for this difference in the time scale, reporting of disability is done on the biennial time scale; however, if a person first developed a work disability in the intermediate years, that person is considered to have a work disability at the next survey round. For example, if a person first develops a disability in the 1987 survey round, they are considered to have acquired the disability in 1988.

[3] A limitation in the measure of obesity is used is that it is based on respondent self-report. Thus measures of height and weight may be noisy, and for some respondents biased. Given the likely measurement error, in supplementary analyses I replicate the logistic regression analyses changing the cut point for obesity. Table 1.20 shows the likelihood of developing a work disability differentiating respondents by whether they are overweight (bmi $\geqslant 25$) and Table 1.19 shows the likelihood of developing a work disability differentiating respondents by whether they are severely obese (bmi $\geqslant 35$). These results are consistent with prior work showing a health gradient by weight with severe obesity accompanying higher risk.

an individual to become obese and work disabled, this study also controls for respondent smoking and drug use which may be negatively associated with obesity but potentially related to the likelihood an individual will develop a work disability. Measures of smoking and drug use are whether a person reported using any drugs in the prior year at the 1984 survey round and whether they had smoked in the 30 days prior to the survey round in 1984. [4]

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[4] Although smoking status might be better operationalized as a time varying covariate, there are insufficient measures in the NLSY to do so. This is a limitation.

1.4 Analytic Strategy

To achieve the study aims of assessing whether an early onset of obesity affects the risk and timing of a work disabling condition, the analytic strategy contains four components: a logistic regression to generate a person's propensity of being obese, a propensity score weighted logistic regression to determine whether obesity predicts a respondent's likelihood of developing a work disability, a propensity score stratified logistic regression analysis to determine if the effect of obesity differs by a person's propensity to become obese, and an event history analysis to determine whether and how obesity affects the timing of disability. In using propensity score stratification for both the logistic regression and event history analysis, I am able to estimate the degree to which a person's likelihood of becoming obese influences the association of obesity with subsequent work disability.

Modeling Selection into Obesity:

Logistic regression is used to estimate an individual's propensity of being obese by the 1985 survey round. Covariates in the selection model include a respondent's race, education, average ASVAB scores, smoking history, poverty history, and drug use. The propensity score is estimated as the predicted probability that a respondent will be obese conditional on these covariates and is estimated using logistic regression. [5]

Estimating Effect of Obesity on Likelihood of Work Disability Adjusting for Selection:

To assess the relation between obesity and the likelihood a person becomes work disabled, logistic regression is used. Four models are fit to estimate the average association between early obesity and whether or not a person develops a work disability. First, I assess the relation of obesity to disability with no covariates. Next, I assess the relation of obesity to work disability adjusting for covariates including respondent's race, education, asvab

---

[5] Estimation of probabilities is taken by for each observation as $p=\frac{exp(\alpha+\sum_{i=1}^{N} B_i X_i)}{1+exp(\alpha+\sum_{i=1}^{N} B_i X_i)}$

scores, smoking history, poverty history, and drug use. I then fit a model to measure obesity's link to work disability weighting by the propensity score. Finally, I measure the effect of obesity on work disability adjusting for the same covariates but again applying the propensity score weighting. Propensity score weights are derived by weighting treated cases to have a probability weight of shown in Algorithm 1. . Given the debate about the most appropriate estimator for estimating the effect of a treatment (in this case, early obesity) on an outcome (here, work disability), in sensitivity analyses I use nearest neighbor propensity score matching within a .05 caliper and nearest neighbor matching with three matched neighbors to estimate the effect of obesity on work disability.

Algorithm 1 Propensity Score Weighting

```
$1/\hat{p}\, where\, X = 1$ (Treated)
$1/(1-\hat{p})\, where\, X = 0$ (Controls)
Calculated from $\ln\left(\frac{\hat{p}}{1-\hat{p}}\right) = \beta_0 + \beta_1 X$
```

Testing for Heterogeneous Effect of Obesity on Likelihood of Work Disability: In addition to estimating the average effect of obesity for work disability, I also look at the effects of obesity stratifying on the propensity to be obese. Three propensity score stratum are constructed based on the predicted likelihood a person will become obese using the prediction equation described above. Stratum are constructed to have an equal number of obese respondents, treated cases, in each propensity score stratum. Thus, the first propensity score stratum represents the effect of obesity on disability likelihood for obese persons who are unlikely to become obese (in the bottom tertile of predicted probabilities). The second propensity score stratum represents the effect for individuals with about an average likelihood of becoming obese (in the second tertile of predicted probabilities). The third propensity score stratum represents the effect of obesity on disability for persons identified as having a high risk of becoming obese (in the top tertile of predicted probabilities). The goal in doing this subsequent analysis is to determine if there are differential effects of obesity on disability based on the relative likelihood that an individual will become obese.

Event History Analysis for Predicting Wait Time to Work Disability:

When assessing whether and how obesity affects the timing of work disabiity, event history analysis is used. First, non-parametric Kaplan Meier survival curves are generated for the obese and non-obese subgroups. After using the Kaplan Meier curves to estimate the unadjusted survival differences across the obese and non-obese, I move to adjusting for potential confounding factors using a Cox Proportional Hazard model. The Cox model specifics a proportional hazard with an unspecified baseline hazard, $h_0(t)$, as shown in 2

Algorithm 2 Cox Proportional Hazard Model

```
$h_i(t) = h_0(t) * exp\{\beta_1 x_{i1} + \beta_2 x_{i2} ...\}$
```

Covariates included in the model are again a respondent's race, education, asvab scores, smoking history, poverty history, and drug use. Model fit is assessed by checking the proportional hazard assumption testing for any time varying effects using the Schonfeld residuals (doing both graphical comparison and Chi Square Tests) and Cox-Snell Residuals. and as indicated from these tests, moving to a model with time varying effects and/or a fully parametric model. As shown in the results, the preferred parametric model has a baseline hazard with a gamma distribution. Finally, this analysis looks at whether the hazard of developing a work disability differs across person's propensity to become obese. This is done by again applying propensity score weighting to the Cox Hazard model and running propensity score stratified models.

Respondents exit the analysis when they first miss a survey round or develop a disability. Missing cases are censored because attrition and disability may be correlated and this study is exclusively interested in the wait time to the first occurrence of disability. A concern in conducting this analysis is that individuals may experience a disability prior to the first survey round; however, as the NLSY surveys individuals close to the start of their working career, this bias is likely limited. However, individuals who do report a disability prior to the 1986 survey round are excluded from this analysis. Characteristics of respondents dropped from the analysis because of a pre-existing disability are summarized in Table 1.3.

1.5 Results

Descriptive Statistics:

Descriptive statistics that show the distribution of relevant covariates by a obesity status are shown in Table 1.1 and statistics showing the distribution of covariates by whether a person went on to develop a work disability are shown in Table 1.2. These descriptives show that the obese are disproportionally likely to develop a work disability (31% vs. 20%) and face a host of other disadvantages. The obese sample is more likely to be black or hispanic, economically disadvantaged, having lived in a household with an income below the federal poverty line or failing to achieve a high school equivalence, and lower skilled, performing worse on the administerd ASVAB test. An area where the obese are advantaged compared to the non-obese sample is in smoking and drug use, perhaps related to the physiological effects of drug use and smoking.

Estimating Propensity to become Obese:

As shown in Table 1.4. Race/ethnicity, socioeconomic status, smoking/drug use, and cognitive skills are used to predict obesity at the 1985 survey round. Only a few of these factors are statistically significant predictors of obesity, whether the respondent had smoked in the 30 days prior to the 1984 survey and the respondent's standardized score on the ASVAB reading and math sections. A prior history of smoking decreased the likelihood a respondent would be obese at the 1985 survey round (OR .65) and respondents with higher scores on the ASVAB test had a lower likelihood of being obese. (OR .788). Overall, these selection factors only explain a small proportion of the variance in obesity status, the pseudo R squared is .0193.

Effects of Obesity on the Likelihood of Developing a Work Disability:

Each observation is assigned a propensity to become obese based on predicted probabilities from the logistic regression described in the previous section. As described in the Analytic Strategy, obesity's association with the likelihood a respondent will develop a work disability is estimated with and without relevant controls and with and without propensity score weights. These estimates are shown in Table 1.5. A key finding in this estimation is that the effect of early obesity on the likelihood an individual will develop a work disability does not seem substantially altered by the inclusion of the covariates nor the propensity score weighting.

In sensitivity analyses which compare the effects of obesity on the probability a person becomes work disabled, the consistency of obesity's effect is confirmed. The percent change in the average likelihood a person will become disabled by their obesity status is consistent across the propensity score weighted, propensity score matched and unweighted logistic regression results. These results are shown in Table 1.15.

A subsequent analysis looks at the effect of obesity on the the likelihood a person will develop a work disability across propensity score strata shown in Table 1.6. In this analysis, I find no consistent trend in the effect of obesity on work disability across propensity score strata. In a model, with three propensity score strata, the association of obesity with the likelihood a person will develop a work disability appears greatest in the first propensity score stratum, with the lowest predicted probability of obesity, (OR 2.668) approaches 1 for the second stratum (OR 1.004), and increases for the third propensity score stratum, with the highest predicted probability of obesity, (OR 1.802). However, in subsequent analyses where the number of strata are changed from 3 strata to 2 strata, associations are quite different. Now for the first stratum, where individuals have a relatively lower predicted likelihood of becoming obese, the association of obesity to work disability is not statistically significant (OR 1.553), and in the second stratum, where individuals have a relatively higher predicted likelihood of becoming obese, the association of obesity to work disability is statistically significant (OR 2.161) and are shown in Table 1.7. In other words, there appears to be no clear-cut association between the likelihood a person becomes obese and the effect being obese has on their likelihood of work disability. [6] Given the sensitivity of findings to

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[6] I also did an analysis with 4 propensity score strata. Again, no clear or consistent pattern. These results are shown in Table 1.8

the number of propensity score strata, results do not suggest a clear or consistent pattern in the effects of obesity across the propensity score strata. [7]

Overall findings across logistic regression and matching analyses suggests that obesity has an association with work disability and this estimated association remains robust to the inclusion of covariates and by controlling for factors which might influence selection into obesity and subsequent work disability.

Effects of Obesity on the Timing of a Work Disability:

Having established that obesity does have an effect on the likelihood an individual will develop a work disability and that this effect cannot be explained by selection processes, this analysis next looks at how obesity affects the time at which work disability occurs. While it is clear that obesity affects the likelihood of developing a disabling condition, it is less certain if this endures over the life course. Preliminary evidence suggesting an enduring effect of obesity is presented in the Kaplan Meier Survival Curve shown in Figure 1.1. The obese subgroup has a lower probability of remaining in the sample without a disability compared to their non-obese counterparts. Similar results are shown in the Cox proportional hazard model. Initially, two Cox models are run: the first shows the effect of obesity without covariates, and the second model estimates the influence of obesity adjusting for potentially confounding factors. As with the logistic regression results, obesity increases the relative hazard of a work disability occurring and the results are robust to the inclusion of covariates. The hazard ratio in a model without covariates was 1.772 and with covariates the hazard ratio was 1.751. Schoenfeld residuals from the Cox model also suggested that smoking history is better modelled including a main effect of smoking and an interaction of time. Incorporating the time varying effect of smoking, the association of obesity to work disability timing remained robust and statistically significant with a

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[7] Balance checks for the three strata model reveal that the sample is well matched within stratum as evidenced by the standardized differences across treated (respondents with obesity) and control (non-obese respondents). A few slight deviations from the preferred 10% difference in standardized differences should be noted. In stratum 1 and stratum 2, there is greater standardized difference for white respondents and on birth year. Both of these differences are less than 20%These differences can be seen in 1.9.

hazard ratio of 1.750. Results from all three Cox models are shown in Table 1.10. Chi square tests from the final model fit suggest no violation of the proportionality assumption and are shown in Table 1.13. Survival curves illustrating differences in the proportion of the sample expected to remain without a work disability by obesity status are shown in Figure 1.2. Cox-Snell residuals were also checked, see Figure 1.3, and revealed good fit at early time periods but some deviation near the point of right truncation. [8]

Findings from propensity score weighted models were very similar to the unweighted estimates seen above as shown in Table 1.11. The hazard ratio of obesity on work disability is 1.8 (HR in unweighted model was 1.751). The Cox Snell residuals also showed similar fit to the unweighted model as shown in Figure 1.4. As was the case in the logistic regression models, the propensity score stratified Cox models do not reveal a consistent trend in the association of obesity on work disability by propensity score strata. Stratified results for a three strata model are shown in Table 1.12 [9]

A finding across models is that the relative effect of obesity appears to be robust to the inclusion of covariates,and in sensitivity analyses, I find no evidence of an interactive effect across other covariates included in the model. Although the relative effect of obesity is the same across subclassification analyses, the absolute effect of obesity on work disability may differ by other covariates in the model. Given prior research suggesting that disability risk differs by race, and socioeconomic position associated with race, Table 1.14 and Figure 1.5 shows the Cox estimated probability of surviving to each survey round without a work disability for average black, hispanic and white respondents. [10] It demonstrates that the

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[8] This deviation looks pretty significant,but seems driven in part by a small number of events and high censorship at later survey rounds. I did check for better model fit in a parametric model (and even trying piecewise models). Of these parametric models, the AIC suggested that the gamma model was the best fit, but the parameters that should be significant for a gamma distribution were not statistically significant. All of these results can be seen in my sensitivity results. Table 1.16 shows the comparison of AIC across parametric models. Table 1.17 illustrates coefficients from the best fitting gamma model. Figure **??** shows the Cox Snell Residuals from the best fitting gamma model. Finally, Table 1.18 shows the predicted probabilities of remaining without a work disability. Results from this prediction are quite similar to the Cox model.

[9] I do not describe these in greater detail because results were not consistent and I think models were underpowered. When going to publish this chapter, I would likely drop the stratified models.

[10] In the Cox model, it appears that the risk of disability is lower for black and hispanic respondents as the hazard ratio is below 1. However, this would only be the case if black and hispanic respondents had

average black respondent has a lower likelihood of remaining without a work disability and early life obesity appears most detrimental for the average black respondent, reducing their probability of surviving to the the thirteenth survey round by 10 points (84% for non-obese blacks and 74 % for obese blacks). Hispanic and white respondents have similar risks of developing a work disability by the thirteenth survey round. By the thirteenth survey round, 91% of non-obese white and hispanic respondents are expected to remain without a work disability while 85% of obese white and hispanic respondents are expected to remain without a work disability.

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comparable traits to whites; they do not. Instead, hispanic and black persons are systematically disadvantaged on other covariates and thus the average black person has a greater risk of work disability and the risk for hispanics is about comparable to whites.

1.6 Discussion and Limitations

Overall, this study finds that early obesity is a significant predictor of whether and when an individual will develop a work disability. Results show that the effects of obesity are robust even when incorporating possible factors that select an individual to become obese such as race, cognitive ability, and socioeconomic status. There is also little evidence of effect heterogeneity. When stratifying by a person's propensity to become obese, results remain relatively robust. That the relative effect of obesity on work disability seems to remain constant across propensity score groups, race, cognitive ability, and education would suggest that the relative influence of obesity is constant across race and socioeconomic subgroups. However, because African American respondents in the sample are disadvantaged on other characteristics that increase disability risk, obesity has a greater impact on their likelihood of remaining disability free.

When making these claims, a few limitations should be acknowledged. First, to limit heterogeneity, this paper restricted analysis to a few birth cohorts. Over time, the degree to which obesity is selected by the factors studied may have drastically changed; however, this analysis is beyond the scope of the paper. Next, all models predicting work disability are run for the male members of the cohort. The association of obesity to work disability may differ for women, but they were not included in the analysis because they have less consistent work histories and because pregnancy, which can affect obesity, work disability, and subsequent employment is difficult to measure given the structure of this data. Finally, unobserved factors, especially genetics, may influence the likelihood a person becomes unable to work and becomes obese. However, genetic data is not available for this cohort, and the mechanisms through which genetics may affect obesity and health are somewhat undetermined.

In spite of these limitations, this study establishes that major factors often believed to be associated with obesity and disability, do not seem to modify obesity's effect on the likelihood or time when a person develops a work disability and establishes that an early onset of obesity has serious implications for the subsequent work abilities of members of this cohort study.

Future work can expand on this research in a few key ways. First, it can examine the risk of work-disability for women modifying these models to account for work exits due to pregnancy. Next, future work should examine the intermediate exposures such as changes in weight and occupational activities that may mediate the effects of obesity for employment outcomes. Finally, estimates generated here can be used to estimate the economic consequences of early onset of obesity at young and middle ages.

1.7 Tables

	Mean	**SD**	**Count**	**Min**	**Max**
Non-Obese					
Took Any Illegal Substance in Year Prior to 1984 Survey	0.332	0.471	2187	0	1
Smoked Cigarettes in 30 Days Prior to 1984 Survey	0.442	0.497	2244	0	1
Hispanic Ethnicity	0.156	0.363	2244	0	1
Race-Black	0.262	0.440	2244	0	1
Race-White	0.582	0.493	2244	0	1
Less than High School Education at 1984 Survey	0.182	0.386	2190	0	1
Household Income Below Poverty Line at 1979 Survey	0.228	0.420	2153	0	1
Averaged Reading and Math ASVAB Score 1980 Survey	0.103	1.056	2007	-3	2
Year of Birth	1959.488	1.225	2244	1957	1961
Ever Developed a Work Disability Between 1986 and 2010	0.201	0.401	1036	0	1
Obese					
Took Any Illegal Substance in Year Prior to 1984 Survey	0.291	0.455	206	0	1
Smoked Cigarettes in 30 Days Prior to 1984 Survey	0.364	0.482	209	0	1
Hispanic Ethnicity	0.215	0.412	209	0	1
Race-Black	0.258	0.439	209	0	1
Race-White	0.526	0.501	209	0	1
Less than High School Education at 1984 Survey	0.223	0.417	206	0	1
Household Income Below Poverty Line at 1979 Survey	0.246	0.432	199	0	1
Averaged Reading and Math ASVAB Score 1980 Survey	-0.127	1.045	188	-2	2
Year of Birth	1959.512	1.225	209	1957	1961
Ever Developed a Work Disability Between 1986 and 2010	0.313	0.466	115	0	1
Total					
Took Any Illegal Substance in Year Prior to 1984 Survey	0.328	0.470	2393	0	1
Smoked Cigarettes in 30 Days Prior to 1984 Survey	0.435	0.496	2453	0	1
Hispanic Ethnicity	0.161	0.368	2453	0	1
Race-Black	0.261	0.439	2453	0	1
Race-White	0.578	0.494	2453	0	1
Less than High School Education at 1984 Survey	0.186	0.389	2396	0	1
Household Income Below Poverty Line at 1979 Survey	0.230	0.421	2352	0	1
Averaged Reading and Math ASVAB Score 1980 Survey	0.083	1.057	2195	-3	2
Year of Birth	1959.490	1.225	2453	1957	1961
Ever Developed a Work Disability Between 1986 and 2010	0.212	0.409	1151	0	1
Observations	2453				

Table 1.1: Descriptive Statistics by Obesity Status

	Mean	SD	Count	Min	Max
Never Developed Work Disability					
Took Any Illegal Substance in Year Prior to 1984 Survey	0.313	0.464	906	0	1
Smoked Cigarettes in 30 Days Prior to 1984 Survey	0.396	0.489	908	0	1
Hispanic Ethnicity	0.143	0.350	908	0	1
Race-Black	0.275	0.447	908	0	1
Race-White	0.581	0.494	908	0	1
Less than High School Education at 1984 Survey	0.120	0.325	908	0	1
Household Income Below Poverty Line at 1979 Survey	0.146	0.354	867	0	1
Averaged Reading and Math ASVAB Score 1980 Survey	0.254	1.011	848	-3	2
Year of Birth	1959.487	1.241	908	1957	1961
BMI Above 30 at 1985 Survey	0.087	0.282	907	0	1
Developed a Work Disability					
Took Any Illegal Substance in Year Prior to 1984 Survey	0.359	0.481	245	0	1
Smoked Cigarettes in 30 Days Prior to 1984 Survey	0.604	0.490	245	0	1
Hispanic Ethnicity	0.171	0.378	245	0	1
Race-Black	0.392	0.489	245	0	1
Race-White	0.437	0.497	245	0	1
Less than High School Education at 1984 Survey	0.314	0.465	245	0	1
Household Income Below Poverty Line at 1979 Survey	0.294	0.456	235	0	1
Averaged Reading and Math ASVAB Score 1980 Survey	-0.542	0.966	229	-3	2
Year of Birth	1959.588	1.240	245	1957	1961
BMI Above 30 at 1985 Survey	0.148	0.355	244	0	1
Total					
Took Any Illegal Substance in Year Prior to 1984 Survey	0.323	0.468	1151	0	1
Smoked Cigarettes in 30 Days Prior to 1984 Survey	0.441	0.497	1153	0	1
Hispanic Ethnicity	0.149	0.356	1153	0	1
Race-Black	0.300	0.458	1153	0	1
Race-White	0.551	0.498	1153	0	1
Less than High School Education at 1984 Survey	0.161	0.368	1153	0	1
Household Income Below Poverty Line at 1979 Survey	0.178	0.383	1102	0	1
Averaged Reading and Math ASVAB Score 1980 Survey	0.084	1.052	1077	-3	2
Year of Birth	1959.508	1.241	1153	1957	1961
BMI Above 30 at 1985 Survey	0.100	0.300	1151	0	1
Observations	1153				

Table 1.2: Descriptive Statistics by Work Disability Status

	Mean	SD	Count	Min	Max
In Analytic Sample					
Took Any Illegal Substance in Year Prior to 1984 Survey	0.336	0.472	3030	0	1
Smoked Cigarettes in 30 Days Prior to 1984 Survey	0.438	0.496	3210	0	1
Hispanic Ethnicity	0.139	0.346	3210	0	1
Race-Black	0.240	0.427	3210	0	1
Race-White	0.622	0.485	3210	0	1
Less than High School Education at 1984 Survey	0.163	0.369	3041	0	1
Household Income Below Poverty Line at 1979 Survey	0.201	0.401	3074	0	1
Averaged Reading and Math ASVAB Score 1980 Survey	0.118	1.024	2735	-3	2
Year of Birth	1959.381	1.220	3210	1957	1961
Ever Developed a Work Disability Between 1986 and 2010	0.212	0.409	1153	0	1
BMI Above 30 at 1985 Survey	0.085	0.279	2453	0	1
Dropped from Sample Due to Pre-1986 Disability or Attrition					
Took Any Illegal Substance in Year Prior to 1984 Survey	0.257	0.439	113	0	1
Smoked Cigarettes in 30 Days Prior to 1984 Survey	0.552	0.499	116	0	1
Hispanic Ethnicity	0.147	0.355	116	0	1
Race-Black	0.310	0.465	116	0	1
Race-White	0.543	0.500	116	0	1
Less than High School Education at 1984 Survey	0.327	0.471	113	0	1
Household Income Below Poverty Line at 1979 Survey	0.321	0.469	109	0	1
Averaged Reading and Math ASVAB Score 1980 Survey	-0.411	1.085	102	-3	2
Year of Birth	1959.509	1.146	116	1957	1961
Ever Developed a Work Disability Between 1986 and 2010	0.000	0.000	33	0	0
BMI Above 30 at 1985 Survey	0.186	0.391	97	0	1
Total					
Took Any Illegal Substance in Year Prior to 1984 Survey	0.333	0.471	3143	0	1
Smoked Cigarettes in 30 Days Prior to 1984 Survey	0.442	0.497	3326	0	1
Hispanic Ethnicity	0.139	0.346	3326	0	1
Race-Black	0.242	0.428	3326	0	1
Race-White	0.619	0.486	3326	0	1
Less than High School Education at 1984 Survey	0.169	0.375	3154	0	1
Household Income Below Poverty Line at 1979 Survey	0.205	0.404	3183	0	1
Averaged Reading and Math ASVAB Score 1980 Survey	0.099	1.030	2837	-3	2
Year of Birth	1959.386	1.217	3326	1957	1961
Ever Developed a Work Disability Between 1986 and 2010	0.207	0.405	1186	0	1
BMI Above 30 at 1985 Survey	0.089	0.285	2550	0	1
Observations	3326				

Table 1.3: Descriptive Statistics for Excluded Versus Kept Cases

	Predictors of Obesity at 1985 Survey Round
Took Any Illegal Substance in Year Prior to 1984 Survey	0.852
	(-0.87)
Smoked Cigarettes in 30 Days Prior to 1984 Survey	0.650*
	(-2.48)
Hispanic Ethnicity	1.486
	(1.84)
Race-Black	0.920
	(-0.38)
Less than High School Education at 1984 Survey	0.978
	(-0.10)
Household Income Below Poverty Line at 1979 Survey	0.917
	(-0.45)
Averaged Reading and Math ASVAB Score 1980 Survey	0.788*
	(-2.40)
1958.Year of Birth	0.838
	(-0.45)
1959.Year of Birth	0.784
	(-0.62)
1960.Year of Birth	0.880
	(-0.33)
1961.Year of Birth	0.706
	(-0.89)
Observations	2060

Exponentiated coefficients; *t* statistics in parentheses

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.4: Logistic Regression Predicting Obesity at the 1985 Survey Round

	(1) No Covariates	(2) Covariates	(3) No Covariates PS Weighted	(4) Covariates PS Weighted
BMI Above 30 at 1985	1.814**	1.891*	1.758*	1.822*
Survey	(2.76)	(2.58)	(2.34)	(2.26)
Took Any Illegal		1.597*		1.450
Substance in Year Prior to 1984 Survey		(2.50)		(1.01)
Smoked Cigarettes in 30		1.762**		1.801
Days Prior to 1984 Survey		(3.21)		(1.77)
Hispanic Ethnicity		0.783		0.911
		(-0.98)		(-0.22)
Race-Black		0.655		0.685
		(-1.92)		(-0.92)
Less than High School Education		1.372		1.129
at 1984 Survey		(1.48)		(0.24)
Household Income Below		1.645*		1.235
Poverty Line at 1979 Survey		(2.43)		(0.51)
Averaged Reading and Math		0.445***		0.444***
ASVAB Score 1980 Survey		(-7.15)		(-3.89)
1958.Year of Birth		1.019		1.610
		(0.04)		(0.67)
1959.Year of Birth		0.942		1.716
		(-0.13)		(0.78)
1960.Year of Birth		1.377		2.048
		(0.72)		(1.05)
1961.Year of Birth		1.146		1.669
		(0.31)		(0.73)
Observations	1151	1026	1026	1026

Exponentiated coefficients; *t* statistics in parentheses
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.5: Logistic Regression Predicting Work Disability by the 2010 Survey Round

	(1) Stratum 1	(2) Stratum 2	(3) Stratum 3
BMI Above 30 at 1985 Survey	2.668*	1.004	1.802
	(2.37)	(0.01)	(1.36)
Took Any Illegal Substance in Year Prior to 1984 Survey	1.756*	0.704	1.840
	(2.00)	(-0.82)	(1.15)
Smoked Cigarettes in 30 Days Prior to 1984 Survey	1.798	0.703	1.044
	(1.41)	(-0.56)	(0.07)
o. Hispanic Ethnicity	1	3.725	1.735
	(.)	(1.76)	(1.06)
Race-Black	0.557	0.808	0.674
	(-1.66)	(-0.54)	(-0.75)
Less than High School Education at 1984 Survey	2.230*	1.118	0.820
	(2.19)	(0.30)	(-0.47)
Household Income Below Poverty Line at 1979 Survey	3.183***	0.807	1.112
	(3.39)	(-0.57)	(0.25)
Averaged Reading and Math ASVAB Score 1980 Survey	0.550**	0.261***	0.177***
	(-2.70)	(-3.51)	(-4.78)
1958.Year of Birth	1.900	1.394	0.313
	(0.57)	(0.43)	(-1.49)
1959.Year of Birth	1.623	0.606	0.560
	(0.43)	(-0.62)	(-0.73)
1960.Year of Birth	1.852	2.118	0.846
	(0.55)	(1.00)	(-0.23)
1961.Year of Birth	1.783	0.833	0.475
	(0.52)	(-0.22)	(-0.91)
Observations	438	360	217

Exponentiated coefficients; *t* statistics in parentheses
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.6: Logistic Regression Predicting Work Disability by the 2010 Survey Round Stratified by Propensity Score 3 Strata Model

	(1) Strata 1	(2) Strata 2
Ever Developed a Work Disability Between 1986 and 2010 Surveys		
BMI Above 30 at 1985 Survey	1.553	2.161*
	(1.21)	(2.15)
Took Any Illegal Substance in Year Prior to 1984 Survey	1.574*	1.502
	(2.03)	(1.04)
Smoked Cigarettes in 30 Days Prior to 1984 Survey	2.365**	1.373
	(2.98)	(0.79)
Hispanic Ethnicity	0.439	1.197
	(-1.48)	(0.48)
Race-Black	0.634	0.805
	(-1.60)	(-0.57)
Less than High School Education at 1984 Survey	1.625	1.001
	(1.72)	(0.00)
Household Income Below Poverty Line at 1979 Survey	1.907*	1.226
	(2.39)	(0.61)
Averaged Reading and Math ASVAB Score 1980 Survey	0.542***	0.308***
	(-3.79)	(-4.80)
1957b.Year of Birth	1	1
	(.)	(.)
1958.Year of Birth	1.891	0.583
	(0.79)	(-0.91)
1959.Year of Birth	1.396	0.783
	(0.41)	(-0.40)
1960.Year of Birth	2.211	1.031
	(0.99)	(0.05)
1961.Year of Birth	1.767	1.009
	(0.71)	(0.02)
Observations	688	338

Exponentiated coefficients; *t* statistics in parentheses

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.7: Logistic Regression Predicting Work Disability by the 2010 Survey Round Stratified by Propensity Score 2 Strata Model

	(1) Strata 1	(2) Strata 2	(3) Stata 3	(4) Strata 4
BMI Above 30 at 1985 Survey	2.209	1.159	4.308**	1.299
	(1.59)	(0.26)	(2.74)	(0.50)
Took Any Illegal Substance in Year Prior to 1984 Survey	1.790	1.636	1.977	2.112
	(1.65)	(1.25)	(1.02)	(1.07)
Smoked Cigarettes in 30 Days Prior to 1984 Survey	2.081	2.903	5.076	0.463
	(1.16)	(1.52)	(1.33)	(-1.01)
o. Hispanic Ethnicity	1	0.519	0.352	5.140*
	(.)	(-0.83)	(-0.89)	(2.05)
Race-Black	0.615	0.747	1.200	0.692
	(-1.15)	(-0.70)	(0.35)	(-0.49)
Less than High School Education at 1984 Survey	1.922	1.451	1.574	0.525
	(1.41)	(1.01)	(0.88)	(-1.22)
Household Income Below Poverty Line at 1979 Survey	3.366**	1.424	2.899	0.587
	(2.96)	(0.87)	(1.84)	(-1.00)
Averaged Reading and Math ASVAB Score 1980 Survey	0.571*	0.616	0.729	0.0985***
	(-2.03)	(-1.20)	(-0.46)	(-4.24)
1957b.Year of Birth	1	1	1	1
	(.)	(.)	(.)	(.)
1958.Year of Birth	1.445	1.039	1.299	0.323
	(0.86)	(0.04)	(0.25)	(-1.19)
1959.Year of Birth	1.061	0.760	0.849	1.409
	(0.15)	(-0.29)	(-0.14)	(0.37)
1960.Year of Birth	0.593	2.135	1.849	0.782
	(-1.00)	(0.87)	(0.65)	(-0.27)
1961o.Year of Birth	1	1.356	2.854	0.427
	(.)	(0.31)	(0.83)	(-0.86)
Observations	308	366	202	136

Exponentiated coefficients; *t* statistics in parentheses
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.8: Logistic Regression Predicting Work Disability by the 2010 Survey Round Stratified by Propensity Score 4 Strata Model

	Stratum 1	**Stratum 2**	**Stratum 3**
Year of Birth	0.132	-0.193	0.093
Took Any Illegal Substance in Year Prior to 1984 Survey	0.042	-0.007	0.011
Smoked Cigarettes in 30 Days Prior to 1984 Survey	-0.089	0.086	0.058
Hispanic Ethnicity	-0.032	0.025	-0.015
Race-Black	-0.084	0.096	0.001
Race-White	0.116	-0.120	0.014
Less than High School Education at 1984 Survey	0.013	0.047	-0.078
Household Income Below Poverty Line at 1979 Survey	-0.032	0.058	-0.029
Averaged Reading and Math ASVAB Score 1980 Survey	0.078	-0.062	0.086

Table 1.9: Standardized Differences Across Propensity Score Strata

	(1) No Covariates	(2) Covariates	(3) Time Varying Effect of Smoking
BMI Above 30 at 1985 Survey	1.772**	1.751**	1.750**
	(2.84)	(2.62)	(2.62)
Took Any Illegal Substance in Year Prior to 1984 Survey		1.331	1.332
		(1.68)	(1.69)
Smoked Cigarettes in 30 Days Prior to 1984 Survey		1.810***	3.135***
		(3.59)	(3.11)
Hispanic Ethnicity		0.596*	.602*
		(-2.07)	(-2.03)
Race-Black		0.722	.729
		(-1.67)	(-1.62)
Less than High School Education at 1984 Survey		0.972	.967
		(-0.15)	(-.17)
Household Income Below Poverty Line at 1979 Survey		1.472*	1.465*
		(2.16)	2.14
Averaged Reading and Math ASVAB Score 1980 Survey		0.492***	.492 ***
		(-6.89)	(-6.88)
1958.Year of Birth		0.867	.863
		(-0.39)	(-0.40)
1959.Year of Birth		0.753	.751
		(-0.76)	(-.76))
1960.Year of Birth		0.924	.922
		(-0.21)	(-0.22)
1961.Year of Birth		0.832	.832
		(-0.50)	(-0.50)
Interaction of Time and Smoking			.928 ***
			(-1.71)
Observations	18636	16428	16428

Exponentiated coefficients; *t* statistics in parentheses
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.10: Cox Proportional Hazard Predicting Work Disability between 1986 and 2010

	(1) No Covariates	(2) Covariates	(3) Time Varying Effect of Smoking
BMI Above 30 at 1985 Survey	1.841** (2.77)	1.801* (2.52)	1.803* (2.55)
Took Any Illegal Substance in Year Prior to 1984 Survey		1.015 (0.04)	0.996 (-0.01)
Smoked Cigarettes in 30 Days Prior to 1984 Survey		2.153** (2.68)	8.188** (3.27)
Hispanic Ethnicity		0.489 (-1.62)	0.491 (-1.61)
Race-Black		0.760 (-0.77)	0.779 (-0.72)
lhs1984		0.911 (-0.20)	0.914 (-0.19)
Averaged Reading and Math ASVAB Score 1980 Survey		0.504*** (-3.33)	0.509*** (-3.29)
[1em] 1958.Year of Birth		1.062 (0.09)	1.045 (0.06)
1959.Year of Birth		1.229 (0.30)	1.217 (0.29)
1960.Year of Birth		1.060 (0.09)	1.047 (0.07)
1961.Year of Birth		1.054 (0.08)	1.056 (0.08)
Household Income Below Poverty Line at 1979 Survey		1.085 (0.23)	1.097 (0.26)
Interaction of Time and Smoking			0.841* (-2.29)
Observations	16428	16428	16428

Exponentiated coefficients; *t* statistics in parentheses
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.11: Cox Model Weighted

	(1) Strata 1	(2) Strata 2	(3) Strata 3
Took Any Illegal Substance in Year Prior to 1984 Survey	1.821* (2.33)	0.468 (-1.77)	1.488 (0.86)
BMI Above 30 at 1985 Survey	2.669** (2.92)	0.941 (-0.13)	1.557 (1.22)
Smoked Cigarettes in 30 Days Prior to 1984 Survey	1.711 (0.66)	1.293 (0.31)	2.761 (1.03)
Hispanic Ethnicity	2.14e-20 (.)	1.959 (0.89)	1.341 (0.63)
Race-Black	0.836 (-0.59)	0.748 (-0.86)	0.801 (-0.51)
lhs1984	1.435 (1.12)	0.817 (-0.62)	0.636 (-1.20)
Household Income Below Poverty Line at 1979 Survey	2.264** (2.96)	0.812 (-0.60)	1.063 (0.17)
Averaged Reading and Math ASVAB Score 1980 Survey	0.677* (-1.98)	0.263*** (-3.90)	0.266*** (-4.72)
[1em] 1958.Year of Birth	1.693 (0.51)	1.146 (0.21)	0.343 (-1.77)
1959.Year of Birth	1.830 (0.58)	0.353 (-1.43)	0.559 (-1.00)
1960.Year of Birth	1.786 (0.56)	1.163 (0.23)	0.512 (-1.17)
1961.Year of Birth	1.703 (0.51)	0.458 (-1.08)	0.578 (-0.92)
Interaction of Time and Smoking	1.055 (0.52)	0.909 (-1.26)	0.869 (-1.05)
Observations	7236	5698	3494

Exponentiated coefficients; *t* statistics in parentheses
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.12: Cox Propensity Score Stratified Model

Variable	**Rho**	**ChiSq**	**DF**	**P-Value**
Took Any Illegal Substance in Year Prior to 1984 Survey	-0.02092	0.07	1	0.7938
BMI Above 30 at 1985 Survey	0.0216	0.08	1	0.776
Smoked Cigarettes in 30 Days Prior to 1984 Survey	-0.12817	2.53	1	0.112
Hispanic Ethnicity	0.01073	0.02	1	0.8867
Race-Black	0.06896	0.82	1	0.3651
Less than High School Education at 1984 Survey	0.00261	0	1	0.9722
Household Income Below Poverty Line at 1979 Survey	-0.02892	0.15	1	0.6946
Averaged Reading and Math ASVAB Score 1980 Survey	-0.01276	0.03	1	0.8609
1958.Year of Birth	0.09768	1.64	1	0.2002
1959.Year of Birth	-0.01614	0.04	1	0.8326
1960.Year of Birth	0.00404	0	1	0.9577
1961.Year of Birth	0.04253	0.31	1	0.5771
Interaction of Time and Smoking	0.11805	2.22	1	0.136
Global Test		11.01	13	0.6098

Table 1.13: Chi Square Tests for Proportionality Final Cox Model

Survey	Black Non-Obese	Black Obese	Hispanic Non-Obese	Hispanic Obese	White Non-Obese	White Obese
1	1.00	0.99	1.00	1.00	1.00	1.00
2	0.99	0.99	1.00	0.99	1.00	0.99
3	0.99	0.97	0.99	0.99	0.99	0.99
4	0.98	0.96	0.99	0.98	0.99	0.98
5	0.97	0.95	0.98	0.97	0.98	0.97
6	0.96	0.92	0.97	0.96	0.97	0.96
7	0.94	0.89	0.96	0.94	0.96	0.94
8	0.93	0.88	0.96	0.93	0.96	0.93
9	0.92	0.86	0.95	0.92	0.95	0.92
10	0.91	0.84	0.95	0.91	0.95	0.91
11	0.89	0.82	0.94	0.89	0.94	0.89
12	0.87	0.78	0.92	0.87	0.92	0.87
13	0.84	0.74	0.91	0.85	0.91	0.85

Table 1.14: Predicted Work Disability-Free Survival by Race

1.8 Figures

Figure 1.1 Kaplan Meier Survival Curve by Obesity Status

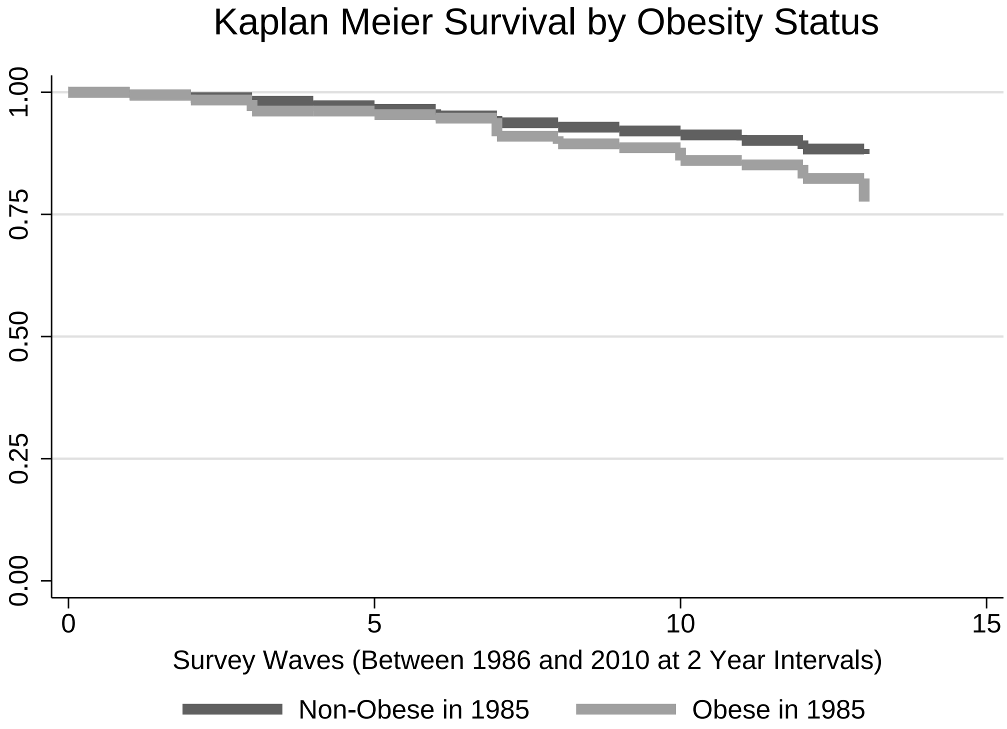


Figure 1.2 Cox Proportional Hazard Survival Curve by Obesity Status Adjusting for Covariates

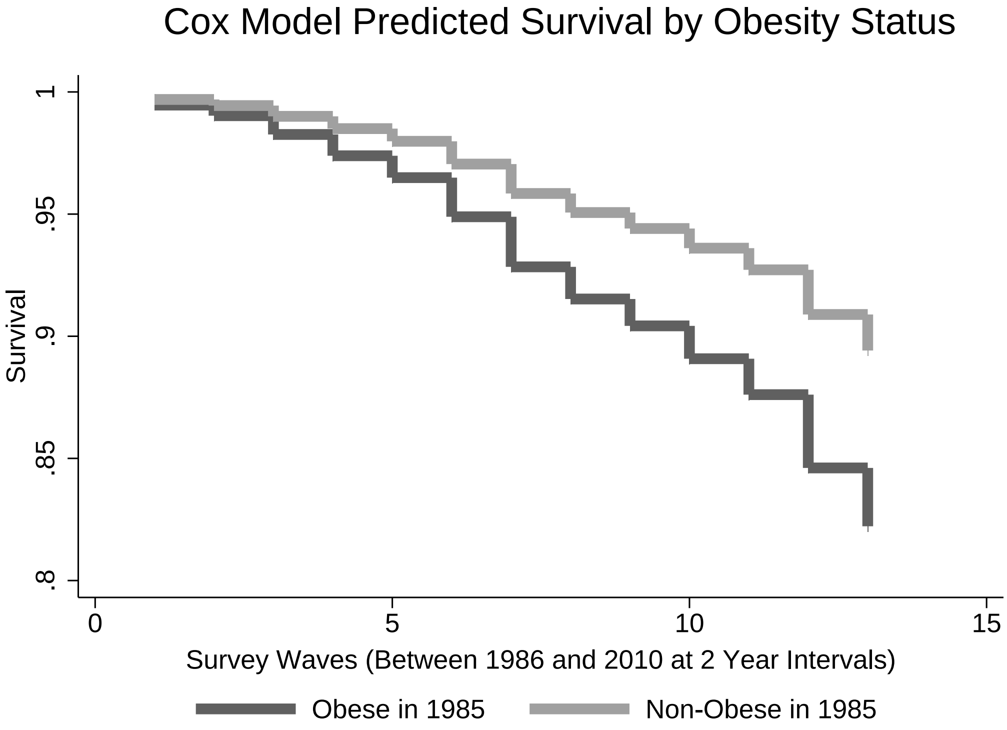


Figure 1.3 Cox Snell Residuals for Best Fitting Cox Model

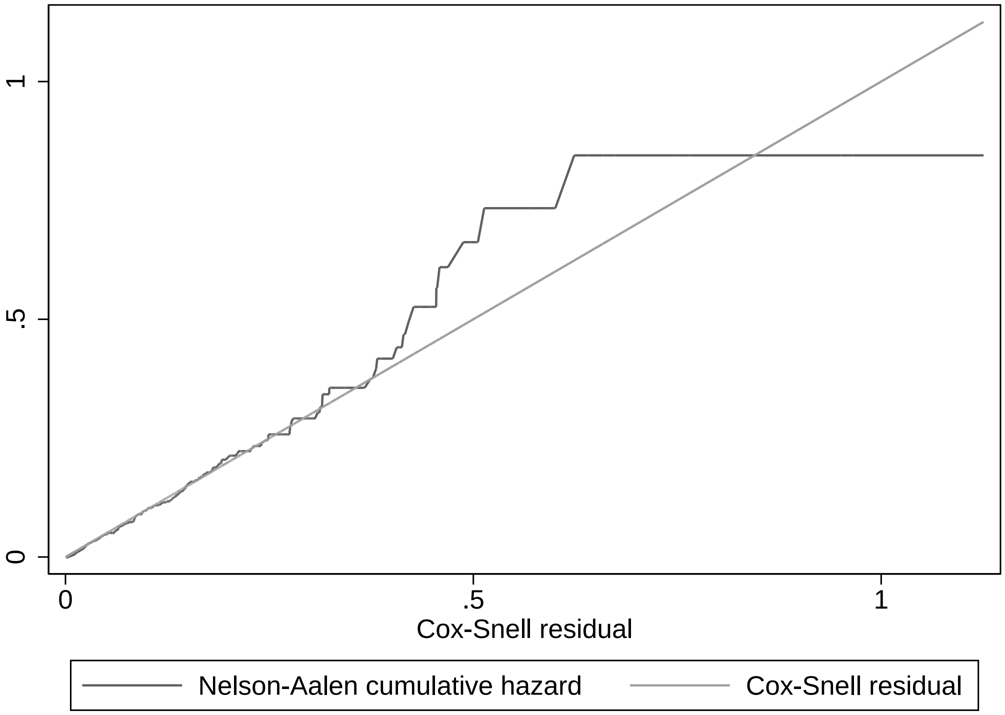


Figure 1.4 Cox Snell Residuals for Best Fitting Cox Propensity Score Weighted Model

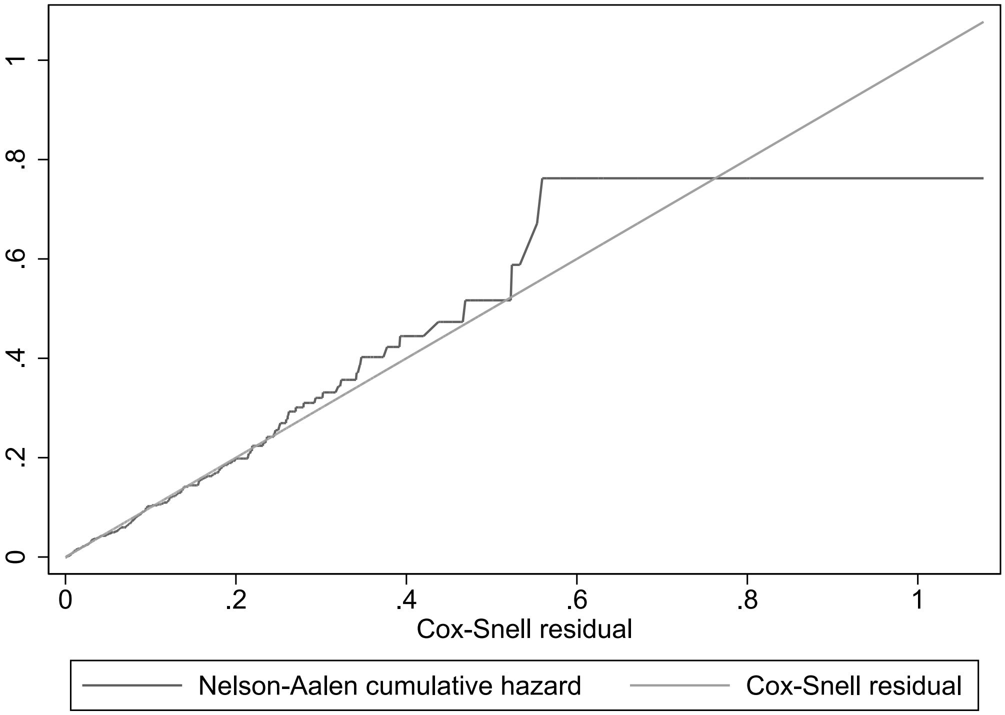
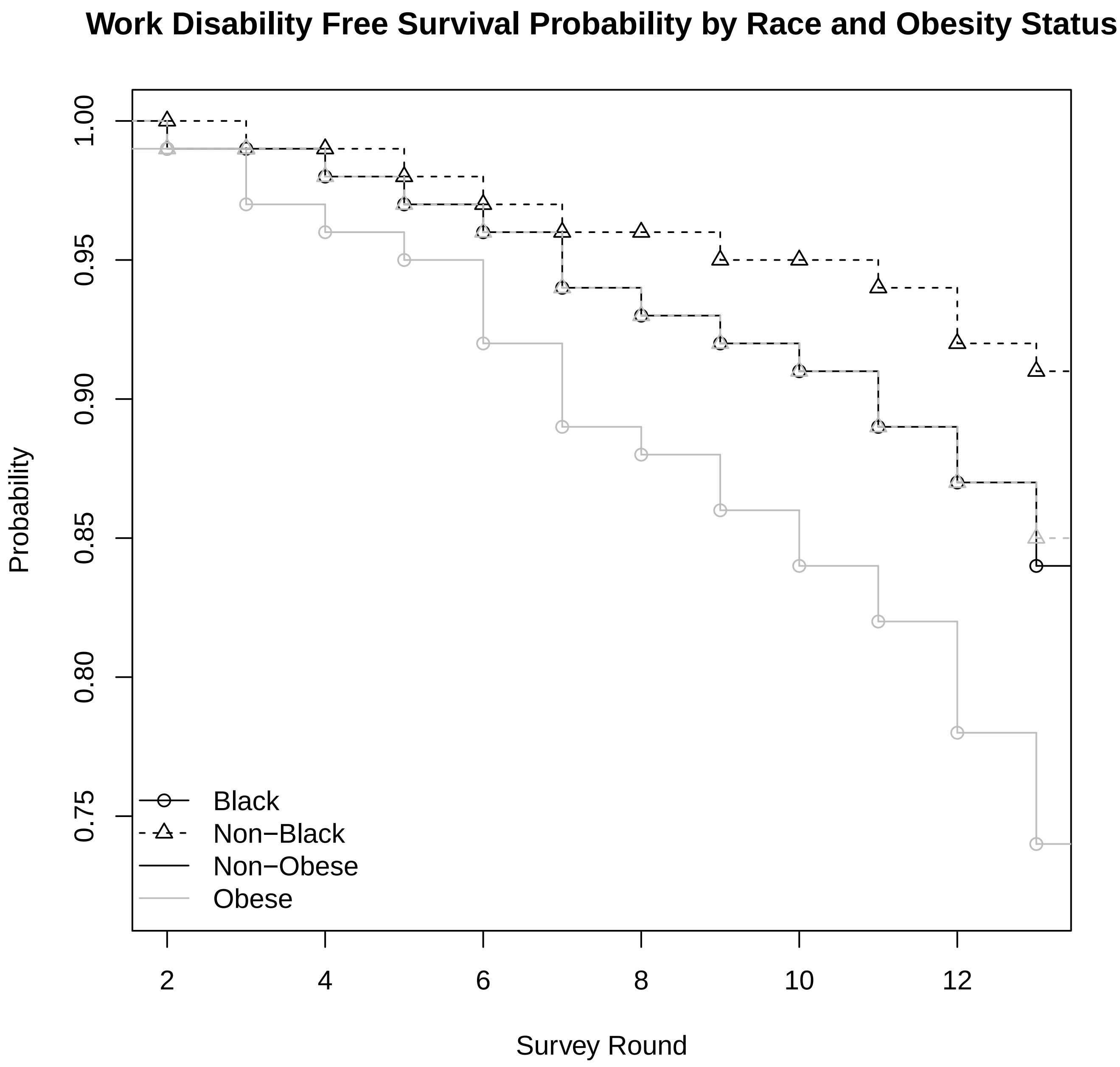


Figure 1.5 Survival Probability by Race and Obesity Status Predicted from Cox Model



1.9 Sensitivity Results

	Avg. Treatment Effect	Avg. Change in Probability of Work Disability
Nearest Neighbor Matching Matching (NN=1 Caliper=.05)	.13**	
Nearest Neighbor Matching (NN=3 Caliper=.05)	.09**	
Logistic Regression (No Covariates)		.11*
Logistic Regression (Covariates)		.11*
Logistic Regression (Covariates PS Weighted)		.10*

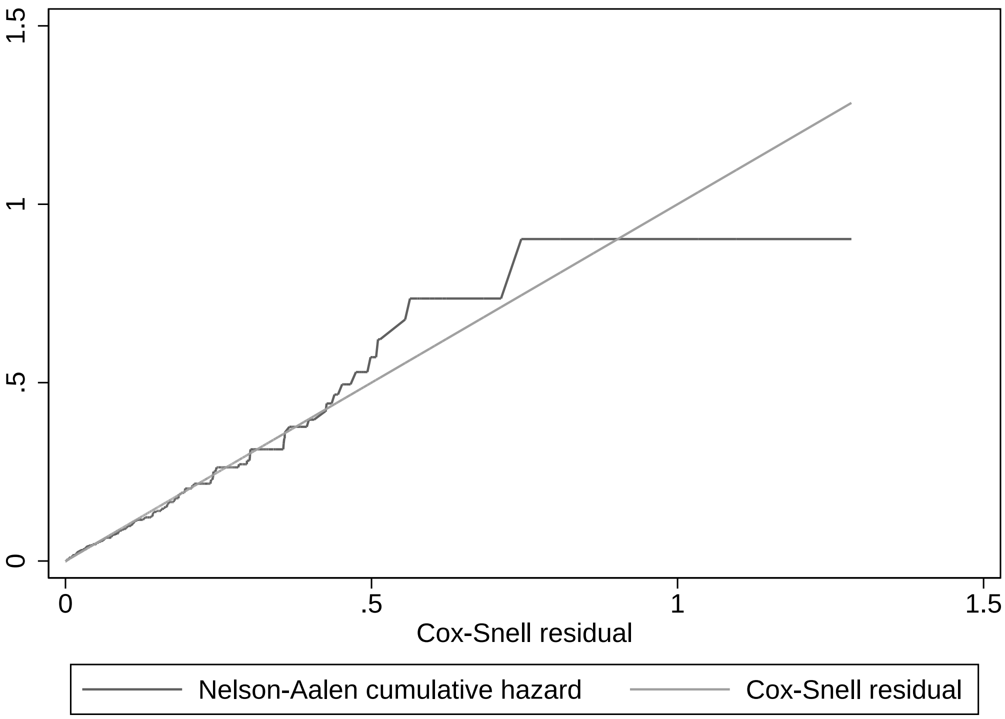
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.15: Effect of Obesity on Likelihood of Work Disability Across Estimators

	(1) AIC
Weibull	1166
Gamma	1165.396
Gompertz	1170.295
Lognormal	1178.288
Loglogistic	1168.615

Table 1.16: AIC Across Parametric Models

Figure 1.6 Cox Snell Residuals for Best Fitting Gamma Model



	(1) Gamma Model
Took Any Illegal Substance in Year Prior to 1984 Survey	-0.128
	(-1.68)
BMI Above 30 at 1985 Survey	-0.242**
	(-2.60)
Smoked Cigarettes in 30 Days Prior to 1984 Survey	-0.925***
	(-6.91)
Hispanic Ethnicity	0.256*
	(2.22)
Race-Black	0.170
	(1.88)
lhs1984	0.0194
	(0.23)
Household Income Below Poverty Line at 1979 Survey	-0.167*
	(-2.07)
Averaged Reading and Math ASVAB Score 1980 Survey	0.334***
	(6.07)
1957b.Year of Birth	0
	(.)
1958.Year of Birth	0.0630
	(0.39)
1959.Year of Birth	0.126
	(0.77)
1960.Year of Birth	0.0272
	(0.17)
1961.Year of Birth	0.0834
	(0.53)
Interaction of Time and Smoking	0.0839***
	(5.27)
Constant	3.559***
	(8.65)
ln_sig	
Constant	-2.039
	(-0.45)
kappa	
Constant	3.707
	(0.22)
Observations	16428

t statistics in parentheses
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.17: Results from Best Fitting Gamma Model

Survey	Black Non-Obese	Black Obese	Hispanic Non-Obese	Hispanic Obese	White Non-Obese	White Obese
1.00	1.00	1.00	1.00	1.00	1.00	1.00
1.96	1.00	0.99	1.00	1.00	1.00	1.00
3.04	0.99	0.99	1.00	0.99	0.99	0.99
4.00	0.98	0.97	0.99	0.99	0.99	0.99
4.96	0.98	0.96	0.99	0.98	0.99	0.98
6.04	0.96	0.94	0.98	0.97	0.98	0.97
7.00	0.95	0.92	0.97	0.96	0.97	0.95
7.96	0.94	0.89	0.97	0.94	0.96	0.94
9.04	0.92	0.86	0.96	0.93	0.95	0.92
10.00	0.90	0.83	0.94	0.91	0.94	0.90
10.96	0.88	0.80	0.93	0.89	0.93	0.88
12.04	0.85	0.75	0.92	0.87	0.91	0.86
13.00	0.82	0.71	0.91	0.84	0.90	0.83

Table 1.18: Predicted Disability Free Survival by Race from Gamma Model

	(1) No Covariates	(2) Covariates	(3) No Covariates PS Weighted	(4) Covariates PS Weighted
BMI Above 35 at 1985 Survey	2.761*	2.661	4.079*	9.007***
	(2.16)	(1.90)	(2.27)	(3.88)
Took Any Illegal Substance in Year Prior to 1984 Survey		1.570*		15.46***
		(2.41)		(3.73)
Smoked Cigarettes in 30 Days Prior to 1984 Survey		1.744**		0.979
		(3.16)		(-0.04)
Hispanic Ethnicity		0.797		0.172*
		(-0.91)		(-2.29)
Race-Black		0.648*		1.664
		(-1.97)		(0.61)
Less than High School Education at 1984 Survey		1.320		0.827
		(1.30)		(-0.24)
Household Income Below Poverty Line at 1979 Survey		1.636*		2.376
		(2.40)		(1.05)
Averaged Reading and Math ASVAB Score 1980 Survey		0.439***		0.365**
		(-7.28)		(-3.09)
1957b.Year of Birth		1		1
		(.)		(.)
1958.Year of Birth		0.981		0.690
		(-0.04)		(-0.48)
1959.Year of Birth		0.906		0.653
		(-0.22)		(-0.52)
1960.Year of Birth		1.340		1.387
		(0.66)		(0.43)
1961.Year of Birth		1.096		1.430
		(0.21)		(0.50)
Observations	1151	1026	1026	1026

Exponentiated coefficients; *t* statistics in parentheses
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.19: Sensitivity Analysis BMI Cutpoint Severely Obese

	(1) No Covariates	(2) Covariates	(3) No Covariates PS Weighted	(4) Covariates PS Weighted
BMI Above 25 at 1985 Survey	0.922 (-0.56)	0.905 (-0.58)	0.869 (-0.89)	0.864 (-0.82)
Took Any Illegal Substance in Year Prior to 1984 Survey		1.547* (2.34)		1.495 (1.79)
Smoked Cigarettes in 30 Days Prior to 1984 Survey		1.695** (3.00)		1.596* (2.21)
Hispanic Ethnicity		0.823 (-0.78)		0.893 (-0.37)
Race-Black		0.645* (-2.00)		0.640 (-1.75)
Less than High School Education at 1984 Survey		1.328 (1.33)		1.477 (1.30)
Household Income Below Poverty Line at 1979 Survey		1.630* (2.39)		1.359 (1.20)
Averaged Reading and Math ASVAB Score 1980 Survey		0.436*** (-7.33)		0.447*** (-6.18)
1957b.Year of Birth		1 (.)		1 (.)
1958.Year of Birth		1.002 (0.01)		1.177 (0.34)
1959.Year of Birth		0.918 (-0.19)		0.849 (-0.33)
1960.Year of Birth		1.349 (0.67)		1.347 (0.62)
1961.Year of Birth		1.093 (0.20)		0.939 (-0.13)
Observations	1151	1026	1026	1026

Exponentiated coefficients; *t* statistics in parentheses
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.20: Sensitivity Analysis BMI Cutpoint Overweight

2 DOES OBESITY MODIFY THE EFFECT OF OCCUPATIONAL HISTORIES FOR ADULTS MOVING INTO RETIREMENT

Abstract

This study examines whether the effect of occupational histories on the health trajectories of older Americans differs by a person's obesity status around age 50. Using the 1992-2010 waves of the Health and Retirement Study (HRS), I employ longitudinal growth curve analysis to model trajectories of activities of daily living (ADL) limitations for obese and non-obese white men in the HRS cohort. Findings suggest that non-obese persons in physically demanding occupations have a lower rate of accumulation of ADLs as they age; however, no such protective association between physically demanding occupation and accumulation of ADLs is observed for the non-obese. Additional analysis shows that the association of physical activity with later life health is especially protective for non-obese persons facing other occupational disadvantages. 10% of non-obese persons would be expected to develop an ADL limitation if working in a job with high physical activity and low decision-making compared to 28% of obese persons. This work suggests differential associations of occupational activities for later life health depending on a person's obesity status at mid-life; however, future work should consider the mechanisms underlying these differences.

2.1 Introduction

The U.S. population is getting older and heavier. In 2010, some 40.2 million adults were above the age of 65 and 35% of adults over the age of 65 were obese (Fakhouri et al., 2012). While the U.S. life expectancy has been rising, improvements in health at older ages remain uncertain and unequal (Woolf et al., 2013). Obesity has become a growing population health concern, and while negative effects of obesity for health have been well established,it is less clear if there are heterogeneous effects of obesity for the health of older Americans. Following on cumulative (dis)advantage theory, which suggests that trajectories of (dis)advantage on health tend to compound over time (DiPrete and Eirich, 2006), one might expect that the individuals most affected by obesity are those with the greatest disadvantages. Obesity may compound previous stressors, and differences in health are accentuated over time. One potential salient stressor for U.S. adults is occupation exposures, the physical and mental strains that occur during working hours. Occupational exposures may compound the effects of subsequent health risks like obesity.

However, research is limited on how occupational activities may affect health moving into retirement and whether the effects of occupational exposures on subsequent health differ by whether or not a person is obese around the time they retire. This study builds on current literature by examining whether the effects of past occupational exposures on old age health differ by whether an individual is obese around retirement age. By employing longitudinal growth curve models, I can determine whether differences accumulate over time or remain relatively static in addition to examining whether there are differential associations of occupational exposures for obese versus non-obese persons.

2.2 Literature Review

Effects of Obesity for Health at Older Ages

The negative effects of obesity for health at older ages have been well established. Obesity after age 50 has been linked to increased risk and accelerated time to cognitive and physical declines. Obesity has been linked to various types of cancer, heart disease, stroke, and muscoloskeletal disorders (Field et al., 2001) (Bianchini et al., 2002), all of which are chronic conditions that may impair daily activities. Prior work has indeed suggested that obesity is linked to a greater risk of limitations on a number of activities of daily living

Effects of Obesity by Occupational Exposures

In 2013 the average American worker spent 1788 hours on the job (Statistics, 2009). What happens during employed time may shape a person's aging trajectory depending on the physical and mental demands of employment. Past research has theorized that individuals who have poor physical or psychosocial work environments may be more likely to develop health problems and impairments (Kuper and Marmot, 2003). Another strand of research in this area suggests that the effects of employment for health are gradational, that is as occupational standing increases so too does health. (Adler and Ostrove, 1999).

Stress has been suggested as a key mechanism explaining differences in the effects of occupations for health (Marmot, 2004). Individuals holding higher status occupations do *not* have fewer occupational strains, but rather higher status occupations are accompanied by greater autonomy, allowing workers who hold such jobs to better control their work environment and manage occupational demands (Marmot, 2004). The ability to control their work environment is hypothesized to afford individuals better coping skills on and off the job. Evidence supporting the autonomy hypothesis emerges from the Whitehall studies which found that individuals employed in higher status occupations were less likely to engage in risky health behaviors, but those that did faced fewer health consequences compared to counterparts working in lower status occupations. A noted limitation of this research is that it can be difficult to separate occupational influences from other dimensions of status, like education, which select an persons into occupations (Hout and DiPrete, 2006) and are highly related to perceived status and feelings of control [1].

Beyond psycho-social theories suggesting an influence of occupations for later health, the environment and physical tasks related to occupations may have direct consequences for health. Physically demanding occupations are associated with greater levels of joint and back pain at retirement **??**. The effects of occupational tasks may vary by a persons's weight. Prior studies examining obese person's health risks have found that obese persons engaged in physically demanding occupations are more likely to become injured on the job (Poston et al., 2011) (Haukka et al., 2012). Additionally, given that obese persons have been found to have a greater risk of musculoskeletal pain net of occupational exposures, it is possible that past occupational physical strains may compound an existing propensity for developing pain.

Whereas physically demanding occupations can carry risks of acute injury, especially for the obese, sedentary occupations may also carry health risks. Some analyses find that individuals engaged in highly sedentary occupations have been found to be more likely to develop serious chronic conditions including coronary heart disease, diabetes mellitius, and certain types of cancer (Van Uffelen et al., 2010). However, poor data quality and difficulty in accurately measuring sedentary behaviors (Proper et al., 2011) have hampered strong conclusions about the association of low occupational physical activity with subsequent health outcomes.

Despite past theories suggesting that occupation may matter for later life outcomes and the effects of occupation may vary by obesity status, limited data is available on how obesity may modify the effects of occupational exposures as individuals move into

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[1] One could trace the selection process back further and argue that underlying expectations Hauser (1972) or non-cognitive traits, especially persistence (Heckman and Rubinstein, 2001) are underlying factors strongly influencing education and occupational attainment processes, thus any comparison by education or occupation is flawed given that different types of people self-select into these categories

retirement. One notable exception is a recent analysis of a middle aged French cohort which finds that for only obese persons, physically demanding occupations are associated with greater pain following retirement (**?**). However, this study is limited in that it relies on retrospective reporting, may not reflect the experiences of American cohorts, and conducts only a cross-sectional analysis.

It is worth acknowledging here that although there is some extensive theory suggesting that occupations may matter for later life health, empirical evidence of occupational effects for health has been more mixed. A recent study using the Health and Retirement Study, the same data source employed in this project, finds that whereas an individual's longest held occupation may predict an individual's initial health, it is not related to subsequent changes in self-reported health (Gueorguieva et al., 2009). However, other studies do find a significant effect of occupations and that the effects of occupation for health do expand over time (Pietiläinen et al., 2011). There are a few potential explanations for these discordant effects. First, it is possible that the effects of occupation do differ depending on the health outcome. Another explanation for the contradictory findings is that occupational effects may aggregate for individuals of some race, sex, and behavioral backgrounds but not for other persons. Third, how occupations are measured may alter the effects of occupation observed.[2]

Objectives

This study contributes to existing literature by examining whether the effects of longest held occupation differ by whether or not a person is obese as they approach retirement age. A novel aspect of this study is that it contains very detailed measures of occupational exposures and information about the duration of those exposures. Specifically, this study explores the following research questions:

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[2] Note, I know given this prior mixed evidence it would make more sense to have a paper just looking at the effects of occupation for subsequent health. Michal and I have been working on just such a manuscript. When that project is published, I will just cite our results and drop this paragraph

Research Question 1: Which characteristics of occupational exposures affect the health trajectories of older adults?

Hypothesis 1: Individuals in more prestigious occupations such as managerial and professional occupations have better health outcomes following retirement, as do persons who have more decision making power within their occupation. Individuals who were engaged in physically demanding occupations will have poorer health outcomes following retirement.

Research Question 2: Does the influence of past occupational exposures on later life health differ by whether a person is obese around retirement ages?

Hypothesis 2: The health of obese persons is more affected by past occupational exposures. Differences in the health of obese and non-obese individuals become exacerbated over time. Specifically, we should expect potentially divergent effects of occupational physical activity which may be differentially harmful for obese individuals.

2.3 Data

Data on health and occupational classification comes from the *Health and Retirement Study* (HRS), a longitudinal survey of community-dwelling, middle-aged and older Americans containing information on both socioeconomic conditions and health status. (Juster and Suzman, 1995).

Baseline occupation data was taken from the 1992 survey round when the HRS administered a full battery of questions on respondents occupational histories. Baseline data on limitations in activities of daily limitation was taken from the 1998 survey. Limitations in ADLs prior to 1998 was not measured because response options differ for the earlier survey rounds. Follow-up information on limitations in ADLs were collected at two year intervals between 1998 and 2010. I additionally exclude respondents who were female, non-white, or part of another birth cohort, because occupational histories differ by race, sex, and cohort, and there is a limited sample size to run stratified models for each sub-group.

Data on the activities a person performs in their occupation is taken from the O*NET database. O*NET is a comprehensive listing of occupations that contains detailed data on the requirements and activities related to particular occupations. Established in 1998 as an update to the DOT database the O*NET database has been updated 19 times to better reflect occupational activities and changes in the responsibilities associated with activities over time. This study uses data from O*NET version 19 published in 2010. O*NET and HRS data are merged using the respondent's detailed three digit census occupation code. As occupations in the O*NET database are identified using 2010 O*NET SOC codes, the O*NET codes are converted to census codes using the Federal Crosswalk System for converting occupational codes.

Dependent Variable

The key dependent variable is the number of activities of daily limitation (ADLs) a person reports. This measure summarizes a person's difficulty in walking across a room, getting in and out of bed, dressing, bathing, and eating. The scale ranges from zero (no limitations) to five (limited in all domains). Activities of daily living are used to summarize a person's overall health status because reported ADL limitations have been shown to be predict functional status (Idler and Benyamini, 1997; Fried et al., 2001). ADL limitations are measured at survey rounds 4-10 as these are the only rounds where comparably worded measures of ADL limitations are available.

Independent Variables

This study examines whether the effect of occupational exposures on trajectories of ADLs is differs by a person's obesity status. Obesity is defined by whether a person has a body mass index (bmi) above 30 at the first survey round. BMI is calculated using an individual's self-reported height and weight. Obesity is measured at the baseline survey round so that it may be treated as a moderator of subsequent trends in health limitations.

Occupational exposure is measured in two ways in this analysis: the activities a person performs on the job and the general occupational title. The activities performed on the job are: general levels of physical activity, levels of problem solving and decision-making, levels of interpersonal relationships, and levels of creative thinking on the job. Measures are selected because past health and policy research suggests that obese persons who work in physically demanding jobs are more likely to develop health impairments and research in social stratification has suggested that the degree to which a person can have control and feel valued in their job as evidenced by decision making, problem solving, and the development of relationships, can offset some occupational strains. These measures are continuous scales ranging from 0 (not done on the occupation) to 6 (a major component of the occupation) with approximately a normal distribution. Across analyses, the scales are standardized to have a mean and 0 and a standard deviation of 1 and thus reflect the relative strain of particular occupations.

A person's more general occupational title is an administrative HRS code and divides jobs into one of eight categories: professional, managerial, clerical, sales, production, operations, service, and farming. Individuals are considered to have a longest held occupation if they spent more than ten years in any one occupation. Longest held occupation is the measure used as a proxy for cumulative exposures to occupational strain. [3]

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[3] These eight categories are collapsed from 16 categories available for HRS cohort:

1. managerial specialty oper (managerial)
2. prof specialty opr/tech sup (professional)
3. sales (sales)
4. clerical/admin supp (clerical)
5. svc:prv hhld/clean/bldg svc (service)
6. svc:protection (service)
7. svc:food prep (service)
8. health svc (service)
9. personal svc (service)
10. farming/forestry/fishing (farming)
11. mechanics/repair (production)

These two measures of occupational exposure are used because they may capture different dimensions of a person's work history. Occupational titles may capture the relative standing of individuals and potentially some industrial exposures while work activities may reflect an individual's day to day exposures and physical and mental strains while on the job.

One concern in linking O*NET data to the respondent HRS employment histories is that the occupational responsibilities of particular respondents may differ from the more general responsibilities quantified in the O*NET codes. As a check of our occupational measures, in a sensitivity analysis we look at how well O*NET measures predict HRS respondents' answers to a respondent's report of occupational physical activity [4].

Controls

Respondent's educational attainment (less than high school, high school graduate, some college, completed four year college degree or greater) and whether the respondent ever smoked are controlled for in some models. These measures are controls in this analysis because education may select an individual to be eligible for particular occupations and controlling for this selection process may lead to a more conservative estimate of the effect of occupations net of education. A history of smoking is also controlled for because smoking onset often begins in young adulthood, smoking history is an important predictor of health trajectories,and smokers are less likely to be obese compared to non-smoking peers.

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12. constr trade/extractors (production)
13. precision production (production)
14. operators: machine (operations)
15. operators: transport, etc (operations)
16. operators: handlers, etc (operations)

[4] While some information on respondent's occupational tasks is available from the HRS data, these measures are not used in the main analysis because information is only available for a respondent's currently held occupation

2.4 Analytic Strategy

Analysis consists of two parts. First, I estimate some descriptive statistics which illustrate the distribution of work activities by occupational title, highlight the distribution of occupational titles initial limitations in daily living for the sample, examine the correlations among different dimensions occupational activities, and establish that individual reports of occupational activity correlate with measures in the O*NET database.

Having established this background information about the sample and measures of occupation, I use longitudinal growth curve modeling to estimate whether obesity at midlife changes the effects of past occupational exposures. Longitudinal growth curve modeling is used here to predict for obese and non-obese subgroups, the expected probability of having between 0 and 5 limitations at each time point as a function of specified covariates. I treat the number of ADL limitations as a count variable with a zero inflated Poisson distribution and obtain parameter values using a maximum likelihood estimator with robust standard errors. The exact model specification is shown in Algorithm 4

Algorithm 4 Zero Inflated Poisson Specification

$$\nu_i = \exp(\alpha + x_i'\beta)$$

$$\Pr(y_i|x_i) = \begin{cases} \pi_i + (1-\pi_i)\exp(-\nu_i) & y_i = 0 \\ (1-\pi_i)\frac{\exp(-\nu_i)\nu_i^{y_i}}{y_i} & y_i \geqslant 1 \end{cases}$$

The interpretation of this model can be divided into two parts: first, the likelihood an individual in the sample will report no ADL limitations over the entire period of follow-up (sometimes called a structural zero), and second, the expected number of limitations an individual is expected to develop,conditional on not being a structural zero.

Occupation is allowed to affect both the probability of an individual being free of ADL limitations (i.e. being in the structural zero category) and, for those not identified as structural zeros, the expected number of ADL limitations. For individuals identified as not being structural zeros, occupation is allowed to influence two parameters in the model: 1. the intercept or the number of limitations a person is expected to have at the start of analysis and 2. the slope or the expect trend in accumulation of limitations across time.

In estimating these models, the structure of the longitudinal growth curves can incorporate linear or quadratic terms and a covariance between the intercept and slope. The preferred model is chosen based on the BIC and statistical significance of the structural parameters.

To test whether the association of occupational exposures to limitations in activities of daily limitations varies across the obese and non-obese sub-samples, stratified models are run for two groups:

1. Non-obese persons employed in an occupation for 10 or more years
2. Obese persons employed for 10 or more years

For the individuals employed for more than 10 years, four models are run. The first models ADLs as only a function of the physical activities a person performs in their longest held occupation. The second model measures ADLs as a function of physical activities and incorporates education and smoking controls. The third model examines the effects of not only occupational physical activity but decision-making, creative thinking and interpersonal relationships while adjusting for the education and smoking controls. While this study focuses on whether there is a differential effect of occupational physical activity on health outcomes for obese versus non-obese persons, by adding other indicators of occupational activities, this analysis can also test if effects of occupational physical activity are offset by other rewards and demands of jobs. The fourth and final model predicts ADL limitations as a function of occupational title. [5]. Examining the effects of occupation can provide more insight into whether some types of occupations are differentially harmful or beneficial for the later health of obese versus non-obese persons.

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[5] When looking at the activities done in an occupation, members of the armed forces are kept in the sample, but they are dropped in the analysis of occupational titles as there was insufficient sample size

When interpreting results across these models, I focus on comparing two estimated quantities across models fit for obese and non-obese persons: the average number of limitations expected at a survey round and the probability of being without a health limitation when covariates are held at the mean. [6]

Additional Model Specifications

As a check on whether results are sensitive to the measure of health chosen and model selected, I run an additional model specification where time of death is the dependent variable and the baseline hazard is modelled using a Gompertz distribution ($\lambda = exp\{\alpha + \beta t\}$). In this specification, I again control for education and smoking history but now, due to limited power, pool obese and non-obese persons in a single model and measure work activities as the activities done in the longest held occupation weighted by standardized duration spent on the job. Model fit is assessed using a Cox-Snell residual test and tests for differences in the association of work activities with the hazard of dying are done by including interaction terms between obesity and the work activity in the survival model.

2.5 Results

Descriptive Results

Descriptive statistics for the full eligible sample and by obesity status are shown in Tables 3.3 and 3.4. Approximately 500 observations are lost in the analytic sample due to attrition or mortality prior to when the health trajectory questions were measured. To determine the degree to which exit from the study is affects the representativeness of the sample, t-tests

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[6] I focus on estimates generated from these models rather than structural parameters themselves because the allowed correlations between the model intercepts and slopes makes interpretation of individual parameters tricky. I have also generated predicted probabilities for having specific numbers of limitations. Thus, if someone had a reason to think more than 1 or 2 limitations would be especially harmful, that could be calculated from my estimates. A potential limitation of this study is that I look only at the count of limitations and do not identify associations with particular limitations in ADLs. Such analysis, while potentially interesting, is outside of the scope of the present paper.

are used to compare the characteristics of respondents included in the sample to those who attrite. Results from these tests can be seen in Table 2.4. Comparison of individuals who remain within the sample compared to those who attrite reveals that attriters are less likely to be in professional occupations, more likely to be in operations occupations, have lower levels of interpersonal relationships on the job, are more likely to have not completed high school, and are less likely to have finished college.

The distribution of health limitations at the start of analysis for the obese and non-obese sub-samples is also shown in Figure 2.1. On average, non-obese respondents have .14 health limitations compared to obese respondents who have .18 health limitations, and a t-test showed the mean number of limitations was not statistically different across obese and non-obese persons. Given that there is a zero inflated poisson distribution of limitations and a great majority of obese and non-obese respondents have no limitations, differences in the number of limitations for obese and non-obese respondents are also examined in a series of t-tests [7] Table 2.2 shows that there are statistically significant differences in the distribution of limitations for the obese and non-obese respondents. Non-Obese persons are significantly more likely to have no limitations and significantly less likely to have one ADL limitation. 92% of non-obese respondents have no health limitations at survey round 4 compared to 88% of obese respondents. 4% of non-obese respondents have one health limitation compared to 7% of obese respondents. [8]

An interesting contribution of this study is that occupational exposures are measured both by an individual's general occupational title and by the the activities an individual performs while on the job. Figures 2.2, 2.3, 2.4, and 2.5 demonstrate that looking within occupational titles, there is considerable variability in the job activities and the occupational grouping alone does a poor job predicting what an indvidual's occupational responsibilities

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[7] Technically a negative binomial distribution does a slightly better job of characterizing the distribution of ADLs. I do not use the negative binomial in my modeling strategy because of limited program support. Mplus has algorithms better designed for zero inflated poisson distributions. I acknowledge this limitation, but realistically, the differences between the zero inflated negative binomial and poisson distributions are small

[8] T-test Table shows 2 sided t-test. Difference are still significant when doing a 1 sided test

will entail.

I also examine the correlation of occupational activities with each other. Consistent with prior work (Marmot, 2004), occupational physical actvity is negatively correlated with decision-making power, development of interpersonal relationships, and creative thinking on the job. Creative thinking, inter-personal relations, and decision-making power are positively correlated with each other. [9] An additional concern in conducting this analysis is whether O*NET measures of occupational activities are actually related to the type of work respondents report doing. One descriptive check suggests that this is indeed the case. Table 2.5 shows that a 1 standard deviation in the O*NET measure of occupational physical activity is associated with respondents being over twice as likely to report that they frequently engage in physical activities while employed (OR= 2.364).

Longitudinal Growth Models

When looking at the effect of occupational physical activity, we see significant effect differences for obese and non-obese persons. Model 1 which models limitations in ADLs as a function of physical activity. Results from this model show countervailing trends for the obese and non-obese. For the non-obese, there is evidence of non-linearity in the rate at which limitations are accrued; based on the BIC, the preferred model includes a quadratic trends. Physical activity has no effect on the initial number of limitations or rate at which they accrue. For the obese, limitations increase at a linear rate and physical activity is associated with an increase in the initial number of limitations and has no effect on the rate at which limitations accumulate. Although these differences may seem subtle, substantively changing the level of occupational physical activity has very different implications for the obese and non-obese. Figure 2.18 shows the expected proportion of the sample with a

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[9] Because of the high positive correlation between creative thinking, inter-personal relationships, and decision-making on the job, I was concerned about near multi-collinearity in the longitudinal growth curve models. Thus, in a subsequent analysis I ran an additional lcg which only modelled physical activity and decision-making power on the job. I chose decision-making power because it most closely maps on to Marmot's idea of occupational autonomy.

given number of limitations for the obese and non-obese assuming the level of occupational physical activities is set at 0, the approximate mean for the sample. When all covariates are set to the means, 92% of the non-obese sample is expected to be without any limitations in activities of daily living while only 89% of the obese sample is without any limitation at the fourth survey round. By the tenth survey round, 87% of the non-obese are expected to be have no ADL limitations while only 79% of the obese sample is expected to be without any limitations. Figures 2.6, 2.7 , and 2.8 graphically show the trends of average limitation for the obese and non-obese subsamples when occupational physical activity is set at 0, -2, and 2. With mean levels of physical activity, the obese have, on average significantly greater impairment than the non-obese (.06 limitations for the obese compared to .04 for the non-obese at the 10th survey round). However, when physical activity is 2 standard deviations below the mean, the non-obese and obese have comparable limitations (approximately .035 limitations at the 10th survey round). When physical activity is 2 standard deviations above the mean, the obese once again have higher levels of impairment (.10 limitations for the obese compared to .03 for the non-obese at the 10th survey round). In other words, when a person's job history was sedentary, there is no difference in the accumulation of ADL limitations by whether that person subsequently is obese or non-obese. But, when a person's job history was physically demanding, subsequent obesity is associated with a greater accumulation of ADL limitations.

Once education and smoking controls are added to the model, levels of occupational physical activity have no effect for the obese or the non-obese. Additionally, there is now evidence of non-linearity in health trajectories for the obese, as evidenced by the BIC. However, the growth curves seem to reflect similar trajectories compared to the model without controls. Figure 2.19 shows the expected proportion of the sample with a given number of limitations for the obese and non-obese assuming the level of occupational physical activities is set at 0 and other covariates are set to the sample mean. At the fourth survey round 92% of the non-obese sample is expected to have no ADL limitations while

89% of the obese are expected to be without limitations. By the tenth survey round, 87% of the non-obese sample is expected to remain without a health limitation while 81% of the obese sample is expected to be without limitations. Figures 2.9, 2.10 , and 2.11 graphically show the trends of average limitation for the obese and non-obese subsamples when occupational physical activity is set at 0, -2, and 2 and other covariates are set to the sample mean. Although the influence of physical activity is attenuated, again at medium and high levels of physical activity, the obese have higher levels of limitations compared to the non-obese. However, at low levels of physical activity, the obese and non-obese have comparable levels of limitation across survey rounds.

Finally, after adjusting for the effect of other occupational activities, occupational physical activity becomes marginally significant and is negatively associated with the rate of limitation accumulation for the non-obese but has no significant effect on initial levels of limitations for the non-obese. Occupational physical activity has no statistically significant effect for obese persons but is negatively associated with the initial level of limitations and is positively related to the rate of accumulation in limitations over time. Figures 2.12, 2.13 , and 2.14 graphically show the trends of average limitation for the obese and non-obese subsamples when occupational physical activity is set at 0, -2, and 2 and other covariates are set to the sample mean. In these graphs, there is no longer the consistent pattern observed in the previous graphs. Now when occupational activity is set two standard deviations below the mean, the obese have higher levels of impairment compared to the non-obese at all survey rounds. At two standard deviations above the mean, the obese and non-obese have similar levels of impairment across survey rounds 4-7 and the obese have higher levels of impairment at survey rounds 9 and 10. [10]

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[10] Given that there is a strong negative correlation between physical activity and occupational decision-making, creative thinking and interpersonal relationships, it might be unreasonable to assume that a person has very high/low levels of physical activity on the job but average values on these other variables. Subsequently in sensitivity analyses, I ran models where physical activity is set to either 0, -2, and 2 and other occupational activities are set to 0,1, or -1. In running this analysis, I can compare outcomes for obese and non-obese persons in more realistic jobs, low physical activity, high decision-making power, or high physical activity, low decision-making power. Results from this subsequent analysis are shown in Figures 2.23, 2.24 , and 2.25

As mentioned previously, there was a high correlation across occupational creative thinking, interpersonal relationship, and decision-making activities. Near multicollinearity may make identification of significant effects difficult. Thus, I run one final analysis where I examine the effects of physical activity controlling only for occupational decision-making. Findings are similar to the previous model which controls for all occupational markers. For the non-obese, occupational physical activity reduces the rate of ADL accumulation. The only difference is that now the effect of occupational physical activity is statistically significant. Results from this subsequent analysis are shown in Figures 2.15, 2.16 , and **??**

Additional Analyses

As a check on the results found in the trajectory models, I examine whether similar differences between obese and non-obese persons occur when mortality is the outcome. Results from a survival analysis where the baseline hazard is characterized by a Gompertz distribution are shown in Table 2.8 with checks on the Cox-Snell residuals shown in **??**. Results from this survival analysis are consistent with results found in the longitudinal growth curve models. Occupational physical activities are marginally associated with a lower mortality hazard for non-obese persons after controlling for other work activities, education, smoking, and obesity. There is a marginally significant interaction between obesity status and occupational physical activity such that the obese have a higher hazard of dying and an the interaction of obesity and physical activity reveals that increasing levels of occupational physical activity is associated with a higher risk of dying for persons who are obese at around retirement ages. For the non-obese the hazard ratio is .90 for a standard deviation increase in physical activity and for the obese the hazard ratio is 1.07. Unlike in the longitudinal growth curve models, other occupational activities are associated with mortality risk. Increases in occupational decision-making at the longest held occupation is associated with higher mortality risk while occupational relationship building and creative thinking are associated with a lower risk of death. However, although these occupational activities were associated with mortality, interactions between these occupational activities and obesity were not statistically significant.

2.6 Discussion and Limitations

This study finds evidence to suggest that occupation may influence patterns of limitation accumulation at retirement ages. Returning to the two research questions previously posed, this study finds features of occupations do affect subsequent health trajectories and the effects differ across obese and non-obese persons. In particular, I find that occupational physical activity is protective for persons who are non-obese around retirement age but has no effect on the health trajectories of obese persons after controlling for covariates that select an individual into particular occupations and other occupational activities.

An interesting finding across models is that for individuals engaged in high levels of physical activity, obese persons have greater levels of of ADL limitations compared to the non-obese before other occupational activities are controlled for. However, after adjusting for occupational activities and holding other occupational activities at their mean values, differences between obese and non-obese persons are much attenuated. This suggests that, although not statistically significant, having even average levels of occupational decision-making, creative thinking, and interpersonal relationships, may offset differences in the effects of occupational activity for obese persons compared to the non-obese. However, when other occupational activities are set to values typically observed for occupations (high physical activity, low decision-making, creative thinking, and inter-personal relationships and low physical activity, high decision-making, creative thinking, and inter-personal relationships) we again see differences in the effects of physical activity such that the obese have a greater number of impairments compared to the non-obese.

While this study finds some evidence to suggest that aspects of occupational exposure do matter for subsequent health trajectories, it does have a number of limitations that need to be acknowledged. First, the study is limited to examining the occupational experiences of white men in the HRS birth cohort. The results are not generalizable to other sex-race groups nor to men who died before the HRS study began. Given that 28% of men born in 1930 did not survive to age 60 and 15% of men born in 1940 did not survive to age 50 (when the HRS study began), this selection is quite substantial (See Figure 2.26.

Additionally, measures of occupation are limited on a few dimensions. First, measures of occupational activity were taken from 2010 but are intended to measure occupational activities which occurred before 1992. Although one sensitivity check showed that the physical activities respondents reported at their current job was highly related to the O*NET occupation measures from their longest held occupation, these measures may still not completely represent respondents' occupational activities if occupational tasks were different in the years prior to 1992.

An additional limitation of this analysis is that the occupational measures are restricted to individuals who worked at least 10 years in a single occupation. Thus, the sample excludes individuals who had unstable job histories. It additionally cannot aggregate total occupational exposures because the survey does not have complete information on all occupations respondents held.

2.7 Figures

Figure 2.1 Distribution of Limitations in Activities of Daily Living at Survey Round 4

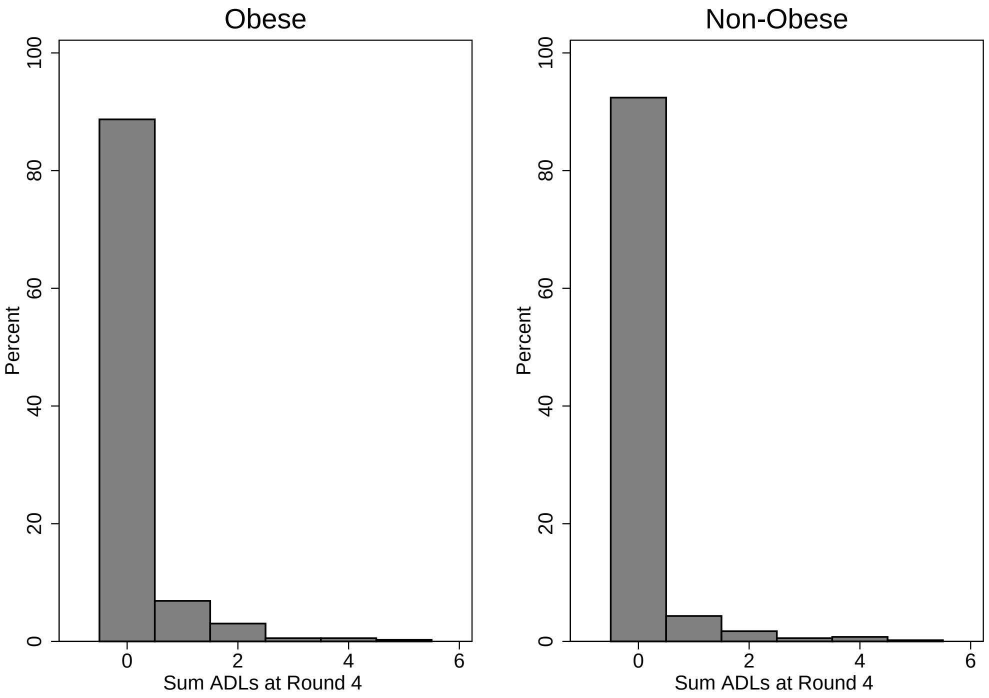


Figure 2.2 Physical Activity by Occupation Type

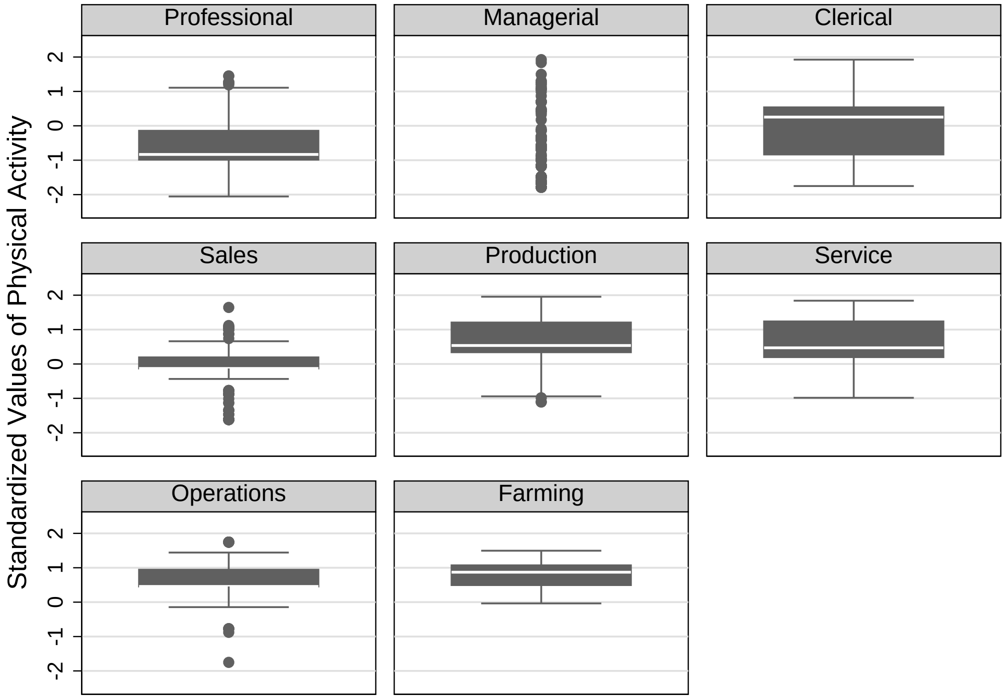


Figure 2.3 Creative Thinking by Occupation Type

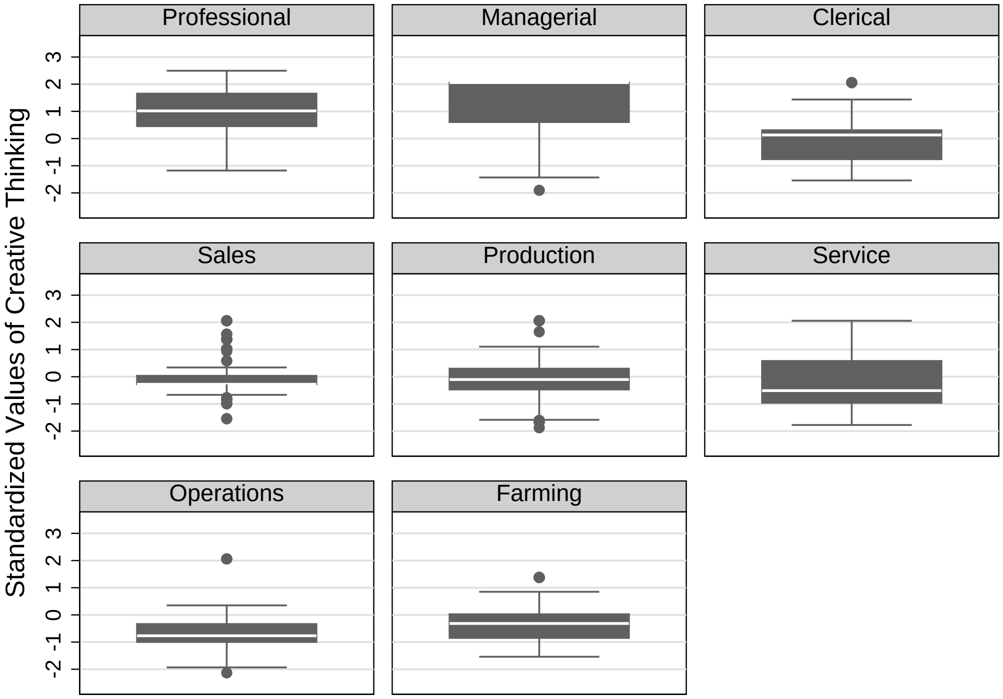


Figure 2.4 Decision Making by Occupation Type

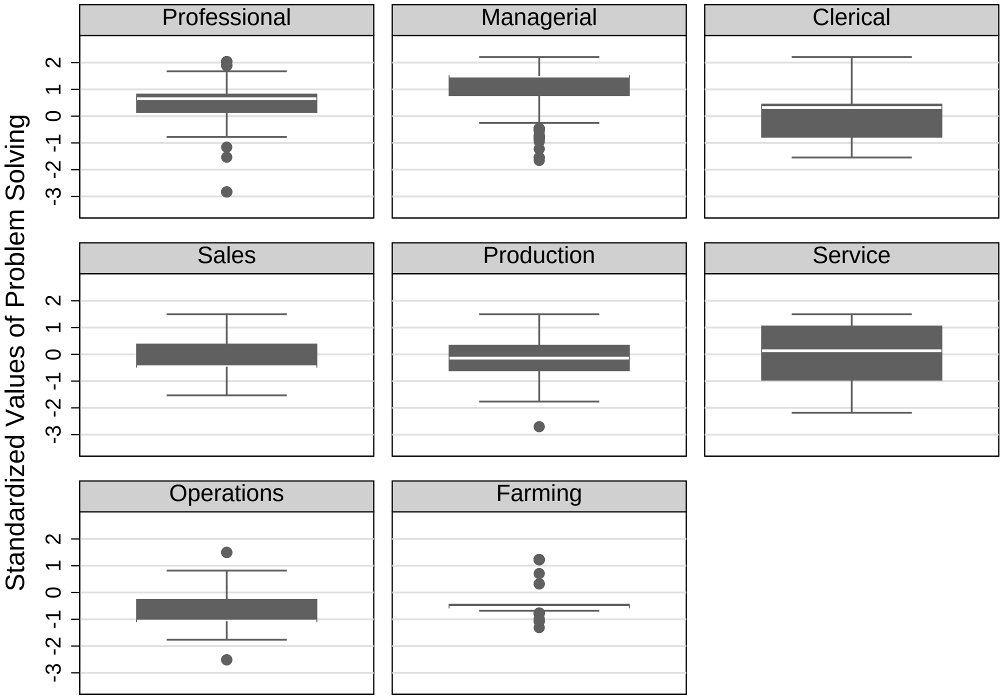


Figure 2.5 Interpersonal Relationships by Occupation Type

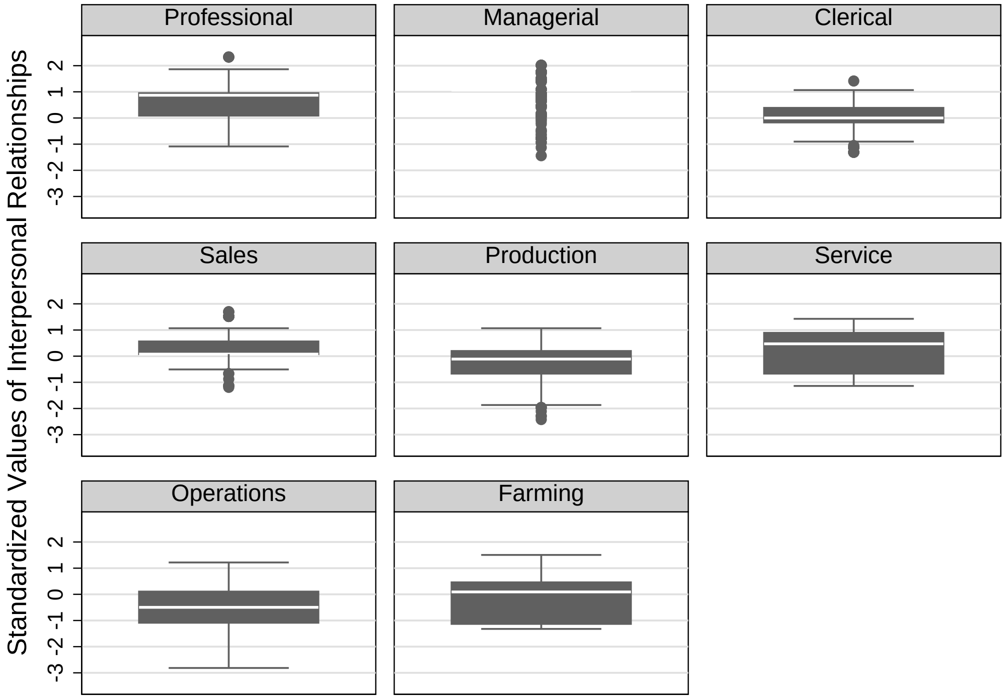


Figure 2.6 Average Number of Limitations with Physical Activity at Mean

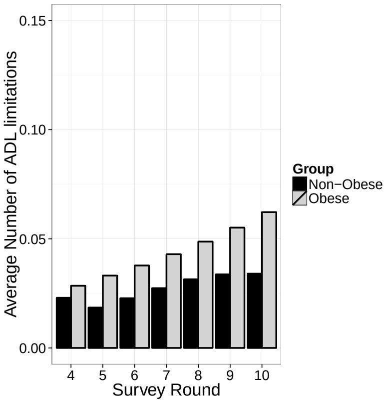


Figure 2.7 Average Number of Limitations with Physical Activity Two Standard Deviations Below the Mean

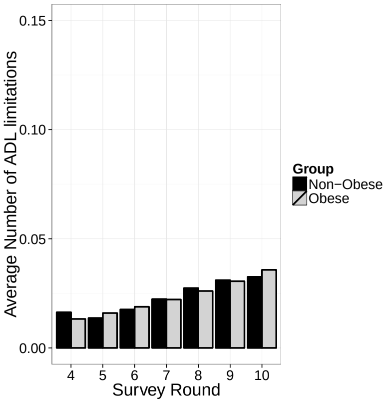


Figure 2.8 Average Number of Limitations with Physical Activity Two Standard Deviations Above the Mean

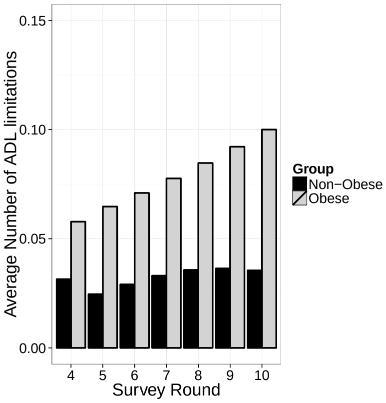


Figure 2.9 Average Number of Limitations with Physical Activity at Mean with Controls

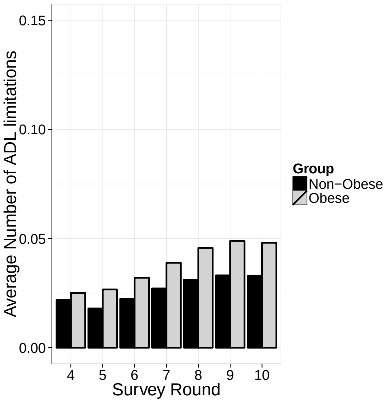


Figure 2.10 Average Number of Limitations with Physical Activity Two Standard Deviations Below the Mean with Controls

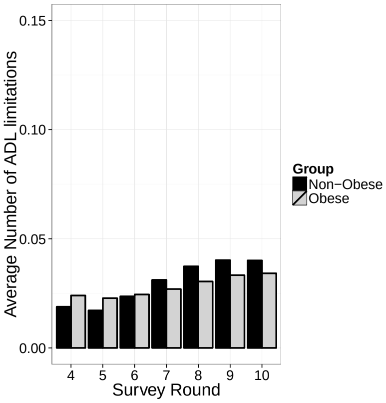


Figure 2.11 Average Number of Limitations with Physical Activity Two Standard Deviations Above the Mean with Controls

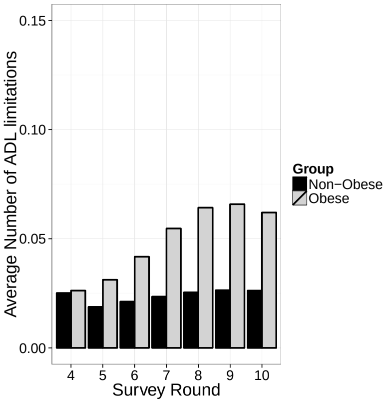


Figure 2.12 Average Number of Limitations with Physical Activity at Mean Controlling for Demographics and Occupational Activities

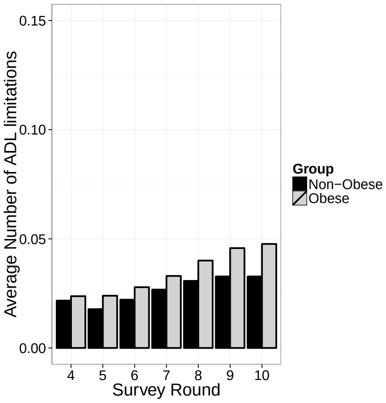


Figure 2.13 Average Number of Limitations with Physical Activity Two Standard Deviations Below the Mean Controlling for Demographics and Occupational Activities

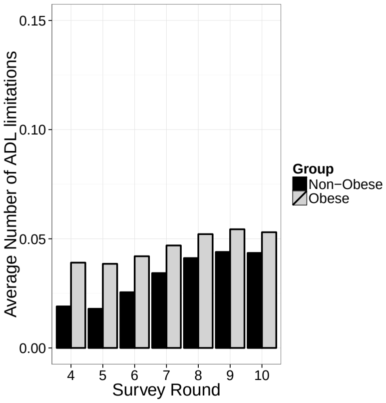


Figure 2.14 Average Number of Limitations with Physical Activity Two Standard Deviations Above the Mean Controlling for Demographics and Occupational Activities

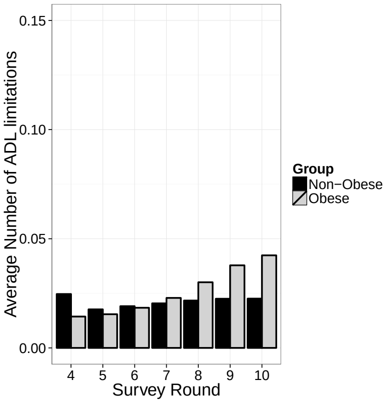


Figure 2.15 Average Number of Limitations with Physical Activity at Mean Controlling for Demographics and Occupational Decision-Making

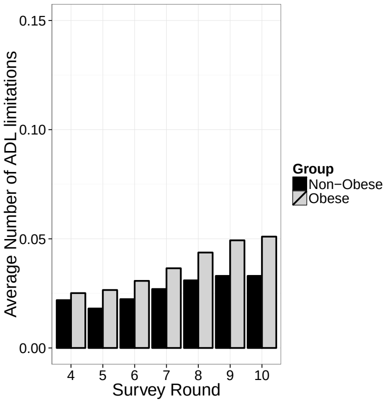


Figure 2.16 Average Number of Limitations with Physical Activity Two Standard Deviations Below the Mean Controlling for Demographics and Occupational Decision-Making

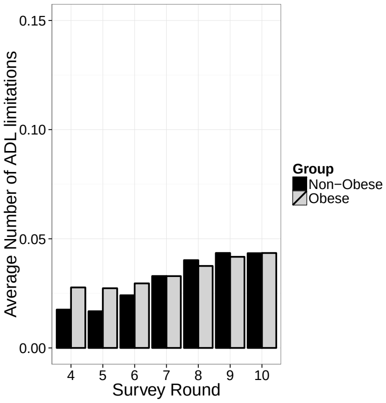


Figure 2.17 Average Number of Limitations with Physical Activity Two Standard Deviations Above the Mean Controlling for Demographics Occupational Decision-Making

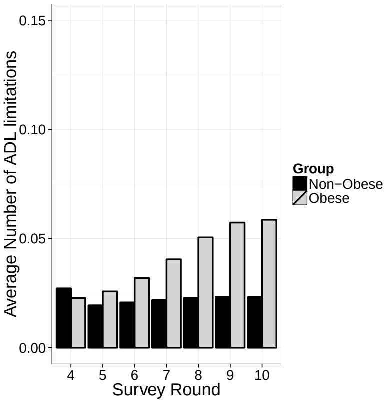


Figure 2.18 ADL Limitations for Non-Obese Persons Modeling Only Physical Activity

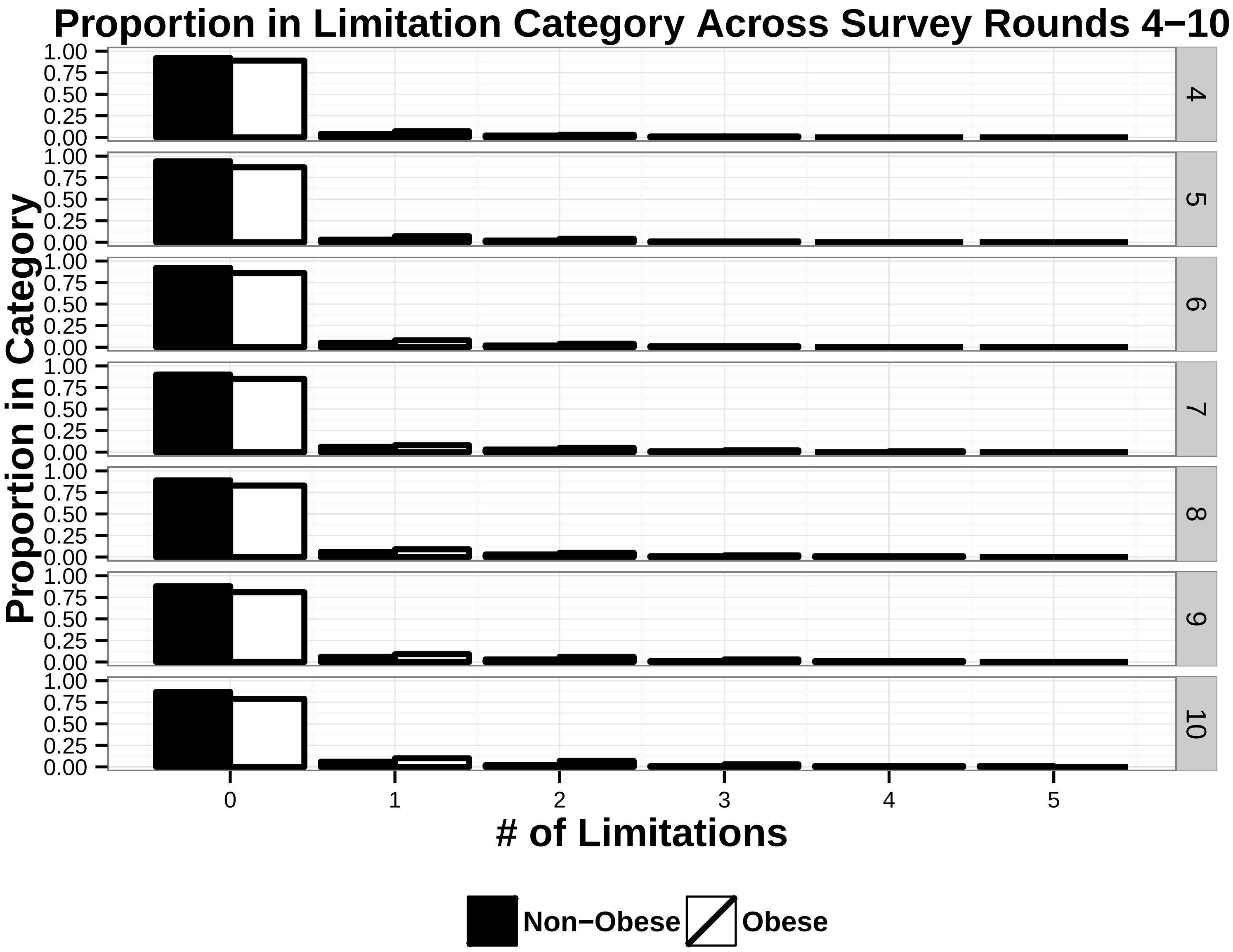


Figure 2.19 ADL Limitations for Non-Obese Persons Modeling Physical Activity and Controls

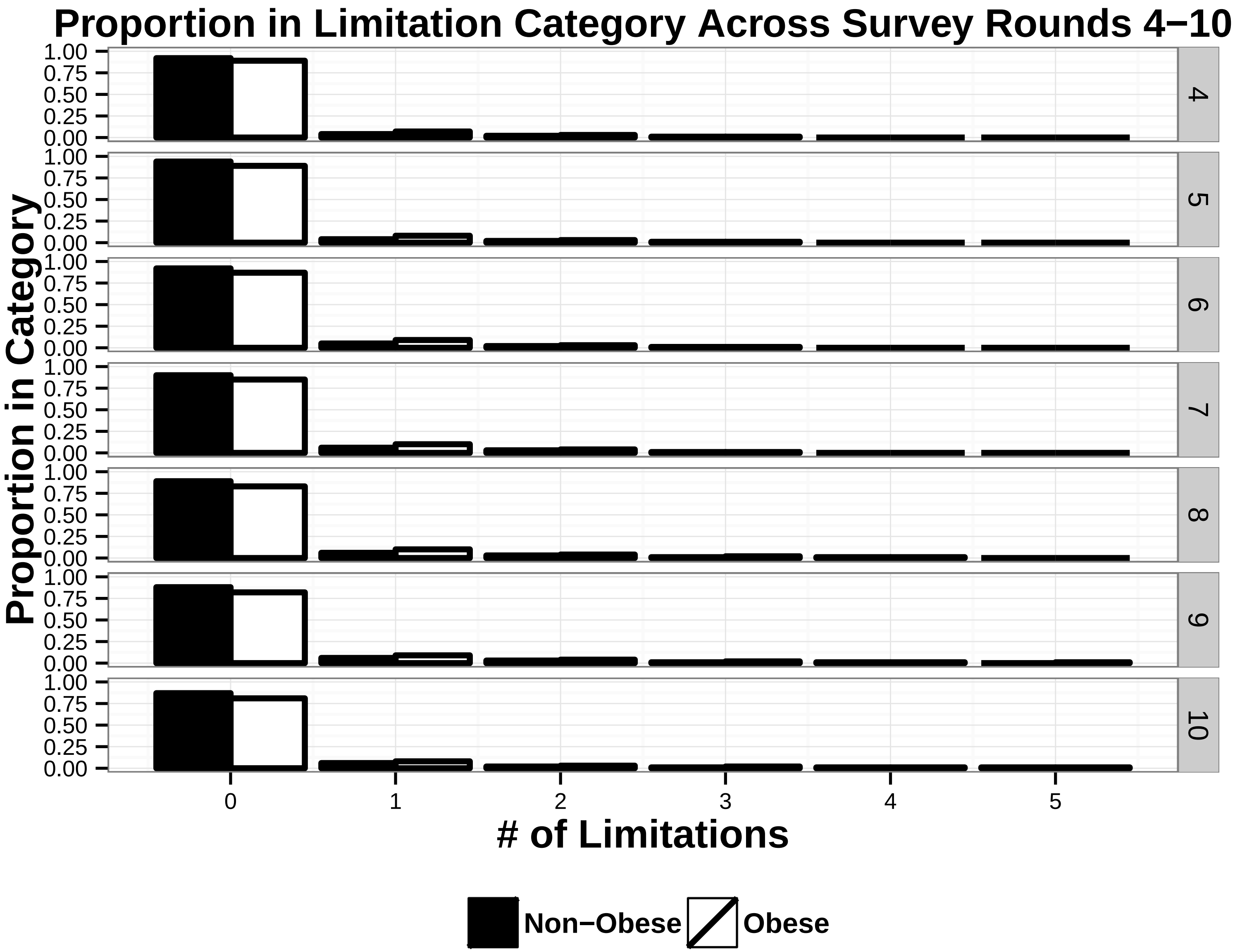


Figure 2.20 ADL Limitations for Non-Obese Persons Modeling All Occupational Activities and Controls

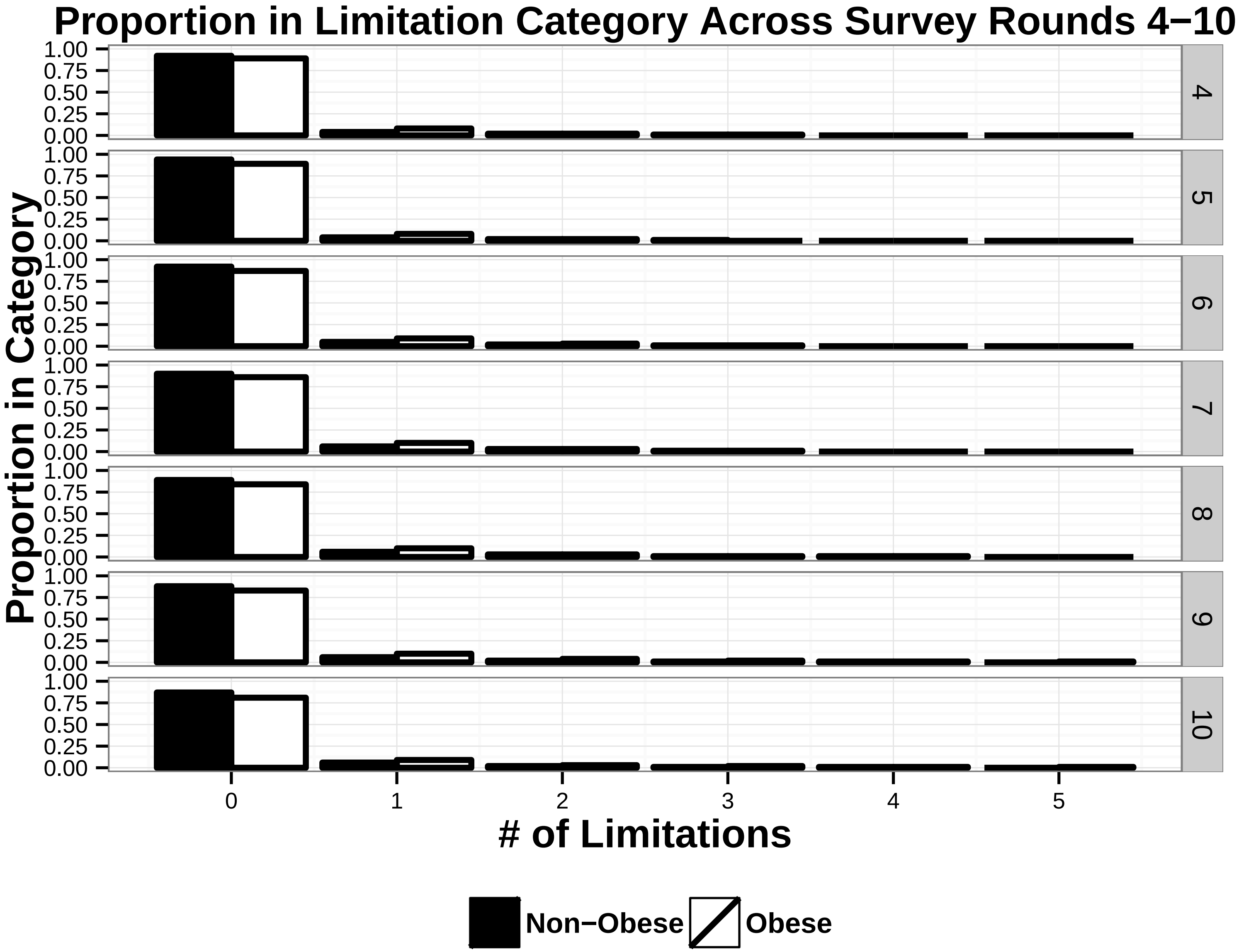
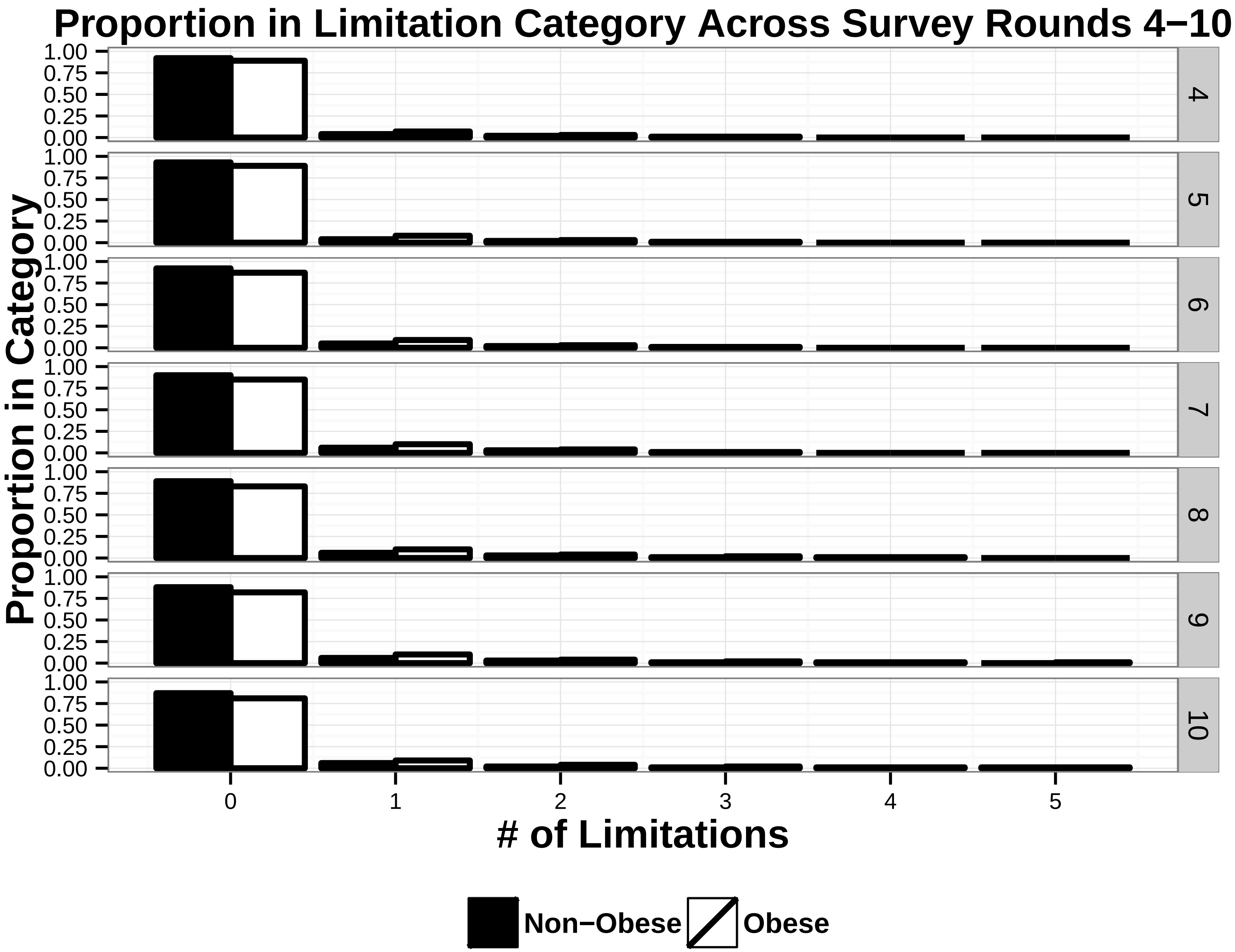


Figure 2.21 ADL Limitations for Non-Obese Persons Modeling Occupational Decision-Making and Controls



2.8 Tables

	Mean
Longest Occ Professional Round 1	0.17
Longest Occ Manager Round 1	0.22
Longest Occ Clerical Round 1	0.05
Longest Occ Sales Round 1	0.08
Longest Occ Production Round 1	0.24
Longest Occ Service Round 1	0.04
Longest Occ Operations Round 1	0.17
Longest Occ Farming Round 1	0.02
Level of General Occ Physical Activities	0.03
Level of Occ Creative Thinking	0.27
Level of Occ Interpersonal Relationships	0.21
Level of Occ Decision Making and Problem Solving	0.15
Obese at Round 1	0.20
Respondent Ever Smoked Round 1	0.73
Less than HS Diploma	0.25
Completed HS	0.31
Some College	0.20
Observations	2169

Table 2.1: Eligible Sample Descriptives

ADLS Round 4	**T Value**	**P(Diff)**	**Non-Obese Mean**	**Obese Mean**
0 Limitations	2.27	0.02	0.92	0.89
1 Limitation	-2.03	0.04	0.04	0.07
2 Limitations	-1.56	0.12	0.02	0.03
3 Limitations	0.02	0.99	0.01	0.01
4 Limitations	0.43	0.66	0.01	0.01
5 Limitations	-0.24	0.81	0.00	0.00
Observations	1797		1434	363

Table 2.2: Differences in Count of Limitations at Survey Round 4 for Obese and Non-Obese Respondents

	Non-Obese	**Obese**	**Total Sample**
Longest Occ Professional Round 1	0.18	0.12	0.17
Longest Occ Manager Round 1	0.22	0.21	0.22
Longest Occ Clerical Round 1	0.05	0.05	0.05
Longest Occ Sales Round 1	0.08	0.09	0.08
Longest Occ Production Round 1	0.24	0.24	0.24
Longest Occ Service Round 1	0.04	0.05	0.04
Longest Occ Operations Round 1	0.16	0.19	0.17
Longest Occ Farming Round 1	0.02	0.03	0.02
Level of General Occ Physical Activities	0.01	0.10	0.03
Level of Occ Creative Thinking	0.29	0.19	0.27
Level of Occ Interpersonal Relationships	0.23	0.13	0.21
Level of Occ Decision Making and Problem Solving	0.17	0.06	0.15
Respondent Ever Smoked Round 1	0.73	0.73	0.73
Less than HS Diploma	0.24	0.30	0.25
Completed HS	0.31	0.32	0.31
Some College	0.20	0.20	0.20

Table 2.3: Eligible Sample Descriptives

	Mean In-Sample	Mean Attrite	T Statistic	P Value
Longest Occ Professional Round 1	0.18	0.11	3.47	0.00
Longest Occ Manager Round 1	0.21	0.23	-1.00	0.32
Longest Occ Clerical Round 1	0.05	0.05	0.10	0.92
Longest Occ Sales Round 1	0.09	0.08	0.50	0.62
Longest Occ Production Round 1	0.24	0.23	0.29	0.77
Longest Occ Service Round 1	0.04	0.05	-1.53	0.13
Longest Occ Operations Round 1	0.16	0.20	-1.79	0.07
Longest Occ Farming Round 1	0.02	0.02	-0.12	0.90
Less than HS Diploma	0.24	0.32	-3.15	0.00
Completed HS	0.31	0.32	-0.23	0.81
Some College	0.20	0.20	-0.10	0.92
Level of General Occ Physical Activities	0.02	0.08	-1.21	0.23
Level of Occ Creative Thinking	0.28	0.24	0.58	0.56
Level of Occ Interpersonal Relationships	0.23	0.14	1.94	0.05
Level of Occ Decision Making and Problem Solving	0.15	0.12	0.50	0.61
Less than HS Diploma	0.24	0.32	-3.15	0.00
Completed HS	0.31	0.32	-0.23	0.81
Some College	0.20	0.20	-0.10	0.92
Completed College Degree	0.25	0.17	3.57	0.00
Obese (BMI Above 30) Round 1	0.20	0.21	-0.19	0.85

Table 2.4: Differences in Eligible vs. Analytic Sample

	(1) Respondent Reports Lots of Physical Activity at Job
Level of General Occ Physical Activities	2.364^{***}
	(11.61)
Observations	1898

Exponentiated coefficients; *t* statistics in parentheses

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 2.5: Relation of O*NET Measures to Respondent Reports

	Obese Physical Activity Only	Non-Obese Physical Activity Only	Obese Physical Activity and Controls	Non-Obese Physical Actvity and Controls	Obese All Covariates	Non-Obese All Covariates	Obese Physical Activity Decision Making	Non-Obese Physical Activity Decision Making
Intercept	0.399†	0.196	-0.034	0.126	-0.273	0.139	-.084	0.190
Slope	-0.017	-0.021	0.057	-0.051	0.057	-0.071†	.034	-0.077*

Table 2.6: Effects of Occupational Physical Activities Across Models

	Decision Making	Creative Thinking	Physical Activity	Interpersonal Relationships
Decision Making		.87	-.58	.68
Creative Thinking	.87		-.59	.64
Physical Activity	-.58	-.59		-.61
Interpersonal Relationships	.68	.64	-.61	

Table 2.7: Correlation of Occupational Activities

2.9 Sensitivity Results

	(1) White Men
Ever Smoked	0.789***
	(7.87)
Less than HS Diploma	0.506***
	(4.84)
Completed HS	0.251*
	(2.36)
Some College	0.296**
	(2.59)
Obese at First Survey Round	0.222**
	(2.75)
Interaction of Obesity and Occupational Physical Activity	0.173^{+}
	(1.89)
Physical Activities 2010 Measure	-0.101^{+}
	(-1.90)
Decision Making Problems 2010 Measure	0.168*
	(2.35)
Interpersonal Relationships 2010 Measure	-0.129*
	(-2.34)
Creative Thinking 2010 Measure	-0.101^{+}
	(-1.68)
Constant	-5.952***
	(-40.08)
gamma	
Constant	0.250***
	(18.66)
Observations	29251

t statistics in parentheses

$^{+}$ $p < 0.10$, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 2.8: Influence of Obesity and Occupational Activities on Mortality–Results from a Gompertz Model

Figure 2.22 Cox Snell Residuals from Gompertz Model

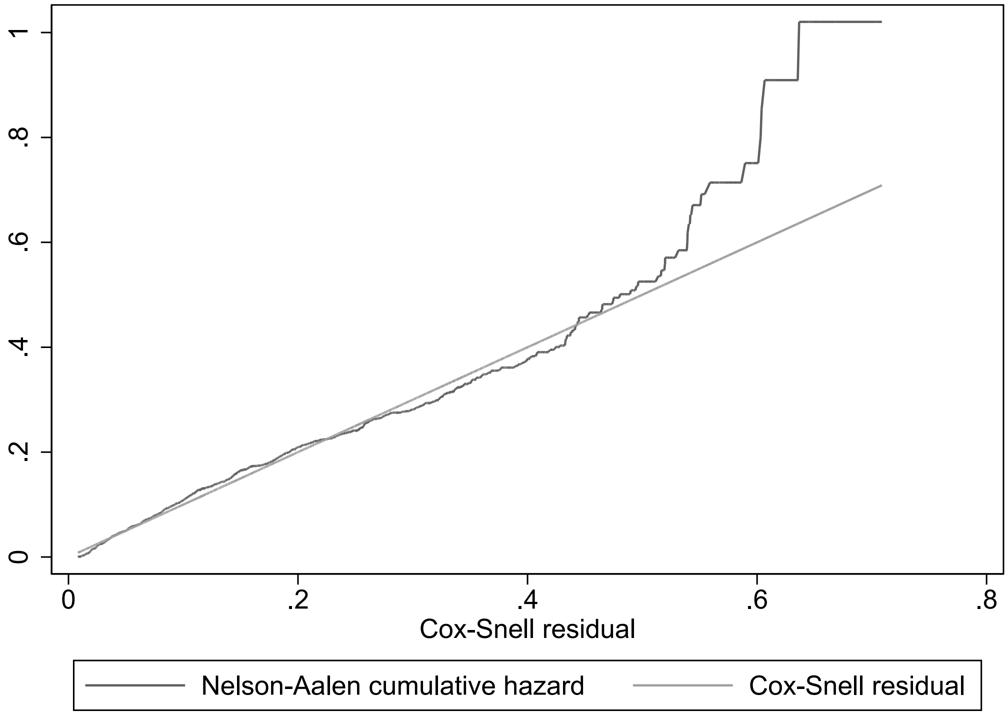


Figure 2.23 Average Number of Limitations with Physical Activity at Mean Controlling for Demographics and Occupational Activities

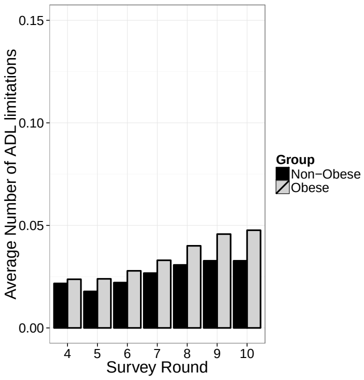


Figure 2.24 Average Number of Limitations with Physical Activity Two Standard Deviations Below the Mean Controlling for Demographics and Setting Other Occupational Activities to One Standard Deviation Above the Mean

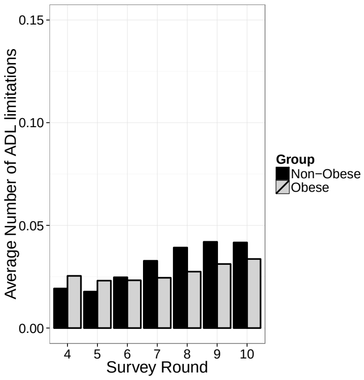


Figure 2.25 Average Number of Limitations with Physical Activity Two Standard Deviations Above the Mean Controlling for Demographics and Setting Other Occupational Activities to One Standard Deviation Below the Mean

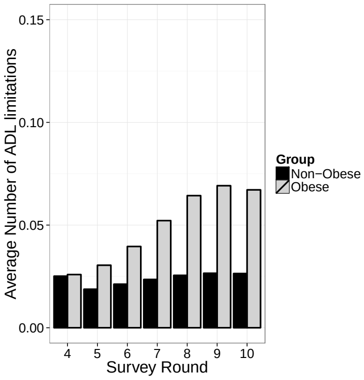
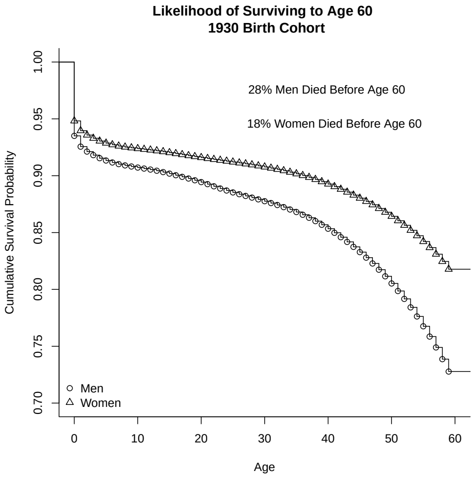


Figure 2.26 Mortality Prior to HRS Study



3 CAN WASTING EXPLAIN THE OBESITY-MORTALITY PARADOX?

Abstract

Recent controversy has emerged regarding the true relation between obesity and mortality at older ages. While epidemiological literature consistently finds that obesity predisposes individuals to develop serious chronic conditions, work in this same literature also shows no significant differences in mortality risk among obese versus non-obese persons. A few studies even suggest that being overweight or slightly obese may be protective, reducing mortality risk. These findings present a puzzle, why are overweight and obese persons more likely to develop serious chronic conditions but less likely to die? In this analysis, I test one explanation for this observed contradiction, that weight loss due to the onset of chronic disease may explain why obese persons appear more likely to get sick but less likely to die. Using data from the Health and Retirement Study, this paper uses multi-state models to test why it is that obese persons appear to be more likely to develop chronic conditions but less likely to die. Findings support that disease wasting may explain the obesity mortality paradox. At any given time, both the non-ill and ill obese have a lower risk of dying compared to their non-obese counterparts. However, across survey rounds, obese persons have a greater likelihood of transitioning to an ill state which carries with it a higher risk of mortality. Once ill, individuals are more likely to become non-obese, and the non-ill non-obese have the greatest mortality risk. Simulation analysis shows that a greater proportion of a simulated non-ill obese cohort would die compared to a cohort of non-ill non-obese. Missing data likely cannot explain these findings and study limitations, such as the failure to measure some chronic diseases associated with obesity and disease wasting, mean that the influence of disease wasting is likely underestimated.

3.1 Introduction

Over the past thirty years, the prevalence of obesity in the United States has risen dramatically. 30 Percent of U.S. adults are obese and two thirds are above a normal body weight (Ogden et al., 2014). The rise in obesity has strong implications for population health. Obesity has been shown to increase the risk for developing a host of chronic diseases, cancers, and musculoskeletal conditions (Field et al., 2001) . Research has linked obesity to an elevated risk of many chronic diseases including diabetes, gallstones, hypertension, heart disease, and stroke (Field et al., 2001). Similarly a body of case-control and cohort studies have found that obesity increases risk for colorectal, breast, endometrial, renal, and oesophagal cancers (Bianchini et al., 2002). Finally, obesity has been found to contribute to musculoskeletal conditions of the hip, knee, ankle, foot and shoulder (Wearing et al., 2006). Not only have these studies found consistent associations between excess body fat and disease risk, but they have also established some of the biological mechanisms through which obesity elevates disease risk and dose-response associations between body fat and disease risk. Together, the body of evidence would suggest a causal relation between excess body fat and disease risk.

However, the relation of obesity to all cause mortality is highly contested. Population based epidemiological studies have found significant disease risks associated with obesity, but studies in the same literature have not found a consistent relation between obesity and mortality. A recent meta-analysis of 100 studies looking at the relation between obesity and mortality found that overweight and class 1 obese populations have a lower relative risk of dying compared to persons at a normal body weight (Flegal et al., 2013). Even among class 2 and class 3 obese, mortality was found to be only marginally higher than in normal weight persons (RR=1.29) (Flegal et al., 2013).

The modest and inconsistent associations between obesity and mortality contrast the strong and enduring associations found between obesity and a host of chronic diseases. The contradiction across these literatures presents a paradox: Why do obese persons get sicker but have a lower risk of dying? If obesity does exert a substantial effect on mortality not captured in current models, then the effect of obesity on the population's life expectancy is substantially under-estimated as are the demographic and economic costs of obesity.

This paper answers this question by using a multi-state model to test whether obese persons are more likely to develop chronic diseases which cause them to lose weight and subsequently die prematurely, what I call the disease wasting hypothesis. The paper consists of four parts. In the first, I review explanations for the obesity-mortality paradox and discuss how disease wasting may explain the incongruity. During part two, I review the data available to test the disease wasting hypothesis and the measures used throughout this study. In section three, I describe the methods and highlight some key findings. Section four concludes and links findings from this study to the broader literature.

3.2 Literature Review

Explanations for the Obesity-Mortality Paradox

Research has struggled to explain the obesity mortality paradox, why the obese appear to be more likely to become chronically ill but less likely to die. It is here important to acknowledge that some people do not view the non-association between obesity and mortality as paradoxical. For example, some work argues that obese persons can be fit but fat. A person who is obese but lives an otherwise healthy, physically active, lifestyle may not have much of an elevated disease or mortality risk (Lee et al., 2009). While this argument can be demonstrated to apply to individuals,it likely does not hold at the population level because relatively few obese persons are physically fit (Duncan, 2010).

Starting from the position that there is an obesity-mortality paradox, I'll review three possible explanations, omitted variables, selection, and flawed measurements before moving to describe the disease wasting hypothesis explored in this paper. All of these explanations might partially account for some of the discrepancy in the obesity, disease, mortality relation, but none of them seems to fully explain the phenomenon.

One explanation for the obesity mortality paradox is that studies fail to account for all relevant factors that may affect both bmi and mortality. Omitted variables that might explain the obesity mortality paradox are those which may reduce bmi but increase mortality risk or vice versa. Smoking is one factor that simultaneously affects bmi and mortality (Willett et al., 2013) and is especially important to consider because more than half of men born in 1920 ever smoked cigarettes and rates of smoking remained high for cohorts born through the 1950s. Smoking can increase the metabolic rate and cause smokers to have a lower body mass index (Chiolero et al., 2008). However, smoking also elevates an individual's risk for developing several chronic diseases and of dying from all causes. This has a key implication for the association of obesity to disease and mortality. As smokers are more likely to be at a normal body weight, studies which look at differences in chronic disease and mortality risk between normal weight and obese persons will tend to under-estimate differences by weight category.

Another possibility is that the apparent obesity-mortality paradox is an artifact of a selection bias. Population surveys may recruit subjects that are not representative of the U.S. population, as they may exclude chronically ill individuals, who also have an elevated risk of dying. As obese individuals are more likely to be chronically ill, the chronically ill obese may disproportionately be the non-responders, and a healthy obese population is being compared to a more diverse population of non-obese persons. This explanation would suggest that study design and respondent selection may account for the paradox. However, while there is some empirical support for this theory (Masters et al., 2013), others have challenged the methods used to generate these findings (Wang, 2014).

Others still have maintained that the weak association of obesity to mortality is due to a flawed measure of obesity, bmi. The body mass index, a common metric used to classify individuals as obese, is only a proxy for an individual's weight status and subsequent mortality risk (Vina et al., 2013). Definitions of obesity which rely strictly on bmi ignore systematic variation in bmi and body fat by race, muscle mass, and age. Past studies have consistently found that Asians have a lower body mass index but a higher body fat composition (Wang et al., 1994). Similarly, body mass index has been shown to be unreliable in athletes often overestimating body fat at a given bmi (Ode et al., 2007). Finally, holding constant body mass index it has been shown that percent body fat increases as individuals age (Gallagher et al., 1996) and thus bmi may have greater implications for older versus younger adults. Because some non-obese persons are classified as obese based on bmi, the observed association between obesity and mortality may be lower than the true association if we were to measure body fat directly.

Given noted limitations of body mass index, an alternative method of measuring bmi has been proposed. Some researchers have found that when bmi trajectories are modeled instead of bmi at a static period, there is a stronger association of body mass index with mortality (Zheng et al., 2013). However a limitation of this approach is that the construction of these trajectories is subjective and modeling trajectories does not allow examination of the factors that may shape weight changes across time.

While it is possible that error in measuring obesity, omitted variables, and selective attrition may partially explain the discrepancy between the expected and observed obesity mortality association, it also possible that at any given age obese persons have a lower risk of dying, but over time, obesity increases mortality risk. This may occur if obesity predisposes individuals to develop chronic illnesses which eventually cause them to lose weight, become non-obese, and die prematurely.

Obesity has been shown to increase the risk for a number of chronic diseases which can in turn cause weight loss, sometimes referred to as cachexia. Caxchexia is sometimes defined as a weight loss of over 10 percent, systematic inflammation, and loss of appetite, and by this classification, is estimated to affect over 2 percent of the population (Tan and Fearon, 2008). However, across disciplines and empirical studies, there is some disagreement about the precise definition of cachexia and the period of time over which people lose weight to receive the diagnosis (Evans et al., 2008).

However, while the population prevalence of disease wasting is relatively low, it can be extremely high among persons with certain chronic diseases. Three diseases in particular are highly related to obesity, often cause cachexia, and significantly elevate mortality risk, chronic obstructive pulmonary disease, chronic kidney disease, and cancer (Tan and Fearon, 2008). Individuals with these conditions are extremely likely to develop disease wasting: 27 percent of patients with COPD, 86 percent of end stage cancer patients, and some 40 percent of patients with chronic kidney disease (being treated with dialysis) (Tan and Fearon, 2008) are estimated to have experienced this weight loss.

Not only is the prevalence of wasting extremely common for these obesity-related diseases, but undergoing extreme weight and muscle loss is also associated with a poorer clinical prognosis. Disease wasting is especially hard to diagnose for the obese and is particularly harmful because it is less likely to be identified until extreme weight loss occurs. Untreated disease wasting substantially reduces life expectancy among patients with any of these conditions (de Mutsert et al., 2008; Anker et al., 1997; Tan and Fearon, 2008) and is especially predictive of a poor cancer prognosis (Tan and Fearon, 2008). The effects of this disease related weight loss for mortality are somewhat difficult to compare across diseases because the definition of wasting is inconsistently defined and poorly measured. Further evidence that disease wasting may explain why obese individuals appear more likely to get sick but less likely to die is evidenced by population studies looking at the relationship between mortality and weight loss. A recent meta analysis of these studies shows that intentional weight loss among the unhealthy obese lowered mortality, but unintended weight loss substantially increased mortality risk (Harrington et al., 2009). Together the clinical and population literatures suggest that weight loss related to disease increases a person's mortality risk.

Study Objective

Prior literature has suggested a consistent association between obesity and disease while finding mixed evidence relating obesity to mortality. However, four propositions have been well established in this literature:

1. Obesity increases the risk for developing a number of chronic diseases.
2. Chronic diseases associated with obesity are also linked to disease wasting.
3. Obesity related chronic diseases and subsequent weight loss substantially increase an individual's mortality risk.
4. Substantial survey attrition may compromise inferences about mortality risk by obesity status.

While these propositions have separately been established in a number of studies, this study proposes to test all four conjectures jointly using a multi-state model shown in Figure 3.3. In doing so, this study hopes to explain why obesity is strongly related to the onset of chronic diseases but only moderately associated with mortality–obese individuals lose weight when they become sick, weakening the observed obesity mortality association.

Using data from the Health and Retirement Study, this study uses a multi-state model to estimate: the effect of obesity on the likelihood that an individual will become chronically ill, the effect of chronic disease on the likelihood that an individual will transition to a different weight category, and finally, the effect of chronic disease and weight loss on mortality. By estimating these parameters jointly, this study will test whether the paradoxical relationship between obesity and mortality can be explained by wasting due to disease.

3.3 Data

Data for this project comes from the Health and Retirement Study (HRS). The HRS began collecting data in 1992 when researchers with the study recruited a nationally representative sample of adults aged 50 and over. Biennially, respondents in the HRS are reinterviewed on topics related to their health, employment, and overall wellbeing. Identical information is collected on the spouses of married respondents. While the HRS contains information on multiple cohorts and spouses of targeted individuals, this analysis uses data only from the main HRS cohort, persons born between 1931-1941, to reduce heterogeneity in age and survey information available. Across analyses, no missing values are imputed. The decision not impute is based on prior speculation that sample selection may be non-random and selective attrition may partially explain the obesity mortality paradox.

Independent and Dependent Variables

Obesity is the key independent variable across analyses. Obesity is measured using bmi according to Center for Disease Control cutpoints. Height and weight used to calculate respondent bmi are self-reported by respondents. [1] A limitation of using self-reported height and weight diet is that these measures are prone to respondent mis-reporting which may be especially acute at younger and older ages (Stommel and Schoenborn, 2009). Because of concerns of misclassification, the multi-state analysis pools individuals into two categories: non-obese ($<$30 kg/m^2) and obese ($\geqslant$30 kg/m^2). An analysis comparing measured height and weight to self-reported height and weight found that among individuals who are obese, close to 95% of obese persons correctly identified themselves as obese (Stommel and Schoenborn, 2009).

Chronic disease is a key mediator that may explain the previously noted paradoxical relation between obesity and mortality. The measure of chronic disease is constructed

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[1] In the 2006 survey round, there was an anthropometric questionnaire where height and weight were directly measured, this could be used as a check on respondent self-reports.

by pooling several diseases to construct what I call an ill state. The diseases included in the ill state are a new reporting of: lung disease, heart disease, stroke, diabetes, or cancer. Because these diseases have persistent consequences for a person's health, I assume that once a person enters the ill state they cannot return to a non-ill state.

Mortality is the final outcome of this study. Information on a person's date of death is available in the form of a tracker file where HRS respondents are identified as deceased based on a probabilistic match made from the death registry reported to the NCHS. For a few cases in the HRS sample, there is contradictory information on whether or not a person is deceased. These cases are dropped from analysis.

Controls

In the HRS, education (less than high school, high school equivalence, some college, or college graduate), smoking history (smoked more than 100 cigarettes prior to the first survey round), and birth year (born before 1935 or on/after 1935) are treated as time invariant factors that may affect both an individual's propensity to become obese and to develop a chronic illness. Given past speculation that smoking may partially explain the obesity-mortality paradox, smoking is particularly important to adjust for in models.

Individuals who identify as African American or hispanic are excluded from this analysis because appropriate bmi cutpoints for obesity have been suggested to vary by race (Rahman and Berenson, 2010; Carroll et al., 2008) and mortality risks differ by greatly by race (Hummer et al., 2004). [2]

Stratification Criteria Given different levels of morbidity and mortality, all models are run separately by gender.

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[2] In an ideal world, I would run race and gender stratified models. I have not done the race stratified analysis yet because of power concerns

3.4 Analytic Strategy

The key contribution of this paper is to test whether the paradoxical relation between obesity and mortality can be more fully explained by examining how obesity may select individuals into both disease and weight loss, both of which carry a higher mortality risk. It also uses the fairly novel approach of not censoring cases when they are not observed at a survey round but rather placing missing cases in distinct states in our multi-state model to allow for non-random attrition and explicitly quantify how much selective missingness may affect results.

The analysis consists of two parts. First, I fit a multi-state model to assess the relation of obesity to mortality longitudinally. Next, to quantify the impact of transitions across states on differential mortality between obese and non-obese persons, I conduct a simulation analysis which assesses how mortality would change depending on the distribution of persons in starting states.

Multi-State Model

Using a multi-state model, I estimate transition probabilities across seven states as shown in Figure 3.3. The seven discrete, mutually exclusive states are:

1. Non-Ill/Non-Obese–No Chronic Disease (no lung disease, heart disease, stroke, diabetes, or cancer), BMI Below 30
2. Non-Ill/Obese –No Chronic Disease (no lung disease, heart disease, stroke, diabetes, or cancer), BMI Above 30
3. Ill/Non-Obese–Chronic Disease (lung disease, heart disease, stroke, diabetes, or cancer), BMI Below 30
4. Ill/Obese–Chronic Disease (lung disease, heart disease, stroke, diabetes, or cancer), BMI Above 30

5. Missing, Previously Non-Ill–Previously had no chronic disease (no lung disease, heart disease, stroke, diabetes, or cancer) (been in states 1 or 2) in survey round prior to missing data.
6. Missing, Previously Ill–Previously had chronic disease (lung disease, heart disease, stroke, diabetes, or cancer) (been in states 3 or 4) in survey round prior to missing data.
7. Deceased

In the HRS, people may be observed in any state, but missing (previously non-ill/previously ill) or deceased at the first survey round. Respondents are dropped from the analysis if at the first survey round, they had missing values on one of the variables used to classify people into these states because it is unclear whether they were ill or non-ill prior to when the person did not provide a response. Non-ill individuals are allowed to transition between any of the seven states. Once ill, individuals are allowed to transfer across ill and missing states, but are not permitted to return to the non-ill state because of the enduring health consequences of prior diagnosis with a serious chronic disease. Transitions into a deceased state are absorbing, assuming perfect matching from the probabilistic match with mortality in the NCHS. Individuals with missing data rounds between survey rounds 2 and 10 are not dropped from the sample because the missing state is substantively meaningful to investigate. While we are unable to say what respondent health and obesity experiences were at rounds when they had missing data, if the obese were more likely to become missing, this might support prior hypotheses that the obese population is less representative than the non-obese population, thereby explaining the obesity mortality paradox.

The model fit is a Markov continuous time model which assumes that the probability of transitioning across states is independent on time spent in a current state. The transition intensity matrix Q(t) with (r,s) entry given by Algorithm 5 and individual transition probabilities dictated by Algorithm 6.

Algorithm 5 Transition Intensity Matrix Continuous Time Markov Model

$$\mu_{rs}(t) = \lim_{\delta t \downarrow 0} \frac{P\{X(+\delta t)=s|X(t)=r\}}{\delta t}$$

Notation taken from (Titman and Sharples, 2010)

Algorithm 6 Transition Probability Continuous Time Markov Model

$$p_{rs}(u, t+u) = P\{X(t+u) = s|X(u) = r\}$$

Notation taken from (Titman and Sharples, 2010)

The key parameters in this model are the expected probabilities of transitioning across states. Specifically, if the obesity-mortality paradox that other researchers have observed is operating in this data, we should expect that for at least some time periods, the healthy obese have a lower risk of dying than the non-obese. However, if disease wasting is operating, we should expect that over time the probability of transitioning to a non-obese ill state is going to be higher compared to transitions into an obese-ill state. For this disease wasting to be explaining the obesity mortality-paradox, the probability of dying for those previously in an ill-non-obese state should be higher compared to the probability of dying for those previously in the non-ill obese state.

A complication in this analysis is the possibility of selective attrition that may differ across the obese and non-obese. If attrition is downwardly biasing association between obesity and mortality, we should expect that the obese are more likely to transition to one of the missing states and these missing states are accompanied by a higher risk of mortality.

<u>Simulations</u> Because of the complexity in interpreting the results from the multi-state model and quantifying how the initial distribution of persons across states may affect mortality, this paper uses simulations to demonstrate how transitions across states affects the probability of dying over the 10 survey rounds of HRS data (spanning a roughly 18 year period from 1992 to 2010). To do this I estimate the proportion of the sample that is expected to die over the follow-up period given transition matrices estimated from the real HRS cohort but modifying the distribution of persons across starting states. Specifically, the following artificial cohorts are constructed:

1. People are assigned to starting state of non-ill non-obese.
2. People are assigned to starting state of non-ill obese.
3. People are assigned to a starting state of non-obese with 40% starting in a ill state and 60% starting in an non-ill state, the ratio of ill to non-ill among the non-obese.
4. People are assigned to a starting state of obese with 60% starting in a ill state and 40% starting in an non-ill state, the ratio of ill to non-ill among the obese.

Varying the starting distribution of people across states allows me to show the extent of transitions across states and the extent to which changing the distribution of health and obesity around age 50 may affect later life mortality. Key outcomes in these simulation scenarios are the proportion of the cohort expected to die by the tenth and final survey round. To account for some uncertainty in the transition probabilities, this analysis additionally constructs pseudo confidence intervals that reflect the mean state occupancy across 100 simulations and the minimum and maximum number of people occupying states across survey rounds. [3]

3.5 Results

Descriptive Results

Table 3.3 shows the characteristics of the analytic sample at baseline. Consistent with our expectations for this birth cohort, substantially more men than women smoked, 75% of men smoked compared to only 55% of women and more men completed college 24% compared to 16 %İhe prevalence of chronic diseases was also higher for men at baseline with 31% of men having a chronic illness versus 26% of women. Rates of obesity are comparable across genders, about 19% of both men and women are obese at the first survey round.

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[3] Simulation results of course assume that the transition probabilities across states would remain the same if the distribution of starting states was to be altered. This may be a a somewhat heroic assumption.

Table 3.4 shows descriptive statistics stratifying by both gender and obesity status. Again similar trends across genders remain; however we also observe that compared to their same gender counterparts obese persons are less likely to have ever smoked, are more likely to have a chronic condition, and are on average less educated.

The total state transitions for men and women are shown in Tables 3.1 and **??**. A few key findings should be taken from these tables. First, although this is a sample of older adults, the majority of transitions observed are from a healthy non-obese state back to a healthy non-obese state. Second, more men and women die from an ill state than from a non-ill state. Third and finally, for both men and women, relatively more transitions occur from the ill-obese state to the non-obese ill state. This final pattern is consistent with theories of disease wasting which suggest that individuals lose weight following the onset of a chronic illness.

Multi State Models

The estimated transition matrix for men and women are presented in Tables 3.5 and 3.6. In both of these transition matrices, results are striking similar. The probability of a non-ill non-obese person dying, at any given survey round, is higher than the probability of a non-ill obese person. Men have a 1.58% chance of dying if they are non-ill/non-obese and a 1.51% chance of dying if they are non-ill obese. women have a .91% chance of dying if they are non-ill/non-obese and a .87% chance of dying if they are non-ill obese. While the probability of ill persons (both obese and non-obese) dying is higher than the non-ill, again the ill non-obese have a higher probability of dying compared to the ill obese. For men, the probability of dying from an ill/non-obese state is 6.10% and for non-obese men, the probability of dying from an ill/obese state is 4.85%$\dot{\text{F}}$or women, the probability of dying from an ill/non-obese states is 4.53%

Although individuals have a greater likelihood of dying while in a non-obese state compared to obese persons of comparable health, obese men and women are both more likely to transition to an ill state if they were previously in the obese/non-ill state. 8.98% of non-obese non-ill men may be expected to transition to an ill state compared to 11.89% of obese non-ill men. 6.05% of non-obese non-ill women would be expected to transition to an ill stat compared to 9.36% of obese non-ill women.

Both men and women did have a substantial risk of not being interviewed in a survey panel. The relative likelihood of having missing data by weight status does differ slightly by gender. Non-obese men have a greater risk of having missing data than obese men, the probability of moving into a missing state after previously being non-ill is 5.60% for non-obese men and and 4.76% for obese men. The probability of moving into a missing state after previously being ill is 4.22% for non-obese men and 3.83% for obese men. For women, the obese have a greater risk of having missing data compared to the non-obese. 5.38% of non-obese women are expected to move into a missing state after previously being non-ill compared to 5.90% of obese women. After having previously been ill, non-obese women have a 4.49% probability of moving into a missing state compared to 5.30% for obese women. Although distinguishing missing cases by prior health is computationally more demanding and substantively more complex, the transition matrix shows that this distinction is important for a study of mortality. For men the probability of dying from a missing previously non-ill state is 1.57% and it increases to 4.63% for men in the missing previously ill state. Women in the missing previously non-ill state have a 1.19% chance of dying, and women in the previously ill state have a 3.27% chance of dying.

Finally, Tables 3.7 and 3.8 show the overall model fit by comparing the observed versus model predicted percent of the sample in each state at each time period. Overall, both models fit the data relatively well although there is some evidence that mortality is underpredicted at early survey rounds. Alternative model specifications explored, including a semi-Markov specification where the probability of transitioning across states was conditional on the time spent in a current state and a piecewise model where all transitions were allowed to change at particular survey rounds, did not yield substantially improved fit. [4]

Simulations

While results from these transition matrices are suggestive, simulations can help us understand the substantive significance of these transition probabilities across survey rounds.

Tables 3.9 and 3.10 show the number of deaths by the 10th survey round for men and women respectively. Comparing scenarios 1 and 2, where all persons are set to be non-ill but obesity status is varied. Comparing these scenarios we see that the expected number of death is higher for both genders in scenario 2 where all persons are obese compared to scenario 1. 2157 men are expected to die when all start as non-ill/non-obese compared to 2246 when all start as non-ill/obese. While mortality is lower for women, differences across these scenarios are similar. 1381 women are expected to die when starting as non-ill/non-obese compared to 1525 starting as non-ill/obese. These results suggest that over the surveyed period, the slightly lower mortality risk of the non-ill/obese is offset by their greater likelihood of moving into an ill state. For both genders, mortality risk is substantially higher when in scenarios 4 and 5 where all persons are obese but the distribution of health is set to what is observed for each weight status at baseline. As expected, given that a fairly high proportion of the sample now occupies an ill state, 40% in scenario 3 and 60% in scenario 4, mortality for both genders is substantially higher compared to scenarios 1 and 2. For both genders, scenario 4, where all persons are obese carries with it the highest risk of mortality, 3177 men per 10,000 and 2573 women per 10,000 are expected to die by the 10th survey round. Overall, these simulations suggest that across survey rounds, cohorts with more obese persons have higher mortality. [5]

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[4] In this section, I do not talk about the influence of particular covariates on the transition probabilities. It is interesting to note that no covariates have a statistically significant impact on any transition probability (for men and women). However, in an additional analysis, I ran a multinomial logistic regression predicting starting state which showed that all of the influence of these covariates is in predicting the health and weight status of a person at the first survey round

[5] I ran some additional simulation runs where I distributed everyone to obese and non-obese persons equally to the ill and non-ill states. It is interesting to note that in these scenarios the mortality risk was higher

3.6 Discussion and Limitations

Findings derived from the multi-state model would suggest that disease wasting hypothesis may explain the obesity-mortality paradox previously noted in other literature.

Although a survey attrition has been suggested as a competing explanation for the obesity-mortality paradox, I find little evidence to support this hypothesis. In explicitly modelling transitions to missing states, I find little evidence to suggest that the missingness is systematically biasing the estimates of mortality. Similar rates of missingness were observed among obese and non-obese persons and among the ill and non-ill sub-groups.

Instead this study finds empirical support for the following (See Tables 3.5 and 3.6 for review):

1. At any given period, obese individuals have a lower risk of dying compared to their non-obese counterparts.
2. Over time, obese non-ill are more likely to become ill compared to their non-obese counterparts.
3. Once ill, individuals are more likely to become non-obese than they are obese.
4. Chronically ill non-obese persons have the greatest mortality risk.

Simulation analyses demonstrate that the obese are more likely than non-obese persons to transition to a chronically ill state, and the onset of chronic illness appears associated with a transition into a non-obese state that carries with it a higher mortality risk.

While these findings are suggestive, a few limitations in this approach should be noted. First, while disease wasting is commonly defined as a change in weight (as well as tissue composition), this study proxies the wasting process as a move from an obese to a non-obese

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for the non-obese, driven by the high mortality for the non-obese/ill state. However, given that we should expect a higher disease risk for the obese, I opted not to show these results. It is possible that the disease risk for obese and non-obese groups could equalize if health within the obese category is heterogeneous and the frail obese die out, then the robust obese group may be relatively less likely to develop chronic illness or die. I don't have the data for formulating this type of cross-over model, but it's an interesting idea.

state. As a consequence, this study may miss wasting processes that occur within the obese and non-obese categories. Additionally, this study assumes that weight loss in a chronically ill state is due to disease wasting and indicative of greater frailty. However, if people intentionally lose weight to help manage a chronic disease, as might be recommended for persons with cardiovascular disease or diabetes, then some moves from an ill obese state to an ill non-obese state might be protective. Future models could account for these transitions by conditioning on whether a person intentionally lost weight.

Given that there is a two year period between survey interviews, this study may also fail to capture some state transitions that occur due to aggressive diseases. Individuals with aggressive diseases may transition from an obese to non-obese state and subsequently die in a matter of months, not years. This limitation likely causes this study to underestimate the extent to which disease wasting leads to an underestimate of the obesity-mortality association.

Similarly, the measure used to capture chronic disease is not comprehensive and relies on a respondent reporting that they have been diagnosed with a condition. If such conditions are associated with weight loss and higher mortality, then we might be over-estimating mortality in the non-ill states and failing to capture transitions to ill states with a higher risk of mortality.

Despite these limitations in measurement and follow-up, findings from this study would suggest that the obesity mortality "paradox" is in fact an artifact of limited follow-up of individuals. If one were to look at a particular point in time and adjust for a person's health state, it would appear that the obese have a lower risk of dying; however, in fitting a multi-state model and expanding analysis to 10 waves of data, we can see that obesity pre-disposes individuals to enter health states that have a higher risk of mortality. Findings from this study illustrate the importance of taking a life course approach to the study of health and the social factors that influence health and mortality risks.

3.7 Tables and Figures

Transition	To: Non-Ill Non-Obese	Non-Ill Obese	Ill Non-Obese	Ill Obese	Missing Pre. Non-Ill	Missing Pre. Ill	Deceased
From: Non-Ill Non-Obese	7585	474	870	49	627	0	135
Non-Ill Obese	304	1778	73	286	144	0	33
Ill Non-Obese	0	0	6732	545	0	404	521
Ill Obese	0	0	487	2788	0	159	179
Missing Pre. Non-Ill	231	55	63	26	2024	0	37
Missing Pre. Ill	0	0	145	84	0	1068	63

Table 3.1: Transitions HRS White Males

Transition	To: Non-Ill Non-Obese	Non-Ill Obese	Ill Non-Obese	Ill Obese	Missing Pre. Non-Ill	Missing Pre. Ill	Deceased
From: Non-Ill Non-Obese	10067	495	747	45	743	0	96
Non-Ill Obese	346	2104	57	267	205	0	20
Ill Non-Obese	0	0	6741	478	0	420	374
Ill Obese	0	0	395	2827	0	227	165
Missing Pre. Non-Ill	272	101	62	39	2356	0	32
Missing Pre. Ill	0	0	182	156	0	1102	49

Table 3.2: Transitions HRS White Females

	Female		Male		Overall			
	Mean	SD	Mean	SD	Mean	SD	Min	Max
Less than HS Diploma	0.229	0.420	0.260	0.438	0.244	0.429	0.0	1.0
Completed HS	0.392	0.488	0.304	0.460	0.350	0.477	0.0	1.0
Some College	0.215	0.411	0.200	0.400	0.208	0.406	0.0	1.0
Completed College Degree	0.164	0.371	0.237	0.425	0.199	0.399	0.0	1.0
Ever Smoked Round 1	0.553	0.497	0.747	0.435	0.645	0.478	0.0	1.0
Obese (BMI At or Above 30)	0.188	0.390	0.190	0.392	0.189	0.391	0.0	1.0

Table 3.3: Descriptive Statistics by Gender

	Female Non-Obese		Female Obese		Male Non-Obese		Male Obese		Overall			
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Min	Max
Less than HS Diploma	0.221	0.415	0.273	0.446	0.251	0.434	0.279	0.449	0.244	0.429	0.0	1.0
Completed HS	0.394	0.489	0.417	0.493	0.301	0.459	0.327	0.469	0.350	0.477	0.0	1.0
Some College	0.216	0.412	0.198	0.399	0.198	0.398	0.207	0.405	0.208	0.406	0.0	1.0
Completed College Degree	0.169	0.375	0.111	0.314	0.250	0.433	0.187	0.390	0.199	0.399	0.0	1.0
Ever Smoked Round 1	0.562	0.496	0.516	0.500	0.750	0.433	0.734	0.442	0.645	0.478	0.0	1.0
Obese (BMI At or Above 30)	0.260	0.439	0.364	0.481	0.314	0.464	0.408	0.492	0.189	0.391	0.0	1.0

Table 3.4: Descriptive Statistics by Gender and Obesity

	Non-Ill/ Non-Obese	Non-Ill/ Obese	Ill/ Non-Obese	Ill/ Obese	Missing– Prev. Non-Ill	Missing– Prev. Ill	Deceased
Non-Ill/Non-Obese	0.8027	0.0388	0.0765	0.0090	0.0553	0.0020	0.0158
Non-Ill/Obese	0.0935	0.7224	0.0324	0.0865	0.0476	0.0027	0.0151
Ill/Non-Obese	0.0000	0.0000	0.8400	0.0568	0.0000	0.0422	0.0610
Ill/Obese	0.0000	0.0000	0.1147	0.7986	0.0000	0.0383	0.0485
Missing– Previously Non-Ill	0.0811	0.0199	0.0264	0.0109	0.8452	0.0008	0.0157
Missing– Previously Ill	0.0000	0.0000	0.0938	0.0532	0.0000	0.8068	0.0463
Deceased	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	1.0000

Table 3.5: Transition Matrix Men

	Non-Ill/ Non-Obese	Non-Ill/ Obese	Ill/ Non-Obese	Ill/ Obese	Missing– Prev. Non-Ill	Missing– Prev. Ill	Deceased
Non-Ill/Non-Obese	0.8412	0.0340	0.0541	0.0064	0.0538	0.0015	0.0091
Non-Ill/Obese	0.0966	0.7394	0.0222	0.0714	0.0590	0.0028	0.0087
Ill/Non-Obese	0.0000	0.0000	0.8572	0.0526	0.0000	0.0449	0.0453
Ill/Obese	0.0000	0.0000	0.0958	0.8071	0.0000	0.0530	0.0442
Missing– Previously Non-Ill	0.0832	0.0299	0.0216	0.0129	0.8403	0.0009	0.0112
Missing– Previously Ill	0.0000	0.0000	0.1059	0.0879	0.0000	0.7735	0.0327
Deceased	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	1.0000

Table 3.6: Transition Matrix Women

	Non-Ill/ Non-Obese	Non-Ill/ Obese	Ill/ Non-Obese	Ill/ Obese	Missing– Previously Non-Ill	Missing– Previously Ill	Deceased
Observed	46.3043	11.7918	23.6411	8.5994	5.5795	2.2721	1.8119
Observed	40.0633	10.6126	25.0503	9.2321	7.4777	2.9623	4.6017
Observed	34.0811	9.606	25.9994	10.9865	8.4843	4.0552	6.7875
Observed	28.9042	8.6281	26.4596	11.5329	9.4334	5.0618	9.9799
Observed	24.1012	7.1038	27.61	13.1723	9.5197	5.0906	13.4024
Observed	19.8735	5.7521	28.4153	13.4599	9.9799	6.1835	16.3359
Observed	16.5085	5.407	28.3578	14.0926	9.6347	6.4136	19.5858
Observed	13.6037	4.084	27.5237	14.6678	9.9511	7.0751	23.0946
Observed	10.0949	3.365	27.6675	12.9134	10.325	7.7941	27.8401
Model Predicted	46.6443	11.0889	24.8555	9.3959	3.7156	1.4266	2.8733
Model Predicted	38.7719	9.892	26.1161	10.414	6.2399	2.689	5.8771
Model Predicted	32.5469	8.7725	26.835	11.2205	7.8796	3.7823	8.9632
Model Predicted	27.5793	7.7553	27.1643	11.8294	8.8671	4.7112	12.0934
Model Predicted	23.5775	6.8475	27.2106	12.2623	9.3784	5.4858	15.238
Model Predicted	20.3225	6.0469	27.0495	12.543	9.546	6.1189	18.3732
Model Predicted	17.6489	5.3458	26.735	12.6948	9.4698	6.625	21.4806
Model Predicted	15.4315	4.7341	26.3064	12.7394	9.2247	7.0184	24.5455
Model Predicted	13.5749	4.2015	25.7923	12.6957	8.8663	7.313	27.5563

Table 3.7: Observed versus Predicted Men

	Non-Ill/ Non-Obese	Non-Ill/ Obese	Ill/ Non-Obese	Ill/ Obese	Missing– Previously Non-Ill	Missing– Previously Ill	Deceased
Observed	48.9488	11.2668	22.4259	8.0593	5.876	2.2642	1.159
Observed	44.1779	10.5391	22.8571	8.9757	7.6011	3.504	2.345
Observed	39.7844	9.4879	23.558	10.0539	8.8949	4.2588	3.9623
Observed	35.5795	8.4906	23.3154	10.9434	10.0809	5.6873	5.903
Observed	31.3477	7.6011	24.3396	11.5364	10.8625	5.8221	8.4906
Observed	27.1429	7.2237	25.6873	12.6954	11.3208	5.876	10.0539
Observed	23.1267	7.062	26.0377	13.4771	11.4286	6.1725	12.6954
Observed	20.9434	6.1186	26.5768	13.504	11.0782	6.5499	15.2291
Observed	16.9542	4.9865	25.7951	13.504	11.9137	7.0081	19.8383
Model Predicted	49.7021	11.6013	22.3233	9.0343	3.8692	1.5096	1.9603
Model Predicted	43.2418	10.3806	23.166	9.8211	6.6099	2.7613	4.0193
Model Predicted	37.919	9.3407	23.7795	10.5158	8.4931	3.7979	6.1539
Model Predicted	33.4989	8.4475	24.2119	11.1146	9.7278	4.6545	8.3449
Model Predicted	29.7981	7.6739	24.4987	11.6188	10.4747	5.3599	10.5759
Model Predicted	26.6732	6.9986	24.6668	12.0326	10.8577	5.9379	12.8333
Model Predicted	24.0118	6.4047	24.7362	12.3622	10.9715	6.4079	15.1056
Model Predicted	21.7256	5.8787	24.7228	12.6146	10.889	6.7864	17.383
Model Predicted	19.7453	5.4097	24.6387	12.797	10.6657	7.0868	19.6569

Table 3.8: Observed versus Predicted Women

Min	Mean	Max	Scenario
2049	2157.38	2223	Scenario 1
2113	2245.84	2319	Scenario 2
2783	2925.51	3004	Scenario 3
3068	3176.64	3285	Scenario 4
2646	2736.26	2827	Scenario 5

Table 3.9: Results Across a 100 Simulations: Number of Deaths Per 10,000 by 10th Survey Round–Men

Min	Mean	Max	Scenario
1266	1381.31	1523	Scenario 1
1436	1524.81	1618	Scenario 2
2014	2138.91	2216	Scenario 3
2491	2572.55	2675	Scenario 4
1897	1981.02	2059	Scenario 5

Table 3.10: Results Across a 100 Simulations: Number of Deaths Per 10,000 by 10th Survey Round–Women

Figure 3.1 Multi-State Model

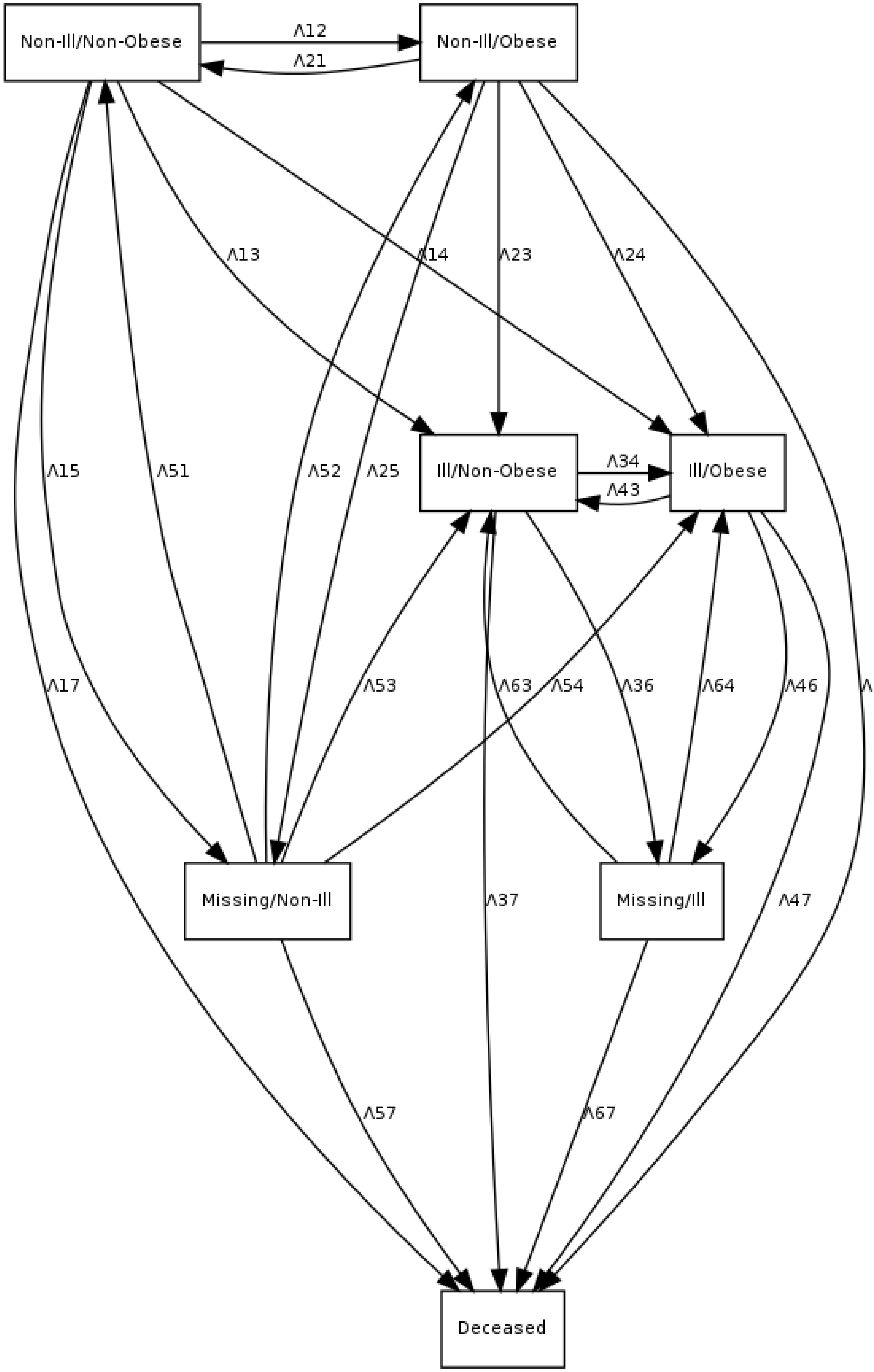


Figure 3.2 Full Simulation Results–Men

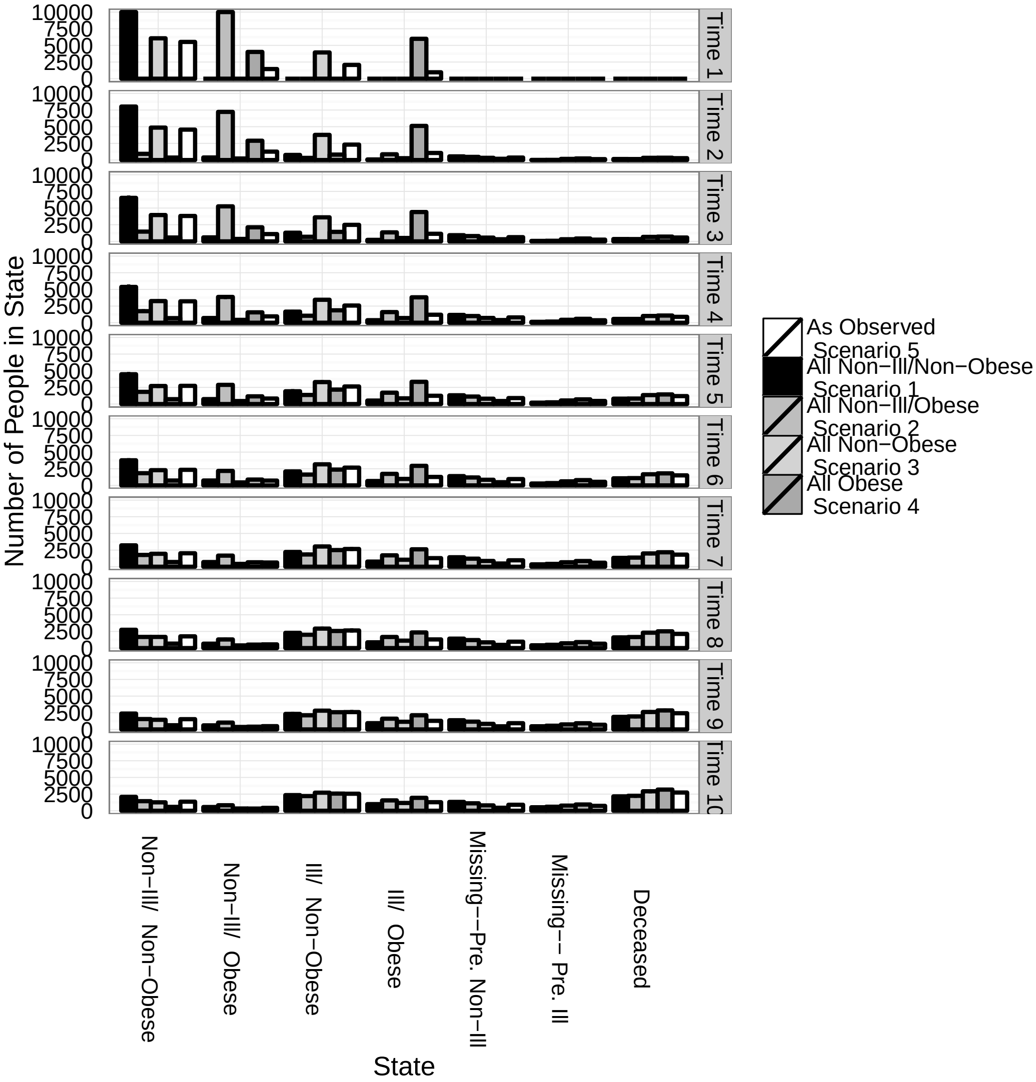
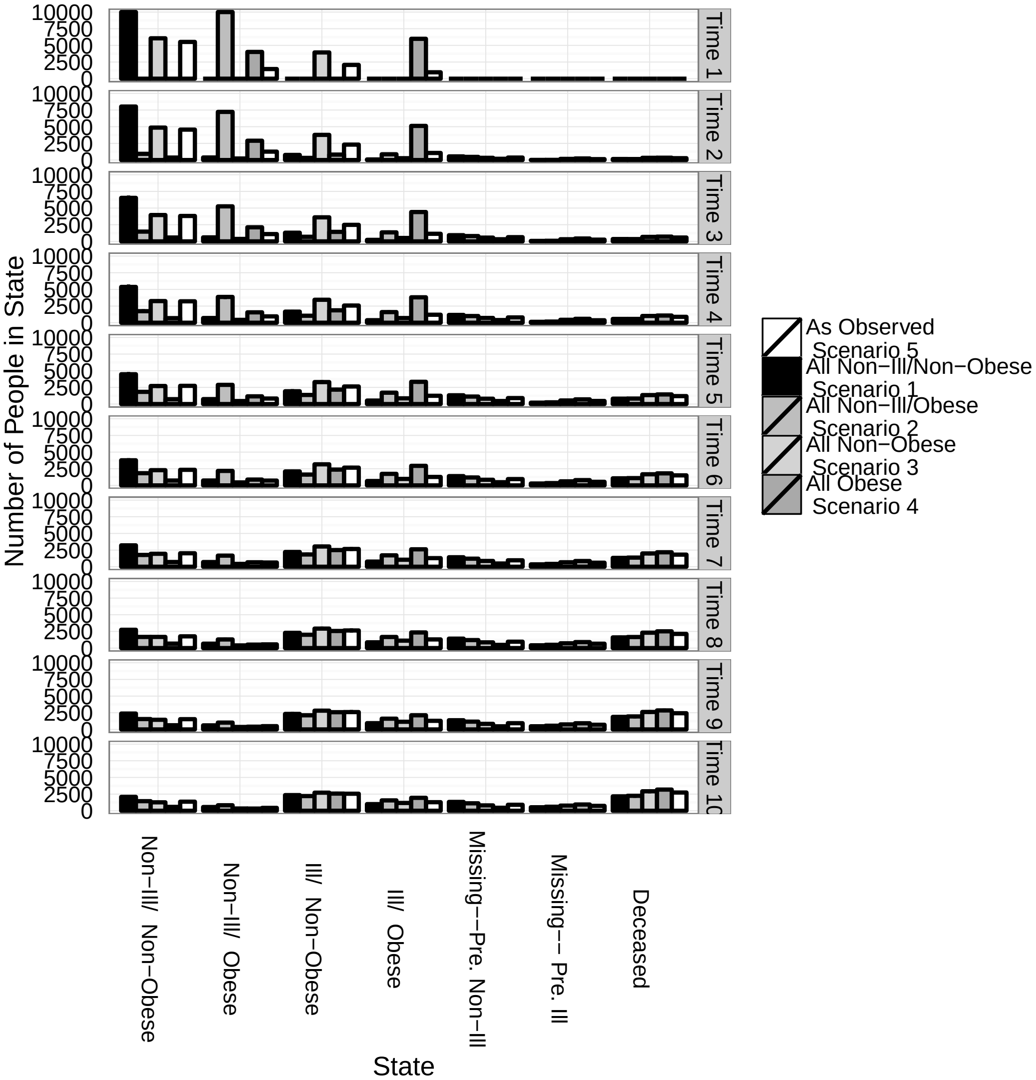


Figure 3.3 Full Simulation Results–Women



3.8 Conclusion

This dissertation has presented three analyses examining the health implications of obesity across the life course. Results presented confirm consistent associations of obesity with adverse health outcomes at young, middle, and older ages. Although there is some evidence that obese persons are disadvantaged on other dimensions, selection into obesity does not appear to explain the health disadvantages obese persons experience.

In chapter 1, I show that obesity in early adulthood is associated with a greater risk of developing a disabling condition that forces a person out of the labor force. The association of obesity to work disability remains robust to the inclusion of factors that increase a person's risk of becoming obese. However, the absolute impact of obesity on the probability that a person becomes work disabled is largest for African Americans suggesting that obesity may exacerbate other disadvantages for this group. In chapter 2, I show that the health disadvantages associated with obesity extend into retirement. Obese persons working in physically demanding occupations are more likely to develop limitations in activities of daily living and ultimately die compared to their non-obese counterparts. Given that physically demanding jobs are generally lower on the social hierarchy, this finding again suggests that obesity may perpetuate health disadvantage among persons of lower social standing. In chapter 3, I show that the appearance of a mortality advantage for obese persons at older ages is an artifact of studies failing to account for weight loss often associated with the onset of serious chronic disease.

While this study cannot examine the implications of obesity over the complete life course nor how the first onset of obesity may influence life time health implications, findings from this paper do suggest that the health consequences of obesity accumulate as people age, cannot be explained by selection forces, and exacerbate existing health inequalities. As the prevalence of obesity has dramatically risen, the negative enduring consequences of obesity will be salient for a greater share of the United States population. These trends in obesity point to a pressing need to understand the implications of obesity for health over the life course, how the severity of obesity may influence health risks, and how timing and duration spent obese may play a role in determining health outcomes.

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