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THE "WEIGHT" OF SOCIO-ECONOMIC STATUS, RACE/ETHNICITY, AND GENDER: A SYSTEMATIC EXAMINATION OF OBESITY AND ITS CO-MORBIDITIES

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THE "WEIGHT" OF SOCIO-ECONOMIC STATUS, RACE/ETHNICITY, AND GENDER: A SYSTEMATIC EXAMINATION OF OBESITY AND ITS CO-MORBIDITIES

Dissertation

A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy in the
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at the University of Kentucky

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ABSTRACT OF THE DISSERTATION

THE "WEIGHT" OF SOCIO-ECONOMIC STATUS, RACE/ETHNICITY, AND GENDER: A SYSTEMATIC EXAMINATION OF OBESITY AND ITS CO-MORBIDITIES

Although extensive research exists on the association between SES and obesity and its patterning across separate gender and racial/ethnic groups, critical gaps remain. In particular, the majority of studies on the SES-BMI association have examined it in additive models without simultaneously considering the influence of gender and race/ethnicity. An additional limitation of the current obesity scholarship concerns the lack of scholarship addressing the interplay between social factors, such as SES, race/ethnicity, gender, and proximate health risk factors, such as BMI, in shaping obesity-related chronic health outcomes, especially considering that health outcomes may vary in the extent to which they may be controlled by individual behavior.

By utilizing the 2011-2014 cycle of the National Health and Nutrition Examination Survey (NHANES) (N=19,931), this dissertation addresses key gaps in the literature on the social patterning of obesity and certain obesity co-morbidities – arthritis and indicators of cardiovascular health, namely blood pressure, plasma fasting glucose and HDL cholesterol – across diverse social groups. This research project has been situated in the fundamental social causes of disease (FSCD) theoretical framework, which situates individual risk factors within the larger socio-cultural structures, making this broader context the key mechanism explaining disparities in health outcomes.

Research findings uncovered stark gendered racial disparities in overweight and obesity that were not attenuated by high income and education, placing Black American women at an increased risk of having a higher BMI. In addition, results reveal that the influence of different indicators of social status vary across health outcomes. Specifically, social factors did not modify the BMI-arthritis relationship, while the effect of BMI on blood pressure was amplified for Hispanic adults. Further, with respect to fasting plasma glucose and HDL cholesterol, the negative effect of BMI was amplified for White adults. In addition, the effect of BMI on HDL cholesterol was also amplified for individuals with higher educational attainment and household income. Overall, the results from this dissertation provide novel insights on the ways distal social factors interact

simultaneously with each other and with more proximal health risk factors to produce variations in individual weight and weight-related health disparities.

KEYWORKDS: Obesity, Obesity Co-morbidities, Socioeconomic Status (SES), Race/Ethnicity, Gender, Fundamental Social Causes of Disease (FSCD)

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CHAPTER I: INTRODUCTION

The prevalence of overweight and obesity has almost doubled in the past 30 years in the United States (Flegal et al. 2012). While the association between excess body weight and illness has been known for decades, the alarming increase in the upper tail of the distribution has been labeled an epidemic and was declared a major public health issue in the early 2000s. In 2011-2012, more than two thirds of American adults were either overweight or obese, and 6.4% were extremely obese.

It is also important to note that while all segments of the U.S. population have been affected by the increasing rates of obesity, one of the common over-generalizations in the obesity research is that variation in body weight is to a large degree attributable to individual socio-economic status (SES), i.e. household income, wealth, and educational attainment. Such simplification of the complex relationship between social status and the risk of obesity is problematic for two reasons. First, the association between overweight/obesity and SES varies by gender, race/ethnicity, and age (Clarke et al. 2009; McLaren 2007; Wang and Beydoun 2007; Zhang and Wang 2004b). Secondly, the SES-obesity gap has been narrowing and weakening over time, particularly among women (Clarke et al. 2009; Zhang and Wang 2004a; Zhang and Lauderdale 2005). The shifting patters in body weight may suggest that, to some extent, increasing obesity rates could be attributed to individual health behaviors, however, the continuous lower weight advantage among White women and men (lower BMI levels relative to racial minorities) suggest that larger social forces are at play.

While the relationship between different obesity categories and negative health outcomes, including all-cause mortality, has been recently problematized by medical

sociologists and social epidemiologists, social disparities of obesity are cause for concern. Mainly, because higher body mass has been linked to lower self-esteem, poor body image, eating disorders, depression, and psychological distress as a consequence of weight discrimination (Faith, Matz, and Jorge 2002; Graham and Felton 2005; Luppino et al. 2010; Stunkard, Faith, and Allison 2003). Thus, increased risk of obesity among more disadvantaged social groups would indicate a relative worsening of physical health and quality of life.

Although extensive research exists on the association between SES and obesity and its patterning across separate gender and racial/ethnic groups, critical gaps remain. In particular, the majority of studies on the SES-BMI association either focus on gender or racial differences – ignoring SES – or examine SES inequality without simultaneously considering gender and race/ethnicity. Additionally, the systematic analyses that do exist, are either outdated or limited by a lack of nationally representative data. As a result, we know little about more recent, national trends of the relationship between socio-economic status and obesity among diverse gender and racial/ethnic groups. Given such limitations, it is difficult to make generalized statements about the combined impact of gender, race/ethnicity, and socio-economic inequality on disparities in body weight.

A second critical gap concerns the relatively small amount of research addressing the role of obesity in shaping chronic health outcomes among different SES, racial/ethnic, and gender groups. As an example, existing research suggests that diabetic or cardiovascular health problems, such as high blood pressure or high blood glucose levels (with obesity being the main "biological" explanatory mechanism), are generally greater among racial and ethnic minorities and individuals of lower SES. However, it is less

clear how social factors (e.g. SES, race/ethnicity, gender) interplay with proximate factors (e.g. BMI) in shaping chronic health outcomes. That is, does increase in BMI equally affect the chronic health outcomes among different social groups? Because few studies have directly examined health risk accumulation across different social groups, our knowledge is limited on how distal causes of health status may condition the effects of more proximate health risk factors (Shafer and Ferraro 2011). Additionally, it is unknown if the interaction between distal and proximal health risks varies across different health outcomes. Thus, more research is needed to assess if and to what extent gender, race/ethnicity, and SES amplify or weaken the effects of obesity on diverse health outcomes.

In this dissertation, I contend that – aside from demonstrating the SES gradient in body weight and chronic illnesses – it is time to pose and explain theoretical models that address the multiple dimensions of social and economic advantage and disadvantage and the ways they produce disparities in obesity and its health consequences. In order to address limitations in the existing literature, the proposed research will be situated in the fundamental social causes of disease (FSCD) theoretical framework. The essential claim of FSCD is that the negative relationship between SES and health persist because it involves access to resources that can be utilized to avoid individual risk factors. Resources include knowledge, money, power, prestige, and social connections. Individuals of higher SES tend have better access to health information and can more easily implement behavioral changes to avoid health risks; for instance, smoking, diet and physical activity (Link and Phelan 1995). The FSCD is a promising theoretical framework for examining the SES-obesity and the SES-obesity-comorbidities

relationships as it situates individual risk factors within the larger socio-cultural structures, making these broader contexts the key mechanisms explaining disparities in health outcomes. Additionally, it not only encourages us to ask questions about the antecedents of variation in obesity, but also the health consequences of obesity for different social groups. FSCD theory has been utilized by medical sociologists studying relationships between SES, health behaviors, and diverse health outcomes, including mortality (Phelan et al. 2004), smoking (Link and Phelan 2009), cancer screening tests (Link et al. 1998), and diabetes-self management (Lutfey and Freese 2005). However, this theoretical perspective has seldom been applied to the study obesity and obesity-related health outcomes. It should be noted that much of the previous research grounded in the FSCD approach has examined a single social indicator of health inequalities (e.g. SES or gender). The research will utilize the theory in a novel way and consider the interplay among multiple fundamental social causes of health (race/ethnicity, gender, and SES) to examine both in-between and within group differences.

By utilizing the 2011-2014 cycle of the National Health and Nutrition Examination Survey (NHANES), this study will apply the fundamental social causes of disease theoretical framework to explore how and why social and economic contexts put certain individuals at risk of being obese and the multiple ways that structural factors and individual behaviors lead to obesity-related health outcomes. Therefore, the main research questions guiding this dissertation include the following:

1. Does race/ethnicity and gender shape the SES gradient in obesity?

2. Do distal social factors (i.e. SES, race/ethnicity and gender) shape the effect of obesity on arthritis and cardiovascular health indicators, such as diastolic and systolic blood pressure, fasting plasma glucose, and HDL cholesterol?

Thus, the main goals of this dissertation are to 1) provide further evidence that studying social disparities in obesity rates based on additive models of risk accumulation do not adequately address the interaction of distal social factors in shaping population health, and 2) assess the conditions under which distal social factors and more proximate health risk factors exert a conjoined effect on selected health outcomes, which vary in the degree to which they can be controlled.

This dissertation addresses key gaps in the literature on the social patterning of obesity and certain obesity co-morbidities – namely, arthritis and indicators of cardiovascular disease, including blood pressure, plasma fasting glucose, and HDL cholesterol – across diverse social groups in distinct but interrelated analytical chapters. Although each chapter addresses unique research questions, they are rooted in a unified theoretical perspective, which emphasizes the systematic identification of the ways in which health is distributed across multiple intersecting social dimensions (Braveman 2009). Racial, gendered, and socio-economic inequalities in health have been addressed in depth in the fields of medical sociology, epidemiology, and public health. However, this research has primarily relied on additive models controlling for gender, race/ethnicity, and SES instead of studying their unique and multiplicative effects. Such research practice has left us with an incomplete conceptualization of disparities in health. Individual health is not shaped by either gender or race, but rather by a combination of these social dimensions, and should be modelled as such. This research project is one of

the few that seeks to uncover and articulate a more nuanced picture of social disparities in obesity and select health outcomes.

Chapter Outlines

The first dissertation chapter provided a brief overview of the problems to be explored in this dissertation, while discussing the gaps in the current SES-obesity and obesity-comorbidity scholarship and identifying the guiding research questions for this study. The following chapter (Chapter 2) provides a more thorough review of the theoretical framework informing this dissertation: the fundamental social causes of disease (FSCD) approach. Beginning with an overview of the historical perspectives on health inequalities research, this chapter offers a detailed look at the risk factor epidemiology literature as well as competing approaches to health disparities, with a special focus on the FSCD approach and its extension to studying SES-obesity and obesity-comorbidity links. Chapter 3 focuses on discussing previously published scholarship on the social disparities in obesity and five obesity-related health outcomes – arthritis, systolic and diastolic blood pressure, fasting plasma glucose and HDL cholesterol. While reviewing the literature, I also underscore the link between my proposed research questions and the ways that the FSCD approach informs them.

In Chapter 4, the National Health and Nutrition Evaluation Survey (NHANES) data are introduced, with an emphasis on the survey objectives and complex sample design. In addition, data collection instruments, issues associated with interviewee training, and data quality are addressed. The 2011-2014 sample of the NHANES study is also described in depth and the main measures are introduced. Further, a brief explanation of how these measures relate to the theoretical approach and facilitate testing

the key relationships between the variables are provided. Data analysis strategies and descriptive statistics are also provided in Chapter 4.

The focus of Chapter 5 is to answer the first proposed research question, which asks whether the association between indicators of socio-economic status (i.e. education and income) and BMI are moderated by gender and race/ethnicity. I test for joint effects of social status indicators to better understand how intersecting dimensions of inequality influence the distribution of BMI. The results from this chapter provide an important understanding of the relationship between socio-economic status and body weight, and in particular, how the advantage of higher income and education in its association with lower BMI do not affect all gender and racial/ethnic groups evenly.

Building on the Chapter 5 findings, the main goal of Chapter 6 is to answer the second research questions, which asks whether gender, race/ethnicity, and SES moderate the association between BMI and arthritis as well as four indicators of cardiovascular health – systolic and diastolic blood pressure, fasting plasma glucose and HDL cholesterol. This chapter clarifies whether the health damaging effect of BMI is amplified or diminished for already disadvantaged social groups. In addition, using different health outcomes as dependent variables that vary in the extent to which they are effected by individual health behavior allows me to investigate whether risk accumulation is dependent on the health outcome in question.

The final chapter (Chapter 7) of the dissertation briefly discusses the most significant findings of the previous chapters and emphasizes the theoretical and methodological contributions of this research project. In addition, potential policy

implications are discussed. Finally, limitations of the current research are disclosed in light of the potential ways they could be addressed in future studies.

CHAPTER 2: THEORETICAL BACKGROUND

The first dissertation chapter has provided a brief overview of the problems to be explored in this dissertation, while discussing the gaps in the current SES-obesity and obesity-comorbidity scholarship and identifying the guiding research questions for this study. By situating the research in the fundamental social causes of disease (FSCD) approach, this dissertation will provide researchers, practitioners, and policy makers a clearer understanding of the intricate relationship between social factors, body weight and health outcomes associated with increased body weight. The following chapter will provide a more thorough review of the theoretical framework informing this dissertation, the FSCD approach. Beginning with an overview of historical perspectives on health inequalities research, this chapter will offer a detailed look at the risk factor epidemiology literature as well as competing approaches to health disparities, with a special focus on the FSCD approach and its extension to studying SES-obesity and obesity-comorbidity links.

Social Inequalities in Health: A Historical Perspective

Social stratification is one of the tenets of sociological inquiry, and the association between socio-economic status and health could be viewed as a classical problem in the study of structure and agency. The primacy of social class as a major determinant of health has been known for centuries and propagated by the advocates of social medicine in the nineteenth century, who observed a strong association between social conditions that the poor lived and worked in (including poor sanitation, overcrowding and inadequate diet) and health.

In the 1800s in Great Britain, Edwin Chadwick reported class-stratified death statistics in his "Report on the Sanitary Condition of the Laboring Population of Great Britain" and Friedrich Engels published the "Conditions of the Working Class in England" documenting the features of urban life stratified by social class as etiology of disease (Chadwick 1842; Engels 1845). At around the same time in Germany, Rudolf Virchow advocated for the idea of medicine as a social science and argued that social structures of oppression were major causes of illness and death (Virchow 1848). Further, in 1977 the British Labour Government funded a research project with a goal to classify the British population into 6 social classes, based on the occupation of the household head, to assess the evidence of inequalities in health. The issued report is now universally known as the Black Report. The results of the report suggested that the risk of mortality in the lowest class was twice as high relative to the higher class. Moreover, health inequalities were widening over time even though the mortality rates as a whole had been decreasing. Interestingly, the widening of inequalities was not due to the increase in mortality in the lowest classes, but a sharp decrease in mortality in the highest classes (Feinstein 1993). Similar findings were confirmed throughout the 1980s in the Whitehall studies of the British Civil Service, which demonstrated a linear gradient of increasing morbidity and mortality at each lower grade (Marmot et al. 1991).

While social class-mortality associations were being monitored fairly thoroughly in Europe and Canada, in the United States, research has predominantly been focused on racial differences in health (DuBois 1899; Williams and Collins 2001). For instance, the influential study "The Philadelphia Negro" by DuBois (1899) uncovered the detrimental effects of structural inequalities on various aspects of the lives of inner city Blacks,

including disparities in health. The early analyses of the relationship between SES and mortality could be traced back to the 1950s and the early work of August B. Hollingshead on social class and mental illness, which uncovered the power of social environment on the distribution of health outcomes (Hollingshead & Redlich 1953, 1958). Later, in 1973 Evelyn Kitagawa and Philip Hauser presented results from two studies using two distinct data sets, entitled "Differential Mortality in the United States: A Study in Socioeconomic Inequality." By linking death certificates with census information on household income and educational attainment information for 340,000 individuals who died during May-August 1960, the authors found that income and education had independent effects on mortality. Further, when mortality was disaggregated into 23 major causes of death, education was consistently inversely related to death from heart disease for men and women. The second study collected information on census tracts in the Chicago metropolitan area and the surrounding suburbs in 1930, 1940, 1950, and 1960. The study used data on median rental payments to divide tracks into 5 categories. In each time period, the mortality rate in the lowest SES class was 60% higher than the highest SES class (Feinstein 1993). Consistent with these findings, in 1993 Paul Menchik used data from the National Longitudinal Survey of the 1966-1983 period to examine the relationship between household wealth and mortality. Not surprisingly, he found that increase in wealth was associated with a decrease in mortality rates, controlling for a number of other characteristics (Feinstein 1993).

Other U.S. scholars investigating the relationship between SES and mortality in the late 1980s and early 1990s reported similar findings, with some deviation based on the choice of SES indicator (Feinstein 1993). Even though these pioneering studies

suffered from fundamental methodological issues, their main conclusion has remained valid over time, and has spurred further interest in the relationship. In fact, more recent research studies have included more accurate mortality measures as well as different indicators of SES. It is important to add that the study of SES-health association was not limited to purely academic purposes. As an example, in Latin America, the work of Salvador Allende (1939) criticized the working conditions as a primary cause of health inequalities, and was one the driving forces of the socialist reform movement.

Risk Factor Approach to Health Inequalities

Despite the deep historical roots of scholarship examining socioeconomic differentials in health, epidemiologists – the dominant scientists in the field of the study of population health – have historically paid little attention to socio-economic status as a determinant of health inequality. During the nineteenth and early twentieth centuries, in the United States and other developed countries efforts were made to improve the physical environment among the working class. They included improvements in sanitation, nutrition, water supply, and housing, as well as promoting access to immunization and medical professionals. Broad improvements in public health led to an increase in life expectancy, based on which, many scientists predicted the disappearance of health inequalities. However, while the prevalence of infectious diseases as a major factor in producing mortality has declined, they have been replaced with chronic illnesses, such as cardiovascular disease, diabetes and cancers. Thus, in spite of the aforementioned developments, social inequalities in health have persisted (Williams 1990).

As chronic diseases proved to be multi-factorial as opposed to having a single

etiological agent, environmental, behavioral and psycho-social factors became the focus of epidemiologists monitoring disease trends in order to uncover the causal connections between risk factors and poor health. By the 1970s, epidemiology delivered strong evidence that behavioral and psychosocial risk factors, such as smoking, poor diet, lack of exercise, chronic stress, self-efficacy and sense of control, were associated with significantly increased risk of morbidity and mortality from chronic illness (Adler and Ostrove 1999; House 2002). Based on the model of risk factor epidemiology, in an effort to reduce inequalities in health, the intervening or more proximate modifiable risk factors, should be eliminated. Thus, in the traditional epidemiological approaches to health inequalities, socio-economic status, which is more distal in the chain of disease causation, became of secondary interest. Even when scholars recognized the disproportionate distribution of psychosocial predictors of disease among low SES individuals, inadequate attention was paid to the systematic examination of social and cultural structures that could affect their unequal distribution in the first place (Williams 1990). For instance, socio-economic status was considered only insofar as it influenced the more proximate risk factors of disease. It was presumed that distal social factors, such as SES, race and gender, were of little relevance once intervening mechanisms were accounted for, and they were relegated to serving as control variables in epidemiological studies. Such focus on proximal causes of disease in the health sciences has been perceived as a scientific advancement in moving beyond mere correlations to a better understanding of causal relationships (Phelan et al. 2004).

While public-health initiatives and interventions based on the risk factor approach had a positive effect on the overall population health by reducing the incidence of life-

threatening chronic diseases, such approaches have been met with criticism. Medical sociologists have criticized the epidemiological explanations of health disparities for their limited is gaze to the most proximate risks of morbidity and mortality, while failing to see broader social relationships and social contexts that condition the capacity to modify or eliminate risk factors, thus rendering a less effective approach to addressing health disparities (Link and Phelan 1995; Phelan, Link, and Tehranifar 2010; Link and Phelan 2010).

Link and Phelan (1995) note that focus on the more proximate risk factors resonates with the cultural and belief system of the Western nations that emphasize individual responsibility and self-control, yet the authors find such approach to health problematic. First, focusing on proximate risk factors, according to the authors, shies away from of larger social-structural conditions of disease that are beyond individual control. Health behavior is induced and constrained by the social context. For instance, an individual of low SES may know that smoking is bad for one's health, yet this may be their only coping mechanism for daily stressors (Williams 1990). Thus, individuals may choose to engage in behavior that is damaging to their health not because they are ignorant but because they have more pressing issues of every-day survival, such as putting food on a table. A lack of consideration of the broader social contexts that affect resource distribution, and in turn constrain individual actions, may result in policy solutions that do not challenge inherent social inequalities. Thus, policy decisions may only successfully affect individuals who have the needed resources to change health behavior while leaving the health of the most vulnerable populations unchanged. Further, such policies perpetuate the culture of "blaming" the individual and stigmatizing the

"lifestyles of the poor", in turn leading to even more detrimental health effects. In addition, it has been argued that identification of proximate risk factors can exacerbate inequalities in health. Because the benefits newfound capacity to control diseases are not equally distributed throughout the society, social groups that have greater access to resources – such as knowledge, social connections and money – will be more likely to benefit from such advancements, while reproducing the social gradient in health for more disadvantaged groups (Link and Phelan 1995; Phelan et al. 2004).

Competing Explanations for Social Inequalities in Health

Some of the alternative explanations of the SES gradient in health include the artifactual and the social drift hypotheses (Adler and Ostrove 1999). Proponents of the artifactual hypothesis, most influentially advocated by Kadushin (1964), argue that individuals from lower SES experience illness differently. Specifically, they suggest that individuals from low SES backgrounds do not have poorer health outcomes but feel and act sicker relative to their higher SES counterparts because of certain cultural and structural factors. However, no empirical evidence exists to support the claims that the artifactual hypothesis can account for SES differences in health, especially considering the persistence of a strong relationship between SES and all-cause mortality (Phelan et al. 2004). The authors add that "material and social resources and the deliberate use of them are critical factors in maintaining socioeconomic differentials in mortality and they [empirical findings] should point attention toward the fundamental importance of the societal distribution of resources – who gets what and how much of it – in shaping the strong socioeconomic gradients in mortality" (p. 280).

According to the social drift hypothesis, individuals who have poorer health are

prevented from participating in the labor market and maintaining adequate income levels, and therefore may drift downwards on the socio-economic ladder (Williams 1990). While some studies have provided support for the social drift hypothesis, it is not sufficiently broad enough to explain the SES gradient in mortality and morbidity. For instance, research indicates that the social drift hypothesis is likely to be supported for diseases that have an early onset and more profound effects later in the life-course, such schizophrenia (Adler and Ostrove 1999).

Thus, even though there is some evidence to support the claim that health status does influence socio-economic status, evidence is more compelling for social causation. Highly educated people and those who are economically well off not only report better self-rated health and physical functioning, but also have lower levels of morbidity and mortality. In contrast, low educational attainment and economic hardship are associated with high rates of infectious and chronic illnesses, poor self-reported health, disability, lower life expectancy, and expedited decline when sick (House et al. 1990; McIntyre 1997; Mirowsky and Hu 1996; Mirowsky and Ross 1998; Moore and Hayward 1990; Reynolds and Ross 1998; Ross and Wu 1995; Wilkonson 1986; Williams 1990).

In addition to the previously discussed hypotheses, health care utilization scholars have argued that inadequate medical care, especially preventative care, can partially explain SES differentials in health. Evidence does suggest that individuals with lower income and education, as well as racial minorities, are less likely to have insurance coverage, have less access to health services, are less likely to have routine examinations and screening procedures, and the treatments that they receive tend to be delayed or of a lower quality (Goldman and Smith 2002; Katz and Hofer 1994; Lantz, Weigers, and

House 1997; Williams 1990; Williams and Collins 1995). Nonetheless, evidence equalizing access to medical care does not reduce inequalities in health; the persistence of the SES gradient in health has been observed in countries with national health insurance (i.e., United Kingdom and Great Britain) where inequalities in access to medical care have been virtually eliminated (Marmot, Kogevinas, and Elston 1987; Roos and Mustard 1997). In addition, having health insurance has been found to show no noticeable effect on health. Specifically, Ross and Mirowsky (2000) have demonstrated that persons with private health insurance do not differ significantly from the uninsured based on self-reported health, physical functioning, and chronic conditions, while those with public insurance report worse health and more chronic conditions. Most importantly, their research has shown that changes in SES (e.g. increased income, being employed), not changes in insurance status, are associated with better health. Thus, evidence shows that SES differentials in health seem to lie outside the medical system.

However, it is necessary to note that medical sociologists whose area of expertise includes heath service research, state that despite the fact that disparities in health service utilization account only for a small part of variation in inequality in health, they do explain illness experiences and health outcomes among social groups who navigate the highly stratified medical system (Wright and Perry 2010). For instance, Lufty and Freese (2005) conducted ethnographic research in order to document the persistence of the inverse relationship between SES and health outcomes among diabetes patients during their routine clinic visits. Having collected data from two clinics, one primarily serving Caucasian middle and upper-middle class patients and the other primarily serving uninsured working class and ethnic minority patients, the authors uncovered complex

pathways operating at individual and institutional levels through which accumulation of knowledge and resources introduced advantages to higher SES patients and constrained health management for lower SES patients. The authors concluded that "durable relationships between encompassing variables like SES and health may represent an accumulation of many small, pervasive advantages that can be expected to be renewed as the particulars of disease treatment change over time" (Lutfey and Freese 2005:1361). Thus, access to medical care is still a desirable policy goal as it can be perceived as an intervening mechanism between social structure and the health of the poor; though it would not completely eliminate the root causes of inequalities in health (Williams 1990).

Fundamental Social Causes of Disease (FSCD) Approach

Medical sociologists have long recognized that social status is an important explanatory factor of health disparities and can have direct and indirect effects on health and health behaviors. However, the most prominent contributors to the "fundamental causality" approach are Link and Phelan (Link 2008; Link et al. 1998, 2008; Link and Phelan 1995, 1996, 2002, 2009; Phelan et al. 2004, 2010). The fundamental social causes of disease (FSCD) theory was first introduced in the seminal article "Social Conditions as Fundamental Causes of Disease" in the *Journal of Health and Social Behavior*, where the authors provided a theoretically sound and empirically robust explanation of why the association between SES and health has persisted across different places and time (Link and Phelan 1995). Within their work, Link and Phelan argue that factors such as socioeconomic status, gender, and race/ethnicity can be thought of as fundamental social causes of disease because they "involve access to resources that can be used to avoid risks or to minimize the consequences of disease once it occurs" (1995:87).

The resources mentioned by Link and Phelan (1995) include money, knowledge, power, prestige, and beneficial social connections. It has been recently proposed that intelligence may be another resource that could potentially account for the relationship between SES and health, however longitudinal analyses have provided no support for such hypotheses (Link et al. 2008). Individuals of higher SES have access to better neighborhoods and better paying jobs, they can more easily access health information, and adjust their behavior in order to avoid risk factors associated with disease, and even if they become sick, they have access to better medical care and other resources to manage their health. The aforementioned resources are flexible, meaning that those of higher SES will always be in an advantageous position to benefit from technological and scientific advancements in medicine despite the disease profile or its associated risk factors.

This claim is very well illustrated with the examples of cancer screening and smoking. Ability to detect deadly cancers early is a fairly recent technological advancement. Before the cancer screenings were available, resources had no influence over access to the procedures, as they did not exist. However, once the screenings for cervical and breast cancer became available, individuals with higher income and education could use their resources to gain access to the procedures, thereby creating a new link between SES and health (Link et al. 1998). Similarly, smoking knowledge and behavior show a strong social patterning. In the 1950s, as the first reports about the negative health outcomes of smoking emerged, only a minority believed that smoking was linked to cancer, and there was no clear SES gradient in smoking knowledge. However, over the following forty years, as more people began to believe that smoking increases the risks of cancer, a steep educational gradient occurred. Consequently,

individuals of higher SES were more likely to quit smoking, contributing to the creation of the inverse association between SES and smoking (Link 2008).

Therefore, social conditions are fundamental social causes of disease because they can be reproduced under different circumstances and involve different risk factors and disease outcomes. Further, because a fundamental social cause is distal in the chain of causation, it may be related to numerous disease outcomes through a multitude of pathways involving more proximate risk factors (Link et al. 1998; Link and Phelan 1995, 1996; Phelan and Link 2005). According to Freese and Luftey (2001) "fundamental causality" is more than saying "SES causes health;" it implies that if individual's SES, gender, race or social networks were different, their health outcomes would be different too (p. 68). Thus, a fundamental cause is analytically different from any proximate cause of health because a multitude of causal mechanisms connect X and Y. While risk factors mediating the relationship may change or be eradicated, new mechanisms will emerge sustaining the fundamental relationship between higher SES and better health outcomes (Freese and Luftey 2001).

In addition, fundamental social causes have the greatest impact on health problems that are easily preventable relative to those that are difficult to prevent due the nature of disease itself or lack of clinical expertise about potential treatments (Phelan et al 2004). By using the National Longitudinal Mortality Study, the authors have found that mortality from causes of death that are highly preventable, are significantly and more strongly related to socioeconomic status. Furthermore, these findings hold across different gender and ethnic groups. The authors add that,

> *these findings strongly suggest that material and social resources and the deliberate use of them are critical factors in maintaining*

> *socioeconomic differentials in mortality and they should draw attention toward the fundamental importance of the societal distribution of resources – who gets what and how much of it – in shaping the strong socioeconomic gradients in mortality (p. 280).*

Even though socio-economic status as a fundamental social cause has received the majority of scholarly attention, gender and race/ethnicity are also examples of social status patterning disease. For instance, women report worse physical health than men, yet men die younger than women even though the leading causes of mortality (i.e. heart disease, cancer, and stroke) are the same for both genders (Rieker and Bird 2008). In addition, women experience substantially higher rates of depression than men at all ages, while men are more likely to suffer from substance abuse and anti-social behavior. The life-time rates of dependence on alcohol and tobacco are also considerably higher among men than women (Rieker and Bird 2008). Other researchers have explored the effect of race/ethnicity on the persistence of health disparities between African Americans and Caucasians in the Unites States (Williams and Collins 1995, 2001). Specifically, they have argued that persistent racial segregation in the U.S. is a fundamental social cause of racial health disparities as it has restricted the access of racial minority groups to education and employment, and shaped their disadvantaged social and economic conditions at both individual and structural levels. Pescosolido and colleagues (2008) have considered gender as well as race/ethnicity and socio-economic status as fundamental social causes of alcohol dependence, with their conclusions demonstrating support for the fundamental social causes of disease theory.

In brief, the fundamental social causes of disease theory involves four key mechanisms: 1) fundamental causes influence multiple disease outcomes; 2) fundamental causes affect health through a multitude of risk factors; 3) the relationship between a

fundamental cause and health involves access to resources that can be used to minimize risks or reduce the consequences of disease if it occurs; and 4) the association is reproduced over time via the replacement of intervening mechanisms (Phelan, Link, and Tehranifar 2010:29).

While some of the previously discussed research projects attempted to test the fundamental social causes of disease approach via quantitative and qualitative means, the proposed dissertation projected is not intended to test the theory but rather use it as a guiding framework in forming research questions and hypotheses. It should also be added that not all essential features of the theory will be addressed in this project due to methodological limitations. Specifically, while SES is associated with multiple disease and health risk factors, I will focus on obesity and five obesity-related health outcomes – arthritis, systolic and diastolic blood pressure, fasting plasma glucose, and high density lipoprotein (HDL) cholesterol.

Second, due to data limitations, I will not be able to explicitly test the multiple possible mechanisms linking fundamental social causes with obesity and obesity comorbidities. Additionally, when answering the second research question (Chapter 6), I will limit my analysis to operationalizing obesity as the core intervening mechanism connecting social factors with arthritis and four indicators of cardiovascular health. I am fully aware, however, that increase in body weight is just one of many mechanisms that link social factors to chronic illness.

Third, by operationalizing indicators of social status, such as gender, race/ethnicity, and SES (education and income), I will be assuming that the more advantaged groups not only have resources at their disposal but also use them with a

purpose of protecting their health. Because of limitations imposed by secondary data, I will not able to model access to and the use of potential resources more explicitly.

Finally, because of constrictions of cross-sectional data, I will not be able to make inferences about the SES and health relationship over time. Nonetheless, I will be able to draw broad conclusions if my research findings are supported by previous research.

CHAPTER 3: PREVIOUS RESEARH ON SOCIAL DETERMINANTS OF OBESITY AND SELECT OBESITY-RELATED HEALTH OUTCOMES

The previous chapter has provided an in depth review of the main theoretical approach guiding this dissertation – the fundamental social causes of disease (FSCD) approach. In Chapter 3, I will review previously published research investigating the social disparities in obesity and five chosen health outcomes associated with obesity – arthritis, systolic and diastolic blood pressure, fasting plasma glucose, and HDL cholesterol. Beginning with an overview of obesity as a highly medicalized condition, I will focus on establishing a connection between my proposed research questions and the ways that FSCD approach informs them.

Social Determinants of Obesity

What is Obesity?

From the biomedical perspective obesity develops when energy intake exceeds energy expenditure, which increases accumulation of excess fat that leads to impaired health (Krauss et al. 1998). Most commonly, obesity is measured by the Body Mass Index or BMI (kg/m^2). Government health agencies, such as Center for Disease Control (CDC) and the National Institutes of Health (NIH) have been relying on BMI as a primary measure of overweight and health since the 1990s. Based on BMI, individuals are classified into four main weight categories: underweight (BMI<18.5), normal weight (BMI=18.5-24.5), overweight (BMI=24.5-30) and obese (BMI>30) (World Health Organization 1995). The obese category can also be differentiated between grade I obesity (BMI=30-34.9), grade II obesity (BMI=35-39.9) and grade III obesity (BMI>40) (World Health Organization 1995).

It is worth noting that BMI is a poor and problematic measure for several reasons. First, as a proxy legitimizing the label of "obesity", it was never intended to be used as an indicator of individual health risk; BMI was developed in the process trying to test whether mathematical laws of probability are applicable to humans, and only years later was co-opted by the insurance companies to predict early deaths, and analyze the general health of the population. In addition, the proposed categorizations of weight standards are arbitrary and had fluctuated until the 1988 NIH panel, where the standard was set for BMI of 25 to indicate overweight and BMI of 30 to indicate obesity. Implementation of aforementioned changes "made" thousands of Americans obese overnight (Oliver 2006). Finally, BMI does not differentiate between lean and fat body mass, which leads to overrepresentation of certain groups of individuals as overweight (e.g. athletes).

Even though BMI is correlated with a number of negative health outcomes, some researchers suggest that waist circumference (WC) may provide an independent effect when predicting health risks over and above that of BMI. Even though waist circumference adds very little predictive power of disease risk in individuals with BMIs ≥35, it is a more robust measure of cardiovascular health risks among individuals who are categorized as normal or overweight. High waist circumference has been shown to be associated with an increased risk for type 2 diabetes, dyslipidemia, hypertension, and cardiovascular disease (CVD) in individuals whose BMI ranges between 25 and 34.9 kg/m^2 (Chan et al. 1994). Despite its increased predictive accuracy over BMI alone, WC also suffers from certain shortcomings. As an example, there are gender, ethnic, and age-related variations in body fat distribution that affect the predictive validity of waist circumference. For instance, women have a higher percentage of body fat than men, but it

does not increase their health risks (Conway et al. 1995; Dowling and Pi-Sunyer 1993). Table 3.1 provides cut-off points for different obesity levels and associated disease risk when using BMI and waist circumference.

[Insert Table 3.1 about here]

Despite its problematic nature, BMI remains the dominant measure for diagnosing obesity and associated health risks in national and international settings. It is a popular and inexpensive tool, which is prone to misclassification errors and can only indirectly measure body fat. It should be noted though that despite the fact that BMI may fail to distinguish between healthy and problematic body fat at an individual level, its widespread and longstanding application has allowed for national and international health organizations to collect and publish population-level data and make comparisons across sub-groups, regions, and time. It is also worth mentioning here that in the majority of research published on the SES-obesity relationship, BMI has been chosen as the primary outcome of interest. In order to maintain consistency and ability to compare findings across publications, BMI will be used as an indicator of obesity in the current study. However, knowing that robustness of BMI when classifying individuals into different weight categories varies by gender and ethnicity, sensitivity analyses will be performed using waist circumference as an indicator of abdominal obesity.

Medicalization of Obesity

Medicalization is the process by which previously non-medical problems become defined as medical (Conrad 2008). According to Conrad (2015), medicalization, is distinguished by several characteristics: (1) the issue of definition is central to medicalization, (2) there are degrees to which problems are medicalized, (3)

medicalization categories are elastic, (4) physician involvement in medicalization is variable, and (5) medicalization is bi-directional, meaning there can be both medicalization and de-medicalization (p. 19). According to Sobal (1995), during the past century, the conception of body fat as "badness" shifted to medicalization of obesity as a health risk factor and sickness in itself, to the de-medicalization of large bodies as socially and politically acceptable. It should be noted though that medicalized and de-medicalized conceptions of obesity may exist at the same time and be evoked by different actors and social groups based on their goals and agenda. In other words, while some activist groups may be fighting for "fat acceptance", the pharmaceutical and medical industries may be promoting weight loss drugs and bariatric surgeries.

According to historical analyses, while fatness was widely socially acceptable in traditional societies, it quickly fell out of fashion in industrial societies, where resources for food were plentiful. In the 20th century United States, a slender body rather quickly became a way to demonstrate not only wealth but also the moral virtues of will power and self-restraint (Sobal and Stunkard 1989). Fostered by the Christian Church, the fat body became associated with gluttony, greed, poor self-impulse, and lack of personal control. As food, fashion, and pharmaceutical companied started promoting thin bodies, stigmatization of fatness became widely prevalent and powerful (Saguy 2013).

Although medical claims about the negative health effects associated with high adiposity levels have been made for decades, the medicalization of obesity did not take off until the 1950s, when the medical community and their allies started making increasingly frequent and persuasive claims that they should be responsible for tacking the issue of increasing fatness in the society. The process of medializing body fat

included multiple steps, such as the creation and application of the label of obesity, development of scientific measures (BMI) to justify a diagnosis, establishment of professional organizations, journals, and conferences focusing on promoting the medicalized framing of obesity, and, finally, invention of pharmaceutical and medical treatments for the condition (Sobal 1995). For instance, from 1977-1978, the previously used term of "corpulence" for defining body fatness was changed to "obesity". The scientific terminology about obesity was also broadened by including eating disorders and other metabolic disorders. As an example, low levels of activity were relabeled from laziness to chronic fatigue and lethargy (Sobal 1995). Further, the medical community negotiated and achieved a designation of disease status for obesity and its inclusion in the ICD-9. In addition, the idea that obesity is not only a disease but also a risk factor was developed in the 1970s. Obesity was promoted as one of the main risk factors for cardiovascular disease, which also made it the major contributor to the leading cause of death in the U.S. (Sobal 1995). Such developments were used to legitimate weight-loss clinics and weight-loss drugs, and promote their vested interests. Food service, fashion, and weight loss industries also emerged as promoters of the medical obesity model. Finally, organizations, such as the Association for the Study of Obesity, were being formed to advance the medicalization of obesity, and specialty journals, such as the *International Journal of Obesity and Obesity Research* followed. Special events, such as the NIH conference on obesity were being held to further legitimize obesity as an attention-worthy medical issue (Oliver 2006; Saguy 2013).

Efforts to Challenge the Medical Model of Obesity and Obesity-Mortality Link

The scientific community in medical and public health fields have, over the last few decades, demonstrated a persistent association between higher body weight and increased blood cholesterol, hypertension, diabetes mellitus, and increased risk of cardiovascular disease, musculoskeletal disorders, and some cancers (Dixon 2010; Guh 2009; Kraus et al. 1998; Mokdad 2003; Must et al. 1999). Such findings have been often communicated by the media as scientific truth framing public opinion and reinforcing the idea that obesity should concern society as a whole.

More recently, however, the relationship between obesity and all-cause mortality has become a source of controversy among obesity scholars and the broader community of social epidemiologists and medical sociologists, who started questioning the BMI-all cause mortality and morbidity link. For instance, the findings of a recently published meta-analysis by Flegal and colleagues (2012) support the claim that variations in the BMI – all-cause mortality findings could be accounted for by the use of different definitions and measurements of obesity. In particular, after reviewing 143 published manuscripts, the authors have found that obesity overall, as well as grade II and grade III obesity were significantly and positively associated with all-cause mortality, while grade I obesity had no significant effect on higher mortality, and overweight was associated with significantly lower all-cause mortality, controlling for age, gender and smoking status. Their findings are consistent with an older meta-analysis, which observed a U-shaped relationship between BMI and mortality (Troiano et al. 1996). In addition, the previously mentioned study has called into attention the negative health effects of being underweight; the relative risk of BMI<18.5 has been found to have more detrimental

health effects than even higher levels of obesity (BMI>30) (Flegal et al. 2005). According to Campos and colleagues (2006), the biggest issue with the most widely cited statistical analyses examining the BMI-mortality relationship, also co-opted by the media, is the lack of adjustment for important confounding factors, such as fitness, exercise, diet quality, weight cycling, and drug use, which tend to attenuate the effect of pure body fat on health. Thus, those that are overweight and obese do not necessarily face higher mortality and morbidity risk if their lifestyles are relatively health-oriented.

At the same time, national activist groups, such as the National Association to Advance Fat Acceptance, have challenged the obesity-morbidity relationship, pointing to the detrimental health effects of weight-based discrimination and yo-yo dieting (Kwan 2009). Advocates of the de-medicalization of obesity challenged the idea that fatness on its own is pathological. They also argued that individual body weight is the wrong target of interventions because body size itself is irrelevant to health and because much of the weight loss interventions have had limited success (Kraschnewski et al. 2010). The fat rights activist groups have taken their claims one step further and argued that fat rights should be seen as any other human rights. They perceive fat as a part of self-identity and claim that fighting weight-based discrimination is closely related to other social issues, such as struggles against racial and sex discrimination, as well as LGBTQ rights (Saguy 2013).

Consequences of Obesity Medicalization

While medicalization of obesity may potentially reduce discrimination and stigma among health care professionals by emphasizing the causes of obesity that are beyond individual control, its broader consequences are more nuanced. The medicalized

framework of obesity might lower the threshold between what is considered a "normal" and "sick" body, increasing the number of people who may be perceived as requiring medical intervention due to the inherent pathology of their body size. Additionally, fears of becoming fat may also contribute to disordered eating or encourage smoking, which has been associated with weight loss. Medicalized perceptions about body weight may also be psychologically harmful to those who may not have previously thought that they needed to lose weight or to those who have tried to lose weight and failed. Furthermore, while investment in medical procedures and the development of weight-loss drugs may lead to safe and higher quality options of health care for individuals, it also implies an increasing expansion of the biomedical approaches to and control of individual bodies (Saguy 2013).

Feminist scholars have raised concerns that bio-medical knowledge is deeply entrenched in patriarchal ideas and exerts domination over female bodies, making them objects of control by a male-dominated profession (Wry and Deery 2008). Overall, American culture puts more pressure on women than men to conform to narrowly defined beauty expectations and punishes them more harshly for deviating from the aesthetic ideals of slim bodies (Bordo 2003; Wolf 1991). From this perspective, medicalization of female fatness is profoundly gendered, and serves as a vehicle of female oppression. It marks women's inferior status relative to men by shifting focus from female empowerment and competence in multiple domains of social life to superficial markers of their appearance, reducing them to objects of sexual attraction (Jeffreys 2005).

The medicalized frames surrounding body size intersect not only with gender but also race and class in fundamental ways. The concerns over and public discourses about

the obesity epidemic in the United States have largely been imbued with class and racial overtones. For instance, the poor and minority women are viewed as irresponsible "fat" people who use the social welfare programs to sustain their unbridled appetites. In addition, while an anorexic child may be treated as a victim of illness that is beyond her control, an obese child is portrayed as an outcome of parental neglect including too much sedentary activity, such as TV watching, and poor food choices, such as fast food and sugary drinks (Saguy 2013). The medicalization of obesity has led to attributing responsibility to individuals, as opposed to making larger structural linkages and focusing on addressing broader social inequalities. It reinforces the view that fat people have a moral obligation to try to "get over" their disease, even if the process involves practices that may ultimately damage their health more significantly than their weight. Such practices also punish people for being fat, and the poor and racial minorities are disproportionately subject to this form of punishment (Saguy and Riley 2005).

Obesity is not just a medical diagnosis, it is also a social issue that various social groups and industries wish to define and re-define. Even though critical obesity research does not unequivocally state that high body weight is healthy, it urges us to exercise caution when discussing the obesity-mortality and morbidity link. Most importantly, such scholarship demonstrates that the "obesity epidemic" reinforces white, middle class prejudice against the poor and racial minorities, and motivates us to consider broader effects of social inequalities and labelling on health (Campos 2004; Oliver 2006; Saguy 2013).

Gender, Race/Ethnicity, SES and Obesity: What Do We Know?

Socio-economic status is a marker of an individual's structural position in a society and access to social, economic and cultural resources that may vary over time (Duncan et al. 2002). SES is generally measured by education, occupation, income or wealth, and can be linked to obesity through material and cultural pathways (Peralta 2003). A promising line of SES-obesity research could be traced back to Sobal and Stunkard's (1989) review based on a total of 144 studies published between the 1960s and 1980 in developed and developing societies. Their findings suggested that in developed countries, the relationship between socio-economic status and obesity depended on age and gender. Specifically, a strong inverse relationship was observed among women (i.e., women of high socio-economic status yielded lower obesity rates), while the findings among men and children were mixed. Other work has continued to show a strong inverse relationship among women and inconsistent findings among men. For instance, Ball and Crawford (2005), in their meta-analysis of 34 longitudinal studies published between 1980 and 2002 found support for the hypothesis of an inverse relationship between occupational status and weight among women, while the link between income and weight gain was inconsistent.

More recently, in her meta-analysis of 333 studies published since Sobal and Stunkard's (1989) report, McLaren (2007) explored the relationship by using multiple indicators of SES (income, education, occupation, employment, composite-indicator, area-level indicator, assets and material belonging) and a three-category measurement to differentiate the development status of countries. With respect to gender and risk of overweight and obesity, her findings confirmed that women and men with higher

education status are less likely to be obese, while the relationship between body weight and income was inconsistent.

A strikingly similar relationship between SES and body weight has been confirmed in a number of studies conducted in the United States, Canada, United Kingdom and Finland. However, the strength, direction, and significance of the relationship appears to be contingent on the indicators of SES and gender. For instance, in the United States, Zhang and Wang (2004a) observed an overall inverse gradient across income levels. With respect to gender, the burden of obesity was equally distributed across levels of SES among men, while strong disparities were observed among women. They also found that men of higher SES were more likely to be overweight, while the opposite was true for women. Godley and McLaren (2010), using a nationally representative data of the Canadian population, found that women were less likely to be obese if they were more educated and lived in households with higher yearly income. However, for men, the relationship was negative if education was used as an indicator of social status, while it was non-linear and positive for income, controlling for age, marital status and racial background. In a study conducted in Helsinki in 2001, parental socio-economic indicators were found to be stronger predictors of obesity than individual-level SES indicators among women, while barely any associations reached significance among men (Laaksonen, Sarlio-Lähteenkorva, and Lahelma 2004). Similarly, research conducted in Great Britain showed a strong inverse relationship between occupational prestige in women and weight, yet no significant association in men (Wardle and Griffith 2001). A different study using the 1996 Health Survey for England confirmed the finding between obesity and education, but also found a

significant negative relationship between education and obesity for women, though not for men (Wardle, Waller, and Jarvis 2002).

Interestingly, researchers noted that ethnic/racial differences in obesity cannot be fully explained by individual SES (Robert and Reither 2004; Wang and Zhang 2006; Zhang and Wang 2004a, 2004b). For instance, Zhang and Wang (2004a) found a positive relationship between SES and obesity among African American and Mexican American men but a negative relationship for White men. Among women, the relationship between SES and obesity was inverse, however, it was only significant among White women. Scholars also found that the greatest inequality in SES-obesity relationship existed among the Whites, followed by Mexican Americans and Blacks. As such, they concluded that that SES could have a stronger impact on obesity in Whites relative to ethnic minorities. The disproportionate distribution of obesity among racial/ethnic inequalities net of their income has been confirmed in a number of multi-level studies. As an example, Robert and Reither (2004) found that even though individual and community SES partially explained higher mean BMI among Black women, racial disparities persisted. Additionally, most unexplained variation in BMI was due to within-community variance.

With respect to changes in BMI over the past few decades, Chang and Lauderdale (2005), using a four successive waves of the National Health and Nutrition Examination Survey (NHANES), found that among Caucasian women the largest increase in BMI occurred among the near poor and the smallest increase occurred among individuals in the highest income categories, while among African American women the largest increase in BMI occurred among those in the middle income categories. Nonetheless, Black women at the top of income distribution showed a larger mean BMI than White

women in all income distributions, across all time points. Among men, the largest increase occurred among African American men in the highest income categories. Other researchers found that during the same timeframe, the magnitude of the relationship between educational achievement and body weight declined among Caucasian women. Surprisingly, among African American women, the magnitude of the relationship declined as well, however the association changed in direction, such that women with medium-levels of education experienced the highest rates of increase in obesity. Among Caucasian men, highly educated groups became slightly protected from obesity, while education had no protective effect among African American men (Zhang and Wang 2004a). Other studies, using community samples, have largely confirmed the findings of greatest weight increase among racial minorities (Baltrus et al. 2005; Burke et al. 1996).

In order to effectively consider the intersectional nature of gender, race/ethnicity and SES, Ailshire and House (2009) conducted a longitudinal study examining changes in BMI in additive as well as multiplicative models. Using a nationally representative sample of adults followed for 15 years (1986-2002), they found that more and less advantaged groups experienced very different BMI trajectories. Specifically, low income and less educated Black women experienced the largest increase in BMI, while the opposite was true for high income and highly educated White men. Despite the importance of these findings, the authors of the study did not address the weight trajectories among Hispanic adults, thus it is not clear how changes in the BMI in this ethnic group compare to weight changes of White and Black adults. In addition, the data used in this study are more than a decade old, and we are left with a knowledge gap about the ways that the joint effect of gender, race/ethnicity and SES may have affected group

changes in BMI between 2002 and now. While this research project does not address the question of BMI trajectories, it does provide an important update to the literature about the joint relationship between indicators of social status and individual body weight.

Finally, to assess the effect of neighborhood disadvantage on changes in BMI, Ruel et al. (2009) estimated multi-level models predicting weight fluctuation among White and Black women over a 16-year period. While baseline neighborhood disadvantage was associated with BMI and did reduce racial disparities in BMI, it did not predict change over time. Substantial racial disparities persisted after controlling for individual-level SES, socio-demographic characteristics, risk and protective factors.

Application of FSCD to Studying Obesity

The idea that socio-economic status is a fundamental social cause of obesity is not novel. Sobal (1991) has argued that in an environment with abundant food resources where cultural values of thinness dominate, increased body weight may be perceived as an undesirable individual trait and negatively affect one's social position in the society. In contrast, if cultural values support fatness and bigger bodies, they will be predominant in higher social strata. Further, individuals with larger bodies will be more likely to move upwards in the trajectory of social mobility.

As discussed, while the increase in BMI over the last thirty years has affected both genders belonging to different racial/ethnic categories in all income groups, White men and women at the higher income levels maintained a distinct lower BMI advantage at each time point. Graphically, the change in slope remained fairly stable as opposed to the change in the intercept, suggesting that White high-income and highly-educated individuals have continuously had lower BMIs than their lower-income and racial

minority counterparts (Chang and Lauderdale 2005). Link and Phelan (1995) would attribute this trend to the fundamental social causes of disease; individuals in higher socio-economic status have more knowledge and resources to attenuate the effects of risk factors associated with increasing average population weight.

For instance, researchers have found that women with higher levels of education and income engage in health behaviors, such as dieting and exercising, that leads to lower BMI (Godley 2010; Kuhle and Veugelers 2008). In addition, research has demonstrated that socio-economically advantaged women feel more pressure to maintain lower body weight as a marker of social status (Bordo 2003). Finally, women of higher socio-economic status have demonstrated greater body dissatisfaction relative to their counterparts of lower socio-economic status (McLaren and Kuh 2004); (Ogden and Thomas 1999; Wardle and Griffith 2001). Among men, research findings are less clear cut. Some researchers have demonstrated that men of higher SES engage in behaviors, such as working longer hours, consuming more alcohol and not enough fruits and vegetables that could lead to a higher BMI (Godley and McLaren 2010). In a different study, Ball, Mishra and Crawford (2003) have found that among men, increase in income is positively associated with obesity via lower rates of smoking, relative to their low SES counterparts.

On the other hand, evidence suggests that entering a marriage is associated with changes in health behaviors, especially among men. In particular, spouses may act as sources of social control by discouraging unhealthy lifestyles, while encouraging healthier behaviors (Umberson 1992; Tucker and Anders 2001). For instance, earlier research has found that social control attempts within married couples are associated with

decreases in substance use (Bachman et al. 1997), and higher intake of produce, vitamins, and fiber (Schäfer et al. 1999; Tucker, Spiro, and Weiss 1995). Thus, women of higher SES may encourage their spouses to engage in healthier behaviors, protective against weight gain.

The application of FSDC approach when explaining weight gain among racial/ethnic minorities, especially those of higher social class is more complex. It could be argued that even though gender and race have strong ties with prestige, money and power, body weight is not simply a health outcome, but also a characteristic of social significance. Therefore, these associations could potentially be attributed to ideas of masculinity, body demographics, and culturally-bounded weight perceptions (Chang and Lauderdale 2005). For instance, some scholars suggest that in high SES strata, men pursue physical dominance by being larger and more muscular (Grogan 2007; McVey, Tweed, and Blackmore 2005).

Others attribute larger proportions of overweight and obese among racial minorities to historical segregation, which may have resulted in cultural insulation from the ideal of the thin body (Peralta 2003). Evidence does suggest that Black Americans tend to be less concerned about their weight and report greater acceptance of being overweight relative to their Caucasian counterparts (Kemper et al. 1994; Powell and Kahn 1995; Stevens, Kumanyika, and Keil 1994). In addition, African American women recognize their weight as a problem at much higher BMI levels than White women (Dorsey, Eberhardt, and Ogden 2009; Fitzgibbon, Blackman, and Avellone 2000; Rand and Kuldau 1990). This line of thinking suggests that body weight is associated with

cultural gendered and racial nuances, which cannot be fully accounted for by individual education or household income (Ross and Mirowsky 1983; Wang and Beydoun 2007).

On the other hand, neighborhood contexts that are marked by SES and racial inequality, may shape individual behaviors in ways that promote obesity, such as consumption of calorie-dense food, lack of physical activity, smoking, and alcohol consumption (Hall et al. 2003; Kuhle and Veugelers 2008; Matheson, Moineddin, and Glazier 2008; Pampel, Krueger, and Denney 2010; Wardle et al. 2002).

Racial minorities and socio-economically disadvantaged groups are more likely to live in neighborhoods that lack access to full-service grocery stores and farmers' markets where residents could buy fresh produce, whole grains, and dairy products. Instead, they may be limited to convenience stores where fresh products is rare (Larson et al. 2009). Using data from the National Health and Nutrition Examination Survey (1988–2002), one study found that non-Hispanic Blacks, persons in poverty, and those with less than a high school education were less likely to meet US Department of Agriculture fruit and vegetable guidelines relative to non-Hispanic Whites and socioeconomically advantaged individuals (Casagrande et al. 2007). Other studies have shown that Black Americans were the least likely to meet dietary recommendations for fruits, vegetables, and milk, while Mexican Americans were the most likely to meet recommendations for peas, beans and whole grains (Hiza et al. 2013; Kirkpatrick et al. 2012).

In addition to having less access to healthy food, lower SES individuals are more likely to experience food insecurity, which results in cycles of skipping food or overeating. Such coping mechanisms may lead to metabolic changes and accelerated weight gain (Eisenmann et al. 2011; McIntyre et al. 2003). Furthermore, racial minorities

and individuals of lower socio-economic status tend to lead more sedentary lives relative to their higher income counterparts (Andersen et al. 1998). Generally, lower income neighborhoods lack access to safe and attractive places to exercise, including parks, paths, green spaces, and recreational facilities, which make physically active lifestyles less attainable. Also, higher rates of crime make those neighborhoods less safe, which discourages staying outdoors (Lovasi et al. 2009).

The risky behavior framework, associated with lower SES, has also been applied to explain the increasing rates of obesity among Hispanic adults. Much of the research on Hispanic health has focused on Mexicans, who generally demonstrate strikingly good health profiles regardless of their SES (Zsembik and Fennell 2005). It has been noted, however, that cultural buffers erode with acculturation, which implies an increasing prevalence of negative health behaviors (Lara et al. 2005). On the other hand, acculturation has also been found to have a positive effect on health care use and self-perception of health (Lara et al. 2005).

While the effect of acculturation on Latino health is complex, a significant positive relationship between length of stay in the United States and increase in BMI has been confirmed by a number of scholars (Abraido-Lanza 2005; Gordon-Larsen 2003; Lara et al. 2005; Oza-Frank and Cunningham 2010). The excess weight gain has primarily been explained by the adaptation of higher alcohol intake, lower rates of physical activity and poor diet (Gordo-Larsen 2003; Abraido-Lanza 2005). It is not well understood however the extent to which opportunities of upward social mobility may ameliorate the negative association between acculturation and sub-optimal health behaviors, and this warrants future research.

Finally, research points to higher levels of stress experienced by low income and racial/ethnic minority families due to financial pressures, less autonomous work, lack of access to health care, and poor housing, among other factors. Chronic stress may trigger feelings of anxiety and depression, which can lead to unhealthy eating habits and lack of physical activity, both of which are associated with weight gain (Pine et al., 1997; Pine et al., 2001). Racial minorities, particularly Black women, may be in a uniquely disadvantaged position vis-à-vis other race/gender groups due to greater exposure to gender and racial discrimination. In addition to socio-environmental barriers, they may face greater threats from more subtle yet pervasive forms of stress that have been associated with poor health (Hunte and Williams 2009; Vines et al. 2007).

Although a handful of studies have addressed the relationship between gender, racial/ethnic, and socio-economic disparities in body weight using cross-section and longitudinal data, they are rather outdated. They are also limited by lack of nationally representative data and, most importantly, the use of additive statistical models. These limitations make it very difficult to state broad conclusions about those who are the most vulnerable to weight gain, and if it is the same groups that were the heaviest a decade ago. Drawing on FSCD approach, my analytic approach focuses on disentangling the combined influence of gender, race/ethnicity, and socio-economic status on obesity disparities in the U.S. population.

The Obesity-Comorbidity Link: How Does FSCD Fit In?

The health risks associated with increased body fat have been well documented in the fields of medicine and public health. A positive relationship has been established between obesity and risk of type 2 diabetes, cardiovascular disease, gallbladder disease,

osteoarthritis and some cancers (Anandacoomarasamy et al. 2007; Davy and Hall 2004; Dixon 2010; Krauss et al. 1998). This research, while important, provides only a partial picture of the how detrimental obesity really is to individual health. I have previously mentioned that there are gender, racial/ethnic and SES differences in the prevalence of obesity. These group differences persist in the prevalence of health complications, associated with obesity (Cossrow and Falkner 2004). Even though the obesity-comorbidity scholarship has recently been problematized by medical sociologists and social epidemiologists, a loose link between body weight, especially at the higher end of the BMI distribution, and negative health outcomes, remains.

It has been previously discussed that rates of obesity are not equally distributed in the American society, and are more prevalent among women, racial/ethnic minorities and individuals in lower socio-economic positions. Similarly, social disparities in cardiovascular mortality and morbidity in the U.S. society have been well documented (Lewey and Choudhry 2014). While separate literatures have addressed the pervasive nature of these disparities, less is known about the ways that distal social forces and proximate risk factors accumulate to affect individual health outcomes. Additionally, relatively little attention has been paid to potential variations in the effect of obesity by gender, race/ethnicity and social status on more or less modifiable health outcomes. Specifically, it should be expected that for health outcomes that are more biologically-determined, social factors should have little additional or multiplicative influence on their variation. In contrast, for health outcomes that are highly controllable, gender, race/ethnicity and social status – social aspects that confer power and access to resources – should make some social groups better poised to avoid detrimental health effects of

obesity relative to others. The following part of the chapter will lay the theoretical ground for testing the multiplicative effects of obesity and the distal social factors of gender, race/ethnicity, and SES on four indicators of cardiovascular health – namely, systolic and diastolic blood pressure, plasma fasting glucose and low density lipoprotein (HDL) cholesterol – and arthritis.

Select Obesity-Related Health Complications

Cardiovascular disease (CVD) is one of the leading causes of death in the United States, accounting for more deaths annually than any other disorder, including numerous problems, such as stroke, heart attack, heart failure, arrhythmia and heart valve problems (Go et al. 2014). Nonetheless, much of the cardiovascular disease morbidity and mortality could be attributed to modifiable risk factors, such as systolic and diastolic blood pressure, type 2 diabetes, hypercholesterolemia, smoking, excessive alcohol use, poor diet, lack of physical activity, and obesity. Obesity has been implicated as one of the major risk factor for cardiovascular events (Lavie, Milani, and Ventura 2009). Even though CVD mortality and morbidity rates have declined over the past few decades, in 2009-2010, about 47% of American adults had at least one of these CVD risk factors – high blood pressure, high levels of HDL cholesterol, or tobacco use (Fryar, Chen, and Li 2012). In 2011-2012 hypertension alone affected about one third of the U.S. adults, aged 18 and over. Even though hypertension awareness and treatment have been improving, undiagnosed and uncontrollable high blood pressure remains an important health challenge (Nwankwo et al. 2013). In 2011-2012, the age-adjusted prevalence of hypertension among adults aged 18 and older was about 29.1% (Nwankwo et al. 2013). Additionally, in 2009-2010, about 21.3% of adults aged 20 and over had low HDL

cholesterol, which is a risk factor for coronary heart disease (Caroll, Kit, and Lacher 2012).

Diabetes is a group of diseases marked by high levels of blood glucose that results from defective insulin production, action, or both. According to the Centers for Disease Control and Prevention, in 2014, about 9.3% of the U.S. population (29.1 million people) were affected by diabetes (Centers for Disease Control and Prevention 2014). While there are many possible causes for type 2 diabetes and genetic predisposition plays an important role, the major risk factors include aging, obesity, poor diet, and lack of physical activity. This form of diabetes also tends to usually go undiagnosed because hyperglycemia develops gradually and the classic symptoms of diabetes may not be severe enough for the patient to notice for years. Type 2 diabetes also occurs more frequently in individuals with hypertension and dyslipidemia (high plasma triglyceride concentration, low HDL cholesterol concentration and increased concentration of LDL-cholesterol particles) (American Diabetes Association 2004). Thus, overall cardiovascular health, while influenced by biological factors, is highly contingent on individual health behaviors and self-care, such as following a nutritious diet and physical activity plan.

Arthritis is considered to be the most prevalent in the group of musculoskeletal diseases and is one of the most common sources of disability among U.S. adults. The most common form of arthritis is osteoarthritis, and other common rheumatic conditions include gout, fibromyalgia, and rheumatoid arthritis. (Guccione et al. 1994). In 2010-2012 sample of the NHANES, about 22.7% (52.5 million) of adults reported doctor-diagnosed arthritis. The age-adjusted prevalence was significantly higher among women

(23.9%) relative to men (18.6%). With the aging of the U.S. population, the prevalence of arthritis is expected to continue increasing (Barbour et al. 2013). As previously mentioned, the effects of arthritis on individual health and well-being are significant. Arthritis-related activity and work limitations include difficulty walking, bending, kneeling, climbing stairs, and participating in social activities. Adults with arthritis also report significantly worse health-related quality of life with two to four times as many unhealthy days per month than those not diagnosed with arthritis (Furner et al. 2011).

Unlike cardiovascular disease and type 2 diabetes, arthritis is much less modifiable by individual health behaviors. Age is considered to be one of the strongest predictors of arthritis, even though the actual mechanisms through which age increases the prevalence and incidence of arthritis are poorly understood (Felson et al. 2000). In addition, gender is risk factor as women suffer from hand, foot, and knee osteoarthritis more often than men (Shrikanth et al. 2005). Furthermore, women are more likely to suffer more severe arthritis, especially following menopause (Felson et al. 2000).

Excess weight is the primary modifiable factor that is associated with an increased risk of developing arthritis and its radiographic progression. Arthritis-related limitations are also the highest among obese adults. For example, among normal and underweight adults with arthritis, about 16% reported arthritis-related activity limitations compared to about 31% among obese adults with an arthritis diagnosis (Barbour et al. 2013).

Arthritis, as an additional dependent variable, was added to the analyses to test whether gender, race, and SES moderate the relationship when the health outcome is less contingent of social factors and health behaviors and more on biological pathways. In the case of arthritis, the main risk factors are being a female and old age, therefore, I

hypothesize other social factors (i.e. race and SES) will have no effect on the outcome and will be non-significant moderators. In other words, where social inequality has a lesser opportunity to operate, fundamental social causes should not have much influence on the health outcome.

Social Disparities in Arthritis and CVD Health: Gender, Race, and SES

As I have previously mentioned, the prevalence of arthritis in the United States is primarily distributed along gender and age lines. Out of estimated 22.7% of U.S. adults who reported doctor-diagnosed arthritis, 49.7% were 65 years old or older. Arthritis prevalence was also higher among women relative to men. Moreover, some racial/ethnic variation in the prevalence of arthritis was noted. Specifically, arthritis was more prevalent among White (25.9%) and Black (21.3%) adults relative to Hispanic Americans (12.1%). In addition, individuals unable to work or disabled and those with less education also experienced higher prevalence of doctor-diagnosed arthritis (Bolen et al. 2010).

Over the last few decades, cardiovascular health among American adults has improved overall, however the prevalence of cardiovascular health risk factors are not equally distributed across gender, racial/ethnic and SES lines. Specifically, in 2009-2010, the age adjusted prevalence of hypertension was similar among women and men, however, it varied across race with the highest rates distributed among non-Hispanic Blacks (42.1%) compared to non-Hispanic Whites (28%) and Hispanics (26%) (Nwankwo et al. 2013). Additionally, Black adults tended to develop hypertension at earlier ages, and more Black women had high blood pressure relative to other gender and racial groups (Mozaffarian et al. 2014). In a literature review of studies on the distribution of cardiovascular disease risk factors in the U.S. conducted by Kurian and

Cardarelli (2007), Mexican American men did not have a significantly higher prevalence of hypertension relative to Caucasian men, but Mexican American women had a higher prevalence of hypertension relative to Caucasian women, controlling for age and SES.

Further, national estimates of those diagnosed with type 2 diabetes vary by race/ethnicity. After adjusting for population age differences, in 2010-2012, among individuals aged 20 and older, 7.6% non-Hispanic Whites, 9% Asian Americans, 12.8% Hispanics, and 13.2% non-Hispanic Blacks were diagnosed with type 2 diabetes. Compared to non-Hispanic Whites, Hispanics and non-Hispanic Blacks had a 66% and 77% higher risk of being diagnosed with diabetes, respectively (Centers for Disease Control and Prevention 2011).

In addition, Black women are twice as likely and Black men are about 1.5 times more likely to develop type 2 diabetes relative to their Caucasian counterparts, controlling for age, BMI, waist-to-hip ratio, and family history (Brancati et al. 2000). The scholars also note that "racial differences in potentially modifiable risk factors, particularly adiposity, accounted for 47.8% of the excess risk in African-American women but accounted for little of the excess risk in African-American men" (p.2253). In a study using a sample representative of the U.S. population, Black and Mexican American women had higher fasting plasma insulin at equivalent levels of adiposity relative to their Caucasian counterparts, while the relationship was not significant for men. For Black women, the risk of developing diabetes was higher at lean and obese weight relative to Caucasian women, whereas for Mexican American women, being overweight had the largest effect on increased risk, controlling for household income (Palaniappan, Carnethon, and Fortmann 2002). In a different study, (Carnethon et al.

2002) scholars found that non-obese Black women had higher levels of fasting insulin than their non-obese Caucasian counterparts, which could potentially explain the higher incidence of diabetes among non-obese African American women but not men.

Low high-density lipoprotein (HDL) cholesterol is another major risk factor for coronary heart disease. In 201-2014, about 19% of adults at age of 20 had HDL cholesterol levels that are considered too low. Between the two genders, the percentage of adults with low HDL cholesterol was higher for men (27.9%) than for women (10.0%). Among the racial/ethnic groups, the percentage of individuals with low HDL levels was the highest among Hispanic Americans (21.1%), followed by non-Hispanic Whites (19.1%), and non-Hispanic Blacks (13.6%). Additionally, in the same racial groups, the percentages were lower for women relative to men. Among men and women, the percentage was lower for non-Hispanic Blacks relative to Whites and Hispanics (Carroll et al. 2015).

Cardiovascular health disparities are also patterned across SES lines (Lewey and Choudhry 2013). This pattern could be explained by the compounding social, environmental, behavioral, and psychosocial risk factors. In particular, lower SES neighborhoods have less access to healthy food options and recreational facilities, and are more exposed to alcohol and tobacco advertising (Woolf and Braverman 2011). In addition, lower SES groups usually have limited access to high quality health care and lack adequate health insurance, which are associated with lower awareness and control of CVD risk factors and higher rates of cardiac evens (Fowler-Brown et al. 2007). For instance, Chang and Lauderdale (2009) have demonstrated how income disparities in cholesterol levels changed after the introduction of a new drug technology – statins. The

scholars found that the SES gradient in cholesterol levels initially was positive, but became inverse later, indicating that those with more resources gained more control over their cholesterol levels in the era of statin use. In addition to limited access to new technology to prevent or better manage chronic conditions, individuals of lower SES are exposed to stressors that may directly influence cardiovascular health via activation of stress-response systems (McEwen, 1998) and indirectly via unhealthy coping behaviors, such as smoking and consumption of alcohol and fatty foods (Albert, Glynn, & Ridker, 2003; Nettleton, et al., 2006).

Ultimately, these research findings make a number of connections to fundamental social causes of disease (FSCD) theory. Specifically, higher prevalence of obesity-related cardiovascular health risks are more prevalent among ethnic and racial minority populations and individuals of lower SES. While disparities in health outcomes ascribed to race/ethnicity are to a large degree driven by socio-economic inequalities, they are not eliminated when income, education, health insurance coverage, and geographic location are taken into account (Bostean et al. 2013). The remaining differences could potentially be attributed to biological differences in the presentation of cardiac symptoms (King, Khuan, Quan 2009) as well chronic stress associated with experiences of racial discrimination, barriers to CVD diagnosis, and lower quality health care (Wyatt et al. 2003).

The Interplay between Distal and Proximate Risk Factors: What to Expect?

Fundamental social causes of disease (FSCD) approach suggests that individuals who are better off are more likely to avoid health risks by adopting preventative strategies that are available to them. Despite the vast research that demonstrates a robust inverse

association between SES and chronic health, less is know how social factors and health risk factors interact to produce differences in health outcomes. By placing the fundamental causes of gender, race/ethnicity and SES at the center of this research, this study will examine the ways that distal and proximate factors independently and simultaneously influence the effect of obesity on five health outcomes: arthritis, systolic and diastolic blood pressure, fasting plasma glucose, and HDL cholesterol.

There are three theoretically grounded explanations for how distal and proximate risk factors may interact to affect health. First, gender, race/ethnicity, and SES, as well as BMI, may independently affect each health outcome in question, net of the others. That is, social status characteristics may neither amplify nor diminish the effect of BMI on individual health.

Second, it is also possible that social status characteristics amplify the negative effects of BMI for those who are already more disadvantaged – including women, people of color, and low SES individuals. Higher SES groups who are more educated and affluent have more resources at their disposal to counteract the negative effects of BMI by, for instance, eating a healthier diet and exercising regularly, thereby maintaining a better CVD profile. In contrast, individuals who are in socially disadvantaged positions, already have poorer overall health, therefore, the additional burden of increased BMI may exert a stronger health damaging effect. Support for the risk amplification effect has been corroborated by a number of studies. For instance, Pampel and Rogers (2004) found that consequences of smoking were worsened by low SES. In addition, Krueger and Chang (2008) demonstrated that physical inactivity was more health-damaging to those with fewer resources. In a similar line of work, smoking and alcohol consumption were found

to elevate the health risks among Black but not White American adults (Haiman et al. 2006; Sempos et al. 2003; Stranges et al. 2004). Finally, Shafer and Ferraro (2011), in their study focusing the conjoint effects of BMI and education on C-reactive protein (CRP; a measure of chronic inflammation and disability, found that the risk factor amplification effect on health varied not only across social status indicators but also health outcomes. Specifically, they found that the educational gradient amplified the negative effect of BMI on C-reactive protein (CRP) at lower levels of obesity, but failed to amplify the effect of BMI at levels of severe obesity. However, disparities in education amplified the effect of BMI on disability among individuals with high BMI. Overall, these findings suggests that social inequalities have more "room" to operate in creating health disparities at lower levels of BMI if a health outcome has a tighter biological link with obesity(e.g. C-reactive protein). In contrast, in the case of disability, the effect of educational disparities is more pronounced at the higher end of the BMI distribution and matters much less at the level of overweight because the link between obesity and disability is much "looser".

Alternatively, while less support exists for the third explanatory mechanism, lower BMI among lower status groups may have little impact on their CVD health. Individuals of lower SES generally lead less healthy lifestyles, work in more dangerous jobs, experience more stress, and have limited access to quality health care; thus, the effect of additional few pounds may be negligible. In other words, having "normal" weight may bring little health advantage given their already less advantageous life circumstances. According to Blaxter (1990), "unhealthy behaviour does not reinforce disadvantage to the same extent as healthy behaviour increases advantage," (p. 233), thus

maintaining a "normal" body weight would advance the health of higher SES individuals more so than those of lower SES. Blaxter's argument was confirmed in his study of British adults (1991). She found that smoking had a more detrimental health effect on non-manual workers relative to manual workers. Similarly, Duncan, Jones, and Moon (1993) showed that in higher-SES communities, health differences between smokers and non-smokers were amplified, while the differences were less apparent in lower SES communities. Scholars have also found that despite higher levels of obesity among Black adults, obesity was more strongly related to mortality among White but not Black women (Hogue 1987). Finally, in a more recent study among older adults, Schafer, Ferraro, and Williams (2011) demonstrated that the influence of BMI on C-reactive protein levels was amplified for individuals of higher SES (higher levels of education and net worth), providing further support for Blaxter's argument.

Research Questions and Guiding Hypotheses

Based on the previously discussed research findings, the hypothesized associations among the study variables are provided below. The hypothesized conceptual models are provided in Figures 3.1 and 3.2.

Research Question 1: Does race/ethnicity and gender shape the SES gradient in obesity?

Supporting Research Questions:

a) Is the relationship between education and BMI moderated by gender?

b) Is the relationship between education and BMI moderated by race/ethnicity?

c) Is the relationship between education and BMI moderated by gender and race/ethnicity simultaneously?

d) Is the relationship between income and BMI moderated by gender?

e) Is the relationship between income and BMI moderated by race/ethnicity?

f) Is the relationship between income and BMI moderated by gender and race/ethnicity simultaneously?

Guiding Hypotheses:

H1a) Gender will moderate the relationship between education and BMI, such that women with lower levels of educational achievement will have higher estimated BMI, relative to men.

H1b) Race/ethnicity will moderate the relationship between education and BMI, such that less educated racial minorities will have higher estimated BMI, relative to less educated Whites.

H1c) Gender and race/ethnicity will moderate the relationship between BMI and education, such that the least educated women of color will have the highest estimated BMI relative to other least educated gender and racial/ethnic groups. In addition, the most highly educated women of color will have higher estimated BMI, relative to the lowest educated White men and women. Differences in education will have little effect on BMI among men in each racial/ethnic group.

H1d) Gender will moderate the relationship between BMI and income, such that women in lower income categories will have higher estimated BMI, relative to men in lower income categories.

H1e) Race/ethnicity will moderate the relationship between income and BMI, such that racial minorities in the lowest income categories will have higher estimated BMI, relative to poor Whites.

H1f) Gender and race/ethnicity will moderate the relationship between income and BMI, such that women of color in the lowest income categories will have the highest estimated BMI relative to other poorest gender and racial/ethnic groups. In addition, women of color in the highest income categories will have higher estimated BMI relative to men and women of all other racial/ethnic groups in the lowest income categories. Differences in income will have little effect on BMI among men in each racial/gender group, even though the relation is expected to be positive to racial/ethnic minorities.

Research Question 2: Do distal social factors, i.e. SES, race/ethnicity, and gender shape the effect of obesity on arthritis and cardiovascular health indicators, such as diastolic and systolic blood pressure, fasting plasma glucose and HDL cholesterol?

Supporting Research Questions:

a) Is the relationship between BMI and each of the five health outcomes moderated by gender?

b) Is the relationship between BMI and each of the five health outcomes moderated by race/ethnicity?

c) Is the relationship between BMI and each of the five health outcomes moderated by education?

d) Is the relationship between BMI and each of the five health outcomes moderated by income?

Guiding Hypotheses:

H2a) Gender will not moderate the relationship between BMI and the CVD risk factors but it will moderate the relationship between BMI and arthritis, such that women of higher BMI will be more likely to be diagnosed with arthritis.

H2b) Race/ethnicity will moderate the relationship between BMI and the CVD risk factors, such that Black and Hispanic Americans will be at an increased health disadvantage with increasing BMI, relative to Whites.

H2c) Education will moderate the relationship between BMI and the CVD risk factors, such that less educated individuals will be at an increased health disadvantage with increasing BMI, relative to more educated groups.

H2d) Income will moderate the relationship between BMI and the CVD risk factors, such that individuals at lower income groups will be at an increased health disadvantage with increasing BMI, relative to those in higher income categories.

Broadly, this dissertation will extend the fundamental social causes of disease (FSCD) theory by considering the multiplicative effects of SES, race/ethnicity, and gender to analyze sub-group variations in obesity, and subsequently, five obesity-related health outcomes. Although the association between social status and obesity has been well established, understanding of the mechanisms underlying this relationship is limited. Previous efforts to examine this link tended to focus on additive models without the consideration of multiplicative effects between social factors. An important first step in better understanding the social distribution of obesity is to recognize the simultaneous interaction of social processes underlying the variations in individual body weight.

It is also unclear if and to what extend distal and proximate health risk factors influence more and less controllable obesity-related health outcomes. In other words, which social groups experience the most health-damaging effects of obesity? And, does the health damaging effect of obesity vary between more and less preventable health conditions? Existing research cannot fully answer these questions for similar reasons; it has generally relied on traditional additive modeling approaches, which do not allow for the simultaneous evaluation of distal and proximate health risk factors. Because distal and proximate risks cluster together within individuals, it is essential to use analytic methods that explicitly examine their joint influences. This approach is also useful for identifying how proximate health risk factors, such as BMI, interact with group-level characteristics like gender, race/ethnicity, and class that may have varying influence on more and less controllable health outcomes.

By placing the fundamental social causes of disease approach (FSCD) as the guiding framework, this dissertation seeks to provide a more comprehensive understanding of the individual and compound significance of gender, race/ethnicity, and class on the social distribution of obesity and certain obesity comorbidities. The choice of the FSCD framework seems appropriate given that it places structural mechanisms known to be distal social causes of health disparities at the center of analysis. In the following two empirical chapters (Chapters 5 and 6), this project seeks to answer these questions using a 2011-2014 nationally representative sample of American adults. Chapter 5 examines the extent to which social factors interact to influence variation in BMI. Building on the findings from Chapter 5, Chapter 6 examines whether gender, race/ethnicity and SES moderates the BMI-comorbidity link

Table 3.1. Summary Research Questions and Hypotheses

Research Question	Supporting Research Questions	Research Hypotheses
1) Does race/ethnicity and gender shape the SES gradient in obesity?	a) Is the relationship between education and BMI moderated by gender?	H1a) Gender will moderate the relationship between education and BMI, such that women with lower levels of educational achievement will have higher estimated BMI, relative to men.
	b) Is the relationship between education and BMI moderated by race/ethnicity?	H1b) Race/ethnicity will moderate the relationship between education and BMI, such that less educated racial minorities will have higher estimated BMI, relative to less educated Whites.
	c) Is the relationship between education and BMI moderated by gender and race/ethnicity simultaneously?	H1c) Gender and race/ethnicity will moderate the relationship between BMI and education, such that the least educated women of color will have the highest estimated BMI relative to other least educated gender and racial/ethnic groups. In addition, the most highly educated women of color will have higher estimated BMI, relative to the lowest educated White men and women. Differences in education will have little effect on BMI among men in each racial/ethnic group.
	d) Is the relationship between income and BMI moderated by gender?	H1d) Gender will moderate the relationship between BMI and income, such that women in lower income categories will have higher estimated BMI, relative to men in lower income categories.
	e) Is the relationship between income and BMI moderated by race/ethnicity?	H1e) Race/ethnicity will moderate the relationship between income and BMI, such that racial minorities in the lowest income categories will have higher estimated BMI, relative to poor Whites.
	f) Is the relationship between	H1f) Gender and race/ethnicity will moderate the

	income and BMI moderated by gender and race/ethnicity simultaneously?	relationship between income and BMI, such that women of color in the lowest income categories will have the highest estimated BMI relative to other poorest gender and racial/ethnic groups. In addition, women of color in the highest income categories will have higher estimated BMI relative to men and women of all other racial/ethnic groups in the lowest income categories. Differences in income will have little effect on BMI among men in each racial/gender group, even though the relation is expected to be positive to racial/ethnic minorities.
2. Do distal social factors, i.e. SES, race/ethnicity, and gender shape the effect of obesity on arthritis and cardiovascular health indicators, such as diastolic and systolic blood pressure, fasting plasma glucose and HDL cholesterol?	a) Is the relationship between BMI and each of the five health outcomes moderated by gender?	H2a) Gender will not moderate the relationship between BMI and the CVD risk factors but it will moderate the relationship between BMI and arthritis, such that women of higher BMI will be more likely to be diagnosed with arthritis.
	b) Is the relationship between BMI and each of the five health outcomes moderated by race/ethnicity?	H2b) Race/ethnicity will moderate the relationship between BMI and the CVD risk factors, such that Black and Hispanic Americans will be at an increasedhealth disadvantage with increasing BMI, relative to Whites.
	c) Is the relationship between BMI and each of the five health outcomes moderated by education?	H2c) Education will moderate the relationship between BMI and the CVD risk factors, such that less educated individuals will be at an increased health disadvantage with increasing BMI, relative to more educated groups.
	d) Is the relationship between BMI	H2d) Income will moderate the relationship between

	and each of the five health outcomes moderated by income?	BMI and the CVD risk factors, such that individuals at lower income groups will be at an increased health disadvantage with increasing BMI, relative to those in higher income categories.

Table 3.2. Classification of Overweight and Obesity by BMI, Waist Circumference, and Associated Disease Risk

	BMI (kg/m^2)	**Obesity Class**	**Men ≤102 cm (≤ 40 in.) Women ≤88 cm (≤ 35 in.)**	**Men >102 cm (>40 in.) Women >88 cm (>35 in.)**
Underweight	18.5		-----	-----
Normal	18.5 - 24.9		-----	-----
Overweight	25.0 - 29.9		Increased	High
Obesity	30.0 - 34.9	I	High	Very High
	35.0 - 39.9	II	Very High	Very High
Extreme Obesity	≥40	III	Extremely High	Extremely High

Source: WHO (1995)

Figure 3.1. Hypothesized Moderation models with BMI as the Dependent Variable.

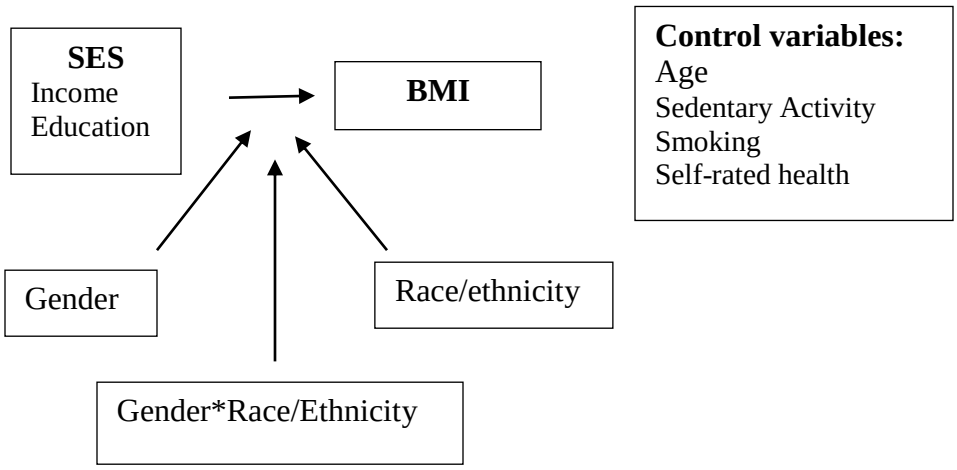
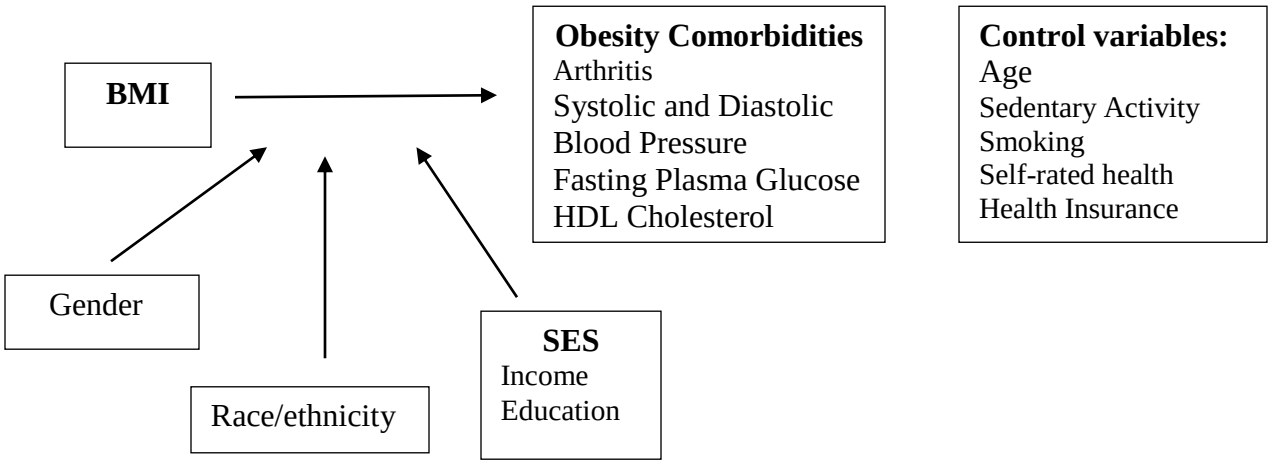


Figure 3.2. Hypothesized Moderation Models with Obesity Co-Morbidities as Dependent Variables



CHAPTER 4: METHODOLOGY

The previous chapter reviewed the existing literature pertaining to obesity and obesity-comorbidities, with a special focus on fundamental social causes of disease (FSCD) theory and its application to SES-obesity and SES-obesity-comorbidity links. The specific research questions that will be analyzed in each of the analytic chapter are described in greater detail in chapter summaries. In addition, detailed research questions and sample hypotheses were introduced the end of Chapter 3. This chapter will detail the methods that will be used to answer the research questions identified in the introductory chapter, beginning with a discussion of the study sample, data collection techniques, and analytic procedures. Chapter 4 will also include descriptive sample statistics.

Data

The data for this research project came from the 2011-2014 cycle of The National Health and Nutrition Examination Survey (NHANES)[1]. NHANES is one of the health and nutrition surveys conducted since 1970s by the National Center for Health Statistics (NCHS), part of the Centers for Disease Control and Prevention (CDC). Data for the NHANES surveys were collected periodically between 1971 and 1994, and in 1999, it became continuous. About 5,000 individuals belonging to all age groups are interviewed at home and complete the health examination component of the survey in a mobile examination center (MEC). Each year's sample as well as any combination of NHANES samples are nationally representative of the resident, non-institutionalized U.S. population. NHANES data are released in two-year cycles in order to have sufficient sample size to obtain stable estimates for certain population subgroups.

[1] http://wwwn.cdc.gov/nchs/nhanes/search/nhanes11_12.aspx

NHANES data are collected by using complex, multistage, probability sampling techniques to select participants residing in each state and D.C. The sampling procedure consists of 4 main stages. First, primary sampling units (PSUs) are selected from strata geographically or by proportions of minority populations. A PSU is usually represented by a county or a group of contiguous counties selected with probability proportional to size (PPS). The majority of strata contain two PSUs. Second, PSUs are divided into segments, usually city blocks of their equivalents, and each segment is selected with PPS. Third, households in each segment are listed and then drawn randomly. Probability of selection of groups is greater in geographic areas where probability of selection for those groups for over-sampling is high. Finally, individuals are chosen from the list of persons residing in selected households. Prospective participants are drawn at random within designated age-sex-race/ethnicity screening sub-domains. On average, 1.6 persons are selected per household.

In order to increase the reliability and precision of health status estimates for certain population sub-groups of particular public health interest, NHANES design includes oversampling. In the 2011-2014 survey, the over-sampled groups included Hispanic persons, Non-Hispanic black persons, Non-Hispanic Asian persons, Non-Hispanic white, and Other individuals at or below 130 percent of the federal poverty level, as well as Non-Hispanic white and Other persons aged 80 years and over. Application of weighting schemes provided by NHANES allows for the estimates from these subgroups to be representative of the relative proportions of these groups in the population as a whole, as the weights account for oversampling and survey nonresponse. NHANES constructed sample weights that take into account survey non-response, over-

sampling, post-stratification, and sampling error. In 2011-2012 a total of 13,431 individuals were selected from 30 different study locations for data collection. Of those selected, 9,756 completed the interview and 9,338 completed the examination at the Mobile Exam Center (MEC). In 2013-2014, 14,332 persons were selected for NHANES from 30 different study locations. Of those selected, 10,175 completed the interview and 9,813 were examined. The total merged sample consisted of 19,931 individuals.

Data Collection Instruments

The screener questionnaire was administered on the doorstep of the household in order to determine the eligibility of survey participants. The relationship questionnaire (or the Screener Module 2) was administered in the selected household to determine the family "units" (e.g. married couple, living together as married) based on questions about the relationships of household members within each household. The family questionnaire was completed for every family unit sampled within the household. The respondent for the family questionnaire was typically the head of household and had to be an adult family member (aged 18 years or older) or an emancipated minor. The family questionnaire was designed to gather household-level data, such as demographic background, occupation, household characteristics, income, and consumer behavior. The sample person questionnaire was administered to all individuals residing in the household. However, eligibility for certain sections of the questionnaire was contingent on age and gender. Participants who were 16 or older as well as emancipated minors were interviewed directly. Survey participants who were under the age of 16 or could not answer the questions themselves, were required to have an adult proxy to provide

information for the survey. The sample person questionnaire was designed to collect data on health conditions and behavior, as well as medication use.

Persons selected to participate in the NHANES survey were also asked to come to a Mobile Exam Center (MEC) for a variety of physical tests and measurements, such as weight, height, and body measurements as well as tests for upper body muscle strength and respiratory health, dental examination and total body scans. Dietary recall data was also collected at the MEC, which involved an extensive interview about the person's food intake over the previous 24 hours. Additionally, data was collected from biospecimens (blood, urine, and other types of specimens) at the MEC to obtain more information about individuals' health and nutritional status. Eligibility for specific laboratory tests was contingent on respondents' gender and age at the time of screening. Generally, information was collected from individuals who were 8 years of age and older. The controlled environment of the MEC allowed for the laboratory data to be collected under identical conditions at each survey location. Survey participants were randomly assigned to exams in the morning or afternoon session, or in the afternoon or evening sessions.

During the visit to MEC, additional information was collected on more sensitive topics, such as reproductive health and alcohol or tobacco use. The MEC interview was conducted in a private setting and consisted of two questionnaires: (a) Computer-Assisted Personal Interviewing (CAPI) Questionnaire conducted by an MEC interviewer, and (b) Audio-Computer-Assisted Self-Interviewing (ACASI) Questionnaire, which allowed the respondents to hear questions through headphones and to read questions on a computer screen in their choice of language. Due to the sensitive nature of the questions in the ACASI questionnaire, no proxies or interpreters were used.

Interviewer Training, Data Quality Insurance, and Questionnaire Languages

All NHANES interviewers were required to complete special training program, which included general interviewing techniques, role-playing exercises, and practice interviews with live respondents – all of which were monitored and evaluated by NCHS staff. In addition, interviewers received a series of cultural competency trainings to help them recognize and respect cultural differences. Further, to ensure data quality, interviewers were frequently observed in the field by their supervisors and NCHS staff to verify that the interviewing protocol was administered correctly. If necessary, interviewers were retrained on survey procedures. Periodically, the interviewers were required to record interviews so that they could be reviewed by NCHS staff. After collection, interview data was reviewed by the NHANES field office staff for accuracy and completeness.

All NHANES questionnaires were translated into Spanish and could be administered in either English or Spanish based on the preference of the respondent. Starting in 2011, selected survey materials were translated into Mandarin Chinese, both traditional and simplified, Korean, and Vietnamese due to over-sampling of Asian-Americans. All translations were carried out using the Institutional Review Board (IRB) approval. A large number of the household interviewers and all MEC interviewers were bilingual in English and Spanish. The respondent could choose their preferred language. Mentally impaired individuals, or participants who were unable to understand English, Spanish, Korean, Vietnamese, or Chinese (traditional/mandarin, simplified/mandarin, or traditional/Cantonese) were not eligible to participate.

Data Recording and Analytic Procedures

NHANES household interview and MEC interview data were recorded electronically. After data collection was completed, the interview data files were transmitted electronically to a central survey database system. The NHANES automated interview systems have built-in edits and checks for many question response options. When unusual, inconsistent, or unrealistic responses were observed, the interviewer was notified immediately and instructed to verify or edit the initial response. During data preparation, variable frequency counts were checked, questionnaire "skip" patterns were verified, and the reasonableness of responses to the questions, and interviewer comments were reviewed. Edits were made to some variables to ensure the completeness, and analytic consistency of the data. Edits were also made, when necessary, to address data disclosure concerns.

Strengths and Limitations of the Data Set

One of the biggest advantages of NHANES that it contains both, the interview component, which consist of questions about socio-demographic, socio-economic characteristics and health-related questions, and the examination component, which consists of laboratory tests and medical, and physiological measurements that are performed by adequately trained personnel. This allows researchers to use accurate data when determining the social distribution of not only diagnosed risk factors and chronic conditions but also undiagnosed ones. It should be noted that while survey physicians do not diagnose medical conditions, they do inform survey participants and refer to a local health center if a health problem is suspected. Specifically for this research project the strength of the data lies in availability of anthropometric measures investigating the SES-

obesity link and biomedical cardiovascular health indicators for investigating the SES-obesity-comorbidity relationship. Additionally, because two cycles, the 2011-2012 and 2013-2014 are merged for all the analyses, reliable estimates can be calculated when conducting sub-group analyses, and conclusions can be generalized to the U.S. population.

One of the limitations of NHANES is cross-sectional design, which allows the possibility of reverse causality bias. To the extent that body weight affects SES, researchers identify a positive relationship between body weight and income among men and negative for women (Averett and Korenman 1996; Baum and Ford 2004; Cawley 2004). Conley and Glauber (2005) have found that increased body mass is associated with decline in women's wages, family income, and the probability of marriage. Even though the mechanisms through which body weight influences one's social standing are not clear, Glass, Hass and Reither (2010) found that heavy women received less post-secondary education than their thinner peers, which in turn adversely affected their occupational prestige.

Another limitation of this data set is limited socio-economic indicators and a lack of psychosocial measures – including racial and gender discrimination, chronic stress, self-efficacy, and mastery experiences – which could potentially mediate the relationship between SES, race/ethnicity, gender, and obesity. Because such variables were not available, the mechanisms through which social factors affect diverse outcomes could not be examined. Finally, due to the small sample size of some ethnic groups, such as Asian or multi-racial, they were recoded as "Other" racial/ethnic category and excluded from the analyses.

Measures

Dependent variables

Body Mass Index (BMI). In order to answer the first research question, I used the continuous BMI measure as the dependent variable. The measure was constructed by dividing weight in kilograms by height in meters squared. Weight and height was measured by survey physicians in the Mobile Examination Center (MEC).

In order to answer the second research question, I used a total of 5 variables. They included four continuous biomedical indicators of cardiovascular health that were available in NHANES – diastolic and systolic blood pressure, fasting plasma glucose, concentration and HLD cholesterol – and a self-reported dichotomous measure of osteoarthritis. The chosen four health risk factors have been identified as robust indicators of cardiovascular health (Lloyd-Jones et al. 2010) and have shown a strong association with BMI (Lavie, Milani, and Ventura 2009). In addition, the association between increased BMI and arthritis has been well documented (Shih 2006).

Systolic and Diastolic Blood Pressure. The first two dependent variables, diastolic and systolic blood pressure were calculated by averaging the data from three, or in some cases four, blood pressure readings collected during the examination at the MEC. After resting quietly for five minutes, three consecutive blood pressure readings were obtained from the study participants. In cases where the blood pressure measurement was incomplete or interrupted, a fourth measurement was taken. According to American Heart Association, and individual is diagnosed with hypertension if their average diastolic blood pressure is 90 mm/Hg or more and/or their average systolic blood pressure is 140 mm/Hg or more (American Heart Association 2013).

Fasting Plasma Glucose. The third dependent variable, fasting plasma glucose, was used as an indicator of type 2 diabetes. Fasting plasma glucose data was collected in the MEC, processed, stored, and shipped to the University of Missouri-Columbia, for analysis. Because glucose was measured in a fasting subsample of persons 12 years and older, fasting sampling weights were used in any analyses including glucose measure. Individuals with fasting glucose levels higher than 126 mg/dL are diagnosed with diabetes (American Diabetes Association 2010).

High Density Lipoprotein Cholesterol (HDL). The fourth continuous dependent variable, total cholesterol, was used as an indicator of blood lipid levels. HDL cholesterol is considered to be "good cholesterol" as it protects against heart disease. Data for LDL cholesterol was also available, but only in the 2011-2012 NHANES wave, therefore, only HDL cholesterol measure was used in the analyses. Blood lipid data was collected in the MEC, processed, stored, and shipped to the University of Missouri-Columbia for analysis. The desirable HDL cholesterol levels for optimal cardiovascular health should be above 60 mg/dL (National Cholesterol Education Program 2001).

Arthritis. Survey respondents were asked if the doctor has ever told them that they had arthritis. The answers to the question were coded into a dummy variable with 1=Yes and 0=No (reference category).

Independent variables

Socio-demographic characteristics included gender, race, age and place of birth.

Gender. Gender was measured as a binary variable with 1 indicating female and 0 indicating male (reference category).

Race/ethnicity. The original race/ethnicity variable was recoded into four categories, representing Whites, African-Americans, Hispanics, and Others. Due to potential small cell issues, Mexican Americans and Other Hispanics were merged into one category of Hispanics. The Others category, including Asians and multi-racial individuals, was excluded from the analyses due to a very small sample size.

Age. Age of respondent was included as a continuous variable ranging from 25 to 80. Individuals older than 80 were coded as 80.Younger individuals were excluded from the analyses in order to create a sample of respondents who have had the opportunity to achieve higher education.

Marital Status. Marital status was measured as a categorical variable with 1=Married/Living together as married, and 2=Separated/Divorced/Widowed, and 3=Never married (reference category).

Place of Birth. Finally, a dummy variable of place of birth will indicate whether the respondent was born in the United States (1), with being born somewhere else representing the omitted category in the analyses (0).

As mentioned previously, income and education were used as primary indicators of **socio-economic status**.

Education. Survey participants aged 20 and above were asked about their highest educational achievement, and their responses were recoded into 4 categories with 1=High school or less (reference category), 2=High school or GED, 3=Some college, and 4=College or higher.

Poverty to Income Ratio (PIR). This measure was provided by NHANES, calculated based in The Department of Health and Human Services (HHS)

poverty guidelines, which are issued each year in the Federal Register. PIR was calculated by dividing family (or individual) income by the poverty guidelines specific to the survey year. Income to poverty ratio ranged between 1 and 5, with values above 5 recoded into 5. Ratios below 1 indicate that the income for the respective family is below the official definition of poverty, while a ratio of 1.00 or greater indicates income above the poverty level. A ratio of 1.25, for example, indicates that income was 125% above the poverty threshold. In the sample PIR had a mean of 2.84 and a standard deviation of 1.68.

Because **individual health behaviors and characteristics** differ across the socio-economic gradient, they were adjusted for in the analyses (Lantz et al. 2001; Pampel et al. 2010). All health behavior measures were based on self-reports.

Smoking. Participants who have reported having ever smoked a cigarette but who do not smoke currently were classified as former smokers, participants who have indicated that they smoke currently were classified as current smokers, while those who do not currently smoke and have never smoked in their lifetime, were classified as never smokers. In the analyses, individuals who have never smoked in their lifetime were used as a reference category.

Sedentary Behavior. Sedentary behavior was measured as a continuous variable asking the respondents to indicate the "Number of minutes spent sitting on a usual day (excluding sleep)." This variable was recoded in to a measure of the number of hours spent sitting on a usual day. This measure had a mean of 6.83 and a standard deviation of 3.29 with a range between 0 and 16.

Self-Rated Health. Subjective health status was measured by asking survey respondents, "Would you say your health in general is…?. The answer categories were coded into 1=Excellent, 2=Very Good, 3=Good, 4=Fair, and 5=Poor. Perceived fair or poor health status was used as the reference category in the analyses.

Health Insurance. Health insurance status was measured as a binary variable with 1=Respondent had health insurance and 0=Respondent did not have health insurance (reference category).

Analytic approaches

The 2011-2014 cycle of NHANES data was downloaded from the Centers for Disease Control website and imported into Stata, version 13 for statistical analyses. *Svyset* command was used to declare the data as complex survey data, specify the variable that contains identifiers for the primary sampling units (PSUs), and 4-year sample weights were calculated and specified. A *svy* prefix was added before running all descriptive and inferential analyses in order to achieve reliable estimates due to complex survey design. The standard errors were estimated using Taylor series linearization, which is a method that incorporates sample weights and accounts for complex sample design.

Sample weights for NHANES participants include adjustments for unequal selection probabilities and certain types of non-response (in-home interview or examination at MEC), as well as an adjustment to estimates of population sizes for specific age, sex, and race/ethnicity categories. Two sets of sample weights were included in the demographics data file, and were used in the majority of analyses. In addition, special subsamples have their own sample weights, which account for the

additional probability of selection into the subsample, as well as the additional non-response. Because it is recommended to use the weight of the smallest sample subpopulation, the fasting glucose subsample weights were used for any analyses that included such data. Additionally, glucose levels were measured for a smaller sample (subsample) of individuals, which was done to reduce participant burden. Nonetheless, each subsample was selected to be a nationally representative sample. Participants aged >=25 years who were not pregnant at the time of examination and had valid data on all main outcome variables were included in the final sample for analyses. It consisted of about 9,809 participants (48.24% female and 51.76% males). As previously mentioned, descriptive analyses will be provided further in this chapter. Specifically, means and standard deviations will be calculated for the continuous variables, and percentages will be provided for the categorical variables. Bivariate analyses will also be performed at the beginning of each analytical chapter to establish group differences between the study variables.

To address the first research question, "Does race/ethnicity and gender shape the SES gradient in obesity?" a generalized linear modeling (GLM) approach with BMI as a continuous dependent variable was utilized. Generalized linear models are an extension of ordinary linear regression models, and accommodate non-normally distributed continuous variables without manual transformations. GLM uses maximum likelihood estimation (MLE) rather than ordinary least squares (OLS) to estimate the parameters, and thus relies on large-sample approximations. Because the dependent variable was not normally distributed, I used Gamma probability distribution to estimate the models. The coefficients, when using GLM, are not directly interpretable, therefore, I used the

margins command to estimate predicted values of the dependent variable based on different values or levels of the independent variables. After fitting a model, I inspected the data for cases that could potentially be outliers or have high leverage using the Cook's D statistic. Over a thousand of cases were identified as having a Cook's D value higher than 1. I reran the regression without the outliers, however, it had no effect on coefficients or standard errors, and the estimation sample size did not decrease. This indicated that the dropped cases were not relevant to my analyses, therefore I decided not to exclude them from the final analyses.

A total of 7 models were analyzed. In the first model, indicators of socio-economic status (income and education) were modelled as predictors of obesity, controlling for gender, age, race/ethnicity, marital status, country of birth, sedentary activity and smoking status. Because the relationship between age and BMI as well as income and BMI was not linear, I included quadratic terms for these variable. The following six models were tested for two-way and three-way interaction effects between indicators of socio-economic status, gender, and race/ethnicity. In the second model, I tested the interaction term between education and gender. In the third model, I conducted a moderation analysis between income, measured as PIR, and gender. The fourth and fifth models were included to test the interaction effects between race/ethnicity x education and race/ethnicity x PIR. In the final two models, I ran two, three way interaction effects between gender, race/ethnicity, and the two indicators of SES. To the ease of interpretation of interaction effects, I used the *margins* command to estimate predicted values and the *marginsplot* command to graph them.

To address the second research question, "Do distal social factors, i.e. SES, race/ethnicity and gender shape the effect of obesity on osteoarthritis and cardiovascular health indicators, such as diastolic and systolic blood pressure, fasting plasma glucose, and HDL cholesterol?", one binary and a series of OLS regression analyses were conducted with 5 cardiovascular indicators as dependent variables. Unlike BMI, mean systolic and diastolic blood pressure, fasting plasma glucose and total cholesterol were distributed approximately normal. I ran all the models using GLM approach in order to conduct sensitivity analyses, however, regression coefficients and standard errors were very similar, thus, I decided to use OLS approach for the ease of interpretation. A total of five models were analyzed for each of the five dependent variables.

In the first model, each of the dependent variables was regressed on BMI, sociodemographic and socio-economic characteristics, controlling for individual health behaviors, self-rated health and insurance status. After fitting the regression models, I inspected the data for cases that could potentially be outliers or have high leverage using the Cook's D statistic (*predict dbeta* command in Stata for logistic regression and *predict cooksd* command in Stata for OLS regression). I reran the regressions without the influential cases, however, it had no effect on coefficients or standard errors, and the estimation sample size did not decrease. This indicated that the dropped cases were not relevant to my analyses, therefore I decided not to exclude them from the final analyses.

The second model included an interaction term between gender and BMI. The third model included an interaction term between race/ethnicity and BMI. Finally, in the last two models, interaction analyses were conducted to assess the moderation effects between BMI x education and BMI x income. I also tested for three-way interactions

between BMI and significant social factors. Even though some of them did show statistical significance, no clear patterns were observed, therefore they were not included in the study results section. I used the *margins* command to estimate predicted values and the *marginsplot* command to graph them.

All two-way interaction terms, despite their significance, were left in the models, but only significant ones were discussed in the dissertation text. To facilitate better understanding of the results, predicted probabilities were calculated for each model and graphic representations of significant interaction effects were provided. Differences between predicted values were tested with a Wald test, using Bonferroni adjustment for multiple group comparison.

Descriptive Statistics

[Insert Table 4.1 about here]

As displayed in Table 4.1, the average age of the sample was about 50 years, and a little over half of the respondents were women (51.79%). The majority of respondents were White (68.96%), while 11.34% were Black, and 14.27% were Hispanic. Almost two thirds of the sample were married (65.44%) and the majority of them were born in the United States (81.77%). With respect to socio-economic characteristics, there was substantial variation in education, with about one third of the sample having some college or college degree and higher education (30.4)% and 32.45% respectively), while about 16% of the sample had less than a high school education. There was also substantial variation by poverty to income ratio with a mean of 3.00 and a standard deviation of 1.44. Moving forward to individual health indicators, about 50% of the sample had never smoked and about 20% were current smokers. The average survey participant generally

spent about 6 hours sitting per day. Further, about two thirds of the sample perceived their health as excellent, very good, or good. Finally, 82.02% of the sample had health insurance.

The findings indicate that the average BMI in the sample was 29.19 with a standard deviation of 5.95. Based on the BMI cut-offs, about half of the sample were overweight or obese. The mean diastolic blood pressure was 71.16 with a standard deviation of 10.44 and the mean systolic blood pressure was 122.87 with a standard deviation of 15.18. Based on these results, about 16% of the sample qualified for a hypertension diagnosis (Systolic BP>=140 mm Hg or diastolic BP>=90 mm Hg). Further, the mean fasting plasma glucose in this sample was 106.53 with a standard deviation of 29.16, which indicates that about 11% of the sample qualified for a type 2 diabetes diagnosis (Fasting plasma glucose>=126 mg/dL). The average HDL cholesterol in this sample was 53.11 with a standard deviation of 13.92. Based on these findings, about 27% of the sample had HDL blood cholesterol levels that were too low for optimal cardiovascular health (HDL total cholesterol< 60 mg/dl). Finally, almost one third of the sample (27.52%) had been diagnosed with arthritis.

Table 4.1. Weighted Means and Proportions of Sample Characteristics (NHANES 2011-2014, N=9,809)

	Mean (SD)/%	Range
Body Mass Index (BMI)	29.19 (5.95)	13.40-82.90
Diastolic Blood Pressure (mm/Hg)	71.16 (10.44)	0-130
Systolic Blood Pressure (mm/Hg)	122.87 (15.18)	64-234
Fasting Plasma Glucose (mg/dL)	106.53 (29.16)	39-421
Mean HDL Cholesterol (mg/dL)	53.11 (13.92)	10-175
Arthritis (Yes)	27.52%	
Age (25+)	50.44 (13.56)	25-80
Gender:		
Female	51.79%	
Male	48.21%	
Race/Ethnicity:		
White	68.96%	
Black American	11.34%	
Hispanic	14.27%	
Other	5.42%	
Marital Status:		
Married/Living together	65.44%	
Separated/Divorced/Widowed	20.95%	
Never Married	13.61%	
Place of birth		
United States (Yes)	81.77%	
Highest Grade of School Completed:		
Less than High School	16.29%	
High School or GED	20.80%	
Some college	30.45%	
College or More	32.45%	
Poverty to Income Ration (PIR)	3.00 (1.44)	0-5
Smoking Status:		
Current Smoker	19.64%	
Former Smoker	25.75%	
Never Smoked	54.61%	
Sedentary Behavior (hours/day)	6.69 (2.89)	0-16
Subjective Health Rating:		
Excellent	10.67%	
Very Good	31.39%	
Good	39.42%	
Fair	15.75%	
Poor	2.78%	
Health Insurance (Yes)	82.02%	
N	9,809	

CHAPTER 5: DO GENDER AND RACE/ETHNICITY CONDITION THE SES GRADIENT IN OBESITY?

In the previous chapter, univariate statistics were used to describe the distribution of the main variables of interest in the study sample. The central focus of this chapter is to examine whether the effects of gender, race/ethnicity, and SES on BMI are cumulative or multiplicative. Based on the literature discussed in previous chapters, I argue that gender, race/ethnicity, and SES intersect to shape mean BMI outcome. Specifically, I predict that women of color will have the highest predicted mean BMI regardless of their education or income, while White women and men, and especially those of high SES will have the lowest predicted body weight. If the two-way and three-way interactions are not significant, this would suggest that each fundamental cause – gender, race/ethnicity and SES – *independently* contribute to an increase or decrease in mean BMI. On the other hand, significant interactions would mean that fundamental causes *condition each other* in their effect on individual body weight.

To address the first research question, "Does race/ethnicity and gender shape the SES gradient in obesity?" I utilized a generalized linear modeling (GLM) approach with BMI as a continuous dependent variable. Generalized linear models are an extension of ordinary linear regression models, and accommodate non-normally distributed continuous variables without manual transformations. GLM uses maximum likelihood estimation (MLE) rather than ordinary least squares (OLS) to estimate the parameters, and thus relies on large-sample approximations. Because the dependent variable was not normally distributed, I used Gamma probability distribution to estimate the models. The coefficients, when using GLM, are not directly interpretable, therefore, I primarily relied

on the *margins* command in Stata to estimate predicted values of the dependent variable based on different values or levels of independent variables.

I estimated a total of 7 models. In the first model, I included indicators of socio-economic status (income and education) as independent correlates of obesity, controlling for gender, age, race/ethnicity, marital status, country of birth, sedentary activity and smoking status. The following six models focused on testing for two-way and three-way interaction effects between indicators of socio-economic status, gender and race/ethnicity. In the second model, I tested the interaction term between education and gender. In the third model, I conducted a moderation analysis between income, measured as PIR, and gender. The fourth and fifth models, I included interaction effects between race/ethnicity x education and race/ethnicity x PIR. In the final two models, I tested three-way interaction effects between gender, race/ethnicity and the two indicators of SES.

Results from Bivariate Analyses

On average, in the 2011-2014 NHANES, across gender, women had significantly higher mean BMI than men (29.39 and 28.82 respectively) ($p<.001$). Across races, Black adults had a significantly higher mean BMI (31.21) relative to White adults (28.99), and the same pattern applied to Hispanic adults (29.92) ($p<.001$). Each gender and racial/ethnic group had a mean BMI value that was considered "overweight".

[Insert Table 5.1. about here]

Findings from simple linear regression analyses presented in Table 5.1 suggest that SES characteristics differ across gender and race/ethnicity lines. Within White adults, the overall mean BMI did not differ significantly between the two genders.

However, women had lower estimated average BMI relative to men in the highest education (27.19 versus 28.09) ($p<.05$) and income (27.23 versus 28.54) categories ($p<.001$). At the lowest income category, however, women had a significantly higher BMI than men. Among Black adults, women had a significantly higher BMI overall (32.92 versus 29.11) and across all socio-economic indicators ($p<.001$). The only exception was the highest PIR category, where the mean BMI difference did not reach statistical significance. The BMI distribution among Hispanic adults was similar across gender lines, even though Hispanic women had an overall higher mean BMI relative to men (30.29 versus 29.54) ($p<.05$). In addition, Hispanic women who had less than a high school education had a significantly higher mean BMI (30.59 versus 29.50) ($p<.001$). Finally, women in the lowest income category also had a significantly higher BMI relative to men (30.69 versus 29.81) ($p<.05$).

When comparing other gender and racial/ethnic groups to White men, results reveal a clear weight disadvantage among women of color. Mean BMI of Black Women was significantly higher in all SES categories when compared to White men. Among Hispanic women, those in the lowest income and education categories also exhibited a higher BMI relative to White men. Taken together, the patterns of differences in social determinants of health and a variety of other characteristics show that those in privileged positions, namely White men and women, tend to have significantly lower estimated body weight.

Results from Multivariate Analyses

[Insert Table 5.2 about here]

Table 5.2 presents results from multivariate GLM analyses examining additive and multiplicative effects of gender, race/ethnicity and SES the effects on BMI. According to Model 1, the baseline model, being a female was associated with an increase in mean BMI ($p<.001$). For instance, a predicted mean BMI for a woman was 29.69 versus 29.14 for a man, holding other covariates constant. Similarly, racial/ethnic minorities, namely African American and Hispanic adults were estimated to have higher mean BMI relative to White adults ($p<.001$). As an example, the predicted mean BMI for White adults was 29.01, while it was 31.30 and 30.04 for Black American and Hispanic adults, respectively. Increase in age had a non-linear association with BMI. Specifically, increase in age had a positive effect at younger ages and a negative effect at older ages ($p<.001$). The relationship between marital status and BMI did not reach statistical significance in this model. Further, individuals with college or greater levels of education were estimated to have a lower mean BMI relative to those with less than a high school education ($p<.001$). Those with less than high school education had a predicted mean BMI of 29.66, while the mean BMI for those who attended college or more was 20.01. Increase in income, or poverty to income ratio, to be more precise, also had a non-linear association with BMI. Specifically, increase in PIR was associated with an increase in BMI at its lower levels and a decrease in BMI at its higher levels. The predicted fit curve for BMI based on PIR is presented in Figure 5.1. Being born in the United States as opposed to somewhere else was associated with an increase in mean BMI ($p<.001$). With respect to health behaviors, current smokers scored lower on the mean BMI ($p<.001$) and increased engagement in sedentary activity had a positive association with a mean BMI ($p<.001$).

Does the effect of education on BMI vary by gender?

Model 2 presents results from an interaction between gender and education. The significance of the interaction term suggests that the relationship between BMI and education is simultaneously contingent on gender. The predicted mean BMI values are presented in Figure 5.2. Specifically, at the lowest level of education, men had a significantly lower estimated mean BMI relative to women (28.90 versus 30.47) ($p<.001$). However, at the highest level of education women had a lower predicted mean BMI value when compared to men (27.70 versus 28.33) ($p<.05$). Within gender, the difference in mean estimated BMI between the lowest and the highest educational achievement category was significant for men and women, however, the difference was much larger among women (.58 versus .27). This trend indicates that increase in education has a stronger effect on decrease in BMI for women relative to men.

Does the effect of education on BMI vary by race/ethnicity?

Model 3 presents results from an interaction between race/ethnicity and education. The interaction is only significant for Black American adults and only at the educational level of college and above. These results suggest that the relationship between education and BMI is simultaneously contingent on race, however only when comparing White and Black adults. The effect on education on BMI does not differ significantly between White and Hispanic adults. The predicted mean BMI values are presented in Figure 5.3. The results indicate that Black adults with a college or higher education had an estimated mean BMI (31.25) significantly higher than the BMI of White adults with less than high school education (28.99) ($p<.001$). Within race/ethnic groups, White and Hispanic adults with a college or higher education had a significantly lower

estimated mean BMI relative to other education categories, however, within Black adults, the mean estimated BMI did not depend on levels of educational achievement. These findings suggest that the effect of increases in education on BMI is not equivalent across the three racial/ethnic groups. Most importantly, the highest level of education does offer a protective weight effect to White and Hispanic adults, while offers no protective effects to Black Americans.

Does the effect of education on BMI vary by gender and race/ethnicity simultaneously?

Model 4 presents results from a three-way interaction between race/ethnicity, gender and education. The significance of this interaction suggest that the relationship between education and BMI is simultaneously moderated by gender and race/ethnicity. Among men, at the lowest education category, the mean estimated BMI of Hispanic men (28.59) was significantly higher relative to Black (28.15) and White men (28.54) ($p<.05$), however there was no significant difference between Black and White men (see Figure 5.4). At the highest educational category, however, White men had a lower estimated mean BMI (28.14) relative to Hispanic (29.10) and Black men (29.65) ($p<.05$). Among women, at the lowest education level, Black adults had a higher mean estimated BMI (32.09) relative to Whites (29.96) and Hispanics (30.67) ($p<.001$). There was no significant difference between Whites and Hispanics. Within race, estimated mean BMI for Black women who had attended college or more did not differ significantly from those who only had less than a high school degree. White and Hispanic women's mean estimated BMI was significantly lower if they had attended college relative to less than high school. Overall, Black women had a higher estimated mean BMI than White or Hispanic women at all education categories, except for high school or GED.

Between gender comparisons revealed that highest educated Black women (32.09) had a significantly higher BMIs than men regardless of their education level or race. As a comparison, the highest educated White women had a significantly lower estimated mean BMI (27.25), than White men with college education (28.14) and men with less than high school education (28.54) ($p<.05$). In sum, these results suggest that Black women have a weight disadvantage regardless of their educaiton within and between genders and racial/ethnic groups, while White women with at least college education have a weight advantage over all the other groups.

Does the effect of income on BMI vary by gender?

Model 5 presents results from an interaction between gender and income, measured as poverty to income ratio (PIR). Based on these findings, increase in family income was associated with a decrease in mean estimated BMI among women, while no significant change was observed among men. As displayed in Figure 5.5, at the lowest income level, the mean estimated BMI for women was 31.32, while for men it was only 28.49 ($p<.001$). In contrast, at the highest income category, the mean estimated BMI for women was 28.89, while the mean estimated BMI for men was 28.90 ($p<.001$). In sum, women had a consistent significantly higher mean estimated BMI at lower PIR and mid-range PIR categories, even though the difference between men's and women's BMI decreased with increasing PIR, until became statistically insignificantly at PIR of three and above. PIR of three indicates a family income of 300% above the federal poverty ratio, and also is the mean PIR for the full sample.

Does the effect of income on BMI vary by race/ethnicity?

Model 6 presents results from an interaction between race/ethnicity and income, measured as poverty to income ratio (PIR). Based on these results (see Figure 5.6), there was no significant difference in estimated mean BMI among the three racial/ethnic groups at the lowest income category, however, at the highest income category, Black adults had a significantly higher predicted mean BMI (30.94) when compared with Hispanic (29.42) and White (28.09) adults ($p<.001$). The same trend of Black adults having the largest weight disadvantage over Whites and Hispanics held across all, except for the lowest PIR categories. Additionally, the mean BMI was predicted to significantly decrease for Whites and Hispanics, while it remained fairly consistent for Black adults. There were no significant differences in predicted mean BMI between White and Hispanic adults regardless of the PIR category. For instance, while White and Hispanic adults had an estimated mean BMI of about 30 at the lowest PIR category, at the highest PIR category their estimated BMI was about 28 ($p>.001$). Overall, these results suggest that increase in income is associated with a decrease in mean BMI, but only for White and Hispanics adults.

Does the effect of income on BMI vary by gender and race/ethnicity simultaneously?

Model 7 presents the results from a three-way interaction between race/ethnicity, gender, and income, measured as PIR (see Figure 5.7). Among men, at the lowest and highest income levels, there were no significant group differences. Similarly, within racial/ethnic groups, the mean BMI did not differ significantly based on PIR level among White, Black and Hispanic men. Among women, at the lower end of income distribution, no significant group differences were observed. Within racial groups, the mean BMI was

predicted to decrease with the increase in income for White and Hispanic women, but no significant difference was detected for Black women. Actually, Black women's estimated mean BMI remained significantly higher relative to the other two groups, regardless of income level. Across gender and racial/ethnic categories, Black women had a significantly higher mean estimated BMI, when compared to women and men in all racial/ethnic categories and across all income levels.

Chapter Summary

The average body weight in the United States has been on the rise over the past several decades, and has affected individuals of all gender, racial/income and socio-economic groups. However, it is not completely clear which social groups are at an increased risk of becoming obese with the rising average weight of Americans. Previous research has demonstrated that women, people of color, and individuals of lower socio-economic status are more likely to have higher BMIs relative to their White higher SES counterparts. Despite the importance of these findings, few studies have examined the intersecting effects of social factors on obesity, and rather have tended to examine their separate influences, or assume that their effects are additive in nature. The analyses conducted in this chapter extend the work on social disparities in obesity prevalence by addressing the potential multiplicative effects gender, race/ethnicity, and socio-economic status on social distribution of individual BMI. I anticipated that these factors would intersect in ways that would put women of color at an increased risk of obesity regardless of their educational status and income level. In contrast, I expected White men and women of the highest social status to be the most protected from weight gain. My

hypotheses were largely supported, demonstrating the importance of joint effects of multiple social statuses.

Findings of the additive model revealed that being a female and being a racial minority were associated with a higher mean BMI, net of each other and covariates. In contrast, having a college degree or greater level of education and living in more prosperous households were associated with a lower mean BMI, net of each other and covariates. These results were expected given the vast literature demonstrating individual effects of gender, race/ethnicity, and SES on obesity prevalence in the U.S. Research findings became more nuanced once I tested for moderating effects. Two-way and three-way interactions showed that the combined effects of gender and race with SES indicators played a significant role in patterning the mean BMI distribution.

Specifically, education had a strong effect on women relative to men such that at the lower levels of education women had higher estimated BMI scores, yet at the highest level of educational achievement, the mean BMI among women was much lower relative to that of men. The effect of increase in educational achievement was also contingent on race/ethnicity. To be more precise, increase in education offered some protective effect from weight gain to White and Hispanic adults, however Black Americans had an estimated higher mean BMI regardless of their education level. When gender and race/ethnicity were simultaneously interacted with education, only minor variation in BMI was observed among men across all racial/ethnic groups, however, among women, a strong racial pattern emerged. That is, increase in education, especially at the higher end of the distribution, was associated with a sharp decreased in mean predicted BMI among White and Hispanic women, whereas higher educational achievement did not buffer the

race effect among Black women. In addition, Black women were estimated to have higher mean BMI at all levels of education when compared men and women in other racial/ethnic categories.

The effect on income on obesity was also moderated by gender and race/ethnicity, even though the patterns were largely similar to those of education. Between the two genders, women at the lowest income levels had much higher estimated BMI levels relative to men. In fact, their weight was predicted to reach Class I obesity, while economically disadvantaged men were only estimated to be overweight. At the higher income levels, however, the differences became insignificant. With respect to race/ethnicity, increase in income was associated with a decrease in mean BMI among Whites and Hispanics, while the weight of Black Americans was predicted to remain constant at about a Class I obesity level. Finally, the three-way interaction revealed little change in mean estimated BMI among men in all racial/ethnic groups, but a sharp decrease in BMI among White and Hispanic women. Not surprisingly, income had no protective effect from the risk of obesity among Black women. In fact, their mean BMI was estimated to maintain higher relative to individuals within and across other gender and racial/ethnic groups. Overall, consistent with the fundamental causes of disease (FSCD) theory, my findings suggest that different systems of inequality shape individual health outcomes that would be overlooked in more traditional research paradigms focusing on proximate risk factors.

The next chapter will build upon these results by examining the role of BMI at the intersection of gender, race/ethnicity and social class when examining five obesity-related health outcomes – arthritis and four indicators of cardiovascular health. The goal

of this chapter is two-fold. First, I will examine whether the health-damaging effect of obesity is contingent on gender, race/class, and SES. Second, I will examine whether the risk accumulation is dependent on the health outcome in question.

Table 5.1. Mean BMI and Distribution of SES characteristics by Gender/Race (NHANES 2011-2014, N=9,809)[ab]

	WM	WW	BM	BW	HM	HW
Mean BMI	28.99	28.99	29.11	32.92*†	29.54	30.29*
	(4.25)	(5.37)	(9.87)	(11.34)	(6.18)	(8.09)
Highest Grade of School Completed:						
Less than High School	28.76	29.11	28.26	33.40*†	29.50	30.59*†
	(4.83)	(5.59)	(10.69)	(11.55)	(5.24)	(7.18)
High School or GED	29.74	30.35	29.18	32.81*†	29.59	31.31*
	(4.40)	(5.19)	(8.99)	(10.74)	(6.16)	(7.63)
Some college	29.80	30.10	29.39	33.49*†	29.83	30.38
	(4.14)	(5.28)	(8.41)	(9.92)	(6.28)	(7.60)
College or More	28.09	27.19*	30.00†	32.13*†	29.15	27.76
	(3.01)	(3.90)	(7.63)	(9.59)	(4.62)	(6.18)
Poverty to Income Ration (PIR)						
0-3	29.17	30.33*	28.55	33.33*†	29.81	30.69*†
	(5.03)	(5.93)	(9.48)	(10.77)	(5.83)	(7.61)
3-5	28.92	27.98*	30.37†	32.52*†	29.18	29.60
	(3.06)	(3.92)	(8.40)	(9.73)	(5.28)	(6.95)
5	28.54	27.23*†	30.38	32.43†	28.66	28.65
	(2.77)	(3.55)	(7.29)	(11.65)	(4.92)	(7.39)

[a] WM, WW, BM, BW, HM, HW refers to White men, White women, Black men, Black women, Hispanic men, and Hispanic women.

[b] Standard deviations reported in parentheses.

* p<.05 for comparison between men and women within racial/ethnic group

† p< .05 for comparison of race/ethnicity and gender groups to White men

Table 5.2 GLM Regression of BMI on Socio-demographic and Socio-economic Characteristics (N=9,809)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	b	SE	b	SE	b	SE	b	SE	b	SE
Female	.01*	.01	.06***	.01	.01*	.01*	.04*	.02	.09***	.01
Black	.06***	.01	.06***	.01	.05*	.02	-.02	.02	.06*	.02
Hispanic	.05***	.01	.05***	.01	.07***	.02	.08***	.02	.05***	.02
Age	.01***	.01	.01***	.01	.01***	.01	.01***	.01	.01***	.01
Age^2	-.01***	.01	-.01***	.01	-.01***	.01	-.01***	.01	-.01***	.01
Widowed/Divorced/Separated	-.01	.01	-.01	.01	-.01	.01	-.01	.01	-.01	.01
Never Married	.01	.01	.01	.01	.01	.01	-.01	.01	.01	.01
High School or GED	.02	.01	.03	.01	.03	.02	.04*	.02	.02	.01
Some College	.01	.01	.03**	.01	.02	.01	.04*	.02	.01	.01
College and Above	-.06***	.01	-.02*	.01	-.06***	.01	-.02	.02	-.06***	.01
PIR	.01	.01	.01	.01	.01	.01	.01	.01	.02*	.01
PIR^2	-.01*	.01	-.01*	.01	-.01*	.01	-.01*	.01	-.01**	.01
Born in the U.S.	.06***	.01	.06***	.01	.07***	.01	.07***	.01	.06***	.01
Former Smoker	.01	.01	.02*	.01	.01	.01	.02*	.01	.01	.01
Current Smoker	-.08***	.01	-.08***	.01	.01***	.01	-.08***	.01	-.08***	.01
Sedentary behavior	.01***	.01	.01***	.01			.01***	.01	.01***	.01
Female x High School or GED			-.02	.01						
Female x Some College			-.04*	.01						
Female x College and Above			-.08***	.02						
Black x High School or GED					- .02	.02				
Black x High Some College					.01	.02				
Black x College and Above					.07**	.02				
Hispanic x High School or GED					-.03	.03				
Hispanic x Some College					-.03	.02				
Hispanic x College and Above					-.02	.02				
Male x Black x High School or GED							.01	.03		

Male x Black x Some College	.02	.03		
Male x Black x College and Above	.08*	.04		
Male x Hispanic x High School or GED	-.04	.02		
Male x Hispanic x Some College	-.04*	.02		
Male x Hispanic x College and Above	-.02	.02		
Female x White x High School or GED	-.01	.02		
Female x White x Some College	-.04	.02		
Female x White x College and Above	-.07*	.03		
Female x Black x Less than High School	.13***	.03		
Female x Black x High School or GED	.06	.04		
Female x Black x Some College	.08*	.04		
Female x Black x College and Above	.10*	.04		
Female x Hispanic x Less than High School	-.01	.02		
Female x Hispanic x High School or GED	-.04	.03		
Female x Hispanic x Some College	-.07*	.03		
Female x Hispanic x College and Above	-.11*	.04		
Female x PIR x PIR^2			-.03***	.01
Black x PIR x PIR^2				
Male x Black x PIR x PIR^2				
Male x Hispanic x PIR x PIR^2				
Female x White x PIR x PIR^2				
Female x Black x PIR x PIR^2				
Female x Hispanic x PIR x PIR^2				

Note: Omitted categories: Male, White, Married, Not born in the U.S., Never Smoked
*p<.05; **p<.01; ***p<.001

Table 5.2 (Continued) GLM Regression of BMI on Socio-demographic and Socio-economic Characteristics (N=9,809)

	Model 6		Model 7	
	b	SE	b	SE
Female	.02*	.01	.09***	.01
Black	.03	.02	.02	.02
Hispanic	.06**	.02	.06**	.02
Age	.01***	.01	.01***	.01
Age^2	-.01***	.01	-.01***	.01
Widowed/Divorced/Separated	-.01	.01	-.01	.01
Never Married	.01	.01	-.01	.01
High School or GED	.02	.01	.02	.01
Some College	.01	.01	.01	.01
College and Above	-.06***	.01	-.06***	.01
PIR	.01	.01	.02*	.01
PIR^2	-.01*	.01	-.01*	.01
Born in the U.S.	.06***	.01	.06***	.01
Former Smoker	.01	.01	.02*	.01
Current Smoker	-.08***	.01	-.08***	.01
Sedentary behavior	.01***	.01	.01***	.01
Female x High School or GED				
Female x Some College				
Female x College and Above				
Black x High School or GED				
Black x High Some College				
Black x College and Above				
Hispanic x High School or GED				
Hispanic x Some College				
Hispanic x College and Above				
Male x Black x High School or GED				

Male x Black x Some College				
Male x Black x College and Above				
Male x Hispanic x High School or GED				
Male x Hispanic x Some College				
Male x Hispanic x College and Above				
Female x White x High School or GED				
Female x White x Some College				
Female x White x College and Above				
Female x Black x Less than High School				
Female x Black x High School or GED				
Female x Black x Some College				
Female x Black x College and Above				
Female x Hispanic x Less than High School				
Female x Hispanic x High School or GED				
Female x Hispanic x Some College				
Female x Hispanic x College and Above				
Female x PIR x PIR^2	.01**	.01		
Black x PIR x PIR^2	-.01	.02		
Male x Black x PIR x PIR^2			.01	.01
Male x Hispanic x PIR x PIR^2			-.01	.01
Female x White x PIR x PIR^2			-.03***	.01
Female x Black x PIR x PIR^2			.01	.01
Female x Hispanic x PIR x PIR^2			-.03***	.01

Figure 5.1 Predicted Mean BMI Values by Poverty to Income Ratio (PIR)

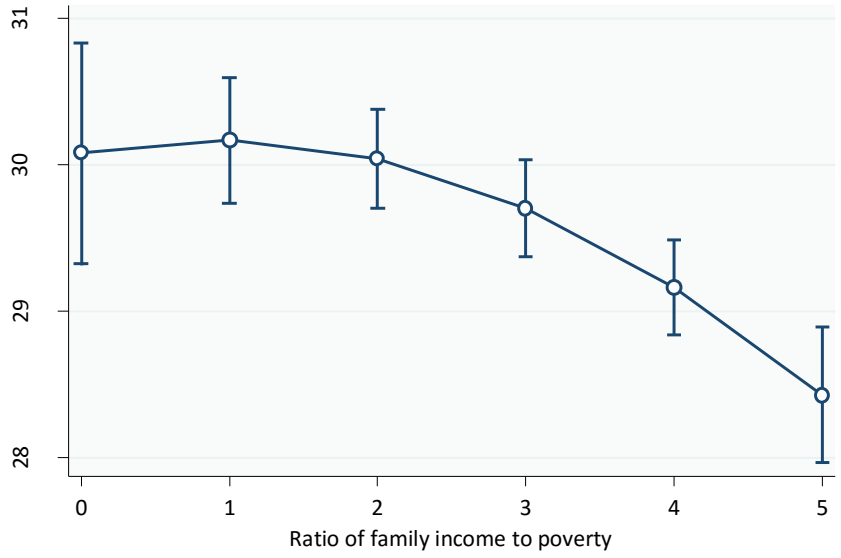


Figure 5.2. Predicted Mean BMI Values by Gender and Education

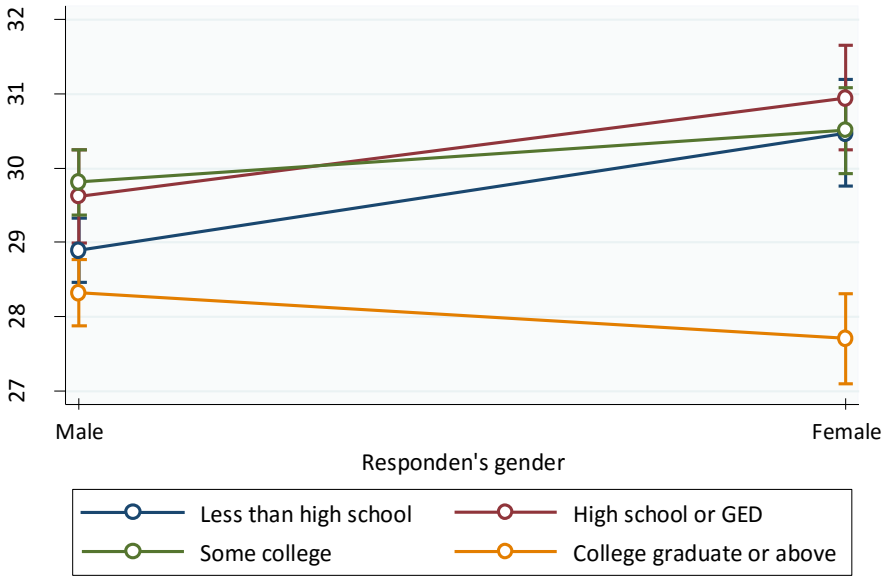


Figure 5.3. Predicted Mean BMI Values by Race/Ethnicity and Education

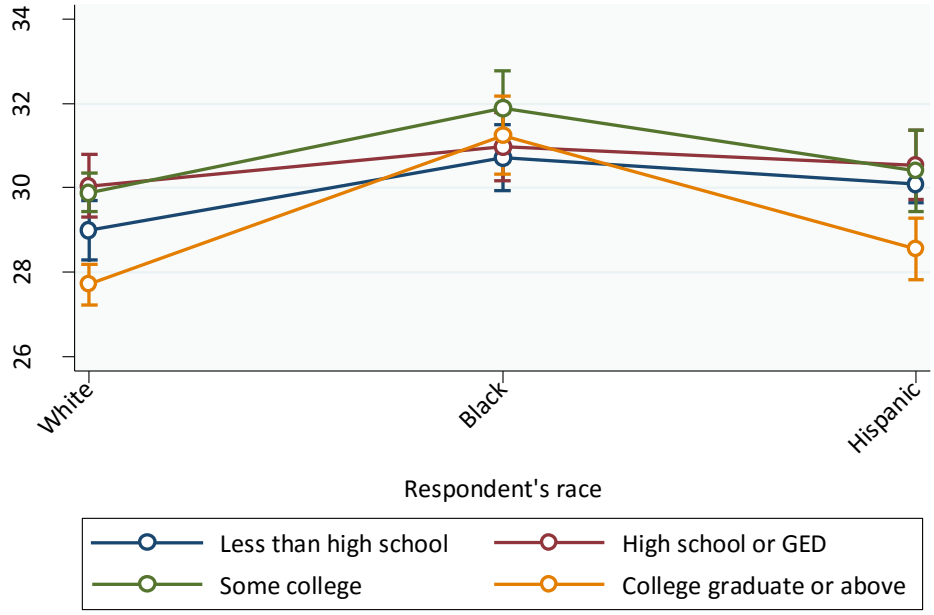


Figure 5.4. Predicted Mean BMI Values by Gender, Race/Ethnicity and Education

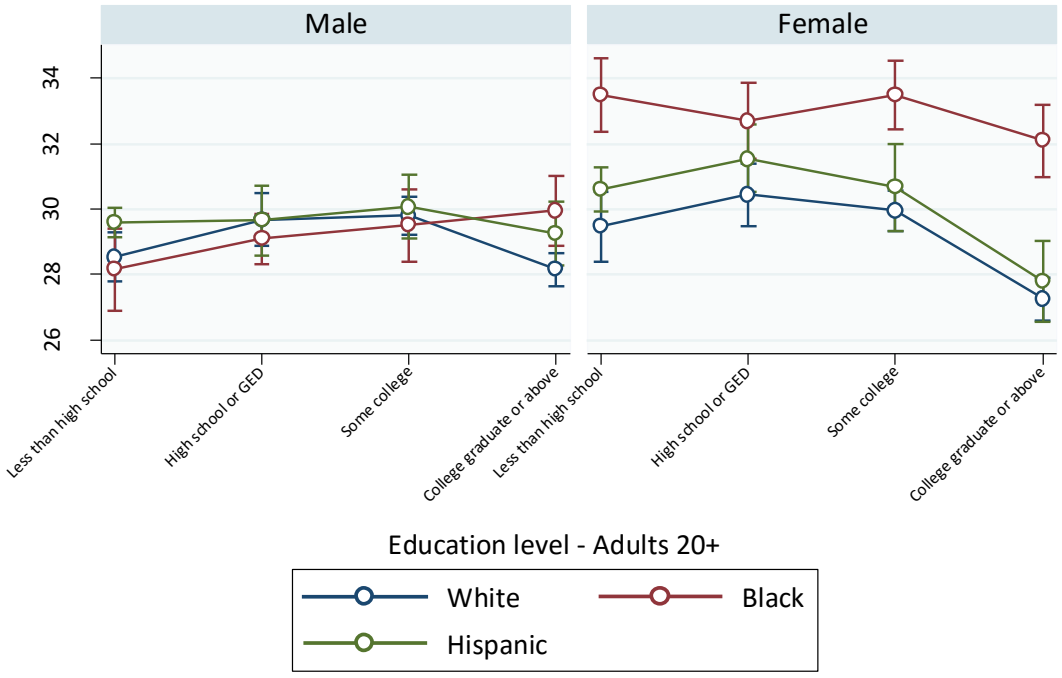


Figure 5.5. Predicted Mean BMI Values by Gender and Poverty to Income Ratio (PIR)

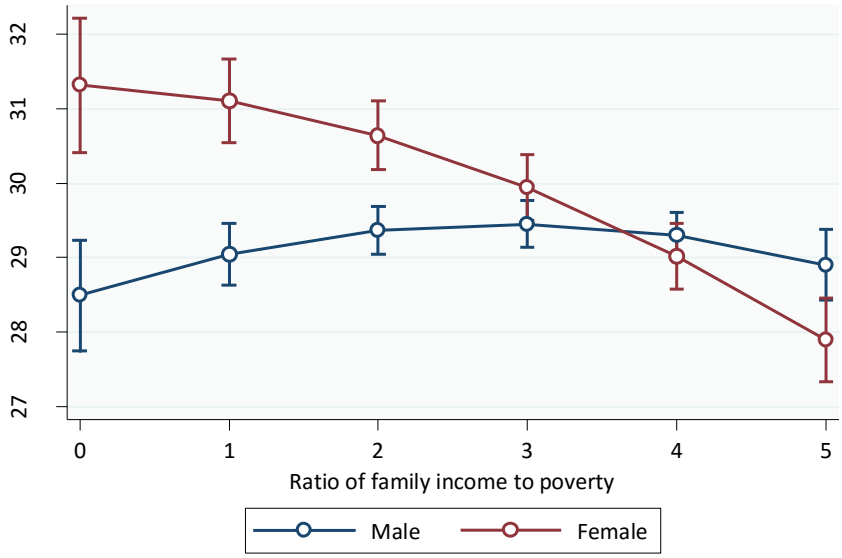


Figure 5.6. Predicted Mean BMI Values by Race and Poverty to Income Ratio (PIR)

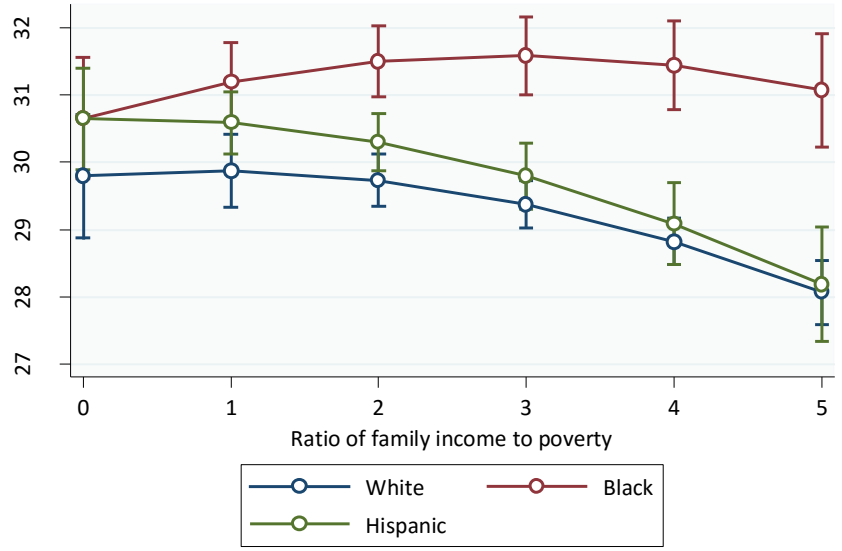
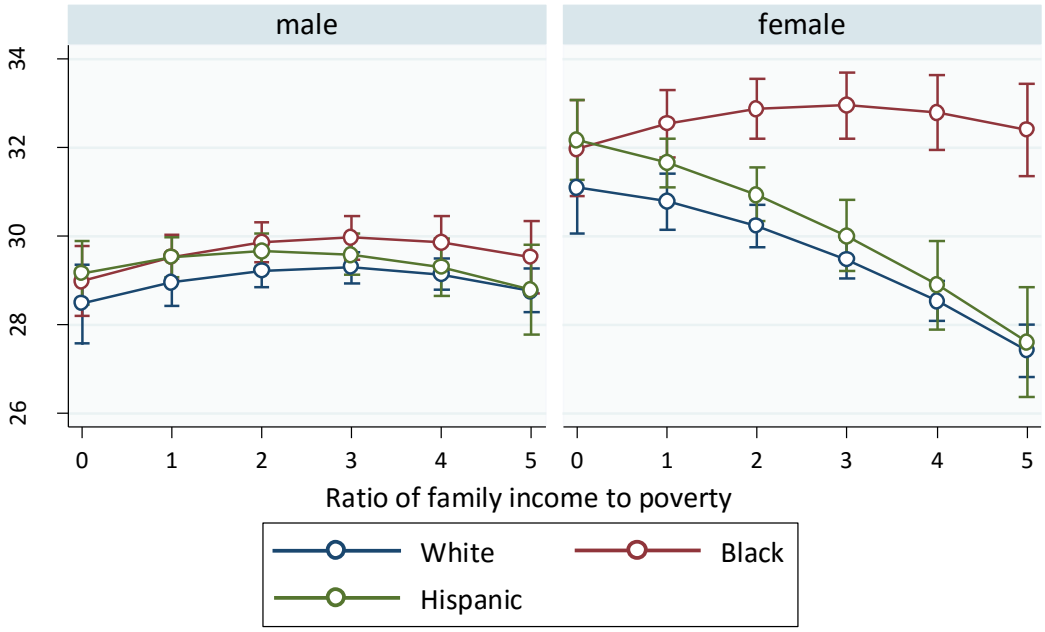


Figure 5.7. Predicted Mean BMI Values by Race/Ethnicity and PIR



CHAPTER 6: DO GENDER, RACE/ETHNICITY AND SES MODERATE THE BMI GRADIENT IN CARDIOVASCULAR HEALTH?

To address the second research question, "Do distal social factors, i.e. SES, race/ethnicity and gender shape the effect of obesity on arthritis and cardiovascular health indicators, such as diastolic and systolic blood pressure, fasting plasma glucose, and HDL cholesterol?", one binary regression analyses for arthritis as the dependent variable as well as a series of OLS regression analyses were conducted with 4 cardiovascular indicators as dependent variables. Unlike BMI, mean systolic and diastolic blood pressure, fasting plasma glucose and total cholesterol were approximately normally distributed. A total of five models were analyzed for each of the five dependent variables. Because previously discussed research suggests that waist circumference (WC) may be a better correlate of cardiovascular health outcomes, I ran sensitivity analyses using WC as the independent variable in models with all five outcomes. Because the results did not differ between the two measures, I decided to use BMI for continuity and ability to compare across studies.

In the first model, each of the dependent variables was regressed on BMI, sociodemographic, and socio-economic characteristics, controlling for individual health behaviors, self-rated health, and insurance status. The second model included an interaction term between gender and BMI. The third model included an interaction term between race/ethnicity and BMI. Finally, in the last two models, interaction analyses were conducted to assess the moderation effects between BMI x education and BMI x income.

Results from Bivariate Analyses

As it was discussed in Chapter 4, in the NHANES 2011-2014 full sample, the mean diastolic blood pressure was 71.16 with a standard deviation of 11.53 and the mean systolic blood pressure was 122.80 with a standard deviation of 16.71. Further, the mean fasting plasma glucose in this sample was 106.53 with a standard deviation of 29.16. The average HDL cholesterol in this sample was 53.08 with a standard deviation of 14.40. Finally, almost one third of the sample (27.39%) had been diagnoses by a physician with arthritis.

Table 6.1 presents results from simple regression analyses comparing the distribution of arthritis, mean systolic and diastolic blood pressure (BP), mean fasting plasma glucose and mean HDL cholesterol among, gender, racial/ethnic and SES lines.

[Insert Table 6.1 about here]

Based on the results, prevalence of diagnosed arthritis was higher among women and White adults relative to men and racial/ethnic minorities. With respect to SES, individuals who had college or higher education, had lower rates of arthritis diagnosis, when compared to those who hadn't finished high school. Finally, people of higher than mid- PIR categories, had lower rates of arthritis diagnosis, relative to those in the lower income category. Mean systolic and diastolic blood pressure were also unequally distributed along gender, racial, and SES lines. Women had a lower mean blood pressure relative to men, and, as one might expect, Black adults had higher mean blood pressure, relative to Whites. Interestingly, Hispanic and White adults had comparable blood pressure levels. Differences in educational achievement also were significantly associated with mean differences in blood pressure, such that those who had attended college or had

higher levels of educational attainment had significantly lower mean systolic and diastolic blood pressure relative to those who had less than a high school education. Similarly, those in the highest income category had a lower mean systolic and diastolic blood pressure relative to those in the lowest income categories. Further, gender differences were present across fasting plasma glucose and HDL cholesterol, with women having lower mean levels relative to men. Mean fasting plasma glucose was the highest among Hispanic adults, while mean HDL cholesterol was the highest among Black adults. Individuals with lower educational status and family income had significantly higher levels of mean fasting plasma glucose, even though significant differences in HDL cholesterol were only notable between those in the highest and lowest income and education categories.

Overall, these results clearly indicate that racial and ethnic minorities as well as individuals of lower SES are disproportionately represented among the risky levels of CVD indicators. It is instructive to note that, as discussed in the previous chapter, rates of obesity are also the highest among racial minorities at lower and higher education and income levels. Having identified that there are notable differences in indicators of cardiovascular health between gender, racial/ethnic, and SES groups, Tables 6.2-6.6 display results of multiple regression analyses with the four CVD risk factors and arthritis individually regressed on relevant socio-demographic characteristics, socio-economic factors, and health-related variables with the inclusion of interaction terms between gender, race/ethnicity, SES indicators, and BMI.

Results from Multivariate Analyses

Arthritis

[Insert Table 6.2 about here]

Based on Model 1 of Table 6.2 being female was associated with an increase in the odds of having an arthritis diagnosis by a factor of 1.83 (OR=1.82, p<.001). Race was also a significant independent correlate of arthritis diagnosis. Specifically, being Black was associated with a decrease in odds of having an arthritis diagnosis by 32% (OR=.68, p<.01) and being Hispanic was associated with a decrease in odds of having an arthritis diagnosis by 38% (OR=.62, p<.001), relative to White adults. With respect to socio-economic status, educational attainment was not significantly associated with arthritis diagnosis, while the relationship between increase in income, measured as Poverty to Income Ratio (PIR), and having been diagnosed with arthritis formed a non-linear pattern (p<.01). Specifically, the predicted probability decreased at the lower end of PIR distribution, levelled off at PIR levels of about 3 and 4, and increased at a slow rate at the highest end of the PIR distribution. Predicted probabilities are presented in Figure 6.1.

Additionally, the relationship between increase in age and having been diagnosed with arthritis also formed a non-linear pattern (p<.001). Predicted probabilities are presented in Figure 6.2. Based on the graph, the predicted probability of being diagnosed with arthritis increased at a slower rate at younger ages, while the rate of increase accelerated beyond age of 45, and reached its peak at the age of 70. On average, for each unit increase in BMI, the odds of arthritis diagnosis were increased by a factor of .05, controlling for covariates (p<.001). When it comes to health behaviors, being a current smoker relative to having never smoked was associated with increased odds of arthritis

diagnosis by a factor of 1.56 (p<.001). Sedentary behavior had no significant association with arthritis diagnosis. Self-rated health had a positive association with arthritis diagnosis, such that rating one's health as poor as opposed to excellent was estimated to increase the odds of arthritis diagnosis by a factor of 6.51 (p<.001), while rating one's health as very good was estimated to increase the odds of arthritis diagnosis only by factor of 1.61 (p<.001). The statistically significant difference between the two estimates was confirmed by an Adjusted Wald test. Finally, the odds of arthritis diagnosis were 1.78 times greater for those with health insurance (p<.001).

In Model 2, an interaction term between gender and BMI was added to the analyses, however it was not statistically significant. The non-significance of the interaction term indicates that the effect of body weight on the odds being diagnosed with arthritis did not differ between men and women. An interaction term between BMI and race/ethnicity was added in Model 3, but it also did not reach statistical significance. This suggests that the effect of BMI on the odds of arthritis diagnosis was not contingent on individuals' race/ethnicity. Surprisingly, in Models 4 and 5, neither educational status nor income moderated the BMI-arthritis relationship. Overall, two conclusions could be drawn based on these results. First, the influence of social factors, such as gender, race/ethnicity, and SES, on arthritis are additive and not multiplicative in their nature. Second, there is no amplification effect of higher body weight on arthritis diagnosis. These findings are consistent with earlier research which shows that older age, being a female, and having a higher body weight are the main risk factors of arthritis.

Systolic Blood Pressure

[Insert Table 6.3 about here]

Findings from Model 1 in Table 6.3 indicate that gender and race were significant correlates of one's mean systolic blood pressure. Specifically, being a female was associated with a 3.11 unit decrease in mean systolic BP, controlling for covariates ($p<.001$). Black adults were at a higher risk of increased systolic BP relative to Whites ($b=5.43$, $p<.001$), while the relationship was not significant for Hispanics. While income was not significantly associated with mean systolic BP, being a college graduate, as opposed not having a high school degree, was associated with a decrease in mean systolic BP ($b=-2.44$, $p<.05$). Age and mean systolic BP were associated in a non-linear pattern. The increase in mean systolic BP was slower between the ages of 25 and 55, while it accelerated among those 55 years old and above. Predicted values are presented in Figure 6.3. Importantly, BMI was associated with mean systolic BP, such that higher mean BMI predicted higher mean systolic BP ($b=.31$, $p<.001$). Neither smoking status nor self-rated health were significantly associated with mean systolic BP. Paradoxically, increase in sedentary behavior was a significant predictor of lower mean systolic BP ($b=-.22$, $p<.01$). Finally, there was no significant relationship between health insurance status and mean systolic BP.

In Model 2, an interaction between BMI and gender was added, but it was not significant. An interaction between BMI and race/ethnicity was added in Model 3, which was significant, but only when comparing Hispanic and Black adults ($b=.20$, $p<.05$). Models 4 and 5 included interaction terms between BMI*education and BMI*income. While the BMI-systolic blood pressure relationship was not moderated by income, it was however dependent on different levels of education. Thus, these interaction analyses suggest synergistic effects between proximate and distal factors that influence one's

blood pressure. With respect to race/ethnicity, the predicted mean systolic blood pressure levels were significantly different among the three groups at normal and overweight BMI levels. Not surprisingly, Black adults faced the largest health disadvantage (125.09), while Hispanic adults had the lowest mean predicted systolic BP levels (115.55). However, while the health disadvantage of Black adults prevailed at all BMI levels, the difference between mean predicted systolic BP values of Hispanics and Whites became statistically insignificant at BMI of about 35 and higher (Class II and extreme obesity). This suggests that the nature of the association between BMI and mean systolic BP is dependent on one's race/ethnicity, and the largest group differences are present at lower and less health-threatening BMI levels (Refer to Figure 6.4).

When it comes to the interaction effects between BMI and one's highest educational achievement, significant group differences were only observed between those with less than a high school education and those who had attended college or more ($b=-.24$, $p<.05$). At normal and overweight levels of BMI (between 20 and 29), those with the lowest educational achievement had a significantly higher estimated mean systolic BP score (124.89.), relative to those with a college education (116.83) ($p<.05$). Interestingly, the mean difference in BP between the two groups was estimated to decrease at the extreme obesity levels of BMI about 40 (128.03 versus 124.77) ($p<.01$). This suggests that for those who are less educated, the increase in BMI does not accelerate the rate of increase in their blood pressure, while the opposite is true for the highest educated individuals (Refer to Figure 6.5).

Diastolic Blood Pressure

[Insert Table 6.4 about here]

Based on the baseline model (see Model 1, Table 6.4), as expected, gender and race were significantly associated with mean diastolic BP. Women had a lower mean diastolic BP relative to men (b=-2.38, p<.001) and Hispanic adults had a lower mean diastolic BP relative to White adults (-1.43, p<.05). Results also indicated that neither education nor income were significant correlates of diastolic BP. Further, increase in age formed a non-linear association with diastolic BP. The predicted mean diastolic blood pressure values by age are presented in Figure 6.6. Specifically, between the ages 25 and 50, diastolic blood pressure was predicted to increase, while its levels were estimated to start decreasing sharply after the age of 55. Such a different pattern from changes in systolic blood pressure are not an anomaly, as isolated systolic hypertension (high systolic blood pressure and low diastolic blood pressure) is fairly common among older adults (Pinto 2007). Increase in BMI was associated with .15 unit increase in mean diastolic BP, controlling for covariates (p<.001). The mean BP was estimated to decrease with an increase in sedentary behavior (b=-.14, p<.001). Finally, rating one's health as poor as opposed to excellent was associated with a decrease in mean systolic BP (b=-3.42, p<.05).

Gender and race/ethnicity were the only two significant moderators in this analysis (Model 2 and Model 3). As displayed in Figure 6.7, at the lower end of the BMI distribution, the estimated mean diastolic blood pressure levels were about equivalent among women and women. However, with increasing levels of BMI, men experienced the increase in mean estimated diastolic blood pressure levels at faster rates relative to women. At the BMI level of extreme, or Class IV obesity, men had significantly higher

estimated mean diastolic blood pressure (75.53) when compared to women (70.89) ($p<.001$).

With respect to the interaction term between BMI and race/ethnicity, it was only significant for the Hispanic category ($p<.01$). Specifically, at the low end of the BMI range, Hispanic Americans had significantly lower mean estimated diastolic BP levels (67.49) relative to Whites (69.94) and Blacks (70.62) ($p<.001$). However, as BMI reached the obesity cut-off of 30 and continued to increase, the significant difference between the three groups disappeared. Overall, increases in BMI had a minimal effect on the increase in estimated mean diastolic BP among White and Black adults, however, an increase in body weight accelerated the increase in estimated mean diastolic BP for Hispanic adults (see Figure 6.7).

Fasting Plasma Glucose

[Insert Table 6.5 about here]

Table 6.5 displays the results of regressing fasting plasma glucose on socio-demographic, socio-economic, and health characteristics. Model 1 reveals that being a female was associated with a decrease in mean fasting plasma glucose levels, net of other covariates ($b=-4.73$, $p<.001$). On average, Hispanic Americans had higher estimated mean glucose levels relative to White Americans ($b=4.42$, $p<.001$) while no significant group difference was observed for Black adults. Income was not significantly associated with fasting plasma glucose levels, but college graduates had lower estimated mean glucose levels, when compared to those who had less than high school education ($b=-3.70$, $p<.05$). Age was associated with mean fasting plasma glucose levels in a non-linear fashion. Specifically, increase in mean glucose levels was sharper among younger individuals, while started levelling off past the age of 55 (see Figure 6.9). Further,

increase in BMI was associated with about 1 unit increase in mean fasting plasma glucose ($p<.001$). Neither smoking status, sedentary behavior, nor having health insurance were significant correlates of glucose levels. Finally, rating one's health as fair as opposed to excellent was associated with higher mean levels in fasting plasma glucose ($b=12.4$, $p<.01$).

Among the four interaction terms, only race/ethnicity was found to be a significant moderator of the BMI-fasting plasma glucose relationship, however, this was only the case for the Black racial group ($b=-.54$, $p<.05$). Specifically, at the lower end of BMI distribution, White adults had lower levels of mean plasma serum glucose relative to Black adults; however, at the high end of the BMI distribution, group differences disappeared. While the increase in BMI was associated with an increase in fasting plasma glucose among all racial/ethnic groups, the rate of increase appeared to be faster for White adults, while it was more constant for Black and Hispanic Americans (see Figure 6.10).

HDL Cholesterol

[Insert Table 6.6 about here]

The results for HDL cholesterol as displayed in Table 6.6, followed a slightly different pattern when compared to those of previously discussed indicators of CVD health. Specifically, being a female was associated with 11 unit increase in HDL cholesterol, on average ($b=11.05$, $p<.001$). Hispanic Americans had lower estimated mean HDL cholesterol levels, relative to Whites, controlling for covariates ($b=-.46$, $p<.001$). Both income and education were independently associated with an increase in mean HDL cholesterol levels. For example, those with a college education had a 3.14 unit higher estimated mean HDL level, relative to the least educated group ($p<.001$).

There was also a clear income gradient, such that each unit increase in PIR was associated with a .77 unit increase in mean HDL cholesterol ($p<.001$). The relationship between age and HDL cholesterol followed a non-linear pattern, such that a sharper increase in mean estimated HDL cholesterol could be observed among individuals between the ages of 25 and 50, while the increase tapered off past about 55 years (refer to Figure 6.11 for predicted probabilities). With respect to health behaviors, being a former smoker was a significant correlate of increase in mean HDL levels and spending more time sedentary was associated with a decrease in mean HDL levels ($p<.05$). Finally, having poorer self-rated health significantly predicted a decrease in HDL levels.

The BMI-HDL cholesterol relationship was moderated by race/ethnicity, education and income. As shown in Figure 6.12, at lower levels of BMI, Hispanic adults had significantly lower levels of HDL cholesterol (55.91), when compared to Black and White adults (61.28 and 60.69 respectively) ($p<.001$). While increase in BMI was associated with a decrease in HDL cholesterol among all three racial/ethnic groups, the decreased was much sharper for White adults relative to racial minorities. At the highest levels of BMI, the mean BMI of White adults reached similar levels of Hispanics, while Black adults maintained the health advantage of higher mean HDL cholesterol.

Figure 6.13 illustrates differences in the effects of BMI by educational achievement levels. While more highly educated adults had higher mean HDL levels at normal and overweight levels of BMI, the significant difference between them disappeared at the high end of BMI distribution. Overall, increase in BMI was associated with a decrease in estimated mean HDL cholesterol levels among all educational groups, the decrease was the sharpest for those who were the most educated.

Finally, differences in the effect of BMI on HDL cholesterol are presented in Figure 6.14. Based on the results, increase in BMI significantly affected all income groups, such that the mean HDL cholesterol level was estimated to decrease as BMI increased. However, the effect of BMI was much stronger for individuals from high income categories relative to the lower income groups. While at the lower BMI end of the distribution, higher SES individuals had the health advantage of higher mean HDL cholesterol, those differences became statistically insignificant at higher BMI levels.

Chapter Summary

The main goal of this chapter was to examine the role of BMI at the intersection of gender, race/ethnicity and social class in shaping five obesity-related health outcomes – arthritis and four indicators of cardiovascular health. First, I wanted to examine the multiplicative effect of distal and more proximate risk factors on health. Second, I was curious to test whether risk accumulation was contingent on the health outcome in question. The persistent link between social status and health has been well demonstrated. Similarly, the negative health outcomes of obesity have received increasing attention from scholars in medical and public health fields. However, we know little about the interaction effects between social status indicators and obesity on chronic health outcomes. Based on previously discussed research, I hypothesized that individuals of more disadvantaged social statuses would experience larger negative health effects of increased body weight. Furthermore, I chose arthritis and cardiovascular health as dependent variables because I wanted to test whether the moderating effect of social factors would be more pronounced if the health outcome was more easily controllable. Because cardiovascular health is largely dependent on individual health behaviors, I

expected that there would be more opportunities for social status to generate inequalities in the distribution of health indicators, such as hypertension, blood glucose, or blood cholesterol levels. Following that logic, I expected that social factors would have little impact above and beyond BMI for the probability of being diagnosed with arthritis.

With arthritis as a health outcome, being a female was associated with increased odds of arthritis diagnosis, while being a racial minority was associated with decreased odds of diagnosis. Additionally, increase in age and increase in BMI were significant correlates of arthritis diagnosis, net of covariates. Interestingly, increase in income also had a protective effect from potential arthritis diagnosis. Neither gender, nor race/ethnicity or SES moderated the BMI-arthritis link. This finding suggests that social status indicators and BMI are *independently* significant predictors of this health outcome. However, the distal social factors are irrelevant for the effect of BMI on arthritis (i.e. there is no multiplicative effect).

When considering cardiovascular health outcomes, increase in BMI was a significant correlate of worsening health. Moderation analyses revealed that the effect of BMI on blood pressure was amplified for Hispanic adults, while it was irrelevant among Hispanic and White adults. It should also be noted that Black adults had higher estimated mean blood pressure across all levels of BMI. With respect to fasting plasma glucose and HDL cholesterol, the negative effect of BMI was amplified for White adults, but not Hispanics or Blacks. In addition, the effect of BMI on HDL cholesterol was also amplified for individuals with higher educational achievement and household income. Overall, these results suggest the effect of increasing body weight are contingent on different indicators of social status, however, their effect is not uniform and depends of

health outcome in question. Further interpretation and implications of the study findings will be discussed in the following chapter.

Table 6.1 Weighted Means and Proportions for Dependent Variables by Gender, Race/Ethnicity, and SES

	Arthritis	Systolic BP	Diastolic BP	Fasting Plasma Glucose	HDL Cholesterol
Male	.23	124.24 (13.64)	72.42 (10.29)	108.48 (28.31)	47.67 (12.34)
Female	.34[a]	121.85[a] (15.21)	69.88[a] (9.64)	104.69[a] (28.01)	58.20[a] (14.70)
White	.31	122.78 (11.49)	71.05 (7.93)	105.45 (20.99)	53.63 (11.98)
Black	.26[b]	127.50[b] (23.99)	72.36[b] (17.44)	107.10 (48.61)	55.16[b] (22.08)
Hispanic	.18[b]	120.59[b] (18.13)	70.57 (2.38)	110.47[b] (44.24)	49.59[b] (15.32)
Less than high school	.33	125.97 (19.36)	69.72 (13.81)	110.33 (36.26)	50.01 (15.63)
High school or GED	.30	125.44 (15.41)	71.71[c] (11.14)	110.39 (33.94)	51.33[c] (14.03)
Some College	.31	122.89[c] (14.12)	71.50[c] (9.12)	107.06[c] (26.98)	52.53[c] (14.09)
College and above	.23[c]	120.00[c] (11.19)	71.08[c] (8.00)	101.38[c] (19.55)	56.36[c] (13.05)
PIR<3	.31	124.42 (17.18)	70.79 (11.95)	108.87 (35.94)	51.15 (15.08)
PIR>3<5	.26[d]	121.56[d] (11.21)	71.57 (7.75)	104.28 (19.98)	54.95[d] (12.79)
PIR=5	.26[d]	121.50[d] (10.66)	72.26[d] (7.11)	102.41[d] (17.15)	56.73[d] (12.60)
N	8,557	8,227	8,227	3,712	8,066

Notes: For continuous variables, standard deviations presented in parentheses.
[a] Significantly different from Male
[b] Significantly different from White
[c] Significantly different from Less than HS
[d] Significantly different from PIR<3
$^{*}p<.05$; $^{**}p<.01$; $^{***}p<.001$

Table 6.2 Binary Logistic Regression of Arthritis on Socio-demographic, Socio-economic and Health Characteristics (N=8,557)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	OR	SE	OR	SE	OR	SE	OR	SE	OR	SE
Female	1.83***	.17	2.18*	.80	1.84***	.17	1.83***	.17	1.83***	.17
Black	.68**	.07	.69**	.07	.91	.24	.68**	.07	.68**	.07
Hispanic	.62***	.06	.63***	.06	.53	.17	.62***	.06	.62***	.06
High School or GED	.94	.13	.94	.13	.94	.13	1.36	.77	.94	.13
Some College	1.17	.14	1.17	.14	1.17	.14	1.52	.61	1.17	.14
College Graduate and Above	.96	.16	.96	.16	.96	.16	1.15	.57	.96	.16
Poverty to Income Ratio	.69**	.08	.68**	.08	.69**	.08	.69**	.08	.70*	.11
Poverty to Income Ratio^2	1.05**	.02	1.05**	.02	1.05**	.02	1.05**	.02	1.05**	.02
Age	1.18	.03	1.18***	.03	1.18***	.03	1.18***	.03	1.18***	.03
Age^2	.99***	.01	.99***	.01	.99***	.01	.99***	.01	1.00***	.01
BMI	1.05***	.01	1.05***	.01	1.05***	.01	1.06***	.01	1.05***	.01
Former Smoker	1.22	.15	1.22	.15	1.22	.15	1.22	.15	1.22	.15
Current Smoker	1.56***	.17	1.56***	.17	1.56***	.17	1.56***	.16	1.56***	.17
Sedentary behavior	1.00	.01	1.00	.01	1.00	.01	1.00	.01	1.00	.01
Very Good	1.61**	.23	1.61**	.23	1.61**	.23	1.61**	.24	1.61**	.23
Good	2.08***	.33	2.09***	.33	2.08***	.33	2.08***	.35	2.09***	.33
Fair	3.17***	.60	3.17	.60	3.17***	.60	3.17***	.63	3.18***	.59
Poor	6.51***	1.64	6.51	1.65	6.50***	1.65	6.49***	1.65	6.52***	1.66
Insured	1.78***	.19	1.78	.19	1.78***	.19	1.78***	.19	1.78***	.19
BMI x Female			.99	.01						
BMI x Black					.99	.01				
BMI x Hispanic					1.01	.01				
BMI x High School or GED							.99	.02		
BMI x Some College							.99	.01		
BMI x College and Above							.99	.02		
BMI x PIR									1.00	.01
F	18.70***		16.81***		17.38***		15.84***		20.25***	

*p<.05; **p<.01; ***p<.001

Table 6.3 OLS Regression of Systolic Blood Pressure on Socio-demographic, Socio-economic and Health Characteristics (N=8,227)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	b	SE	b	SE	b	SE	b	SE	b	SE
Female	-3.11***	.41	1.23	2.55	-3.10***	.40	-3.05***	.41	-3.03***	.41
Black	5.43***	.62	5.52***	.62	7.15**	2.29	5.39***	.62	5.41***	.61
Hispanic	-.51	.56	-.52	.54	-6.44*	2.85	-.49	.56	-.51	.55
High School or GED	-.27	.79	-.28	.79	-.28	.80	-4.05	4.07	-.28	.80
Some College	-1.60*	.67	-1.62*	.67	-1.59*	.67	-7.02*	3.37	-1.61*	.67
College Graduate and Above	-2.44*	.98	-2.44*	.98	-2.44*	.98	-9.47**	3.24	-2.39*	.97
Poverty to Income Ratio	-.10	.18	-.11	.18	-.10	.18	-.09	.18	-1.28	.66
Age	-.08	1.00	-.08	.10	-.08	.10	-.09	.10	-.09	.10
Age^2	.01***	.01	.01***	.01	.01***	.01	.01***	.01	.01***	.01
BMI	.31***	.03	.41***	.07	.30***	.04	.16*	.08	.20**	.01
Former Smoker	-.15	.51	-.19	.50	-.15	.51	-.15	.51	-.13	.51
Current Smoker	1.14	.77	1.17	.77	1.11	.07	1.11	.77	1.13	.77
Sedentary behavior	-.22**	.07	-.22**	.07	-.22**	.70	-.22**	.07	-.22**	.07
Very Good	-.05	.70	-.03	.70	-.02	.70	-.16	.69	-.14	.69
Good	1.11	.58	1.11	.58	1.15	59	.95	.59	.96	.57
Fair	1.02	.84	1.03	.83	1.06	.84	.88	.84	.89	.81
Poor	-.83	2.18	-.82	2.17	-.89	2.16	-.88	2.20	-.86	2.19
Insured	-1.07	.70	-1.11	.69	-1.07	.69	-1.07	.70	-1.05	.70
BMI x Female			-.15	.09						
BMI x Black					-.05	.07				
BMI x Hispanic					.20*	.09				
BMI x High School or GED							.13	.13		
BMI x Some College							.18	.11		
BMI x College and Above							.24*	.11		
BMI x PIR									.04	.02
R^2	.21		.21		.21		.21		.21	
F	54.65***		49.50***		43.69***		38.78***		49.01***	

*p<.05; **p<.01; ***p<.001

Table 6.4 OLS Regression of Diastolic Blood Pressure on Socio-demographic, Socio-economic and Health Characteristics (N=8,227)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	b	SE	b	SE	b	SE	b	SE	b	SE
Female	-2.38***	.29	3.30*	1.60	-2.40***	.28	-2.39***	.29	-2.39***	.29
Black	.46	.67	.57	.68	-.35	1.87	.47	.68	.46	.67
Hispanic	-1.44*	.57	-1.46*	.57	-6.33***	1.49	-1.43*	.58	-1.44*	.57
High School or GED	1.28	.85	1.26	.85	1.28	.85	2.44	2.61	1.28	.85
Some College	.97	.65	.96	.66	.99	.65	.39	2.22	.97	.65
College Graduate and Above	.24	.84	.24	.85	.23	.84	1.91	2.66	.23	.84
Poverty to Income Ratio	.11	.17	.09	.17	.11	.17	.11	.17	.21	.43
Age	1.15***	.06	1.14***	.06	1.15***	.06	1.15***	.01	1.15***	.06
Age^2	-.01***	.01	-.01***	.01	-.01***	.01	-.01***	.01	-.01***	.01
BMI	.16***	.03	.28	.04	.14**	.04	.18*	.08	.17**	.04
Former Smoker	-.52	.40	-.57	.40	-.51	.39	-.52	.39	-.52	.40
Current Smoker	-.14***	.04	-1.01*	.49	-1.08*	.49	-1.03*	.49	-1.05*	.49
Sedentary behavior	-.14***	.04	-.14**	.04	-.14***	.04	-.14***	.04	-.14***	.04
Very Good	.38	.56	.40	.56	.41	.57	.41	.56	.39	.56
Good	.59	.61	.60	.60	.63	.61	.65	.61	.60	.60
Fair	-.27	.71	-.27	.71	-.23	.71	-.23	.71	-.26	.71
Poor	-3.42*	1.38	-3.41*	1.37	-3.48*	1.38	-3.44*	1.40	-3.42*	1.38
Insured	-.40	.53	-.45	.52	-.41	.53	-.40	.53	-.40	.53
BMI x Female			.-19**	.05						
BMI x Black					.03	.07				
BMI x Hispanic					.16**	.06				
BMI x High School or GED							-.04	.08		
BMI x Some College							.02	.08		
BMI x College and Above							-.06	.09		
BMI x PIR									-.01	.01
R^2	.10		.11		.11		.10		.10	
F	44.60***		47.93***		39.01***		33.63***		40.17***	

*p<.05; **p<.01; ***p<.001

Table 6.5 OLS Regression of Fasting plasma Glucose on Socio-demographic, Socio-economic and Health Characteristics (N=3,712)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	b	SE	b	SE	b	SE	b	SE	b	SE
Female	-4.74***	.80	6.25	7.01	-4.54***	.81	-4.77***	.79	-4.60***	.82
Black	-.60	1.56	-.34	1.54	16.05*	5.92	-.49	1.58	-.67	1.56
Hispanic	4.42***	1.11	4.42***	1.12	14.06	7.83	4.47***	1.11	4.44***	1.09
High School or GED	.63	1.87	.63	1.83	.62	1.85	3.54	8.49	.71	1.87
Some College	-.84	1.46	-.84	1.47	-.89	1.46	-4.05	8.38	-.76	1.48
College Graduate and Above	-3.70**	1.70	-3.63*	1.67	-3.54*	1.67	2.48	7.59	-3.48*	1.71
Poverty to Income Ratio	-.23	.39	-.26	.39	-.19	.38	-.23	.39	-2.67	1.95
Age	.91**	.27	.92**	.26	.89**	.26	.92**	.26	.90**	.27
Age^2	-.01*	.01	-.01*	.01	-.01*	.01	-.01*	.01	-.01*	.01
BMI	1.02***	.11	1.26***	.23	1.14***	.15	1.06***	.19	.80***	.17
Former Smoker	-1.04	1.70	-1.06	1.68	-1.08	1.69	-1.05	1.69	-1.02	1.69
Current Smoker	-2.84	1.83	-2.68	1.80	-2.74	1.80	-2.70	1.88	-2.88	1.83
Sedentary behavior	.24	1.18	.25	.18	.23	.19	.24	.18	.23	.18
Very Good	.29	1.16	.26	1.17	.34	1.17	.37	1.21	.10	1.19
Good	2.26	1.69	2.19	1.69	2.21	1.7	2.50	1.73	1.93	1.72
Fair	12.41**	3.64	12.35**	3.57	12.49**	3.60	12.56**	3.63	12.22**	3.55
Poor	11.32	6.94	11.22	7.01	11.78	6.83	11.05	6.99	11.32	6.93
Insured	1.11	1.39	.96	1.38	1.22	1.37	1.05	1.40	1.21	1.36
BMI x Female			-.37	-.25						
BMI x Black					-.53*	.21				
BMI x Hispanic					-.32	.28				
BMI x High School or GED							-.10	.30		
BMI x Some College							.11	.30		
BMI x College and Above							-.21	.28		
BMI x PIR									.08	.07
R^2	.15		.15		.15		.15		.15	
F	25.04***		31.30***		21.41***		19.95***		21.31***	

*p<.05; **p<.01; ***p<.001

Table 6.6 OLS Regression of HDL Cholesterol on Socio-demographic, Socio-economic and Health Characteristics (N=8,066)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	b	SE	b	SE	b	SE	b	SE	b	SE
Female	11.05***	.44	9.26**	2.41	10.96***	.43	10.94***	.43	10.88***	.43
Black	4.83	.49	4.79***	.49	-3.22	2.22	4.93***	.48	4.86***	.48
Hispanic	-.46***	.44	-.46	.44	-5.53*	2.61	-.44	.45	-.46	.46
High School or GED	1.00	.68	1.00	.68	1.02	.68	7.75*	.3.68	1.01	.69
Some College	1.35*	.67	1.35*	.67	1.38*	.67	7.52**	2.58	1.35	.68
College Graduate and Above	3.14***	.73	3.13***	.73	3.07***	.72	17.00***	2.83	2.97***	.76
Poverty to Income Ratio	.77***	,18	.77***	.18	.76***	.18	.76***	.18	3.61***	.71
Age	.34**	.09	.34**	.09	.34**	.09	.34**	.09	.34**	.09
Age^2	-.01*	.01	-.01*	.01	-.01*	.01	-.01*	.01	-.01*	.01
BMI	-.73***	.04	-.77***	.06	-.79***	.05	-.48***	.06	-.47***	.06
Former Smoker	1.15*	.53	1.16*	.52	1.17*	.53	1.13*	.54	1.10*	.52
Current Smoker	-.46	.69	-.47	.69	-.48	.69	-.38	.68	-.43	.69
Sedentary behavior	-.15*	.06	-.16*	.06	-.15*	.06	.-16*	.06	-.15*	.06
Very Good	-1.55	.92	-1.55	.92	-1.52	.92	-1.30	.91	-1.34	.92
Good	-3.13**	.82	-3.13**	.82	-3.06**	.82	-2.74**	.81	-2.78**	.83
Fair	-3.41**	1.01	-3.41**	1.01	-3.36**	.99	-3.10**	.99	-3.14**	1.00
Poor	-3.96*	.1.63	-3.97*	1.63	-4.10*	1.63	-3.94*	1.59	-3.97*	1.58
Insured	-.57	.79	-.55	.78	-.62	.77	-.59	.80	-.61	.81
BMI x Female			.06	.07						
BMI x Black					.26***	.07				
BMI x Hispanic					.17*	.08				
BMI x High School or GED							-.23*	.11		
BMI x Some College							-.21*	.08		
BMI x College and Above							-.48***	.09		
BMI x PIR									-.10	.02
R^2	.26		.26		.26		.26		.26	
F	35.39***		46.01***		33.91***		26.19***		35.07***	

*p<.05; **p<.01; ***p<.001

Figure 6.1 Predicted Probability of Having Arthritis Diagnosis by Poverty to Income Ratio (PIR)

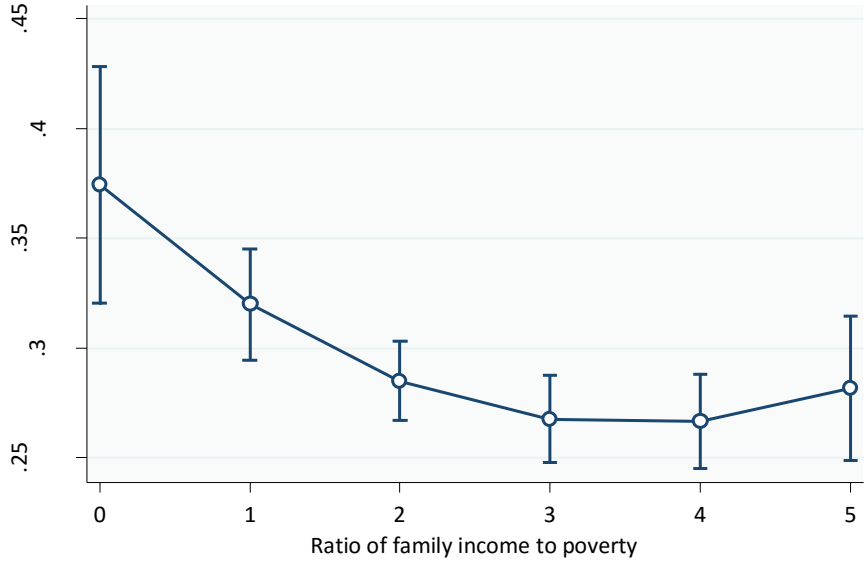


Figure 6.2 Predicted Probability of Having Arthritis Diagnosis by Age

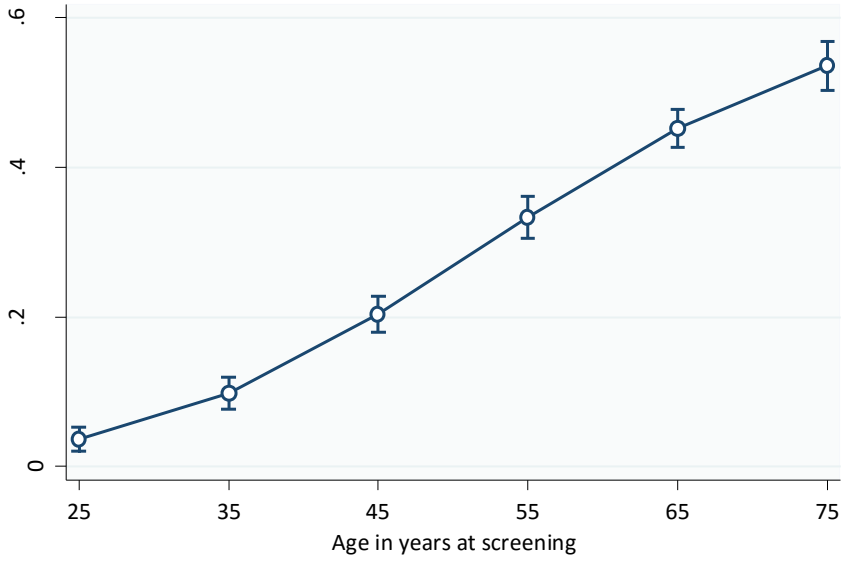


Figure 6.3 Predicted Mean Systolic Blood Pressure Values by Age

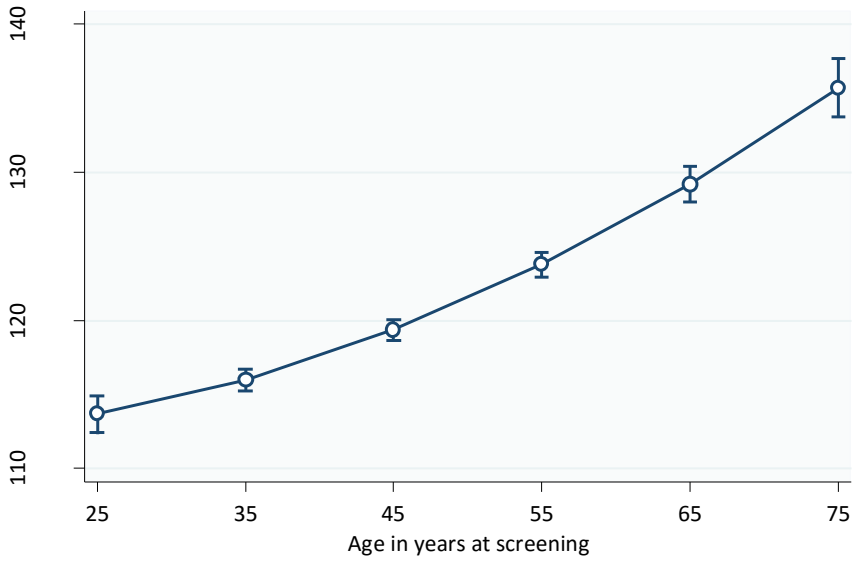


Figure 6.4 Predicted Mean Systolic Blood Pressure Levels by BMI and Race/Ethnicity

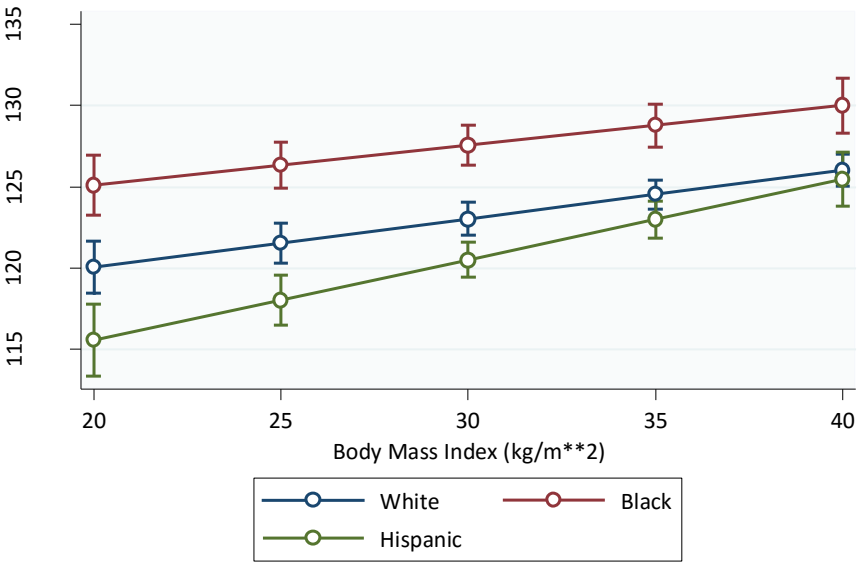


Figure 6.5 Predicted Mean Systolic Blood Pressure Levels by BMI and Education

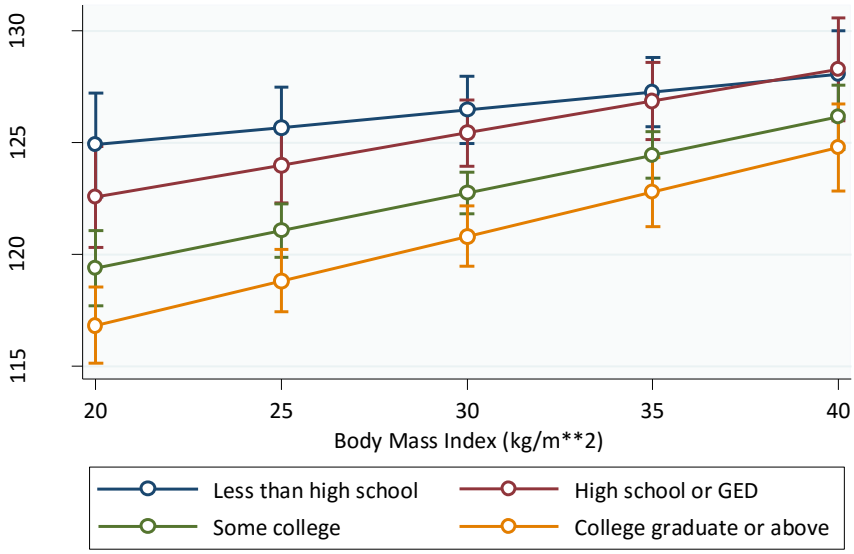


Figure 6.6 Predicted Mean Diastolic Blood Pressure Levels by Age

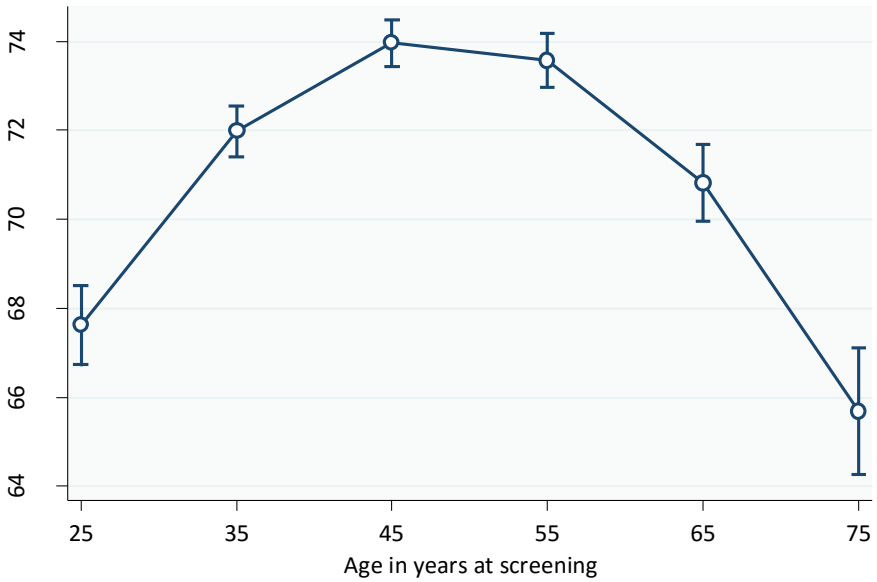


Figure 6.7 Predicted Mean Diastolic Blood Pressure Levels by Gender

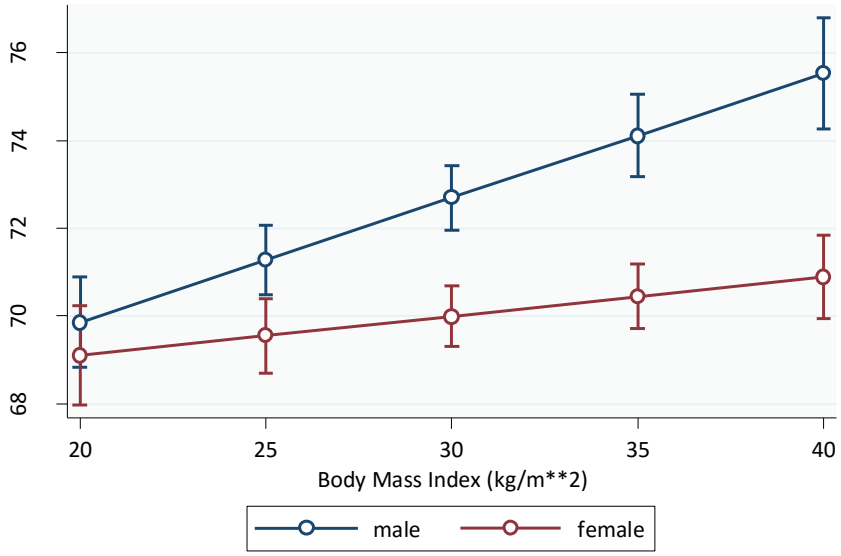


Figure 6.8 Predicted Mean Diastolic Blood Pressure Levels by Race/Ethnicity

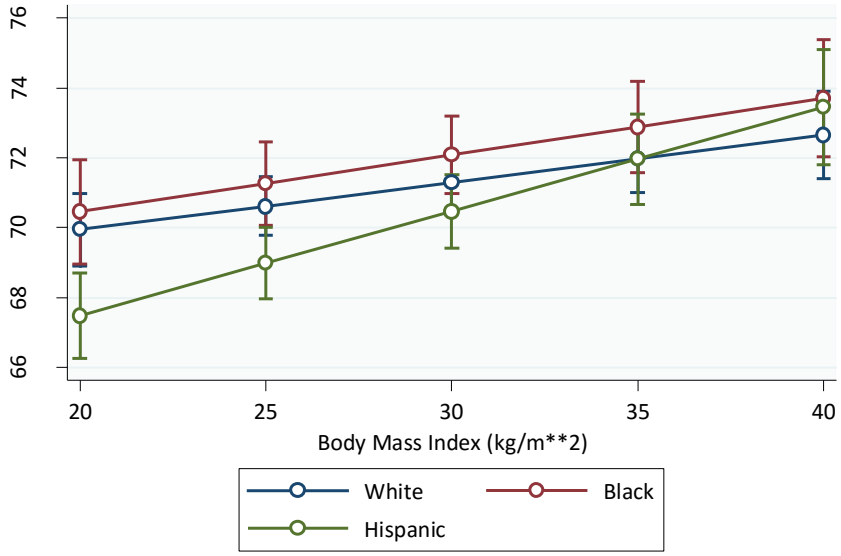


Figure 6.9 Predicted Mean Fasting plasma Glucose Levels by Age

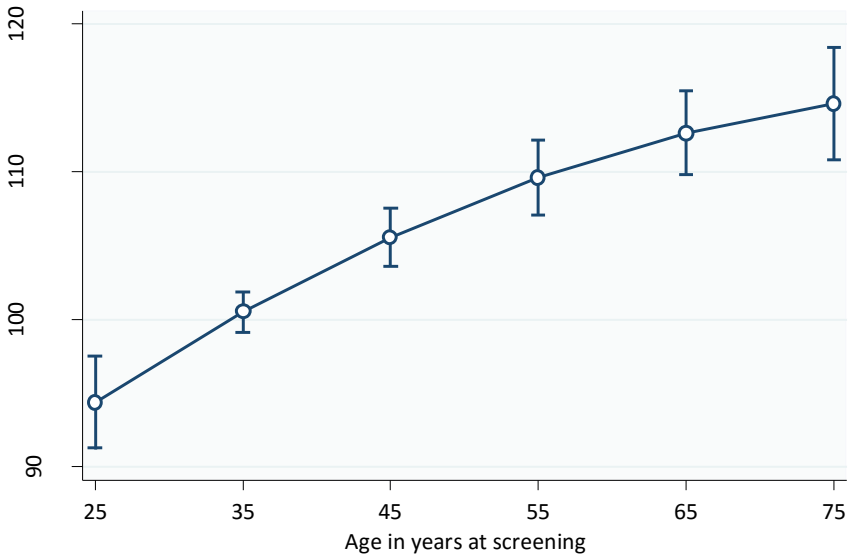


Figure 6.10 Predicted Mean Fasting plasma Glucose Levels by Race/Ethnicity and BMI

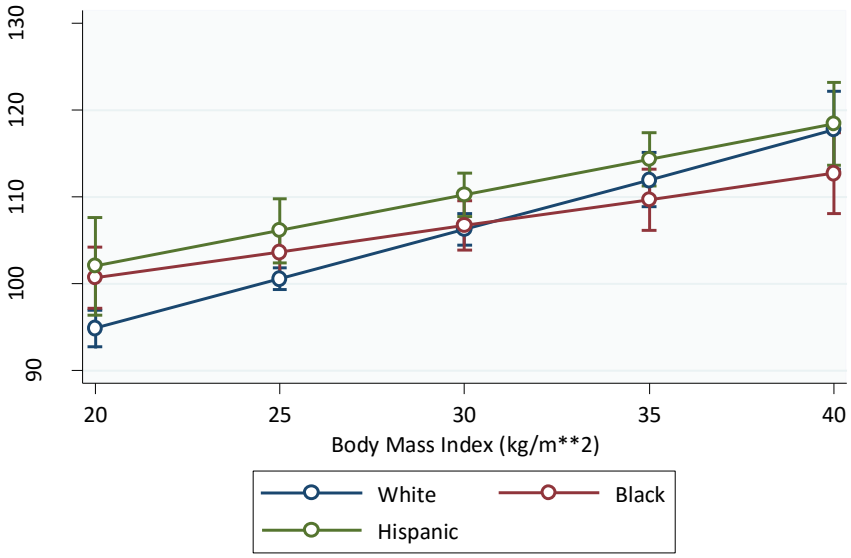


Figure 6.11 Predicted Mean HDL Cholesterol Levels by Age

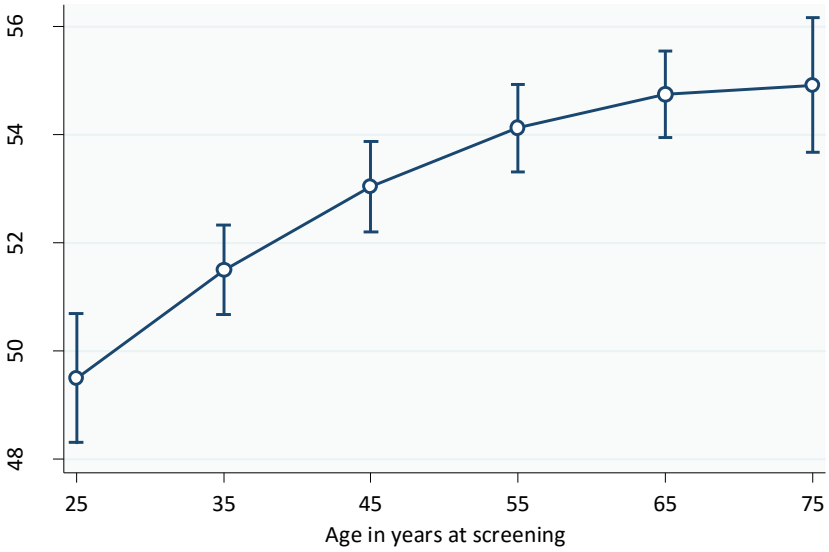


Figure 6.12 Predicted Mean HDL Cholesterol Levels by Race/Ethnicity and BMI

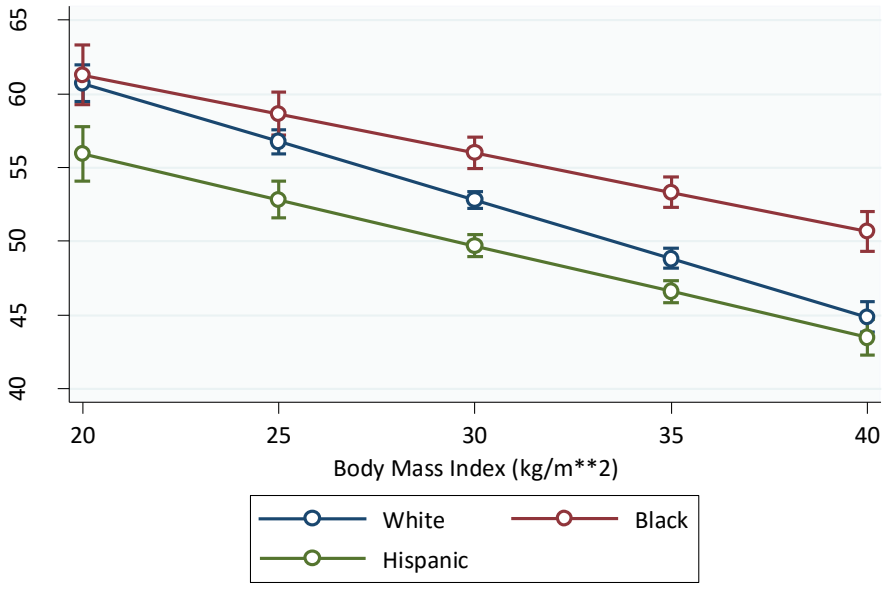


Figure 6.13 Predicted Mean HDL Cholesterol Levels by Education and BMI

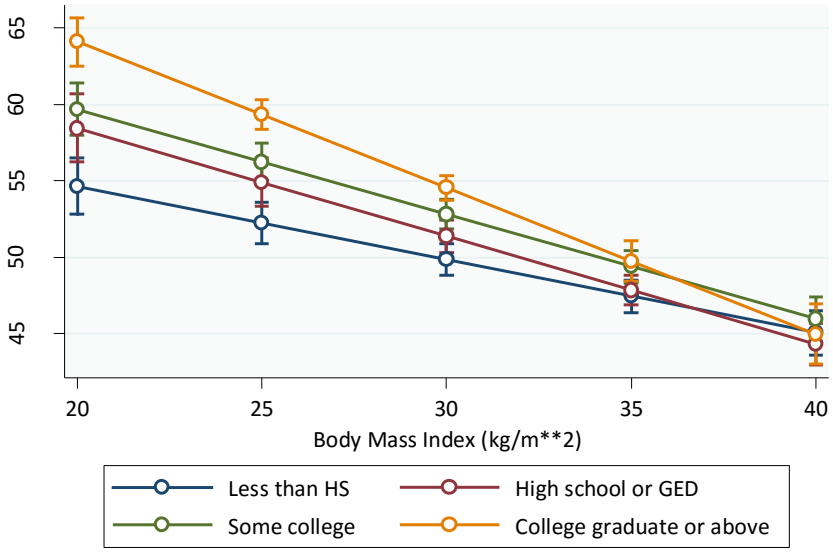
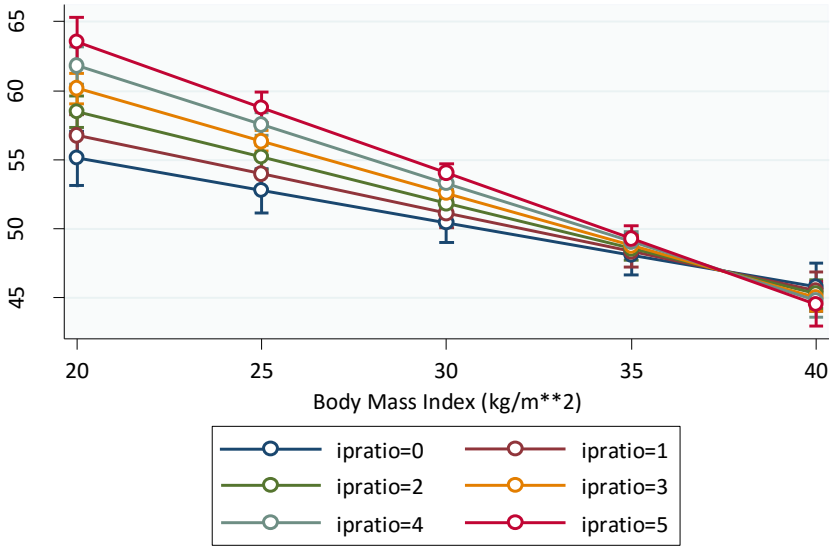


Figure 6.14 Predicted Mean HDL Cholesterol Levels by Poverty to Income Ratio (PIR) and BMI



CHAPTER 7: DISCUSSION, STUDY IMPLICATIONS, AND LIMITATIONS

Study Motivation and Limitations of Previous Research

The increase in average body weight in the United States has affected individuals of all gender, race/ethnicity, and socio-economic groups. However, research has continuously demonstrated that the largest increase has occurred among the most socio-economically disadvantaged individuals and racial minorities (Ailshire and House 2011; Zhang and Wang 2004a). Even though the SES gap has been recently appeared to be narrowing, White adults – and especially those with more education and income – have consistently maintained a lower BMI advantage relative to their lower SES and racial/ethnic minority counterparts (Chang and Lauderdale 2005). The persistence of such social patterning of overweight and obesity suggests that distal, structural social factors rather than more proximate modifiable health behaviors, are involved and should be positioned as points of departure in obesity disparities research.

The association between social status and obesity has been well documented and dates back to studies of late 1900s (Stunkard 1989). However, the SES-obesity link is not that straightforward, and varies by gender, race/ethnicity, and age. Even though a number of researchers have further examined this complex relationship, fewer studies have addressed the differences in obesity prevalence simultaneously along socio-economic, racial/ethnic and gender lines. Previous scholars have either focused on explaining the social distribution of being obese and/or overweight along gender and SES lines without simultaneously considering race/ethnicity or along racial/ethnic and gender lines without simultaneously considering the effects of income and education (for examples refer to Chang and Lauderdale 2005; Clarke et al. 2009; Wardle et al. 2002). Additionally, and

most importantly, the few studies that have interrogated the effects of all three indicators of social status have tended to focus on the separate influences of these social status indicators, rather than assuming that their effects could be multiplicative (Zhang and Wang 2004a, 2004b). Such analyses have left us with an incomplete understanding of social disparities associated with being overweight and/or obese. Finally, much of the findings from cross-sectional research on social disparities in BMI and longitudinal research on social disparities in weight gain are largely outdated. Thus, we have limited knowledge of which social groups are at an increased risk of becoming overweight and obese with the rising average weight of Americans.

Another major gap that I have identified in the obesity disparities literature, is the lack of studies investigating the role of obesity in shaping chronic health outcomes among different SES, racial/ethnic, and gender groups. The association between obesity, especially at the higher end of the BMI distribution, and negative health outcomes has been well documented (Must et al. 1999). The importance of social conditions in shaping population health has also been established (House 1990, Link and Phelan 1995). Additionally, studies that addressed the interplay between proximate and distal social factors have produced inconsistent findings. Some of the studies demonstrated a health risk amplification effect among the socially disadvantaged. For example, Pampel and Rogers (2004) found that negative health consequences of smoking were amplified by low SES. Krueger and Chang (2008) noted that physical inactivity was more health-damaging to those with less resources. In addition, a number of scholars found that alcohol consumption and smoking were more health damaging to Black relative to White adults (Haiman et al. 2006; Sempos et al. 2003; Stranges et al. 2004).

In contrast, other studies have found a lack of an amplification effect of health risk factors among the socially disadvantaged groups relative to their resource-rich counterparts. For example, Blaxter (1990) found that smoking had a more detrimental health effect on non-manual workers relative to manual workers. Similarly, Duncan, Jones, and Moon (1993) showed that in higher-SES communities, health differences between smokers and non-smokers were amplified, while the differences were less apparent in lower SES communities.

However, considerably fewer studies have examined whether higher levels of obesity are more health damaging for some social groups compared to others. Results from a fairly old study demonstrated that despite higher levels of obesity among Black adults, obesity was more strongly related to mortality among White women (Hogue 1987). A more recent study found that the amplification effect of BMI varied not only across social status indicators but also across health outcomes (Shafer and Ferraro 2011). Specifically, their research findings showed that the educational gradient amplified the effect of BMI on C-reactive protein (CRP; a measure of chronic stress) at low lower levels of obesity, but failed to amplify the effect of CRP at the levels of severe obesity. More intriguingly, disparities in educational achievement amplified the effect of BMI on disability among individuals who were classified as Class I and II obese. Overall, these results suggest that because BMI is more tightly related to inflammation, there is more "room" for the educational gradient to contribute to health inequalities at lower BMI levels. However, because the link between obesity and disability is "looser", the effect of educational inequalities is more prominent at the high end of the BMI distribution.

Despite the new insights brought by Shafer and Ferraro's (2011) study, the authors only considered the moderating effect of education, and did not test for possible interactions between BMI and other indicators of social status – such as gender, race/ethnicity, and income. Additionally, increase in adiposity has been linked with a number of other chronic conditions, including type 2 diabetes, hypertension, and hypercholesterolemia (Must et al. 1999), and it is not yet clear whether indicators of social status amplify or diminish the effects of BMI on other obesity-related health outcomes.

Motivated by the findings of the Shafer and Ferraro (2011) study, I investigated whether gender, race/ethnicity, and SES amplified the effects of body weight on cardiovascular health. In the United States, cardiovascular disease is the leading cause of death among men and women, and obesity has been identified as the primary mechanism leading to cardiovascular mortality (Go et al. 2014). In addition, because Shafer and Ferraro (2011) found varying effects of obesity of two health outcomes – CRP and disability – I wanted to test whether such variations would exist between chronic health outcomes that are more or less controllable by individual health behaviors. I chose arthritis as a control condition because, unlike cardiovascular health, it is largely independent of individual health behaviors, and mostly affects older adults, and, in particular, women (Felson et al. 2000).

To address the aforementioned gaps in the literature, I used the most recent data from the National Health and Nutrition Evaluation Survey (NHANES, 2011-2014), to answer two distinct yet analytically related research questions: 1) Do race/ethnicity and gender simultaneously shape the SES gradient in obesity?; and 2) Do distal social factors,

i.e. SES, race/ethnicity, and gender, shape the effect of obesity on arthritis and cardiovascular health indicators, such as diastolic and systolic blood pressure, fasting plasma glucose, and HDL cholesterol?

I theoretically grounded my research questions in the fundamental social causes of disease approach (FSCD). According to this theory, social factors are distal yet fundamental causes of health inequalities because they involves access to resources that can be utilized to avoid individual risk factors (Link and Phelan 1995). This approach has been widely used to examine disparities in various health behaviors and chronic health outcomes, such as smoking (Link and Phelan 2009), cancer screening tests (Link et al. 1998) and diabetes-self management (Lutfey and Freese 2005), yet has been seldom used in obesity scholarship. Guiding my research questions and hypotheses with this theory was appropriate because it places health risk factors, such as obesity, within the larger socio-cultural context, making them the key mechanism explaining disparities in health outcomes.

By theoretically situating research questions about disparities in the prevalence of obesity and the influence of overweight and obesity on chronic health, my goal was twofold. First, I wanted to provide further evidence that conceptualizing dimensions of social inequality as distinct is inadequate in fully understanding gender, racial/ethnic, and SES inequalities in health. As Williams and colleagues (2010) stated, gender, race/ethnicity, and socio-economic status matter for health outcomes both separately and in combination. We need to better understand what happens when these factors interact (p. 93). Because "purified" effects of each social factor are impossible to disentangle in the reality of the social world, they should be not be modelled as such. Second, I wanted

to investigate under what conditions distal social factors of gender, race/ethnicity, and SES interact with more proximate health risk factors to exert a multiplicative effect on health outcomes.

Research Findings and Study Implications

The focus of Chapter 5 was to address the first research question: Do race/ethnicity and gender simultaneously shape the SES gradient in obesity? Analyses conducted in this chapter add to the scholarship on social disparities in obesity prevalence by addressing the multiplicative effects gender, race/ethnicity, and socio-economic status on social distribution of individual BMI. Based on FSCD approach and previously conducted research in the field, I hypothesized that these indicators of social status would intersect in ways would put women of color at an increased risk of obesity regardless of their educational status and income level. In contrast, I expected White men and women of the highest income and education to be the most protected from higher levels of BMI. My findings confirmed these hypotheses and demonstrated the importance of the multiplicative effects of social statuses on body weight.

The results from moderation analyses uncovered stark gendered racial disparities in overweight and obesity that were not attenuated by high income and education, placing Black American women at a greater risk than Whites of having a higher BMI. Specifically, I found that increase in educational achievement not only affected body weight in opposite directions between men and women, but it also had a stronger overall effect on women's BMI relative to men's. For instance, at the lower levels of education, women had higher estimated mean BMI scores relative to men, yet at the highest level of educational achievement, the mean estimated BMI among women was much lower

relative to that of men, which supported hypothesis H1a. Similarly, in support with hypothesis H1d, I found that women at the lowest income levels had much higher estimated mean BMI levels relative to men. However, these differences became insignificant at the higher income levels. In fact, while increase in income exerted a protective effect from weight gain among women, it did not change the predicted mean BMI values for men.

This finding suggests that better access to material and cultural resources has a protective health effect for women, but not for men. While this finding has been confirmed in some previous research, it is still not clear why socio-economic advantages benefit women more so than men (Godley and McLaren 2010, McLaren 2007; Stunkard 1989; Wardle and Griffith 2001; Zhang and Wang 2004a). Evidence from studies conducted in the highly developed countries shows that individuals in higher socio-economic groups engage in healthier behaviors that also prevent from weight gain, such as consuming more fruits and vegetables and less fat (Power 2009). In addition, women of higher social status tend feel more pressure than men and lower status women to maintain lower body weight as a marker of their superior position in the society (Saguy 2013). Women of higher SES have also demonstrate greater body dissatisfaction relative to their lower SES counterparts (McLaren & Kuh 2004; Wardle & Griffith 2001). In contrast, norms of masculinity, such that larger bodies are valued as a sign of physical dominance, may promote engagement in physical activity that increases muscle mass, which in turn, increases BMI (Power 199). Finally, research has shown that women tend experience weight-based discrimination at much lower levels of BMI relative to men, which suggests that higher body weight among men is considered more socially

acceptable (Fikkan & Rothblum, 2012). These mechanisms, however, have not been tested directly in this study and future research should more closely examine the complexities associated with the effects of social determinants of health.

I also hypothesized that race/ethnicity would moderate the SES-BMI relationship, such that poor and less educated racial minorities will have higher estimated mean BMI levels relative to their lower SES White counterparts (hypotheses H1b and H1e, respectively). In partial support of my hypotheses, I found that higher educational achievement and income were protective against having a higher BMI among White and Hispanic, but not Black adults. In fact, Black adults maintained a higher mean estimated BMI regardless of their income and education. The weight contrast between socio-economically advantaged groups was particularly apparent when comparing Black women with individuals across and within racial/ethnic groups. Their mean BMI was estimated to remain at the "obese" category regardless of their income and education. While this finding may seem troubling, it is consistent with other studies where higher obesity prevalence among women of color could not be explained by individual and community-level SES (Robert and Reither 2004, Zhang and Wang 2004a, 2004b).

It is not clear why socio-economic advantage does little to protect women of color from weight gain but it could be potentially attributed to different notions surrounding beauty and body ideals. For instance, Rucker and Cash (1992) found that Black women have greater body satisfaction and find heavier bodies more attractive. In addition, Black women tend to perceive their body weight as normal even when they are officially categorized as overweight based on BMI measurement (Allan, Mayo, and Michel 1993; Flynn and Fitzgibbon 1996; Rand & Kuldau 1990). In contrast, Black women may be

particularly vulnerable to weight-based gain due to experiences of gendered and racial discrimination. The persistence of racial disparities in health as an outcome of discriminatory experiences has been well documented (Williams and Mohammed 2009), and the independent effect of racial discrimination on an increase in BMI has also been confirmed in a number of studies (Hunte and Williams 2009; Wines et al. 2007).

In addition to confirming some of the findings from previous studies, this study offers some new insights, particularly when it comes to weight disparities between Hispanics versus other racial/ethnic groups. While some scholars observed no statistically significant association between BMI and SES among Hispanic men and women (Khan, Sobal, and Martonell 1997), others have found a positive association among Hispanic men, yet no significant association among women (Zhang and Wang 2004a). While I expected the SES-BMI relationship among Hispanic adults to closely resemble that of Blacks, I was surprised to find that higher income and education among Hispanic adults was a significant buffering mechanism.

The rates of overweight and obesity have been on the rise among Hispanic Americans, and are now close to the prevalence observed among Black Americans (Odgen et al. 2014). However, when education and income are taken into consideration, the differences in BMI between Hispanic and White adults disappears. Even at lower income and education levels, Hispanic adults, like White adults, are estimated to have mean BMI scores that would place them in the overweight category. This finding suggests that Hispanic and White adults may overcome the barriers for maintaining a healthy weight with better access to material and cultural resources granted by better education and higher income. However, this finding may also mean that more educated

and well-off Hispanic women who have acculturated in the U.S. society, are more likely to embrace the U.S. cultural norms of beauty, preferring thin female bodies. Even though this hasn't been tested directly, researchers have found that Hispanic women who were born in the U.S. or emigrated before the age of 17 reported body dissatisfaction at rates similar to those of White women living in the U.S. (Lopez et al. 1995). Similarly, second-generation Hispanic women were more likely to engage in disordered eating patterns and showed more bulimic symptoms relative to first-generation immigrants (Chamorro & Flores-Ortiz, 2000).

It should be noted that in order to better understand that SES gradient in BMI among Hispanic adults, country of birth and acculturation status should be taken into consideration in future research. The increasing rates of obesity among Hispanic Americans are usually attributed to adoption of unhealthy lifestyles (Lara et al. 2005), but most studies do not differentiate between U.S.-born and non-U.S. born individuals. Such comparisons are, however, important because Hispanics are not a monolithic group and substantial health variations between ethic subgroups do exist, yet this heterogeneity has not been adequately appreciated in the majority of health research (Zsembik and Fennell 2005). With respect to obesity, some researchers have found a much weaker education gradient in BMI among non-U.S. born Mexican adults relative to U.S. born Mexican and White adults (Godman et al. 2006). Additionally, evidence suggests that the educational gradient in obesity emerges or becomes stronger with acculturation (Frank and Akresh 2013). Thus, it is crucial to better understand how and which Hispanic sub-groups are able to utilize health-promoting resources and the ways that their unique cultural and lifestyle characteristics interact with SES in producing variation in health outcomes.

The findings from three-way interactions were of particular importance as they made the weight disadvantage of Black women stand out. Such analyses are also novel methodologically as they have seldom been utilized to analyze joint influences of different social statuses on health outcomes. I hypothesized that gender and race/ethnicity would moderate the SES-BMI relationship such that the poor and least educated women of color would have the highest estimated BMI relative to other least educated gender and racial/ethnic groups. In addition, I expected most educated women of color to have higher estimated BMI relative to the lowest educated White men and women. I also projected that differences in education or income would have little effect on BMI among men in each racial/ethnic group (H1c and H1f).

In support to my hypotheses, I found that increases in education and income, especially at the higher end of the distribution, were associated with a sharp decrease in mean predicted BMI among White and Hispanic women. In contrast, higher educational achievement and higher income did not buffer the race effect among Black women. In addition, Black women were estimated to have higher mean BMI at all levels of education, which placed them in the "obese" category, when compared men and women in other racial/ethnic categories. Finally, the three-way interaction revealed little change in mean estimated BMI with the increase in income and education among men in all racial/ethnic groups.

Findings from three-way interactions provide a clear overall summary of the previously discussed relationships and underscore two important points. First, overall SES disparities in BMI are stronger among women relative to men. In other words, the effect of higher SES on individual body weight has greater salience for women. Second,

the effect of income and education among Black women is minimal, as the most educated and well-off Black women are estimated to have higher BMI when compared to the least educated poorest White and Hispanic men and women. In contrast, White and Hispanic women with at least a college education and household income corresponding to the mid-range of PIR are estimated to gain a lower BMI advantage, which is further predicted to increase with increasing SES. Finally, the pattern of the estimated income and education effect on BMI among White and Hispanic women is strikingly similar, and future research should seek to elucidate the mechanisms underlying this similarity.

Extending the research findings of Chapter 5, the main goal of Chapter 6 was to answer the research question: Do distal social factors (i.e. SES, race/ethnicity, and gender) shape the effect of obesity on arthritis and cardiovascular health indicators, such as diastolic and systolic blood pressure, fasting plasma glucose, and HDL cholesterol? The goal of this chapter was to examine the multiplicative effect of distal and more proximate risk factors on health outcomes that vary in the extent to which they may be controlled by individual behavior. Based on previously discussed research, I hypothesized that individuals of more disadvantaged social statuses and racial/ethnic minorities would experience larger negative cardiovascular health effects of increased body weight. Furthermore, I hypothesized that there would be no amplification in the case of arthritis. While the latter hypothesis was confirmed by my research findings, some of the results were opposite from what was predicted.

With respect to arthritis diagnosis, I found no interaction effects between BMI and gender, race/ethnicity, or SES, which supported hypotheses H2a, H2b, H2c, and H2d, but failed to support hypothesis H1b. Being a female was associated with increased odds

of arthritis diagnosis, while being a racial minority was associated with decreased odds of arthritis diagnosis. Additionally, as expected, increase in age and increase in BMI were significant independent correlates of arthritis, net of covariates. Interestingly, increase in income also had a protective effect from potential arthritis diagnosis. The non-linear pattern formed by the relationship between PIR and arthritis is interesting, and has not been well addressed in the literature. It could be hypothesized that individuals with lower incomes may be disproportionately employed in the manual labor sector, which increases the risk for joint "wear and tear" and injury. In contrast, most individuals in the mid- and higher income levels work more sedentary jobs, decreasing their risk of arthritis. This explanation has been partially supported by research demonstrating that some types of arthritis are more prevalent in economically deprived areas; however this association was attenuated by high obesity levels (Reyes et al. 2015). Overall, my findings demonstrate that social status indicators and BMI are significantly *independently* associated with arthritis, however, the distal social factors are irrelevant for the effect of BMI on arthritis diagnosis.

When considering cardiovascular health outcomes, increase in BMI was a significant correlate of worsening health in all four outcomes of interest; however, moderation analyses uncovered interesting patterns of variation in vulnerability to CVD. With respect to systolic and diastolic blood pressure, being a female and having at least some college education were associated with a lower mean blood pressure. Interestingly, being Black relative to White was linked to an increase in mean systolic blood pressure, while being Hispanic relative to White was linked to a decrease in mean diastolic blood pressure. The reasons underlying these discrepancies are not clear and call for further

investigation. Overall, the highest rates of hypertension have been observed among Black Americans, while Mexican Americans, and men in particular, have been reported to have lower rates of hypertension when compared to Whites, after controlling for age and socio-economic status (Kurian and Cardelli 2007). Some aspects of such patterning could partially be seen in my results.

The moderation analyses produced two significant and interesting findings. First, I observed a significant interaction between gender and diastolic blood pressure, such that men have higher estimated mean diastolic blood pressure values at higher BMI scores relative to women, which I did not expect to find (hypothesis H2a). Overall, while CVD risk factors for men and women are similar, women (up to about their mid-50s) tend to have lower incidence of hypertension, relative to men, though, their blood pressure – especially systolic blood pressure – tends to rise more sharply after mid-50s. There is some evidence that the protective effects of estrogen could at least partially explain this pattern (Rieker and Bird 2008). This may explain why the interaction was significant for diastolic and not systolic blood pressure. Because younger women tend to be protected more of high systolic blood pressure than men, men may also be more susceptible to some risk cardiovascular risk factors more than women. As I did not conduct three-way interactions between gender, BMI, and age, my findings present only a partial picture of possible gendered fluctuations in blood pressure that occur throughout the life-course. Further research is needed to better understand the mechanisms that give rise to such patterns.

Second, I observed a significant interaction between race/ethnicity and systolic and diastolic blood pressure, such that at lower levels of BMI, Hispanic Americans had

lower estimated mean diastolic and systolic blood pressure levels relative to Black and White Americans. However, at the higher end of the BMI distribution, the estimated mean level of diastolic blood pressure were similar in all three groups. Thus, the amplification effect was present, but only in the Hispanic group. This finding provides only partial support for my hypothesis (H2b), as I expected the effect of BMI to be amplified among Black Americans as well.

The overall higher blood pressure among Black Americans, despite the BMI value, could be attributed to higher rates of diagnosed and undiagnosed hypertension in that racial group, which has been well documented (Nwanko et al. 2013). However, the amplification effect among the Hispanics but not Whites is not clear given that the prevalence of hypertension is similar in both groups. It is possible that SES could be a contributing factor. Specifically, higher body weight may be less health damaging among White adults due to the residual confounding effect of their overall higher socio-economic status and better health practices. More research is needed to explain this relationship. For instance, considering the influence of acculturation and country of origin could give rise to a different situation, where certain Hispanic sub-groups or only long-term U.S. residents may experience the health-damaging effects. Some research has suggested that the general health status of non-US born Hispanics generally gets worse over time, but it is not clear if that pattern holds when considering the multiplicative effect of ethnicity and health risk factors on different health outcomes (Gordon-Larsen et al. 2003). In addition, Bostean and colleagues (2014) found that the disparities in cardiovascular health based on race/ethnicity and nativity status varied based on educational achievement, such that those with better education were better off overall.

Thus, in future studies scholars should further investigate whether education and ethnicity-nativity interact to influence differences in cardiovascular health.

Finally, the significant moderation effect between BMI and education when identifying the significant correlates of blood pressure, was only significant for systolic BP and was in the opposite direction from what I had hypothesized (H2c). In addition, when I tested for the interaction effect by running a binary regression examining the association between the independent variables and hypertension, the interaction term was not significant. Income also did not significantly moderate the BMI-BP relationship, thus hypothesis H2d was not supported for this cardiovascular health risk factor.

Racial/ethnic variations were also prevalent in fasting plasma glucose analyses. Based on the main model effects, women and those with higher education were estimated to have lower mean glucose levels, and Hispanic adults were predicted to have lower levels of mean fasting plasma glucose relative to Whites, controlling for covariates. The interaction effects between race/ethnicity and BMI revealed a distinctive finding inconsistent with the hypothesized one – that is, there was an amplification effect of BMI for Whites but not Blacks or Hispanics (H2b). This effect is striking given the generally lower rates of type 2 diabetes among Whites and their higher SES status relative to racial and ethnic minorities. As previously mentioned, in 2011-2012, among individuals aged 20 and older, 7.6% non-Hispanic Whites, 12.8% Hispanics, and 13.2% non-Hispanic Blacks were diagnosed with type 2 diabetes (Centers for Disease Control and Prevention 2011).

The lack of an amplification effect among Black Americans could partially be explained by their higher susceptibility to diabetes. For instance, research has found that

Black adults are more likely to develop diabetes at earlier ages and lower BMI relative to White adults (Brancati et al. 2000; Carnethon et al. 2002). In addition, Hispanic adults, especially Mexicans of the indigenous heritage, have a stronger genetic predisposition for adult onset of diabetes, which may not be exacerbated much by increasing BMI. Racial minorities are also more likely to engage in risky health behaviors, including tobacco use, alcohol consumption, and lower rates of physical activity (Gordon-Larsen et al. 2003; Williams and Collins 1995). In contrast, White adults may be more vulnerable to the health-damaging effects of higher body weight given their generally better health profile. That is, White individuals may experience a greater health loss from higher BMI relative to people of color and ethnic minorities who, due to their already low SES, experience less harm from higher body weight. Thus, healthy behaviors would grant more health benefits to Whites rather than Blacks or Hispanics.

A similar pattern emerged when analyzing HDL cholesterol and the negative effect of BMI among different racial/ethnic and SES groups. With respect to race/ethnicity, the negative effect of BMI was amplified for White adults but not Black or Hispanic adults. This finding was the opposite from the hypothesized relationship (H2b). In addition, the effect of BMI on HDL cholesterol was also amplified for individuals with higher educational achievement and household income, which failed to support hypotheses H2c and H2d. This finding could be interpreted in light of Blaxter's (1990) argument that individuals of lower SES and racial/ethnic minorities get little advantage in cardiovascular health from having lower body weight because of repeated exposure to a number of other health-damaging factors.

The explanation of amplification effect of BMI among Whites and individuals of higher SES for fasting blood glucose and HDL cholesterol is, however, inconsistent with the explanation of the vulnerability of Hispanic adults for hypertension. In the case of hypertension, I argued that the negative effect of BMI among White adults may be offset by their higher SES and access to resources needed to protect health. Following that logic, the question arises, why aren't White adults protected by their SES in the case of blood sugar and HDL cholesterol outcomes? This discrepancy could be potentially attributed to differences in biological, psychosocial, and/or coping mechanisms that have not been examined, or lack of simultaneous stratification by race/ethnicity and SES in all of my analyses. Regarding the latter explanation, it is possible that there is some variation in the extent to which White adults experience an amplification effect based on their SES status. In other words, the negative health effects of BMI among White adults with higher education and income may be stronger relative to White adults with lower education and income, and vice versa. I believe that further analyses are needed to uncover the complex mechanisms that may link race/ethnicity and SES with BMI and, in turn, different cardiovascular health outcomes.

Regarding the former explanation, some risk factors may have a more adverse impact on racial minorities relative to Whites, even if the overall levels of the aforementioned risk factors are lower or equal in the two groups. This statement has been supported by research on negative health effects of alcohol and tobacco consumption among diverse racial/ethnic groups (Sempos et al. 2003; Stranges et al. 2004). In contrast, several studies have found that some risk factors damage the health of White adults more strongly relative to Blacks. As an example, the rates of obesity are higher among Black

adults, however obesity is more strongly associated with mortality among White than Black women (Calle et al. 1999; Stevens 2000). In addition, persistent poverty is positively related to wasting and stunting in Whites and Hispanics, but not in Blacks (Hogue et al. 1987). Williams and colleagues (2010) pointed out that due to earlier and elevated exposure to some risk factors among Black Americans, these patterns could reflect a habituation effect that weakens the health-damaging effect of certain risk factors. Relatedly, it is possible that variation in the extent to which obesity may exert a negative effect on different indicators of hypertension or type 2 diabetes could be attributed to biological/genetic differences in racial/ethnic vulnerability and/or conditions under which certain cultural, psychosocial, and coping mechanisms can exacerbate or weaken the effects of obesity.

It is critical to note though that while BMI was associated with worse cardiovascular health, none of the CVD risk indicators reached the established risky levels, at overweight, Class I, and Class II obesity categories. This suggests that while BMI is a significant cardiovascular health risk factor, on its own, it does not pose a major health threat. It has been demonstrated that the well-established CVD health risk factors explain only about two thirds of the variation in cardiovascular disparities in the United States (Canto and Iskandrian 2003). Moreover, Zhang, Wang, and Huang (2009) found that between 1971 and 2004, ethnic disparities in type 2 diabetes increased among normal weight and overweight adults, while minimal disparities were observed among the obese. Additionally, some research suggests that being overweight could serve as a protective factor and predict better survival rates among individuals with hypertension and heart failure (Lavie et al. 2009). This is especially important given the established medical and

public health literature on obesity as the key biological gateway to compromised cardiovascular health. While my research results are limited in the extent to which they capture the complexity of cardiovascular health disparities, they do send an important message and underscore the findings of critical obesity research: having higher body weight does not necessarily result in poor physical health.

Overall, while the findings from Chapter 5 consistently supported my research hypotheses, the relationships revealed in Chapter 6 were much more nuanced and complex. Specifically, I expected that gender would not moderate the relationship between BMI and cardiovascular health indicators, while I hypothesized that it would significantly moderate the BMI-arthritis relationship. Based on my results, this hypothesis was partially supported as gender did moderate the BMI-diastolic blood pressure relationship, however it was not a significant moderator of the BMI-arthritis relationship. Further, I hypothesized that race/ethnicity would moderate the relationship between BMI and the CVD risk factors, such that Black and Hispanic Americans will be at an increased health disadvantage with increasing BMI relative to Whites. This hypothesis was also only partially supported. While, as expected, I found a BMI amplification effect among Hispanic adults for increase in systolic and diastolic blood pressure, I also found that increase in BMI made White adults more vulnerable for increasing levels of plasma glucose and decreasing levels of HDL cholesterol. Moreover, the hypothesis that increase in mean BMI would put the less educated at CVD risk (H2c) was not supported. Surprisingly, education was only significant for the BMI-HDL link. However, the moderation effect was opposite from what I had expected. In particular, more educated adults were placed at greater CVD risk with increasing BMI levels,

relative to their less educated counterparts. Similarly, my last hypothesis (H2d) was not supported, as income moderated the BMI-HDL link in the opposite direction expected, while the moderation effect was not significant for other cardiovascular health risk factors.

Study Implications

Methodological Implications

This study offers not only a conceptual contribution to the extension of fundamental social causes of disease (FSCD) theory, but also a notable methodological contribution in its use of anthropometric measures and biomarkers of obesity, type 2 diabetes, hypercholesterolemia, and hypertension that were available in the most recent waves of NHANES data (2011-2014). Because of potential biases associated with self-reports of health, my research results provide current accurate estimates that can be generalized to the U.S. population.

Theoretical Implications

In their (1995) article, Link and Phelan (1995) noted that in addition to socio-economic status, other social factors, such as race/ethnicity and gender could be considered as fundamental social causes of disease. While SES has received the most scholarly attention, researchers have also found support for gender and race/ethnicity as important social status categories and determinants of health, yet they are seldom examined more in depth independently or in combination. In addition, prior research grounded in the FSCD theoretical approach, has primarily focused on examining the health effect of one social status indicator at a time; thus know relatively little how much gender, race/ethnicity, and SES matter for health *in combination*. In addition, few

scholars have looked into the interplay between distal and proximate health risk factors on health, and we need more research that clarifies the mechanisms through which social status indicators and health risks accumulate to produce inequalities in health. My research extends the application of fundamental social causes of disease (FSCD) approach not only by considering individual and joint effects of three fundamental causes of disease – gender, race/ethnicity, and socio-economic status – but also by exploring the ways that distal social factors and proximate health risk factors interact to produce variations in health outcomes.

Finally, this study is one of the few that has supported the hypothesis of the health risk amplification effect among higher SES groups as opposed to individuals of lower social standing and racial minorities (Blaxter 1990; Calle et al. 1999; Hogie et al. 1987; Stevens 2000). Inconsistency in research findings leaves this field of inquiry with support for very contrasting arguments on the cumulative harm of low socio-economic status and more proximate health risk factors. Particularly, it demonstrates that the patterning of social factors and health risk factors outcomes is neither simple nor straightforward, particularly when considering diverse health outcomes. While some progress has been made to address the interdependencies between distal and proximate health risk factors, more research that would elucidate the pathways through which SES and race/ethnicity interact with different health risk factors to produce inequalities in health is necessary. Understanding the joint effects of distal social factors and more proximate health risk factors will also require greater attention to biological mechanisms and genetic predispositions as social, behavioral, and biological mechanisms jointly affect patterns of health (Das 2013).

Policy Implications

In terms of practical and policy applications, results of this study underscore the importance of addressing social inequalities in order to improve population health. In support to the main ideas of FSCD approach, the results of this research lead to different policies and interventions for addressing health inequalities than would the modifiable risk factor approach. That is, based on the risk-factor approach, a policy goal would be to identify the modifiable risk factors that link social factors to obesity and obesity-related health outcomes, and try to eliminate them. Following such logic, health interventions would target individual eating habits, engagement in physical activity, and reducing chronic stress. However, while addressing these intervening factors may be beneficial to health among those with more recourses, it would not improve overall levels of population health or reduce inequalities in health.

This point is well illustrated by my research findings. For instance, in the Chapter 5 analyses, Black women were estimated to have higher mean BMI relative to men and women of other racial/ethnic groups regardless of their income and education, controlling for individual health behaviors. Thus, in addressing the disproportionate rates of obesity among racial minorities by targeting more proximate risk factors would likely do little good for both poor women of color, and also for those who are already affluent and living healthy lives. In contrast, addressing the broader issues of gendered and racial discrimination would benefit individuals irrespective of their resources or behaviors. In addition, increased media attention to and further perpetuation of the medicalized obesity narrative would likely do more psychological harm than good to women who are already under constant pressure to fit in narrowly defined standards of beauty. In contrast,

enactment of strict anti-discriminatory work policies and equal gender pay would likely offer health benefits to all social groups. Limited success of behavioral interventions has also been supported by observational studies and clinical trials demonstrating difficulty in sustaining weight loss (Kraschnewski et al. 2010; Levy et al. 2007). Individual body weight is affected by a number of factors, which, other than behavior, include genetic, hormonal, and metabolic influences. These factors may predispose some individuals to obesity despite their eating and physical activity habits and vice versa.

In Chapter 6, I demonstrated that higher SES and being White amplified the negative health effect of BMI on HDL cholesterol and fasting plasma glucose, while variation in BMI had little effect on these health outcomes among racial/ethnic minorities. This means that reduction in weight would be beneficial to individuals of higher SES, while it would do little to improve the cardiovascular health among the more disadvantaged social groups. This finding also suggests that contrary to common media portrayals of the poor and minorities as embracing the culture of irresponsibility, obesity, and possibly other health behaviors, may not play a major role in cardiovascular health disparities. In all, my research results closely mirror the main claims of the FSCD showing that low status groups and racial/ethnic minorities would benefit little in terms of their cardiovascular health from improvement in some isolated aspects of their lifestyles and behavior. This claim is also supported by research studies which have demonstrated that cardiovascular risk factors – including smoking, high blood pressure, high cholesterol, and diabetes – do not fully account for or explain the excess burden of cardiovascular disease (CVD) in the population (Greenland et al. 2003; Magnus and Beaglehole 2001). There is reason to suspect that cumulative exposure to multi-

dimensional stressors, such as early childhood adversities, job strain, underemployment or unemployment, environmental hazards, social isolation and lack of social support, and discriminatory experiences may explain some of the remaining variation in CVD inequalities. However, up to date psychosocial stress remains an understudied contributor to SES–CVD gradient and little data exists on the effectiveness of stress-reduction interventions (Albert, Slopen, and Williams 2013).

Taken together, the evidence here reaffirms that getting rid of one proximate health risk factor without changing the underlying social inequalities yields little population health benefit and maintains disparities in health. Another important but less frequently articulated point that my findings support is that health interventions should avoid exclusively targeting the poor. This is ineffective because some racial/ethnic groups may experience poor health outcomes regardless of their socio-economic status, and all social groups could benefit from physical activity and healthier diets.

While such final remarks may seem to be painting a grim picture of the persistence of health disparities, it is valuable to note that despite the observation that the resource-rich have continuously been better off relative to their low-SES counterparts, the rates of obesity have levelled off (Odgen et al. 2014). Moreover, the cardiovascular disease mortality rates have declined over the past few decades due to improvement and availability of treatment options and lower smoking rates (Steptoe and Kiwimaki 2013). I would, however, like to emphasize that in order to improve the health at the population level, health interventions should either focus on reducing disparities in socio-economic resources or more equal distribution of salubrious resources across individuals from diverse social statuses. Chang and Lauderdale (2009) argue that health interventions that

are inexpensive and fairly easy to disseminate may be successful at reducing the SES gradient in health because they are not restricted to those who have access to the newest medical inventions. Such arguments were supported by Goldman and Lakdawalla (2005), who demonstrated a decrease in SES-CVD disparities with the introduction of beta-blockers for hypertension control.

Broader health policy recommendation following FSCD and these findings of this study are tightly connected to addressing social inequalities and should become a part of the national agenda. Examples of the some of the ways to reduce health disparities without specifically targeting individual behaviors include increasing the minimum wage, improving access to and the quality of housing for low income individuals, safer neighborhood environments and access to healthy food and recreational facilities, better unemployment and disability benefits, policies that address racial, gender and weight discrimination in diverse domains of social life, universal health insurance coverage, greater numbers of affordable community clinics, and similar initiatives of this type. In sum, contextualizing risk factors and identifying what social factors put people at particular health risk should be the starting point of the improvement in overall population health.

Limitations and Future Research

Despite the importance of these results, this study has several limitations that should be addressed in future research. First, due to data restrictions, I could not investigate the mechanisms that link indicators of higher social status with lower BMI and vice versa. While a number of scholars have looked into the role of individual health behaviors, it is still not clear why higher SES is associated with lower BMI among

women but not men. In addition, the studies that made efforts to address this question lack nationally representative data and measures of health behavior, psycho-social resources and stress variables. A tedious exploration of mediating pathways including health behaviors, psychosocial, and community-level measures could provide some insights into diverse material and cultural influences on variations in individual weight. In my analyses, I was only able to control for smoking status and sedentary behavior in addition to self-appraised health and insurance status. Consistent adjustment for possible confounding factors is crucial in considering the overall interpretations of the findings, and future studies should try to address this issue.

In addition, I have previously mentioned that the protective effects of income and education among Hispanic Americans in terms of BMI and cardiovascular health, and the extent to which they are moderated by acculturation, age at the time of immigration, and country of origin are not clear. Previous studies have noted that the overall health of non-U.S. born Hispanics is better relative to the U.S. born Hispanics but it starts to deteriorate with assimilation to mainstream U.S. culture (Abraido-Lanza, Chao, and Florez 2005). In contrast, other researchers have found that the acculturation process has a positive influence on adoption of preventative health care measures and better self-perceptions of health (Lara et al. 2005). It is still, however, unclear how socio-economic status interacts with the acculturation process in producing diverse health outcomes among Hispanic adults.

It is also important to note that due to restrictions associated with using secondary data, I could not performed more fine-tuned analyses of the effects of social factors on obesity and obesity comorbidities among different ethnic subgroups within the Hispanic

group. While much of what we know about Hispanic health in the United States is based on research performed on Mexican American samples, Hispanics are not a monolithic ethnic group and there are stark variations between individuals of Mexican, Puerto Rican, and Cuban origin, among others (Zsembik and Fennell 2005). Because I treated all adults of Hispanic origin as a monogamous group, it is very likely that my research conclusions are too simplistic given the group-specific variations in health.

In addition, type 2 diabetes, hypertension, and hypercholesterolemia are highly co-morbid, and having two of the three or all three conditions at once is quite common. While using different indicators of cardiovascular health as dependent variables uncovered important patterns in variations in health, future analyses should examine the effect of obesity and the moderating effects of SES, race/ethnicity, and gender on a constellation of co-morbid conditions as correlated outcomes in order to present a more realistic picture of the disparities in cardiovascular health in the U.S. population. Furthermore, in considering health outcomes associated with obesity, more research focused on general aspects of well-being and life satisfaction is needed, especially as weight discrimination has been found to be associated with negative physical and psychological health outcomes (Carr and Friedman 2005; Puhl and Heuer 2010; Shafer and Ferraro 2011).

Finally, due to used cross-sectional study design, my research findings should be interpreted with caution. Even though the majority of research has confirmed that social status is a strong determinant of health, some studies have found that obesity, especially among women, may negatively affect one's life chances and up-ward mobility (Glass, Hass, and Reither, 2010; Pudrovska et al. 2014). Similarly, poor health and chronic

illness has been found to be associated with a lower SES due to inhibited participation in the labor market (Adler and Ostrove 1999).

It should also be noted that the purpose of this study was not to test the FSCD approach, but to utilize it as a guiding framework for developing research questions and hypotheses. Whereas this theory has been widely used by medical sociologists and has proved its utility for understanding the persistence of inequalities in health, it is not without limitations. While it is widely agreed that excellent health is a desirable outcome and shaped by health behaviors among the majority of individuals, certain countervailing mechanisms may break the SES-health association. Status attainment has been identified as one of these potential mechanisms, and supported by previous research. For instance, Luftey and Freese (2005) in their ethnographic study of diabetes self-management found that some higher SES patients did not always follow their disease management plan due to the desire to stay thin and maintain occupational success.

While I did not consider testing for potential countervailing mechanisms in this study, systematic examination of them should be undertaken in the future. Analyses of pursuit of social status could possibly reveal engagement in unhealthy eating behaviors and psychological distress among White women who seek to maintain a slim body or Hispanic women who pursue the narrow Western definitions of female beauty. Additionally, ideals of masculinity as denial of the need for preventative health care may reveal the complexities of poorer men's cardiovascular health and disease management. Thus while in most circumstances, higher SES is compatible with individual behaviors and utilization of recourses that do lead to better health, it is important to identify

situations under which the association between social status and health may be diminished.

Despite these limitations, this study identifies promising avenues for future research that can enrich our understanding of how the social dimensions shape health outcomes. Future work should extend this study to considering the joint effect of gender, race/ethnicity, and SES and other health risk factors, including diet, exercise, smoking, and alcohol consumption. Future research should also examine how distal and more proximal health risk factors condition the social distribution of other chronic conditions. Finally, the incorporation of contextual factors into studies of the SES-BMI and SES-BMI-comorbidities nexus can provide a more comprehensive application of the FSCD theory and emphasize the interaction of individual and contextual characteristics in shaping health outcomes.

Conclusions

Overall, the results of this dissertation complicate the traditional story about obesity being a problem of the poor by showing that distal social forces interact simultaneously and with more proximal health risk factors to produce variations in individual weight and weight-related health disparities. In doing so, they encourage scholars to think more broadly about the SES-health relationship and to recognize the multiple mechanisms that intersect to shape health outcomes. Several main conclusions could be drawn from the research findings:

1) Social factors are important determinants of individual health, and should not be overlooked in future research. Instead of controlling for gender, race, or SES, I urge scholars to pay closer attention to combinations of these indicators of social status, as

they play a fundamental role in shaping population health. In addition, testing for multiplicative, in addition to cumulative, effects of multiple social statuses reveals a broader picture of health disparities that may otherwise be missed.

2) Socio-economic resources do not equally benefit the health of Whites, Blacks, and Hispanics. My research findings further exemplify the vulnerabilities of Black Americans, and Black women in particular, with respect to obesity and cardiovascular health, more broadly. While this finding is not at all surprising given the ample literature on racial health disparities, it warrants further investigation on how race "gets under the skin" and sustains inequalities in health.

3) Body weight on its own does not preclude an individual from having good health, which supports the obesity medicalization argument. In addition, my study results underscore the limitations of BMI, which is commonly used as an indicator of general health status. My research findings also emphasize the need to stop the war on obesity, view health more holistically, and consider the negative health consequences of weight-based discrimination.

4) Proximate and distal causes of disease interact in their influence on health, and more research is needed to better understand the multifarious ways in which gender, race/ethnicity, and SES can channel health risk factors to create and sustain health inequalities. Based on my research findings, while social factors may amplify the negative effect on obesity for more advantaged groups for some health outcomes, they may exert little or no effect on other health outcomes. Risk accumulation across achieved and ascribed social statuses is a rich area of study, and more research should

focus on uncovering the multiple interacting mechanisms that may lead to better population health.

Table 7.1. Summary Research Questions, Hypotheses and Findings

Research Question	Supporting Research Questions	Research Hypotheses	Corroborated?
1) Does race/ethnicity and gender shape the SES gradient in obesity?	a) Is the relationship between education and BMI moderated by gender?	H1a) Gender will moderate the relationship between education and BMI, such that women with lower levels of educational achievement will have higher estimated BMI, relative to men.	Yes
	b) Is the relationship between education and BMI moderated by race/ethnicity?	H1b) Race/ethnicity will moderate the relationship between education and BMI, such that less educated racial minorities will have higher estimated BMI, relative to less educated Whites.	Yes
	c) Is the relationship between education and BMI moderated by gender and race/ethnicity simultaneously?	H1c) Gender and race/ethnicity will moderate the relationship between BMI and education, such that the least educated women of color will have the highest estimated BMI relative to other least educated gender and racial/ethnic groups. In addition, the most highly educated women of color will have higher estimated BMI, relative to the lowest educated White men and women. Differences in education will have little effect on BMI among men in each racial/ethnic group.	Yes
	d) Is the relationship between income and BMI moderated by gender?	H1d) Gender will moderate the relationship between BMI and income, such that women in lower income categories will have higher estimated BMI, relative to men in lower income categories.	Yes
	e) Is the relationship between income and BMI moderated by race/ethnicity?	H1e) Race/ethnicity will moderate the relationship between income and BMI, such that racial minorities in the lowest income categories will have higher estimated BMI, relative	Yes

		to poor Whites.	
	f) Is the relationship between income and BMI moderated by gender and race/ethnicity simultaneously?	H1f) Gender and race/ethnicity will moderate the relationship between income and BMI, such that women of color in the lowest income categories will have the highest estimated BMI relative to other poorest gender and racial/ethnic groups. In addition, women of color in the highest income categories will have higher estimated BMI relative to men and women of all other racial/ethnic groups in the lowest income categories. Differences in income will have little effect on BMI among men in each racial/gender group, even though the relation is expected to be positive to racial/ethnic minorities.	
2. Do distal social factors, i.e. SES, race/ethnicity, and gender shape the effect of obesity on arthritis and cardiovascular health indicators, such as diastolic and systolic blood pressure, fasting plasma glucose and HDL cholesterol?	a) Is the relationship between BMI and each of the five health outcomes moderated by gender?	H2a) Gender will not moderate the relationship between BMI and the CVD risk factors but it will moderate the relationship between BMI and arthritis, such that women of higher BMI will be more likely to be diagnosed with arthritis.	Partially
	b) Is the relationship between BMI and each of the five health outcomes moderated by race/ethnicity?	H2b) Race/ethnicity will moderate the relationship between BMI and the CVD risk factors, such that Black and Hispanic Americans will be at an increased health disadvantage with increasing BMI, relative to Whites.	Partially

	c) Is the relationship between BMI and each of the five health outcomes moderated by education?	H2c) Education will moderate the relationship between BMI and the CVD risk factors, such that less educated individuals will be at an increased health disadvantage with increasing BMI, relative to more educated groups.	No
	d) Is the relationship between BMI and each of the five health outcomes moderated by income?	H2d) Income will moderate the relationship between BMI and the CVD risk factors, such that individuals at lower income groups will be at an increased health disadvantage with increasing BMI, relative to those in higher income categories.	No

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Williams, David R. and Chiquita Collins. 1995. "US Socioeconomic and Racial Differences in Health: Patterns and Explanations." *Annual Review of Sociology* 21:349–86.
Williams, David R. and Chiquitta Collins. 2001. "Racial Residential Segregation: A Fundamental Cause of Racial Disparities in Health." *Public Health Reports* 116(5):404-16.
Williams, David R., and Selina A. Mohammed. 2009. "Discrimination and racial disparities in health: evidence and needed research." *Journal of behavioral medicine* 32(1):20-47.
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Wright, Eric R. and Brea L. Perry. 2010. “Medical Sociology and Health Services Research Past Accomplishments and Future Policy Challenges.” *Journal of Health and Social Behavior* 51(1 suppl):S107-19.
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Zsembik, Barbara A., and Dana Fennell. 2005. "Ethnic variation in health and the determinants of health among Latinos." *Social science & medicine* 61(1):53-63.

VITA

GABRIELE CICIURKAITE

EDUCATION

2011 M.A., Sociology, East Carolina University
Thesis: Policy Reforms Targeting Care for Older Adults in Lithuania
Chair: Arunas Juska, PhD

2008 B.A., Sociology, Vilnius University
Thesis: Information and Communication Technologies (ICTs) as a Means For a Socially Active Life of Older Adults

RESEARCH AND TEACHING INTERESTS

Medical Sociology, Health Disparities, Mental Health, Quantitative Methods

PEER-REVIEWED ARTICLES

Juska, Arunas and **Gabriele Ciciurkaite**. 2015. "Older-age Care, Politics, Policy and Institutional Reforms in Lithuania." *Ageing and Society* 35(4): 725-749.

Kimuna, Sitawa, Yanyi K. Djamba, **Gabriele Ciciurkaite** and Survana Cheruki. 2013. "Domestic Violence in India: Insights from the 2005-2006 National Family Health Survey." *Journal of Interpersonal Violence* 28(4): 773-807.

PEER-REVIEWED BOOK CHAPTERS

Brown, Robyn L. and **Gabriele Ciciurkaite**. Forthcoming. "'I Get by with a Little Help from My Friends: Social Support and Mental Health." In *A Handbook for the Study of Mental Health (3rd edition),* edited by Teresa Scheid and Tony Brown. Oxford University Press.

Mooney, Patrick H., **Gabriele Ciciurkaite,** and Keiko Tanaka. 2014. "The Food Policy Council Movement in North America: A Convergence of Alternative Local Agrifood Interests?" In *Alternative Agrifood Movements: Convergence and Divergence*, edited by Douglas Constance, Marie-Christine Renard, and Marta G. Rivera-Ferre. Emerald Group Publishing Limited.

MANUSCRIPTS UNDER REVIEW

Ciciurkaite, Gabriele. "Time Out: A Critical Evaluation of Hyperlink Network Analysis."

Ciciurkaite, Gabriele and Brea L. Perry. "Body Weight, Perceived Weight Stigma and Mental Health among Women: Insights from the Modified Labeling Approach."

Schoenberg, Nancy, **Gabriele Ciciurkaite,** and Mary Kate Greenwood. "Community to Clinic Navigation (CCN) to Improve Diabetes Outcomes among a Rural Health Inequities Population."

Perry, Brea L., **Gabriele Ciciurkaite**, Christy Freadreacea Brady and Justin Garcia. "Partner Effects on Obesity-Related Health Behaviors: A Test of the Normative Body Size, Social Regulation, and Behavior Modeling Explanations."

MANUSCRIPTS IN PROGESS

Ciciurkaite, Gabriele Robyn L. Brown. "Food insecurity and psychological distress: Understanding the salience of work and family roles for gender disparities." Proposal accepted for submission to Volume 18 of Advances in Medical Sociology (Volume Editor Sara Shostak)

Ciciurkaite, Gabriele, Yelena Tarasenko, and Nancy Schoenberg. "Physical Activity among Rural Appalachians Participating in an Energy Balance Intervention: Does Self-Efficacy Matter?" In preparation for submission to *Health Education and Behavior*

Snell-Rood, Claire, **Gabriele Ciciurkaite,** and Nancy Schoenberg. "Depression and Readiness to adopt Physical Activity: Do Psychosocial Constructs in the Trans-Theoretical Model Account for Mental Health?" In preparation for submission to *American Journal of Health Promotion*

FELLOWSHIPS, HONORS AND AWARDS

2016	The O'Donnell Award for Outstanding Academic Performance, Department of Sociology, University of Kentucky
2016	Doris Wilkinson Award for the best paper in medical sociology, Department of Sociology, University of Kentucky (co-winner)

2013 Clifford C. Clogg Fellowship to attend the ICPSR Summer Program, University of Michigan. Awarded up to three graduate students in PhD programs ($2,300)

2012 Howard Beers Summer Fellowship, Department of Sociology, University of Kentucky ($1,000)

2011-2013 T. Marshall Hahn Graduate Student Fellowship, University of Kentucky ($5,000)

GRANT EXPERIENCE

University of Kentucky, Poverty Research Center in cooperation with the Economic Research Service (USDA) and University of Illinois, *FoodAPS Research Initiative.* "Food Insecurity, SNAP and Obesity Revisited: The Complex Relationship between Fundamental Social Causes and Health Outcomes." 2014. Role: Co-PI. Budget: $40,000 (Not funded)

RESEARCH EXPERIENCE

2015-present Research Assistant, University of Kentucky, Department of Sociology (PI: Claire Renzetti, Ph.D.) Project: "Religiosity, Religious Self-Regulation and Intimate Partner Violence"

Research Assistant, University of Kentucky, College of Social Work (PI: Michele Staton-Tindall, Ph.D.) Project: "Women's Intervention to Stop HIV/HCV (WISH) Project" NIH/NIDA R01 DA033866)

2014 Research Assistant, University of Kentucky, Department of Agricultural Economics (PI: Karen Rignall, Ph.D.) Project: "Economic impact of migration in Mgoun valley, Morocco"

2014-2015 Research Assistant, University of Kentucky, College of Medicine, Department of Behavioral Sciences (PI: Nancy Schoenberg, Ph.D.) Projects: "An Intergenerational Community-based Participatory Research (CBPR) Intervention to Reduce Appalachian Health Disparities" (NIH R01 DK081324); "Clinic to Community Navigation (CCN) to Improve Diabetes Outcomes"

2013-2014 Graduate Assistant, University of Kentucky, Quantitative Initiative for Policy and Social Research (QIPSR) (Director: Tom Janoski, Ph.D.)

2011-2013 Research Assistant, University of Kentucky, Department of Sociology (PI: Patrick H. Mooney, Ph.D.) Project: "The Food Policy Council Movement in North America"

2009-2010 Research Assistant, East Carolina University, Department of Sociology (PIs: Bob Edwards, Ph.D, Kenneth Andrews, Ph.D., and Neal Caren, Ph.D.
Project: assessment of newspaper coverage of environmental organizations in NC

PROFESSIONAL DEVELOPMENT

2015 Graduate Certificate in Applied Statistics. Department of Statistics, University of Kentucky

Intensive workshop on Item Response Theory (IRT). Quantitative Initiative for Policy and Social Research (QIPSR), University of Kentucky

Research Grant Building and Review Team (REGBART-G). Quantitative Initiative for Policy and Social Research (QIPSR), University of Kentucky

2014 Social Network Analysis workshop. Module: Advanced SNA. Mini module: Networks and Health. Links Center for Social Network Analysis, University of Kentucky

Structural Equation Modeling with STATA. Quantitative Initiative for Policy and Social Research (QIPSR), University of Kentucky

ICPSR Summer Program. Courses audited: Maximum Likelihood Estimation. University of Michigan

2013 ICPSR Summer Program. Courses taken: Applied Longitudinal Analysis, Structural Equation Modeling with Latent Variables. University of Michigan

Social Network Analysis workshop. Module: Advanced SNA. Links Center for Social Network Analysis, University of Kentucky

Applied Survey Data Analysis workshop. Quantitative Initiative for Policy and Social Research (QIPSR) Applied Statistics Lab (ASL), University of Kentucky

2012 Social Network Analysis workshop. Module: Analyzing Social Network Data. Links Center for Social Network Analysis, University of Kentucky

PRESENTATIONS

2015 Ciciurkaite, Gabriele. "Feeling Fat and Sad? The Relationship between Body Weight, Weight-Based Discrimination and Psychological Well-Being." Annual Sociological Association meetings, Chicago, IL

Staton-Tindall, Michele, J. Matthew Webster, Carl Leukefeld, Jennifer Havens, Carrie Oser, Megan Dickson, & Gabriele Ciciurkaite. "Women's Intervention to Stop HIV/HCV (WISH)." College on Problems of Drug Dependence meetings, Phoenix, AZ

2014 Ciciurkaite, Gabriele. "The Complex Relationship between Food Insecurity and Obesity: What Do we Know and Not Know about the Issue of Measurement?" Rural Sociological Society meetings, New Orleans, LA

2013 Mooney, H. Patrick, Gabriele Ciciurkaite, and Keiko Tanaka. "The Diffusion of the Food Policy Council Movement in North America." Rural Sociological Society meetings, New York, NY

2012 Ciciurkaite, Gabriele. "Examination of the Hyperlink Network Structure of the Community Food Security Coalition." Rural Sociological Society meetings, Chicago IL

Mooney, H. Patrick, Gabriele Ciciurkaite and Keiko Tanaka. "The Food Policy Council Movement in North America: A Convergence of Alternative Local Agrifood Interests?" International Rural Sociological Association meetings, Lisbon, Portugal

Ciciurkaite, Gabriele and Patrick H. Mooney. "The Food Policy Council Movement: An Exploratory Study." Southern Rural Sociological Association meetings, Birmingham, AL

2011 Ciciurkaite, Gabriele. "Path-Dependent Trajectories of Social Care for Older Adults in Lithuania." Southern Conference on Slavic Studies meetings, Alexandria, VA

2010 Sitawa, Kimuna, Gabriele Ciciurkaite and Survana Chernuki. "Gender-based Violence in India: Insights from the 2005-2006 National Family Health Survey." American Society of Criminology meetings, San Francisco, CA

Ciciurkaite, Gabriele and Arunas Juska. "NGOs as Social Service Providers for Older Adults in Lithuania." Southern Gerontology Society meetings, Richmond, VA

Juska, Arunas and Gabriele Ciciurkaite."Old Age Care Reforms in Lithuania." Southern Sociology Society meetings, Atlanta, GA

TEACHING EXPERIENCE

University of Kentucky

2016 Instructor, Quantitative Initiative for Policy and Social Research (QIPSR) Workshop: *Introduction to SPSS*

2015 Instructor, Quantitative Initiative for Policy and Social Research (QIPSR) Workshops: *Introduction to Stata* and *Stata Graphics*

Instructor, Quantitative Initiative for Policy and Social Research (QIPSR) Workshop: *Introduction to SPSS*

2014-2015 Teaching Assistant, Department of Sociology, University of Kentucky Graduate level *Introduction to Quantitative Political Methodology* and *Research Design and Analysis*

2014 Instructor, Quantitative Initiative for Policy and Social Research (QIPSR) Workshop: *Introduction to Stata*

2014 Primary Instructor, Department of Sociology, University of Kentucky *Quantitative Sociological Analysis (SOC 303)*

2013 Teaching Assistant, Department of Sociology, University of Kentucky *Introduction to Sociology*

2011 Teaching Assistant, Department of Sociology, East Carolina University Graduate-level *Social Statistics*

2010 Teaching Assistant Department of Sociology, East Carolina University *Social Problems*

SERVICE

Professional

Occasional Reviewer

2014-present Journal of Substance Abuse Treatment, Social Science and Medicine

Associations

2009-present Member, American Sociological Association
Member, Society for the Study of Social Problems
Member, Southern Sociological Society

2011-2012 Member, Rural Sociological Society
Member, Southern Rural Sociological Association

2009-present Alpha Kappa Delta: International Sociology Honor Society

Department

2013-present Graduate Student Representative, Quantitative Initiative for Policy and Social Research (QIPSR), University of Kentucky

2010-2011 Co-Secretary, Alpha Kappa Delta: International Sociology Honor Society, East Carolina University

2009-2010 Vice President, Alpha Kappa Delta: International Sociology Honor Society, East Carolina University