THE SOCIAL DETERMINANTS OF OBESITY

by

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To my family,
this accomplishment belongs to you as well

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ABSTRACT

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Obesity has become a major social and public health concern in the United States. The risk for obesity is not evenly distributed across racial/ethnic and socioeconomic groups and we know little about how obesity risk differs according to experiences in important life settings or how experiences accumulate over the life course to influence adult obesity. This dissertation begins by documenting how social disparities in body mass index (BMI) trajectories have changed in the U.S. population during a time of rapid growth in obesity rates. Drawing on intersectionality theory, I examine the multiple and interactive effects of inequality and find increasing racial and socioeconomic disparities in BMI trajectories over time, particularly among the youngest adults, such that black women with medium to high education and low to medium income levels experienced substantially larger increases in BMI over time, while white men with high education or high income levels experienced the least growth. I then investigate the relationship between early-life socioeconomic position (SEP) and adult BMI trajectories and determine which theoretical models of life course processes best explain how early-life SEP comes to influence adult BMI. I find enduring effects of early-life SEP that are

heavily mediated by adult characteristics and some evidence that racial/ethnic inequality in BMI is anchored in experiences in early-life, though life course SEP did not fully account for black/white differences in BMI trajectories. I also find that socioeconomic disadvantage accumulates over the life course and that early-life disadvantage in combination with adult disadvantage results in significantly higher BMI. In the third analytic chapter, I examine differences in BMI by different types of relationship change over time and different levels of relationship quality (i.e., stress and social support). I find that people who are continuously in a relationship have higher BMI than those who remain single and that entering a relationship is associated with a subsequent increase in BMI while exiting a relationship is associated with a decrease in BMI. I also find that BMI increases more for those individuals who are in stressful relationships.

CHAPTER I

INTRODUCTION

In a review of the stigma associated with being obese, Cahnman (1968) noted that although obesity was clearly a social phenomenon, it was "…hardly ever mentioned in the writings of sociologists" (p. 283). More than 40 years later, obesity is still seldom discussed in the sociological literature, and has received almost no attention outside of research on stigma and prejudice. Most of the scholarship on obesity comes from the fields of medicine and public health. Thus, obesity is often placed entirely in the province of public health. While obesity is indeed a significant public health issue given its association with disease, disability, and even death, obesity is also a social problem with both social causation and consequences. For instance, there are large racial, ethnic, gender, socioeconomic, and age differences in obesity. Moreover, social inequality in obesity suggests that the causes of obesity are social as well as biological and behavioral.

A sociological approach to the study of obesity could provide new insight into identifying those groups most at risk of obesity, thus improving our understanding of obesity-related inequality. In addition, using social theories linking macroscocial factors with individual experiences to identify the social causes of obesity could provide greater understanding of the unequal distribution of obesity. Studies of obesity as a social phenomenon can also contribute to the sociological literature by further developing existing theoretical frameworks.

The Rising Significance of Obesity

Some have argued that the United States is experiencing an obesity epidemic and have further suggested that the global burden of obesity represents a pandemic. Whether the current state of obesity prevalence in the U.S. population should be labeled an epidemic is hotly debated. Nevertheless, population-level rates of overweight and obesity have increased dramatically in last few decades. Overweight is often considered in tandem with obesity because increasing rates of overweight and obesity both reflect weight gain across the population. In this dissertation I primarily use language that focuses on obesity, but my primary aim is to understand the broader phenomenon of adult weight gain.

Substantial increases in overweight and obesity prevalence have been found in two leading national health surveys, the National Health and Nutrition Examination Survey (NHANES) and the Behavioral Risk Factor Surveillence System (BRFSS). NHANES data show increases in the prevalence of both overweight and obesity in the population aged 20 to 74 at ever wave of data collection, since data collection began in 1960. Between 1960 and 1980, the prevalence rates remained fairly stable (Flegal et al. 1998, Kuczmarski et al. 1994), but beginning in 1980 increases became much more dramatic. Overweight increased from 46.0% in 1976-1980 to 54.4% in 1988-1994 and continued to increase to 64.5% in 1999-2000 (Flegal et al. 1998; Flegal et al. 2002). By 2003/2004, over two-thirds (66.3%) of adults age 20 and older were classified as overweight (Ogden et al. 2006). Obesity also increased during this period from 14.5% in 1976-1980 to 22.5% in 1988-1994 and again to 30.5% by 1999-2000 (Flegal et al. 1998; Flegal et al. 2002). Obesity rates further increased to 32.2% in 2003/2004 (Ogden et al. 2006). BRFSS data

confirm increases in obesity from 1991 to 2000 (Mokdad et al. 1999; Mokdad et al 2001).[1]

With over two-thirds of adults classified as overweight and over one-third of adults classified as obese, it is perhaps not surprising that overweight and obesity are one of the Leading Health Indicators in Healthy People 2010 (HSS 2000), which was designed to provide objectives for improving the nation's health. Indicators are selected based on their "importance as public health issues" and because they "reflect the major health concerns in the United States at the beginning of the 21st century" (HSS 2000). To put the inclusion of overweight/obesity as an indicator in context, other indicators include heath care access, tobacco use, substance abuse, and mental health. Further signs that overweight and obesity have become major public health concerns include the 2001 Surgeon General's report highlighting overweight and obesity as serious public health issues (HHS 2001) and the recent National Institute of Health initiative to encourage and provide funds for obesity prevention research (Kumanyika 2003).

Physical and Mental Health Consequences of Obesity

Prior studies have consistently found a relationship between obesity and poor health. Obese individuals are at increased risk of experiencing cardiovascular disease (Bierman and Brunzel 1992; Eliahou, Shechter, and Blau 1992; Kenchaiah et al. 2002) respiratory disorders (Visscher and Seidell 2001), diabetes (Bonadonna and Defronzo 1992; Must et

[1] Estimates from the BRFSS tend to be about 10 percentage points lower than those reported using NHANES data. However, BRFSS data likely significantly underestimate obesity prevalence due to the sample design of the survey and the method used to measure height and weight. BRFSS uses a telephone sample and individuals without phones are more likely to be of low socioeconomic status, a factor associated with obesity. In addition, while NHANES uses objectively measured height and weight, BRFSS relies on individual self-reports. Self-reports tend to underestimate weight, particularly among overweight individuals, and overestimate height.

al. 1999), and certain cancers (Visscher and Seidell 2001). Some research even suggests that obese adults have higher rates of mortality and lower overall life expectancy (Allison et al. 1999; Fontaine et al. 2003; Manson et al. 1995; Peeters et al. 2003). The negative health effects of obesity may explain why obese individuals experience more hospital stays per year and spend more time in the hospital during those stays (Schafer and Ferraro 2007).

In addition to being a risk factor for multiple diseases, obesity can affect the quality of daily living. For instance, the more excess weight an individual has, the more likely she or he is to experience joint pain (Allison et al. 1999; Peeters et al. 2003) and functional limitations later in life (Peeters et al. 2004). Using a nationally representative study that tracked individuals over 20 years, Ferraro and Kelley-Moore (2003) found that individuals who had been obese or who were chronically obese had subsequently higher levels of lower body disability. Similarly, Himes (2000) found that among older adults, being obese was associated with increased limitations in performing activities of daily living (ADLs), including walking.

In addition to affecting one's physical health and quality of life, being obese may also be psychologically harmful. Obese individuals are more likely to experience depression (Ross 1994), have lower self-acceptance (Carr and Friedman 2005), and report feeling sad or worthless, and are less likely to report being happy or satisfied but more likely to (Carr, Friedman, and Jaffe 2007). However, being obese does not directly cause psychological harm. Instead, psychological distress results from experiences of physical strain, weight-related discrimination, interpersonal conflict, and the effort to conform to socially acceptable weight standards. Ross (1994) found that depression associated with

obesity is largely attributable to the psychological toll of dieting and being in poor physical health. Similarly, Carr and Friedman (2005, 2007) report that the positive association between psychological distress and weight largely results from the physical strain, discriminatory treatment, and stressful interpersonal interactions related to being overweight and obese. Thus being obese is not directly harmful to one's psychological health.

Obesity (and overweight) has become a significant population health concern not only because it has increased so dramatically in such a short period of time, but also because obesity is considered to be one of the major contributors to a number of preventable causes of death. In addition to the public health impact, I contend that the secular trends in obesity prevalence represent an important social issue as well, due to the many negative social consequences associated with being obese in the current U.S. social context. Further, it is important to remember that although obesity may be a population-level problem, it is experienced at the individual-level and can be a deeply personal issue.

Social Consequences of Obesity

In addition to causing psychological distress, social stigma related to obesity is also associated with prejudice that leads to mistreatment and discrimination against obese individuals. Obesity is a highly stigmatized condition that has become a social liability to those who are rejected by current cultural standards of acceptable body composition (Cahman 1968, Maddox, back, and Liederman 1968). Obese individuals may be the last acceptable targets of discrimination in the U.S. (O'hara 1996). Crandal (1995), for instance, likens weight-related prejudice to racism.

The stigma associated with obesity primarily arises from a misplaced belief that obesity is solely the result of personal actions, thus holding the individual entirely responsible. Obesity is as likely to be considered a health behavior – such as smoking, drinking, diet, etc. – as it a health condition. Health behaviors are often thought to be the individual's own responsibility (Knowles 1977), while getting sick is more likely to be considered outside the control of individuals. For instance, DeJong (1980) used an experimental study to determine that obese individuals who could not demonstrate non-behavioral causes of their obesity were less positively evaluated than those who had a medical basis for being obese.

The consequences of obesity stigma range from negative interpersonal interactions to institutional discrimination. Obese individuals are more likely to have less supportive and more problematic relationships with family members (Crandal 1995; Carr and Friednman 2006). In addition, obese individuals experience more discrimination in the workplace. Experimental research has shown that overweight and obese individuals are more likely to be negatively evaluated during hiring procedures (Rothblum 1992; Larkin and Pines 1979), and are therefore more likely to be exposed to weight-related hiring prejudice. Obese individuals are also more likely to experience a wage penalty (Loh 1993), report being fired because of their weight (Rothblum et al. 1990), and have lower promotion prospects (Brink 1988). The economic penalty of being obese, however, seems to accrue primarily to women (Pagan and Davila 1997). Obesity-related prejudice also extends to medical care. Obese individuals are viewed negatively by health care providers (Schwartz et al. 2003), and as a consequence receive lower quality medical care (Hebl and Xu 2001; Wee et al. 2000).

Perceptions of interpersonal mistreatment and institutional discrimination are fairly common among obese individuals. Over 40% of obese individuals report experiencing weight-related interpersonal mistreatment, and perceptions of mistreatment increase with weight (Falkner et al. 1999). In addition, obese individuals are about 50% more likely to report having experienced major discrimination compared to normal weight individuals (Carr and Friedman 2005). The social consequences of discrimination and mistreatment may have enduring effects for obese individuals due to the negative psychological consequences of exposure to discrimination (Carr and Friedman 2006; Kessler, Mickelson, and Williams 1999)

Organization of Dissertation

Strategies designed to reduce obesity that focus on individual causes of obesity have proven to be ineffective, especially in reducing the excess burden among the most disadvantaged who continue to beat higher risk. In this dissertation obesity is addressed as a population-level social problem shaped by a lifetime of exposures and experiences. For instance, socioeconomic conditions and health behaviors in adulthood that are related to obesity are the product of a lifetime of exposures and experiences. Yet we know relatively little about how obesity risk differs according to experiences in different social contexts, nor do we know how these experiences accumulate over the life course. In this dissertation, I seek to further develop a Sociology of Obesity that conceptualizes obesity as a social condition, with consequences for health, that develops over time and is shaped by individual social characteristics and experiences.

This dissertation contributes to a sociology of obesity by (1) documenting how social disparities in body mass index (BMI) trajectories have changed in the U.S. adult population during a time of rapid growth in obesity rates, (2) assessing the relationship between life course socioeconomic position (SEP) and adult BMI trajectories, and (3) examining the influence of relationship structure and quality on adult BMI.

Chapter II investigates racial and socioeconomic disparities in individual BMI growth trajectories 1986 to 2001/2002, a period of rapid increase in obesity rates. I use data from the Americans' Changing Lives (ACL), a nationally representative sample of the U.S. population in 1986, to estimate multilevel models of inter-individual differences in BMI trajectories. My analytic strategy for examining disparities is strongly informed by the theory of intersectionality. According to this paradigm, health inequalities are embedded in relationships defined by intersections of race, gender, and class - to which I also add age, an additional dimension of inequality that was not included in the original articulations of intersectionality theory. Whereas traditional health disparities scholarship investigates dimensions of inequality separately, I use a multiplicative framework that incorporates the interaction of race, gender, socioeconomic position, and age to examine the full extent of disparities in BMI growth over time. This framework accounts for important variations by group membership and provides a more detailed and complete picture of the nature of disparities.

I expect that fully multiplicative models will better detail the extent of social disparities in BMI trajectories, compared to a more typical additive approach. Based on prior cross-sectional and longitudinal studies of disparities in obesity change over time and theories of intersectionality and human development over the life course, I expect to

find, 1) higher BMI growth rates among blacks, which are primarily driven by higher rates among black women, 2) lower BMI growth among high educated white men and women and high income white men and, 3) higher BMI growth among high educated and high income black women, and possibly black men. Furthermore, I expect to find racial disparities in BMI growth among women but not necessarily men, and to see larger socioeconomic disparities in BMI growth among whites than blacks. The first study to take a fully intersectional approach to social disparities in the growth of obesity, I hypothesize that growth in obesity should be greatest among lower socioeconomic black women, and least among higher socioeconomic white men. Finally, I expect all of these social disparities in BMI growth to be more pronounced at the younger end of the adult age distribution, a time in the life course when weight gain is most pronounced.

Chapter II shifts the focus of the dissertation from an analysis of the inequality in BMI trajectories to an examination of the social factors that contribute to those BMI trajectories. I investigate the relationship between early-life SEP and adult BMI trajectories among women and determine which theoretical models of life course processes – Pathway Model, Latency Model, Cumulative Disadvantage Model – best explain the mechanisms linking early-life SEP to adult BMI. I also examine the extent to which life course SEP accounts for the large racial and ethnic disparities in BMI trajectories among U.S. women.

I utilize 15 years of adult weight history data and retrospective reports of early-life socioeconomic conditions from the ACL to estimate multilevel models of BMI change to assess the role of early-life SEP in shaping adult BMI trajectories. I first examine the independent effects of early-life SEP. I then examine the effects of early-life

SEP adjusted for adult SEP and other adult characteristics. I then turn to summary measures of accumulation of socioeconomic disadvantage to determine if there is a multiplicative effect of early-life and adult disadvantage. Finally, I analyze the differences in adult BMI trajectories according to patterns of educational mobility over the life course.

In Chapter IV, I examine differences in BMI associated with changing relationship status and quality. I use the ACL to estimate ordinary least squares regression models of (1) the effect of wave to wave relationship transitions on BMI and (2) the effect of relationship stress and spousal/partner social support on BMI. Based on prior empirical studies of relationship status and BMI I expect to find that, compared to remaining in a relationship, exiting a relationship will result in a lower BMI and entering a relationship will result in a higher BMI. I argue that the negative effect of relationships on BMI challenges an assumption in the literature on relationships; that being in a relationship is universally beneficial. Furthermore, I contend that the benefits of relationships depend largely on the quality of those relationships. Drawing from theoretical perspectives on the importance of relationship stress and support, I hypothesize that individuals who experience more stress in their relationships will have a higher BMI and that those who report high levels of spousal/partner positive social support will have a lower BMI.

In Chapter V, I synthesize findings from the three previous chapters, discuss the limitations of this research, and consider the broader implications of the findings presented in this dissertation.

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CHAPTER II

THE UNEQUAL BURDEN OF RISING OBESITY: AN INTERSECTIONAL APPROACH TO UNDERSTANDING RACIAL AND SOCIOECONOMIC DISPARITIES IN BMI TRAJECTORIES FROM 1986-2001

The total population burden of obesity in the United States has increased steadily over the past three decades, with overall prevalence rates more than doubling (Flegal et al. 1998; Ogden et al. 2006). Though women, African Americans, and individuals of low socioeconomic status have higher obesity rates (Chang and Lauderdale 2005; Okuson et al. 2004; Zhang and Wang 2004), it is unclear if the recent overall population growth in obesity is driven by accelerated obesity rates among these "higher risk" groups, by the remaining population (i.e., whites, men, individuals with high income and education) catching up to these groups, or by similar increases across all population groups. In the first case, racial and socioeconomic disparities would be *increasing*; in the second, though overall obesity prevalence would rise, the disparities would be *decreasing*; and in the last case, the existing disparities would remain similar over time.

This epidemiological trend of rising obesity is of public health concern, not only because it may lead to increased morbidity and mortality (Allison et al. 1999; Flegal et al. 2005; Fontaine et al. 2003; Kenchaiah et al. 2002; Mokdad et al. 2001; Must et al. 1999; Vischer and Seidell 2001) but also because a rise in racial and socioeconomic disparities in obesity would suggest a relative worsening of health among already vulnerable populations. Furthermore, health interventions designed to reduce overall obesity

prevalence would be more effective if they could target the populations in which the increases in obesity are actually occurring. Despite its salience as both a sociological and public health issue, there is surprisingly little research examining the racial and socioeconomic disparities in *changes* in obesity rates.

The existing research faces several limitations. First, most studies examining obesity disparities have described only cross-sectional relationships (Flegal et al. 1998; Kuzcmarski et al. 1994; Ogden et al. 2006; Okuson et al. 2004). Cross-sectional data provide static snap-shots of the population at a given time, but they do not provide insight into individual change over time. For instance, the increase in obesity found in studies using cross-sectional data may in some degree be a reflection of changing population composition by age, race/ethnicity, socioeconomic position, gender, or other factors instead of a reflection of actual individual growth. Furthermore, while cross-sectional studies establish that obesity rates are increasing in the total population, they generally offer limited insight into whether some groups are experiencing more, or less, of the overall increase. Those studies that have used longitudinal data to examine racial and socioeconomic disparities in individual change typically use only two measurement occasions, thereby treating change simply as the difference between values at two time points. However, this approach to studying individual change can not establish clearly whether the change between the two time points reflects random fluctuations in the data or true change over time (Rogosa, Brandt, and Zimowski 1982; Rogosa and Willet 1985; Singer and Willett 2003). This makes it difficult to determine from existing literature if racial and socioeconomic disparities in obesity are changing over time.

The literature on racial and socioeconomic disparities in obesity is further limited by an incomplete conceptualization of disparities. Studies of obesity disparities tend to address a limited set of dimensions of inequality, and although some cross-sectional studies do explore the interactive effect of several different dimensions of inequality, most studies of *change* focus exclusively on additive effects. However, it is the intersection of race, gender, and socioeconomic status that leads to the differential distribution of health risks (Krieger, Williams, and Moss 1993), thereby producing subgroup variation in health outcomes such as obesity, with age also playing an important role, especially in the case of attributes such as weight and obesity, which vary systematically over the life course. Thus, a proper conceptualization of disparities requires a simultaneous examination of the *multiple and interacting* demographic and socioeconomic determinants of obesity. Prior research, however, does not adequately explore disparities that exist at the intersections of gender, race, socioeconomic status, and age. For example, research shows that African American women have higher obesity prevalence than white women but it is unclear if this is the case for *all* African American women or if there is variation by age, education, or income in the difference between race/gender groups. Failure to account for the full set of dimensions of inequality - gender, race, and class – as well as potential age variation may mask important disparities in obesity, particularly in its change over time.

In this paper I attempt to address limitations in the current literature by using growth curve models to examine racial and socioeconomic disparities in body mass index (BMI) trajectories from 1986-2001 in a nationally representative U.S. sample. The growth curve analysis takes advantage of four waves of data to construct BMI

trajectories, thereby increasing the precision and reliability of estimates of change and trajectories. This study advances obesity disparities research by applying a theoretically-based model of inequality. Drawing from intersectionality theory I utilize a multidimensional and multiplicative framework for examining obesity disparities that is based on the intersection of multiple dimensions of inequality (e.g., gender, race, and class). Moving beyond the additive effects of these categories allows me to examine the full set of disparities in obesity growth. Furthermore, this study extends the existing intersectional paradigm by applying it to health disparities, and also by highlighting the importance of accounting for age variation in changes of disparities by race/ethnicity, gender, and socioeconomic position. To my knowledge this is the first study to examine *both racial and socioeconomic* disparities in BMI *trajectories* and to do so using a representative national sample of U.S. adults with detailed attention to the nuanced differences between population subgroups whose social standing lies at the intersection of gender, race, income and education levels, and age.

Prior Research on Disparities in Obesity Prevalence and Change

Cross-Sectional Evidence for Changing Racial and Socioeconomic Disparities in Obesity Prevalence

Replicated cross-sectional studies show considerable racial disparities in obesity prevalence between blacks and whites. Obesity is defined as having a body mass index of 30 or higher (NHLBI 2000), which translates into a weight of at least 174 pounds for an average height woman (64 inches) and 204 pounds for an average height man (69 inches). According to the most recent national data, 45% of blacks over age 20 are obese

compared to 30.6% of whites, a difference of nearly 15 percentage points (Ogden et al. 2006). This black-white disparity appears to be much larger than it was in the late 1970s when there was a difference of only 9.8 percentage points between the two groups (Flegal et al. 1998). These data suggest that blacks experienced more of the overall increase in obesity compared to whites.

However, in addition to racial variation in changes in obesity prevalence rates, there are important sex differences within race groups. In the aggregate it appears that black individuals have much higher obesity prevalence rates than white individuals, and that the disparity has increased over time. However, according to national data there are currently no significant differences in obesity rates between black and white men and this has been the case for the past three decades (Flegal et al. 1998; Ogden et al. 2006). Among women however, blacks are significantly more likely to be obese compared to whites. In 2003/2004 just over 30% of white women were obese compared to nearly 54% of black women (Ogden et al. 2006). This difference of 24 percentage points constitutes a sizeable increase in the black-white disparity, compared to rates in the late 1970s when there was only a 15.2 percentage point difference between the two groups (Flegal et al. 1998). Thus, increasing racial disparities in obesity seem to be the result of increasing racial differences among women and not men.

The secular trend towards an increase in obesity has also been subject to socioeconomic variation, both in the total population and within gender and race groups. Although obesity rates have increased at all levels of education and income, certain groups appear to have experienced greater increases than others (Chang and Lauderdale 2005; Zhang and Wang 2004). As a result, the nature of the relationship between

socioeconomic status and obesity has changed over time. For instance, Mokdad et al. (1999) found an inverse, linear relationship between education and obesity in the total population that has weakened over time. However, the extent to which this relationship has changed over time differs considerably by gender and race. Zhang and Wang (2004) found that the negative association between education and obesity has been observed primarily among white women, with much weaker relationships seen in black women and black and white men. Furthermore, while higher education tended to be protective for white women and men and, to a lesser extent, black women, the secular trend for black men shows that those with higher education had higher average BMI. This study indicates that education differences observed in the 1970s, 1980s, and 1990s in white women and men and black women were no longer significant by 1999/2000, but that the opposite is true for black men. It is less clear if observed racial and gender differences at comparable levels of education persisted over time.

As with education, obesity has increased at nearly all income levels but the magnitude of the change differs by race and gender. In an examination of the secular trend in associations between income and obesity, Chang and Lauderdale (2005) found greater increases in obesity prevalence at lower levels of income for white women and men, but among black women and men obesity increased the most at higher levels of income. In fact, black men at the highest income level transitioned from having the lowest obesity rates (relative to lower income black men) in 1971/1974 to having the highest rates in 1999/2000. In addition, as a result of these secular trends, the strong negative association between income and obesity initially observed in both white and black women attenuated by 1999/2000 for black women, while becoming stronger for

white men (Chang and Lauderdale 2005). This, study suggests that over time the protective effect of higher income has increased for white women and men but has decreased for black women and, most dramatically, for black men. While these secular trends highlight the changing nature of income disparities within race/gender groups, they provide only limited insight into changes in disparities between race/gender groups.

In sum, repeated cross-sectional studies of obesity prevalence rates indicate that racial disparities in obesity have been increasing, but that this is largely due to differences found between women, not men. In addition, while higher socioeconomic position has continued to be protective for white women and men over time, similarly positioned black women and men have become less advantaged in terms of obesity. While these cross-sectional studies describe secular changes in the population, they do not describe individual change, making it difficult to determine conclusively that the apparent changes in racial and socioeconomic disparities reflect actual subgroup differences in change over time and not just changing population composition. For instance, the apparent leveling of education disparities for most race/gender groups may be the result of population shifts in people of different weights across education levels. Thus, the existing research from cross-sectional data on trends in population obesity is useful for describing racial or socioeconomic disparities in prevalence rates at a given time, and for contrasting with rates from other periods, but it is not possible to draw conclusions about how individuals changed over time. Longitudinal studies of individual change are essential for answering questions about changing racial and socioeconomic disparities.

Racial and Socioeconomic Disparities in Individual Change

Longitudinal studies of obesity change in individuals have consistently found racial disparities among women, with mixed results for men. In a study of black-white differences in 10-year weight change in a national sample of adults aged 25-44 years, Kahn and Williamson (1991) found that BMI increased more for black women than it did for white women but that BMI in black men did not increase significantly more than white men, even after adjusting for education and income. Similarly, a study of race differences in weight trajectories over 34 years among adults aged 21 and older in Alameda California reported significantly higher weight gain among black women compared to white women, but no significant difference among men (Baltrus et al. 2005). In contrast, a multi-city study of young adults, aged 18-30 years, found significantly higher weight gain in both black men and women, compared to their white counterparts (Burke et al. 1996). Thus, prior studies consistently point to significant race differences in rates of BMI and weight change among women, while the results for men are less consistent.

Studies of socioeconomic disparities in obesity change have reported conflicting results, partly because some studies examine racial differences within socioeconomic categories while other studies examine socioeconomic differences within race categories. One study of middle aged women analyzed black-white differences in BMI change over time by level of education and found no significant effect of education on race differences (Lewis et al. 2005). In a multi-community, probability study of middle aged men and women, Mujahid et al. (2005) examined socioeconomic differences in 5-year

BMI change in models stratified by race and gender and found an inverse relationship between education and BMI change among white men and women. Among black men and women, on the other hand, they found a *positive* relationship between education and BMI change, though this relationship was not statistically significant for black men. Finally, in analyses stratified by race and gender, Kahn and Williamson (1991) found an inverse relationship between education and BMI change in both men and women. Some of these studies also included income but found no significant effect of income (Kahn and Williamson 1991) or did not report on the effect of income (Baltrus et al. 2005). Mujahid and colleagues (2005) found a significant positive effect of income on BMI change for black and white women, though there was no significant effect for men.

The inconsistencies in findings from prior longitudinal work on obesity disparities, especially socioeconomic disparities, may be somewhat accounted for by differences in study design, but may also result from using only two measurement occasions to study change. Studies of individual change in obesity typically utilize only two data points, which increases the possibility of measurement error and also selection bias. Studies that use only two data points to analyze change can not distinguish true change from random fluctuations in the data, making it difficult to draw conclusions about the nature of change in the outcome of interest (Singer and Willett 2003). In addition, because individuals can only be included in the analysis if they have a measurement for both occasions, those who dropped out after the first measurement occasion are not included in the analysis. This creates the potential for selection bias, which may lead to conflicting results between studies. Both measurement error and selection bias can be reduced by including more measurement occasions and analyzing

the *trajectory* of change (Rogosa, Brandt, and Zimowski 1982; Rogosa and Willett 1985). One exception is Baltrus and colleagues (2005), who analyzed trajectories of individual change using more than two measurements of BMI. Their study, however, was limited to a specific population (residents of Alameda County, California) and so the results are not necessarily generalizable to the U.S. population. Furthermore, like much of the prior work on obesity disparities, the Blatrus et al. study uses an additive approach to studying disparities which does not provide a complete picture of racial and socioeconomic disparities in obesity change.

Multiplicative Framework for Understanding Obesity Disparities

Prior studies of obesity disparities, particularly those that focus on longitudinal change, are limited by an incomplete conceptualization of disparities. In most of these studies gender and race are treated as being independent from class and are modeled as additive effects. Those studies that do make comparisons using multiple dimensions of inequality rarely do so for more than two dimensions. As a result, none of the existing research on disparities in obesity change has examined both racial and socioeconomic disparities *simultaneously and interactively*. For instance, Mujahid et al (2005) analyzed education and income disparities within race/gender groups, but did not examine these socioeconomic disparities across gender or race groups. This approach to studying obesity disparities derives from scholarly traditions that tend to treat gender, race, and class separately. However, treating these dimensions as separate and distinct is problematic because it ignores important relationships between different dimensions of inequality.

I use a multiplicative framework that incorporates the interaction of race, gender, socioeconomic position, and age to examine the full extent of disparities in obesity prevalence and rates of change. This framework accounts for important variations by gender, race, education, income, and age and provides a more detailed and complete picture of disparities. The idea of multiple, interacting dimensions of inequality is articulated most clearly by the theory of intersectionality, which endeavors to highlight the fundamental relationships between socio-cultural categories and identities (Crenshaw 1991; Hill Collins 1990). Whereas traditional health disparities scholarship investigates dimensions of inequality separately, the intersectional approach considers dimensions of inequality to be co-constructed. According to this paradigm, health inequalities are embedded in relationships defined by intersections of race, gender, and class - to which I also add age, an additional dimension of inequality that was not included in the original articulations of intersectionality theory.

The development of intersectionality theory has been paralleled in health disparities research with a move towards a more complete conceptualization of disparities. For instance, Kessler and Neighbors (1986) warned against setting race and social class against each other as predictors of health, arguing instead for an approach that considers the interactive effect of race and class. Because racial differences are more pronounced at certain levels of socioeconomic status (SES), modeling the joint influence of race and SES as additive effects is often an inadequate analytic strategy for revealing the full extent of racial and socioeconomic disparities in health (cf. also Farmer and Ferraro 2005). Most studies do not explicitly consider that the dimensions of inequality are not additive but multiplicative. Rather than being distinct, these dimensions of

inequality form specific combinations of identities that reflect unique historical experiences which cannot be captured by simply adding effects across categories (Krieger et al. 1997). Individuals do not experience obesity in terms of either gender or class or race but rather as individuals with specific combinations of gender, race, and class attributes – and I would further this to include age as well.

Previous studies of disparities in obesity change do not adequately account for variation by age, which also is not much considered in theories of intersectionality. Though previous studies include age as a potential confounder, no study examines racial and socioeconomic disparities by age. This is a potentially serious limitation because obesity rates vary considerably across age groups, reflecting life course and/or cohort variation in the way weight changes with age. It is essential that studies of obesity disparities account for age variation because changes in weight status have been shown to follow an age trajectory that is based in biology. Obesity prevalence tends to increase with age among adults in the 20-69 age group, but decreases with age thereafter (Flegal et al. 2002; Kuzcmarski et al. 1994). The observed age group variation in obesity rates is likely due to an inherent age trajectory of weight gain (Sheehan et al. 2003; Willet, Dietz, and Colditz 1999) as well as due to cohort effects (Reynolds and Himes 2007). Further, strong historical or period effects are often felt most keenly by those at more formative, often younger, ages (Mannheim 1952), and this may be accentuated in the case of obesity and BMI which increases more for biological reasons in early adulthood. Age is also an important factor to consider because it is a social phenomenon that has inherent significance for sociological studies of health disparities, both because aging is a fundamentally social process and because society is stratified by age (Foner 1975; Riley

1987). In fact, prior research has found that racial and socioeconomic disparities vary widely by age (House, Kessler, and Herzog 1990; House et al. 1994; Ross and Wu 1996). Thus, examinations of racial and socioeconomic disparities in obesity should also consider the effect of age variation.

In addition to broader applications in health inequality research, the intersectional approach to studying health can inform research on obesity disparities. This requires the development of analytic strategies that identify specific groups (characterized by race, class, gender, and age) most at risk of obesity, instead of assuming homogeneity within broad identity categories. Intersectionality theory provides a central perspective for this study with implications for the experience of BMI growth among subgroups of the population. Using a multiplicative framework for understanding disparities, I expect that additive models of BMI trajectories will not reveal the full extent of racial and socioeconomic disparities. Instead, models that explicitly examine differences in the interactions between gender, race, socioeconomic status, and age will more fully detail the extent of disparities in BMI trajectories.

Current Study

This paper extends the literature on disparities in obesity change over time by investigating racial and socioeconomic disparities in individual BMI growth trajectories in a nationally representative sample of U.S. adults. I examine both the additive and multiplicative effects of race, sex, education and income on 15-year change in BMI from 1986 to 2001. Using the theory of intersectionality as a starting point, I employ a multiplicative approach to understanding health disparities. I expect that fully

multiplicative models will better detail the extent of social disparities in obesity prevalence and growth rates, compared to a more typical additive approach. Based on prior cross-sectional and longitudinal studies of disparities in obesity change and theories of intersectionality and human development over the life course, I expect to find: 1) higher BMI growth rates among blacks, which are primarily driven by higher rates among black women, 2) lower BMI growth among high educated white men and women and high income white men, and 3) higher BMI growth among high educated and high income black women, and possibly black men. Furthermore, I expect to find racial disparities in BMI growth among women but not necessarily men, and to see larger socioeconomic disparities in BMI growth in whites than blacks. And as the first study to take a fully intersectional approach to social disparities in the growth of obesity, I hypothesize that growth in obesity should be greatest among lower socioeconomic black women, and slightest among higher socioeconomic white men. Finally, I expect all of these social disparities in BMI growth to be more pronounced at the younger end of the adult age distribution when weight gain is most pronounced.

DATA AND METHODS

Data

This research uses data from the Americans' Changing Lives (ACL) survey, a 15-year longitudinal study of the noninstitutionalized U.S. adult population age 25 and older (House, Kessler, and Herzog 1990; House et al. 1994). The study sample was obtained using a stratified, multistage area probability sample with an oversampling of both adults aged 60 and older and black adults. In 1986 (wave 1) face-to-face interviews were

conducted with 3,617 respondents, representing 70% of sampled households and 68% of sampled individuals. In 1989 (wave 2) follow-up face-to-face interviews were successfully completed with 2,867 (83%) of the surviving wave 1 respondents. A second follow-up was conducted in 1994 (wave 3) via telephone or, when necessary, via face-to-face interviews with 2,562 respondents or their proxies (n=164), representing 83% of wave 1 survivors. In 2001 and 2002 (wave 4), approximately 15 years after the initial interview, a fourth wave of follow-up was conducted via telephone, or face-to-face interviews when necessary, with 1,787 respondents or their proxies (n=95), representing 74% of the surviving original ACL sample.

Measures

Body Mass Index

BMI is calculated by dividing self-reported weight (in kilograms) by height (in meters) squared. Respondents were asked about their height in the baseline interview and were asked about their weight in every wave of data collection for which they were interviewed.[3] Those missing on height or weight (2.4%, 1.9%, 0.98%, and 2.2% of the W1, W2, W3, and W4 samples, respectively) were given imputed BMI values derived from sex-specific prediction equations that accounted for respondent's age, race, and prior height and weight (when available).

[3] Although height is only measured at baseline, this is not expected to have a notable effect on the results because the overall effect of height loss on BMI is modest (Sorkin, Multer, and Andres 1999). With increasing age, particularly after age 80, loss of height may produce an "artifactual" increase in BMI. Thus it is possible that BMI growth in older respondents will be estimated slightly differently than it would in studies where height is measured at every wave, though it is arguable whether this increases or decreases "error" in the estimation of body mass since the loss of height may primarily reflect spinal or postural compression as much as or more than any real change in body length.

Self-reports of height and weight are subject to reporting error but the error is sufficiently low that self-reports are considered to be reliable estimates of BMI (Bolton-Smith et al. 2000; Nawaz et al. 2001; Palta et al. 1982) and are thus not expected to bias results. Moreover, I compared self-reports of BMI in ACL wave 2 (1989) with national estimates of directly measured BMI from the NHANESIII-Phase I (1988-1991) and found minimal differences, ranging from lows of 0.1 BMI points for black men and 0.3 BMI points for white men and black women, to a high of 0.8 BMI points for white women (Kuczmarski et al. 1994).

Sociodemographics.

I include only white and black individuals in the analysis because the number of respondents from other racial and ethnic groups is insufficient to form a third comparison group (n=130 or 3.6% of the original sample). Race and gender are measured using dummy variables for *black* and *male*, respectively. Differences by combinations of race and gender characteristics are measured using dummy variables representing one of four race/gender categories: *white women*, *black women*, *black men*, and *white men*. I treat white women as the reference category because I am particularly interested in the contrast between white and black women.

According to prior work, the relationship between age and BMI is non-linear, and in addition there is considerable variation by cohort (Flegal et al. 2002; Kuczmarski et al. 1994; Reynolds and Himes 2007). I account for age differences in BMI by grouping individuals into the following 15-year age groups, based on their age in 1986: 25-39, 40-54, 55-69, and 70-84 (dummy coded using 25-39 as the reference). These age groups

were chosen to reflect the natural age trajectory of weight gain where weight gain begins, accelerates, decelerates, and ultimately declines, respectively. Individuals aged 85 and over were excluded from the analysis because they had an insufficient number of observations, due to death or drop out, to be treated as a distinct group in the growth curve analysis (n=71 or 1.96% of the original sample).

I use educational attainment and household income in 1986 as our measures of socioeconomic status. Educational attainment is represented by a three category dummy variable indicating less than 12 years, 12 years, and more than 12 years of schooling, with the last category treated as the reference. In preliminary analysis I found little substantive difference between individuals with some college and individuals with a college degree. I also choose a 3-category specification of education due to small sample sizes for those with more than 16 years of education. Family income, defined as the total pre-tax annual income of the respondent and his/her spouse/partner in 1986, is used as a measure of the respondent's economic situation at baseline. The income variable is coded into three categories representing less than $10,000, $10,000-$29,999, and $30,000 or more (reference category). These categories were created based on 1986 income and poverty levels to approximately represent below poverty level, at poverty to median income level, and above median income level, respectively. Income information was imputed for 311 cases that were missing on income using a regression-based imputation strategy (House et al. 1994).

Statistical Analysis

Growth curve analysis (Hedeker 2004; Singer and Willett 2003) is used to examine additive and multiplicative models of race, gender, age, education, and income

disparities in individual BMI trajectories from 1986 to 2002. Growth curve models are ideal for analyzing inter-individual differences in BMI growth over time (Heo et al. 2003). The individual BMI trajectory is jointly composed of an intercept for initial BMI status at baseline in 1986, a coefficient for the annual BMI growth rate, as well as random effects variances.

The slope for BMI growth is a function of time (plus covariates). In this study time is measured with a linear term representing annual growth where the first wave of data collection (1986) is treated as *time* = 0 and each follow-up observation is measured in terms of the number of years elapsed since the baseline interview (approximately 2.5, 7.5 and 15 years for wave 2, wave 3, and wave 4, respectively).[4] Thus, a one unit change in *time* is equivalent to annual change in BMI, also referred to as the annual growth.

Baseline BMI and annual growth, the components of the BMI trajectory, are modeled simultaneously in a two-level model, as a function of time-invariant individual characteristics. At level-1 I model the individual growth trajectory, which describes how individual BMI changes over time. At level-2 I model the inter-individual differences in these changes, which tell us how baseline BMI and annual growth, the components of the individual growth trajectory, vary across individuals. I specify the level-1 individual growth model as follows:

$$BMI_{ij} = \beta_{0j} + \beta_{1j}(TIME_{ij}) + \varepsilon_{ij} \; ; \quad \varepsilon_{ij} \sim N(0, \sigma_{\varepsilon}^2) \, ,$$

[4] Examination of empirical growth plots for randomly selected respondents showed that the functional form for BMI growth for most of the sample is approximately linear. Linear specification of the growth parameter is often the best approximation of change given a small number of data points; in this case four (Singer and Willet 2003). Furthermore, there is little to be gained from incorporating a quadratic term. We conducted a likelihood-ratio (LR) test for model fit using a linear vs. quadratic specification. We found a slight improvement in the LR of the quadratic specification over the linear specification, but the quadratic parameter itself was not significant and resulted in a reduction in the deviance of only 0.05%.

where β_{0j} represents the baseline BMI value and $\beta_{1j}(TIME_{ij})$ represents the annual BMI growth rate for person j, and ε_{ij} represents the normally distributed random effect or error variance with mean 0 and variance σ_{ε}^{2}. Both β_{0j} and β_{1j} are fixed effects coefficients that are further modeled as functions of covariates Z in the level-2 submodels:

$$\beta_{0j} = \gamma_{00} + \gamma_{01}Z_{1j} + \gamma_{02}Z_{2j} + \ldots + \gamma_{0k}Z_{kj} + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}Z_{1j} + \gamma_{12}Z_{2j} + \ldots + \gamma_{1k}Z_{kj} + u_{1j}$$

where u_{0j} and u_{1j} represent the random effects variances in the models. The covariates Z include the independent variables age, race, gender, education, income, and any multiplicative combinations therein as well as control variables. For each model I present both fixed effects coefficients (representing the initial BMI and rate of change in BMI) as well as the random effects variances, though I focus primarily on the fixed effects in our interpretation of models.

Wave-specific sample weights are applied in all descriptive statistics and statistical analyses. The wave 1 sample weight includes adjustment for the differential probability of selection at baseline and non-response rates, and a post stratification adjustment to the 1986 age/race/sex/region specific Census estimates of the U.S. population. Sample weights for waves 2, 3 and 4 adjust for non-response and attrition. Statistical analyses are conducted using HLM software (Raudenbush and Bryk 2002).

Attrition Correction

I correct for non-random attrition over the course of the study by using maximum likelihood (ML) estimation via the expectation maximization (EM) algorithm, an effective correction method for bias related to sample attrition (Little and Rubin 2002).

Under the assumption that the attrition process is conditional on a combination of unobserved variables as well as random chance, growth curve modeling with the ML-EM method produces unbiased coefficients in two ways. First, the ML-EM method retains full information on all sample members, even if they were not present at all waves (Feng et al 2006; Little and Rubin 2002). Thus, sample members are not dropped based on unobserved selection criteria. Second, I include covariates in our models that are associated with both the outcome and the likelihood of attrition, thereby strengthening our ability to correct for bias due to attrition (Cnaan, Laird, and Slasor 1997; Collins, Schafer, and Kam 2001; McArdle and Hamagami 1992). I also include controls for attrition due to death (respondents who died between 1986 and 2001) and non-response (respondents who were not present at all waves) in all models to control for any residual effect of attrition. Sample sizes used to estimate final models are reported in Appendix A and Appendix B.

RESULTS

I begin by calculating the unconditional intraclass correlation coefficient (ICC), a measure of the variability in BMI found between people, for the total sample and for each of the four race/gender groups. The ICC for the full sample is 0.804 (20.09/(4.91+20.09)). This ICC indicates that about 80% of the total variability in BMI over time is due to variation between individuals. The remaining variation is due to differences found within individuals over time, which reflects a combination of real change in BMI between measurement occasions, and some degree of measurement error. The ICCs for each race/gender group are as follows: 0.807 (21.53/(5.15+21.53)) for white

women, 0.742 (27.64/(9.61+27.64)) for black women, 0.773 (15.47/(4.54+15.47)) for black men, and 0.821 (16.42/(3.58+16.42)) for white men. According to these subgroup ICCs, most (77%-82%) of the total variability in BMI exists between individuals even within race/gender groups, which may be accounted for by adjusting for other covariates such as age, education, and income. The magnitude of these ICCs represents preliminary evidence for the importance of considering race/gender differences in addition to other characteristics such as education, income, and age.

I next present descriptive statistics – means and standard deviations for BMI and percentages for the remaining variables – for each race/gender category in Table 2.1. Among both men and women, BMI was higher for blacks throughout the study period. However, for men the race differences were negligible and not significant. Consistent with findings from national samples, black women had higher BMI values than their male counterparts, while for whites the opposite was found. At the beginning of the study each race/gender group had a BMI value considered to be in the overweight category (BMI>25.0), except white women who had a value very near the threshold between normal and overweight (24.8). In addition, each race/gender group experienced steady increases in BMI over the 15-year study period. Only for black women, however, did this increase through wave 4, combined with the higher initial value, result in an average BMI (30.4) that is considered obese (BMI>30.0). The age distribution is similar across race/gender groups, though white women have a slightly older age composition. White men and women have more years of schooling and higher incomes than their black counterparts.

[TABLE 2.1 ABOUT HERE]

Additive Effects

I first examine age, gender, race, and socioeconomic differences in BMI trajectories, assuming no interactive effects, results shown in Table 2.2. Differences in the BMI trajectory are examined in terms of *initial BMI*, which may be thought of as the cross-sectional differences in 1986, and a*nnual growth in BMI*, or the change in BMI over time.

In Model 1 I examine the differences by race and gender, controlling for age. Differences in initial BMI follow the typical age pattern with the highest initial BMI found among the middle aged (40-69), and with the oldest age group having only slightly higher BMI than the youngest. The annual growth rates of BMI between 1986 and 2001, however, are clearly greatest among the youngest (25-39) age group, consistent with the period effect of the obesity epidemic which has had its greatest impact at younger ages. These age effects, also seen in Table 3, concur with results from prior studies showing that weight gain accelerates through midlife, and then decelerates in older age.

Model 1 also shows that men have a higher initial BMI than women and that black individuals have a higher initial BMI than whites. In terms of annual growth in BMI between 1986 and 2001, men experienced smaller increases compared to women, as evidenced by the negative growth coefficient (0-.034, $p<.01$), but blacks experienced larger increases in BMI (.032, $p<.05$) than whites. These results indicate that over time the gender gap has been decreasing while the race gap has increased.

In Model 2 I added education and income. The differences in initial BMI and annual growth in BMI by age, gender, and race that were found in Model 1 are relatively unchanged with the addition of education and income. Consistent with prior work, I find an inverse relationship between education and initial BMI, but no significant difference by income. Thus, a purely additive model indicates educational (but not income) disparities in initial BMI levels (presumably reflecting residues of prior history or individual development prior to age 25), but no evidence of socioeconomic disparities in annual BMI growth by either education or income over the period. Thus a purely additive analysis suggests that the "obesity epidemic" has increased racial disparities in obesity, and left socioeconomic disparities largely unchanged.

[TABLE 2.2 ABOUT HERE]

Interactive Effects

Modeling solely additive effects, however, does not capture the effects that interactions among social characteristics may have on BMI, and may obscure the real changes in social disparities in BMI over time. Thus, I also modeled the interactive effects of gender, race, education, income, and age. The results for race, gender and education are presented in Table 2.3, with model numbering continued from Table 2.2.

Model 3 in Table 2.3 shows the results of interacting race with gender via a set of race/gender categories. The race difference in *initial BMI* observed in Table 2.2 is primarily a difference between women: black women had an initial BMI of 27.0 (24.19+2.81) compared to 24.19 for white women, while initial differences between

black and white men are negligible. Thus, the positive gender coefficient for initial BMI in Table 2 appears to reflect the fact that white men have a larger initial BMI than white women, since black men have a lower initial BMI than black women. The race/gender disparities in initial BMI shown in Model 3 mirror the race/gender disparities found in previous studies of cross-sectional differences in obesity.

The second column of Model 3 further specifies the disparities in annual BMI growth found in Table 2.2. Model 3 shows that the lower rate of growth among men (-.034) relative to women observed in Table 2 can be attributed almost entirely to the lower growth experienced by white men (-.034, $p<.01$). Model 3 further shows that the racial disparity in growth rates in Table 2 reflects, in about equal degrees, a higher rate of growth (.033. n.s.) among black women relative to white women, and a lower rate of growth (.034, $p<.001$) among white men relative to black men.

[TABLE 2.3 ABOUT HERE]

I also examined the interactive effects of race, gender, and education, and present the results in Model 4 of Table 2.3. The first column shows that education is inversely related to initial BMI for all race/gender groups. However, the relationship is much weaker among men than women. These findings are largely consistent with prior research, though I found more of an education gradient among men than has previously been reported. The strongest effects are for low-educated (<12 years) black women, who began the study with a baseline BMI of 28.62 (23.53+5.23=28.76).

The annual growth rates reveal a complex and partially unexpected pattern of results. Educational differences in BMI growth rates are not significant among white women, but among white men, growth rates are relatively lower among the most educated. In contrast, among black men and women, the highest rates of growth are among the *more* educated, especially those with 12 years of education. By examining the interactive effects of race, gender, and education I found heretofore undiscovered (in our Table 2.2 and prior data) socioeconomic disparities and gained a more nuanced understanding of racial disparities in annual growth BMI. For instance, I found that the effect of education on the racial disparity in BMI has acted in opposite directions among men, with BMI increasing more rapidly among better educated black men and among less educated white men. Thus, I found increased racial disparities in BMI among both higher and lower educated men and increased socioeconomic disparities among both black and a white men. In addition, the larger BMI growth rates found among higher educated black women result in increased racial disparities among better educated women (but not less educated ones), and reduced socioeconomic differences in BMI among black women over the course of the study period.

Though not shown here, I also examined the interactive effects of race, gender and income. I found a positive relationship between income and initial BMI among women but no relationship among men, a pattern consistent with prior research. I also found high-income white men had a negative BMI growth coefficient (-.056 $p<.05$), experiencing the least growth over time relative to other groups. Thus, the income differences in BMI remained largely unchanged over the period, with the exception of

increasing racial disparities among high-income men, and increasing socioeconomic disparities among white men.

Age-Specific Effects

Thus far age has been treated as a control, with results shown in the previous tables (as well as results from prior studies of BMI change) representing an averaged age effect across racial and socioeconomic groups. However, since age may interact with other characteristics in important ways that are masked in models that treat age as an additive effect, I investigated those interactions via age-specific models, with results shown in Table 2.4. I did not find the strong education gradient in baseline BMI in white women that was found in prior models or prior research. However, I did find a strong negative association between education and *initial BMI* in black women in the first three age groups (ages 25 to 69). Low-educated black women in these age groups have the highest initial BMI levels. I found no consistent relationship between education and initial BMI in men.

[TABLE 2.4 ABOUT HERE]

The largest differences in annual BMI growth are found between white and black women aged 25-39, especially high school-educated women. Black women in this education category experienced the most growth in BMI – about 0.35 points per year

(0.183+0.163), or a 5.2 point increase over the 15 years. The lowest growth was seen among high educated white men in all age groups except 55-69. These models also indicate that low-educated black men also experienced relatively low growth, though the effect is only significant among those aged 40-54. I found some additional differences in BMI growth across gender/race/education groups in the last two age groups (ages 55 to 84), but given limited sample sizes used to estimate these effects (see Appendix 2.A), these coefficients must be interpreted with caution and will not be discussed.

These results indicate that a much more complete picture of racial and socioeconomic disparities in BMI trajectories, both in terms of starting point and growth, emerges only after accounting for the interactive effects of race, gender, education, and age. I found that race and education disparities have in fact increased over time among adults aged 25-39, and to a lesser extent adults aged 40-54. Thus, examining racial and socioeconomic differences by age reveals disparities in BMI trajectories that were not apparent either in prior research or in our prior models, which merely controlled for age.

Age-specific race/gender/income differences in BMI trajectories are shown in Table 2.5. Initial BMI was not strongly patterned by income levels for any race/gender group, except that low-income black women aged 25-39 had the highest initial BMI ($23.23 + 4.44 = 27.67$; $p<.001$). Income is associated with annual BMI growth, especially for black women aged 25-39 and white men aged 25-54. Among black women aged 25-39, BMI growth appears to be inversely related to income, with a total increase in BMI points over the period of 5.1 ([0.187+0.152]*15) and 5.3 ([0.187+0.166]*15) for black women in the middle- and low-income categories, respectively –considerably higher than for high-income white women aged 25-39, who experienced a total increase of only 2.8

BMI points over the period. White men aged 25-54 at the highest income level had even lower growth (-0.061 p<.05and -0.063 p<.10 for 25-39 and 40-54, respectively) than high-income white women. In contrast, black men aged 40-54 also experienced less growth at both the middle (-0.120 p<.10) and low (-0.098 p<.01) income levels, though these effects should be interpreted with caution as they were estimated using relatively small sample sizes (see Appendix 2.B). I also found some indication of decreasing disparities in annual BMI growth at older ages, but again the results must be considered in light of small subgroup sample sizes (see Appendix 2.B).

[TABLE 2.5 ABOUT HERE]

Similar to the findings from Table 2.4, these results indicate important age differences in race/gender and socioeconomic disparities in BMI growth trajectories. For instance, the largest differences by race, gender, and income were found among adults aged 25-39. This indicates that while racial and socioeconomic disparities may not be increasing in the aggregate, they have been increasing among the youngest adults.

In sum, the patterning of growth in BMI levels is truly intersectional, especially in the portion of the life course (ages 25-39) most impacted by the rising obesity rates. In this relatively young age bracket, both poor and (somewhat anomalously) better educated black women have experienced massive increases in BMI of 5+ points in only 15 years, enough to move a person from normal weight to obese, or overweight to morbidly obese. In contrast, high-SES (high-educated and high-income) white men have shown the least increase in BMI, less even than high-SES white females. These strong intersections of

race, gender, and class with age, are simply not evident in prior research – nor in our simpler models in Tables 2.2 and 2.3.

CONCLUSION

Although it is clear that obesity in the United States has increased dramatically over the last several decades, it is less clear whether the increase in obesity has been distributed evenly along social dimensions of race, gender, class, and age. Using growth curve analysis, I examined racial and socioeconomic disparities in individual change in BMI to determine whether the total increase in obesity from 1986-2001 was concentrated among certain racial and socioeconomic groups. Drawing on the theory of intersectionality, I applied a multiplicative framework to examine differences in BMI trajectories by combinations of race, gender, and socioeconomic position (education and income), and extended this approach to include age as well. The findings revealed a more nuanced picture of racial and socioeconomic disparities than has emerged from any previous research.

Our findings confirm some of the results found in prior work on disparities in obesity change, but also yield important new insights. For instance, previous longitudinal studies of disparities in individual change in obesity over time have generally found that black women experienced greater increases compared to white women and that, compared to white men, black men experienced similar increases in obesity change (Baltrus et al. 2005; Burke et al. 1996; Kahn and Williamson 1991). Our data also indicate that black women have experienced more growth in BMI over time compared to white women, but

further reveal that this disparity exists largely among the youngest adults (aged 25-39). However, contrary to the prior findings of few racial differences among men, I found that white men, at least at higher levels of education and income, have experienced *less* growth than black men, among the youngest adults (ages 25-39). Thus our study suggests a widening of racial disparities in obesity over time among women, as was expected, but also among men, which has not been demonstrated in prior work.

Prior work has not focused on changes in gender differences or disparities in obesity. Our Table 2 suggested that gender differences in BMI have declined during the obesity epidemic as males, who had *higher initial levels of BMI*, have shown a *lower rate of annual growth in BMI*. However, further analysis reveals opposite trends for gender differences among whites vs. blacks. The overall evidence of declining gender disparities in obesity is entirely a function of *white males* having *lower rates of annual growth in BMI* than white females, while among blacks gender differences have increased as black females have experienced *higher rates of growth in BMI* than black males. All of this has been most true at younger ages.

I also found age-race-gender-specific evidence of socioeconomic disparities in *annual growth in BMI*, which have not been articulated in prior work on socioeconomic differences on changes in obesity over the course of the obesity epidemic. Thus, among adults aged 25-39, and to a certain degree those aged 40-54, I found that low income black women were the worst off in terms of growth in BMI, while high education and income white men were the best off since they experienced the least amount of growth in BMI. This contrast between lower class black women and upper class white men exemplifies the premise of the theory of intersectionality, which posits that individuals

are arrayed on a spectrum of inequality with the least advantaged (i.e. lower class black women) at one end and the most advantaged (i.e. upper class white men) at the opposite end. In terms of the obesity epidemic, lower class black women have experienced the worst outcomes, on top of their higher initial levels of BMI, while upper class white men have experienced a relatively better outcome.

This study demonstrates how conceptualizing dimensions of inequality as separate and distinct, as is done in the additive approach, is inadequate for fully detailing the extent of obesity disparities. Using a multiplicative approach that recognizes the unique intersection of multiple dimensions of inequality, such as race, gender, and class produces a more nuanced and informed picture of obesity disparities. However, even this intersectional approach does not adequately characterize the changing nature of disparities. Examining racial and socioeconomic disparities in BMI trajectories by age group revealed a more detailed picture of disparities than merely controlling for age. In age-stratified models I found increasing racial and socioeconomic disparities primarily among adults aged 25-39, and to a lesser extent among adults aged 40-54.

The intersectional perspective stresses the importance of accounting for the unique lived experiences of different individuals, yet writings on the theory of intersectionality do not incorporate age as an important dimension of the lived experience (Crenshaw 1995; Hill Collins 1990). This is a particularly important omission with regard to intersectional approaches to studying obesity disparities since trajectories of BMI growth have a natural aging pattern and may also vary by cohort membership. I found increasing racial and socioeconomic disparities in obesity growth among younger adults, a finding that would have been masked had I simply treated age as a control and not an

important additional dimension along which to study obesity differences. Thus, the intersectional approach to understanding health disparities can be enhanced by integrating perspectives on cohort differences and human development over the life course.

The analysis in this study benefited from the use of growth curve analysis, which allowed us to directly model individual change over time. Most prior studies of disparities in obesity change have relied on only two observation points for measuring change. By using multiple observations in a growth curve analysis, I was able to isolate the effect of random fluctuations between observations from the real changes taking place over time. Thus, this represents a methodological advance over prior studies that use only two observation points to study change.

This study, the first to simultaneously examine racial and socioeconomic disparities in BMI trajectories, identifies those groups who have experienced a greater burden of the increase in obesity across the U.S. population. The findings, when taken in light of the health impact of obesity, paint a dismal picture of the future of adult health disparities. Some groups, particularly lower class black women, may have potentially even poorer health moving forward than they do now, while upper class white men may become increasingly advantaged. Understanding trends and projections for the future of obesity, and health disparities more generally, requires a focus on younger ages because these individuals have yet to experience the full extent of weight gain associated with the aging process. Finally, the increasing social disparities found in this study argue strongly for recognition of the obesity epidemic as a major sociological problem in addition to being an important public health issue.

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TABLES

Table 2.1. Descriptive Statistics by Race and Gender (N=3426)

	Black Women	White Women	Black Men	White Men
Mean BMI				
1986	27.6 (5.8) †‡	24.8 (4.9) †‡	26.2 (4.2) †	26.1 (4.1) †
1989	28.1 (6.2) †‡	25.3 (5.0) †‡	26.7 (4.4) †	26.6 (4.4) †
1994	28.6 (5.9) †‡	25.7 (5.1) †‡	27.3 (4.1) †	27.0 (4.6) †
2001/2002	30.4 (6.7) †‡	26.7 (5.6) †‡	28.5 (4.8) †	27.9 (4.9) †
Age				
25-39	43.2%	37.4% †	44.0%	45.0% †
40-54	27.5%	25.5%	32.0%	23.5%
55-69	18.5%	23.0% †	16.5%	21.8% †
70-84	10.9%	14.1% †	7.5%	9.8% †
Education				
>12 years	30.0% ‡	40.5% †‡	38.1% ‡	48.8% †‡
12 years	29.9%	36.1% †	23.2%	29.0% †
<12 years	40.1% ‡	23.5% ‡	38.6% ‡	22.2% ‡
Income				
$30,000+	21.8% †‡	39.5% †‡	22.4% †‡	47.2% †‡
$10,000-30,000	34.6%	41.0%	43.8%	40.6%
< $10,000	43.6% †‡	19.5% †‡	33.8% †‡	12.2% †‡
Unweighted N	*763*	*1,385*	*390*	*888*
Weighted N	*228*	*1,586*	*172*	*1,440*

Notes: BMI = body mass index. Numbers represent weighted means with stadard deviations in parentheses. Baseline values for age, education, and income are reported.

† Denotes significant (at 95% Confidence Level) sex differences within race.

‡ Denotes significant (at 95% Confidence Level) reace differences within sex.

Table 2.2. Independent Effects of Race, Gender, Age, Education, and Income on Initial BMI and Annual BMI Growth

	Model 1				Model 2			
	Initial		Annual Growth		Initial		Annual Growth	
Constant [a]	24.35 (0.16)	***	0.195 (0.009)	***	23.97 (0.19)	***	0.182 (0.011)	***
Age (ref= age 25-39)								
Age 40-54	1.59 (0.20)	***	-0.050 (0.011)	**	1.45 (0.20)	***	-0.047 (0.011)	**
Age 55-69	1.64 (0.23)	***	-0.207 (0.013)	***	1.25 (0.23)	***	-0.206 (0.013)	***
Age 70-84	0.83 (0.31)	*	-0.304 (0.021)	***	0.33 (0.32)		-0.300 (0.022)	***
Male (ref=female)	1.05 (0.16)	***	-0.034 (0.009)	**	1.10 (0.16)	***	-0.032 (0.010)	*
Black (ref=white)	1.64 (0.24)	***	0.032 (0.015)	*	1.44 (0.25)	***	0.034 (0.016)	*
Education (ref=12+ years)								
12 years of education					0.55 (0.19)	*	0.018 (0.011)	
<12 years of education					1.42 (0.23)	***	-0.024 (0.014)	
Income (ref=$30,000+)								
Income $10,000-29,999					0.19 (0.18)		0.016 (0.011)	
Income < $10,000					0.10 (0.26)		0.017 (0.016)	
Attrition (ref=not missing)								
Death	-0.37 (0.24)		-0.077 (0.020)	**	-0.51 (0.24)	†	-0.073 (0.021)	**
Non-Response	-0.52 (0.18)	*	-0.009 (0.013)		-0.69 (0.18)	**	-0.008 (0.013)	
Variance Components								
Level 1: Within-person	2.628				2.628			
Level 2: Between-person	19.57 (0.00)	***	0.0317 (0.00)	***	19.30 (0.00)	***	0.0315 (0.00)	***
Goodness of Fit								
Deviance (-2 LL)	52,641				52,617			
BIC (smaller is better)	80,461				80,404			

†p<.10; *p<.05; **p<.01; ***p<.001 (two-tailed tests)

Note: Regression coefficients with standard errors in parentheses are shown. Standard errors are calculated using robust estimation. N = 3426. BIC = Bayesian Information Criterion.

[a] Constant represents a white female aged 25-39 in Model 1 and a white female aged 25-39 with more than 12 years of education and having a houshold income more than 30k in Model 2.

Table 2.3. Interactive Effect of Race/Gender (Model 3) and Race/Gender/Education (Model 4) on Initial BMI and Annual BMI Growth

	Model 3		Model 4	
	Initial	Annual Growth	Initial	Annual Growth
Constant [a]	24.19 (0.16) ***	0.195 (0.009) ***	23.53 (0.21) ***	0.192 (0.011) ***
Age (ref=25-39)				
40-54	1.62 (0.20) ***	-0.050 (0.011) **	1.50 (0.20) ***	-0.049 (0.011) **
55-69	1.66 (0.22) ***	-0.207 (0.013) ***	1.30 (0.23) ***	-0.205 (0.013) ***
70-84	0.86 (0.31) **	-0.303 (0.021) ***	0.37 (0.31)	-0.296 (0.022) ***
White Female	-ref-	-ref-	- -	- -
>12 years of education	- -	- -	-ref-	-ref-
12 years of education	- -	- -	0.90 (0.27) **	0.016 (0.015)
<12 years of education	- -	- -	2.31 (0.30) ***	-0.024 (0.019)
Black Female	2.81 (0.32) ***	0.033 (0.020)	- -	- -
>12 years of education	- -	- -	2.08 (0.57) ***	0.048 (0.033)
12 years of education	- -	- -	3.23 (0.57) ***	0.092 (0.036) *
<12 years of education	- -	- -	5.23 (0.50) ***	-0.024 (0.033)
Black Male	1.51 (0.36) ***	-0.005 (0.024)	- -	- -
>12 years of education	- -	- -	2.33 (0.58) ***	0.033 (0.036)
12 years of education	- -	- -	2.37 (0.72) ***	0.039 (0.048) *
<12 years of education	- -	- -	2.49 (0.58) ***	-0.086 (0.042) *
White Male	1.37 (0.17) ***	-0.034 (0.010) **	- -	- -
>12 years of education	- -	- -	2.08 (0.25) ***	-0.043 (0.014) *
12 years of education	- -	- -	2.35 (0.29) ***	-0.020 (0.017)
<12 years of education	- -	- -	2.46 (0.32) ***	-0.023 (0.021)
Attrition (ref=not missing)				
Death	-0.38 (0.24)	-0.077 (0.020) **	-0.46 (0.24)	-0.074 (0.021) ***
Non-Response	-0.52 (0.18) *	-0.009 (0.013)	-0.66 (0.18) ***	-0.009 (0.013)
Variance Components				
Level 1: Within-person	2.644		2.703	
Level 2: Between-person	19.37 (0.00) ***	0.0315 (0.00) ***	18.93 (0.00) ***	0.0307 (0.00) ***
Goodness of Fit				
Deviance (-2 LL)	52,613		52,560	
BIC (smaller is better)	80,425		80,306	

†p<.10; *p<.05; **p<.01; ***p<.001 (two-tailed tests)
Note: Regression coefficients with standard errors in parentheses are shown. Standard errors are calculated using robust estimation. N = 3426. BIC = Bayesian Information Criterion.
[a] Constant represents a white female aged 25-39 in Model 3 and a white female aged 25-39 with more than 12 years of education and having a houshold income more than 30k in Model 4.

Table 2.4. Age Stratified Models of Race/Gender by Education Differences in Inital BMI and Annual BMI Growth.

	25-39		40-54		55-69		70-84	
	Initial	Annual Growth	Initial	Annual Growth	Initial	Annual Growth	Initial	Annual Growth
Constant [a]	23.32 (0.31) ***	0.183 (0.016) ***	24.71 (0.43) ***	0.165 (0.023) ***	25.66 (0.41) ***	-0.039 (0.023) †	24.68 (0.53) ***	-0.066 (0.037) *
White Female								
>12 years of education								
12 years of education	1.29 (0.48) *	0.001 (0.026)	0.23 (0.50)	0.013 (0.034)	5.26 (0.75)	0.079 (0.029) *	0.40 (0.64)	-0.088 (0.048) †
<12 years of education	0.65 (0.68)	0.046 (0.038)	2.29 (0.53) **	-0.032 (0.044)	0.89 (0.82) ***	-0.058 (0.032)	1.56 (0.57) *	-0.072 (0.045)
Black Female								
>12 years of education	1.79 (0.86) **	0.096 (0.049) *	2.79 (1.32) **	-0.031 (0.075)	3.11 (1.74) **	-0.043 (0.105)	3.66 (2.14) **	-0.047 (0.176)
12 years of education	2.96 (0.92) ***	0.163 (0.057) **	4.01 (1.27) ***	0.050 (0.078)	3.47 (1.25) **	-0.196 (0.090) *	2.13 (2.01)	0.145 (0.203) ***
<12 years of education	5.96 (1.16) ***	0.092 (0.073)	5.10 (1.18) ***	-0.097 (0.072) †	5.51 (0.80) ***	-0.032 (0.052)	2.89 (0.89) ***	-0.094 (0.075)
Black Male								
>12 years of education	2.35 (0.82) ***	0.046 (0.049)	2.75 (1.59) **	0.007 (0.103)	2.43 (1.58) *	-0.043 (0.105)	0.40 (2.73)	0.129 (0.259)
12 years of education	3.50 (1.27) **	0.080 (0.087)	1.50 (1.36) †	0.006 (0.088)	2.35 (1.81) †	-0.196 (0.090)	-0.10 (3.53)	0.277 (0.337)
<12 years of education	2.50 (1.49) **	-0.067 (0.102)	3.17 (1.16) **	-0.197 (0.076) **	1.31 (0.93)	-0.032 (0.052)	1.70 (1.13) **	-0.008 (0.111)
White Male								
>12 years of education	2.23 (0.41) **	-0.039 (0.022) †	2.68 (0.60) **	-0.080 (0.033) *	0.91 (0.55)	0.048 (0.033)	1.27 (0.78)	-0.110 (0.067) †
12 years of education	3.33 (0.50) ***	-0.013 (0.027)	2.99 (0.68) ***	-0.044 (0.037)	-0.08 (0.56)	0.028 (0.034)	0.93 (0.75)	-0.112 (0.071) †
<12 years of education	2.92 (0.76) **	-0.015 (0.044)	3.31 (0.80) **	-0.049 (0.045)	1.64 (0.53) *	-0.005 (0.034)	1.16 (0.61) †	-0.007 (0.055)
Attrition (ref=None)								
Death	-0.22 (0.77)	0.026 (0.088)	0.27 (0.62)	-0.031 (0.066)	-0.66 (0.29)	-0.094 (0.024) **	-0.48 (0.40)	-0.092 (0.031) **
Non-Response	-0.56 (0.32)	-0.016 (0.023)	-1.12 (0.43) *	-0.021 (0.031)	-0.73 (0.33) *	0.000 (0.025)	-0.33 (0.40)	0.060 (0.039)
Variance Components								
Level 1: Within-person	2.99		3.35		2.33		2.44	
Level 2: Between-person	18.39 (0.00) ***	0.027 (0.00) ***	20.06 (0.00) ***	0.029 (0.00) ***	18.44 (0.00) ***	0.031 (0.00) ***	17.83 (0.00) ***	0.041 (0.00) ***
Unweighted N	*1011*		*625*		*1098*		*692*	

†p<.10; *p<.05; **p<.01; ***p<.001 (two-tailed tests)

Note: Regression coefficients with standard errors in parentheses are shown. Standard errors are claculated using robust estimation.

[a] Constant represents a white female with more than 12 years of education.

Table 2.5. Age Stratified Models of Race/Gender by Income Differences in Inital BMI and Annual BMI Growth.

	25-39		40-54		55-69		70-84	
	Initial	Annual Growth	Initial	Annual Growth	Initial	Annual Growth	Initial	Annual Growth
Constant [a]	23.23 (0.33) ***	0.187 (0.017) ***	25.04 (0.39) ***	0.170 (0.021) ***	25.85 (0.39) ***	-0.002 (0.022) *	23.45 (0.80) ***	-0.087 (0.051) †
White Female								
Income $30,000+								
Income $10,000-29,999	0.96 (0.47)	0.012 (0.025)	1.40 (0.62) †	-0.015 (0.034)	0.40 (0.47)	-0.013 (0.028)	2.45 (0.85) **	-0.062 (0.056)
Income < $10,000	1.81 (0.71) †	-0.014 (0.040)	0.60 (0.85)	-0.009 (0.050)	1.39 (0.55) **	-0.037 (0.034)	2.22 (0.86) **	-0.022 (0.059)
Black Female								
Income $30,000+	3.12 (1.08) ***	-0.006 (0.064)	2.56 (1.28) **	-0.011 (0.074)	2.97 (2.24) †	-0.058 (0.131)	4.49 (6.64)	-0.025 (0.402)
Income $10,000-29,999	2.14 (0.91) **	0.152 (0.053) **	4.40 (1.17) ***	-0.070 (0.066)	4.55 (1.10) ***	-0.023 (0.075)	4.09 (1.70) **	0.119 (0.141)
Income < $10,000	4.44 (0.93) ***	0.166 (0.056) **	3.77 (1.33) **	-0.006 (0.089)	4.51 (0.81) ***	-0.131 (0.053) *	4.17 (1.10) ***	-0.094 (0.084) **
Black Male								
Income $30,000+	2.70 (1.07) ***	0.045 (0.061)	2.58 (1.22) ***	-0.045 (0.080)	2.79 (1.33) *	-0.062 (0.083)	2.19 (3.90)	-0.136 (0.503)
Income $10,000-29,999	3.43 (0.92) ***	0.022 (0.063)	2.85 (1.14) **	-0.120 (0.073) †	1.91 (1.23) *	-0.030 (0.083)	1.92 (2.00)	0.137 (0.173)
Income < $10,000	0.99 (1.45)	0.045 (0.087)	-2.52 (2.12) ***	-0.098 (0.142) **	-0.45 (1.19)	0.073 (0.096)	3.03 (1.37) **	0.014 (0.127)
White Male								
Income $30,000+	2.90 (0.44) ***	-0.061 (0.023) *	2.63 (0.55) ***	-0.063 (0.030) †	0.28 (0.52)	-0.016 (0.031)	1.46 (1.12)	-0.032 (0.081)
Income $10,000-29,999	2.33 (0.47) ***	0.001 (0.025)	2.32 (0.67) **	-0.048 (0.037)	1.08 (0.49)	-0.011 (0.030)	2.82 (0.89) **	-0.109 (0.068)
Income < $10,000	3.12 (0.77) **	0.003 (0.046)	2.43 (1.06) *	-0.177 (0.061) †	-0.56 (0.70)	0.045 (0.051)	2.04 (0.96)	0.076 (0.076)
Attrition (ref=None)								
Death	-0.26 (0.79)	0.014 (0.088)	0.40 (0.63)	-0.035 (0.067)	-0.48 (0.30)	-0.104 (0.024) **	-0.54 (0.41)	-0.538 (0.412)
Non-Response	-0.58 (0.32)	-0.020 (0.022)	-0.92 (0.44) *	-0.025 (0.031)	-0.37 (0.33)	-0.020 (0.025)	-0.34 (0.40)	-0.340 (0.398)
Variance Components								
Level 1: Within-person	2.98		3.39		2.33		2.60	
Level 2: Between-person	18.53 (0.00) ***	0.027 (0.00) ***	20.40 (0.00) ***	0.029 (0.00) ***	18.83 (0.00) ***	0.034 (0.00) ***	17.71 (0.00) ***	0.037 (0.00) ***
Unweighted N	*1011*		*625*		*1098*		*692*	

†p<.10; *p<.05; **p<.01; ***p<.001 (two-tailed tests)

Note: Regression coefficients with standard errors in parentheses are shown. Standard errors are claculated using robust estimation.

[a] Constant represents a white female with a houshold income more than 30k.

APPENDICES

Appendix 2.A. Wave-Specific Sample Sizes by Race/Gender, Age, and Education

	White Women			Black Women			Black Men			White Men		
	<HS	HS	Coll+	<HS	HS	Coll+	<HS	HS	Coll+	<HS	HS	Coll+
Wave 1												
Age												
25-39	48	117	165	56	94	93	24	31	69	31	91	192
40-54	47	82	99	65	45	37	42	28	20	31	50	79
55-69	166	188	128	152	46	26	83	16	18	106	77	92
70-84	165	92	88	115	17	17	49	4	6	75	37	27
Wave 2												
Age												
25-39	38	98	147	44	69	67	19	16	52	16	75	167
40-54	38	76	89	54	37	29	30	20	15	22	43	70
55-69	127	168	117	122	36	23	58	13	12	81	68	82
70-84	127	77	70	83	10	13	39	1	3	46	20	20
Wave 3												
Age												
25-39	37	98	148	41	58	69	15	18	48	19	79	167
40-54	39	71	84	50	32	30	25	17	13	26	45	72
55-69	118	152	107	97	29	20	47	9	12	69	56	76
70-84	95	61	67	60	8	12	17	2	3	26	15	15
Wave 4												
Age												
25-39	34	90	138	24	47	52	9	14	36	19	72	149
40-54	29	66	80	31	26	26	16	14	11	20	36	60
55-69	74	119	82	52	14	15	20	6	8	27	44	49
70-84	28	32	29	15	1	3	5	0	0	8	4	3

Note: Sample sizes are unweighted.

Appendix 2.B. Wave-Specific Sample Sizes by Race/Gender, Age, and Income

	White Women			Black Women			Black Men			White Men		
	<$10k	10-30k	30k+	<$10k	10-30k	30k+	<$10k	10-30k	30k+	<$10k	10-30k	30k+
Wave 1												
Age												
25-39	51	146	133	107	90	46	25	65	34	31	136	147
40-54	37	79	112	58	57	32	14	45	31	19	49	92
55-69	134	229	119	148	62	14	52	39	26	45	126	104
70-84	163	153	29	120	27	2	42	14	3	43	80	16
Wave 2												
Age												
25-39	44	119	120	82	65	33	22	36	29	18	114	126
40-54	29	70	104	47	46	27	10	31	24	14	39	82
55-69	107	198	107	114	54	13	36	32	15	32	107	92
70-84	130	118	26	86	18	2	32	11	0	24	51	11
Wave 3												
Age												
25-39	40	120	123	70	64	34	16	36	29	21	114	130
40-54	31	64	99	41	41	30	8	28	19	16	41	86
55-69	94	180	103	90	44	12	25	25	18	22	96	83
70-84	92	104	27	63	15	2	12	9	1	14	36	6
Wave 4												
Age												
25-39	31	115	116	56	44	23	13	25	21	16	104	120
40-54	22	60	93	22	39	22	4	19	18	10	36	70
55-69	55	137	83	49	25	7	10	13	11	9	55	56
70-84	27	46	16	14	4	1	3	2	0	5	4	6

Note: Sample sizes are unweighted.

CHAPTER III

A LIFE COURSE APPROACH TO UNDERSTANDING THE RELATIONSHIP BETWEEN SOCIOECONOMIC POSITION AND ADULT BMI TRAJECTORIES

Research on the relationship between socioeconomic position (SEP) and adult obesity in the United States has primarily focused on adult SEP. Prior studies report inverse associations between adult SEP - measured using education and income - and body mass index (BMI) among white, African American, and Mexican American women (Chang and Lauderdale 2005; Croft et al. 1992; Kahn, Sobal and Martorell 1997; Pawson, Martorell and Mendoza 1991). However, early-life SEP may also be an important determinant of adult BMI. Adult socioeconomic attainment is heavily influenced by family background characteristics such as parental occupation and education (Blau and Duncan 1987; Sewell, Haller, and Portes 1969). Thus early-life SEP likely influences adult BMI, if not directly, then at least indirectly via adult socioeconomic attainment processes.

Early-life socioeconomic conditions may be instrumental in shaping later life inequality in a number of health outcomes, including obesity. National data show large racial and ethnic differences in obesity among women with much higher obesity prevalence found among African American and Mexican American Women compared to

white women; very little racial and ethnic variation is found among men (Flegal et al. 2002; Ogden et al. 2006). Racial and ethnic disparities in obesity may reflect differential access to socioeconomic resources as well as differential exposure to disadvantage over the life course (Williams and Collins 1995).

Increasingly, researchers are utilizing a life-course approach to identifying and understanding the determinants of adult health (Alwin and Wray 2005). There is growing evidence that the origins of poor adult health, and by extension adult health inequality, may lie in experiences earlier in life (Kuh and Ben Shlomo 2004). Prior studies have reported associations between early-life SEP and adult obesity risk (Baltrus et al. 2005; Greenlund et al. 1996; Langenberg et al. 2003; Power, Manor, and Davey Smith 2003; Power et al. 2005), but the processes through which early-life socioeconomic conditions affect obesity risk in later life are not articulated in the existing research. Furthermore, little is known about how early-life SEP matters for understanding the large racial and ethnic differences in obesity observed among U.S. women.

This paper seeks to better understand the processes linking early-life SEP to adult obesity by examining the association between life course SEP and individual BMI trajectories among women over 15 years of their adult life course. I focus specifically on how these processes operate among women because obesity rates are much higher among women compared to men (Ogden et. al. 2006) and because prior studies show associations between measures of SEP and obesity among women, while weaker or no associations are found for men (Chang and Lauderdale 2005; Croft et al. 1992; Zhang and Wang 2004). Focusing on women also allows me to examine the extent to which

early-life and adult socioeconomic conditions explain racial and ethnic variation in adult BMI trajectories, which is greater among women.

Life Course Approach to Adult Health

The life course as a theoretical orientation emerged in the 1960s out of a need to understand how variability in human development from birth through adulthood is related to personal biography within socio-historical contexts. The life course perspective on individual development takes the "long view" and emphasizes the importance of early life experiences and opportunity structures in determining later life outcomes (Elder 1994). Individual lives are organized according to a range of social processes that unfold over the life course (Dannefer 1987). The life course perspective has been synthesized in a range of theoretical and empirical studies that seek to understand the links between experiences and outcomes within the individual life course, including research on the implications of historical change for determining cohort experiences (Ryder 1965), cohort variability of aging patterns (Riley 1987), effects of early life conditions on adult development (Clausen 1991), age and cohort variation in stress processes (Pearlin and Skaff 1996), cohort and intergenerational linkages to family development (Bengston and Allen 1993; Rossi and Rossi 1990), and stability and change in criminal careers (Sampson and Laub 1992; Uggen and Massoglia 2004).

The life-course perspective is also increasingly being applied in studies of the determinants of adult chronic disease. Adult health is the long-term outcome of the "…physical and social exposures [experienced] during gestation, childhood, adolescence, young adulthood, and later life" (Kuh and Ben-Shlomo 2004: pg. 5). A growing body of

research, for instance, finds that exposure to early life socioeconomic disadvantage is associated with poor health behaviors and worse health in adulthood. Prior studies have found that adults from a low socioeconomic position in early-life are more likely to engage in risky health behaviors such as smoking, drinking, poor diet, and physical inactivity (Lynch, Kaplan, and Salonen 1997; Power et al. 2005). Socioeconomic disadvantage in early life is also associated with an increased risk of cardiovascular disease (Lawlor, Smith, and Ebrahim 2004; Ramsey et al. 2007; Smith and Hart 2002; Wamala, Lynch and Kaplan 2001), Type II diabetes (Lidfeldt et al. 2007), and cancer (de Kok et al. 2007), poorer adult cognitive and psychosocial functioning (Harper et al. 2002; Kaplan et al. 2001), and higher mortality (Beebe-Dimmer et al. 2004; Hart 1998; Hayward and Gorman 2004). In addition, several studies have found that the accumulation of socioeconomic disadvantage over the life course is associated with worse adult health (Ljung and Hallqvist 2006; Power, Manor, and Matthews 1999; Singh-Manoux et al. 2004; Wamala, Lynch, and Kaplan 2001). Among the studies that reported the effects of early-life adjusted for adult SEP, the general finding is that the effects of early-life are often substantially diminished with the inclusion of adult SEP, suggesting that much of their effects are mediated through later SEP. Nevertheless, early-life indicators of SEP often remain significant predictors of adult health (Beebe-Dimmer et al. 2004; de Kok et al. 2007; Harper et al. 2002; Hayward and Gorman 2004; Ramsey et al. 2007; Wamala, Lynch, and Kaplan 2001).

Prior studies that have examined the relationship between early-life SEP and adult obesity risk among women have found significant effects of early-life SEP. For instance, several studies from the UK and Europe found a higher risk of adult obesity among those

with manual social class origins, even after adjusting for adult social class (Langenberg et al. 2003; Power, Manor, and Davey Smith 2003; Power et al. 2005). In a randomly selected multi-community sample of U.S. black and white adults aged 18-30 years at baseline, Greenlund et al. (1996) observed a negative effect of father's education, adjusted for participant's education, on baseline BMI and seven-year BMI change, but only among white women; they reported finding a significant effect of father's education among black women only in models unadjusted for adult education. Power et al. (2005) examined data from a 1965 community study of U.S. adults and found that women aged 30-50 whose fathers held manual occupations had an elevated risk of obesity in adulthood, even after adjusting for adult occupation, though effects of early life were not statistically significant. Using the same data source, Baltrus et al. (2005) examined racial differences in weight trajectories over 34 years among adults aged 17 to 40 years in 1965, and found that adjustment for childhood socioeconomic position - defined using father's occupation or education- reduced the black-white difference in baseline weight and accounted for the race gap in weight gain. However, the coefficients for early-life and adult predictors were not reported, so it is unclear what the nature of the relationship is between these predictors and weight trajectories and whether early-life effects remained after adjusting for adult socioeconomic characteristics.

Several studies report associations between early-life SEP and adult obesity risk in the U.S., but these studies are limited by the use of community-based samples, the absence of Hispanic individuals in the data, and study periods that are not current, thus making it difficult to generalize from these studies to the U.S. population. It is important to determine if the associations found between early-life socioeconomic conditions and

adult weight and BMI hold in a representative sample of U.S. adults, and to include other minority groups such as Hispanics, to better understand the biopsychosocial processes through which early-life has its effects on outcomes in later-life.

The empirical evidence suggests that early-life SEP matters for adult obesity, but the extent to which the effects of early-life SEP are direct or indirect remains poorly specified, along with the pathways involved in both kinds of effects. Thus, although the extant research on life course determinants of adult health provides evidence for the significance of early life experiences in understanding later life health outcomes, life course studies would be enriched by paying more attention to the theoretical processes by which life-course experiences accumulate over time to influence outcomes in later life.

Models of Life Course Processes

"Critical period" models and "accumulation of risk" models are the major theories of life-course processes that have been developed and applied in the epidemiologic literature (Ben-Shlomo and Kuh et al. 2002, Lynch and Davey Smith 2005). Critical period models postulate that exposures during specific periods of the life course have a long-term impact on the development of adult disease. These models, also referred to as latency models (Hertzman et al. 2001), emphasize long term programming in early life and are the basis for Barker's (1990) 'fetal origins of adult disease' hypothesis. Accumulation of risk models focus on the accumulation of exposures over the life course and the resulting cumulative effects on health. Accumulation of risk models should further be specified as additive or multiplicative models of cumulative exposure to disadvantage. In additive models, the number, duration, and severity of exposures to

disadvantage are expected to affect health in a purely additive relationship. In multiplicative models, on the other hand, exposures in early life interact with exposures later in life to produce steeper cumulative disadvantage to health than would be produced by either alone.

I focus on three life course pathways that link early-life socioeconomic exposures to later-life outcomes, shown in Figure 1. In the pathway model (a), differential exposure to early-life circumstances may initiate "chains of risk" where the experience of disadvantage at one point in the life course produces further disadvantage later in the life course, resulting ultimately in health inequalities. According to this model, early-life SEP affects health in later-life through its association with adult SEP.

The latency model (b) is a variant of the pathway model that allows for effects of early-life exposure that are independent of intervening experiences. Thus, early-life SEP has an independent direct effect on adult health, even if some of the association operates via adult SEP. Socioeconomic conditions in early-life may initiate developmental health trajectories that are well established before adulthood by increasing the risk of exposure to stressors and harms, for instance, that cause health declines in later-life.

I refer to the final conceptual pathway as the cumulative disadvantage model (c). In the pathway and latency models, disadvantage in early life combines with disadvantage in later life in an additive fashion, whereas in the cumulative disadvantage model, early- and later-life disadvantage combine in a multiplicative fashion. The interaction in this model is shown in Figure 1 via the broken lines from SEP during one period of the life course intersecting the relationship between SEP in another period of the life course and the outcome.

[FIGURE 3.1 ABOUT HERE]

Early-Life Disadvantage as an Inequality Generating Process

These theoretical models of life course processes can be used to understand the effects of early-life experiences on the level of health at a given point in the adult life course, but also on the *development or trajectory of health* throughout adulthood. For instance, the outcome Y in Figure 1 can represent BMI at some point in time and it can also represent change in BMI over time. Prior studies of life course determinants of BMI are generally limited to assessments of the effects of early-life on BMI at one point in the adult life course. Only Baltrus et al. (2005) considered the effects of early-life on BMI change over time. Early-life experiences may generate inequality in health as it develops over the life course.

Early-life shapes access to health-related resources and opportunities, creating a situation in which disadvantaged groups are exposed to health-compromising conditions across the life course with little opportunity to improve their health (Wadsworth 1997; Williams and Collins 1995). This may be particularly relevant in the case of weight gain because early-life inequality may place disadvantaged individuals on a trajectory of weight gain that unfolds throughout adulthood. Early experiences of socioeconomic disadvantage have been shown to be important determinants of health at a given point in adulthood. However, early experiences may also influence trajectories of health decline such that individuals who experience disadvantage also experience steeper declines in health. Thus, in addition to finding a relationship between early-life SEP and adult health

status at a given point in adulthood, we would further expect early-life SEP to influence health as it develops throughout adulthood. However, the concept of early-life factors generating adult health inequality has not been examined in the literature on life course approaches to adult health.

The idea that the effects of disadvantage accumulate over time has roots in Merton's (1968) work on the "Matthew effect in science" in which Merton argued that early career achievement predicted subsequent achievements, resulting a widening of the gap between the "haves" and the "have-nots." The theory of cumulative disadvantage thus emerged as a framework for understanding inequality-generating processes as they unfold over time. The intersection of theories of cumulative disadvantage and life course provide an ideal framework for studying the emergence of health inequality and inter-individual divergence in health trajectories (Dannefer 2003; O'Rand 1996). This framework has been applied in research demonstrating that health inequality observed in old age is developed throughout adulthood (House, Lantz, and Herd 2005; Ross and Wu 1996). For instance, Wilson, Shuey, and Elder (2007) explored cumulative effects of socioeconomic advantage as a mechanism for explaining disparities in health trajectories. They found that individuals with more educational attainment and greater wealth experienced better health over the adult life course. Similarly, Mirowsky and Ross (2008) found a divergence in health by level of education as individuals aged.

For the most part, cumulative disadvantage has been used to understand inter-individual divergence in health by adult socioeconomic position. There are fewer studies, however, that have used cumulative disadvantage as a framework for explaining racial and ethnic differences in health trajectories, and no studies, to my knowledge, that have

explored early-life SEP as an explanation for divergence in adult health trajectories. The experience of adverse life events, which is socially patterned by race and ethnicity, may have enduring effects over the life course thus accounting for racial and ethnic differences in health trajectories. It is possible, therefore, that the large racial/ethnic differences in obesity among adult women are well established prior to adulthood.

Current Study

The primary aim of this study is to determine the pathways through which early-life SEP influences adult BMI trajectories over 15 years of the adult life course. This study seeks to answer the following questions: (1) Does early-life SEP influence adult BMI trajectories primarily through adult SEP, as described in the pathway model, (2) Does early-life SEP influence BMI independent of adult characteristics through latency effects, and (3) Is there a cumulative effect of socioeconomic disadvantage on adult BMI trajectories? Furthermore, if socioeconomic disadvantage accumulates over the life course to influence BMI trajectories, (4) is the combined effect additive or multiplicative? A secondary aim of this study is to determine the extent to which life-course SEP accounts for the large racial and ethnic disparities in BMI trajectories among U.S. women.

While prior studies of life course determinants of adult obesity have typically examined the outcome of interest at only one point in adulthood, this study examines the effect of life course SEP on within-individual adult weight trajectories over 15 years at different points in the life course. The BMI trajectory is composed of baseline BMI and BMI change. Thus I am able to examine the effects of life-course SEP on BMI at the

beginning of the study period as well as in divergence in BMI over time (change in the trajectory). This is the first study to my knowledge that examines the effects of life course SEP on adult obesity using a nationally representative survey of U.S. adults.

DATA AND METHODS

Data

This study uses longitudinal data spanning 15 years of adulthood and retrospective data on early life socioeconomic experiences from the Americans' Changing Lives (ACL). The ACL is a stratified, multi-stage area probability sample of 3,617 non-institutionalized adults 25 years and older living in the United States in 1986, with oversampling of adults aged 60 and older and African Americans. The baseline response rate was 68% for sampled individuals and 70% for sampled households. Follow up interviews were conducted in 1989 with 2,867 respondents, 1994 with 2,562 respondents or their proxies (n = 164), and 2001/2002 with 1,787 respondents or their proxies (n = 95), representing 83%, 83%, and 74% of surviving respondents respectively. The analysis is restricted to female respondents who make up between 62% and 64% of the sample at each wave. Because questions about early-life experiences were asked only during the Wave 2 interview, 429 women who were not present for the Wave 2 interview were excluded from the analytic sample. Excluding these women from the analyses did not change the substantive findings. The final analytic sample consists of 1,809 women aged 25-84 in 1986.

Wave-specific sample weights are applied in all analyses. The wave 1 sample weight includes an adjustment for the differential probability of selection at baseline and

non-response, and a post stratification adjustment to the 1986 age/race/sex/region specific Census estimates of the U.S. population. Sample weights for waves 2, 3 and 4 adjust for panel non-response and attrition. In addition to the weight adjustment, I employ three strategies to reduce the likelihood that multivariate results are affected by non-random attrition over the course of the study. First, all models are estimated using maximum likelihood (ML) estimation via the expectation maximization (EM) algorithm, which corrects for bias related to sample attrition (Feng et al 2006; Little and Rubin 2002). The models also include covariates that are associated both with BMI and the likelihood of attrition, thus further correcting for bias due to attrition (Cnaan, Laird, and Slasor 1997; Collins, Schafer, and Kam 2001; McArdle and Hamagami 1992). Finally, I include controls for attrition due to death (respondents who died between 1986 and 2001) in all models to control for any residual effect of subsequent death on BMI. I also estimated models that further controlled for non-response but the results were unchanged.

Measures

Body Mass Index

Self-reported height and weight were used to determine respondents' body mass index (BMI), calculated as squared height in meters divided by weight in kilograms. Self-reports of height and weight are considered to be reliable estimates of BMI, though they are subject to reporting error, with women tending to underreport their weight (Bolton-Smith et al. 2000; Nawaz et al. 2001; Palta et al. 1982; Willett, Dietz and Colditz, 1999). Respondents were asked about their height in the baseline interview and were asked about their weight in every wave of data collection for which they were interviewed. BMI

values were imputed for cases missing on height or weight (2.4%, 1.9%, 0.98%, and 2.2% of the W1, W2, W3, and W4 samples, respectively) using sex-specific prediction equations that accounted for respondent's age, race, and, when available, prior height and weight.

Early-Life Socioeconomic Position

Respondents were asked to report retrospectively on their early-life experiences up until the age of 16, including whether the respondent lived with both parents, the highest level of education attained by the respondent's parent(s), the respondent's relative financial situation, and whether the respondent had to work before the age of 16 to help out the family financially. These early-life socioeconomic factors – parental education, family structure, and financial situation – shape the life chances of young people, differentiating between individuals who grew up with privilege and opportunities and those who did not (Furstenberg 2003). In addition, prior work shows that children whose parents had lower educational attainment and who are from single-parent and low-income households are more likely to be overweight and obese (Kimm et al. 2006; Miech et al. 2006; Strauss and Knight 1999). Thus, these early-life measures reflect conditions that both promote childhood obesity and that determine socioeconomic achievement in later-life.

Respondents were also asked to report on the number of years of school completed by their mother and father. A measure of parental education was created by taking the highest number of years of school completed by either parent and categorizing into 0 to 11 years of education, 12 years of education, and 13 to 17 years of education,

with the highest education category treated as the reference. I use a combined parental education measure because it reflects the highest amount of educational capital available to the respondent when growing up. In addition, analysis using only mother's or only father's education or including both mother's and father's education produced substantively similar results to those using the combined parental education measure. Intact (natural) family is measured with the question, "Up to the age of 16 did you live with both your natural parents?" and is coded so that yes = 0 and no = 1. Early-life relative financial situation was assessed with the following question, "Compared with the average family in your community at the time you were growing up, was your family better off financially, about average, or worse off?" Response categories were coded such that better than average = 1, average = 2, and worse than average = 3, with better than average treated as the reference. Finally, I include an indicator of whether the respondent worked before the age of 16 to help support the family, where a response of yes = 1 and no = 0.

There was a significant amount of missing data on early-life measures, particularly parent's education, so I imputed values for these variables to retain the maximum number of cases for multivariate analysis. I used Stata's hotdeck procedure[4] (Mander and Clayton 2000) to impute missing values for intact family (n = 8), mother's education (n = 355), father's education (n = 498), financial situation (n = 3), and worked before 16 (n = 7).

[4] The hotdeck procedure imputes missing data for a variable X by using the approximate Bayesian bootstrap to first draw n values at random with replacement from the observed values of X and then draw another n values with replacement from the first draw of n values to use as imputed values. Furthermore, I specified that missing values for X be imputed by sampling from the observed values of X of matched subjects, who are matched using covariate patterns constructed from other early life predictors, age group, and race. Because imputed values are obtained from other observed values in the sample, an advantage of hotdecking is that the imputed values will be valid values.

Adult Socioeconomic Position

I include the respondent's level of education in 1986 and wave-specific measures of family income and financial difficulty to measure adult SEP. Years of educational attainment is categorized to indicate 0 to 11 years of schooling, 12 years of schooling, and 13 to 17 years of schooling (the reference). Family income and difficulty paying bills are used to measure the respondent's economic situation at each wave. Family income, defined as the total pre-tax annual income of the respondent and his/her spouse/partner, is measured with three dummy variables representing low, middle, and high tertiles of income, where the highest tertile is treated as the reference. Difficulty paying bills is based on a question asking "How difficult it is to meet the monthly payments on family bills? Is it extremely difficult, very difficult, somewhat difficult, slightly difficult, or not difficult at all?" and is measured using dummy variables indicating extremely or very difficult (these two categories were collapsed to increase cell size), somewhat difficult, slightly difficult, or not difficult at all, with the last category treated as the reference.

Accumulation of Socioeconomic Disadvantage

In addition to examining the effect of individual measures of socioeconomic conditions across the life course, I want to determine if there is a cumulative effect of socioeconomic disadvantage on BMI trajectories. I constructed summary scores of accumulated disadvantage in early-life and later-life, where higher scores represent more accumulated disadvantage. These scores were derived using the measures described above, which have all been coded so that the most disadvantaged state is equal to 1 and the least disadvantaged state is equal to 0. In the case of measures with three categories, I

assigned a value of 0.5 to the middle category to represent an intermediary level of disadvantage. The early-life accumulated disadvantage score was derived by summing the values across the following measures: non-intact family (yes = 0, no = 1), parental education (more than 12 years = 0, 12 years = 0.5, less than 12 years = 1), relative financial situation (better off = 0, average = 0.5, worse off = 1), and worked before the age of 16 (no = 0, yes = 1). Early-life accumulated disadvantage scores range from 0-4; the mean is 1.6 (std. dev. 0.88). The later-life accumulated disadvantage score was created using measures of socioeconomic position at baseline(1986): respondent educational attainment (more than 12 years = 0, 12 years = 0.5, less than 12 years = 1), family income in 1986 (highest tertile = 0, middle tertile = 0.5, lowest tertile = 1), and difficulty paying bills in 1986 (not at all difficult = 0, somewhat/slightly difficult = 0.5, very/extremely difficult = 1). Later-life accumulated disadvantage scores range from 0-3; the mean is 1.59 (std. dev. 0.91).

Additional Covariates

Race/ethnicity is measured using dummy variables for non-Hispanic white (the reference category), non-Hispanic black, and Hispanic. I will hereafter refer to these race/ethnic categories as white, black, and Hispanic for brevity. Prior studies show that the shape of BMI trajectories varies with age. Weight is highest among middle aged adults and relatively lower among younger and older adults, indicating that adults gain weight through early and middle adulthood but that this weight gain slows with increasing age. (Flegal et al 2002; Kuczmarski et al 1994; Reynolds and Himes 2007). I control for age differences in baseline BMI by including linear and quadratic age terms,

deviated from 25 years - the youngest age at baseline - to make the intercept more meaningful. Although I include a quadratic effect of age on baseline BMI to capture the U-shaped relationship between BMI and age, I only include a linear effect of age on BMI change because weight changes linearly with age. I also include an interaction between black and age for BMI growth because the black/white difference in growth rates varies with age (see Chapter II).

I adjust for wave-specific family structure with controls for marital status and the presence of children in the household. Prior work shows that married individuals are more likely to be obese and gain more weight over time, compared to individuals who are not married, (Chou et al. 2002; Jeffrey and Rick 2002) and that parity is associated with higher body weight and weight gain (Lahmann et al. 2000). Marital status is measured using dummy variables indicating currently married (the reference), divorced/separated or widowed, and individuals who have never married. I do not have information on parity so I use an approximate indicator, presence of children under the age of 18 in the household coded so that no = 0 and yes = 1.

Prior studies have shown associations of smoking status and physical activity with body weight, so I also include adjustments for wave-specific smoking and activity. Individuals who smoke and those who engage in physical activity generally weigh less. Smoking status is measured using dummy variables indicating if the respondent is a current smoker (reference), a former smoker, or has never smoked. Physical activity is derived from three questions about how often the respondent "typically works in the garden or yard," "takes walks," and "engage(s) in active sports or exercise." Response categories are: often = 4, sometimes = 3, rarely = 2, or never = 1. An index of physical

activity was created by first reverse coding the items then summing across the three items. Indices created using measures in 1989, 1994, and 2001/2002 were then standardized using the mean and standard deviation of the 1986 index. The index ranges from the lowest level of physical activity to the highest level of physical activity.

Analytic Strategy

I first examine the distribution of the variables used in the analysis in the full sample and by race/ethnic group. In addition, I examine the bivariate correlations between all measures of socioeconomic conditions to determine the likelihood of encountering multi-collinearity. I then estimate individual linear growth models to determine the effect of life course SEP on both baseline BMI and BMI change over time. Time is measured with a linear term representing annual growth where the first wave of data collection (1986) is treated as *time* = 0 and each follow-up observation is measured in terms of the number of years elapsed since the baseline interview, approximately 2.6 (range: 2.2-2.9), 7.7 (range: 7.2-8.2), and 15.5 (range: 14.9-16.6) years for wave 2, wave 3, and wave 4, respectively. The individual growth model is specified as follows:

(1) $Y_{it} = \beta_{0i} + \beta_{1i}(TIME_{ij}) + \varepsilon_{it}$; $\varepsilon_{ij} \sim N(0, \sigma_{\varepsilon}^2)$,

where Y_{it} is the BMI for person *i* at time *t*, β_{0i} is the term for baseline BMI, β_{1j} is the term for change in BMI from baseline until time *t*, and ε_{ij} is a normally distributed random effect or error variance with mean 0 and variance σ_{ε}^2. Both baseline BMI(β_{0j}) and BMI change (β_{1j}) are fixed effects coefficients that are further modeled as functions of baseline covariates as follows:

(2) $\beta_{0i} = \gamma_{00} + \gamma_{01}B + \gamma_{02}H + \gamma_{03}Z_1 + \ldots + \gamma_{0k}Z_k + u_{0i}$

(3) $\beta_{1i} = \gamma_{10} + \gamma_{11}B + \gamma_{12}H + \gamma_{13}Z_1 + \ldots + \gamma_{1k}Z_k + u_{1i}$

where B is the term for the black/white difference, H is the term for the Hispanic/white difference, $Z_{1\text{-}k}$ are covariates, and u_{0j} and u_{1j} represent the random effects variances in the models. All variables are included in each model as predictors of both baseline BMI and BMI change (the interaction with time). Analyses are conducted using the HLM software Version 6.0 (Raudenbush and Bryk 2002).

RESULTS

Means and standard deviations or percentages for wave-specific BMI, age in 1986, and early-life socioeconomic characteristics are presented in Table 1 for the full sample and separately for whites, blacks, and Hispanics. BMI increased over time in all groups and at each wave black women had higher BMI compared to white women. Parental educational attainment was lower among black and Hispanic women compared to white women. In addition, black and Hispanic women were more likely to grow up in a single-parent household and to work before age 16. Compared to white women, Hispanic women were more likely to report a worse relative financial situation growing up and less likely to report a better relative financial situation growing up. Black women, on the other hand, were less likely to report a worse relative financial situation compared to white women and more likely to report a better financial situation. This may reflect the comparative nature of the measure, which is based on an assessment of one's early-life financial situation compared to others from the same community. The other early-life measures indicate that black women were more likely to have grown up in a disadvantaged environment compared to white women, but they may not have been

worse off financially compared to the other individuals growing up in the same disadvantaged communities.

[TABLE 3.1 ABOUT HERE]

Summary statistics for later-life socioeconomic characteristics, family structure, and health behaviors by survey year are presented in Table 3.2. The majority of the sample had completed at least 12 years of education by the time of the baseline interview in 1986. The distribution of income and difficulty paying bills is similar across waves, though the overall financial situation of the sample improved. The majority of the sample was married at each wave and the proportion of children in the household under age 18 decreased over time from 42% in 1986 to 24% in 2001. In 1986, 78% of sample members were current or former smokers, decreasing to 64% by 2001. On average, individuals rarely or sometimes engaged in physical activity with little variation across waves.

[TABLE 3.2 ABOUT HERE]

Measures of socioeconomic position are likely correlated with each other, both within a given point in the life course (early-life or later-life) and across the life course, potentially introducing multi-collinearity into the models. Thus, I examined bivariate correlations of measures of socioeconomic position in early-life and later-life. I use adult socioeconomic measures from 1986; the pattern of correlations did not vary if I used measures from other years. Table 3.3 shows that the highest correlation among early-life

measures is between the measure of relative financial situation and working before age 16 (r = 0.25). The highest correlation in later-life is between educational attainment and income (r = 0.48). The highest correlation between early-life and later-life measures is found for parental and respondent education (r = 0.43), as would be expected. Overall, correlations among measures from the same point in the life course were generally low, suggesting that each measure largely represents a distinct aspect of socioeconomic position. Low correlations also suggest the potential for modest collinearity at most.

[TABLE 3.3 ABOUT HERE]

Pathway and Latency Effects

I first determine whether early-life SEP influences adult BMI trajectories. I then evaluate the extent to which pathway and latency models explain the process through which early-life SEP affects adult BMI. Result from growth curve models predicting baseline BMI and BMI change are presented in Table 3.4. Model 1 shows the race/ethnic disparities in baseline BMI and BMI over time, adjusting only for age and mortality. Baseline BMI was 2.92 points higher for blacks and 1.30 points higher for Hispanics, compared to whites. White women experienced an annual increase of 0.253 BMI points. Black women experienced a significantly higher annual increase in BMI (0.151) compared to white women, but this difference decreased with age (-0.006). There were no statistically significant differences in BMI change between white and Hispanic women.

In Model 2 I add measures of early-life SEP. Individuals whose parental educational attainment was less than 12 years were 1.29 ($p<.01$) points higher on baseline BMI. Living in a single-parent household was not related to baseline BMI. Having worse financial situation growing up is negatively associated with baseline BMI: individuals growing up in worse financial situations were 0.99 ($p<.10$) points lower on baseline BMI. Individuals who worked to support their family were 0.98 ($p<.05$) higher on baseline BMI. The black/white gap in baseline BMI was reduced by about 15% after accounting for early-life SEP. With the inclusion of early-life SEP, there are no statistically significant differences between Hispanic and white women. In terms of BMI growth over time, coming from a worse relative financial situation is associated with an additional annual increase of 0.70 ($p<.05$) points. The black/white difference in BMI growth is not reduced with the inclusion of early-life measures. In analyses not reported here, I also tried stepping variables in and out one variable at a time and found that effects of early-life measures were similar to those reported in the model including all variables (Model 2).

Model 3 shows the effect of further adjusting for adult SEP, where education was measured only in 1986 and the income and difficulty paying bills variables were measured at each wave and treated as time-varying covariates. Individuals with less than 12 years of education were 1.93 ($p<.001$) points higher on baseline BMI. BMI trajectories did not vary according to income and difficulty paying bills. With the inclusion of adult SEP, the effect of parental education and working before age 16 was reduced. Although reduced, early-life effects remain, suggesting that early-life SEP is associated with adult BMI independent of adult SEP, and thus operates through a latency process. An

examination of the effect of individual adult SEP measures showed that early-life effects were also partially mediated by adult educational attainment. For instance, the association of parental education with baseline BMI is partially mediated by the respondent's own educational attainment, suggesting that early-life SEP operates via adult SEP to influence adult BMI, the process described in the pathway model. The inclusion of adult SEP also resulted in an increase in the association between worse financial situation and baseline BMI. The black/white gap is further reduced, by an additional 10%, by accounting for adult SEP. The positive association between worse financial situation and annual BMI growth is reduced after adjusting for adult SEP (again primarily the effect of including respondent educational attainment). There is no effect of adult SEP on BMI growth and the black/white difference is similar to that in prior models. I also examined the effect of adult SEP without adjustment of early-life SEP (results not shown) and found similar patterns as those in Model 3.

Further adjustment for adult family structure and health behaviors in Model 4 has little effect on the associations of early- and later-life SEP on BMI trajectories. The black/white gap in baseline BMI is further reduced, by an additional 5%, but remains significant. The black/white difference in BMI growth gets larger, compared to the difference in Model 1, suggesting that black and white individuals with similar family structure and health behaviors are even more divergent in terms of BMI growth.

[TABLE 3.4 ABOUT HERE]

Cumulative Disadvantage

In addition to assessing processes that treat early- and later-life socioeconomic experiences as independent factors, I also wanted to determine if instances of socioeconomic disadvantage cluster together to produce cumulative effects of disadvantage on BMI trajectories, and if cumulative disadvantage has purely additive effects or if the effect is multiplicative. Table 3.5 shows the results for the accumulation of socioeconomic disadvantage adjusted for age, and later-life family structure and behaviors. The effects of cumulative disadvantage experienced in early-life are presented in Model 1. Early-life cumulative disadvantage is positively related to baseline BMI, with each one unit increase in the cumulative score associated with a 0.46 ($p<.01$) point increase in BMI. Early-life cumulative disadvantage is also positively related to BMI growth, where a one unit increase in the cumulative score is associated with an annual increase in BMI of 0.022 ($p<.10$) points. Model 2 shows the effects of later-life cumulative disadvantage on BMI trajectories. Later-life accumulation of disadvantage is also positively associated with baseline BMI, with a 1.11 ($p<.001$) point increase in BMI for every one unit increase in the disadvantage score. There is no association of later-life cumulative disadvantage with BMI growth.

Model 3 shows the additive effects of early-life and later-life accumulation of socioeconomic disadvantage. Controlling for later-life accumulation of disadvantage, there is no longer an effect of early-life accumulated disadvantage on baseline BMI. However, the effect of early-life disadvantage on BMI growth remains. Adjusting for early-life cumulative disadvantage has little effect on the relationship between later-life cumulative disadvantage and baseline BMI. I determine if there is a multiplicative effect of early-life and later-life cumulative disadvantage by testing the interaction between

early-life scores and later-life scores; the result is shown in Model 4. I find a significant interaction of early- and later-life accumulation of socioeconomic disadvantage, but only for baseline BMI. This indicates that the effects of cumulative disadvantage on adult BMI are multiplicative rather than additive, meaning that it is the combination of cumulative disadvantage across periods of the life course that matters for determining BMI at a single point in adulthood. Individuals who experienced the highest levels of cumulative disadvantage, both in early- and later-life, have significantly higher adult BMI than those who experienced high levels of disadvantage during only one period.

[TABLE 3.5 ABOUT HERE]

I present results for cumulative socioeconomic disadvantage using a linear specification, but I also conducted the analysis using a categorical specification (results not shown). The results from models using categorical specifications of cumulative disadvantage indicate that the positive effect of early-life disadvantage on baseline BMI is driven primarily by those with the highest scores (3-4 points) and that the effect of early-life cumulative disadvantage is non-linear. Similarly there is a much larger effect of later-life cumulative disadvantage on baseline BMI for those with the highest score (3 points), though the effect does increase monotonically. Although the effects of cumulative disadvantage on BMI trajectories are not truly linear in nature, the interpretation of results treating the effects as linear is substantively similar to the interpretation using a categorical specification. In addition, using linear terms for

cumulative socioeconomic disadvantage allowed me to test the two-way interaction between early-life scores and later-life scores.

The significant interaction between early- and later-life experiences of disadvantage indicates that it is the combination of experiences in early-life and later-life that matter for adult BMI. Cumulative disadvantage processes that are multiplicative in nature indicate that what matters for health is the experience of patterns of advantage and disadvantage over the life course. To illustrate this point, I created patterns of educational mobility using parental educational attainment and respondent educational attainment. Individuals whose parents had more than 12 years of education and who themselves had more than 12 years of education experienced low disadvantage in early-life and in later-life (low-low), whereas, individuals who parents had less than 12 years of education and who themselves achieved less than 12 years of education experienced high disadvantage in early-life and in later-life (high-high). Individuals who achieved a higher level of education than their parents experienced upward educational mobility, while those who achieved less education than their parents experienced downward mobility.

The results for educational mobility are presented in Table 3.6. Compared to those who were never disadvantaged in terms of education, individuals who were upwardly mobile, meaning they experienced some disadvantage in early-life but were less disadvantaged in later-life, had higher baseline BMI (0.77 $p<.10$). Individuals whose parents had 12 years of education and who themselves had 12 years of education and thus experienced moderate disadvantage in both early- and later-life also had comparatively higher baseline BMI (1.04, $p<.10$). Individuals who experienced downward mobility were 1.6 ($p<.01$) points higher on baseline BMI. The individuals with the highest

baseline BMI, by far (3.6, p<.001), experienced a combination of high levels of disadvantage in both early- and later-life. These results confirm that the combination of disadvantage in early-life and later-life leads to higher adult BMI than experiencing disadvantage during only one life course period.

DISCUSSION

Although prior research has found a relationship between early-life socioeconomic conditions and adult obesity, the processes that underlie this relationship have received very little attention. Several theoretical models have been proposed to explain the link between early life exposures and health status in adulthood (Ben-Shlomo and Kuh et al. 2002; Lynch and Davey Smith 2005). The primary aim of this study was to explain how early life matters for adult BMI trajectories. In this study I focused on pathway, latency, and cumulative disadvantage models.

I found that early-life socioeconomic disadvantage is positively associated with both baseline BMI and BMI change. Though the effects of early-life are mediated heavily through adult SEP, the results confirm that early-life socioeconomic experiences have enduring effects on adult weight and weight gain. Thus, the latency model of life course processes may best represent the pathway by which specific early-life socioeconomic conditions affect adult BMI trajectories. This model allows for early-life socioeconomic conditions to operate via adult socioeconomic attainment and also to have direct effects on health in later-life. The positive relationship between early-life financial situation and baseline BMI and BMI change, net of adult characteristics, indicates that economic hardship in early life sets individuals on a trajectory of weight gain over and above the

normal age-related weight gain trajectories, thus producing divergence over time. It is important to note, however, that adult socioeconomic attainment is also an important predictor of BMI and that adult characteristics have an effect on BMI even after accounting for socioeconomic origins.

The results suggest that early–life may be a significant period during which adult weight trajectories are established. Traditional critical period models focus on biological mechanisms linking early-life origins to adult outcomes and these processes are thought to occur primarily during gestation or in the initial stages of development following birth. However, the critical period for exposure to socioeconomic disadvantage likely extends throughout childhood. Studies have shown that early-life socioeconomic disadvantage increases childhood risk for overweight and obesity (Kimm et al. 2006; Miech et al. 2006; Strauss and Knight 1999), which may increase the risk of becoming obese as an adult (Whitaker et al. 1997). Early-life socioeconomic position determines behavioral habits in children and exposure to environmental factors that contribute to obesity. For instance, Miech et al. (2006) found that early-life socioeconomic position is associated with poorer diets and lower levels of physical activity in children. Gordon-Larsen and colleagues (2006) found that children living in low SEP areas have access to fewer facilities for physical activity which in turn was associated with less physical activity and higher weight status. Lower early-life socioeconomic position is also found to be related to worse diet and physical activity habits in adulthood (Lynch et al. 1997). Early life socioeconomic position may also cluster with other early life factors that predict adult obesity such as maternal and infant body weight (Curhan et al. 1996) and perinatal nutritional surpluses and deficits (Owen et al. 2005).

I also found evidence for cumulative effects of socioeconomic disadvantage on adult BMI trajectories. Early-life disadvantage interacts with later-life disadvantage and is associated with higher baseline BMI, though it is not associated with BMI change. This finding indicates that adult BMI increases with increasing exposure to socioeconomic disadvantage, but that the effect is stronger when disadvantage is higher in both early- and later-life. For instance, individuals who were disadvantaged in terms of educational achievement in both early-life and as adults had much higher BMI compared not only to those who did not experience any disadvantage, but also compared to those who experienced disadvantage during only one period of the life course.

A secondary aim of this study was to determine if early-life SEP accounted for the large racial and ethnic differences in BMI trajectories among women. I found that early life socioeconomic position accounted for some of the observed black/white difference in baseline BMI and fully accounted for baseline differences between Hispanic and white women. However, there was no effect of early-life SEP on BMI change. Relatively little of the large gap between black and white women in baseline BMI was explained by life course SEP, adult family structure, and adult health behaviors, and the difference in BMI growth actually increased with the inclusion of these factors.

Adult obesity has become a major health concern in the United States due to the recent, rapid increase in prevalence rates and the positive association between obesity and a number of chronic health conditions, including three of the leading causes of death: diabetes, heart disease, and cancer (Kenchaiah et al. 2002; Mokdad et al. 2003; Must et al. 1999; Vischer and Seidell 2001). Understanding the early-life determinants of obesity is useful because adult weight trajectories are likely established, if not in childhood and

adolescence, then certainly in early adulthood. Thus, policies focused on reducing obesity should target risk factors across the life course. Research shows that disparities in health, and the socioeconomic factors that contribute to health, emerge in early-life and accumulate throughout adulthood. Thus, while reducing the large racial and ethnic disparities in obesity among U.S. women requires addressing the underlying social factors that produce inequality, it is important to consider when these factors emerge over the life course.

This study is the first to use a life course framework to understand how early-life and later-life socioeconomic conditions influence adult obesity over a 15-year period of the adult life course, using a nationally representative sample of U.S. women, including Hispanic women in addition to black and white women. An additional strength of this study is that multiple measures of socioeconomic conditions in early-life and adulthood are used to get a better assessment of socioeconomic position. Finally, while existing studies of life course SEP and adult obesity typically rely on one or two measures to determine early-life and adult SEP, this study utilizes multiple measures of SEP. Krieger and colleagues (1997) caution against relying on a single measure of socioeconomic position because an individual measure may not adequately depict how racial/ethnic disparities in health are shaped by socioeconomic position. Using multiple measures of socioeconomic position in childhood and adulthood may also decrease measurement error associated with any given variable.

There are a few limitations to the study that should be noted. Although several different measures of early-life socioeconomic position were used in this study, parental occupational class was not used. Parental occupation is an indicator of early-life

socioeconomic conditions that has been used in a number of prior studies. This information was not available for this study, but the inclusion of four different measures of socioeconomic position in early-life, particularly parent's education, likely reflect early-life socioeconomic position as well as, or better, than parental occupation

An additional potential limitation to this study is that the measures of cumulative socioeconomic disadvantage used in the analysis ignore the timing and severity of exposures to disadvantage. The measures assume that any given exposure has the same value regardless of when it occurred in the life course, and that exposures are of equal severity in their association with the outcome. It is possible that experiences during certain periods in the life course matter more for BMI trajectories and that the effects of certain exposures may be more severe than others. Finally, I only have information on adult exposure to socioeconomic disadvantage at the time of interview. Thus, I do not know what the respondent was exposed to between age 16 and the time of the interview. Future work should examine cumulative experiences of socioeconomic disadvantage over a longer period of time, with particular attention to the transition from early life to early adulthood.

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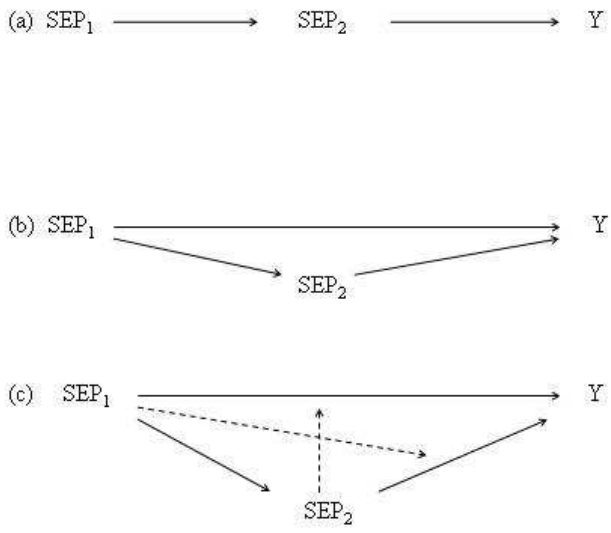
FIGURES

Figure 3.1. Life Course Pathways Linking Early-Life SEP to Later-Life Outcomes: (a) Pathway Model, (b) Latency Model, (c) Cumulative Disadvantage Model (Additive and Multiplicative Effects)



Note: SEP_1 = Early-life socioeconomic position; SEP_1 = Adult socioeconomic position Y = Outcome

TABLES

Table 3.1. Descriptive Satisitics for Body Mass Index (1986-2001), Age (1986), and Early-Life Socioeconomic Characteristics for All Women and by Race/Ethnicity.

	All		White		Black		Hispanic	
	Mean/%	Sd	Mean/%	Sd	Mean/%	Sd	Mean/%	Sd
Body Mass Index								
1986	25.17	(5.05)	24.83	(4.87)	27.85	(5.78)	25.73	(4.94)
1989	25.62	(5.25)	25.22	(5.02)	28.12	(6.19)	26.47	(5.47)
1994	26.05	(5.26)	25.63	(5.12)	28.79	(5.83)	27.45	(5.26)
2001	27.15	(5.71)	26.72	(5.54)	30.52	(6.67)	27.96	(5.51)
Age (years) 1986	47.20	(15.96)	48.17	(16.22)	46.71	(15.22)	41.66	(13.57)
Early-Life SEP								
Parent Education								
< 12 years	0.42		0.37		0.57		0.71	
12 years	0.33		0.36		0.30		0.20	
> 12 years	0.25		0.27		0.12		0.09	
Intact Family	0.23		0.20		0.41		0.32	
Relative Financial Situation								
Worse than Average	0.15		0.14		0.12		0.36	
Average	0.72		0.74		0.71		0.54	
Better than Average	0.13		0.12		0.17		0.10	
Worked Before Age 16	0.16		0.12		0.25		0.38	
Unweighted N	1809		1112		576		86	

Note: Figures are weighted using 1986 sampling weight, column Ns are unweighted.

Table 3.2. Descriptive Satisitics for Socioeconomic Characteristics, Family Structure, and Health Behaviors in Adulthood, by Survey Year.

	1986					1989					1994					2001				
	%	Mean	Sd	Min	Max	%	Mean	Sd	Min	Max	%	Mean	Sd	Min	Max	%	Mean	Sd	Min	Max
Later-Life SEP																				
Respondent Education																				
< 12 years	0.23																			
12 years	0.35																			
> 12 years	0.41																			
Income[a]																				
Lowest Tertile	0.32	7.9	(3.9)	2.5	12.5	0.29	7.5	(3.2)	1.1	12	0.24	9.1	(3.6)	1.2	15	0.27	13.7	(5.6)	0	24
Middle Tertile	0.29	22.3	(4.1)	17.5	27.5	0.30	21.6	(4.7)	12.4	30	0.34	25.4	(6.5)	15	37	0.36	39.2	(9.2)	24	55
Highest Tertile	0.39	53.4	(22.8)	35.0	110	0.41	60.1	(35.9)	31.0	250	0.42	72.4	(50.1)	37.2	500	0.37	112.6	(94.2)	56	1000
Difficulty Paying Bills																				
Very/Extremely	0.09					0.08					0.07					0.07				
Slightly	0.22					0.19					0.21					0.16				
Somewhat	0.26					0.25					0.25					0.25				
None at All	0.42					0.48					0.46					0.52				
Family Structure																				
Marital Status																				
Married	0.66					0.63					0.63					0.60				
Div/Sep/Widowed	0.26					0.30					0.31					0.35				
Never married	0.08					0.07					0.06					0.05				
Kids in HH < 18 years	0.46					0.42					0.32					0.24				
Health Behaviors																				
Smoking Status																				
Current	0.28					0.24					0.21					0.16				
Former Smoker	0.50					0.49					0.48					0.48				
Never Smoker	0.22					0.26					0.30					0.36				
Physical Activity		-0.05	(0.98)	-2.47	1.50		-0.29	(0.97)	-2.36	1.50		-0.02	(0.96)	-2.37	1.50		-0.10	(0.92)	-2.37	1.33
Unweighted N			1809					1809					1495					1060		

Note: Figures are weighted using wave-specific sampling weight, column Ns are unweighted.

a. Household income in $1000's reported by income tertile.

Table 3.3. Pearson Correlations of Early-Life and Later-Life Measures of Socioeocnomic Position

		Early-Life				Later Life		
		(1)	(2)	(3)	(4)	(6)	(7)	(8)
Early-Life	(1) Parental Education	1.00	–	–	–	–	–	–
	(2) Intact Family	-0.05	1.00	–	–	–	–	–
	(3) Relative Financial Situation	-0.15	0.15	1.00	–	–	–	–
	(4) Worked Before Age 16	-0.14	0.12	0.25	1.00	–	–	–
Later Life	(6) Respondent Education	0.43	-0.19	-0.15	-0.17	1.00	–	–
	(7) Income Tertiles[a]	0.31	-0.13	-0.06[b]	-0.14	0.48	1.00	–
	(8) Difficulty Paying Bills[a]	-0.01[b]	0.09	0.05[b]	0.01	-0.09	-0.28	1.00

Note: Figures are weighted using 1986 sampling wieght. Correlation coefficients are significant at $p<.05$ unless otherwise noted.

a. Measured in 1986.

b. Not statistically significant.

Table 3.4. Effects of Early-Life and Adult SEP on Baseline BMI and BMI Change, Weighted Multilevel Regression Coefficients (SE)

	Model 1			Model 2			Model 3			Model 4		
Baseline BMI (ref = White)	22.26	(0.40)	***	22.17	(0.61)	***	22.30	(0.60)	***	21.69	(0.64)	***
Black	2.92	(0.32)	***	2.48	(0.35)	***	2.21	(0.36)	***	2.10	(0.36)	***
Hispanic	1.30	(0.65)	*	0.76	(0.64)		0.24	(0.65)		0.02	(0.64)	
Other	-0.56	(0.74)		-0.80	(0.79)		-0.60	(0.76)		-0.98	(0.75)	
Age	0.23	(0.03)	***	0.21	(0.03)	***	0.21	(0.03)	***	0.21	(0.03)	***
Age^2	0.00	(0.00)	***	0.00	(0.00)	***	0.00	(0.00)	***	0.00	(0.00)	***
Early-Life SES												
Parent Education (ref= > 12 yrs)												
< 12 years	–			1.29	(0.39)	**	0.96	(0.42)	*	0.97	(0.42)	*
12 years	–			0.54	(0.39)		0.38	(0.39)		0.39	(0.39)	
Intact Family	–			0.11	(0.36)		-0.17	(0.34)		-0.02	(0.33)	
Relative Financial Situation (ref=Better than Average)												
Worse than Average	–			-0.99	(0.55)	+	-1.08	(0.55)	*	-1.18	(0.55)	*
Average	–			-0.30	(0.44)		-0.40	(0.44)		-0.49	(0.44)	
Worked Before Age 16	–			0.98	(0.40)	*	0.73	(0.40)	+	0.75	(0.40)	+
Adult SES												
Education (ref= > 12 yrs)												
< 12 years	–			–			1.93	(0.45)	***	1.94	(0.44)	***
12 years	–			–			0.18	(0.35)		0.21	(0.35)	
Income (ref= High Tertile)												
Low Tertile	–			–			-0.03	(0.16)		0.18	(0.17)	
Middle Tertile	–			–			0.05	(0.11)		0.15	(0.12)	
DifficultyPaying Bills (ref= None)												
Somewhat	–			–			0.00	(0.10)		-0.02	(0.10)	
Slightly	–			–			0.00	(0.12)		-0.02	(0.12)	
Very/Extremely	–			–			-0.28	(0.21)		-0.27	(0.21)	
Family Structure												
Marital Status (ref = Married)												
Div/Sep/Widowed	–			–			–			-0.54	(0.16)	***
Never married	–			–			–			-0.36	(0.33)	
Kids in HH < 18 years (yes= 0)	–			–			–			-0.08	(0.13)	
Health Behaviors												
Smoking Status (ref = Current)												
Former Smoker	–			–			–			1.40	(0.27)	***
Never Smoker	–			–			–			0.83	(0.16)	***
Physical Activity	–			–			–			-0.26	(0.06)	***
BMI Change												
Intercept (ref= White)	0.253	(0.017)	***	0.236	(0.031)	***	0.225	(0.030)	***	0.216	(0.031)	***
Black	0.151	(0.039)	***	0.154	(0.040)	***	0.152	(0.040)	***	0.160	(0.040)	***
Hispanic	0.051	(0.044)		0.037	(0.042)		0.046	(0.042)		0.057	(0.042)	
Other	0.047	(0.061)		0.049	(0.060)		0.036	(0.064)		0.039	(0.063)	
Age	-0.007	(0.001)	***	-0.007	(0.001)	***	-0.007	(0.001)	***	-0.007	(0.001)	***
Black*Age	-0.006	(0.001)	***	-0.006	(0.001)	***	-0.005	(0.002)	***	-0.005	(0.002)	***
Parent Education												
< 12 years	–			-0.013	(0.023)		-0.013	(0.024)		-0.008	(0.023)	
12 years	–			-0.008	(0.022)		-0.008	(0.023)		-0.004	(0.023)	
Intact Family				0.012	(0.020)		0.019	(0.020)		0.016	(0.019)	
Relative Financial Situation												
Worse than Average	–			0.070	(0.034)	*	0.064	(0.033)	+	0.063	(0.033)	+
Average	–			0.012	(0.026)		0.011	(0.025)		0.010	(0.025)	
Worked before age 16	–			0.011	(0.026)		0.026	(0.026)		0.030	(0.026)	
Respondent Education												
< 12 years	–			–			-0.028	(0.027)		-0.028	(0.026)	
12 years	–			–			0.029	(0.018)		0.028	(0.018)	
Dead by Wave 4	-0.097	(0.034)	**	-0.097	(0.034)	**	-0.102	(0.033)	**	-0.102	(0.033)	**

Note: ***p<.001, **p<.01, *p<.05, +p<.10 (two-tailed tests).

Table 3.5. Cumulative Effect of Early-Life and Later-Life Socioeconomic Disadvantage on Baseline BMI and BMI Change, Weighted Multilevel Regression Coefficients (SE).

	Model 1			Model 2			Model 3			Model 4		
Early-Life Disadvantage	0.46	(0.18)	**	–			0.18	(0.17)		-0.52	(0.35)	
Later-Life Disadvantage	–			1.11	(0.18)	***	1.06	(0.18)	***	0.29	(0.39)	
Early-Life*Later-Life										0.49	(0.23)	*
Time*												
Early-Life Disadvantage	0.022	(0.012)	+	–			0.024	(0.012)	*	0.001	(0.022)	
Later-Life Disadvantage	–			-0.004	(0.010)		-0.010	(0.010)		-0.038	(0.027)	
Early-Life*Later-Life										0.018	(0.018)	

Note: ***p<.001, **p<.01, *p<.05, +p<.10 (two-tailed tests). Models adjust for age, race/ethnicity, family structure, and health behaviors.

Table 3.6. Effects of Educational Mobility on Baseline BMI and BMI Change, Weighted Multilevel Regression Coefficients (SE).

		Baseline BMI			BMI Growth	
	Unweighted N	β	(se)		β	(se)
Low-Low (ref)	210	–			–	
Upwardly Mobile	681	0.77	(0.42)	+	0.016	(0.024)
Med-Med	197	1.04	(0.54)	+	0.001	(0.027)
Downwardly Mobile	242	1.60	(0.56)	**	0.013	(0.032)
High-High	474	3.10	(0.50)	***	-0.037	(0.035)

Note: ***p<.001, **p<.01, *p<.05, +p<.10 (two-tailed tests). Models adjust for age, race/ethnicity, family structure, and health behaviors. Low = >12 years of education, Med = 12 years of education, High = <12 years of education, Upwardly Mobile = Adult education higher than parental education level, Downwardly Mobile = Adult education lower than parental education level.

CHAPTER IV

DO SPOUSAL AND PARTNER RELATIONSHIPS MATTER FOR OBESITY? DIFFERENCES IN BODY MASS INDEX BY RELATIONSHIP TRANSITIONS AND QUALITY

Are relationships always good for one's health and well being? A number of studies find that being married is associated with better health-related behaviors and lower mortality (Waite 1995). However, the health-promoting effects of being in a relationship most likely depend on the quality of the relationship. For instance, individuals in relationships characterized by high levels of conflict and low levels of social support may not receive the health benefits thought to accrue to those in relationships and may even fare worse in terms of health and well being compared to those not in a relationship.

Both the quantity and quality of relationships are thought to be causally related to health, benefiting health either via main effects on health or as buffers against life stressors. Research suggests that being socially integrated and having social ties, in other words simply being in relationships, improves health (Berkman and Glass 2000). Relational content, the quality of social relationships, has also been linked to health and may be an important mechanism linking social relationships to health (House, Umberson, and Landis 1988). Although relationship structure tends to be positively

related to health, the health benefits of relational content depend on the nature of that content. Positive relational content, such as social support, – is thought to be health promoting while negative relational content, such as demands and conflicts, is thought to be health damaging.

Body mass index (BMI) is one of the few health outcomes for which married individuals are not advantaged over single individuals. Those who are married or who have been married have higher average BMI and are more likely to be obese compared to single people (Chou et al. 2002; Sobal, Rauschenbach, and Frongilli 1992). In addition, prior research suggests that transitions into and out of relationships result in weight gain and weight loss. A study of worksite health promotion found that over 4 years men and women who entered marriage had higher BMI compared to those with no change in marital status and that women who became unmarried had lower BMI (Jeffrey and Rick 2002). Conversely, a national study spanning 3 years found that men and women who exited from marriage had lower BMI than those who remained married, but that men and women who entered marriage did not have higher BMI (Umberson 1992). Another national study with a 10-year follow-up found that men who exited marriage lost weight, women who entered marriage gained weight, and individuals who were never married lost weight, compared to those who remained in a relationship (Sobal, Rauschenbach, and Frongilli 2001). However, with such a long lag between the relationship transition and follow-up, it is difficult to determine if the transition caused the weight change or if events that occurred between baseline and follow-up caused the weight change. Prior studies of the effect of marital transitions on weight change provide some mixed evidence

suggesting that entry into marriage is associated with weight gain and exit from marriage is associated with weight loss.

Obesity has become a serious health concern both at the individual and population level (Allison et al. 1999; Flegal et al. 2005; Fontaine et al. 2003; Kenchaiah et al. 2002; Mokdad et al. 2001; Must et al. 1999; Vischer and Seidell 2001). Past research suggests that being in a relationship is associated with higher BMI and increased weight gain and that exiting a relationship may be related to weight loss. Research on the associations between relationship transitions and weight change is fragmented and needs more intensive examination of relationship changes and BMI changes. In addition, the role of relationship quality in determining BMI has not been explored. The potentially negative impact of relationships on BMI deserves more attention both because it is important to understand the determinants of adult weight gain but also because it may improve our understanding of how relationships affect health in both positive and negative ways. In this paper I use a nationally representative study of U.S. adults to assess 1) the effect of experiencing different relationship transitions on BMI and 2) the impact of relationship quality on BMI.

Relationship Status and Health

A large body of animal and human research has found health promoting effects of being in social relationships (House, Landis, and Umberson 1998). The degree to which individuals are socially integrated and the extent of their social networks has been linked to morbidity and mortality in a number of studies (Berkman and Glass 2000; Seeman 1996). Social ties with friends and families are thought to play a central role in

maintaining good health and buffering against threats to health. Prior research has identified biological and social mechanisms linking relationships to health. Physiological pathways linking social integration to health are found in studies that show lower immune function and higher stress load among those who are socially isolated, the converse of social integration (Seeman 1996). Prior research has also identified social and behavioral pathways linking social integration/isolation to health (Klinenberg 2008). Marital relationships have particular importance for health since these are often the closest social ties that many adults have.

Although the benefits of marriage are still debated, there is a significant amount of evidence showing that marriage is generally positively related to health and well being and is protective against mortality (Berkman and Glass 2000; Hu and Goldman 1990; Gove 1973; Hughes and Booth 1981; Kobrin and Hendershot 1977; Verbrugge 1979; Waite and Gallagher 2000). However, marriage is not positively related to all health outcomes. For instance, in a study of the effects of relationship on health behaviors, Umberson (1992) found that married men and women were less likely to engage in unhealthy behaviors such as smoking and excessive alcohol consumption. However, being married was associated with higher BMI and less physical activity. In addition, Umberson found that the transition out of marriage increased risky behaviors among men, but decreased BMI among both men and women. Thus, social relationships such as marriage may not exert a positive influence over all domains of health.

Mechanisms Linking Relationships to Health

Mechanisms by which marriage (and other relationships) benefits health include processes of social support and social control that inhere in social relationships. Researchers argue that social relationships, such as marriage, provide health benefits through processes of social control and self-regulation (Ross 1995; Umberson 1987, 1992). In addition the social support provided by a spouse or partner may help individuals deal with stressful situations in ways that are not harmful to their health.

Social support has been defined in a number of ways, but is commonly conceptualized in terms of emotional and instrumental support. Emotional support refers to the things people do that make us feel loved and cared for and instrumental support refers to the different types of assistance others provide. Supportive relationships are generally characterized as providing emotional sustenance and instrumental support (House, Umberson and Landis 1988). Processes of social support may operate at all times to influence health, or may only operate in the presence of stressors, to buffer against their negative effects on health (Cohen and Wills 1985).

Another aspect of relationships thought to benefit health is social control or regulation. Social control refers to the process through which relationships regulate health behaviors. Umberson (1987) found that social control, an important aspect of family integration, acts as a deterrent for participation in negative health behaviors. In Umberson's study, families exerted social control on individuals in a way that encouraged healthy behaviors and discouraged unhealthy behaviors. However, the presence of social control did not influence BMI. Thus, social control does not positively influence all health behaviors.

A more complete understanding of how relationships affect health should therefore include a focus on both the positive and negative sides of social interactions (Rook 1984). For instance, social ties do not necessarily equate with supportive relationships. In addition, although married individuals are more socially integrated, the relationship itself may be a source of stress. There are several pathways through which marital relationships negatively affect health. For instance, martial relationships characterized by demands and conflicts may undermine the physical health of wives and husbands by decreasing psychological well being and increasing psychological distress (Ross, Mirowsky, and Goldstein, 1990). Marital stress has been found to decrease immunologic response and increase stress reactivity (Kiecolt-Glaser et al 1997; Kiecolt-Glaser et al 1998) Empirical evidence suggests that chronic stress, produced for instance by prolonged exposure to stressful relationships, stimulates the production of stress hormones such as cortisol which are related to abdominal obesity (Bjorntorp 2001). In addition, the quality of the martial relationship may bear directly on whether wives and husbands engage in health-enhancing behaviors (Umberson 1987; Wickrama, Conger, and Lorenz 1995).

Current Study

The aim of this study is to determine how BMI differs by transitions into and out of relationships and to assess the impact of relational content on BMI. I examine both negative (stress) and positive (support) aspects of relationship quality. Prior research suggests that people in relationships have higher BMI and that BMI changes in different ways for people who transition into and out of relationships. However, no studies have

examined the impact of relationship stress and social support on BMI. Prior conceptual and empirical work on relationship quality would suggest that the impact of relationships on health differs according to levels of stress and social support generated by these relationships. Furthermore, the effect of transitions into and out of relationships may differ according the quality of the relationship. Exiting from a low quality relationship could have positive health effects, while entering a low quality relationship could have negative health effects. Conversely, exiting a high quality relationship could have negative health effects, while entering a high quality relationship could have positive health effects.

This study addresses three hypotheses which are informed by prior theoretical and empirical work on relationship status, stress, and social support.

1) Compared to individuals who stay in a relationship, transition out of a relationship is associated with lower BMI, transition into a relationship is associated with higher BMI, and remaining single is associated with lower BMI.

2) High relationship stress at baseline is positively associated with BMI at follow-up for those who remain in the relationship. BMI is higher for those who transition into a stressful relationship. BMI is lower for those who exit a stressful relationship.

3) High spousal/partner social support at baseline is negatively associated with BMI at follow-up for those who remain in the relationship. BMI is lower for those who transition into a relationship characterized by high social support. BMI is higher for those who exit a relationship characterized by high social support.

DATA AND METHODS

Data

I analyze data from the Americans' Changing Lives (ACL), a 15-year longitudinal panel study of noninstitutionalized adults aged 24 and older in the contiguous United States. The ACL is a stratified, multistage area probability sample with an oversampling of adults aged 60 and older and black adults. Initial face-to-face interviews were conducted in 1986 (n = 3,617) with follow up interviews occurring in 1989 (n = 2,867), 1994 (n = 2,562), and 2001/2002 (1,787). Analyses are weighted to take account of different rates of selection and differential non-response. In addition a post stratification weight is applied that makes the ACL sample representative of the age, gender, and race distribution of the population age 24 and older living in the United States in 1986.

Measures

Descriptive statistics for all dependent and independent variables are presented in Table 4.1 separately for women and men. I present the dependent variable, BMI, for respondents who were present for the 2001/2002 wave of the survey, though the analysis uses BMI measures from 1986, 1989 and 1994 (this is discussed in more detail in the description of the analytic strategy), and independent variables measured at baseline (1986). All figures presented in Table 1 are weighted.

Body Mass Index

BMI is calculated by dividing self-reported weight (in kilograms) by height (in meters) squared. Respondents were asked about their height in the baseline interview and

were asked about their weight in every wave of data collection for which they were interviewed. Those missing on height or weight (2.4%, 1.9%, 0.98%, and 2.2% of the W1, W2, W3, and W4 samples, respectively) were given imputed BMI values derived from sex-specific prediction equations that accounted for respondent's age, race, and prior height and/or weight (when available). At baseline women had a mean BMI of 25.1 and men had a mean BMI of 26.2. Mean BMI increased over the study period for both men and women.

Relationship Transitions

Change in relationship status was defined based on the individual's report of current marital or cohabitation status. At each wave individuals indicated whether they were currently married, divorced, separated, widowed, or had never been married. In addition, individuals were asked whether they were involved in an intimate relationship. I combined responses to these two questions to create four categories of relationship change: continuously married or living with someone, exited a marital or cohabiting relationship (i.e., married or cohabiting at baseline but not married or cohabiting at follow-up), entered a marital or cohabiting relationship (i.e., not married or cohabiting at baseline but married or cohabiting at follow-up), previously married but not in a relationship (i.e. reported being divorced, separated, or widowed at baseline and follow-up), and never married.

At baseline, 66% of women and 80% of men were married or cohabiting, 27% of women and 10% of men were divorced, separated, or widowed, and 7% of women and 10% of men had never been married and were not in a relationship. During the study

period 1,681 individuals were in a relationship continuously between waves, 488 individuals experienced an exit from a relationship, 290 individuals experienced an entrance into a relationship, 999 individuals were single between waves but had been married at some point in the past, and 225 individuals were single between waves and had never been married.

Relationship Quality

In each wave respondents were asked a series of questions about their marital/cohabiting relationships. These measures were used to create indices of relationship stress and spousal/partner positive social support. Marital stress is based on three questions: (a) "how often would you say the two of you typically have unpleasant disagreements or conflicts?" (never = 1, less than once a month = 2, about once a month = 3, 2 or 3 times a month = 4, about once a week = 5, 2 or 3 times a week = 6, daily or almost daily = 7), (b) "how satisfied are you with your marriage?" (completely satisfied = 1, very satisfied = 2, somewhat satisfied = 3, not very satisfied = 4, not at all satisfied = 5), and (c) "how often do you feel bothered or upset by your marriage?" (never = 1, rarely = 2, sometimes = 3, often = 4, almost always = 5). The metric for the last two items was expanded from 1-5 to 1-7 and the index was created by taking the arithmetic mean of the items. Wave-specific scale reliabilities ranged from α=0.63 to α=0.68. The average score on the stress index was 2.6 for women and 2.5 for men, indicating that most respondents perceived some stress in their relationship but that few respondents reported very high levels of stress, but men had a maximum stress score of 6.

I am interested in assessing the contrast between individuals who experience high levels of relationship stress and individuals who experience relatively less stress

Therefore, I created a categorical stress variable distinguishing between four levels of stress based on the continuous stress measure described above. The categories are: 1 - no stress (score = 0), 2 - very little stress (score = 0-1), 3 - little to medium stress (score = 1-3), and 4 - high stress (score > 3). Among women, 4% reported no stress, 31% experienced very little stress, 56% reported more than a little stress, and 9% reported high stress. The distribution was similar for men, though a lower proportion of men reported high stress (7%) and a slightly higher proportion reported no stress (6%).

The spousal positive support index is derived from responses to two questions: (a) "how much does your husband/wife make you feel loved and cared for?" and (b) "how much is your husband/wife willing to listen when you need to talk about your worries or problems?" Response categories are not at all = 1, a little = 2, some = 3, quite a bit = 4, and a great deal = 5. The index was constructed by taking the arithmetic mean for the two items. Wave-specific scale reliabilities ranged from α=0.58 to α=0.67. The average support score was 4.1 for women and 4.3 for men, indicating that most individuals experienced very high levels of social support from their spouses or partners and that few individuals felt they received very little support.

I also created a categorical support variable to better capture the contrast between individual who have highly supportive relationships and individuals who have relatively lower support. The four categories of support are: 1 - little to no support (score = 1-3), 2 - some support (score = 2-2.5), 3 - fairly high support (score = 1.5), and 4 - high support (score > 3). Among women, 16% reported no or little support, 31% had some support, 24% reported fairly high support, and about 30%% reported high support. The

distribution was similar for men, though a higher proportion of men reported fairly high to high support.

Controls

I include controls for demographic characteristics, socioeconomic status (SES), parental status, and health behaviors. Demographic controls include age (centered on 24 years) and race (2 = black, 1 = Hispanic, 0 = white). The mean age at baseline (1986) for women was 48.6 and the mean age for men was 46.1. About 81% of women are white, 13% are black, and 7% are Hispanic. Among men, 83% of respondents are white, 10% are black, and 7% are Hispanic. SES measures include education (centered on 12 years) and household income (logged). The average number of years of educational attainment is around 12 years for both women and men. Women had slightly lower household income at baseline than men ($28,000 vs. $33,000).

The respondent's parental status may affect spousal/partner relationship quality so I include a dummy variable for parental status at each wave indicating whether respondents have children under the age of 18 in the household (1 = yes, 0 = no). About 45% of women and men had children under age 18 present in the household at baseline.

Individuals experiencing marital/partner conflict may engage in unhealthy behaviors such as smoking to cope with relationship stress. On the other hand, individuals in a supportive relationship may be more likely to engage in good health habits such as physical activity. Both smoking and physical activity have been shown to be important predictors of weight and thus may be important confounders in the association between relationship stress and support and BMI. Therefore, I also include

adjustments for smoking and physical activity. Smoking status is measured using dummy variables indicating if the respondent is a current smoker (reference), a former smoker, or has never smoked. At baseline about 30% of women were current smokers, 21% were former smokers, and 50% had never smoked. Among men, about 31% were current smokers, 36% were former smokers, and 33% had never smoked. Physical activity is derived from three questions about how often the respondent "typically works in the garden or yard," "takes walks," and "engage(s) in active sports or exercise." Response categories are: often = 4, sometimes = 3, rarely = 2, or never = 1. An index of physical activity was created by first reverse coding the items then summing across the three items and standardizing. The index ranges from the lowest level of physical activity to the highest level of physical activity.

Analytic Strategy

For the multivariate analysis I created person-period records for each respondent to capture changes in the dependent and independent variables between waves. The first period reflects change that occurred between 1986 and 1989, and contains information about BMI in 1986 and in 1989, indicators for change in relationship status between 1986 and 1989 socioedemographic characteristics measured in 1986, and relationship quality reported in 1986. The second period reflects changes that occurred between 1989 and 1994 and the third period reflects changes that occurred between 1994 and 2001/2002. In the second period In the analysis I include an indicator variable for the person-period (1986-1989, 1989-1994, and 1994-2001/2002) that adjusts for the number of years between time 1 and time 2 observations.

I treat BMI as a continuous outcome and use ordinary least squares multiple regression models with robust standard errors, estimating models separately for women and men. In the analysis I adjust for the clustering of multiple observations per person. In the models I regress BMI at time 2 (1989, 1994, or 2001/2002) on predictors from time 1 (1986, 1989, or 1994). All analyses are conducted using Stata version 10.0SE software.

I first examine the associations between different types of relationship transitions and BMI. I then restrict the analyses to individuals who were in a relationship *both* at baseline and follow-up or *either* at baseline or follow-up to assess the nature of the association between relationship quality, stress and support, and BMI. Thus I conduct analysis of relationship quality and BMI separately for those who were continuously in a relationship, those who exited a relationship, and those who entered a relationship.

RESULTS

I begin by describing the difference in BMI by type of relationship status change between waves. Figure 4.1 shows unadjusted mean BMI change by relationship transitions between time 1 and time 2, for women and men. Women who stayed in a relationship between time 1 and time 2 experienced about a half-point increase in BMI. Women who exited a relationship experienced a decrease in BMI and women who entered a relationship experienced a one point increase in BMI. Women who had never been married experienced an increase in BMI, while those who had previously been married but were single at time 1 and time 2 experienced a slight increase in BMI. A similar pattern is observed for men, but men who exited a relationship experienced a

larger decrease in BMI and men who entered a relationship experienced an increase in BMI of 1.5 points.

[FIGURE 4.1 ABOUT HERE]

I then examine the effect of relationship transitions on BMI in multivariate models presented in Table 4.2. Model 1 shows BMI associated with different relationship transitions adjusted only for age, race, and years between waves (indicator of the person-period under observation). Among both women and men, those who exited a relationship over the period had significantly lower BMI (β = -1.03 women, β = -1.87 men) compared to those who were continuously in a relationship. I adjust for potential confounders in model 2 by controlling for education, income, and parental status. The difference between those who exited a relationship and those continuously in a relationship over the period increased for both men and women after making this adjustment. In addition, women who entered a relationship had significantly lower BMI (β = -1.18) after adjusting for SES and parenting factors. Adjusting for health behaviors in model 3 slightly reduced the associations from model 2 for both women and men, but overall had little effect on the associations.

An individual's weight at any given time may largely reflect prior weight status. To control for the potential confounding of prior weight, I further adjust for baseline BMI in model 4. The coefficients in model 4 reflect BMI change from baseline. Even after making this adjustment, women and men who exited a relationship had significantly lower BMI compared to those who stayed in a relationship (β = -0.65 women, β = -1.06

men). However, while entering a relationship was associated with lower BMI in the prior model, after adjustment for baseline BMI, entering a relationship is associated with a significantly higher BMI ($\beta = 0.51$). In addition, model 4 shows that men who entered a relationship had higher BMI ($\beta = 0.91$) compared to those continuously in a relationship. I suspect this change between models reflects the fact that thinner people are more likely to enter relationships. Thus, the transition from the single state into a relationship may result in weight gain above and beyond that experienced by those who are continuously in a relationship, but individuals continuously in a relationship tend to have higher BMI at any given time compared to individuals who had only recently entered a relationship. Finally, model 4 shows that women who reported being single, and had not previously been married, had significantly lower BMI ($\beta = -0.44$) compared to those continuously in a relationship, after adjusting for baseline BMI. Thus, in addition to having lower BMI compared to married individuals as has been reported in prior studies, single individuals who had never been married also gain less weight over time compared to those in a relationship.

[TABLE 4.2 ABOUT HERE]

I next turn to assessing the impact of relationship quality on BMI. Only individuals who reported being in a relationship were asked about their relationship quality. Thus the analysis of relationship quality is limited to those who were continuously in a relationship, those who exited a relationship, and those who entered a

relationship. Furthermore, I analyze relationship quality separately for each type of relationship transition.

The impact of relationship stress on BMI for those who were continuously in a relationship is presented in Table 4.3. According to model 1, adjusting only for age, race, and years between waves, individuals who report very little stress and high relationship stress have higher BMI, but with only marginally significant effects ($\beta = 0.68$ and $\beta = 1.21$, $p<.10$). Further adjusting for SES and parental status in model 2 reduces the differences in BMI and the differences seen in model 1 are no longer statistically significant. Adjustment for health behaviors in model 3 has little effect. However, after adjusting for baseline BMI in model 4 the positive association of high stress and BMI becomes statistically significant $\beta = 0.49$, $p<.05$). There is also a marginally significant positive association between the no stress category and BMI. There are no patterns of association between relationship stress and BMI among men.

[TABLE 4.3 ABOUT HERE]

Table 4.4 shows the effect of spousal/partner support on BMI. Model 1 shows marginally significant positive effects of some support and high levels of support on BMI. The adjustment for SES and parental status in model 2 and health behaviors in model 3 result in little change to the coefficients. Adjusting for baseline BMI in model 4 reduces the effect of support on BMI, though the positive association between the category for some support and BMI remains significant ($\beta = 0.35$, $p<.05$). This suggests that lower levels of support are associated with higher BMI, though this does not extend

to those who reported having no support. There are no patterns of association between relationship stress and BMI among men.

[TABLE 4.4 ABOUT HERE]

I also examined the impact of relationship stress and spousal/partner support on BMI among those who exited a relationship and those who entered a relationship. I only report the fully adjusted models in Table 4.5; there was little variation in coefficients across models with fewer adjustments. Among women, there is no effect of stress on BMI for those who exited a relationship. This may reflect the fact that individuals are likely exiting from unhappy relationships and thus we might even expect to see that those who left a high stress relationship lost weight after exiting the relationship. In fact there is a negative coefficient for the high stress category, but the effect is not significant. Among women who entered a relationship, those who reported no stress from the new relationship had significantly lower BMI ($\beta = -2.09$, $p<.05$). There were no patterns of association among men. The impact of support is shown on the right side of the table. There are no patterns of association between support and BMI among for those who exited or entered a relationship.

[TABLE 4.5 ABOUT HERE]

CONCLUSION

Previous research suggests that the transition into marriage results weight gain (Umberson 1992; Sobal, Rauschenbach, and Frongilli 2001) while the transition out of marriage results in weight loss (Jeffrey and Rick 2002; Sobal, Rauschenbach, and Frongilli 2001). The present findings show that exit from a relationship is related to lower BMI and entry into a relationship is related to higher BMI compared to staying in a relationship. In contrast to the prior studies, these results were found for both men and women. In addition, similar to findings reported by Sobal, Rauschenbach, and Frongilli (2001) women who had never been in a relationship had lower BMI compare to those who were continuously in a relationship. These findings support Hypothesis 1 - being in a relationship and transitioning into a relationship has a negative impact on weight and never being in a relationship or exiting form a relationship is positively related to weight – and suggest that the health benefits of relationships are not universal and at least do not extend to BMI.

As predicted in Hypothesis 2, individuals who remained in a relationship characterized by high levels of stress had higher BMI. The results also suggest that individuals who reported no exposure to stress also had higher BMI, though the finding was not statistically significant at $p<.05$. The positive association between BMI and the absence of relationship stress may reflect the curvilinear pattern of marital satisfaction over the course of a marriage (Rollins and Feldman 1970). Individuals in the "honeymoon" stage that occurs in the initial years of marriage may be more likely to report having no relationship stress. But these individuals may also be more likely to gain weight over time as they adjust to the health habits of their partners. Because I had

limited information about the duration of the relationships I could not control for relationship duration in the models.

Individuals who enter into a relationship tend to gain weight, but the results show that individuals who entered less stressful relationships had lower BMI. This finding provides additional support for Hypothesis 2. However, there were no effects of stress for those who exited a relationship. There appears to be no health benefit of leaving a stressful relationship context. However, leaving a stressful relationship may actually increase overall stress if the loss of a relationship also results in a loss of financial security, relationships with extended family and friends, and additional burdens related to work and parenting. It is difficult to disentangle the effects of stressful relationships from the stressful effects that occur as a result of transitions out of relationships. Overall, these results suggest that negative relational content, experiencing demands and conflicts that promote stress, is positively associated with BMI.

Social support had virtually no impact on BMI. The results showed that individuals who remained in a relationship with low levels of social support had higher BMI. However, I did not find a significant effect of being in a relationship that is totally unsupportive. This mixed finding makes it difficult to conclude that social support is strongly related to BMI. Moreover, I found no effect of social support for those who exited or entered a relationship. Thus, I find little support for Hypothesis 3. The lack of effects of support on BMI may be due to the highly skewed distribution of responses on questions related to social support. Nearly everyone reports having a supportive spouse/partner, making it difficult to create a category with an adequate number of individuals in it that reflects absolutely no support. In addition, the social support

measure used in this study is a measure of emotional support. Instrumental support may be a more informative measure of support because getting help with parenting or housekeeping tasks might facilitate better eating and exercise habits and thus may matter more for weight than having someone to talk to or feeling loved.

All effects of relationship quality were found for women only; no effects were found for men. Prior research suggests that the effects of marital stress may be more severe for women than men, either because women actually experience more stress or because women internalize stressful experiences in a way that is more physiologically harmful. Women consistently report lower marital quality than men, suggesting that women may perceive more marital problems and are more likely to appraise the marital relationship as being stressful (Umberson et al. 1996). Relationships may also be more salient to the well-being of women than they are to men (Kessler and McLeod 1984). Research indicates that women experience stronger adverse affects of marital stress, displaying heightened physiological response (Kiecolt-Glaser et al. 1996; Kiecolt-Glaser et al. 1998). Thus, gender differences in appraisal of relationship quality and physiological response to stress may explain why BMI is related to relationship quality only among women.

This study, the first to assess the impact of relationship transitions and relationship quality on BMI, shows that the health promoting effects of being in relationships, and staying in relationships, may not extend to all health outcomes. Although relationships may be protective against chronic health problems and risky health behaviors, they are not protective against weight gain. Whether conceptualized as a health behavior, a health risk factor, or a health outcome, obesity is a condition that is

not necessarily improved by being in a relationship. However, the benefits of relationships depend strongly on relational content. Negative relational content, such as experiencing conflict and too many demands, is positively associated with weight gain among those who remain in or enter into relationships. Thus, in addition to relationship status and changes in status, the quality of relationships also determines weight gain. Assertions that relationships are universally good for one's health should be tempered by the findings reported in this study. Instead, we should continue to examine the health effects of both relationship structure and relational content using a variety of health outcomes to determine when social relationships do and do not promote health and the mechanisms that link relationships to health.

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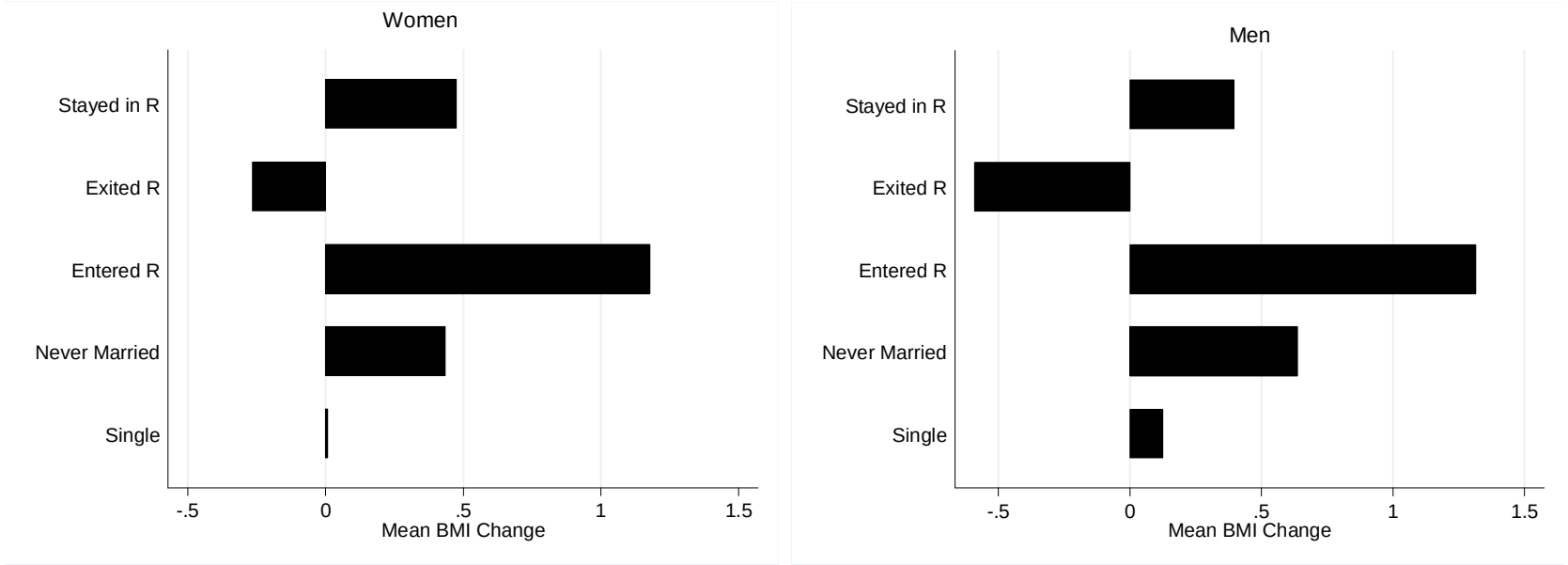
FIGURES

Figure 4.1. Mean BMI Change by Relationship Transition Types for Women and Men



Note: R = Relationship, BMI = Body mass index. BMI Change is the difference between BMI at Time 2 and BMI at Time 1. Positive change values indicate an increase in BMI over time and negative values indicate a decrease over time.

TABLES

Table 4.1. Descriptive Statistics for Dependent and Idnependnet Variables

	Women				Men			
	Mean/%	(sd)	Min	Max	Mean/%	(sd)	Min	Max
BMI 1986	25.1	(5.2)	15.5	55.1	26.2	(3.9)	18.1	44.9
BMI 1989	25.7	(5.4)	16.1	54.9	26.8	(4.3)	18.1	46.9
BMI 1994	26.3	(5.4)	16.1	54.7	27.4	(4.4)	18.4	50.2
BMI2001	27.3	(5.8)	16.1	57.6	28.0	(4.9)	16.2	52.5
Marital Status								
Married	0.66				0.80			
Div/Sep/Wid	0.27				0.10			
Never married	0.07				0.10			
Stress index	2.6	(1.1)	1	7	2.5	(1.0)	1	6
Stress Categories								
1 - No stress	0.04				0.06			
2 - Very little stress	0.31				0.32			
3 - Little to med stress	0.56				0.56			
4 - High stress	0.09				0.07			
Social Support Index	4.1	(0.9)	1	5	4.4	(0.7)	1	5
Support Categories								
1 - Little to no support	0.16				0.07			
2 - Some support	0.31				0.23			
3 - Fairly high support	0.24				0.25			
4 - High support	0.30				0.44			
Age at basline	48.5	(16.9)	24	96	46.1	(16.0)	25	92
Race								
White	0.81				0.83			
Black	0.12				0.10			
Hispanic	0.07				0.07			
Education, years	12.1	(3.0)	0	17	12.5	(3.3)	0	17
Household income	28.1	(23.9)	2.5	110	33.0	(24.1)	2.5	110
Kids < 18	0.44				0.45	(0.5)	0	1
Smoking Status								
Current smoker	0.30				0.31			
Former smoker	0.21				0.36			
Never smoker	0.50				0.33			
Physical activity	-0.13	(1.0)	-2.47	1.5	0.14	(0.9)	-2.36	1.5
Unweighted N	1791				1012			
Weighted N	1619				975			

Notes: Figures represent weighted means, or percentages where appropriate, and standard deviations (sd).

Table 4.2. Unstadardized Coefficients (Standard Errors) from Oridnary Least Squares Regression Models of BMI on Change in Relationship Status

	Women				Men			
	Model 1	Model 2	Model 3	Model 4	Model 1	Model 2	Model 3	Model 4
Relationship status[a]								
Exited relationship	-1.03 (0.35) **	-1.28 (0.34) ***	-1.24 (0.35) ***	-0.65 (0.25) **	-1.87 (0.47) ***	-2.02 (0.49) ***	-1.99 (0.48) ***	-1.06 (0.21) ***
Entered relationship	-0.48 (0.46)	-1.18 (0.48) *	-1.10 (0.47) *	0.51 (0.24) *	-0.52 (0.48)	0.04 (0.47)	0.01 (0.46)	0.91 (0.22) ***
Not in relationship	0.09 (0.37)	-0.52 (0.40)	-0.47 (0.40)	0.07 (0.11)	-0.75 (0.45) †	-0.51 (0.47)	-0.35 (0.49)	0.10 (0.18)
Never in relationship	-0.42 (0.87)	-1.03 (0.88)	-1.34 (0.85)	-0.44 (0.21) *	-0.61 (0.69)	0.04 (0.71)	-0.09 (0.72)	0.26 (0.30)
Age at basline	0.01 (0.01)	-0.03 (0.01) *	-0.04 (0.01) **	-0.04 (0.00) ***	-0.02 (0.01) *	-0.01 (0.01)	-0.02 (0.01) †	-0.03 (0.01) ***
Years between waves	0.34 (0.03) ***	0.40 (0.04) ***	0.39 (0.04) ***	0.15 (0.02) ***	0.29 (0.04) ***	0.31 (0.05) ***	0.31 (0.05) ***	0.09 (0.03) **
Black[b]	3.26 (0.37) ***	2.82 (0.39) ***	2.62 (0.39) ***	0.43 (0.12) ***	0.29 (0.38)	0.05 (0.41)	0.03 (0.41)	0.07 (0.12)
Hispanic[b]	1.61 (0.72) *	0.83 (0.79)	0.71 (0.78)	0.30 (0.23)	0.36 (0.72)	0.02 (0.75)	-0.39 (0.75)	-0.15 (0.17)
Education, years		-0.23 (0.07) **	-0.22 (0.07) **	-0.04 (0.02) †		-0.12 (0.08)	-0.13 (0.07) †	0.00 (0.02)
Household income		-0.61 (0.19) **	-0.59 (0.19) **	0.04 (0.07)		-0.03 (0.26)	-0.03 (0.25)	-0.08 (0.08)
Kids < 18		-0.84 (0.38) *	-0.93 (0.38) *	-0.21 (0.13)		1.10 (0.39) **	0.99 (0.39) *	0.30 (0.14) *
Former smoker[c]			0.73 (0.40) +	0.00 (0.15)			1.47 (0.36) ***	-0.04 (0.13)
Never smoker[c]			1.33 (0.39) ***	0.06 (0.11)			1.42 (0.42) ***	-0.02 (0.14)
Physical activity			-0.60 (0.16) ***	0.06 (0.05)			-0.60 (0.17) ***	0.03 (0.06)
Baseline BMI				0.92 (0.01) ***				0.96 (0.02) ***
Constant	24.13 (0.35) ***	34.42 (2.10) ***	33.58 (2.07) ***	2.99 (0.80) ***	26.50 (0.39) ***	27.48 (2.47) ***	27.05 (2.43) ***	2.46 (0.90) **
N (observations)	4057	4057	4057	4057	2309	2309	2309	2309
N (individuals)	1791	1791	1791	1791	1012	1012	1012	1012
R^2	.053	.081	.098	.784	.029	.043	.069	.804

*** p<.001; ** p<.01; * p<.05; † p<.10 (two-tailed tests)

Notes: BMI = Body mass index.

[a] Omitted group is those who were continuously in a relationship.

[b] Omitted group is whites.

[c] Omitted group is current smokers.

Table 4.3. Unstadardized Coefficients (Standard Errors) from Oridnary Least Squares Regression Models of BMI on Relationship Stress for Individuals Continuously in a Relationship

	Women				Men			
	Model 1	Model 2	Model 3	Model 4	Model 1	Model 2	Model 3	Model 4
Stress 1	1.20 (0.84)	0.78 (0.80)	0.81 (0.78)	0.88 (0.47) †	-0.32 (0.60)	-0.43 (0.62)	-0.52 (0.62)	0.16 (0.23)
Stress 2	0.68 (0.36) †	0.55 (0.35)	0.57 (0.35)	0.08 (0.13)	0.03 (0.35)	0.02 (0.34)	0.00 (0.34)	-0.02 (0.12)
Stress 3[a]	-ref-	-ref-	-ref-	-ref-	-ref-	-ref-	-ref-	-ref-
Stress 4	1.21 (0.63) †	0.92 (0.60)	0.74 (0.56)	0.49 (0.25) *	0.68 (0.74)	0.71 (0.71)	0.58 (0.68)	0.14 (0.25)
Age at basline	0.03 (0.01) *	0.00 (0.02)	-0.01 (0.02)	-0.03 (0.01) ***	-0.02 (0.01) †	0.00 (0.02)	-0.01 (0.02)	-0.02 (0.01) ***
Years between waves	0.33 (0.05) ***	0.43 (0.05) ***	0.43 (0.05) ***	0.16 (0.03) ***	0.28 (0.04) ***	0.31 (0.06) ***	0.31 (0.06) ***	0.08 (0.03) *
Black[b]	3.13 (0.56) ***	2.49 (0.56) ***	2.29 (0.57) ***	0.20 (0.20)	0.14 (0.48)	-0.12 (0.51)	-0.15 (0.52)	0.11 (0.13)
Hispanic[b]	1.18 (0.90)	0.15 (0.98)	0.10 (0.96)	0.20 (0.23)	0.39 (0.79)	-0.05 (0.85)	-0.47 (0.85)	-0.15 (0.21)
Education, years		-0.25 (0.09) **	-0.24 (0.09) **	-0.04 (0.03)		-0.11 (0.09)	-0.12 (0.09)	0.01 (0.02)
Household income		-0.97 (0.26) ***	-0.93 (0.26) ***	0.01 (0.10)		-0.10 (0.32)	-0.10 (0.32)	-0.09 (0.10)
Kids < 18		-0.29 (0.47)	-0.39 (0.47)	0.01 (0.16)		1.33 (0.46) **	1.20 (0.45) **	0.40 (0.17) *
Former smoker[c]			0.23 (0.55)	-0.14 (0.17)			1.40 (0.41) ***	-0.02 (0.16)
Never smoker[c]			1.28 (0.53) *	0.06 (0.15)			1.34 (0.49) **	-0.10 (0.15)
Physical activity			-0.65 (0.22) **	0.13 (0.07) †			-0.80 (0.20) ***	-0.04 (0.07)
Baseline BMI				0.94 (0.02) ***				0.96 (0.02) ***
Constant	23.38 (0.43) ***	37.27 (2.74) ***	36.33 (2.72) ***	2.32 (1.10) *	26.53 (0.44) ***	27.82 (3.02) ***	27.34 (2.98) ***	2.24 (0.96) *
N (observations)	1896	1896	1896	1896	1586	1586	1586	1586
N (individuals)	915	915	915	915	714	714	714	714
R^2	.051	.094	.115	.812	.024	.041	.071	.823

*** p<.001; ** p<.01; * p<.05; † p<.10 (two-tailed tests)

Notes: BMI = Body mass index.

[a] Omitted group is most the prevalent category of stress.

[b] Omitted group is whites.

[c] Omitted group is current smokers.

Table 4.4. Unstadardized Coefficients (Standard Errors) from Oridnary Least Squares Regression Models of BMI on Relationship Support for Individuals Continuously in a Relationship

	Women				Men			
	Model 1	Model 2	Model 3	Model 4	Model 1	Model 2	Model 3	Model 4
Support 1	0.78 (0.54)	0.69 (0.53)	0.67 (0.52)	0.13 (0.20)	-0.64 (0.67)	-0.46 (0.65)	-0.65 (0.69)	0.28 (0.26)
Support 2	0.72 (0.39) †	0.71 (0.39) †	0.73 (0.38) †	0.35 (0.16) *	-0.03 (0.53)	-0.05 (0.52)	-0.08 (0.50)	-0.06 (0.16)
Support 3[a]	-ref-	-ref-	-ref-	-ref-	-ref-	-ref-	-ref-	-ref-
Support 4	0.70 (0.42) †	0.60 (0.40)	0.69 (0.40) †	0.17 (0.15)	-0.64 (0.42)	-0.54 (0.42)	-0.60 (0.42)	-0.08 (0.13)
Age at basline	0.03 (0.01) *	0.00 (0.02)	-0.01 (0.02)	-0.03 (0.01) ***	-0.03 (0.01) +	0.00 (0.02)	-0.02 (0.02)	-0.02 (0.01) ***
Years between waves	0.32 (0.05) ***	0.42 (0.05) ***	0.43 (0.05) ***	0.16 (0.03) ***	0.29 (0.04) ***	0.31 (0.06) ***	0.31 (0.06) ***	0.08 (0.03) *
Black[b]	3.16 (0.56) ***	2.48 (0.56) ***	2.26 (0.57) ***	0.23 (0.20)	0.27 (0.46)	-0.01 (0.49)	-0.03 (0.50)	0.10 (0.14)
Hispanic[b]	1.18 (0.90)	0.11 (0.97)	0.05 (0.96)	0.19 (0.23)	0.40 (0.83)	-0.05 (0.88)	-0.48 (0.87)	-0.14 (0.21)
Education, years		-0.27 (0.09) **	-0.25 (0.09) **	-0.05 (0.03) †		-0.10 (0.09)	-0.10 (0.09)	0.00 (0.02)
Household income		-0.97 (0.26) ***	-0.93 (0.26) ***	-0.01 (0.10)		-0.11 (0.33)	-0.10 (0.32)	-0.09 (0.10)
Kids < 18		-0.28 (0.47)	-0.39 (0.47)	0.01 (0.16)		1.30 (0.45) **	1.14 (0.43) **	0.40 (0.16) *
Former smoker[c]			0.21 (0.55)	-0.14 (0.17)			1.42 (0.41) ***	-0.01 (0.16)
Never smoker[c]			1.29 (0.54) *	0.06 (0.15)			1.36 (0.49) **	-0.09 (0.15)
Physical activity			-0.67 (0.22) **	0.11 (0.07)			-0.84 (0.20) ***	-0.03 (0.08)
Baseline BMI				0.94 (0.01) ***				0.97 (0.02) ***
Constant	23.15 (0.47) ***	37.27 (2.75) ***	36.21 (2.70) ***	2.51 (1.11) *	26.90 (0.55) ***	28.06 (3.12) ***	27.62 (3.06) ***	2.29 (0.95) *
N (observations)	1899	1899	1899	1899	1590	1590	1590	1590
N (individuals)	915	915	915	915	714	714	714	714
R^2	.049	.093	.116	.811	.026	.042	.074	.824

*** p<.001; ** p<.01; * p<.05; † p<.10 (two-tailed tests)

Notes: BMI = Body mass index.

[a] Omitted group is the most prevalent category of support.

[b] Omitted group is whites.

[c] Omitted group is current smokers.

Table 4.5. Unstadardized Coefficients (Standard Errors) from Oridnary Least Squares Regression Models of BMI on Relationship Stress or Support for Individuals Who Exited a Relationship or Who Entered a Relationship

	Stress				Support			
	Women		Men		Women		Men	
	Exiters	Enterers	Exiters	Enterers	Exiters	Enterers	Exiters	Enterers
Category 1	1.08 (0.78)	-2.09 (0.70) **	-1.05 (1.10)	1.14 (0.74)	0.07 (0.61)	0.73 (0.70)	0.19 (0.76)	-0.04 (0.99)
Categroy 2	0.63 (0.55)	-1.10 (0.64) †	0.38 (0.45)	-0.49 (0.65)	-0.11 (0.53)	1.13 (0.72)	-0.02 (0.63)	-0.57 (0.68)
Category 3[a]	-ref-	-ref-	-ref-	-ref-	-ref-	-ref-	-ref-	-ref-
Category 4	-0.50 (0.52)	-0.07 (0.77)	0.35 (0.74)	0.54 (0.81)	-0.31 (0.69)	0.31 (0.59)	0.27 (0.68)	-0.16 (0.60)
Age at basline	-0.06 (0.02) **	-0.05 (0.02) +	-0.05 (0.02) *	-0.03 (0.02)	-0.05 (0.02) **	-0.06 (0.02) *	-0.03 (0.02) *	-0.03 (0.02)
Years between waves	0.14 (0.11)	0.20 (0.13)	0.06 (0.12)	0.29 (0.12) *	0.15 (0.10)	0.19 (0.13)	0.04 (0.11)	0.28 (0.11) *
Black[b]	1.35 (0.54) *	-0.33 (0.56)	-0.36 (0.93)	-0.26 (0.62)	1.11 (0.51) *	-0.38 (0.57)	0.10 (0.77)	-0.02 (0.58)
Hispanic[b]	0.11 (1.07)	-1.17 (1.03)	-0.81 (0.85)	0.25 (0.65)	0.27 (1.06)	-1.03 (1.09)	-0.70 (0.75)	0.15 (0.68)
Education, years	-0.16 (0.13)	0.02 (0.11)	-0.08 (0.09)	-0.06 (0.09)	-0.20 (0.14)	0.04 (0.10)	-0.01 (0.07)	-0.04 (0.10)
Household income	0.23 (0.22)	-0.42 (0.30)	0.20 (0.30)	0.24 (0.29)	0.30 (0.21)	-0.29 (0.33)	0.20 (0.34)	0.10 (0.30)
Kids < 18	-1.04 (0.72)	-1.46 (0.53) **	-0.72 (0.58)	1.17 (0.59) *	-1.18 (0.76)	-1.55 (0.52) **	-0.23 (0.47)	0.92 (0.60)
Former smoker[c]	0.17 (0.50)	0.35 (0.53)	0.80 (0.63)	-0.36 (0.69)	0.10 (0.48)	0.21 (0.52)	0.70 (0.51)	-0.25 (0.70)
Never smoker[c]	1.15 (0.84)	-0.18 (0.62)	-0.42 (0.71)	0.38 (0.56)	0.91 (0.80)	-0.06 (0.59)	-0.83 (0.57)	0.45 (0.57)
Physical activity	0.31 (0.19) †	-0.18 (0.24)	0.18 (0.26)	0.18 (0.27)	0.35 (0.18) †	-0.13 (0.26)	0.12 (0.27)	0.35 (0.27)
Baseline BMI	0.77 (0.04) ***	0.92 (0.06) ***	0.88 (0.09) ***	0.88 (0.06) ***	0.77 (0.04) ***	0.95 (0.06) ***	0.96 (0.07) ***	0.90 (0.06) ***
Constant	6.23 (2.50) *	8.02 (3.44) *	2.61 (4.44)	1.72 (3.20)	6.15 (2.41) *	5.51 (3.73)	-0.70 (3.17)	2.85 (3.44)
N (observations)	335	175	112	99	334	178	111	101
N (individuals)	333	171	110	98	332	174	109	99
R^2	0.684	0.753	0.774	0.794	0.695	0.747	0.813	0.797

*** p<.001; ** p<.01; * p<.05; † p<.10 (two-tailed tests)

Notes: BMI = Body mass index.

a Omitted group is the most prevalent category of stress or support.

b Omitted group is whites.

CHAPTER V

CONCLUSION

This dissertation extends the literature on obesity by examining emergent disparities in BMI trajectories and by investigating the association between adult BMI and life course experiences of socioeconomic disadvantage and the structure and quality of social relationships in adulthood. With this research I sought to, (1) obtain a more detailed picture of changes in BMI disparities during a period of rapid growth in obesity rates, and (2) gain a better understanding of the social experiences that shape weight gain trajectories over the adult life course. Below I synthesize findings from the three previous chapters, discuss the limitations of this research, and consider the broader implications of the findings presented in this dissertation.

In Chapter II, I utilized longitudinal data from the American's Changing Lives (ACL), spanning 15 years from 1986 to 2001/2001, to investigate racial and socioeconomic disparities in individual BMI growth trajectories. Whereas prior studies of BMI disparities, and health disparities research in general, has investigated dimensions of inequality separately, I used a multiplicative framework that incorporates the interaction of race, gender, socioeconomic position, and age to examine the full extent of disparities in BMI growth over time. Drawing on the theory of Intersectionality, I hypothesized that

fully multiplicative models would better detail the extent of social disparities in BMI trajectories, compared to the more typical additive approach used in prior studies.

The findings confirm some of the results found in prior work on disparities in obesity change, but also yield important new insights. I found complex interactive effects of age, gender, race, and class, which manifest powerful effects of intersectionality. In particular, black women with medium to high education and low to medium income levels experienced substantially larger increases in BMI over time, while white men with high education or high income levels experienced the least growth. Increasing disparities were evident primarily among adults aged 25-39, the ages most predictive of future changes in adult trajectories of obesity. This contrast between lower class black women and upper class white men exemplifies the premise of the theory of intersectionality, which posits that individuals are arrayed on a spectrum of inequality with the least advantaged (i.e. lower class black women) at one end and the most advantaged (i.e. upper class white men) at the opposite end. The unequal distribution of weight gain over time provides an empirical illustration of how the most socially disadvantaged groups often bear the negative consequences of social phenomenon, such as rising population obesity. These findings highlight the sociological and health importance of investigating changes in obesity disparities intersectionally by race, gender, class, and age.

After documenting the increasing disparities in BMI over time, I shifted to an examination of the social factors that contribute to those BMI trajectories. In Chapter III, I used 15 years of adult BMI history and retrospective reports of early-life socioeconomic conditions from the ACL to assess the role of early-life SEP in shaping adult BMI trajectories among women. The primary aim of this study was to explain how early life

matters for adult BMI trajectories. In this study I focused on several existing theoretical models of life course processes – Pathway Model, Latency Model, Cumulative Disadvantage Model – to determine which model, or models, best explain the mechanisms linking early-life SEP to adult BMI. I also examined the extent to which life course SEP accounts for the large racial and ethnic disparities in BMI trajectories among U.S. women.

I found that early-life SEP is positively associated with both baseline BMI and BMI change, though the effects of early-life are mediated heavily through adult SEP. The findings confirm that early-life socioeconomic experiences have enduring effects on adult weight and weight gain. I conclude that the latency model of life course processes best represents the pathway by which specific early-life socioeconomic conditions affect adult BMI trajectories, because this model allows for early-life socioeconomic conditions to operate via adult socioeconomic attainment and also to have direct effects on health in later-life. I also found evidence for cumulative effects of socioeconomic disadvantage on adult BMI trajectories. The findings showed that early-life disadvantage interacts with later-life disadvantage to produce higher baseline BMI, though it is not associated with BMI change. This indicates that adult BMI increases with increasing exposure to socioeconomic disadvantage, but that the effect is stronger when disadvantage is higher in both early- and later-life. A secondary aim of this study was to determine if early-life SEP accounted for the large racial and ethnic differences in BMI trajectories among women. I found that early life socioeconomic position accounted for some of the observed black/white difference in baseline BMI and fully accounted for baseline differences between Hispanic and white women. However, there was no effect of early-

life SEP on BMI change. Relatively little of the large black/white gap in baseline BMI was explained by life course SEP, adult family structure, or adult health behaviors and the difference in BMI growth actually increased after accounting for these factors. Overall, these findings suggest that early–life may be a significant period during which adult weight trajectories are established.

In Chapter IV, I focused on the influence of relationship transitions and quality on adult BMI. Using the ACL, I first assessed the effect of wave-to-wave changes in relationship status on BMI. I then examined the influence of relationship quality, characterized by levels of stress and social support, on subsequent BMI. Based on prior empirical studies of relationship status and BMI I expected to find that, compared to remaining in a relationship, exiting a relationship will result in a lower BMI and entering a relationship will result in a higher BMI. I sought to challenge the majority position in the existing literature on relationships and health that relationships are universally beneficial to individuals. Furthermore, I wanted to demonstrate that the benefits of relationships depend largely on the quality of those relationships. Drawing from theoretical perspectives on the importance of relationship stress and support, I hypothesize that individuals who experience more stress in their relationships will have a higher BMI and that those who report high levels of spousal/partner positive social support will have a lower BMI.

The findings confirm my hypothesis that relationships negatively influence BMI. I found that, compared to continuously being in a relationship, exiting from a relationship is related to lower BMI and entering a relationship is related to higher BMI. In contrast to prior studies, I found these effects for both men and women. These findings suggest that

the health benefits of relationships are not universal and at least do not extend to BMI. I also found that individuals who remained in a relationship characterized by high levels of stress had higher BMI and that although individuals who enter into a relationship tend to gain weight, entering less stressful relationships was associated with lower BMI. I did not find any effects of stress on BMI for those who exited a relationship. In addition, I did not find any consistent pattern of effects of social support on BMI.

Effects of relationship quality were found for women only. This is consistent with prior research that suggests that the quality of relationships is more salient to the well-being of women than men, because women tend to internalize relationship stressors in a more physiologically harmful manner. Thus, gender differences in appraisal of relationship quality and physiological response to stress may explain why BMI is related to relationship quality only among women.

This study, the first to assess the impact of relationship transitions and relationship quality on BMI, shows that the health promoting effects of being in relationships, and staying in relationships, may not extend to all health outcomes. Whether conceptualized as a health behavior, a health risk factor, or a health outcome, obesity is a condition that is not necessarily improved by being in a relationship, though this may depend on the quality of the relationship.

In Chapter II, I highlight the issue of increasing racial and socioeconomic disparities in BMI. Although findings from Chapters II and IV illustrate the importance of social experiences in determining adult weight gain, these factors did not fully account for the large racial differences in BMI observed in Chapter II. Baseline differences in BMI were somewhat explained by differential early-life and relationship experiences, but

the disparities in BMI growth were not at all explained by these factors. It is possible that while these factors are important determinants of adult BMI, they are not significantly differentially distributed according to race. Future sociological inquiry into the source of obesity inequality should focus on identifying social causes that are unequally distributed across racial/ethnic groups. For example, recent research showing that neighborhood environments, which are highly socially stratified, influence diet, physical activity, and obesity represents a promising area of inquiry.

The findings presented in this dissertation have important implications for the future state of health disparities in the U.S. and for improving our understanding of obesity as a social phenomenon. Increasing racial and socioeconomic disparities in obesity may foreshadow future increases in disparities in other related chronic health conditions, such as heart disease and diabetes, which are already unequally distributed in the population. Furthermore, lower SES black women, who already experience negative consequences related to race and class inequality, are increasingly at risk for exposure to additional negative experiences (e.g., institutional and interpersonal discrimination) related to weight. Chapters III and IV underscore the problems associated with placing responsibility for being overweight or obese solely on the individual, without regard for differences in people's past and current experiences. Placing the responsibility for weight status and weight gain entirely with the individual ignores the importance of social structures and interpersonal interactions in determining weight gain.

With this dissertation, I sought to further develop a Sociology of Obesity that conceptualizes obesity as a social condition that is, (1) distributed in the population along existing dimensions of inequality (i.e., race, gender, class, and age), that (2) develops

throughout the life course, and (3) is partly shaped by experiences in important social settings. Although it is contested whether the current state of obesity in the U.S. constitutes an epidemic (defined in a medical sense) or merely a moral panic (Flegal 2006; Campos et al. 2006; Lobstein 2006; Saguy and Riley 2005; Boero 2007), it is clear that obesity has both social causes and social consequences. In this debate, sociologists take the position that obesity is more of a socially construct than a real medical condition. Regardless of whether it is truly a disease or merely a social construct, obesity affects people on a deeply personal level. Sociologists are well-positioned to develop a body of knowledge focused on how obesity came to be a social issue and how the social processes that led to the current state of obesity interact with biological and behavioral processes (Crossley 2004).

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