

RACE, ETHNICITY AND THE SOCIAL CONTEXT OF DISADVANTAGE AND ITS
LINKS TO OBESITY AND PHYSICAL ACTIVITY IN ADOLESCENCE AND THE
TRANSITION TO ADULTHOOD

Hedwig Lee

A dissertation submitted to the faculty of the University of North Carolina at Chapel Hill in
partial fulfillment of the requirements for the degree of Doctor of Philosophy in the
Department of Sociology.

Chapel Hill
2009

Approved by:

Kathleen Mullan Harris

Penny Gordon-Larsen

Gunag Guo

Peggy Thoits

Shiriki Kumanyika

ABSTRACT

HEDWIG LEE: Race, Ethnicity and the Social Context of Disadvantage and its Links to Obesity and Physical Activity in Adolescence and the Transition to Adulthood
(Under the direction of: Kathleen Mullan Harris)

My dissertation investigates the relationship between social disadvantage and obesity physical activity outcomes using the National Longitudinal Study of Adolescent Health (Add Health). Using a dynamic longitudinal model, I examine the impact of social disadvantage in adolescence on patterns of obesity and physical activity (PA) across the transition to young adulthood, a period when adult lifestyle behaviors are solidified and major racial/ethnic and socioeconomic disparities become apparent. The highest rates of obesity occur among the most disadvantaged population groups, racial and ethnic minorities, and those with the highest poverty rates and the least education. I draw on a life course framework and the ecological model of human development to conceptualize the mechanisms of social disadvantage that link race/ethnicity and low SES to obesity and low levels of PA. I use data from three waves of Add Health to track obesity and PA trajectories from adolescence (WII: ages 13-19 y.) into adulthood (WIII: ages 19-26 y.) and to measure factors at the individual, family, peer, school and neighborhood levels in adolescence (WI and WII) that shape these trajectories. My three specific aims investigate the role of social disadvantage in the social contexts of young people's lives and determine if they serve as mechanisms through which race/ethnic and SES disparities operate. These mechanisms include: (1) cumulative risk (CR) indices, which refers to the number of risk factors that exists in a child's social environment

with the assumption that disadvantage is related to the accumulation of risk factors rather than a singular exposure; (2) Multilevel socioeconomic disadvantage—disadvantage present in the peer, school, and neighborhood context (e.g., proportion of peers/school/neighborhood with parents who have no high school diploma) and (3) parenting styles and practices. This dissertation contributes to social science research on racial and ethnic and socioeconomic health disparities by utilizing dynamic, interconnected and multilevel conceptualizations of the environment to study obesity and physical activity outcomes. In addition, by identifying alterable mechanisms operating in the social environments of disadvantaged populations that produce obesity and PA disparities, this research can inform policies and interventions aimed at eliminating them.

For Dad, with all my love

ACKNOWLEDGEMENTS

To the casual observer, a doctoral dissertation may appear to be solitary work. However, to complete a project of this magnitude requires a network of support, and I am indebted to many people. I am most especially grateful to my parents, Rotan E. Lee and Graciela Cabret Lee, and my advisor, Kathleen Mullan Harris, for their guidance, support and extraordinary courage.

TABLE OF CONTENTS

LIST OF TABLES	ix
LIST OF FIGURES	xi
Chapter	
I. INTRODUCTION	1
Health Significance of Obesity and Physical Activity in Adolescence and Young Adulthood	5
Disparities in Obesity and Physical Activity and Linkages to Social Disadvantage	6
Life Course Theory and the Ecological Model	7
Conceptual Model	10
Data	12
Longitudinal Analytic Design	14
Organization of My Dissertation	15
References	17
II. USING CUMULATIVE RISK MODELS TO LINK SOCIAL DISADVANTAGE TO OBESITY RISK IN THE TRANSITION TO ADULTHOOD	26
The Cumulative Risk Model	29
Research Questions	36
Conceptual Model	38
Data	38

	Measures	40
	Analytical Approach	54
	Results	57
	Discussion and Conclusion	65
	References	70
III.	MULTIPLE LEVELS OF SOCIAL DISADVANTAGE AND ITS LINKS TO OBESITY RISK IN ADOLESCENCE AND YOUNG ADULTHOOD	95
	Theoretical Framework	98
	Conceptual Model	103
	Specific Aims of Analysis	104
	Data	105
	Measures	107
	Analytical Design	115
	Analytical Methods	116
	Results	119
	Discussion and Conclusion	126
	References	131
IV.	THE INFLUENCE OF PARENTING PRACTICES AND BEHAVIORS IN THE RELATIONSHIP BETWEEN SOCIAL DISADVANTAGE AND PHYSICAL ACTIVITY IN THE TRANSITION TO YOUNG ADULTHOOD	148
	Previous Research on Parenting and Physical Activity	152
	The Influence of Disadvantage and Race/Ethnicity on Parenting Practices	154
	The Mediating Role of Parental Practices	157

Conceptual Model	162
Data	163
Measures	164
Analytical Approach	172
Results	174
Discussion and Conclusion	180
References	187
V. CONCLUSION	204

LIST OF TABLES

Table

2.1 Variable Descriptions, Means and Standard Deviations by Sex	83
2.2 Associations Between Risk Measures and Obesity Status at Waves II and III by Sex (Bivariate Odds Ratios)	85
2.3 Variable Means and Standard Deviations for Cumulative Risk Index and Distributions by Sex	87
2.4 Variable Means and Standard Deviations for Cumulative Risk Index and Distributions by Sex	88
2.5 Mean Cumulative Risk Level by Sex, Race/Ethnicity and Welfare/Poverty Status	89
2.6 Mean Cumulative Risk Level by Sex, Race/Ethnicity and Welfare/Poverty Status	90
2.7 Relationship between Cumulative Risk Index and Obesity Outcomes for Females (N=3,371)	91
2.8 Relationship between Cumulative Risk Index and Obesity Outcomes for Males (N = 3,122)	92
2.9 Estimated Multinomial Odds Ratios for Obesity Trajectories for Female Sample (N=3,371)	93
2.10 Estimated Multinomial Regression Odds Ratios for Obesity Trajectories for Male Sample (N=3,122)	94
3.1. Variable Descriptions, Means and Standard Deviations by Sex	142
3.2. Associations Between Disadvantage Measures and Obesity Status at Waves II and III by Sex	143
3.3. Mean Contextual Family, Peer, School and Neighborhood Disadvantage by Race and Sex	144

3.4. Mean Contextual Peer, School and Neighborhood Disadvantage by Family Disadvantage and Sex	145
3.5. Multilevel Logistic Regression Results for Obese at Wave II and Wave III for Females (N=3,926)	146
3.6. Multilevel Logistic Regression Results for Obese at Wave II and Wave III for Males (N=3,622)	147
4.1. Variable Descriptions, Means and Standard Deviations by Sex	200
4.2. Mean Parenting Measures by Race/Ethnicity (N=8,109)	201
4.3. Mean Parenting Measures by Sex	201
4.4. Mean Parenting Measures by Socioeconomic Status (Poverty/Welfare and Parent Education High School or Less)	201
4.5. Associations Between Disadvantage Measures, Parenting Measures and Physical Inactivity at Waves II and III (Bivariate Odds Ratios)	202
4.6. Multinomial Logistic Regression Models for Change in Physical Activity (Odd Ratio) (N=8,109)	203

LIST OF FIGURES

Figure

1.1 Conceptual Model	25
2.1 Conceptual Model	82
3.1 Conceptual Model	141
4.1 Conceptual Model	199

INTRODUCTION

The striking disparities in burden of illness and death experienced by racial and ethnic minorities, despite improvements in the overall health of the nation, are a serious public health concern facing our country. The Department of Health and Human Services (DHHS 2006), Centers for Disease Control (CDC 2006) and National Institutes of Health (NIH 2000), among other organizations, have all highlighted the need to reduce and eventually eliminate these troubling disparities. Growing racial and ethnic differences in the prevalence of obesity and the morbidity and mortality due to obesity have become a major focus in eliminating health disparities¹.

The highest rates of obesity occur among the most disadvantaged population groups, racial and ethnic minorities, (Flegal et al. 2002; Ogden et al. 2002) and those with the highest poverty rates and the least education (Drewnowski and Specter 2004; Schoenborn et al. 2002; DHHS 2000). Disparities in overweight/obesity trends between both racial and ethnic minorities and whites and the poor and non-poor, appear early in childhood and increase dramatically during adolescence and in the transition to adulthood (Gordon-Larsen et al. 2004a; Harris et al. 2006; McTigue et al. 2002; Ogden et al. 2002; Serdula et al. 1993). In addition, racial and ethnic minorities are disproportionately poor with low levels of education (Iceland 2003), compounding their disadvantage. Racial and ethnic disparities in patterns of

¹ The term “health disparity” has multiple definitions without a clear consensus on its meaning (Carter-Pokras and Baquet 2002). This research uses the National Institutes of Health definition where health disparities are defined as: “...differences in the incidence, prevalence, mortality, and burden of diseases and other adverse health conditions that exist among specific population groups in the United States. Research on health disparities related to socioeconomic status is also encompassed in the definition” (NIH 2000).

physical activity, a key determinant of obesity, are also evident, with ethnic minorities increasingly engaging in less physical activity than whites through adolescence and the transition to adulthood (Gordon-Larsen et al. 2000; 2002; 2004b; Harris et al. 2006). Because adolescence is a critical period when lifestyle and health-related behaviors are established and because the transition period from adolescence to young adulthood is a time of high risk for the development of obesity (Gordon-Larsen et al. 2004a; McTigue et al. 2002), understanding the role social disadvantage plays in influencing obesity and physical activity is critical to understanding racial and ethnic health disparities in these outcomes.

The goal of this dissertation is to examine how social disadvantage relates to obesity and physical activity in adolescence and the transition to young adulthood and how social disadvantage plays a role in the production of health disparities. Measures of race/ethnicity and low individual/family socioeconomic status (SES) have traditionally been used to indicate social disadvantage. In this research, the life course (Elder 1998) and ecological model of human development (Bronfenbrenner 1979) are utilized as overarching theoretical frameworks to guide the conceptualization of dynamic and comprehensive mechanisms of social disadvantage that link race/ethnicity and low SES to obesity and physical activity. These theoretical frameworks recognize that social disadvantage is a complex, multidimensional and dynamic construct that is comprised of diverse factors (e.g., economic resources, education, prestige, power), operating at different levels (e.g., individual, family, peer, school, neighborhood), and working through different causal pathways (e.g., via direct causal effects, determining vulnerabilities or exposures) to affect health outcomes (Bravemen et al. 2005). In addition, these multiple aspects of disadvantage interact dynamically and co-occur to determine health trajectories over time (Boyce et al. 1998; Bronfenbrenner and Ceci

1994).

This dissertation is organized as three separate research articles. Across all articles, the overarching question of interest focuses on the relationship between social disadvantage and obesity and physical activity outcomes in the transition to adulthood. These analyses allow for the investigation of the complex and dynamic relationship between social disadvantage and obesity and physical activity from adolescence and in to young adulthood by using data from three waves (1995, 1996, 2001) of the National Longitudinal Study of Adolescent Health (Add Health). Add Health is a longitudinal, nationally-representative population-based study that includes large samples of blacks, Hispanics and Asians. In addition, Add Health contains an extensive amount of data on multiple domains of social context (e.g. parent-child-relationships and peer networks) measured at multiple levels (e.g. family, school and community) and over time. This research has three specific aims:

Aim 1: To examine the relationship between social disadvantage and obesity in the transition from adolescence into young adulthood employing cumulative risk models.

Cumulative risk models measure the number of risk factors that exists in a child's social environment with the assumption that social disadvantage is related to the accumulation of multiple risk factors rather than a singular exposure (Rutter 1983). The concept of cumulative risk, as an approach to measuring disadvantage, has been heavily used in the human development literature, but has not been used to study obesity risk. These analyses (1) identify measures of risk (e.g. unhealthy diet patterns, neighborhood crime) that increase the likelihood of obesity; (2) identify population subgroups, defined by race/ethnicity, sex and poverty status, that face the highest number of identified risks; (3) determine if risk factors operate in a cumulative manner such that the risk of obesity in adolescence and young

adulthood and the risk of becoming obese from adolescence into young adulthood increases, as the number of factors in a cumulative risk index (defined below) increases; and (4) assess whether cumulative risk operates to explain race/ethnic and poverty disparities in the risk of becoming obese during the transition from adolescence into young adulthood.

Aim 2: To examine the role that multiple levels of social context play in the relationship between social disadvantage and obesity in adolescence and young adulthood employing multivariate multilevel modeling. Research consistently demonstrates that health is

influenced by the social and physical environments in which individuals live (Bravemen et al. 2005; Glass and McAtee; Halfon and Hochstein 2002; Taylor and Repetti 1997). In addition, social disadvantage operates at different levels of social context (e.g. family, peers, school and neighborhood) and interdependently. Multilevel models (Raudenbush and Bryk 2002a; 2002b) can capture these multiple levels of social disadvantage simultaneously.

These analyses (1) create measures of socioeconomic disadvantage at the individual, family, peer, school and neighborhood levels; (2) determine how contextual disadvantage varies by race/ethnicity, sex and poverty status; (3) investigate the bivariate relationships between each level of contextual disadvantage and risk of obesity; and (4) investigate the unique influence of each level of disadvantage on the risk of obesity in adolescence and adulthood using multivariate multilevel modeling.

Aim 3: To better understand the relationship between social disadvantage and physical activity by investigating the mediating role of parenting practices during adolescence between poverty in adolescence and physical activity patterns in the transition from adolescence into young adulthood. Parent-child interactions are an important aspect of a child's social environment and have profound direct and indirect influences on children's

development and health behaviors well into adolescence (Baumrind 1991; Birch and Fisher 1998; Collins et al. 2000; Harris et al. 1998; Harris and Marmer 1996; NRC 2004).

Parenting styles and practices differ by race/ethnicity (García Coll and Pachter 2002; Steinberg et al. 1992; Wu et al. 2003) and SES (Elder et al. 1995; Lareau 2002). The effectiveness of parenting styles and behaviors on child outcomes may also differ across cultural and socioeconomic groups (García Coll and Pachter 2002; Darling and Steinberg 1993). These analyses (1) create measures of parenting behaviors; (2) determine how parenting behaviors differ by race/ethnicity and SES; (3) investigate the relationships between different types of parenting behavior and physical activity patterns; and (4) determine if parenting practices play a mediating role in the relationship between SES and racial/ethnic minority status and physical activity patterns from adolescence and into young adulthood.

Health Significance of Obesity and Physical Activity in Adolescence and Young Adulthood

The prevalence of obesity in the U.S. has risen dramatically in both children and adults (Mokdad et al. 1999; 2000; 2001; Strauss and Pollack 2001) and continues to increase (Mokdad et al. 2003). Within a period of two decades the prevalence of adolescent overweight has tripled and the prevalence of young adult obesity has nearly doubled (Ogden et al. 2002). In addition, recent research comparing prevalence of overweight and obesity among adolescents and adults between 1999 and 2004 show no indication that prevalence rates are decreasing (Hedley et al. 2004; Wang and Beydoun 2007). Overweight in adolescence is strongly linked to obesity in adulthood (Guo and Chumela 1999; Parsons et al. 1999; Whitaker et al. 1997). These observations are also evidenced in the Add Health data.

The incidence of obesity increased dramatically between adolescence and young adulthood; at Wave II, 10.6% of adolescents were overweight/obese and only 6 years later at Wave III, this number doubled to 22.1% of the young adults in the sample. These trends are troubling given that obesity has been linked to numerous morbidity outcomes, such as type 2 diabetes, kidney disease, coronary heart disease, congestive heart failure, stroke, and osteoarthritis and even death (Choudhary et al. 2007; Mokdad et al. 2004; Must et al. 1999; Willet et al. 1999).

Physical activity and inactivity are inextricably linked with obesity. Although the origins of obesity are complex, a simplified model of weight gain shows that it is the result of inadequate physical activity (energy expenditure) to balance food consumption (energy intake). Over the past few decades there has been an overall decline in physical activity among the U.S. population, partly due to increasing sedentary behavior (Adams 2006; Brownson et al. 2005; CDC 2001; 2005). There is a near universal decline in physical activity with age, with the steepest decline occurring between the ages of 13 and 18 (Sallis 2000). Data from Add Health indicate that patterns of physical activity track from adolescence into young adulthood, as levels of physical activity decrease and inactivity increase (Gordon-Larsen et al. 2004b; Harris et al. 2006).

Disparities in Obesity and Physical Activity and Linkages to Social Disadvantage

Disparities in overweight/obesity risk and physical activity behavior are evident in many segments of the population based on race/ethnicity, sex and SES (Brownson et al. 2005; Crawford et al. 2001; Flegal et al. 2002; Ogden et al. 2002; DHHS 2001). For example, although overweight/obesity is observed in all population groups, obesity is particularly common among African American, American Indian, Hispanic and Pacific

Islander women (NWLC 2004). Within gender and socioeconomic groups racial/ethnic disparities in physical activity and obesity persist and grow with age, with minorities generally facing higher risks of obesity and engaging in lower levels of physical activity than whites (Gordon-Larsen et al. 2004b, Harris et al. 2006; Winkelby et al. 1993). What is missing in the research that documents these disparities are the underlying mechanisms that explain these differences. Because race and gender, although social constructs (ASA 2003; Braun 2002; Mays et al. 2003; Ore 2002), are ascribed characteristics of an individual, it is important to understand what other factors related to these characteristics contribute to obesity and physical activity. SES, especially poverty status, has been used as a possible explanation for racial/ethnic and gender differences (Crawford et al. 2001; LaVeist 2005). However, we need a clearer conceptualization of social disadvantage that accounts for the many associated risks faced by the poor and how the contexts and experiences of disadvantage might differ by race/ethnicity and gender. This research contributes substantially to the literature on health disparities by understanding how social disadvantage plays a role in contributing to rises in obesity and declines in physical activity and disparities in these outcomes. A primary focus of this research uses the Add Health data to conceptualize mechanisms of disadvantage in multiple ways that link race/ethnicity and low SES to obesity and physical activity guided by the life course and ecological paradigms.

Life Course Theory and the Ecological Model

Life course theory (Elder 1998) and the ecological model of human development (Bronfenbrenner 1979) serve as overarching frameworks to understand the influence of social disadvantage on obesity trajectories from adolescence into young adulthood. Both

perspectives have developed into dominant theoretical paradigms for studying adolescence and the transition to adulthood (Bronte-Tinkew et al. 2005; Elder 1997; Shanahan 2000; Smetana, Campione-Barr and Metzger 2006) as well as health development (Ben-Schlomo and Kuh 2000; Halfon and Hochstein 2002; Lynch and Smith 2005) and disparities (Pearlin et al. 2005; Reifsnider, Gallagher and Forgione 2005). Each paradigm contains important principles that are highly related to each other yet unique to each theory, that guide how social disadvantage and its influence on obesity risk and behavior are conceptualized in this analysis.

Themes from the Life Course Paradigm

Four central themes define the life course paradigm (Elder 1998). The first theme advances the idea that individuals are connected via a network of shared relationships that are interdependent across generations. A good example of this principle are the relationships between parents and children. The second theme concentrates on the sequencing and timing of life events, which can be viewed as a trajectory. The ordering of events can determine subsequent states and risks. For example, overweight in childhood increases the risk of obesity in adulthood (Dietz 1998; Reilly et al. 2003). In addition, trajectories have the potential to build momentum from reinforcing circumstances. The third theme calls attention to human agency in choice making. Individuals, although influenced by their social world, actively make decisions within the constraints of their social, historical and economic context, family backgrounds and stage in the life course. The fourth theme particularizes the usefulness of studying individuals in their historical context. Time specific events and social and cultural norms have direct ramifications on an individual's current and future well being

and behavior.

Themes from the Ecological Paradigm

The ecological model of human development is also described by four major components (Bronfenbrenner and Morris 1998; Lerner 2005). The first component maintains that humans develop through a process of dynamic interaction between themselves and their social context. Similar to the life course theme of human agency in choice-making, individuals serve to shape and be shaped by their environment as they develop. The second component emphasizes that each individual's biological, cognitive, emotional and behavioral characteristics are influential in shaping the course of his/her development. The third component conceptualizes the context of human development as nested levels or systems, with bi-directional influences within and between systems. Each level of social context represents an increasingly larger level of the social environment and all levels operate together to influence development. The *microsystem* is the innermost level and represents the immediate social settings of the individual (e.g., family, school, peer group and neighborhood). The *mesosystem* represents linkages between these immediate environments (e.g., a child's home and school). The *exosystem* can be described as an external setting that indirectly affects development (such as parent's workplace). The *macrosystem* involves the larger cultural context (e.g., national economy and politics). Similar to the life course theme of "linked lives," the ecological model emphasizes proximal processes, enduring interactions that occur between individuals in the immediate environment, as the primary engine of development. The fourth component of this model, referred to as the *chronosystem*, emphasizes the importance of multiple conceptualizations of time including the order of

events in their historical sequence and context. These themes are also similar to conceptualizations of time in life course theory.

Shared Themes

The life course and ecological models both describe how individuals develop in the context of their social, historical and cultural environments and across time and generations. Both theories capture stability and change in life course transitions and trajectories, such as the transition to adulthood and stability and change in obesity risk over time. Both theories maintain that individuals are influenced by all levels of organization of human life and that these ecologies are complexly and dynamically linked, such as the multiple levels and cumulative nature of disadvantage that exist for the poor (Evans 2004). Both theories emphasize the importance of shared relationships as an important influence of human development, such as the critical role of parent-child relationships in adolescent development (Smetana et al. 2006). Although public health researchers call for dynamic, interconnected and multilevel conceptualizations of the environment in relation to health outcomes (Braveman et al. 2005; Committee on Evaluation of Children's Health 2004; Halfon and Hochstein 2002; Glass and McAtee 2006), few researchers have utilized these approaches to study health outcomes and especially racial/ethnic and socioeconomic health disparities in obesity and physical activity.

Conceptual Model

I use one overarching conceptual model to inform and link the three dissertation articles (See Figure 1.1). The conceptual model draws from the life course and ecological

frameworks. The fundamental assumption of this model is that an individual's obesity and levels of physical activity are dynamic (changeable over time) and affected by multiple aspects of his/her social environment. In Figure 1.1, an individual's family, peers, school and neighborhood represent overlapping environments that influence individual obesity and physical activity, both independently and additively, over time, from adolescence and into young adulthood. These overlapping environments represent aspects of an individual's social world which take place within the political, economic and cultural constraints of the nation. This model is derived from the Committee on Evaluation of Children's Health (2004) conceptualization of the dynamic process of multiple, interacting influences on the evolution of children's health.

Three major features of this model are linked to conceptualizations of social disadvantage in relation to obesity and physical activity over time, and map closely with the three aims of the dissertation research. The first feature is that multiple aspects of disadvantage co-occur to affect obesity risk. While each aspect of the social disadvantage may be individually important, these factors usually do not operate alone, but co-occur over time in the form of *cumulative risks*—which are fundamentally additive. The second feature is that *multiple levels* of disadvantage operate simultaneously and interact over time to influence obesity risk (i.e., social disadvantage in one context increases the effect of social disadvantage in another context). The third feature is that social disadvantage operates through various conditions of the social environment. An especially important context is the family and the *parenting styles and practices* that occur within this context and through which social disadvantage may operate to influence physical activity.

These three features drawn from the life course and ecological models serve to guide

conceptualizations of social disadvantage in the research. Although obesity is the result of complex interactions between multiple genetic and environmental factors (Faith and Kral 2006; Glass and McAtee 2006), analyses focus on the complex interactions of the social environments in which individuals are embedded as a baseline that will permit further investigation of complex biosocial interactions. Genetic factors are thought to contribute substantially to the predisposition toward obesity. However, the sharp increase in population body weight, which has occurred in only a few decades, cannot be explained by genetics alone (Koplan and Dietz 1999; NIH 2004). Understanding racial/ethnic and socioeconomic obesity and physical activity disparities requires a better understanding of the social environments that place individuals at risk for obesity and low levels of physical activity and how these environments systematically differ by race/ethnicity and SES.

Data

I use data from the National Longitudinal Study of Adolescent Health (Add Health) throughout this dissertation. Add Health is a longitudinal, nationally representative, school-based study of ethnically diverse U.S. adolescents in grades 7 to 12 (ages 12 to 19 years). It was designed to explore the causes of health-related behaviors, with an emphasis on the influence of social context. Wave I (WI) occurred during 1994-1995 and involved an in-school and in-home administration of surveys. For the in-school administration, a nationally representative sample of 80 U.S. high schools and 52 middle schools was selected using a stratified cluster design and an In-School Questionnaire was administered to every student attending these schools on a particular day during the 1994-95 school year [N= 90,118]. The in-school administration provides unique data on school context given it represents a census

of all students in each school. In a second level of sampling, adolescents were selected from the school rosters using a gender- and grade-stratified design for an extensive in-home interview in 1995 [N=20,745]. A parent was also interviewed in WI [N= 17,700]. At Wave II (WII: during 1996) adolescents in grades 7-11 at Wave I were followed up with a second in-home interview (seniors at Wave I were not followed up as part of the Add Health design) [N=14,738]. In Wave III (WIII) all WI respondents (including the seniors left out of the eligible sample at WII), now aged 18-26 years (2002) were followed [N=15,197]. Response rates are relatively high for a prospective cohort: WI, 78.9%; WII, 88.2%; and WIII, 77.4% completed in-home interviews. 10,828 respondents completed all three waves of in-home interviews.

A major strength of Add Health is the extensive measures of SES, health, and race/ethnicity and the ability to create measures at multiple levels of social context. In the In-School Questionnaire students were asked to nominate up to 5 male and 5 female friends by locating and recording friends' student IDs listed on the school roster. Because the in-school sample included the entire student population, with nearly all children in the school interviewed, the identification numbers of nominated friends can be linked back to their own in-school questionnaire and characteristics of a respondent's peer group can be determined, such as its racial/ethnic makeup. In a similar fashion, school-level measures can be derived by aggregating the responses of the In-School Questionnaire for all students in their respective schools. In addition, contextual data containing information on the characteristics of the neighborhoods and communities in which Add Health sample members lived in Waves I and II have been linked to individual-level records.

These analyses use data from the WI In-School, In-Home and Parent Questionnaires

and contextual data, as well as the follow-up WII and WIII surveys. These analyses are therefore limited to adolescents who participated in all three waves of the study and have complete measured height and weight data at WII and WIII. Exclusions include seriously disabled respondents and pregnant females, since pregnancy affects weight and may result in obesity missclassification. Given the preceding restrictions, the approximate sample size for most analyses is 8,000 respondents.

Longitudinal Analytic Design

The analytical design is derived from the conceptual model (See Figure 1.1). Longitudinal data from three waves of Add Health are used to measure obesity and physical activity trajectories from adolescence and into young adulthood and to measure factors operating during adolescence that serve to influence these trajectories. Outcomes of interest, represented by the square boxes in Figure 1.1, include trajectories of change in obesity and physical activity patterns from WII (adolescence) to WIII (young adulthood), in addition to static measures of obesity at WII and WIII.² Factors influencing these patterns of change are measures of individual, family, peer, school and neighborhood context at WI and WII, in addition to ascribed social characteristics such as race/ethnicity and sex, measured at WI. This design exploits the longitudinal data and incorporates the temporal order of effects (i.e. factors operating during adolescence prior to the transition to adulthood), which is a necessary first step in establishing a causal relationship. A longitudinal design also allows for the measurement of change and continuity of factors operating in adolescence, and the influences these have on obesity and physical activity outcomes.

One of the major goals of my dissertation is to consider multiple ways to

² WI obesity is not used because height and weight were self-reported at WI.

conceptualize what it means to be disadvantaged (i.e., cumulative risks, multiple levels of social context) in adolescence and its relationship to obesity and physical activity outcomes. The primary ways that disadvantage is measured is through the use of the multiple economic measures available in the Add Health data including: parental income; family poverty status; parental education, and family usage of public assistance prior to the respondent turning 18. A second major goal is to examine whether family processes (i.e., parental practices and behaviors) operate as intervening mechanisms that help to explain the relationship between poverty and physical activity outcomes. The accomplishment of these goals lead to a better understanding of the social environments that place individuals at risk for obesity and lowered levels of physical activity.

Organization of My Dissertation

In Chapter 2, I examine the relationship between social disadvantage and obesity in the transition from adolescence into young adulthood employing cumulative risk models. The cumulative risk model assumes that it is the accumulation of risk factors across a variety of domains, rather than a single risk factor that is important in adversely impacting the developmental outcomes of individuals. As the number of risk factors increase, regardless of the specific risk factor, child development becomes increasingly compromised. In this chapter the focus is on the idea that multiple risk factors co-occur in the lives of disadvantaged persons and have additive effects on obesity outcomes (independent of their unique influences on obesity) and should be modeled to account for these characteristics. This analysis focus on risk factors at mainly the family and neighborhood level.

In Chapter 3 the focus shifts to examining the effects of social disadvantage at

multiple levels on health outcomes under the assumption that individuals are influenced by factors and forces beyond their family and neighborhood environments. I examine the role that multiple levels of social context (i.e., family, peers, school and neighborhood) play in the relationship between social disadvantage and obesity in adolescence and young adulthood employing multivariate multilevel modeling.

In Chapter 4, I take a more micro-level view focusing on the influence of parenting practices and behaviors on their children's behavior. I examine the relationship between social disadvantage and obesity by investigating the mediating role of parenting practices during adolescence in the relationship between social disadvantage in adolescence and physical inactivity patterns in the transition from adolescence into young adulthood.

By exploiting these rich data available in Add Health, this dissertation contributes to understanding obesity and physical activity disparities in four ways: 1) by identifying the multiple risks that the disadvantaged face and constructing parsimonious measures to capture these risks; 2) by measuring disadvantage at multiple levels of social context; 3) by linking disadvantage to parental practices and behaviors; and 4) by determining whether these multiple approaches of conceptualizing social disadvantage provide new insights and knowledge about racial/ethnic disparities in obesity and physical activity. This dissertation contributes to social science research on racial and ethnic and socioeconomic health disparities by utilizing dynamic, interconnected and multilevel conceptualizations of the environment to study obesity and physical activity outcomes. In addition, by using a longitudinal framework, I can capture change in health behaviors and health outcomes and sort out how adolescent social context impacts obesity and physical activity outcomes in the transition to adulthood.

References

- Adams, Jean. 2006. "Trends in Physical Activity and Inactivity Amongst US 14-18 Year Olds by Gender, School Grade and Race, 1993-2003: Evidence from the Youth Risk Behavior Survey." *BMC Public Health* 6: 57.
- American Sociological Association (ASA). 2003. *The Importance of Collecting Data and Doing Social Scientific Research on Race*. Washington, DC: American Sociological Association. Retrieved September 21, 2006 (http://www.asanet.org/galleries/default-file/asa_race_statement.pdf).
- Baumrind, Diana. 1991. "The Influence of Parenting Style on Adolescent Competence and Substance Use." *Journal of Early Adolescence* 11(1):56-95.
- Ben-Shlomo, Yoav and Diana Kuh. 2002. "A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges and Interdisciplinary Perspectives." *International Journal of Epidemiology* 31: 285-293.
- Birch, Leann L. and Jennifer O. Fisher. 1998. "Development of Eating Behaviors Among Children and Adolescents." *Pediatrics* 101:539-549.
- Boyce, W. Thomas, Ellen Frank, Peter S. Jensen, Ronald C. Kessler, Charles A. Nelson and Laurence Steinberg. 1998. "Social context in Developmental Psychopathology: Recommendations for Future Research from the MacArthur Network on Psychopathology and Development." *Development and Psychopathology* 10(1):143-165.
- Braun, Lundy. 2002. "Race, Ethnicity, and Health: Can Genetics Explain Disparities?" *Perspectives in Biology and Medicine* 45(2): 159-174.
- Braveman, Paula A., Catherine Cubbin, Susan Egerter, Sekai Chideya, Kristen S. Marchi, Marilyn Metzler, Samuel Posner. 2005. "Socioeconomic Status in Health Research: One Size Does Not Fit All." *Journal of the American Medical Association* 294(22): 2879-2888.
- Bronfenbrenner, Urie. 1979. *The Ecology of Human Development: Experiments by Nature and Design*. Cambridge, MA: Harvard University Press.
- Bronfenbrenner, Urie and Stephen J. Ceci. 1994. "Nature-Nurture Reconceptualized in Developmental Perspective: A Bioecological Model." *Psychological Review* 101(4):568-586.
- Bronfenbrenner, Urie and Pamela A. Morris. 1998. "The Ecology of Developmental Processes." Pp.992-1028 In *Handbook of Child Psychology: Vol. 1: Theoretical Models of Human Development, 5th Edition*, edited by W. Damon and R. M. Lerner, New York, NY: Wiley.
- Bronte-Tinkew, Jacinta, Brett Brown, Jennifer Carrano and Rebecca Shwalb. 2005. "Logic Models and Outcomes for Youth in the Transition to Adulthood: Report to the DC Children

and Youth Investment Trust Corporation.” Washington, D.C.: Child Trends, Inc., Retrieved September 21, 2006 (www.childtrends.org/Files/Transition_to_Adulthood_Report-FINAL.pdf).

Brownson, Ross C., Tegan K. Boehmer and Douglas A. Luke. 2005. “Declining Rates of Physical Activity in the United States: What Are the Contributors?” *Annual Review of Public Health* 26: 421-443.

Carter-Pokras, Olivia and Claudia Baquet. 2002. “What is a “Health Disparity.” *Public Health Reports* 117: 426-434.

Centers for Disease Control (CDC). 2006. “Eliminating Racial and Ethnic Health Disparities.” Atlanta, GA: Centers for Disease Control, Retrieved September 21, 2006 (<http://www.cdc.gov/omh/AboutUs/disparities.htm>).

Centers for Disease Control and Prevention (CDC). 2005. “Trends in Leisure-Time Physical Inactivity by Age, Sex, and Race/Ethnicity --- United States, 1994—2004.” *Morbidity and Mortality Weekly Report* 54(39): 991-994.

Centers for Disease Control and Prevention (CDC). 2001. “Physical Activity Trends --- United States, 1990--1998.” *Morbidity and Mortality Weekly Report* 50(9):166-169.

Choudhary, Arabinda K., Lane F. Donnelly, Judy M. Racadio and Janet L. Strife. 2007. “Diseases Associated with Childhood Obesity.” *American Journal of Roentgenology* 188:1118-1130.

Collins, W. Andrew, Eleanor E. Maccoby, Laurence Steinberg, E. Mavis Hetherington and Marc H. Bornstein, Marc H. 2000. “Contemporary Research on Parenting: The Case for Nature and Nurture.” *American Psychologist* 5:218-232.

Committee on Evaluation of Children's Health, National Research Council. 2004. *Children's Health, the Nation's Wealth: Assessing and Improving Child Health*. Washington, DC: The National Academies Press.

Crawford, Patricia B., Mary Story, May C. Wang, Lorrence D. Ritchie and Z. I. Sabry. 2001. “Ethnic Issues in the Epidemiology of Childhood Obesity.” *Childhood and Adolescent Obesity* 48(4): 855-878.

Darling, Nancy and Laurence Steinberg. 1993. “Parenting Style as Context: An Integrative Model.” *Psychological Bulletin* 113(3): 487-496.

Dietz, William H. 1998. “Health Consequences of Obesity in Youth: Childhood Predictors of Adult Disease.” *Pediatrics* 101:518-525.

Drewnowski, Adam and S.E. Specter. 2004. “Poverty and Obesity: the Role of Energy Density and Energy Costs.” *American Journal of Clinical Nutrition* 79:6-16.

Elder, Glen H., Jr. 1998. "The Life Course as Developmental Theory." *Child Development* 69:1-12.

Elder, Glen H., Jr. 1997. "The Life Course and Human Development," Pp. 939-991 In *Handbook of Child Psychology, Volume 1: Theoretical Models of Human Development*, edited by R. M. Lerner and W. Damon. New York: Wiley.

Elder, Glen H. Jr., Jacquelynne S. Eccles, Monika Ardel and Sarah Lord. 1995. "Inner-City Parents Under Economic Pressure: Perspectives on the Strategies of Parenting." *Journal of Marriage and the Family* 57(3): 771-784.

Evans, Gary W. 2004. "The Environment of Childhood Poverty." *American Psychologist*. 59(2):77-92.

Faith, Myles S. and Tanja V.E. Kral. 2006. "Social Environmental and Genetic Influences on Obesity and Obesity-Promoting Behaviors." Pp. 279-332 In *Genes, Behavior, and the Social Environment: Moving Beyond the Nature/Nurture Debate*. Edited by L.M. Hernandez and D.G. Blazer. Washington, DC: National Academies Press.

Flegal, Katherine M., Margaret D. Carroll, Cynthia L. Ogden and Clifford L. Johnson. 2002. "Prevalence and Trends in Obesity Among US Adults, 1999-2000." *Journal of the American Medical Association* 288(14): 1723-1727.

García Coll, Cynthia and Lee M. Patcher. 2002. "Ethnic and Minority Parenting." Pp. 1-20 in *Handbook of Parenting, 2nd Edition*, edited by M.H. Bornstein. Mahwah, NJ: Lawrence Erlbaum Associates.

Glass, Thomas A. and Matthew J. McAtee. 2006. "Behavioral Science at the Crossroads in Public Health: Extending Horizons, Envisioning the Future." *Social Science and Medicine* 62:1650-1671.

Gordon-Larsen, Penny, Linda S. Adair, Melissa C. Nelson and Barry M. Popkin. 2004a. "Five-Year Obesity Incidence in the Transition Period between Adolescence and Adulthood: The National Longitudinal Study of Adolescent Health." *American Journal of Clinical Nutrition* 80:569-575.

Gordon-Larsen, Penny, Linda S. Adair and Barry M. Popkin. 2002. "Ethnic Differences in Physical Activity and Inactivity Patterns and Overweight Status." *Obesity Research* 10:141-149.

Gordon-Larsen, Penny, Robert G. McMurray and Barry M. Popkin. 2000. "Determinants of Adolescent Physical Activity and Inactivity Patterns." *Pediatrics* 105(6), Retrieved September 21, 2006 (<http://pediatrics.aappublications.org/cgi/content/abstract/105/6/e83>).

Gordon-Larsen, Penny, Melissa C. Nelson and Barry M. Popkin. 2004b. "Longitudinal Physical Activity and Sedentary Behavior Trends: Adolescence to Adulthood." *American*

Journal of Preventive Medicine 27:277-283.

Guo, Shumei S. and William Cameron Chumlea. 1999. "Tracking of Body Mass Index in Children in Relation to Overweight in Adulthood." *American Journal of Clinical Nutrition* 70(1): 145S-148S.

Halfon, Neal and Miles Hochstein. 2002. "Life Course Health Development: An Integrated Framework for Developing Health, Policy, and Research." *The Milbank Quarterly* 80(3): 433-479.

Harris, Kathleen Mullan, Francesca Florey, Joyce W. Tabor, Peter S. Bearman, Jo Jones, and J. Richard Udry. 2003. The National Longitudinal Study of Adolescent Health: Research Design. Retrieved September 21, 2006 (<http://www.cpc.unc.edu/projects/addhealth/design.html>.)

Harris, Kathleen Mullan, Frank F. Furstenberg, Jr., and Jeremy K. Marmer. 1998. "Paternal Involvement with Adolescents in Intact Families: The Influence of Fathers over the Life Course." *Demography* 35:201-216.

Harris, Kathleen Mullan, Penny Gordon-Larsen, Kim Chantala and J. Richard Udry. 2006. "Longitudinal Trends in Race/Ethnic Disparities in Leading Health Indicators From Adolescence to Young Adulthood." *Archives of Pediatrics and Adolescent Medicine* 160:74-81.

Harris, Kathleen Mullan and Jeremy K. Marmer. 1996. "Poverty, Paternal Involvement, and Adolescent Well-Being." *Journal of Family Issues* 17:614-640.

Hedley, Allison A., Cynthia L. Ogden, Clifford L. Johnson, Margaret D. Carroll, Lester R. Curtin, Katherine M. Flegal. 2004. "Prevalence of Overweight and Obesity Among US Children, Adolescents, and Adults, 1999-2002." *The Journal of the American Medical Association* 291: 2847-2850.

Iceland, John. 2003. *Poverty in America: A Handbook*. Berkeley, CA: University of California Press.

Koplan, Jeffrey P. and William H. Dietz. 1999. "Caloric Imbalance and Public Health Policy." *The Journal of the American Medical Association* 282(16): 1579-1581.

Lareau, Annette. 2002. "Invisible Inequality: Social Class and Childrearing in Black Families and White Families." *American Sociological Review* 67(5): 747-776.

LaVeist, Thomas A. 2005. *Minority Populations and Health: An Introduction to Health Disparities in the United States*. New York, NY: Jossey-Bass.

Lerner, Richard M. 2005. "Foreword: Urie Bronfenbrenner: Career Contributions of the Consummate Developmental Scientist." Pp. ix-xxvi In *Making Human Beings Human:*

Biological Perspectives on Human Development edited by U. Bronfenbrenner, Thousand Oaks, CA: Sage Publications.

Lynch, John and George D. Smith. 2005. "A Life Course Approach to Chronic Disease Epidemiology." *Annual Review of Public Health* 26:1-35.

Mays, Vickie M., Ninez A. Ponce, Donna L. Washington and Susan D. Cochran. 2003. "Classification of Race and Ethnicity: Implications for Public Health." *Annual Review of Public Health* 24: 83-110.

McTigue Kathleen M., Joanne M. Garrett, Barry M. Popkin. 2002. "The Natural History of Obesity: Weight Change in a Large US Longitudinal Survey." *Annals of Internal Medicine* 136: 857-64.

Mokdad, Ali H., Barbara A. Bowman, Earl S. Ford, Frank Vinicor, James S. Marks and Jeffrey P. Koplan. 2001. "The Continuing Epidemics of Obesity and Diabetes in the United States." *The Journal of the American Medical Association* 286: 1195-1200.

Mokdad, Ali H., Earl S. Ford, Barbara A. Bowman, William H. Dietz, Frank Vinicor, Virginia S. Bales and James S. Marks. 2003. "Prevalence of Obesity, Diabetes, and Obesity-Related Health Risk Factors, 2001." *The Journal of the American Medical Association* 289: 76-79.

Mokdad , Ali H., James S. Marks, Donna F. Stroup and Julie L. Gerberding. 2004. "Actual Causes of Death in the United States, 2000" *The Journal of the American Medical Association* 291:1238-1245.

Mokdad, Ali H., Mary K. Serdula, William H. Dietz, Barbara A. Bowman, James S. Marks and Jeffrey P. Koplan. 2000. "The Continuing Epidemic of Obesity in the United States." *The Journal of the American Medical Association* 284:1650-1651.

Mokdad, Ali H., Mary K. Serdula, William H. Dietz, Barbara A. Bowman, James S. Marks and Jeffrey P. Koplan. 1999. "The Spread of the Obesity Epidemic in the United States, 1991-1998." *The Journal of the American Medical Association* 282: 1519-1522.

Must, Aviva, Jennifer Spadano, Eugenie H. Coakley, Alison E. Field, Graham Colditz, and William H. Dietz. 1999. "The Disease Burden Associated With Overweight and Obesity." *The Journal of the American Medical Association* 282: 1523-1529.

National Institutes of Health (NIH). 2000. "National Institutes of Health Strategic Plan to Reduce and Ultimately Eliminate Health Disparities: Fiscal Years 20002-2006." Bethesda, MD: National Institutes of Health, Retrieved September 21, 2006 (www.nih.gov/about/hd/strategicplan.pdf).

National Research Council and Institute of Medicine. 2002. *Community Programs to Promote Youth Development*. Edited by J. Eccles and J. A. Gootman. Washington, DC:

National Academy Press.

National Women's Law Center (NWLC). "Making the Grade on Women's Health: A National and State-By State Report Card." Washington D.C.: National Women's Law Center, Retrieved September 21, 2006 (www.nwlc.org/pdf/HRC04RacialDisparitiesFactSheet.pdf).

Ogden, Cynthia L., Katherine M. Flegal, Margaret D. Carroll and Clifford L. Johnson. 2002. "Prevalence and Trends in Overweight Among U.S. Children and Adolescents, 1999-2000." *Journal of the American Medical Association* 288(14): 1728-32.

Ore, Tracey. 2002. *The Social Construction of Difference and Inequality: Race, Class, Gender, and Sexuality, 2nd Edition*. Columbus, OH: McGraw Hill.

Parsons, T. J., C. Power, S. Logan and C. D. Summerbell. 1999. "Childhood Predictors of Adult Obesity: A Systematic Review." *International Journal of Obesity and Related Metabolic Disorders* 23(Suppl 8): S1-107.

Pearlin, Leonard I.; Scott Schieman, Elena M. Fazio, Elena M. and Stephen C. Meersman. 2005. "Stress, Health, and the Life Course: Some Conceptual Perspectives." *Journal of Health and Social Behavior* 46(2):205-219.

Raudenbusch, Stephen W. and Anthony S. Bryk. 2002a. "A Hierarchical Model for Studying School Effects." *Sociology of Education* 59: 1-17.

Raudenbusch, Stephen W. and Anthony S. Bryk. 2002b. *Hierarchical Linear Models: Applications and Data Analysis Methods, 2nd Edition*. Thousand Oaks, CA: Sage Publications.

Reifsnider, Elizabeth, Martina Gallagher and Bunny Forgione. 2005. "Using Ecological Models in Research on Health Disparities." *Journal of Professional Nursing* 21(4): 216-222.

Reilly, J. J., Methven, E. McDowell, Z.C., Hacking, B. Alexander, D. Stewart, L. and Kehler, C. J. H. 2003. "Health Consequences of Obesity." *Archives of Disease in Childhood*, 88:748-752.

Rutter, Michael. 1983. "Statistical and Personal Interactions: Facets and Perspectives." Pp. 295-319 in *Human Development: An Interactional Perspective*, edited by D. Magnusson and V. Allen. New York, NY: Academic Press.

Sallis, James F. 2000. "Age-Related Decline in Physical Activity: A Synthesis of Human and Animal Studies." *Medicine and Science in Sports and Exercise* 32: 1598-1600.

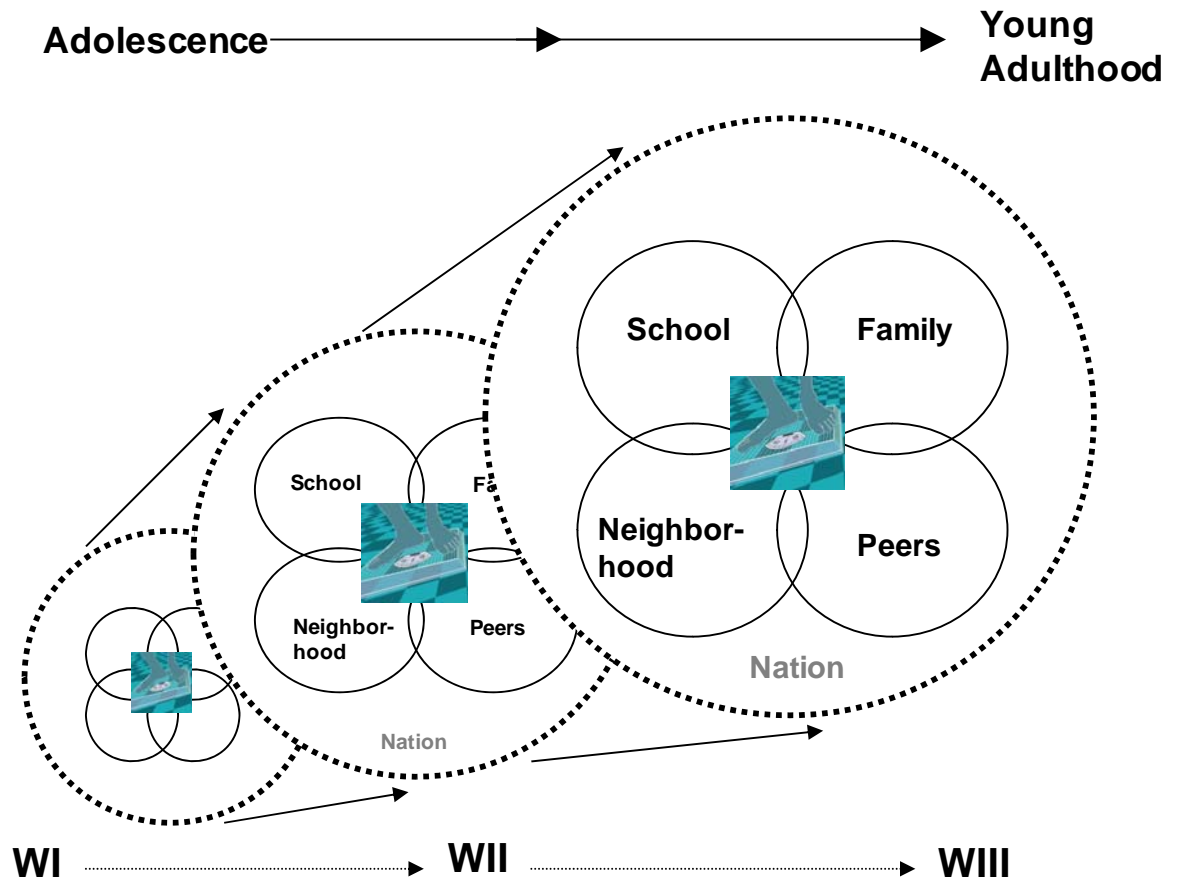
Schoenborn, Charlotte A., Patricia F. Adams, and Patricia M. Barnes. 2002. "Body Weight Status of Adults: United States, 1997-98." *Advance Data* 330:1-15.

- Serdula, Mary K., Donna Ivery, Ralph J. Coates, David S. Freedman, David F. Williamson and Tim Byers. 1993. "Do Obese Children Become Obese Adults? A Review of the Literature." *Preventive Medicine* 22:167-177.
- Shanahan, Michael J. 2000. "Pathways to Adulthood in Changing Societies: Variability and Mechanisms in Life Course Perspective." *Annual Review of Sociology* 26:667-692.
- Smetana, Judith G., Nicole Campione-Barr and Aaron Metzger. 2006. "Adolescent Development in Interpersonal and Societal Contexts." *Annual Review of Psychology* 57:255-284.
- Steinberg, Laurence; Sanford M. Dornbusch and B. Bradford Brown. 1992. "Ethnic Differences in Adolescent Achievement: An Ecological Perspective." 47(6):723-729.
- Strauss, Richard S. and Harold A. Pollack. 2001. "Epidemic Increase in Childhood Overweight, 1986-1998." *The Journal of the American Medical Association* 286: 2845-2848.
- Taylor, Shelley E. and Rena L. Repetti. 1997. "Health Psychology: What is an Unhealthy Environment and How Does It Get Under the Skin?" *Annual Review of Psychology* 48:411-447.
- U.S. Department of Health and Human Services (USDHHS). 2006. "Protecting the Health of Minority Communities." Washington, D.C.: U.S. Department of Health and Human Services, Retrieved September 21, 2006 (<http://www.hhs.gov/news/factsheet/minorityhealth.html>).
- U.S. Department of Health and Human Services (DHHS). 2001. "The Surgeon General's Call to Action to Prevent and Decrease Overweight and Obesity." Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General, Retrieved September 21, 2006 (<http://www.surgeongeneral.gov/topics/obesity/calltoaction/toc.htm>).
- U.S. Department of Health and Human Services (USDHHS). 2000. "Healthy People 2010, 2nd Ed." Washington: DC: US Government Printing Office.
- Wang, Youfa and May A. Beydoun. 2007. "The Obesity Epidemic in the United States—Gender, Age, Socioeconomic, Racial/Ethnic, and Geographic Characteristics: A Systematic Review and Meta-Regression Analysis." *Epidemiologic Reviews* 29:6-28.
- Whitaker, Robert C., Jeffrey A. Wright, Margaret S. Pepe, Kristy D. Seidel and William H. Dietz. 1997. "Predicting Obesity in Young Adulthood from Childhood and Parental Obesity." *The New England Journal of Medicine* 337(13): 869-873.
- Winkleby Marilyn A., Thomas N. Robinson, Jan Sundquist and Helena C. Kraemer. 1999. "Ethnic Variation in Cardiovascular Risk Factors among Children and Young Adults: Findings from the Third National Health and Nutrition Examination, 1988-1994." *The Journal of the American Medical Association* 281(11): 1006-1013.

Willett, Walter C., William H. Dietz and Graham A. Colditz. 1999. "Guidelines for Healthy Weight." *The New England Journal of Medicine* 341: 427-434.

Wu Ying, Bonita F. Stanton, Jennifer Galbraith, Linda Kaljee, Lesley Cottrell, Xiaoming Li, Carole V. Harris, Dawn D'Alessandri, and James M. Burns. 2003. "Sustaining and Broadening Intervention Impact: A Longitudinal Randomized Trial of 3 Adolescent Risk Reduction Approaches." *Pediatrics* 111(1):32-38.

Figure 1.1. Conceptual Model



USING CUMULATIVE RISK MODELS TO LINK SOCIAL DISADVANTAGE TO OBESITY RISK IN THE TRANSITION TO ADULTHOOD

The high rates of obesity and overweight that exist in the US, especially among poor and minority children and adolescents, continue to remain a national public health concern. Large racial/ethnic and socioeconomic disparities in overweight and obesity, add to the significant number of disparities in morbidity and mortality outcomes that exist between the poor and the nonpoor and whites and certain minority groups (i.e., African Americans, Native Americans and Latinos) (DHHS 2000; Kumanyika and Grier 2006; Miech et al. 2006). The highest rates of obesity occur among the most disadvantaged population groups, those with the highest poverty rates and the least education (Drewnowski and Specter 2004; Schoenborn, Adams and Barnes 2002; DHHS 2000). Although there is a general positive relationship between socioeconomic disadvantage and obesity (Flegal et al. 1998, Flegal et al. 2002)¹, what is less understood are the aspects of social disadvantage that contribute to the risk of obesity. In other words, although researchers know that being poor, black, and/or Hispanic places an individual at risk for obesity (Drewnowski and Specter 2004; Gordon-Larsen, Adair and Popkin 2003; Kimm et al. 1996; Patterson et al. 1997; Schoenborn et al. 2002), researchers are less clear about what factors place these population groups at a higher risk for obesity than other population groups.

There has been an extensive amount of research in both the social science and public health arenas that attempts to measure socioeconomic status (SES) (see Braveman et al. 2005

¹ The relationship between socioeconomic status and obesity varies by ethnicity and gender (Flegal et al. 2002; Kumanyika and Grier 2006).

for a review), as well as socioeconomic disadvantage (e.g., Brooks-Gunn and Duncan 1997; Duncan, Brooks-Gunn and Klebanov 1994; Duncan et al. 1998), especially as it relates to health and important child developmental outcomes. One theme that can be drawn from this research is that SES is a complex, multidimensional construct that is comprised of diverse factors (e.g., economic resources, education, prestige, power), operating at different levels (e.g., individual, family, neighborhood), and working through different causal pathways (e.g., via direct causal effects, determining vulnerabilities or exposures) (Braveman et al. 2005). Therefore, a better understanding of the relationship between socioeconomic disadvantage and obesity will entail capturing the many factors that comprise this construct.

The argument that SES (or disadvantage) is a complex, multidimensional construct is not new. The life course (Elder et al. 1995; Elder, Nguyen and Caspi 1985) and ecological perspectives (Bronfenbrenner 2005; Bronfenbrenner and Morris 1998) both contain arguments that social disadvantage should be measured at multiple ecological levels, over time, and via different processes. Capturing the complex and dynamic nature of the social context of disadvantage is especially important when studying the transition to adulthood (Elder 1997; Shanahan 2000) as well as health development, including the development of chronic diseases such as obesity (Ben-Shlomo and Kuh 2000; Halfon and Hochstein 2002; Lynch and Smith 2005). In addition, the transition period from adolescence to young adulthood has been shown to be a lifecycle period of particular risk for the development of obesity (Gordon-Larsen et al. 2004a; McTigue, Garrett and Popkin, 2002).

A related argument originating from the ecological perspective (Bronfenbrenner and Morris 1998; Dannefer 2003) and life course literature (Elder 1998), is the idea that social disadvantage may be cumulative. Social disadvantage is related to the accumulation of

multiple environmental and social risk factors rather than a singular exposure (Evans 2004; Rutter 1979; Sameroff 1987a). Socially disadvantaged groups may be at higher risk for adverse health outcomes because they are exposed to a larger proportion of adverse social and physical environmental conditions than advantaged groups (Evans 2004).

This chapter investigates the relationship between social disadvantage in childhood and adolescence and obesity trajectories from adolescence into young adulthood using cumulative risk models (e.g., Rutter 1979; Sameroff 1987a) and nationally representative data from the National Longitudinal Study of Adolescent Health (Add Health). These models have mainly been used in human development and epidemiological research examining child outcomes such as cognitive development, mental health and behavior problems (e.g., Appleyard et al. 2005; Jones et al. 2002; Rutter 1979; Sameroff 1987a). They have recently been used to investigate child health outcomes (Bauman, Silver and Stein 2006; Evans 2003). This will be the first time these models will be used to examine the relationship between social disadvantage and obesity. These specific aims of this chapter are to:

- (1) Identify measures of risk (e.g., unhealthy diet patterns, neighborhood crime) that increase the likelihood of obesity.
- (2) Identify population subgroups, defined by race/ethnicity, sex and poverty status, that face the highest number of identified risks.
- (3) Determine if risk factors operate in a cumulative manner such that the risk of obesity in adolescence and young adulthood and the risk of becoming obese from adolescence into young adulthood increases, as the number of factors in the cumulative risk index increases.

(4) Determine whether cumulative risk operates to explain race/ethnic and poverty disparities in the risk of becoming obese during the transition from adolescence into young adulthood.

Although, a large and growing body of research has investigated the relationship between SES and obesity among adults (e.g., Casas et al. 2001; Drewnowski and Specter 2004; Flegal et al. 1998; Kuczmarski et al. 1994; Must, Gortmaker and Dietz 1994; Paeratakul et al. 2002; Sobal and Stunkard 1989; Stunkard 1993; Sundquist and Johansson 1998; Zhang and Wang 2003), and among children and adolescents (e.g., Gibson 2004; Goodman 1999; Gordon-Larsen et al. 2003; Haas et al. 2003; Kimm et al. 1996; McMurray et al. 2000; Miech et al. 2006; Nelson, Chiasson and Ford 2004; Wang 2001; Winkleby et al. 1999), no research to date has employed the use of cumulative risk models to investigate this relationship.

The Cumulative Risk Model

A “risk factor” is a term used in epidemiology to define a characteristic that is either directly or indirectly associated with the occurrence of disease or other adverse health outcomes. Risk factors can be fixed at birth (such as sex and race) or acquired via the social and physical environment as a person proceeds through his/her life (such as exposure to violence). “Risk profiles” are produced by creating a multifactorial model that represents the interplay of these multiple fixed and acquired factors. These profiles can be used to identify vulnerable members of our population, on whom prevention strategies and disease treatment can be focused (Risch et al. 2002).

Human development and sociological research on disadvantage, more specifically the

ecological and life course paradigms, also emphasizes the idea that disadvantage is defined by multiple risks (Bronfenbrenner 2005; Bronfenbrenner and Morris 1998; Elder et al. 1985, 1995). Multiple risks co-occur across multiple domains of social context and accumulate over time in disadvantaged populations, with serious implications for future life chances and other developmental outcomes. The most disadvantaged individuals are those who are not only of low socioeconomic status measured in terms of family income or welfare receipt, but individuals who are also living in unstable families and poor and socially disorganized neighborhoods (Elder et al. 1985, 1995; Furstenberg et al. 1999; Gephart 1997; Wilson 1987).

The cumulative risk model assumes that it is the accumulation of risk factors across a variety of domains, rather than a single risk factor that is important in adversely impacting the developmental outcomes of children (e.g., Rutter 1979; Rutter and Quinton 1977; Sameroff 2000). As the number of risk factors increase, regardless of the specific risk factor, child development becomes increasingly compromised (Jones et al. 2002).

Cumulative risk models have their origin in human development and epidemiological research that investigates the effects of risk factors on child psychiatric disorders and cognitive development. The most notable studies that find evidence of cumulative risk on child outcomes are the Isle of Wight studies (Rutter 1979; Rutter et al. 1975, 1976) and the Rochester Longitudinal Study (Sameroff 2000; Sameroff et al. 1987a, 1987b, 1998).

Rutter and colleagues (1975) investigated the prevalence of mental disorders in 10-year old children in the Isle of Wight and the inner borough of London, England. They determined six risk factors within the family environment that significantly correlated with childhood psychiatric disorders including: (1) severe marital discord; (2) low social status;

(3) large family size or overcrowding; (4) paternal criminality; (5) maternal mental disorder and (6) foster placement. A direct association was found between the number of risk factors for child psychiatric disorder and the odds of a child having a clinical problem. While no single risk factor significantly increased risk for disorder, the presence of multiple risk factors contributed to increases in the likelihood of mental disorder in a linear fashion. In particular, a single risk factor did not considerably increase the risk of mental disorder in children, while the presence of two risk factors increased the likelihood of mental disorders four-fold and the presence of four risk factors increased the likelihood of mental disorders ten-fold. Rutter (1979) has argued that multiple risk factors “potentiated” each other so that the risk factors yielded much more of an impact when they were combined than when each risk factor was considered independently (i.e., an exacerbation effect).

Sameroff and colleagues (1987b) collected data over a 20-year period in Rochester, New York to investigate the development of a group of children from the prenatal period through their early childhood. The focus of the study was to investigate the impact of parental schizophrenia on child development. They identified ten environmental risk factors [(1) maternal mental disorder; (2) high maternal anxiety; (3) rigid parental attitudes, beliefs, and values about child development; (4) observations of few positive parent–child interactions; (5) unskilled occupational status; (6) low maternal educational status; (7) minority group status; (8) single parenthood as a measure of low family social support; (9) stressful life events; and (10) large family size] which were combined to create a multiple risk score for each child. Similar to Rutter’s study (1975), they found that the number of risk factors was associated with concurrent behavior problems in preschool (Sameroff et al. 1987b) and with later problem behavior, adolescent mental health and academic problems

(Sameroff et al. 1998).

More recent research on risks finds that cumulative risk is associated with numerous negative child outcomes in mental health, behavior problems and cognitive and language development (e.g., Appleyard et al. 2005; Atzaba-Poria, Pike and Deater-Deckard 2004; Burchinal et al. 2000; Carta et al. 2001; Deater-Deckard et al. 1998; Dekovic 1999; Garbarino and Kostelny 1996; Jones et al. 2002; Liaw and Brooks-Gunn 1994; Stanton-Chapman 2004). Two recent studies have also found associations between cumulative risk and child health outcomes, such as stress, measured by cardiovascular and neuroendocrine parameters, body fat and allostatic load (Evans 2003) and overall health, activity limitation and rates of chronic conditions (Bauman et al. 2006). These studies focused primarily on risk within the family context such as material poverty, single parenthood and low parental education. Evans (2003) also investigated physical (crowding, noise, housing quality) and psychosocial (child separation, turmoil, violence) aspects of the home environment. Taken together, these studies suggest that it is the number of factors in one's background, rather than a particular risk factor that potentially influences child development and health.

It should be noted that the concept of cumulative risk differs from the sociological concept of cumulative advantage/disadvantage. Unlike cumulative risk, cumulative advantage/disadvantage involves a time dimension where advantage/disadvantage accumulates over time compounding initial relative advantage or disadvantage associated with structural location. This process leads to a systematic divergence in life outcomes across individuals or groups over time (Dannefer 1987, 2003; Merton 1968; O'Rand 1996). In addition, cumulative advantage takes many forms and can be represented by multiple statistical and empirical models such as mathematical models for exponential growth

(DiPrete and Eirich 2006). Despite their dissimilarities, both concepts can be used to explain disparities and inequality between individuals and groups of individuals.

Advantages of Cumulative Risk Model

Cumulative risk models are an innovative way to measure social disadvantage as it relates to obesity transition for a number of reasons. As discussed earlier, contextual risk factors usually do not occur in isolation (Duncan et al. 1994, 1998; Evans 2004; Rutter 1979; Sameroff et al. 1987a, 1987b; Moore, Vandivere and Ehrle 2000). Contextual risk factors can be defined as aspects of an individual's environment that are associated with a higher likelihood of poor or negative outcomes such as compromised health. Disadvantaged children are usually exposed to multiple environmental risk factors rather than to a singular exposure (Evans 2004). Concentrations of social and physical risk are usually centered on the poor and ethnic minorities (Schell 1997). For example poverty, poor housing quality, unemployment, father absence, family turmoil, violence and stressful life events are highly interrelated (Evans 2004; Chase-Lansdale and Brooks-Gunn 1995; McLoyd 1990; Wilson 1987). Important health conditions may arise from cumulative risk exposure, especially for low-income populations, that may not be observed by focusing on a singular risk factor in isolation (Evans and Marcynyszyn 2004). In addition, human development is shaped by these multiple and interrelated contextual factors and personal relationships (Bronfrenbrenner and Morris 1998; Elder 1998).

A cumulative risk index is able to capture these multiple and interrelated elements of contextual risk in a simple yet comprehensive way. The measure is the number of risk factors that exists in a child's social environment. In addition, a cumulative risk index

sidesteps the problems that arise by using correlated or overlapping contextual factors in multiple regression models. Including moderate to highly correlated parameters as separate indicators in a regression model can lead to deflated parameter estimates, concealing the actual statistical associations of individual predictors with the outcome variable (Kutner, Nachtsheim and Neter 2004). Using cumulative risk models allows a researcher to take advantage of a set of comprehensive measures of social context at multiple levels (e.g., individual, family, neighborhood) and over time (i.e., multiple waves of data collection) that is provided in the Add Health data. This comprehensive cumulative risk index may yield the most complete estimate of overall risk for a socially disadvantaged child (Luthar 1993). The model also has the capacity to be used by researchers and clinicians to assess the number of risks an adolescent or child can “tolerate” (Jones et al. 2002) before their risk of negative outcomes, such as obesity, increases. In addition, research has shown cumulative risk indices serve as a good control variable or succinct covariate to represent risk, when social risk is a hypothesized moderator (Burchinal et al. 2000).

Disadvantages of the Cumulative Risk Model

There are some disadvantages to using cumulative risk models. The main criticisms stem from the way that the index is constructed. A dichotomous classification of risk exposure is determined for each person either by a statistical cutoff for continuous variables (e.g., upper quartile, greater than one standard deviation above the mean) or on the basis of conceptual categorization for categorical variables (e.g., minority status, below the poverty line). These multiple risk categories are then summed to calculate cumulative risk (Rutter 1983, 1993), regardless of their relative influence on the outcome.

Using this index assumes that environmental, social and personal risk factors are of equal weight and can simply be summed to represent the accumulated amount of adversity faced by an individual (Evans 2003; Burchinal et al. 2000). This stands in stark contrast to traditional multiple regression models, which assume that the impact of each risk factor on the outcome of interest is unique and unequal (Burchinal et al. 2000). However, a cumulative risk metric provides a more parsimonious representation of multiple and correlated risk factors that provides the same explanatory power in one variable as in the total of multiple individual risk factors (e.g., Evans 2003; Rutter 1983, 1993; Sameroff 1998).

An important limitation is that cumulative risk indices cannot render information about the mechanisms through which contextual risk leads to poorer child outcomes (Ackerman et al. 1999) and, therefore, cannot be utilized for intervention or prevention efforts targeted at high risk children (Jones et al. 2002). However, this index can be used to distinguish high-risk children from low-risk children. In addition, this index can be used as an indicator that can be tracked over time to indicate the trend in childhood risk for obesity, similar to indices used to track the risk of adverse child developmental outcomes (Moore et al. 2000). Lastly, this index can provide the public and policymakers with information about children whose social environments may predispose them to obesity risk.

Despite the fact that the cumulative risk model has consistently predicted negative child outcomes in previous research and may serve as a useful indicator of obesity risk, the weaknesses of the model should not be ignored. For exploratory purposes, similar to Burchinal and colleagues (2000), I will compare a model including each risk factor separately to a model including all risk factors together as an index, which will help me to gain insight on the utility of cumulative risk models in studying obesity outcomes.

Research Questions

Four research questions guide the analysis of this paper and inform the conceptual model (See Figure 2.1).² Because I apply a new measurement approach (cumulative risk indices) in the investigation of obesity in adolescence and its trajectory into young adulthood, the research questions are exploratory in nature.

Question 1: *What are the factors that place young people at risk for obesity?* This question will be answered in two steps. First, I will identify measures of risk at the individual, family and neighborhood level that have been discussed in the obesity literature as risk factors for obesity. To take advantage of the wide variety of measures available in the Add Health data, other risk factors discussed in the sociological and public health literature that have been hypothesized to be adversely related to child health will also be incorporated into the analysis. Second, I will perform bivariate analyses to test the relationship between the created risk measures and obesity in adolescence and young adulthood. Measures that are significantly related to obesity will be included in a cumulative risk index.³ A separate risk index will be created for neighborhood risk measures given that it represents a higher level and less proximate measure of social context.

Question 2: *Who faces the highest amounts of cumulative risk?* The poverty literature indicates that the poor and ethnic minorities (i.e., Hispanics and African Americans) face higher levels of contextual risk than the non-poor and whites, respectively (Evans 2004; Chase-Lansdale and Brooks-Gunn 1995; McLoyd 1990; Wilson 1987). It has also been shown that adult females are more likely to be poor than males (Spraggins 2003). To answer this research question I will compare the average level of cumulative risk among these

² A discussion of the conceptual model is provided in the following section.

³ I will also run cumulative risk models using all variables theoretically related to obesity to examine if the same pattern emerges.

population subgroups (i.e., male, female, black, Hispanic, white, poor, non-poor) and combinations of these population subgroups (e.g., black female, Hispanic male, poor white male, non-poor Hispanic female).

Question 3: *Do risk factors operate in a cumulative manner, such that higher levels of risk are associated with higher levels of obesity risk?* To answer this question, I will use logistic and multinomial logistic regression to determine the risk of obesity in adolescence and young adulthood and the risk of becoming or staying obese from adolescence into young adulthood, as the number of factors in the cumulative risk indices increases.

Question 4: *Do measures of cumulative risk mediate the relationship between race/ethnicity, poverty and obesity?* I explore if cumulative risk plays a role in the relationship between poverty (and ethnic minority status) and becoming or staying obese from adolescence and into young adulthood by including the cumulative risk measures as a mediating variables in a multivariate model (see conceptual model).

Answering these questions will help to determine the utility of cumulative risk models as an indicator of obesity risk in the transition from adolescence into young adulthood. This analysis also extends the literature in multiple areas of research. It expands the application of a measure of risk traditionally used in child development and mental health literature. It supports the growing literature suggesting new directions in measuring the complexity and multidimensionality of socioeconomic status when studying health disparities (e.g., Braveman et al. 2005). Lastly, by using cumulative risk as a mediating measure, I help to better explain the relationship between minority status and obesity and poverty status and obesity.

Conceptual Model

The conceptual model (see Figure 2.1) provides the basic relationship between poverty status (and racial/ethnic minority status) and change in obesity status from adolescence into young adulthood. Poverty status acts as a confounder in the relationship between race/ethnicity and obesity. In this conceptual model for longitudinal analysis of Add Health data, the two cumulative risk indices (one at the individual/family level and one at the neighborhood level) mediate the relationship between these traditional measures of disadvantage (i.e., race/ethnicity and poverty status) and change in obesity status. Data from all three waves in Add Health are utilized, where poverty status (and race/ethnicity) is measured in childhood and adolescence, risk is measured in adolescence (Waves I and II), and obesity trajectories are constructed from data in adolescence (Wave II) and young adulthood (Wave III). All controls are measured at Wave I, the first data point in adolescence. The individual, family and neighborhood level factors that will be used to construct the cumulative risk index, as well as the specific theories that help to motivate these factors, are discussed below. Overall, I expect a positive relationship between minority status and obesity, and poverty and obesity in adolescence and young adulthood. I expect that poverty status will attenuate the relationship between race/ethnicity and obesity and I expect both the race/ethnicity and poverty effects on obesity will be mediated by the cumulative risk index.

Data

Data come from the National Longitudinal Study of Adolescent Health (Add Health), which is an ongoing nationally representative, school-based study of adolescents in grades 7

to 12 that began in 1994. It was designed to explore the causes of health-related behaviors, with an emphasis on the influence of social context. In 1994 Add Health administered an In-School Questionnaire to every student attending school from a nationally representative sample of schools. A sample of 80 high schools and 52 middle schools from the U.S. was selected using a stratified cluster design. Using the school rosters, a gender- and grade-stratified sample⁴ was chosen for the In-Home Interview in 1995 (Wave I), given an average of eight months after the In-School Survey, and again in 1996 (Wave II). In Wave III (2001-02), Wave I In-Home respondents were re-interviewed.

A parent, generally the mother, was also interviewed in Wave I. In-home adolescent questionnaires were administered by computer-assisted personal-interview (CAPI), as well as computer-assisted self-interview (CASI) for more sensitive questions. Ultimately, 20,745 in-home interviews were completed in Wave I; 17,713 parents answered child specific questions and 17,669 answered parent specific questions (more than one child was interviewed in some households). 14,738 in-home interviews were completed in Wave II (the seniors in Wave I were not followed). In Wave III 15,197 eligible⁵ original Wave I respondents completed the survey. In Wave I (WI), the age of participants ranged from 12 to 20 years, in Wave II (WII) from 13 to 21 years and Wave III (WIII) from 18 to 26 years.

Over 70% of the schools originally selected for the survey participated. Of the adolescents sampled for the in-home questionnaires, 78.9% participated in WI. Parent interviews are available for 85% of these respondents. Of those eligible for participation in WII, 88.2% completed in-home interviews. Of those eligible for participation in WIII, 77.4% completed in-home interviews.

⁴ Each school provided a roster of all students enrolled. From the rosters and the pool of participants in the in-school survey, adolescents in grades 7 to 12 were sampled to participate in the in-home interview.

⁵ Eligible respondents were all Wave I participants living in the US who were not dead.

The fact that the data set is longitudinal and nationally representative, with extensive measures of socioeconomic status, health, race and ethnicity among other factors, makes it an ideal data set to investigate the relationship between disadvantage and obesity in adolescents. In addition, contextual data containing information on the characteristics of the neighborhoods and communities in which Add Health sample members lived in WI and WII have been linked to individual-level records. Harris and colleagues (2003) provides a more detailed description on the Add Health Study.

This study uses data from the Wave I In -Home and Parent Questionnaires as well as the follow-up Wave II and III surveys. This analysis is therefore limited to adolescents who participated in all three waves of the study, have completed Parent Questionnaires, and have complete measured height and weight data. Exclusions included seriously disabled respondents, pregnant females and racial and ethnic groups other than whites, blacks and Hispanics. After applying these data constraints and deleting the cases with missing data on covariates, the final study sample contains 6,493 (3,371 females and 3,122 males).⁶

Measures

Table 2.1 provides means and standard errors of all measures used in this analysis by sex.

Minority Status: Race and ethnicity are self-reported at WI. Although Add Health allows for rich detail in measures of race and ethnicity, I include only non-Hispanic whites, non-Hispanic blacks and Hispanics in this analysis. Research shows that blacks and Hispanics

⁶ Seniors at Wave I were not followed up in Wave II as part of the design of Add Health and thus the analysis sample is younger than at Wave I. Missing from the analysis sample are respondents who did not have a completed parent questionnaire (therefore, there are fewer immigrants in the sample).

are at a higher risk for obesity than whites and are more likely to be poor than whites. Asians do not share in this higher risk profile despite their minority status (Flegal et al. 2002). For these reasons and because of the overall focus of social disadvantage and the exploratory nature of this analysis, I limit the sample to these racial/ethnic groups. Race and ethnicity are measured in two ways. Dummy variables are used to designate black, Hispanic and white (reference group) respondents. I also explore the use of a dummy variable indicating if an individual is either black or Hispanic (minority) or white (non-minority). Twenty-six percent of total the sample is minority (14% are Non-Hispanic black and 12% are Hispanic).

Welfare/Poverty Status: Welfare/Poverty status is a dichotomous indicator of any welfare receipt before the age of eighteen or family income less than poverty level. This measure is constructed from data on the family's receipt of public assistance or welfare from WI and WII during adolescence in combination with a retrospective report at WIII on the receipt of welfare and public assistance prior to the age of eighteen. Using data from the WI Parent Questionnaire on reported annual income from 1994, family income is categorized as below poverty level if income was less than \$16,000 (roughly the poverty level for a family of four in 1994). I chose a welfare- and income-based measure of poverty over an only income-based measure due to the large proportion of missing data on income ($\approx 20\%$). Twenty-nine percent of the total sample received welfare prior to the age of eighteen and/or was living below poverty at WI. This measure is used as a proxy for economic disadvantage as opposed to social disadvantage.

Sex: A dummy variable is used to represent sex where '1' indicates female and '0' indicates male. This measure is constructed from responses in the WI In-Home Questionnaire and crosschecked with WII and WIII responses. I divide the full sample by sex for this analysis.

Previous research using the Add Health data has shown that there are strong sex differences in the relationship between poverty and obesity, where poverty effects are more important for females than for males (Lee, Harris and Gordon-Larsen 2008).

Urban Block Group: A dummy variable is used to represent if a respondent lives in an urbanized area at Wave I as defined by the Census Bureau.⁷ This measure serves as a control variable in the analysis.

Age: A continuous variable of self-reported age at WI (in years) is also used as a control variable in the analysis.

Risk Factors and Cumulative Risk Index Construction

The measures of risk used in this analysis are individual, family and neighborhood level measures that have been discussed in the sociological and public health literature as being theoretically or empirically linked to obesity or adverse child health outcomes. In order to be consistent with the cumulative risk literature (Appleyard et al. 2005), all risk variables are transformed into dichotomous variables to represent the presence or absence of the risk factor. For continuous variables, subjects who are in the top or bottom 20th to 30th percentile⁸, depending on the measure, are coded as ‘1’. These fairly conservative cutoffs are used to ensure the presence of risk. The measures found to have a statistically significant bivariate relationship with obesity (excluding measures of poverty and minority status) will then be summed in a cumulative risk index (CRI) used to calculate the level of cumulative risk for each respondent.

⁷ The urbanicity code distinguishes block groups that are in completely urbanized areas from those that have any individuals living outside urbanized areas, in rural farm or rural nonfarm locations. This measure is different from the census “urban” designation which also includes places outside urbanized areas of 2,500 or more persons

⁸ This cutoff serves as a rough guideline for the creation of risk measures.

$$\text{CRI} = \sum_{i=1}^n x_i = x_1 + x_2 + x_3 + \dots + x_n$$

Where x represents a statistically significant risk factor (with a value of ‘0’ or ‘1’) and n represents the number of statistically significant risk factors included in the cumulative risk index. A risk factor is equal to ‘1’ if the risk factor is present for the respondent and it is equal to ‘0’ if the risk factor is not present.

Two separate cumulative risk indices are created. The first index includes individual and family risk factors and the second index includes neighborhood risk factors. In addition, I chose to include measures that were significant at WII or WIII in my cumulative risk index for two main reasons. First, although I am interested in change in obesity status from adolescence and into young adulthood, I am also interested in the static life stage measures of obesity. It is important to establish if measures are salient in adolescence and/or young adulthood, before examining change in obesity status from adolescence and into young adulthood. I chose to include measures that were significant at only one of the two waves because many of the measures that are included are theoretically and empirically validated predictors of obesity. For sensitivity analysis, I also run models using risk indices that include all measures of obesity risk (that are theoretically related to obesity or have been shown to be associated with obesity in previous research), even if they are not significantly related to obesity at either wave.

Individual Level Risk Factors

I focus on individual level risk factors in the following domains: eating patterns, sleep and self-esteem. Skipping breakfast, especially in adolescence, increases the propensity to snack and graze on food with high energy densities during the day, which increases the total

fat and saturated fat as a percentage of total daily energy for individuals who skip breakfast compared to individuals who do not skip breakfast (Timlin et al. 2008). A growing number of epidemiological studies observe an association between short sleep duration and obesity (Gangwisch et al. 2005; Kohatsu et al. 2006; Patel et al. 2006; Reilly et al. 2005). Low levels of sleep have also been physiologically linked to heavier weight. Studies have demonstrated possible hormonal mechanisms that act via increased ghrelin and decreased leptin levels, which are positively linked with hunger and satiety, respectively (Spiegel et al. 2004a, 2004b; Taheri 2004). Low self-esteem has also been found to be positively related to obesity in adolescents (e.g., Crossman, Sullivan and Benin 2006). Individuals who suffer from low self-esteem may have internalized negative views about themselves, which makes self-awareness especially painful. Therefore, they use eating as a coping mechanism, particularly using binge eating to reduce painful self-awareness, which can then lead to excessive weight gain (Heatherton and Baumeister 1999). Although the relationships of sleep and low self-esteem with obesity are likely bi-directional, I attempt to sort out the pathway of influence by measuring self-esteem and sleep prior to obesity in a longitudinal model.

Skips Breakfast: Using responses from the WI and WII In-Home Questionnaires, a dummy variable was created to indicate whether the respondent skips breakfast. This variable indicated whether the respondent reported usually eating nothing for breakfast at WI and eating breakfast zero to two times in the seven days prior to their interview at WII. Eleven percent of respondents skip breakfast.

Inadequate Sleep: Using responses from the WI and WII In-Home Questionnaires, a dummy variable is created to indicate whether the respondent reported usually sleeping less

than 7 hours at both Waves I and II.⁹ A higher percentage of females (27%) experience short sleep duration than males (20%).

Low Self-Esteem: Self-esteem is measured from responses in the WI In-Home Questionnaire. Respondents were asked a series of six questions measuring the extent to which a respondent believes he/she feels loved and wanted, feels socially acceptable, likes himself/herself just the way he/she is, is doing everything right, has a lot to be proud of, believes he/she has a lot of good qualities. Respondents answered each question using a 5-point Likert Scale (Alpha=0.85). The answers to these questions are combined in a scale ranging from 1 (low self esteem) to 5 (high self esteem). The respondents with a score of 3.8 or less for reported self-esteem are classified as having low self-esteem. Females are also more likely to have low self-esteem (26%) than males (16%).

Family Level Risk Factors

I focus on family level risk factors in the following domains: cyclical income, health insurance status, family structure, parental education, number of siblings, parental monitoring and care of children, and parental obesity. Trouble paying bills is a proxy for the cyclical income effect that is common in poor, usually welfare or food stamp reliant, households. If parents do not have enough money to pay family bills, and bills are due at the end of each month, this may indicate episodic food shortages with food restriction when money runs low and food binging of calorically-dense foods when money is again available leading to weight gain over time (Townsend et al. 2001). Health policy analysts have also identified lack of access to health care as a possible explanation for social class disparities in child health (Newacheck et al. 2000).

⁹ The exact wording of the question was; “How many hours of sleep do you usually get?”

Studies have found that parental monitoring has an influence on children's food selections which impacts childhood obesity (e.g., Brown and Ogden 2004; Klesges et al. 1991; Robinson et al. 2001; Seibold, Knafl and Grey 2003). Poor parents are less able to monitor their children's diet and physical activity due to stress, lack of emotional resources, and time constraints if they work. Family structure is highly correlated with poverty and is thought to be associated with parental monitoring as well (Hogan and Kitagawa 1985; McLanahan 1995). Parental education, related to poverty and family structure, may also affect parental monitoring, with less-educated parents monitoring their children less than higher-educated parents (Lareau 2002, 2003). Parental monitoring may also be compromised in large families. Research from a number of disciplines suggests that an increase in the number of siblings diminishes the time and material resources that parents can provide to each child, which can negatively affect child outcomes (see Heer 1986 for a review).

The quality of parent-child relationships has also been linked to adolescent obesity outcomes (e.g., Crossman et al. 1996). In addition, child maltreatment has been linked to a number of child developmental outcomes (see Crouch and Milner 1993 for a review) as well as obesity (Gustafson and Sarwer 2004; Selway 2006; Wiederman, Sansone and Sansone 1999; Williamson et al. 2002). Previous research has also found significant positive relationships between adolescent obesity and parental obesity status, which can be contributed to parental role modeling of lifestyle and eating behavior, as well as genetic inheritance, or some combination of genetic propensity and environmental risk (Agras et al. 2004; Whitaker et al. 1997).

Trouble Paying Bills: Using data from the WI Parent Questionnaire, a dichotomous variable is created to indicate whether the respondent's parent reported having trouble paying

bills. This measure is a proxy for cyclical income and is experienced by 17% of the sample.

No Health Insurance: Using data from the WI Parent Questionnaire, a dichotomous variable is created to indicate whether the respondent's parent reported that the respondent had health insurance. Eleven percent of respondents had no health insurance.

Single-Parent or Surrogate Parent Family: Rich detail is available on adolescents' living arrangements. Because risk factors need to be dichotomized, I classify adolescents into two categories. The "at risk" category consists of individual living in single mother, single father and surrogate parent (no biological parent in the household) families. Surrogate families include foster parents, stepparents, grandparents, aunts, uncles, siblings, or other adults who act as parent figures. The "non risk" category consists of two biological or adoptive parents and stepfamilies that include a biological parent. Twenty-four percent of the sample lives in this "at risk" family arrangement.

Parental Education Less than High School: Using data from the WI Parent, In-Home and In-School Questionnaires, parents' education is measured as the higher of either mother's or father's education. The "at risk" category consists of parents with less than a high school degree or GED. Ten percent of the sample has a parent with less than a high school degree.

Large Number of Siblings: Respondents are coded as having a large number of siblings if they reported living with more than two siblings at WI (14% of respondents).

Unshared Family Meal Times: Data from WI was used to create a measure of whether a parent regularly eats the dinner meal with the adolescent during the week. Based on the question, "On how many of the past 7 days was at least one of your parents in the room with you while you ate your evening meal?" Responses of less than 4 days per week were coded as '1', for not eating dinner together regularly. Twenty six percent of the sample did not eat

dinner regularly with their parent(s). Although I cannot determine the quality and quantity of food that children eat when they eat meals with their parents, it has been argued that children who eat with their parents might have better dietary intake and can learn good eating habits, if parents consume healthy foods. Increased frequency in eating meals with parents is also associated with fewer disordered eating behaviors and increased psychosocial well-being, which would protect adolescents from excessive weight gain. In addition, when parents eat with their children, they monitor their behavior and their intake of food. Therefore, eating less with parents may lead to less healthy dietary intake and increased weight gain (Fulkerson et al. 2008).

Lack of Parental TV and Food Monitoring: This measure involves adolescents' TV viewing (inactivity) and food consumption. Parental monitoring of TV viewing is also measured at WI by adolescent responses to the question, "Do your parents let you make your own decisions about how much television you watch?" Parental monitoring of eating is also measured at WI by adolescent responses to the question, "Do your parents let you make your own decisions about what you eat?" Respondents were coded as experiencing a lack of monitoring if the parent did not make the decision about the amount of adolescent TV viewing and food consumption. More than half (68%) the sample had parents who did not monitor their eating or TV habits.

No Curfew: This measure also uses data from WI adolescent responses to the question, "Do your parents let you make your own decisions about the time you must be home on weekend nights?" Respondent were coded as having no curfew if they answered the question affirmatively. A higher percentage of males (34%) than females (25%) have no curfew.

High Parent-Child Conflict: High parent child conflict is measured at WI and WII by

adolescent reports of whether they had a serious argument about their behavior with their mother or father within the last four weeks. If respondents reported having this type of argument in both WI and WII, I code high parent-child conflict as '1'. High conflict is evidenced in twenty four percent of the sample.

Lack of Parental Presence: This measure is constructed from respondent reports of the frequency with which their parents were home when they left for school, when they returned from school and when they went to bed (ranging from “never” to “always”) at WI.

Respondents were coded as having a lack of parental presence if a parent was reported as never being present for one or more of these three daily activities (25% of respondents).

Lack of Parent-Child Interaction: Parent-child interaction is created from data at WI.

Respondents were asked to report the activities they engaged in with each parent in the past four weeks. Respondents could report up to nine activities ranging from going shopping to working on a school project. Respondents were reported as having a lack of parent-child interaction if the average number of activities they engaged in with their parent(s) was less than two in the past four weeks (30% of respondents).

Sexual Abuse: Sexual abuse is also measured using reports from WIII Questionnaire.

Respondents are classified as being sexually abused if they reported that their parent or other adult caregivers had “touched [them] in a sexual way, forced [them] to touch [their parent or caregiver] in a sexual way, or forced [them] to have sexual relations.” Four percent of the sample reported sexual abuse. There are a number of explanations for the relationship between childhood sexual abuse and obesity. One hypothesis is that sexual abuse leads to binge eating, which results in obesity. Binge eating may be the result of the low-self esteem and depression experienced by victims of abuse. Another explanation is that women with a

history of sexual abuse use obesity as a self-protective mechanism or adaptive function that shields them from sexual advances. In Western culture, thinness is a component of female beauty and sexuality. Therefore, obesity can protect a person who has been sexually abused from their sexuality (Gustafson and Sarwer 2004).

Physical Abuse: Physical abuse is measured using reports from WIII Questionnaire.

Respondents are classified as being physically abused if they reported that their parent or other adult caregivers had ever “slapped, hit, or kicked” them. Twenty-eight percent of the sample reported physical abuse. Similar to sexual abuse, childhood physical abuse is also an exposure to trauma in early life that is characterized as an intense negative emotional experience that can lead to low-self esteem and depression. Disordered eating behavior can be used as a coping mechanism that can lead to obesity (Alvarez et al. 2007).

Parent Obese: Using reports from the Parent In-Home Questionnaire, a respondent was coded as having an obese parent if either their biological mother and/or biological father self-reported as being obese. Twenty four percent of the sample has an obese parent.

Neighborhood Level Risk Factors

I focus on neighborhood level risk factors in the following domains: family structure, poverty, unemployment, housing quality, crime and safety. Studies have found that neighborhood poverty is related to physical activity, and, therefore, obesity (e.g., Gordon-Larsen, Nelson and Popkin 2006). Low-SES neighborhoods have reduced access to recreational facilities (Gordon-Larsen et al. 2006) and higher crime rates (e.g., Hannon 2005; Kling, Ludwig, and Katz, 2005), which limit physical activity (Gordon-Larsen, McMurray and Popkin 2000). The measures of risk used in this analysis try to capture the neighborhood

context of social disadvantage.

The census block group (BG) is used as the geographical unit to define neighborhood in this analysis. Census blocks are the smallest geographic area for which the Census Bureau collects decennial census data. In the geographic hierarchy, block groups BGs are the next level above census blocks. A BG is a subdivision of a census tract or block numbering area (BNA) and is composed of a number of census blocks (U.S. Census Bureau 1994). A BG represents the most localized available contextual characteristics of the areas in which individuals reside. In 1990, a BG averaged 452 housing units, or 1,100 people (U.S. Census Bureau 1994).

High Proportion of Female Headed Households in Neighborhood: Neighborhood family structure comes from census data that is attached to the adolescent's home address at WI. I use a measure of the percentage of households in the census block group that are female-headed with own children under the age of 18. The respondents living in neighborhood where 10% or more of the family households are female-headed households are coded as having a high level of female-headed households in their neighborhood. Twenty-two percent of the respondents in this sample live in this type of neighborhood.

High Neighborhood Poverty: Neighborhood poverty also comes from WI block group measures. It is a measure of the proportion of families with income in 1989 below poverty level. The respondents living in neighborhoods where the proportion of families below poverty level in their neighborhood equals or exceeds 19% are coded as having a high level of neighborhood poverty. Twenty one percent of the respondents in this sample live in this type of neighborhood.

High Neighborhood Unemployment: Neighborhood unemployment also comes from WI

BG measures. It is a measure of the total unemployment rate. The respondents living in neighborhoods where the unemployment rate in their neighborhood equals or exceeds 10% are coded as having a high level of unemployment (19% of respondents).

Low Neighborhood Housing Quality: Neighborhood housing quality comes from WI BG measures. It is a measure of the proportion of vacant housing units. Respondents living in neighborhoods where 10% or more of the housing units are vacant are coded as having a low level of housing quality (25% of respondents).

High County-Level Crime: County crime comes from WI county level measures. It is a measure of the total crime rate per 100,000 in the reporting county. Respondents living in counties where the total crime rate exceeds or equals 7,500/100,000 are coded as having a high level of crime.¹⁰ Eighteen percent of respondents in this sample live in counties of this type.

Unsafe Neighborhood: A measure of the respondent's perception of his/her neighborhood being unsafe was created by responses to the question: "Do you usually feel safe in your neighborhood?" at WI. Nine percent of respondents felt unsafe in their neighborhood. It should be noted that this measure is not a neighborhood level measure because it is not aggregated by neighborhood, it is an individual measure of the perception of the neighborhood in which a respondent lives.

Outcome Variable

Obesity and Obesity Trajectories

Body mass index or BMI is used to measure obesity. BMI is a tool for indicating weight status in adults, computed by dividing an individual's body weight in kilograms by the

¹⁰ Add Health contains no neighborhood level measure of crime.

square of his or her height in meters (i.e., $\text{weight}/(\text{height})^2$). BMI is more highly correlated with body fat than any other indicator of height and weight (CDC 2006). For adults over 20 years old, BMI falls into one of four categories: underweight, normal, overweight and obese. Adults with a BMI of 25 to 29.9 are categorized as overweight. Adults with a BMI of 30 or more are considered obese (NHLBI 1998; WHO 2000).

BMI is defined differently for children and adults. Age- and sex-specific BMI percentiles are used as growth references during childhood and adolescence because BMI changes at different rates by age and sex during normal developmental growth. In the United States, the 85th and 95th percentiles, based on nationally representative data from the 2000 growth curves of the Centers for Disease Control and Prevention (CDC), have been recommended for use in classifying persons as being overweight or at risk of overweight (CDC 2004). However, definitions of overweight based on these percentiles are not directly comparable to the adult definitions of obesity using specified cut points (NHLBI 1998, WHO 2000).

The ability to generate comparable prevalence measures between adult and adolescent measures of obesity or to calculate obesity incidence over the transition period from adolescence to young adulthood is limited by discrepancies between adolescent and adult definitions. The International Obesity Task Force (IOTF) developed BMI curves, which link childhood and adolescent BMI centiles to adult cut off points of BMI of 25 and 30 kg/m^2 , and thus, allow greater consistency in the youth versus adult definitions. The BMI curves provide good comparative reference data during this transitional period (Cole et al. 2000). Because this analysis investigates the incidence of obesity from adolescence to young adulthood, the IOTF measures are used to determine obesity status among adolescents and

young adults.

Obesity is measured at WII and WIII using BMI calculated from measured height and weight¹¹ using the IOTF cutoffs. Individuals are classified as obese if their BMI falls above the age- and sex-specific, IOTF 30 kg/m² cutpoint in adolescence at WII. For the young adults at WIII, the adult BMI cut point of 30 kg/m² is used. A 2X2 table of obesity status at WII by obesity status at WIII is then created to identify trajectories of obesity from adolescence into young adulthood. The dependent variable contains four trajectories that capture change and continuity in obesity status from Wave II to Wave III: *Become Obese* (respondent not obese at WII but obese at WIII); *Stay Obese* (obese at both WII and WIII); *Reduce Obesity* (obese at WII but not obese at WIII); and *Stay Non-Obese* (not obese at both waves). Because the proportion of individuals in the trajectory of reduce obesity is too small to analyze, I do not report coefficients for the *Reduce Obesity* outcome category. The *Stay Non-Obese* trajectory will serve as the reference category in analysis using obesity trajectories as outcomes. Table 2.1 shows that 12% of the sample became obese, 10% of the sample stayed obese and 77% of the sample either stayed non-obese or became non-obese at WIII. At WII 11% of the sample was obese and at WIII 22% of the sample was obese, indicating a doubling in the prevalence of obesity in this sample in only 2 years.

Analytical Approach

All analyses will use STATA survey procedures with longitudinal sampling weights to adjust for the clustered sample design and unequal probability of selection at Wave I and attrition in the sample over time so that the results are nationally representative and that bias in standard errors are reduced. Analysis will be guided by the four research questions

¹¹ Height and weight are self-reported at Wave I.

discussed above. Analysis begins with examination of the bivariate relationship between risk factor measures and obesity outcomes using logistic regression in samples divided by sex. If bivariate relationships differ by sex, a separate CRI is created for males and females. Measures found to be significant will be included in a cumulative risk index (CRI).¹² A separate index will be created for the individual and family risk factors and neighborhood level risk factors. I will also construct a total risk index comprising all risks across all levels of measurement to compare differences by population subgroup. The mean level of cumulative risk for female, male, white, black, Hispanic, poor, and non-poor population subgroups and combinations of these populations will be calculated to determine which population subgroup faces the highest amount of cumulative risk. The statistical significance of the differences among means will also be tested.

Analysis continues by using logistic and multinomial logistic regression models to test if the risk of obesity in adolescence and young adulthood and the risk of becoming or staying obese¹³ from adolescence into young adulthood increases as the number of risk factors increases. The number of risk factors, measured by the cumulative risk index, will be operationalized as a set of dummy variables in the regression model representing the count of risks with zero risks as the reference category. Regression models will control for age of respondent and urbanicity. Previous research indicates that living in rural areas increases the likelihood of obesity for children and adolescents (Lutfiyya et al. 2007). The bivariate relationship between accumulated risk and obesity will also be investigated using cross tabulations with chi-squared tests for statistical significance.

Multinomial logistic regression is then employed to examine the relationships

¹² For sensitivity analysis, I will also create indices using all the measures that have been theoretically or empirically linked to obesity in previous research.

¹³ This is compared to the trajectory of staying non-obese.

displayed in the conceptual model (see Figure 2.1). Multinomial regression is appropriate when the dependent variable is an unordered nominal variable with n categories. The procedure estimates the log of the ratio of the probability of being in the n^{th} category relative to a base category (stay non-obese), where the effects of independent variables are measured by the relative risk or odds (Long 1997).

The fundamental model takes the form:

$$\log \left[\frac{P_{ij}}{P_{iJ}} \right] = \beta_j x_i$$

The model is generalized to J categories, with the running index $j=1 \dots, J$. Where P_{ij} is the probability that individual i falls into category j and where x_i is a column vector of variables describing individual i and β_j is a row vector of coefficients for category j . Note that each category is compared with the highest category J .

Multivariate analysis begins with a baseline model of the relationship between race/ethnicity and obesity trajectories. Model 2 enters welfare/poverty status. Models 3 and 4 enter the cumulative risk indices (individual and family; neighborhood) as the intervening mechanisms displayed in Figure 2.1. Model 5 enters control measures for age and urbanicity. This design exploits the longitudinal data and incorporates the temporal order of effects (i.e., factors operating during adolescence prior to the transition to adulthood). The longitudinal design allows for the measurement of factors operating in adolescence and the influences this has on physical activity outcomes in later adolescence and young adulthood. To the extent that cumulative risk mediates the effects of poverty and race/ethnicity, we add to our understanding of the ways in which social disadvantage influences obesity trajectories from adolescence into young adulthood. Analysis adjusts for design effects inherent in the complex stratified cluster sampling used by Add Health.

Results

What are the factors that place young people at risk for obesity?

Table 2.2 shows the relationship between measures of risk and obesity status at WII and WIII for females and males. Minority and welfare/poverty status are only related to obesity in the female sample, supporting previous research (Lee et al. 2008). Females are more likely to be obese at WII and WIII if they are black or Hispanic (versus white) and if they were poor and/or received welfare in childhood (versus no poverty or welfare receipt in childhood). More specifically, female minorities are 82% more likely to be obese at WII¹⁴ and 78% more likely to be obese at WIII than whites. Female respondents who were poor or received welfare in childhood are 86% more likely to be obese at WII and 82% more likely to be obese at WIII than female respondents who were not poor or did not receive welfare in childhood.

Among individual level risk factors, skipping breakfast and inadequate sleep increase the likelihood of obesity at WII or WIII for both males and females. There is a particularly strong relationship between skipping breakfast and obesity at WIII for males. Males who skip breakfast at WI and WII are 159% more likely to be obese at WIII than males who did not skip breakfast. Low self-esteem increases the likelihood of obesity for females.

Among family level risks factors, only parental obesity, cyclical income and physical abuse were significantly related to obesity status at WII or WIII for both males and females. A strong relationship between parental obesity and child obesity exists for both males and females. Males and females having an obese parent are more than 100% more likely to be obese at WII or WIII than males and females who do not have an obese parent. Other

¹⁴ **Note:** This statement is equivalent to stating, “Being a female minority is associated with an 82% increase in the likelihood or odds of becoming obese at WII.”

measures of family risk are significantly related to obesity for females, including single or surrogate family structure, low parent education, having no curfew, and physical abuse. A significant relationship also exists between having a large number of siblings and obesity status for males, but it is in an unexpected direction. Males who have more than two siblings are less likely to be obese at WII than males with fewer siblings. This could be explained by the fact that Add Health only contains information on siblings currently living in the respondent's household. Therefore, this measure could be capturing families with younger children overall who are more active than families with older children who have left the family household. Interestingly, measures of lack of parental monitoring, other than low interaction and having no curfew, were not significantly related to obesity. Not sharing regular meals with parents, lack of parental monitoring of what respondent eats or TV viewing and lack of parental presence when the respondent goes to school, comes home or goes to bed are not significantly related to obesity. High parent-child conflict and sexual abuse are also not significantly related to obesity in the male or female samples.

Although most measures of social disadvantage at the individual and family level (i.e., minority status, poverty status, low parent education) only increase risk of obesity for females, disadvantage at the neighborhood level increases risk of obesity for both males and females. A high percentage of female-headed families, high neighborhood poverty and high neighborhood unemployment are significantly related to obesity for both males and females. Low neighborhood housing quality and respondent perceptions of neighborhood as being unsafe are also significantly related to obesity for females. County level measures of crime and respondent reports of neighborhood safety are not significantly related to obesity.

Because the relationship between the specified risk factors and obesity operate

differently for males and females, separate indices are created for each group. Separate indices will also be created for individual and family risk factors and neighborhood risk factors. Each index contains all significant risk factor relationships with obesity at WII or WIII for each sex group and risk level (i.e., individual and family and neighborhood). For descriptive purposes I will also create a total index that sums risk factors across all levels of social context. In sensitivity analysis, I will also create indices that include all measures that are discussed earlier that are theoretically related to obesity, to determine if the same patterns exist.

The female total cumulative risk index (F-CRI) includes fourteen risk factors: (1) skipping breakfast; (2) inadequate sleep; (3) low self-esteem; (4) parental obesity; (5) trouble paying bills (cyclical income); (6) single or surrogate parent family; (7) low parent education; (8) no curfew; (9) physical abuse; (10) high percentage female headed households in neighborhood; (11) high neighborhood poverty; (12) high neighborhood unemployment; (13) low neighborhood housing quality and (14) respondent perceptions of neighborhood as being unsafe.¹⁵

The female individual and family risk index (IF-F-CRI) includes nine risk factors: (1) skipping breakfast; (2) inadequate sleep; (3) low self-esteem; (4) parental obesity; (5) trouble paying bills (cyclical income); (6) single or surrogate parent family; (7) low parent education; (8) no curfew and (9) physical abuse. The female neighborhood risk index (N-F-CRI) includes five risk factors: (1) high percentage female headed households in neighborhood; (2) high neighborhood poverty; (3) high neighborhood unemployment; (4) low neighborhood housing quality and (5) respondent perceptions of neighborhood as being

¹⁵ Correlations across all risk factor measures do not exceed 0.35 except for correlations between neighborhood female-headed households, poverty and unemployment, which range from 0.71 to 0.79 (Results not shown).

unsafe.

The male total cumulative risk index (M-CRI) contains a subset of the measures in the F-CRI including: (1) skipping breakfast; (2) inadequate sleep; (3) parental obesity; (4) trouble paying bills (cyclical income); (5) physical abuse; (6) high percentage female headed households in neighborhood; (7) high neighborhood poverty and (8) high neighborhood unemployment.¹⁶

The male individual and family risk index (IF-M-CRI) contains five risk factors: (1) skipping breakfast; (2) inadequate sleep; (3) parental obesity; (4) trouble paying bills and (5) physical abuse. The male neighborhood risk index (N-M-CRI) contains three risk factors: (1) high percentage female headed households in neighborhood; (2) high neighborhood poverty and (3) high neighborhood unemployment.

No single respondent experiences all of the risk factors that are entered into the indices. The maximum number of risks experienced by a female is eleven (out of fourteen) and the maximum number of risks experienced by a male is seven (out of eight). Table 2.3 provides the percentage of individuals who fall into each level of risk. For the F-CRI, female respondents experiencing six or more risk factors are collapsed into one category due to the small percentage of individuals experiencing seven or more risks. For the M-CRI, male respondents experiencing five or more risk factors are collapsed into one category. The distributions show that a majority of respondents experience at least one risk, with fewer respondents experiencing very high levels of risk or no risks. The mean level of risk experienced by females is about three risks (out of fourteen risks). The mean level of risk experienced by males is about two risks (out of eight risks).

¹⁶ Although a high number of siblings is significantly related to obesity for males, the relationship is in the opposite direction, and, therefore, is not included in the M-CRI.

Similar patterns exist in the distribution of number of risk factors by individual and family and neighborhood level. Table 2.4 provides the proportion of individuals that fall into each level of risk. The highest category is collapsed for the individual and family cumulative risk indices for both males and females.

Who faces the highest amounts of cumulative risk?¹⁷

Table 2.5 displays the mean level of cumulative risk out of the fourteen risk factors in the F-CRI¹⁸ by sex, race/ethnicity and poverty status subgroups. In general, females experience a slightly higher level of risk than males, despite poverty status or race/ethnicity. Blacks experience the highest levels of risk, with poor blacks experiencing the largest mean levels of risk compared to all other sex, race/ethnicity and poverty status subgroup combinations. In addition, non-poor blacks face levels of risk similar to poor whites. The mean level of risk among non-poor blacks is 3.77 compared to 3.60 among poor whites. Hispanics also face higher levels of risks than whites. Similar patterns are evidenced when comparing the mean levels of cumulative risk out of the nine risk factors in the M-CRI (see Table 2.6).

Do risk factors operate in a cumulative manner, such that higher levels of risk are associated with higher levels of obesity risk?

Table 2.7 shows the results of the logistic and multinomial logistic regression models of the relationship between the number of risks in the Individual and Family CRI (IF-F-CRI) and the Neighborhood CRI (N-F-CRI) and obesity status at WII and WIII and change in

¹⁷ All differences in mean levels of risk discussed in this paper are significant to the $p < 0.05$ level or less, unless stated otherwise.

¹⁸ Mean risk out of the fourteen risk factors of the F-CRI is calculated for male subgroups for comparison purposes.

obesity status from WII to WIII for female sample.¹⁹ Results for males are shown in Table 2.8. The table also displays the percent of respondents who are obese at each wave or become or stay obese from WII to WIII as a function of the number risks in the CRI.

In the female sample, there is a clear gradient in the percentage of respondents who are obese at WII or WIII as the number of risks in the IF-F-CRI increases. Among respondents with no risk factors, 3% were obese at WII and 9% were obese at WIII. Among respondents with seven or more risk factors, 21% and 36% were obese at WII and WIII, respectively. A similar gradient can be evidenced among those who become obese or stay obese from WII to WIII (compared to those who stay-non obese).²⁰ It should be noted that the gradient becomes less clear with risks that are greater than four. Some of this may be a function of sample size. Only 3% of the female sample experience five risks and only 2% of the female sample experience six or more risks. Odd ratios follow a similar gradient. The stepwise increase in odds for each additional risk factor is statistically significant in the female sample for all obesity outcomes.²¹ This increase in odds is substantial. For females, respondents experiencing seven or more risk were 840% more likely to be obese at WII, 465% more likely to be obese at WIII, 244% more likely to become obese and 130% more likely to stay obese from WII to WIII than individuals experiencing zero risks. A similar gradient is evidenced for the N-F-CRI. As the number of neighborhood risks increases, the odds of obesity at WII and WII and become and staying obese from WII to WIII increases. It should be noted that having one risk is not significantly different from having zero risks for the obese at WII and stay obese outcomes. Having all five neighborhood risks is also not

¹⁹ Models control for age and urbanicity.

²⁰ In the discussion of results when “become obese” or “stay obese” are used it is assumed that this state is compared to the reference category of “stay non-obese.”

²¹ It should be noted that experiencing two risks was not statistically significant from experiencing zero or one risk for “stay obese” (versus stay non-obese) in the female sample.

statistically different from having one neighborhood risk for the obese at WII outcome. Again, this may be a function of sample size, given that only 2% of the female sample experiences all five neighborhood risks.

Gradients in the relationship between obesity and number of risks in the IF-M-CRI are also clear for the male sample. The percent of respondents who are obese at each wave as a function of the number risks in the IF-M-CRI steadily increases. This is also the case for those who become obese from WII to WIII. However, having four or more risks is not significantly different from having zero risks for the stay obese category. Gradients are clearer in the relationship between number of risks and obesity at WII and WIII and staying obese from WII to WIII. Despite the less clear gradients in the male sample, in general, the larger the number risks experienced by male respondents, the higher the likelihood that they will be obese at WII or WIII and become obese or stay obese from WII to WIII. Gradients in the relationship between obesity and number of risks in the N-M-CRI for the male sample are less apparent than they are for the IF-M-CRI. For all outcomes having one neighborhood risk or all 3 neighborhood risks is not significantly different from have zero neighborhood risks. Having 2 risks increased the likelihood of obesity at WII and WIII for males and also increased the likelihood of staying obese from WII to WIII. Number of neighborhood risks was not associated with becoming obese for males.²²

Do measures of cumulative risk mediate the relationship between race/ethnicity, poverty and obesity?

Tables 2.9 and 2.10 present the estimated odds ratios for multinomial logistic regression models that examine the relationships displayed in the conceptual model (see

²² Models examining cumulative effects using risk indices including all theoretically relevant variables produced similar results.

Figure 2.1) for female and male samples, respectively. In Model 1 for the female sample (see Table 2.9), there is a significant relationship between being black and the likelihood of becoming obese and staying obese from WII to WIII. Black female respondents are 94% more likely to become obese and 132% more likely to stay obese than white female respondents. Hispanic females are also 65% more likely to stay obese compared to white females. When welfare/poverty status is added in model 2, the relationship between Hispanic ethnicity and stay obese becomes insignificant and the relationship between black and becoming or staying obese is slightly reduced. Poverty status acts as a confounder in the relationship between minority status and obesity as put forth in the conceptual model. Poverty status is significantly related to both becoming obese and staying obese from WII to WIII. Poor females are 58% more likely to become obese and 65% more likely to stay obese from WII to WIII. When the IF-F-CRI is included in Model 3, the relationship between black and obesity status is reduced and poverty status and obesity status becomes insignificant. When the N-F-CRI is included in Model 4, the relationship between black and obesity status becomes insignificant.²³ This suggests that cumulative risk mediates the relationship between these traditional measures of disadvantage and obesity. Model 5 includes controls for age and urbanicity. The relationship between cumulative risk and obesity status remains the same. For each increase in risk in the IF-F-CRI, a female's odds of becoming obese and staying obese from Wave II to Wave III increases by 31% and 47%, respectively. For each increase in risk in the N-F-CRI, a female's odds of becoming obese and staying obese from Wave II to Wave III increases by 15% and 22%, respectively.

Table 2.10 provides multinomial logistic regression results for the male sample.

²³ In models not shown, N-F-CRI was included with only race/ethnicity and poverty status. The N-F-CRI slightly reduced poverty coefficients, but they did not become insignificant.

Neither race/ethnicity nor poverty status are significantly related to the obesity transitions or continuity. However, the IF-M-CRI is significantly related to both becoming obese and staying obese. The N-M-CRI is only significantly related to staying obese. In the final model (Model 5), for each increase in risk in the IF-M-CRI, a male's odds of becoming obese and staying obese from Wave II to Wave III increases by 49% and 86%, respectively. For each increase in risk in the N-M-CRI, a male's odds of staying obese from WII to WII increases by 38%. In these models, the CRI cannot be classified as a mediating measure given that race/ethnicity and poverty status are not significantly related to obesity for males.

Discussion and Conclusion

Using nationally representative data, I assessed the relationship between cumulative representations of risk in childhood and adolescence and obesity transitions and continuity from adolescence into young adulthood. This research is one of the few studies to examine the relationship between models of cumulative risk and health outcomes beyond mental or psychiatric disorders. To my knowledge, it is also the first time these models are used to examine the relationship between social disadvantage and obesity. This analysis contributes to the research on obesity among children and adolescents by using a longitudinal design that allows for the tracking of obesity change and continuity beginning in adolescence and continuing through the transition to young adulthood. This longitudinal design is used to sort out the temporal ordering of the effects by measuring poverty and race/ethnicity in childhood and adolescence, the intervening mechanisms (i.e. mechanisms included in the cumulative risk indices) of the ways in which poverty may operate on obesity during adolescence, and obesity change and continuity from adolescence to young adulthood. This design is effective

for examining prior effects on subsequent outcomes through time.²⁴ Finally, this analysis uses dynamic measures of both poverty and obesity, and a measure of cumulative risk that attempt to capture the multi-factorial causes of obesity.

The analysis reveals that numerous individual, family and neighborhood level factors serve as significant risk factors for obesity in adolescence and young adulthood. The relationship between risk factors and obesity operates differently for males and females and by level of context, necessitating the construction of separate cumulative risk indices for females (F-CRI) and males (M-CRI) and by individual/family risks and neighborhood risks. A larger number of risk factors were significantly related to obesity outcomes for females than for males. The M-CRI was actually made up of a subset of the fifteen risk factors that made up the F-CRI. Females' obesity status is vulnerable to many aspects of the family and neighborhood environment such as low parent education, single or surrogate family structure, lack of parent-child interaction and neighborhood housing quality where is not for males. Interestingly, although family level poverty status was not significantly related to obesity in males, neighborhood poverty was significantly related to obesity in males.

Further analysis showed that females experience slightly higher levels of risk than males. In addition, the poor face higher levels of risk than the non-poor. However, it is blacks who experience the highest levels of risk, especially poor blacks. Non-poor blacks face levels of risk equivalent to poor whites, highlighting the vulnerability of this racial/ethnic group and reinforcing notions that the poor and ethnic minorities face multiple risks and experience more risks the non-poor and whites, respectively.

Analysis found that risk factors do operate in a cumulative manner, such that higher levels of risk are associated with higher levels of obesity risk. However, relationships varied

²⁴ It should be noted that this model is not able to establish causal relationships.

by the CRI index used (i.e., Individual and Family CRI or Neighborhood CRI), by the obesity outcome, and by sex. This relationship was more apparent for the Individual and Family CRI for both the male and female samples. Neighborhood CRI also operated in a cumulative manner for females, but not for males. The gradient between increasing number of risks and likelihood of obesity is also more apparent for staying obese than for becoming obese from WII to WIII (versus stay non-obese or reduce obese). The fact that cumulative risk models work better in determining risk of obesity for females than for males is an interesting finding that deserves further research, as does the lack of a significant relationship between traditional measures of disadvantage (i.e., racial/ethnic minority status and welfare/poverty status) and obesity in males.

Cumulative risk indices also mediated the relationships between race (being black) and poverty status with obesity in the female sample. The Individual and Family CRI mediated the relationship between welfare/poverty status and becoming and staying obese and both the Neighborhood and Individual and family CRI completely mediated the relationship between being black and becoming or staying obese. This means that the experiences captured in the CRI serve as intervening mechanisms through which poverty affects obesity and through which race is related to obesity for females. The relationship between Hispanic ethnicity and staying obese from adolescence into young adulthood is due to the higher rates of poverty among Hispanic females. The CRI was also significant in the male sample but did not serve as an intervening mechanism due to the fact that race/ethnicity and poverty were not significantly related to obesity. This provides evidence that the cumulative risk model is able to capture experiences at the individual, family and neighborhood level faced by adolescents that place them at risk for obesity. The evidence is

quite clear, however, that it is the poor and racial/ethnic minorities who will face more of these risks than other groups.

By utilizing the cumulative risk model as an intervening mechanism through which poverty and race are related to obesity, this research contributes to an understanding of the processes of obesity development during adolescence and in the transition to adulthood. The cumulative risk model provides evidence that individuals facing multiple risks at multiple levels of social context are most vulnerable to becoming obese and staying obese from adolescence and into young adulthood. Although one of the weaknesses of the cumulative risk model is that it cannot identify which aspect of individuals' social context make them most vulnerable to obesity, it does indicate that as the number of risk factors (in a CRI) experienced by an individual increases, the higher the likelihood that he/she will become or stay obese in young adulthood.

Despite weaknesses in the cumulative risk model noted previously, this analysis illustrates its utility as an overall indicator of risk for obesity, especially for females. In addition, the cumulative risk model is able to use the numerous measures available in Add Health at multiple levels of social context in a way that cannot be not be done in a traditional multivariate model where each measure would be a separate predictor. The cumulative risk indicator also contributes to racial/ethnic health disparities research. African Americans may face higher levels of obesity risk because they experience a higher level of contextual risk factors compared to other races, regardless of poverty status. Lifestyle and other modifiable risk factors included in the cumulative risk index, such as short sleep duration and skipping breakfast could be targeted as possible interventions since they work cumulatively, along with other factors to increase obesity risk. In sum, cumulative risk models provide an

alternative and useful approach to capture risks to health that incorporate the multidimensionality and complexity of the social world.

References

- Ackerman, Brian P., Carroll E. Izard, Kristen Schoff, Eric A. Youngstrom and Jen Kogos. 1999. "Contextual Risk, Caregiver Emotionality, and the Problem Behaviors of Six and Seven year Old Children from Economically Disadvantaged Families." *Child Development* 70(6): 1415-1427.
- Agras, Stewart W., Lawrence D. Hammer, Fiona McNicholas and Helena C. Kraemer. 2004. "Risk Factors for Child Overweight: A Prospective Study from Birth to 9.5 Years." *Journal of Pediatrics* 145: 20-45.
- Alvarez, Jennifer, Joanne Pavao, Nikki Baumrind and Rachel Kimerling. 2007. "The Relationship between Child Abuse and Adult Obesity Among California Women." *American Journal of Preventative Medicine* 33(1):28-33.
- Andersen, Ross E., Carlos J. Crespo, Susan J. Bartlett, Lawrence J. Cheskin, Michael Pratt. 1998. "Relationship of Physical Activity and Television Watching with Body Weight and Level of Fatness among Children: Results from the Third National Health and Nutrition Examination Survey." *Journal of the American Medical Association* 279: 938-942.
- Appleyard, Karen, Byron Egeland, Manfred H. M. Van Dulmen and L. Alan Sroufe. 2005. "When More is Not Better: The Role of Cumulative Risk in Child Behavior Outcomes." *Journal of Child Psychology & Psychiatry* 46(3): 235-245.
- Atzaba-Poria, Naama, Alison Pike and Kirby Deater-Deckard. 2004. "Do Risk Factors for Problem Behaviour Act in a Cumulative Manner? An Examination of Ethnic Minority and Majority Children through an Ecological Perspective." *Journal of Child Psychology and Psychiatry* 45(4): 707-718.
- Bauman, Laurie J., Ellen J. Silver and Ruth E.K. Stein. 2006. "Cumulative Social Disadvantage and Child Health." *Pediatrics* 117(4): 1321-1328.
- Ben-Shlomo, Yoav and Diana Kuh. 2002. "A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges and Interdisciplinary Perspectives." *International Journal of Epidemiology* 31: 285-293.
- Braveman, Paula A., Catherine Cubbin, Susan Egerter, Sekai Chideya, Kristen S. Marchi, Marilyn Metzler, Samuel Posner. 2005. "Socioeconomic Status in Health Research: One Size Does Not Fit All." *JAMA* 294(22): 2879-2888.
- Bronfenbrenner, Urie. 2005. *Making Human Beings Human: Bioecological Perspectives on Human Development*. Thousand Oaks, CA: Sage.
- Bronfenbrenner, Urie and Pamela Morris. 1998. "The Ecology of Developmental Processes." In W. Damon (Series Ed.) and R. Lerner (Vo. Ed.), *Handbook of Child Psychology: Vol. 1: Theoretical Models of Human Development* (5th ed., pp.992-1028). New

York: Wiley.

Brooks-Gunn, Jeanne and Greg J. Duncan. 1997. "The Effects of Poverty on Children." *The Future of Children*. 7(2): 55-71.

Brown, Rachel and Jane Ogden. 2004. "Children's Eating Attitudes and Behaviour: A Study of the Modeling and Control Theories of a Parental Influence." *Health Education Research* 19:261-71.

Burchinal, Margaret R., Joanne E. Roberts, Stephen Hooper and Susan A. Zeisel. 2000. "Cumulative Risk and Early Cognitive Development: A Comparison of Statistical Risk Models" *Developmental Psychology* 36(6): 793-807.

Carta, Judith J., Jane B. Atwater, Charles R. Greenwood, Scott R. McConnell, Mary A. McEvoy and Rosanne Williams. 2001. "Effects of Cumulative Prenatal Substance Exposure and Environmental Risks on Children's Developmental Trajectories." *Journal of Clinical Child Psychology* 30(3): 327-337.

Casas, Yoli G., Brian C. Schiller, Christopher A. DeSouza and Douglas R. Seals. 2001. "Total and Regional Body Composition Across Age in Healthy Hispanic and White Women of Similar Socioeconomic Status." *American Journal of Clinical Nutrition* 73(1):13-8.

Centers for Disease Control and Prevention (CDC). 2004. "Overweight Children and Adolescents: Recommendations to Screen, Assess and Manage." Atlanta, GA: Centers for Disease Control, Retrieved April 10, 2004 (www.cdc.gov/nccdphp/dnpa/growthcharts/training/modules/module3/text/module3print.pdf).

Centers for Disease Control and Prevention (CDC). 2006. "BMI—Body Mass Index." Atlanta, GA: Centers for Disease Control, Retrieved September 21, 2006 (<http://www.cdc.gov/nccdphp/dnpa/bmi/index.htm>).

Ceru-Bjork, C., I. Andersson and S. Rossner. 2001. "Night Eating and Nocturnal Eating—Two Different or Similar Syndromes Among Obese Patients?" *International Journal of Obesity* 25: 365–372.

Chase-Lansdale, Lindsay and Jeanne Brooks-Gunn (Eds.). 1995. *Escape from Poverty: What Makes a Difference for Children?* New York: Cambridge University Press.

Cole, Tim J., Mary C. Bellizzi, Katherine M. Flegal, William H. Dietz. 2000. "Establishing a Standard Definition for Child Overweight and Obesity Worldwide: International Survey." *British Medical Journal* 320: 1–6.

Crossman, Ashley, Deborah Anne Sullivan and Mary Benin. 2006. "The Family Environment and American Adolescents' Risk of Obesity as Young Adults." *Social Science and Medicine* 63:2255-2267.

Crouch, Julie L. and Joel S, Milner. 1993. "Effects of Child Neglect on Children." *Criminal Justice and Behavior* 20(1): 49-65.

Dannefer, Dale. 1987. "Aging as Intracohort Differentiation: Accentuation, the Matthew Effect, and the Life Course." *Sociological Forum* 2:211-37.
First citation in article, CrossRef

Dannefer, Dale. 2003. "Cumulative Advantage/Disadvantage and the Life Course: Cross - Fertilizing Age and Social Science Theory." *Journal of Gerontology B* 58:S327-S337.

Deater-Deckard, Kirby, Kenneth A. Dodge, John E. Bates and Gregory S. Pettit. 1998. "Multiple risk factors in the Development of Externalizing Behavior Problems: Group and Individual Differences." *Development and Psychopathology* 10(3): 469-493.

Dekovic, Maja. 1999. Risk and Protective Factors in the Development of Problem Behavior During Adolescence. *Journal of Youth and Adolescence* 28(6): 667-685.

DiPrete, Thomas A. and Gregory M. Eirich. 2006. "Cumulative Advantage as a Mechanism for Inequality: A Review of Theoretical and Empirical Developments." *Annual Review of Sociology* 32: 271-297

Drewnowski, Adam and S.E. Specter. 2004. "Poverty and Obesity: the Role of Energy Density and Energy Costs." *American Journal of Clinical Nutrition*, 79:6-16.

Duncan, Greg J., Jeanne Brooks-Gunn, and Pamela Kato Klebanov. 1994. "Economic Deprivation and Early Childhood Development." *Child Development* 65: 296-318.

Duncan, Greg J., W. Jean Yeung, Jeanne Brooks-Gunn and Judith R. Smith. 1998. "How Much Does Childhood Poverty Affect the Life Chances of Children?" *American Sociological Review* 63(3): 406-423.

Elder Jr., Glen H. 1997. "The Life Course and Human Development," Pp. 939-991 In *Handbook of Child Psychology, Volume 1: Theoretical Models of Human Development*, edited by R. M. Lerner and W. Damon. New York: Wiley.

Elder Jr., Glen H. 1998. "The Life Course as Developmental Theory." *Child Development* 69:1-12.

Elder Jr., Glen H., Jacquelynne S. Eccles, Monkica Ardel and Sarah Lord. 1995. "Inner-City Parents Under Economic Pressure: Perspectives on the Strategies of Parenting." *Journal of Marriage and the Family* 57(3): 771-784.

Elder Jr., Glen H., Tri Van Nguyen and Avshalom Caspi. 1985. "Linking Family Hardship to Children's Lives." *Child Development* 56: 361-375.

Epstein Leonard H., Brian E. Saelens, Jodi Giancola O'Brien. 1995. "Effects of Reinforcing Increases in Activity versus Decreases in Sedentary Behavior in Obese Children." *International Journal of Behavioral Medicine* 2:41-50.

Epstein Leonard H., Brian E. Saelens, Michelle D. Myers and Dominica Vito. 1997. "Effects of Decreasing Sedentary Behaviors on Activity Choice in Obese Children." *Health Psychology* 16:107-113.

Evans, Gary W. 2003. "A Multimethodological Analysis of Cumulative Risk and Allostatic Load Among Rural Children." *Developmental Psychology* 39(5): 924-933.

Evans, Gary W. 2004. "The Environment of Childhood Poverty." *American Psychologist*. 59(2):77-92.

Evans, Gary W. and Lyscha A. Marcynyszyn. 2004. "Environmental Justice, Cumulative Risk, and Health Among Low-Income Children in Upstate New York." *American Journal of Public Health* 94(11): 1942-1944.

Flegal, K. M., M. D. Carroll, R. J. Kuczmarski and C. L. Johnson. 1998. "Overweight and Obesity in the United States: Prevalence and Trends, 1960–1994." *International Journal of Obesity and Related Metabolic Disorders* 22:39–47.

Flegal, Katherine M., Margaret D. Carroll, Cynthia L. Ogden and Clifford L. Johnson. 2002. "Prevalence and Trends in Obesity Among US Adults, 1999-2000." *JAMA* 288(14): 1723-1727.

French, S. A., L. Harnack and R. W. Jeffrey. 2000. "Fast Food Restaurant Use Among Women in the Pound Prevention Study; Dietary, Behavioral and Demographic Correlates." *International Journal of Obesity* 24:1353-1359.

Fulkerson, Jayne A., Dianne Neumark-Sztainer, Peter J. Hannan and Mary Story. 2008. "Family Meal Frequency and Weight Status Among Adolescents: Cross-sectional and 5-year Longitudinal Associations." *Obesity* 16(11): 2529–2534. (doi:10.1038/oby.2008.388).

Furstenberg, Frank, F., Jr., Thomas D. Cook, Jacquelynne Eccles, Glenn H. Elder, Jr. and Arnold Sameroff. 1999. *Managing to Make It: Urban Families and Adolescent Success*. Chicago: University of Chicago Press.

Gangwisch, James E., Dolores Malaspina, Bernadette Boden-Albala and Steven B. Heymsfield. 2005. "Inadequate Sleep as a Risk Factor for Obesity: Analyses of the NHANES I." *Sleep* 28:1289-1296.

Gephart, Martha A. 1997. "Neighborhood and Communities as Contexts for Development." Pp. 1-43. In *Neighborhood Poverty: Context and Consequences for Children Volume I*, edited by J. Brooks-Gunn, G. J. Duncan and J. L. Aber, New York, NY: Russell Sage Foundation.

Garbarino, James and Kathleen Kostelny. 1996. "The Effects of Political Violence on Palestinian Children's Behavior Problems: A Risk Accumulation Model." *Child Development* 67(1):33-45.

Gibson, Diane. 2004. "Long-Term Food Stamp Program Participation is Differentially Related to Overweight in Young Girls and Boys." *Journal of Nutrition* 134:372-9.

Goodman, Elizabeth. 1999. "The Role of Socioeconomic Status Gradients in Explaining Differences in US Adolescents' Health." *American Journal of Public Health* 89(10):1522-8.

Gordon-Larsen, Penny, Linda S. Adair and Barry M. Popkin. 2003. "The Relationship of Ethnicity, Socioeconomic Factors, and Overweight in U.S. Adolescents." *Obesity Research* 11(1):121-9.

Gordon-Larsen, Penny, Linda S. Adair, Melissa C. Nelson and Barry M. Popkin. 2004a. "Five-Year Obesity Incidence in the Transition Period between Adolescence and Adulthood: The National Longitudinal Study of Adolescent Health." *American Journal of Clinical Nutrition* 80:569-75.

Gordon-Larsen, Penny, Robert G. McMurray and Barry M. Popkin. 2000. "Determinants of Adolescent Physical Activity and Inactivity Patterns." *Pediatrics* 105:1-8.

Gordon-Larsen, Penny, Melissa C. Nelson and Barry M. Popkin. 2004b. "Longitudinal Physical Activity and Sedentary Behavior Trends: Adolescence to Adulthood." *American Journal of Preventive Medicine* 27:277-283.

Gordon-Larsen, Penny, Melissa C. Nelson and Barry M. Popkin. 2006. "Inequality in the Built Environment Underlies Key Health Disparities." *Pediatrics*:117(2):417-424.

Gortmaker S. L., A. M. Sobal, K. Peterson, C. A. Colditz and W. H. Dietz. 1996. "Television Viewing as a Cause of Increasing Obesity among Children in the United States, 1986-1990." *Archives of Pediatrics and Adolescent Medicine* 150:536-562.

Gustafson, T.B. and D.B. Sarwer. 2004. "Childhood Sexual Abuse and Obesity." *Obesity Reviews* 5:129-135.

Halfon, Neal and Miles Hochstein. 2002. "Life Course Health Development: an Integrated Framework for Developing Health, Policy, and Research." *Milbank Quarterly* 80:433-79.

Haas, Jennifer S., Lisa B. Lee, Celia P. Kaplan, Dean Sonneborn, Kathryn A. Phillips and Su-Ying Liang. 2003. "The Association of Race, Socioeconomic Status, and Health Insurance Status with Prevalence of Overweight Among Children and Adolescents." *American Journal of Public Health* 93(12):2105-10.

Hannon, Lance E. 2005. "Extremely Poor Neighborhoods and Homicide." *Social Science*

Quarterly 86:1418-1434.[javascript:popRef\('a1'\)](#)

Harris, Kathleen Mullan, Francesca Florey, Joyce W. Tabor, Peter S. Bearman, Jo Jones, and J. Richard Udry. 2003. The National Longitudinal Study of Adolescent Health: Research Design. Retrieved September 21, 2004 (<http://www.cpc.unc.edu/projects/addhealth/design.html>.)

Harris, Kathleen Mullan, Penny Gordon-Larsen, Kim Chantala, J. Richard Udry. 2006. "Longitudinal Trends in Race and Ethnic Disparities in Leading Health Indicators from Adolescence to Young Adulthood." *Archives of Pediatrics and Adolescent Medicine* 160:74-81.

Heatherton, Tod F., and Roy F. Baumeister. 1991. "Binge Eating as Escape from Self-Awareness." *Psychological Bulletin* 110:86-108.

Heer, David M. 1986. "Effect of Number, Order, and Spacing of Siblings on Child and Adult Outcomes: An Overview of Current Research." *Social Biology* 33:1-4 .

Hogan, Dennis P. and Evelyn M. Kitagawa. 1985. "The Impact of Social Status, Family Structure, and Neighborhood on the Fertility of Black Adolescents." *American Journal of Sociology* 90(4):825-55.

Jones, Deborah J., Rex Forehand, Gene Brody and Lisa Armisted. 2002. "Psychosocial Adjustment of African American Children in Single-Mother Families: A Test of Three Risk Models." *Journal of Marriage and the Family* 64: 105-115.

Kant, A. 1995. "Frequency of Eating Occasions and Weight Change in NHANES I Epidemiologic Follow-up Study." *International Journal of Obesity* 19: 468-474.

Kimm, Sue Y.S., Eva Obarzanek, Bruce A. Barton, Christopher E. Aston, Shari L. Similo, John A. Morrison, Z. I. Sabry, George B. Schreiber and Robert P. McMahon. 1996. "Race, Socioeconomic Status, and Obesity in 9-to-10 year-old Girls: The NHLBI Growth and Health Study." *Annals of Epidemiology* 6:266-275.

Klesges Robert C., Risa J. Stein, Linda H. Eck, Terry R. Isbell and Lisa M. Klesges. 1991. "Parental Influence on Food Selection in Young Children and Its Relationships to Childhood Obesity." *American Journal of Clinical Nutrition* 53:859-64.

Kling, Jeffrey R., Jens Ludwig and Lawrence F. Katz. 2005. "Neighborhood Effects on Crime for Female and Male Youth: Evidence from a Randomized Housing Voucher Experiment." *Quarterly Journal of Economics* 120(1): 87-130.

Kohatsu, Neal D., Rebecca Tsai, Terry Young, Rachel VanGilder, Leon F. Burmeister, Ann M. Stromquist and James A. Merchant. 2006. "Sleep Duration and Body Mass Index in a Rural Population." *Archives of Internal Medicine* 66:1701-5.

Kuczmariski, R.J., K.M. Flegal, S.M. Campbell and C.L. Johnson. 1994. "Increasing Prevalence of Overweight Among US Adults: the National Health and Nutrition Examination Surveys, 1960 to 1991." *Journal of the American Medical Association* 272:205-211.

Kumanyika, Shiriki and Sonya Grier. 2006. "Targeting Interventions for Ethnic Minority and Low-Income Populations." *The Future of Children* 16(1):187-207.

Kutner, Michael H., Christopherr J. Nachtsheim and John Neter. 2004. *Applied Linear Regression Models*. New York: Mcgraw-Hill Irwin.

Lareau, Annette. 2002. "Invisible Inequality: Social Class and Childrearing in Black Black Families and White Families," *American Sociological Review* 67: 747-776.

Lareau, Annette. 2003. *Unequal Childhoods: Class, Race, and Family Life*. Berkeley, CA: University of California Press.

Lee, Hedwig, Kathleen Mullan Harris and Penny Gordon-Larsen. 2008. "Life Course Perspectives on the Links Between Poverty and Obesity During the Transition to Young Adulthood." *Population Research and Policy Review*. [Forthcoming]

Liaw, Fong-Ruey and Jeanne Brooks-Gunn. 1994. "Cumulative Familial Risks and Low-Birthweight Children's Cognitive and Behavioral Development" *Journal of Clinical Child Psychology* 23(4): 360-372.

Long, Scott J. 1997. *Regression Models for Categorical and Limited Dependent Variables*. Thousand Oaks, CA: SAGE Publications Inc.

Lutfiyya May Nawal, Martin S. Lipsky, Jennifer Wisdom-Behounek and Melissa Inpanbutr-Martinkus. 2007. "Is Rural Residency a Risk Factor for Overweight and Obesity for U.S. Children?" *Obesity* 15:2348-2356.

Luthar, Suniya S. 1993. "Annotation: Methodological and Conceptual Issues in Research on Childhood Resilience." *Journal of Child Psychology & Psychiatry* 34(4): 441-453.

Lynch, John and George D. Smith. 2005. "A Life Course Approach to Chronic Disease Epidemiology." *Annual Review of Public Health* 26:1-35.

Malina, Robert M. 1996. "Tracking of Physical Activity and Physical Fitness Across the Lifespan". *Research Quarterly for Exercise and Sports* 67(Suppl. 3):S48-S57.

McCrorry, Megan A., Paul J. Fuss, Joy E. McCallum, Manjiang Yao, Angela G. Vinken, Nicholas P. Hays and Susan B. Roberts. 1999. "Dietary Variety within Food Groups: Association with Energy Intake and Body Fatness in Men and Women." *American Journal of Clinical Nutrition* 69: 440-447.

McLanahan, Sara S. 1995. "The Consequences of Nonmarital Childbearing for Women."

Pp. 229-39 in *Report to Congress on Out-of-Wedlock Childbearing*. DHHS Pub (PHS) 95-1257. Hyattsville, MD: National Center for Health Statistics.

McLoyd, Vonnie C. 1998. "Socioeconomic Disadvantage and Child Development." *American Psychologist* 53(2): 185-204.

McMurray, Robert G., Joanne S. Harrell, Shibing Deng, Chyrise B. Bradley, Lori M. Cox and Shrikant I. Bangdiwala. 2000. "The Influence of Physical Activity, Socioeconomic Status, and Ethnicity on the Weight Status of Adolescents." *Obesity Research* 8(2):130-9.

McTigue Kathleen M., Joanne M. Garrett, Barry M. Popkin. 2002. "The Natural History of Obesity: Weight Change in a Large US Longitudinal Survey." *Annals of Internal Medicine* 136:857-64.

Merton, Robert K. 1968. "The Matthew Effect in Science." *Science* 159:56-63.

Miech, Richard A., Shiriki K. Kumanyika, Nicolas Stettler, Bruce G. Link, Jo C. Phelan and Virginia W. Chang. 2006. "Trends in the Association of Poverty with Overweight among US Adolescents, 1971-2004." *Journal of the American Medical Association* 295:2385-2393.

Moore, Kristin Anderson, Sharon Vandivere and Jennifer Ehrle. 2000. "Sociodemographic Risk and Child Well-Being." (http://www.urban.org/UploadedPDF/anf_b18.pdf) Urban Institute Policy Brief.

Must, A., S.L. Gortmaker and W.H. Dietz. 1994 "Risk Factors for Obesity in Young Adults: Hispanic, African American and Whites in the Transition Years, age 16-28 Years." *Biomedicine and Pharmacotherapy* 48:143-156.

National Heart, Lung, and Blood Institute (NHLBI) Obesity Education Initiative. 1998. "NHLBI Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. Clinical Guidelines on the Identification, evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report." *Obesity Research* 6(Supplement): 51S-209S.

Nelson, Jennifer A., Mary Ann Chiasson and Viola Ford. 2004. "Childhood Overweight in New York City WIC Population." *Research and Practice* 94(3): 458-462.

Newacheck, Paul W., Margaret McManus, Harriette B. Fox, Yun-Yi Hung and Neal Halfon. "Access to Health Care for Children with Special Health Care Needs." *Pediatrics* 105(4): 760-766.

O'Rand, Angela M. 1996. "The Precious and the Precocious: Understanding Cumulative Disadvantage and Cumulative Advantage over the Life Course." *The Gerontologist* 36:230-38.

Paeratakul, S., J. C. Lovejoy, D. H. Ryan and G. A. Bray. 2002. "The Relation of Gender,

Race and Socioeconomic Status to Obesity and Obesity Comorbidities in a Sample of US Adults.” *International Journal of Obesity* 26: 1205-1210.

Patel, Sanjay R., Atul Malhotra, David P. White, Daniel J. Gottlieb and Frank B. Hu. 2006. “Association between Reduced Sleep and Weight Gain in Women.” *American Journal of Epidemiology* 164(10): 947-954.

Patterson, M.L., Stern, S. Stern, P.B. Crawford, R.P. McMahon, S.L. Similo, G.B. Schreiber, J.A. Morrison and M.A. Waclawiw. 1997. “Sociodemographic Factors and Obesity in Preadolescent Black and White Girls: NHLBI’s Growth and Health Study.” *Journal of the National Medical Association* 89: 594-600.

Raftery, Adrian. E. 1995. “Bayesian Model Selection in Social Research.” *Sociological Methodology* 25:111-163.

Raitakan Olli T., Kimmo V. K. Porkka, Simo Taimela, Risto Telama, Leena Räsänen and Jorma S. Vllkari. 1994. “Effects of Persistent Physical Activity and Inactivity on Coronary Risk Factors in Children and Young Adults: The Cardiovascular Risk in Young Finns Study.” *American Journal of Epidemiology* 140:195-205.

Reilly, John J., Julie Armstrong, Ahmad R. Dorosty, Pauline M. Emmett, A. Ness, I. Rogers, Colin Steer and Andrea Sherriff, for the Avon Longitudinal Study of Parents and Children Study Team. 2005. “Early Life Risk Factors for Obesity in Childhood: Cohort Study.” *British Medical Journal* 330:1357 Online. Retrieved September 21, 2006 (doi:10.1136/bmj.38470.670903.E0).

Risch, Neil, Esteban Burchard, Elad Ziv and Hua Tang. 2002. “Categorization of Humans in Biomedical Research: Genes, Race and Disease.” *Genome Biology* 3(7): Online. Retrieved September 21, 2006 (<http://genomebiology.com/2002/3/7/comment/2007>)

Robinson, Thomas N., Michaela Keirnan, Donna M. Matheson and K. Farish Haydel. 2001. “Is Parental Control Over Children’s Eating Associated with Childhood Obesity? Results from a Population-Based Sample of Third Graders.” *Obesity Research* 9:306-312.

Rosenheck, R. 2008. “Fast Food Consumption and Increased Caloric Intake: A Systematic Review of a Trajectory Towards Weight Gain and Obesity Risk.” *Obesity Reviews* 9(6):535-547.

Rutter, Michael. 1979. “Protective Factors in Children’s Responses to Stress and Disadvantage.” In M.W. Kent and J.E. Rolf (Eds.), *Primary Prevention of Psychopathology, Vol.3: Social Competence in Children* (pp.49-74).

Rutter, Michael. 1983. “Statistical and Personal Interactions: Facets and Perspectives.” In D. Magnusson and V. Allen (Eds.), *Human Development: An Interactional Perspective* (pp. 295-319). New York, NY: Academic Press.

Rutter, Michael. 1993. "Stress, Coping and Development." In N. Garmezy and M. Rutter (Eds.), *Stress, Coping and Development in Children* (pp.1-41). New York, NY: McGraw-Hill.

Rutter, M, A. Cox, C. Tupling, M. Berger and W. Yule. 1975. "Attainment and Adjustment in Two Geographical Areas, 1: The Prevalence of Psychiatric Disorder." *British Journal of Psychiatry* 126: 493-509.

Rutter, M. and D. Quinton. 1977. "Psychiatric Disorder: Ecological Factors and Concepts of Causation." In: McGurk H., ed. *Ecological Factors in Human Development*. Amsterdam, the Netherlands: North-Holland Publishing Co. 173-187.

Rutter, M.; Tizard, J.; Yule, W.; Graham, P. and Whitmore, K. 1976. "Research Report: Isle of Wight Studies: 1964-1974." *Psychosomatic Medicine*, 6:313-332.

Sameroff, Arnold J., Ronald Seifer, Ralph Barocas, Melvin Zax and Stanley Greenspan. 1987a. "Intelligence Quotient Scores of 4-Year-Old Children: Social-Environmental Risk Factors." *Pediatrics* 79(3): 343-350.

Sameroff, Arnold, Ronald Seifere, Melvin Zax and Ralph Barocas. 1987b. "Early Indicators of Developmental Risk: Rochester Longitudinal Study." *Schizophrenia Bulletin* 13:383-394.

Sameroff, A.J., Bartko, W.T., Baldwin, A. Baldwin, C., and Seifer, R. 1998. "Family and Social Influences on the Development of Child Competence." In M. Lewis and C. Feiring (Eds.), *Families, Risk, and Competence* (pp.161-183). Mahwah, NJ: Erlbaum.

Sameroff, Arnold J. 2000. "Dialectical Processes in Developmental Psychopathology." In A. Sameroff, M. Lewis and S. Miller (Eds.), *Handbook of Developmental Psychopathology* (2nd edn, pp.23-40). New York: Kluwer Academic/Plenum Publishers.

Schell, Lawrence M. 1997. "Culture as a Stressor: A Revised Model of Biocultural Interaction." *American Journal of Physical Anthropology* 102: 67-77.

Schoenborn, Charlotte A., Patricia F. Adams, and Patricia M. Barnes. 2002. "Body Weight Status of Adults: United States, 1997-98." *Advance Data* 330:1-15.

Seibold Esther S., Kathleen Knafl and Margaret Grey. 2003. "The Family Context of an Intervention to Prevent Type 2 Diabetes in High-Risk Teens." *Diabetes Education* 29:997-1004.

Selway, Janet S. 2006. "Childhood Maltreatment and Adult Obesity." *Bariatric Nursing and Surgical Patient Care* 1(4): 273-282.

Shanahan, Michael J. 2000. "Pathways to Adulthood in Changing Societies: Variability and Mechanisms in Life Course Perspective." *Annual Review of Sociology* 26:667-692.

Sobal, Jeffery and Albert J. Stunkard. 1989. "Socioeconomic Status and Obesity: A Review of the Literature." *Psychological Bulletin* 105:260–275.

Speechly, D. P., G. G. Rogers and R. Buffenstein. 1999. "Acute Appetite Reduction Associated with an Increased Frequency of Eating in Obese Males." *International Journal of Obesity* 23:1151–1159.

Spiegel Karine, Rachel Leproult, Mireille L'Hermitte-Balériaux, Geroges Copinschi, Plamen D. Penev and Eve Van Cauter. 2004a. "Leptin Levels are Dependent on Sleep Duration: Relationships with Sympathovagal Balance, Carbohydrate Regulation, Cortisol, and Thyrotropin." *The Journal of Clinical Endocrinology and Metabolism* 89(11): 5762-5771.

Spiegel, Karine, Esra Tasali, Plamen Penev and Eve Van Cauter. 2004b. "Brief Communication: Sleep Curtailment in Healthy Young Men Is Associated with Decreased Leptin Levels, Elevated Ghrelin Levels, and Increased Hunger and Appetite." *Annals of Internal Medicine* 141(11): 846-850.

Spraggins, René E. 2003. "Women and Men in the United States: March 2002 Population Characteristics." (<http://www.census.gov/prod/2003pubs/p20-544.pdf>) March 2003 Current Population Reports, Census Bureau, Washington: DC.

Stanton-Chapman, Tina L. Derek A. Chapman, Ann P. Kaiser and Terry B. Hancock. 2004. "Cumulative Risk and Low-Income Children's Language Development." *Topics in Early Childhood Special Education* 24(4):227-37.

Stunkard, Albert J. 1993. "Obesity and Socioeconomic Status—A Complex Relation." *The New England Journal of Medicine* 329:1036-7.

Sundquist Jan and Sven-Erik Johansson. 1998. "The Influence of Socioeconomic Status, Ethnicity and Lifestyle on Body Mass Index in a Longitudinal Study." *International Journal of Epidemiology* 27(1):57-63.

Taheri, Shahradsad, Ling Lin, Diane Austin, Terry Young and Emmanuel Mignot. 2004. "Short Sleep Duration is Associated with Reduced Leptin, Elevated Ghrelin, and Increased Body Mass Index." *Plos Medicine* 1(3): Online. Retrieved September 21, 2006 (e62 doi:10.1371/journal.pmed.0010062).

Timlin, Maureen T., Mark A. Pereira, Mary Story, and Dianne Neumark-Sztainer. 2008. "Breakfast Eating and Weight Change in a 5-Year Prospective Analysis of Adolescents: Project EAT (Eating Among Teens)." *Pediatrics* 121(3): e638-e645. (DOI:10.1542/peds.2007-1035).

Townsend Marilyn S., Janet Peerson, Bradley Love, Cheryl Achterberg and Suzanne P. Murphy. 2001. "Food Insecurity is Positively Related to Overweight in Women." *Journal of Nutrition* 131:1738-1745.

U.S. Census Bureau, Geography Division. 1994. "Geographic Areas Reference Manual." Geography Division, Bureau of the Census, Washington, DC. Retrieved October 18, 2008. (<http://www.census.gov/geo/www/garm.html>).

U.S. Department of Health and Human Services (DHHS). 2000. "Healthy People 2010." 2nd Ed. Washington: DC: US Government Printing Office.

Wang, Youfa. 2001. "Cross-national Comparison of Childhood Obesity: the Epidemic and the Relationship between Obesity and Socioeconomic Status." *International Journal of Epidemiology* 30:1129-1136.

Whitaker, Robert C., Jeffrey A. Wright, Margaret S. Pepe, Kristy D. Seidel and William H. Dietz. 1997. "Predicting Obesity in Young Adulthood from Childhood and Parental Obesity." *New England Journal of Medicine* 337:869-873.

Wiederman, Michael W., Randy A. Sansone and Lori A. Sansone. 1999. "Obesity Among Sexually Abused Women: An Adaptive Function for Some?" *Women and Health* 29(1): 89-100.

Williamson, D.F., T. J. Thompson, R. F. Anda, W. H. Dietz and V. Felitti. 2002. "Body Weight and Obesity in Adults and Self-Reported Abuse in Childhood." *International Journal of Obesity* 26:1075-1082.

Wilson, William Julius. 1987. *The Truly Disadvantaged*. Chicago: IL University of Chicago Press.

Winkleby, Marilyn A., Thomas N. Robinson, Jan Sundquist, and Helena C. Kraemer . 1999 "Ethnic Variation in Cardiovascular Disease Risk Factors Among Children and Young Adults: Findings from the Third National Health and Nutrition Examination Survey, 1988–1994." *Journal of the American Medical Association* 281:1006–1013.

World Health Organization (WHO). 1995. "Physical Status: The Use and Interpretation of Anthropometry: Report of a WHO Expert Committee" Geneva, Switzerland: World Health Organization (WHO Technical Report Series, No. 854), Retrieved September 21, 2006 (http://whqlibdoc.who.int/trs/WHO_TRS_854.pdf).

Yang, Rea-Jeng, Edward K. Wang, Yeu-Sheng Hsieh and Mei-Yen Chen. 2006. "Irregular Breakfast Eating and Health Status Among Adolescents in Taiwan." *BMC Public Health*. Online. Retrieved September 21, 2006 (<http://www.biomedcentral.com/1471-2458/6/295>)

Zhang, Qi. and Youfa Wang. 2003. "Socioeconomic Inequality of Obesity in the United States: Do Gender, Age and Ethnicity Matter?" *Social Science and Medicine* 58(6):1-17.

Figure 2.1 Conceptual Model

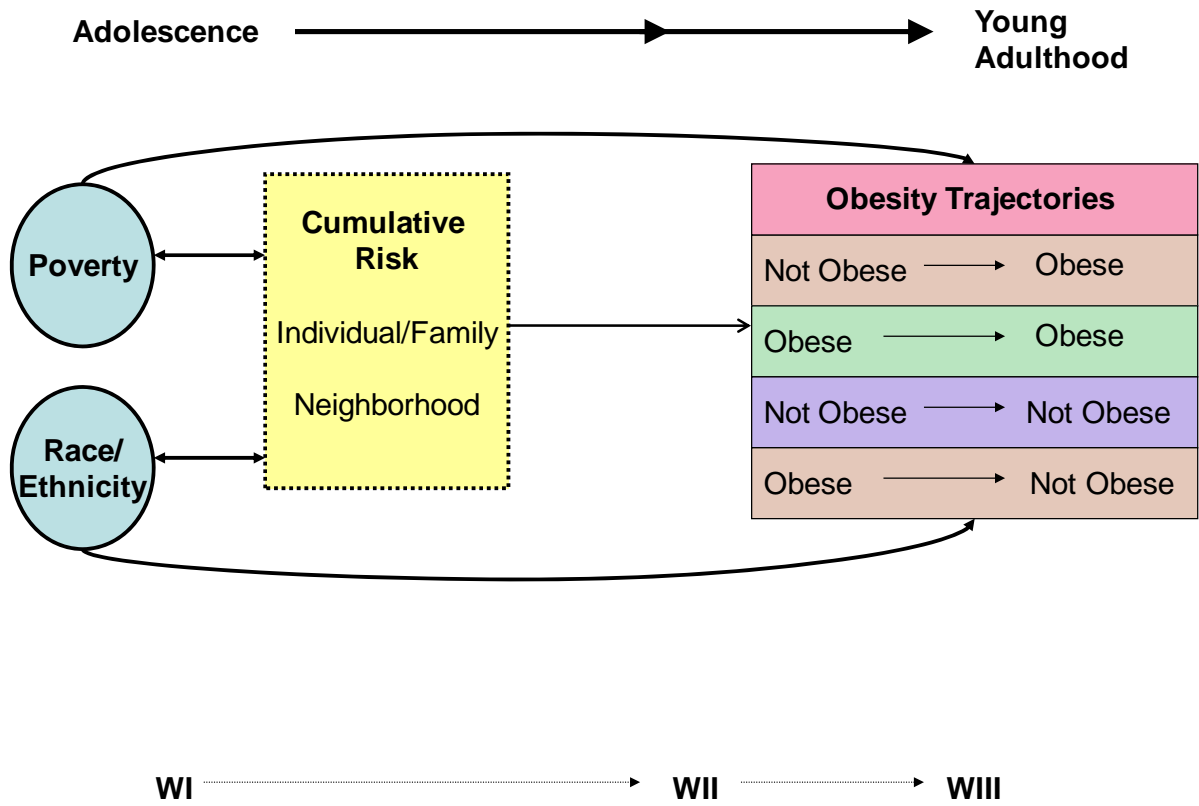


Table 2.1. Variable Descriptions, Means and Standard Deviations by Sex

Variable	Description	Female		Male	
		Mean	SE	Mean	SE
OUTCOME MEASURES					
<i>Change in Obesity Status</i>					
Become Obese	Not obese at WII but obese at WIII	0.13	0.009	0.11	0.007
Stay Obese	Obese at WII & obese at WIII	0.10	0.009	0.09	0.008
Stay Non-Obese	Not obese at WII & not obese at WIII	0.76	0.014	0.77	0.012
Reduce Obese	Obese at WII but not obese at WIII or	0.01	0.002	0.02	0.004
<i>Static Measures of Obesity</i>					
Obese at Wave II	Obese at Wave II	0.11	0.009	0.12	0.009
Obese at Wave III	Obese at Wave III	0.23	0.013	0.21	0.012
SOCIAL DISADVANTAGE MEASURES					
<i>Minority Status</i>					
Minority Status	Respondent reported black or Hispanic	0.26	0.028	0.26	0.028
Non-Hispanic White (Reference Category)	Respondent reported white	0.74	0.028	0.74	0.028
Non-Hispanic Black	Respondent reported black	0.14	0.021	0.14	0.020
Hispanic	Respondent reported Hispanic	0.11	0.018	0.12	0.020
Welfare/Poverty Status	Welfare receipt prior to the age of 18 and/or Family Income less than \$16,000/year	0.29	0.018	0.28	0.019
RISK MEASURES					
<i>Individual Level Risk</i>					
Skips Breakfast	Respondent reported usually skips breakfast at WI and reported skips breakfast 0-2 days in a week	0.13	0.007	0.09	0.008
Inadequate Sleep	Respondent reported usually sleeping less than 7 hours at WI and WII	0.27	0.013	0.20	0.012
Low Self Esteem	WI measure of low-self-esteem	0.26	0.010	0.16	0.009
<i>Family Level Risk</i>					
Parent Obese	Report of mother or father being obese	0.24	0.010	0.24	0.010
Trouble Paying Bills	Parent reports not having money to pay the bills at WI	0.17	0.012	0.16	0.013
No Health Insurance	Parent reports respondent has no health insurance at WI	0.12	0.012	0.10	0.009
Single of Surrogate Parent Family	Single mother, single father or other family structure [<i>Reference Category: 2 Biological/Adoptive parents or step family with one biological parent</i>]	0.23	0.013	0.24	0.014
Large Number of Siblings	Respondent has more than 2 siblings	0.14	0.012	0.14	0.008
Parent Education Less than High School	Highest educated parent completed less than high school or GED	0.10	0.011	0.09	0.012
Lack of Parent TV and Food Monitoring	Parent does not monitor how much TV respondent viewed or what respondent eats at WI	0.69	0.013	0.66	0.016
N		3,371		3,122	

Data are weighted.

Table 2.1 Cont. Variable Descriptions, Means and Standard Deviations by Sex

Variable	Description	Female		Male	
		Mean	SE	Mean	SE
RISK MEASURES					
<i>Family Level Risk Cont.</i>					
No Curfew	Respondent reports having no curfew at WI.	0.25	0.010	0.34	0.014
Unshared Family Meals	Parents eat with respondent less than 4 times/week at WI	0.26	0.013	0.26	0.013
Lack of Parent-Child Interaction	Respondent reports engaging in less than two activities with parent(s) in the past 4 weeks at WI	0.27	0.012	0.33	0.013
Lack of Parental Presence	Respondent reports parent not present for 1 or more of 3 daily activities: leaving/returning for/from school and going to bed	0.27	0.013	0.23	0.011
High Parent-Child Conflict	Respondent had serious argument with parent at WI and WII	0.27	0.010	0.22	0.011
Physical Abuse	Respondent reported physical abuse in childhood	0.27	0.012	0.29	0.012
Sexual Abuse	Respondent reported sexual abuse in childhood	0.04	0.005	0.04	0.005
<i>Neighborhood Level Risk</i>					
High Proportion of Female Headed Households	Respondents live in a block group with 20% or more female headed household with own children < 18 yr. old at WI	0.24	0.028	0.23	0.027
High Neighborhood Poverty	Respondent lives in a block group with 20% or more families below poverty at WI	0.18	0.024	0.19	0.026
High Neighborhood Unemployment	Respondent lives in a block group where the total unemployment rate is greater than or equal to 10%	0.19	0.027	0.18	0.025
Low Neighborhood Housing Quality	Respondent lives in a block group with 10% or more of the housing units are vacant at WI	0.25	0.027	0.25	0.025
High County Level Crime	Respondent lives in a county where crime rates are greater than 7,500/100,000 at WI	0.17	0.033	0.18	0.034
Neighborhood Unsafe	Respondent reports that their neighborhood is unsafe	0.10	0.009	0.08	0.009
<i>Control Measures</i>					
Age at Wave I	Age at WI	14.87	0.121	15.09	0.126
Urban Tract	Respondent lives in an urbanized area	0.51	0.047	0.48	0.046
N		3,371		3,122	

Data are weighted.

Table 2.2. Associations Between Risk Measures and Obesity Status at Waves II and III by Sex (Bivariate Odds Ratios)

	Female		Male	
	Obese Wave II	Obese Wave III	Obese Wave II	Obese Wave III
SOCIAL DISADVANTAGE MEASURES				
Minority Status	1.82** (0.306)	1.78*** (0.239)	1.01 (0.170)	1.00 (0.138)
Welfare/Poverty Status	1.86*** (0.311)	1.82*** (0.239)	1.19 (0.226)	1.18 (0.182)
RISK MEASURES				
<i>Individual Level Risk</i>				
Skips Breakfast	1.68* (0.368)	1.49* (0.244)	1.84* (0.437)	2.59*** (0.442)
Inadequate Sleep	1.14 (0.198)	1.46** (0.153)	1.67** (0.279)	1.48** (0.203)
Low Self Esteem	1.63** (0.294)	1.59*** (0.175)	1.37 (0.276)	1.14 (0.175)
<i>Family Level Risk</i>				
Parent Obese	4.30*** (0.677)	2.80*** (0.294)	3.55*** (0.503)	2.76*** (0.314)
Trouble Paying Bills	1.55* (0.328)	1.43* (0.213)	1.78* (0.416)	1.25 (0.227)
No Health Insurance	1.03 (0.245)	1.33 (0.207)	1.04 (0.294)	1.27 (0.221)
Single of Surrogate Parent Family	1.39* (0.235)	1.45* (0.210)	1.26 (0.217)	1.08 (0.145)
Large Number of Siblings	1.13 (0.260)	1.10 (0.168)	0.50** (0.114)	0.78 (0.138)
Parent Education Less than High School	1.64* (0.420)	2.22*** (0.349)	1.23 (0.309)	1.14 (0.277)
Lack of Parent TV and Food Monitoring	1.12 (0.223)	1.06 (0.120)	1.08 (0.158)	1.07 (0.108)
N	3,371	3,371	3,122	3,122

Data are weighted.

Standard errors are in parentheses

* significant at .05 level

** significant at .01 level

*** significant at .001 level

Table 2.2 Cont. Associations Between Risk Measures and Obesity Status at Waves II and III by Sex (Bivariate Odds Ratios)

	Female		Male	
	Obese Wave II	Obese Wave III	Obese Wave II	Obese Wave III
RISK MEASURES				
<i>Family Level Risk Cont.</i>				
No Curfew	1.55** (0.246)	1.31* (0.150)	1.25 (0.156)	1.08 (0.116)
Unshared Family Meals	1.19 (0.192)	1.04 (0.124)	1.00 (0.175)	1.02 (0.152)
Lack of Parent-Child Interaction	1.26 (0.217)	1.23 (0.143)	1.20 (0.207)	1.05 (0.138)
Lack of Parental Presence	1.03 (0.181)	0.99 (0.105)	1.29 (0.222)	1.09 (0.154)
High Parent-Child Conflict	1.07 (0.167)	1.07 (0.101)	0.91 (0.132)	1.00 (0.113)
Physical Abuse	1.20 (0.21)	1.34** (0.145)	1.04 (0.209)	1.31* (0.178)
Sexual Abuse	0.81 (0.267)	1.45 (0.382)	1.85 (0.672)	1.53 (0.481)
<i>Neighborhood Level Risk</i>				
High % Female Headed Households	1.43* (0.243)	1.62** (0.254)	1.68** (0.281)	1.26 (0.193)
High Neighborhood Poverty	2.09*** (0.380)	2.01*** (0.281)	1.65** (0.295)	1.64** (0.242)
High Neighborhood Unemployment	2.44*** (0.426)	1.95*** (0.336)	1.44 (0.267)	1.54* (0.294)
Low Neighborhood Housing Quality	1.38 (0.236)	1.47* (0.203)	1.14 (0.182)	1.05 (0.142)
High County Level Crime	1.41 (0.293)	1.33 (0.219)	1.13 (0.193)	1.00 (0.146)
Neighborhood Unsafe	1.36 (0.317)	1.39* (0.237)	1.18 (0.272)	1.00 (0.204)
N	3,371	3,371	3,122	3,122

Data are weighted.

Standard errors are in parentheses

* significant at .05 level

** significant at .01 level

*** significant at .001 level

Table 2.3. Variable Means and Standard Deviations for Cumulative Risk Index and Distributions by Sex					
Female			Male		
	Mean	SE	Mean	SE	
Female Cumulative Risk Index			Male Cumulative Risk Index		
Number of Risk Factors			Number of Risk Factors		
0	0.09	0.010	0	0.27	0.019
1	0.19	0.014	1	0.29	0.012
2	0.22	0.012	2	0.20	0.011
3	0.17	0.008	3	0.13	0.012
4	0.14	0.009	4	0.08	0.011
5	0.08	0.008	5+	0.03	0.006
6	0.06	0.006	M-CRI	1.57	0.081
7+	0.06	0.008			
F-CRI	2.88	0.113			
N		3,371			3,122

Data are weighted.

Table 2.4. Variable Means and Standard Deviations for Cumulative Risk Index and Distributions by Sex					
Cumulative Risk Index	Female		Male		
	Mean	SE	Mean	SE	
Female Cumulative Risk Index			Male Cumulative Risk Index		
<i>Number of Individual/Family Risk Factors</i>					
0	0.16	0.010	0	0.37	0.015
1	0.28	0.011	1	0.37	0.012
2	0.26	0.011	2	0.19	0.010
3	0.16	0.008	3	0.06	0.006
4	0.09	0.007	4+	0.01	0.003
5	0.03	0.004			
6+	0.02	0.003			
IF-F-CRI	1.92	0.045	IF -M-CRI	0.97	0.031
<i>Number of Neighborhood Risk Factors</i>					
0	0.50	0.036	0	0.67	0.034
1	0.26	0.021	1	0.16	0.015
2	0.11	0.013	2	0.09	0.014
3	0.08	0.014	3	0.09	0.016
4	0.04	0.007			
5	0.01	0.003			
N-F-CRI	0.96	0.085	N -M-CRI	0.60	0.072
N		3,371			3,122

Data are weighted.

Table 2.5. Mean Cumulative Risk Level by Sex, Race/Ethnicity and Welfare/Poverty Status

Out of 14 Risk Factor F-CRI	Female	Male	Total
Total	2.88 (0.113)	2.73 (0.114)	2.80 (0.109)
White	2.46 (0.105)	2.30 (0.110)	2.38 (0.097)
Black	4.51 (0.194)	4.40 (0.183)	4.45 (0.174)
Hispanic	3.59 (0.173)	3.47 (0.168)	3.52 (0.153)
Poor	4.23 (0.144)	3.97 (0.152)	4.10 (0.130)
Poor White	3.68 (0.153)	3.51 (0.183)	3.60 (0.145)
Poor Black	5.33 (0.249)	5.00 (0.197)	5.17 (0.196)
Poor Hispanic	4.54 (0.220)	4.18 (0.234)	4.36 (0.185)
Non-Poor	2.33 (0.089)	2.24 (0.090)	2.28 (0.084)
Non-Poor White	2.10 (0.088)	1.93 (0.174)	2.02 (0.075)
Non-Poor Black	3.69 (0.174)	3.85 (0.202)	3.77 (0.165)
Non-Poor Hispanic	2.87 (0.183)	3.03 (0.194)	2.96 (0.163)
N	3,371	3,122	6,493

Data are weighted.

Table 2.6. Mean Cumulative Risk Level by Sex, Race/Ethnicity and Welfare/Poverty Status

Out of 8 Risk Factor M-CRI	Female	Male	Total
Total	1.68 (0.087)	1.57 (0.081)	1.63 (0.080)
White	1.39 (0.090)	1.29 (0.074)	1.34 (0.079)
Black	2.89 (0.120)	2.80 (0.131)	2.84 (0.110)
Hispanic	2.07 (0.116)	1.91 (0.093)	1.98 (0.086)
Poor	2.46 (0.105)	2.35 (0.109)	2.41 (0.095)
Poor White	2.09 (0.129)	2.03 (0.131)	2.06 (0.117)
Poor Black	3.27 (0.151)	3.13 (0.146)	3.20 (0.117)
Poor Hispanic	2.59 (0.150)	2.41 (0.130)	2.49 (0.102)
Non-Poor	1.36 (0.074)	1.27 (0.064)	1.31 (0.065)
Non-Poor White	1.18 (0.076)	1.07 (0.056)	1.13 (0.062)
Non-Poor Black	2.50 (0.141)	2.50 (0.151)	2.50 (0.126)
Non-Poor Hispanic	1.68 (0.126)	1.59 (0.145)	1.63 (0.078)
N	3,371	3,122	6,493

Data are weighted.

Table 2.7. Relationship between Cumulative Risk Index and Obesity Outcomes for Females (N=3,371)

Individual and Family CRI	Change in Obesity From WII to WIII							
	Obese at WII		Obese at WIII		Become Obese		Stay Obese	
	Mean	OR	Mean	OR	Mean	OR	Mean	OR
Number of Risk Factors								
0 (Reference Group)	<i>0.025</i>	1.00	<i>0.090</i>	1.00	<i>0.066</i>	1.00	<i>0.025</i>	1.00
1	<i>0.075</i>	3.08*** (0.890)	<i>0.168</i>	2.03** (0.461)	<i>0.102</i>	1.74* (0.483)	<i>0.065</i>	2.91** (0.873)
2	<i>0.105</i>	4.38*** (1.523)	<i>0.236</i>	3.13*** (0.673)	<i>0.139</i>	2.59*** (0.653)	<i>0.097</i>	4.67*** (1.695)
3	<i>0.153</i>	6.69*** (2.593)	<i>0.287</i>	4.07*** (0.940)	<i>0.158</i>	3.27*** (0.865)	<i>0.129</i>	6.72*** (2.715)
4	<i>0.207</i>	9.63*** (3.847)	<i>0.424</i>	7.47*** (1.682)	<i>0.234</i>	5.98*** (1.635)	<i>0.191</i>	12.19*** (4.906)
5	<i>0.211</i>	9.63*** (4.876)	<i>0.368</i>	5.90*** (1.986)	<i>0.174</i>	4.10** (1.751)	<i>0.194</i>	11.04*** (5.983)
6+	<i>0.206</i>	9.40** (6.143)	<i>0.360</i>	5.65*** (2.724)	<i>0.154</i>	3.44* (1.863)	<i>0.205</i>	11.30** (7.697)

Neighborhood CRI	Change in Obesity From WII to WIII							
	Obese at WII		Obese at WIII		Become Obese		Stay Obese	
	Mean	OR	Mean	OR	Mean	OR	Mean	OR
Number of Risk Factors								
0 (Reference Category)	<i>0.081</i>	1.00	<i>0.175</i>	1.00	<i>0.103</i>	1.00	<i>0.072</i>	1.00
1	<i>0.094</i>	1.21 (0.324)	<i>0.235</i>	1.47* (0.244)	<i>0.150</i>	1.58** (0.251)	<i>0.085</i>	1.31 (0.383)
2	<i>0.161</i>	2.19** (0.540)	<i>0.293</i>	1.97*** (0.361)	<i>0.152</i>	1.78* (0.392)	<i>0.141</i>	2.34** (0.629)
3	<i>0.168</i>	2.21** (0.503)	<i>0.316</i>	2.15** (0.491)	<i>0.160</i>	1.89* (0.561)	<i>0.156</i>	2.54*** (0.656)
4	<i>0.193</i>	2.68** (0.907)	<i>0.341</i>	2.45*** (0.582)	<i>0.168</i>	2.12 (0.576)	<i>0.173</i>	3.03** (1.058)
5	<i>0.174</i>	2.36 (1.463)	<i>0.473</i>	4.29** (1.931)	<i>0.298</i>	4.65** (2.411)	<i>0.174</i>	3.70* (2.373)

Standard errors are in parentheses
 * significant at .05 level
 ** significant at .01 level
 *** significant at .001 level

Data are weighted.
 Percentages significant at p < .001 level
 Reference category for Change in Obesity is "Stay Non-Obese"
 Results for the "Reduce Obese" category are not shown.

Table 2.8. Relationship between Cumulative Risk Index and Obesity Outcomes for Males (N = 3,122)

Individual and Family CRI	Obese at Will		Obese at Will		Change in Obesity From Will to Will			
					Become Obese		Stay Obese	
	Mean	OR	Mean	OR	Mean	OR	Mean	OR
Number of Risk Factors								
0 (Reference Group)	0.068	1.00	0.135	1.00	0.079	1.00	0.056	1.00
1	0.115	1.84** (0.358)	0.205	1.66*** (0.222)	0.114	1.58** (0.261)	0.091	1.85** (0.390)
2	0.171	3.07*** (0.577)	0.277	2.42*** (0.369)	0.149	2.26*** (0.413)	0.127	2.99*** (0.631)
3	0.178	3.23*** (0.986)	0.368	3.68*** (0.886)	0.214	3.60*** (1.014)	0.155	4.05*** (1.358)
4+	0.629	25.09*** (11.309)	0.639	11.50*** (5.294)	0.078	2.83 (2.024)	0.561	30.65*** (15.667)

Neighborhood CRI	Obese at Will		Obese at Will		Change in Obesity From Will to Will			
					Become Obese		Stay Obese	
	Mean	OR	Mean	OR	Mean	OR	Mean	OR
Number of Risk Factors								
0 (Reference Group)	0.099	1.00	0.184	1.00	0.106	1.00	0.078	1.00
1	0.128	1.34 (0.308)	0.226	1.34 (0.226)	0.125	1.30 (0.291)	0.101	1.41 (0.339)
2	0.212	2.48*** (0.541)	0.307	2.00*** (0.367)	0.126	1.45 (0.361)	0.181	2.83*** (0.703)
3	0.144	1.56 (0.389)	0.249	1.51 (0.335)	0.134	1.43 (0.409)	0.115	1.66 (0.469)

Standard errors are in parentheses

* significant at .05 level

** significant at .01 level

*** significant at .001 level

Data are weighted.

Percentages significant at $p < .001$ level

Reference category for Change in Obesity is "Stay Non-Obese "

Results for the "Reduce Obese" category are not shown.

Logistic and multinomial logistic regression models control for age and urbanicity.

Table 2.9. Estimated Multinomial Odds Ratios for Obesity Trajectories for Female Sample (N=3,371)
 (Reference Category is "Stay Non-Obese")

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Become Obese	Stay Obese	Become Obese	Stay Obese	Become Obese	Stay Obese	Become Obese	Stay Obese	Become Obese	Stay Obese
<i>Minority Status</i>										
Black	1.94 ** (0.369)	2.32 *** (0.478)	1.72 ** (0.330)	2.02 ** (0.422)	1.61 * (0.314)	1.86 ** (0.380)	1.36 (0.307)	1.44 (0.329)	1.37 (0.316)	1.43 (0.328)
Hispanic (White: Reference Category)	1.33 (0.262)	1.65 * (0.372)	1.22 (0.250)	1.49 (0.345)	1.17 (0.229)	1.42 (0.336)	1.08 (0.227)	1.26 (0.309)	1.19 (0.276)	1.29 (0.348)
Welfare/Poverty Status										
			1.58 ** (0.246)	1.65 ** (0.305)	1.23 (0.194)	1.13 (0.211)	1.14 (0.177)	1.00 (0.177)	1.12 (0.179)	1.00 (0.176)
Individual & Family CRI										
					1.31 *** (0.052)	1.48 *** (0.090)	1.30 *** (0.051)	1.48 *** (0.091)	1.31 *** (0.051)	1.47 *** (0.092)
Neighborhood CRI										
							1.14 * (0.077)	1.22 ** (0.090)	1.15 * (0.079)	1.22 ** (0.088)
Age at WI										
									0.99 (0.047)	1.04 (0.051)
Urbanicity										
									0.79 (0.120)	0.94 (0.189)
Pseudo Log Likelihood	-4240899.2		-4207089.4		-4103025.7		-4086323.1		-4080347.7	
Pseudo R Squared	0.0103		0.0182		0.0425		0.0464		0.0478	

Data are weighted.

Standard errors are in parentheses

* significant at .05 level

** significant at .01 level

*** significant at .001 level

Results for the "Reduce Obese" category are not shown.

Table 2.10. Estimated Multinomial Regression Odds Ratios for Obesity Trajectories for Male Sample (N=3,122)
 (Reference Category is "Stay Non-Obese ")

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Become Obese	Stay Obese	Become Obese	Stay Obese	Become Obese	Stay Obese	Become Obese	Stay Obese	Become Obese	Stay Obese
<i>Minority Status</i>										
Black	1.07 (0.245)	1.14 (0.234)	1.03 (0.236)	1.05 (0.228)	0.93 (0.211)	0.91 (0.206)	0.78 (0.209)	0.65 (0.181)	0.77 (0.203)	0.65 (0.183)
Hispanic (White: Reference Category)	0.90 (0.194)	0.87 (0.232)	0.88 (0.189)	0.83 (0.221)	0.84 (0.178)	0.78 (0.211)	0.79 (0.178)	0.69 (0.194)	0.82 (0.187)	0.72 (0.214)
Welfare/Poverty Status			1.15 (0.175)	1.40 (0.28456)	0.99 (0.154)	1.11 (0.221)	0.91 (0.152)	0.94 (0.199)	0.92 (0.155)	0.93 (0.194)
Individual & Family CRI					1.51 *** (0.112)	1.81 *** (0.169)	1.52 *** (0.112)	1.83 *** (0.176)	1.49 *** (0.113)	1.86 *** (0.175)
Neighborhood CRI							1.18 (0.117)	1.36 ** (0.148)	1.19 (0.120)	1.38 ** (0.153)
Age at WI									1.07 (0.048)	0.95 (0.062)
Urbanicity									0.86 (0.142)	0.91 (0.167)
Pseudo Log Likelihood	-4509854.5		-4502872.5		-4377465.8		-4353701.9		-4335486.4	
Pseudo R Squared	0.0004		0.0019		0.0297		0.0350		0.0390	

Data are weighted.

Standard errors are in parentheses

* significant at .05 level

** significant at .01 level

*** significant at .001 level

Results for the "Reduce Obese" category are not shown.

MULTIPLE LEVELS OF SOCIAL DISADVANTAGE AND ITS LINKS TO OBESITY RISK IN ADOLESCENCE AND YOUNG ADULTHOOD

The large proportion and recently rising number of poor and low-income children¹ living in the United States (US) has been a concern of many social science researchers and public policy officials for decades. Nearly 13 million children –17% of all children – live in poor² families and 23% of all children (15.9 million children) live in low-income³ families (Douglas-Hall and Chau 2007). Between 2000 and 2005, the number of children living in poverty increased by more than 11% (Fass and Cauthen 2006; NCCP 2007). Childhood poverty, especially deep and persistent poverty, is a concern because of its strong relationship to a number of adverse developmental, educational and health outcomes, such as obesity, in childhood, that continue to have negative effects on individuals when they reach adulthood (Blackwell, Hayward and Crimmins 2001; Brooks-Gunn and Duncan 1997; Dietz 1998; Elo and Preston 1992; Hayward and Gorman 2004; Serdula et al. 1993). Compounding the negative effects of family poverty, a large proportion of disadvantaged children must also navigate disadvantaged environments where their neighbors, the students in their schools, as well as their close friends or peers, may also be disadvantaged (Massey 1996; Massey and Denton 1993; Saporito and Sohoni 2007; Wilson 1987). In 1999, about 68% of families in high poverty census tracts (census tracts with a poverty rate of 40% or more) had an income of less than \$30,000, whereas only 14% of families had a yearly income of \$50,000 or more

¹ Children are defined as individuals under the age of 18.

² Poor families are defined as having income below 100% of the Federal Poverty Level (HHS 2007).

³ Low-income families are defined as having an income between 100% and 200% of the Federal Poverty Level (NCCP 2007).

(Bishaw 2005). Similar patterns can be found when investigating the relationship between race/ethnicity and school and neighborhood socioeconomic composition. African American and Hispanic children, especially poor African American and Hispanic children, are more likely than white children to live in neighborhoods and attend schools with higher levels of poverty (e.g., Iceland and Wilkes 2006; Saporito and Sohoni 2007). The fact that a significant portion of poor and minority children are embedded in multiple disadvantaged contexts has serious implications for their development, health and well-being (Jencks and Mayer 1990; Brooks-Gunn, Duncan and Aber 1997a; 1997b; Gephart 1997).

Researchers recognize that social disadvantage at these multiple levels of social context have important effects on the health of children (Braveman et al. 2005; Glass and McAtee 2006; Halfon and Hochstein 2002; Robert 1999; Taylor and Repetti 1997). The ecological model and life course paradigms emphasize the idea that individuals are influenced by the environments or ecological systems in which they are embedded, including the nuclear family, extended family, peer group, neighborhood, and community and institutions such as the school or the workplace (Bronfenbrenner 1979; Bronfenbrenner and Morris 1998; Elder 1997, 1998). Researchers in the social sciences as well as in public health, have attempted to model these multiple levels of context to better capture the multiple ecological systems in which individuals reside and how these contexts might affect their health (For reviews see Ben-Shlomo and Kuh 2002; Diez-Roux 2000; Earls and Carlson 2001; Pickett and Pearl 2001; Robert 1999; Sampson, Morenoff and Gannon-Rowley 2002).

A large amount of research demonstrates the importance of neighborhood, school, peer and family contexts in affecting health and health behaviors (Borsari and Carey 2001; Diez-Roux 2000; Goodman et al. 2003; Harding 2007; IOM 2001; Kawachi and Berkman

2003; Kirby and Kaneda 2005; La Greca, Prinstein and Fetter 2001; Pebley and Sastry 2004; Pickett and Pearl 2001; Prinstein, Boergers and Spirito 2001; Stafford and Marmot 2003). Given the recent rise in obesity in the US, particular attention has focused on this increasing public health problem especially among children and adolescents. There is growing research linking family, peer, neighborhood and school contexts to obesity and physical activity (Boardman et al. 2005; Christakis and Fowler 2007; Cummins and McIntyre 2005; Dietz and Gortmaker 2001; Frumkin 2006; Gordon-Larsen, McMurray and Popkin 2000; IOM 2005; Janssen et al. 2006; Lowry et al. 1996; Morenoff et al. 2006; Richmond et al. 2006; Robert and Reither 2004; Sallis, Prochaska and Taylor 2000; Sallis et al. 2006; Wickrama et al. 2006; Zakarian et al. 1994). In general, the research shows that the more disadvantaged a family, school or community, the more likely an individual in that context is to be obese. However, previous research has only been able to focus on one context or two contexts at a time, and, therefore, has not been able to determine independent contextual effects of each level of the social environment, when all levels of context are considered. In addition, most research utilizes cross-sectional data and focuses solely on children, adolescents or older adults at one point in time, ignoring how the relationship may change across life stages. Therefore, a better understanding of the relationship between socioeconomic disadvantage and obesity will entail capturing multiple levels of disadvantage, over time or across the early life course.

This chapter investigates the longitudinal relationship between multiple levels of social disadvantage in childhood and adolescence and obesity in adolescence and young adulthood using multilevel models (Raudenbush and Bryk 2002a; 2002b) and nationally representative data from the National Longitudinal Study of Adolescent Health (Add Health).

Social disadvantage operates at different levels of social context (e.g. family, peers, school and neighborhood) and interdependently. Multilevel models can capture these multiple levels of social disadvantage simultaneously. Multilevel models can also explicitly recognize the clustering of individuals and adjust for the correlations among individuals who share the same context to correct for their lack of independence. The goal of this analysis is to determine the level of socioeconomic disadvantage that has the strongest association with obesity outcomes and whether multiple levels of disadvantage in adolescence have enduring influences on obesity in young adulthood. This study of contextual disadvantage and its relationship to health is unique in that it will attempt to model multiple levels of social context and its relationship to obesity across the early life course, something previous research on environmental effects and health has not done.

Theoretical Framework

Conceptualization of “Context”

The local social and spatial context in which an adolescent develops can have an important influence on multiple aspects of current and future well-being. Although sociologists, demographers and other population researchers have studied the effects of family, school, peer and neighborhood factors on a variety of outcomes, there is still little research on comprehensive and theoretically driven conceptualizations of context (Entwisle 2007). Guided by Urie Bronfenbrenner’s ecological model of human development (1979), I conceptualize context as systems of environments in which adolescents live, learn, work, mature and interact with others and are subject to regulations and unwritten social norms. These multiple levels of social context include the family, school, peer and neighborhood

community, which are dynamic and interact with each other having direct and indirect implications for adolescent development. It is important to examine contextual influences because they impact available resources and opportunity structures, relationships and ties to others, and accepted norms of behavior (Jencks and Mayer 1990). For the purposes of this analysis, attributes of social contexts, especially structural attributes, have important direct impacts on health behaviors and outcomes.

Contexts of Structural Disadvantage and Links to Health

There has been a strong tradition in both medical sociology and social epidemiology to better understand and explicate the social patterning of disease. One robust finding has been the strong and persistent association between socioeconomic status (SES) and health outcomes (Adler et al. 1994; Braveman 2005; House et al. 1990, 1994). In this analysis, SES is measured using structural dimensions of social disadvantage. Structural disadvantage is a way to distinguish deprivation of groups from individual poverty (Galbraith 1969; Harrington 1997). It is a way to describe the social circumstance in which the poor live. Structural disadvantage is a macrosocial characteristic and is typically measured as aggregations of individual-level attributes of deprivations, such as poverty and low levels of education (e.g., Steffensmeier and Haynie 2000). Structural measures of disadvantage may also include racial ethnic segregation, unemployment rates, percentage of single-parent families and other measures of socioeconomic composition. I focus on structural measures of disadvantage at the neighborhood, school and peer group level measured by parent education, poverty and welfare usage (e.g., percent of school with students whose highest educated parent received a high school degree or less).

In sociology, structural perspectives, especially in the neighborhood research, have been used to explain numerous adverse outcomes in educational achievement, economic position, crime and delinquency and other outcomes for both children and adults (for a review see Sampson et al. 2002). William Julius Wilson, in his seminal book *The Truly Disadvantaged* (1987), highlighted the damaging effects of concentrated poverty and social isolation (in urban cities) on the poor residents that inhabited these areas. Following in this tradition, many others have also linked dimensions of structural disadvantage at the family, school and neighborhood level to multiple outcomes including health and health behaviors (for a review see Robert 1999; Taylor and Repetti 1997). The pervasive effects of these contexts of structural disadvantage, in addition to individual socioeconomic status (SES), on health have also garnered growing attention in the field of public health. A primary goal for public health research is to better understand how structural measures, in and of themselves, serve as risk factors for diseases and other health outcomes.

Writing from a public health perspective, and informed by research in sociology and social epidemiology, Link and Phelan (1995) created a theoretical framework to better explain the important link between structural measures of disadvantage, which they referred to as social conditions⁴, and health. Their two main arguments are that researchers must contextualize individually-based risk factors by investigating what puts individuals at risk of risks. If the goal is to design more effective interventions, then it is essential to identify the way in which people come to be subject to individually based risk factors such as lack of exercise, poor diet, cholesterol or high blood pressure. Following this argument, they asserted that some social conditions such as SES, race/ethnicity and gender are the “fundamental causes” of disease. They argue that these are fundamental causes because they

⁴ The authors define social conditions as factors that relate to an individual’s connections to other people.

determine access to crucial resources,⁵ which assist people in avoiding diseases and their negative consequences, influence multiple disease outcomes via multiple mechanisms and sustain an association with disease outcomes when there are changes in the intervening mechanisms. Because of the nature of fundamental causes, one cannot eliminate the effects of fundamental causes by addressing the mechanisms that link these fundamental causes to a disease in a specific situation. They argue that social conditions have not only a strong, but critical role in disease causation and should not be viewed as distal factors whose primary function is to serve as a starting point that can be explained by more proximal risk factors. The important role of social factors, including SES, is an idea shared by other theorists in sociology and public health that study neighborhood and community effects (Adler et al. 1994; Jencks and Mayer 1990; Robert 1999; Sampson et al. 2002; Wilson 1987).

By definition, a fundamental cause determines access to resources that can be utilized to avoid risks or curtail the effects of a disease once it has occurred, which is why we see such persistent associations between this type of cause and disease outcomes. Using this theoretical framework, we can conceptualize socially disadvantaged environments as shaping an individual's exposure to such risk factors as poor diet and a sedentary lifestyle that might lead to obesity. Poor neighborhoods, schools, and even peer groups create contexts that may increase risk behaviors for obesity.

The theoretical concept of fundamental causes can be expanded using sociological and social psychological theories that attempt to explain potential pathways of peer, neighborhood and school community influences through which these social conditions operate (Anderson 1990, 1999; Cohen et al. 2006; Jencks and Mayer 1990; Leventhal and

⁵ They define resources broadly to include knowledge, money, prestige, power and the kinds of interpersonal resources embedded in social connectedness.

Brooks-Gunn 2003; Massey and Denton 1993; Sampson, Raudenbusch and Earls 1997; Taylor & Repetti 1997; Wilson 1987). However, structural characteristics of the neighborhood, school and peer group continue to have direct effects on many individual health and social outcomes, which cannot be fully explained by a few well-known risk factors (Robert 1999; Sampson et al. 2002). As discussed earlier, the fundamental causes of disease framework argues that because the mechanisms that link disadvantage to health outcomes can change over time, it is more important to first understand the overall influence of the social contexts of disadvantage on health. Neighborhood, school and peer group resources (or lack thereof) influence health behaviors, by creating opportunities and/or barriers to healthy behaviors. In regards to obesity, neighborhood and school quality provide opportunities and access to physical activity and healthy food consumption and create positive or negative social norms that influence these behaviors. Also, peer groups that are created from the school or neighborhood environment can mirror and reinforce these social environments. Following Link and Phelan's (1995) theoretical framework of fundamental causes of disease, this chapter will focus on the importance of understanding the total effects of contextual levels of structural disadvantage, and how they are related to the health of individuals who exist within them.

There is also the possibility that selection bias may be driving contextual effects at the peer, school and neighborhood level. Parents with certain attributes (especially attributes that are not easily observed) may select their children into a particular school or neighborhood context (Brooks-Gunn et al. 1993; Duncan and Raudenbush 1999; Lee and Byrk 1989; Tienda 1991; Manski 1993). For example, parents who are really interested in promoting their child's physical activity and involvement in sports might pick a school where there are

many sports programs and opportunities for physical activity. Parents who are really interested in their family's physical exercise may choose to live in a neighborhood that is accessible to parks and walking paths and other venues for physical activity. This is problematic because instead of capturing true contextual effects of neighborhoods and schools, we may instead be capturing differential selection into schools and neighborhoods or unmeasured factors that affect the choice of both the neighborhood (and/or the school) and one's health. Selection bias can occur at the peer level, when individuals select friends who are similar to them (Evans, Oates and Schwab 1992). In an attempt to minimize selection bias, I control for family background characteristics associated with intentional selection into schools and neighborhoods, and also associated with health. These include parent obesity, parent education level, race/ethnicity and family structure.

Conceptual Model

The conceptual model for these analyses draws from the life course and ecological frameworks. The fundamental assumption of this model is that an individual's obesity risk is dynamic (changeable over time) and affected by multiple aspects of his/her social environment. In this model (See Figure 3.1), an individual's family, peers, school and neighborhood represent overlapping environments that influence individual obesity, both directly and in interaction with each other, over time, from adolescence and into young adulthood. Although these influences overlap in this conceptual model, they represent ever larger aspects of an individual's social world which all take place within the political, economic and cultural constraints of the nation. This model is derived from the Committee on Evaluation of Children's Health (NRC 2004) conceptualization of the dynamic process of

multiple, interacting influences on the evolution of children's health.

The Add Health data provide a rich set of measures for family, peer, school and neighborhood characteristics. The goal of this analysis is to determine which level of socioeconomic disadvantage has the strongest association with obesity outcomes and whether these multiple levels of disadvantage in adolescence have enduring influences on obesity in young adulthood. Therefore, this empirical work will estimate a reduced-form version of the model presented here. The interest is on the total effects of each level of disadvantage on obesity outcomes, which has not been accomplished in previous research. Future work will explore the mechanisms (e.g., institutional resources, relationships and ties, and norms and collective efficacy) through which disadvantage at the family, school, peer and neighborhood level influences obesity once these basic relationships have been established within a longitudinal multilevel framework.

Specific Aims of Analysis

Theory on social context emphasizes the fact that adolescents develop within a complex system of environments that interact and overlap. Multilevel models are used to account for the nesting of individuals within schools (the highest level of social context in this analysis), which subsumes the nesting of peers and neighborhoods within schools.

These analyses will:

- (1) Create measures of socioeconomic disadvantage at the individual, family, peer, school and neighborhood levels.
- (2) Determine how contextual disadvantage varies by race/ethnicity, sex and poverty status.
- (3) Investigate the bivariate relationships between each level of contextual disadvantage and

risk of obesity.

(4) Within a longitudinal framework, investigate the unique influence of each level of disadvantage on the risk of obesity in adolescence and adulthood using multivariate multilevel modeling.

(5) Investigate cross-level interaction between different levels of social context to explore whether social disadvantage at one level varies by social disadvantage at another level.

Data

Data come from the National Longitudinal Study of Adolescent Health (Add Health), which is an ongoing nationally representative, school-based study of adolescents in grades 7 to 12 that began in 1994. It was designed to explore the causes of health-related behaviors, with an emphasis on the influence of social context. In 1994 Add Health administered an In-School Questionnaire to every student attending school from a nationally representative sample of schools. A sample of 80 high schools and 52 middle schools from the U.S. was selected using a stratified cluster design. A subsample of individuals in these schools participated in the In-Home Interview in 1995 (Wave I), given an average of eight months after the In-School Survey, and again in 1996 (Wave II). In Wave III (2001-02) Wave I respondents were re-interviewed.

A parent, generally the mother, was also interviewed in Wave I (Parent In-Home Questionnaire). In-home adolescent questionnaires were administered by computer-assisted personal interview (CAPI), as well as computer-assisted self-interview (CASI) for more sensitive questions. Ultimately, 20,745 in-home interviews were completed in Wave I; 17,713 parents answered child specific questions and 17,669 answered parent specific

questions (more than one child was interviewed in some households). 14,738 in-home interviews were completed in Wave II (the seniors in Wave I were not followed-up). In Wave III 15,197 eligible original Wave I respondents completed the survey. In Wave I, the age of participants ranged from 12 to 19 years, in Wave II from 13 to 20 years and Wave III from 18 to 26 years.

Over 70% of the schools originally selected for the survey participated. Of the adolescents sub-sampled for the in-home questionnaires, 78.9% participated in Wave I. Parent interviews are available for 85% of these respondents. Of those eligible for participation in Wave II, 88.2% completed in-home interviews. Of those eligible for participation in Wave III, 77.4% completed in-home interviews.

In the In-School Questionnaire students were asked to nominate up to 5 male and 5 female friends and to locate and record their student ids from the school roster. Because the in-school sample was a saturated sample, with nearly all children in the school interviewed, the identification numbers of nominated friends can be linked back to their own in-school questionnaire and characteristics of a respondent's peer group can be determined, such as its racial/ethnic makeup. In a similar fashion, school-level measures can be derived by aggregating the responses of the In-School Questionnaire for all students in their respective schools. In addition, contextual data containing information on the characteristics of the neighborhoods and communities in which Add Health sample members lived in Waves I and II have been linked to individual-level records.

The fact that the data set is longitudinal and nationally representative, with extensive measures of socioeconomic status, health, race and ethnicity among other factors and the ability to create measures at multiple levels of social context, makes it an ideal data set to

investigate the relationship between poverty and obesity in adolescence. Harris et al. (2003) provides a more detailed description on the Add Health Study.

This study uses data from the Wave I In -Home and Parent Questionnaires as well as the follow-up Wave II and III surveys. This analysis is therefore limited to adolescents who participated in all three waves of the study, have completed Parent Questionnaires, school information, and have complete measured height and weight data. Exclusions included seriously disabled respondents and pregnant females. After applying these data constraints and deleting the few cases with missing data on covariates, the final study sample contains 7,548 (3,926 females and 3,622 males).

My data requirements for participation in multiple survey components are demanding, but necessary given my research question focuses on multiple levels of social context. Although my sample is somewhat constrained, it is still sufficient given the large oversamples of blacks from high SES families, Chinese, Cubans, and Puerto Ricans in Add Health. In addition, I use sample weights to ensure that my subsample is nationally representative of the school population of adolescents in grades 7 through 12 in 1995.

Measures

Table 3.1 provides means and standard errors of all measures used in this analysis for the sample by sex.

Individual Level Measures and Controls

Race/Ethnicity: Add Health allows for rich detail in measures of race and ethnicity.

Race/ethnicity is self-reported at Wave I and is classified into five race and ethnic groups:

non-Hispanic white (reference group), non-Hispanic black, Hispanic, Asian, or other racial/ethnic group. I control for race/ethnicity because of its possible confounding effects with socioeconomic status.

Sex: This measure is constructed from responses in the Wave I In-Home Questionnaire. This measure was crosschecked with WII and WIII responses. Because research suggests that disadvantage might operate differently in affecting obesity risk for males and females (Lee et al. 2008), analyses will also be run in samples stratified by sex.

Age: Age is a continuous measure of self-reported age at WI. Age ranges from 11 to 21 years of age.

Parent Obese: Using reports from the Parent In-Home Questionnaire, respondents were coded as having an obese parent if either their biological mother and/or biological father were reported as being obese. Parental obesity is used as a control to account for parental role modeling of lifestyle and eating behavior, as well as genetic predisposition for obesity, or some combination of genetic propensity and environmental risk (Agras et al. 2004; Whitaker et al. 1997). About 22% of the sample has an obese parent.

Socioeconomic Disadvantage

The measures of socioeconomic disadvantage used in this analysis are measured at the family, peer, school and neighborhood level. Family level socioeconomic disadvantage is measured in two ways using welfare/poverty status and parent education. Family structure is used as a control. Peer and school disadvantage are measured using parent education. Neighborhood level disadvantage is measured by the education level of adults and percentage female headed household within the census block group in which a respondent lives. I will

also control for racial heterogeneity at the neighborhood level. Because school and neighborhood contexts overlap considerably and are highly correlated, I explore whether there are distinct neighborhood social disadvantage effects in supplementary analysis that include both school and neighborhood or exchange these two measures.

Parent education is used as a measure of socioeconomic disadvantage at each level of social context for multiple reasons. There is a well-established and robust positive relationship between education and a variety of health outcomes (Antonovsky 1967; Crimmins and Saito 2001; Kitagwa and Hauser 1973; Ross and Wu 1995; Vargas, Ingram and Gillum 2000). Education is a socioeconomic indicator that is particularly likely to capture aspects of behavior and lifestyle (Shavers 2007). Parental education, related to poverty and family structure, may also affect parental monitoring, with more educated parents doing a better job at monitoring their children (Lareau 2003), especially behaviors that directly relate to obesity such as physical activity and eating patterns. Further, the education of parents directly affects food and diet quality (Crawford et al. 1995; Deshmukh-Taskar et al. 2007; Patrick and Nicklas 2005; Sausenthaler et al. 2007; Stenhammar, Sarkadi and Edlund 2007). People with higher education levels have a greater awareness of health issues and are better able to make healthy food choices for their children. This is especially relevant during adolescence when lifestyle and health-related behaviors are established. Finally, my choice of parents' education across contexts reflects the empirical problem of collinearity that I found with other aggregate measures such as family structure, unemployment, poverty, etc.

Family Level Disadvantage and Controls

Welfare/Poverty Status: Welfare/Poverty status is a dichotomous indicator of any welfare receipt before the age of eighteen or family income less than poverty level. This measure is constructed from data on the family's receipt of public assistance or welfare from WI and WII during adolescence in combination with a retrospective report at WIII on the receipt of welfare and public assistance prior to the age of eighteen. Using data from the WI Parent Questionnaire on reported annual income from 1994, family income is categorized as below poverty level if income was less than \$16,000 (roughly the poverty level for a family of four in 1994). I chose a welfare- and income-based measure of poverty over an only income-based measure due to the large proportion of missing data on income ($\approx 20\%$). About 28% percent of the total sample received welfare prior to the age of eighteen and/or was living below poverty at WI.

Parental Education High School Diploma (or Equivalent) or Less: Using data from the Wave I Parent Questionnaire, parents' education is measured as the higher of either mother's or father's education. A respondent is considered disadvantaged if his/her highest educated parent has high school degree or equivalent (i.e., GED) or less. About 63% of the sample has a parent with a high school degree or less.

Family Structure: There is rich detail on family of origin living arrangements. Adolescents are classified as those who live with two biological or adoptive parents (reference category), a stepfamily (biological mother and step father or a biological father and step mother), single mother, single father, and surrogate or foster parents (including grandparents, aunts and uncles, other adult relatives, or non-relative adults). I control for family structure because of its possible confounding effects with socioeconomic status.

Peer Level Disadvantage and Controls

Although less studied in relationship to obesity, peer context is highly related to health and health behaviors, especially in adolescence and the transition to young adulthood (Borsari and Carey 2001; Bronte-Tinkew 2005; Christakis and Fowler 2007; Giordano 2003; La Greca et al. 2001; Perry 2000; Prinstein et al. 2001; Sallis et al. 2000; Zakarian et al. 1994). During adolescence peer influences increase as peer groups become more autonomous and less neighborhood-based (Brown 1990; Giordano 2003) and continue to strengthen during the lifecycle transition to adulthood (Bronte-Tinkew 2005). Peers function as credible sources of information, role models of new social behaviors, sources of reinforcement, and bridges to alternative lifestyles (Brown 1990).

Community influences can originate in neighborhoods, schools, or other organizations and can operate through adolescents' peer groups (Jencks and Mayer 1990). Including peer group measures in analysis using school level measures helps to disentangle the larger context of school composition effects from smaller group context of peer relations in a school, an issue previous research in this area has not been able to address due to data limitations. As individuals age, these multiple contexts broaden and deepen and serve to channel and reinforce influences in health trajectories (Halfon and Hochstein 2002; NRC 2004).

Peer Parental Education High School Diploma (or Equivalent) or Less: Continuous measures of percentage of students in a respondent's peer group (as nominated by the respondent) who come from a family where the highest educated parent received a high school degree (or equivalent) or less. An average of 51% of a respondent's peer group has a highest educated parent with a high school degree or less.

No Peer Information: This measure serves as a control measure for respondents who had no friendship information because they reported no friends or reported only non-school friends.

School Level Disadvantage

School Parental Education High School Diploma (or Equivalent) or Less: Continuous measures of percentage of students in a respondent's school who come from a family where the highest educated parent has a high school degree (or equivalent) or less. An average of 51% of a respondent's school peers has a highest educated parent with a high school degree or less.

Neighborhood Level Disadvantage and Controls

The census block group (BG) is used as the geographical unit to define neighborhood in this analysis. Census blocks are the smallest geographic area for which the Census Bureau collects decennial census data. In the geographic hierarchy, BGs are the next level above census blocks. A BG is a subdivision of a census tract or block numbering area (BNA) and is composed of a number of census blocks (U.S. Census Bureau 1994). A BG represents the most localized available contextual characteristics of the areas in which individuals reside, and because I argue that neighborhood influence on obesity operates mainly at the local level, I chose BG over a census tract. In 1990, a BG averaged 452 housing units, or 1,100 people (U.S. Census Bureau 1994). It should be noted that in Add Health, neighborhood block groups are a smaller level of aggregation than schools. Multiple block groups are usually represented in one school.

Neighborhood Adult Education Less than High School: Neighborhood education is a continuous WI BG level measure of the proportion aged 25 years and over with no high school diploma or equivalency.⁶ An average of 27% of the adults individuals living in a respondent's BG do not have a high school diploma or equivalent.

Proportion of Female Headed Households in Neighborhood: Neighborhood family structure is a continuous WI BG level measure of the percentage of households in the BG that are female-headed with own children under the age of 18. An average of 7% of the households in a respondent's BG are female-headed.

Neighborhood Racial Heterogeneity: Neighborhood racial dispersion is a continuous WI BG level of dispersion in racial composition (using the racial categories of white/black/other), based on census definition. I account for neighborhood racial dispersion due to links between neighborhood racial composition and obesity in previous research (Boardman et al. 2005; Wickrama et al. 2006).

Urban Block Group⁷: Categorical measure to designate if respondent lives in an urbanized area from Wave I context data. About half of the sample lives in an urbanized BG.

Outcome Variable

Obesity at WII and WIII: I use body mass index or BMI to measure obesity. BMI is a tool for indicating weight status in adults, computed by dividing an individual's body weight in kilograms by the square of his or her height in meters (i.e., $\text{weight}/(\text{height})^2$). BMI is more highly correlated with body fat than any other indicator of height and weight (NRC 1989).

⁶ There are no census BG measures of adult education of a high school degree or less available.

⁷ The urbanicity code distinguishes block groups that are in completely urbanized areas from those that have any individuals living outside urbanized areas, in rural farm or rural nonfarm locations. This measure is different from the census "urban" designation which also includes places outside urbanized areas of 2,500 or more persons.

For adults over 20 years old, BMI falls into one of four categories: underweight, normal, overweight and obese. Adults with a BMI of 25 to 29.9 are categorized as overweight. Adults with a BMI of 30 or more are considered obese (NHLBI 1998, WHO 2000).

BMI is defined differently for children and adults. Age- and sex-specific BMI percentiles are used as growth references during childhood and adolescence because BMI changes at different rates by age and sex during normal developmental growth. In the United States, the 85th and 95th percentiles, based on nationally representative data from the 2000 growth curves of the Centers for Disease Control and Prevention (CDC), have been recommended for use in classifying persons as being overweight or at risk of overweight (CDC 2004). However, definitions of overweight based on these percentiles are not directly comparable to the adult definitions of obesity using specified cut points (NHLBI 1998, WHO 2000).

The ability to generate comparable prevalence measures between adult and adolescent measures of obesity or to calculate obesity incidence over the transition period of adolescence to young adulthood is limited by discrepancies between adolescent and adult definitions. The International Obesity Task Force (IOTF) developed BMI curves, which link childhood and adolescent BMI centiles to adult cut off points of BMI of 25 and 30 kg/m², and thus, allow greater consistency in the youth versus adult definitions. The BMI curves provide good comparative reference data during this transitional period (Cole et al. 2000). Because I investigate the incidence of obesity in adolescence and young adulthood, I use the IOTF measures to determine obesity status among adolescents and young adults in this analysis.

Using the IOTF cutoffs, I define obesity at Waves II and III using BMI calculated

from measured height and weight. I identify individuals as obese if their BMI falls above the age- and sex-specific, IOTF 30 kg/m² cutpoint in adolescence at Wave II. For the young adults at Wave III, we consistently use the adult BMI cut point of 30 kg/m². At Wave 11% of the sample was obese and at Wave 23% of the sample was obese, indicating a doubling in the prevalence of obesity in this sample in only 2 years.

Analytical Design

The analytical design is derived from the conceptual model (See Figure 3.1). Longitudinal data from three waves of Add Health are used to measure obesity in adolescence and young adulthood and to measure factors operating during adolescence that serve to influence these outcomes. Outcomes of interest include obesity at WII (adolescence) and WIII (young adulthood). Factors influencing obesity are measures of individual, family, peer, school and neighborhood context at WI, in addition to ascribed individual social characteristics such as race/ethnicity and sex, also measured at WI. This design exploits the longitudinal data and incorporates the temporal order of effects (i.e., factors operating during adolescence prior to the transition to adulthood), which is a necessary first step in establishing a causal relationship. A longitudinal design also allows for the measurement of factors operating in adolescence and the influences these have on obesity outcomes in later adolescence and young adulthood. Although a longitudinal design also allows for measurement of change in obesity status from adolescence and into young adulthood, it is important to first investigate how multiple levels of disadvantage at WI operate to affect obesity risk in adolescence (WII) and young adulthood (WIII), separately. Future work will explore change models once these developmental models are established.

STATA GLLAMM commands are utilized for multilevel logistic regression analysis, and include weights for both individuals and schools (Chantala, Suchindran and Blanchette 2005; Rabe-Hesketh and Skrondal 2006; Rabe-Hesketh, Skrondal and Pickles 2004).

Analytical Methods

Analysis follows the five research aims listed at the outset of the paper. I first examine the bivariate associations between measures of disadvantage and obesity at WII and WIII. I then calculate the proportion of disadvantaged individuals at each level of social context by individual poverty, race/ethnicity and sex, to explore variation in contextual disadvantage. For example, the proportion of a respondent's peers with parent education less than a high school diploma will be compared across racial/ethnic groups. Chi-square analyses will be performed to test for differences between subgroups.

Multilevel modeling (Raudenbush and Bryk 2002a; 2002b) is then used to investigate the unique influence of each level of disadvantage on obesity risk in adolescence and young adulthood. Multilevel models explicitly recognize the clustering of individuals and adjust for the correlation among individuals who share the same context to correct for their lack of independence. This analysis employs a two-level model, individuals serve as level 1 and schools serve as level 2. Peer context is associated with individuals (level 1) given that peer groups are nominated from respondents' nominations. Neighborhoods (BGs) are nested within schools (level 2), because the school population of the school pair including a high school and the feeder school (middle or junior high) to that high school draws from multiple neighborhoods within the school boundary, and this is especially the case for private and magnet schools. In addition, adjusting the standard errors for the nonindependence of

adolescents who share the school context (the larger spatial context) automatically adjusts for the nonindependence among adolescents in the same neighborhoods.

STATA GLLAMM commands are used for multilevel logistic regression analysis. To illustrate the analytic techniques used in this analysis, assume a model with only two levels of observation: individual and school. The multilevel model for binary outcomes is conceptually similar to traditional logistic regression with the addition of a school-level error component (u_j). Error across schools is measured by this school level (level 2) residual term, which is assumed to be normally distributed with an expected value of 0 and an unknown variance of σ_u^2 (Little et al. 1996; McCulloch and Searle 2001). The following equation represents a multilevel equation for the probability of being obese, allowing obesity to vary across schools and including an individual level explanatory variable (x_{ij}).

$$\log\left[\frac{P_{ij}}{1-P_{ij}}\right] = \beta_0 + \beta_1 x_{ij} + u_j \quad (\text{Model 1: Combined Model})$$

Model 1 is known as the combined model and can alternatively be represented by 2 models.

$$\log\left[\frac{P_{ij}}{1-P_{ij}}\right] = \beta_{0j} + \beta_1 x_{ij} \quad (\text{Model 2: Level 1}) \quad \beta_{0j} = \beta_0 + u_j \quad (\text{Model 3: Level 2})$$

Model 1 captures the probability (P_{ij}) that the i th individual in the j th school is obese. These models can then be reduced to an additive model in the form of a single equation represented by Model 1. The extent to which residual variation in the log-odds of obesity is situated within or between schools can be estimated by the variance of the level 2 residual (σ_u^2). The

ratio of the level 2 residual variance to the overall residual variance ($\sigma_u^2 + \sigma_e^2$) measures the intraclass correlation (ICC). The variance of the standard logistic regression ($\pi^2/3$) has been suggested to estimate level 1 residual variance when modeling binary outcomes (Guo and Zhao 2000; Snijders and Bosker 1999). Model 4 represents a multilevel equation including individual (x_{ij}) and school level (w_j) explanatory variables. $\log\left[\frac{P_{ij}}{1-P_{ij}}\right] = \beta_0 + \beta_1 x_{ij} + \beta_2 w_j + u_j$

(Model 4)

For these analyses, models also include measures at the peer and neighborhood level. Although not the theoretical focus of this analysis, I test cross-level interactions between individual, peer, and school level disadvantage for exploratory purposes. The purpose is to examine whether social disadvantage at one level varies by social disadvantage at another level, implying multiplicative effects.

Modeling Plan

As discussed earlier, variables are grouped into individual and family-level variables, peer group-level, school-level and neighborhood-level variables. The most basic model (Model 1) for analysis in this study only includes an intercept term (the null model), which is used to calculate the unadjusted ICC. Model 2 includes the individual and family-level variables for race/ethnicity, age, parental obesity status, family structure and socioeconomic disadvantage. Here, controlling for individual characteristics accounts for the aggregation effects of individual characteristics that are confounded with school-level measures (Manski 1993). School level socioeconomic disadvantage is added in Model 3 as a second-level of the multilevel model. Neighborhood level variables are included in Model 4 mainly as controls. Peer group-level measures are added in Model 5. These series of models are run separately

for obesity at WII and WIII. ⁸ After the inclusion of family and individual level variables, school level variables are added first because they represent the highest level of aggregation followed by neighborhood and peer group. I also examine cross-level interactions between individual, peer, and school level disadvantage individually (i.e., individual x school; individual x neighborhood; individual x peer; peer x school; peer x neighborhood and school x neighborhood).

Results

Table 3.2 shows the percentage of respondents obese at WII and WIII (by sex) by each of the measures of social disadvantage that are used in these analyses. Measures of continuous contextual disadvantage at the peer, neighborhood and school level were dichotomized, so that ‘1’ represented the highest levels of risk. About 20% to 30% of the sample fell into this risk group. There was a stronger association between family level disadvantage (poverty status/welfare receipt) for females compared to males, as has been evidenced in previous research (Lee et al. 2008). 13% of females living below poverty or receiving welfare at WI were obese at WII compared to 9% of females who were not in poverty or receiving welfare at WI. The difference in means is not significant for males for obesity at WII. At WIII, 30% of females living below poverty or receiving welfare at WI were obese compared to 20% of females who were not in poverty or receiving welfare at WI. Among males, 25% of males living below poverty or receiving welfare at WI were obese at WIII compared to 21% of males who were not in poverty or receiving welfare at WI. In regards to education, 11% of females with a highest educated parent who had a high school

⁸ To assess proportionate change in level 2 variance across models, σ_u^2 estimates were rescaled because the level 1 variance was approximated with the fixed value ($\Pi^2/3$) (Bauer 2009).

diploma (or equivalent) or less were obese at WII compared to only 7% of females with a highest educated parent who had more than a high school diploma or equivalent. Among males, 14% with a highest educated parent who had a high school diploma (or equivalent) or less were obese at WII compared to 9% with a highest educated parent who had more than a high school diploma or equivalent. At WIII, 27% of females and 25% of males with a low educated parent were obese compared to 17% of females and 18% of males with a non-low educated parent. Significant differences by family structure were only found for females. In summary, we see that disadvantage at the family level was associated with higher levels of obesity at WII and WIII, with some variation by sex.

Differences in mean prevalence of obesity by contextual disadvantage at the peer, school and neighborhood level, were also large and significant for both males and females. For example, 28% of females and 26% of males who attended schools where 60% or more of the student population had a highest educated parent who completed a high school degree or less were obese at WIII compared to 21% of females and 20% of males who attended schools where less than 60% of the student population had parents that were low educated. The significant relationships evidenced between disadvantage and obesity at each level of social context highlight the importance of investigating multiple levels of disadvantage when investigating the relationship between poverty and obesity.

Table 3.3 provides the mean percentages of family, peer, school and neighborhood disadvantage (measured using education) by race and sex. Among both males and females, Hispanics (Male: 73%; Female: 82%) and African Americans (Male: 67%; Female: 72%) had the highest levels of low parent education compared to whites (Male: 59%; Female: 60%) and Asians (Male: 40%; Female: 54%). These differences are statistically significant.

There was a similar pattern for neighborhood education. Hispanics and African Americans lived in neighborhoods where there was a higher percentage of adults over 25 with less than a high school degree compared to whites and Asians. Levels of peer and school parental education were similar across racial groups (except for Asians) at about 50%.

Table 3.4 provides the mean percentages of peer, school and neighborhood disadvantage (measured using education) by family disadvantage and sex. For both males and females, respondents who lived in families where the highest educated parent had a high school degree or less and respondents who received welfare or live below poverty before the age of 18, also had friends, attended schools and lived in neighborhoods that had lower levels of parental/adult education compared to respondents whose highest educated parent received more than a high school degree and who did not receive welfare and lived above poverty in childhood. In summary, the poor faced higher levels of contextual poverty at all levels than the non poor.

Female Multivariate Multilevel Results

Results from the multilevel logistic regression models for females are presented in Table 3.5. Model 1 for the Obese WII outcome, an intercept-only random effects model, indicated a small amount of variation across schools in the prevalence of obesity at WII ($\sigma^2_u = 0.23$, SE=0.09; ICC=0.07). The ICC indicated that about 7% of the variability in the likelihood of being obese at WII was located between schools. Among the race/ethnicity variables, only African American was significantly related to obesity at WII. African Americans were 65% more likely to be obese at WII than whites.⁹ Similar to findings from

⁹ I estimated an alternative model using more detailed ethnicity variables (Mexican, Cuban, Central/South American, Puerto Rican, Chinese, Filipino, other Asian, African and European) and found similar results. The only measure significantly related to obesity at WII was African.

previous research, females who had an obese parent or a parent who completed a high school degree or less were also more likely to be obese at WII. Females with an obese parent were 311% more likely to be obese at WII than females with no obese parent. Females whose highest educated parent received a high school degree or less were 37% more likely to be obese at WII compared to females with higher educated parents. Neither family structure nor family poverty status were significantly related to females' obesity at WII. Model 3 included a measure of the percentage of the students in a school who have parent with a high school degree or less. This measure was positively related to obesity at WII. A 10% increase in the number of students in a school with a low educated parent increases the likelihood of obesity at WII by 28%. Family level parent education became insignificant. Black and parental obesity remained significantly related to obesity at WII. Model 4 included neighborhood level measures. No measures of neighborhood were significantly related to females' obesity. African American, parental obesity and school level education remained significantly related to obesity at WII. Model 5 included the percentage of persons in a female's peer group whose parent had a high school degree or less. This measure was not significantly related to obesity at WII. African American, parental obesity and school level education remained significantly related to obesity at WII. Individual, family and school-level factors explained most of the between school variation (98%).

Model 1 for the Obese WIII outcome, an intercept-only random effects model, indicated a small amount of variation across schools in the prevalence of obesity at WIII ($\sigma_u^2 = 0.21$, SE=0.06; ICC=0.07). The ICC indicates that about 6% of the variability in the likelihood of being obese at WIII is located between schools. Among the race/ethnicity variables, African American, Hispanic and Asian were significantly related to obesity at

WIII. African Americans were 56% more likely to be obese at WIII than whites. Hispanics were 21% more likely to be obese at WIII than whites. Asians were 46% less likely to be obese at WIII than whites. Females who had an obese parent or a parent with a high school education or less were also more likely to be obese at WIII. Females with an obese parent were 217% more likely to be obese at WIII than females with no obese parent. Both measures of family level disadvantage were significantly related to obesity. Females whose highest educated parent received a high school degree or less were 28% more likely to be obese at WIII compared to females with higher educated parents. Females who received welfare or lived below poverty level in childhood were 40% more likely to be obese at WIII than females who had not received welfare or lived below poverty in childhood. Family structure was not significantly related to females' obesity at WIII. Model 3 included a measure of the percentage of the students in a school who have parent with a high school degree or less. This measure was positively related to female obesity at WIII. A 10% increase in the number of students in a school with a low educated parent increased the likelihood of obesity at WIII by 16%. Measures of race, parental obesity and family disadvantage remained significantly related to females' obesity at WIII. However, the coefficients for both poverty/welfare before childhood and parent education were slightly reduced. Model 4 included neighborhood level measures. No measures of neighborhood were significantly related to obesity at WIII. Hispanic was no longer significant in Model 4. However, African American, Asian, parental obesity, family disadvantage and school level education remained significantly related to obesity at WIII. Coefficients for school and family level disadvantage were reduced. Model 5 included the percentage of persons in a female's peer group whose parent had a high school degree or less. This measure was

significantly related to obesity at WIII. A 10% increase in the percentage of a peer group with a low educated parent increases the likelihood of obesity at WIII by 40%. African American, Asian, parental obesity, family disadvantage and school level education remain significantly related to obesity at WIII. Coefficients for school and family level disadvantage were slightly reduced. Individual, family and school-level factors explained most of the between school variation (94%).

In summary, among females, once accounting for all levels of disadvantage, school level disadvantage was most salient in influencing obesity in adolescence (WII). In addition, being African American or having an obese parent increased the likelihood of female obesity at WII. Disadvantage at multiple levels of context had enduring influences on the likelihood of obesity in adulthood. Family, peer and school disadvantage were associated with an increased likelihood of obesity at WIII. In addition, being African American or having an obese parent increased the likelihood of obesity at WIII and being Asian decreased the likelihood of obesity at WIII.

Male Multivariate Multilevel Results

Table 3.6 presents results from the multilevel logistic regression results for males. Model 1 for the Obese WII outcome, an intercept-only random effects model, indicated a small amount of variation across schools in the prevalence of obesity at WII ($\sigma^2_u = 0.15$, SE=0.06; ICC=0.04). The ICC indicated that about 7% of the variability in the likelihood of being obese at WII was located between schools. Race/ethnicity, family structure, welfare/poverty status and parent educations were not significantly related to obesity at WII. Parental obesity was significantly related to obesity at WII. Males with an obese parent were 232% more likely to be obese at WII than males with no obese parent. Model 3 included a

measure of the percentage of the students in a school who have a parent with a high school degree or less. This measure was positively related to obesity at WII. A 10% increase in the number of students in a school with a low educated parent increased the likelihood of obesity at WII by 23%. Parental obesity remained significantly related to obesity at WII. Model 4 included neighborhood level measures. Racial dispersion was significantly related to obesity at WII. An increase in racial heterogeneity increased the likelihood of obesity at WII. Parental obesity and school level education remained significantly related to obesity at WII. Model 5 included the percentage of persons in a male's peer group whose parent had a high school degree or less. This measure was not significantly related to obesity at WII. African American, parental obesity and school level education remained significantly related to obesity at WII.

Model 1 for the Obese WIII outcome, an intercept-only random effects model, indicated a small amount of variation across schools in the prevalence of obesity at WIII ($\sigma^2_u = 0.11$, SE=0.04; ICC=0.03). The ICC indicated that about 3% of the variability in the likelihood of being obese at WIII was located between schools. Race/ethnicity and family structure were not significantly related to obesity at WIII. Age and parent obesity were positively related to obesity. Males with an obese parent were 173% more likely to be obese at WIII than males with no obese parent. Among the family level disadvantage measures, only parent education was significantly related to obesity at WIII. Males whose highest educated parent received a high school degree or less were 23% more likely to be obese at WIII compared to males with higher educated parents. Model 3 included a measure of the percentage of the students in a school who had parent with a high school degree or less. This measure was positively related to obesity at WIII. A 10% increase in the number of students

in a school with a low educated parent increased the likelihood of obesity at WIII by 11%. Measures of parental obesity and age remained significantly related to obesity at WIII. Model 4 included neighborhood level measures. No measures of neighborhood were significantly related to obesity at WIII. School level education, parental obesity and age remained significantly related to obesity at WIII. Model 5 included the percentage of persons in a male's peer group whose parent had a high school degree or less. This measure was not significantly related to obesity at WIII. Parental obesity, age and school level education remained significantly related to obesity at WIII.¹⁰

In summary, among males, once accounting for all levels of disadvantage school level disadvantage was most salient in influencing obesity in adolescence (WII) and young adulthood (WIII). In addition, having an obese parent and neighborhood racial heterogeneity increased the likelihood of obesity at WII. Having an obese parent and age increased the likelihood of obesity at WIII. It should also be noted that the variables explained a lower proportion of between school variation in the male models compared to the female models.

Interactions were also tested for both obesity at WII and WIII and for males and females. The interactions included parent education and peer parent education, parent education and school parent education, parent education and neighborhood adult education, peer education and school education, school education and neighborhood education. These interactions were added separately to the final models. No interactions were significant. Results are not shown.

Discussion and Conclusion

¹⁰ Models were also run excluding neighborhood measure and including only school measures. Results did not differ.

In this paper I assessed the relationship between multiple levels (family, peer, school and neighborhood) of social disadvantage in childhood and adolescence and obesity status in adolescence and young adulthood using multilevel logistic regression models and national data from Add Health. The goal of this analysis was to determine what levels of social context were most salient in influencing obesity risk in adolescence and whether these contexts of disadvantage in adolescence continued to influence obesity outcomes in young adulthood.

Bivariate descriptive statistics indicated that social disadvantage at the family, peer, school and neighborhood level were significantly related to obesity at both WI and WIII for males and females. In addition, disadvantaged respondents attended schools and had peers with a high percentage of low educated parents and lived in neighborhoods with a high proportion of low educated adults. Hispanics and African Americans respondents had parents with the lowest levels of education and lived in neighborhoods where there was a higher percentage of adults with low education compared to whites and Asians. These findings support previous research indicating that poor and minority groups are more likely to live in disadvantaged social contexts. It also shows that disadvantage at multiple levels has influences on obesity in both adolescence and young adulthood.

Multilevel models indicated that when all levels of social disadvantage were included, school level parent education was most salient in influencing obesity in adolescence (WII) and young adulthood (WIII) for both males and females. For females, school level parent education was the only measure significantly related to obesity at WII. In addition, being African American or having an obese parent increased the likelihood of obesity at WII for females. At WIII, family, peer and school disadvantage were associated with an increased

likelihood of obesity at WIII. In addition being African American or having an obese parent increased the likelihood of obesity at WIII and being Asian decreased the likelihood of obesity at WIII for females. For males, school level disadvantage was the only level of disadvantage significantly related to obesity at WII and WIII. In addition, having an obese parent and neighborhood racial heterogeneity increased the likelihood of obesity at WII. Having an obese parent and age increased the likelihood of obesity at WIII.

These findings highlight the importance of school context in influencing the health outcomes of both male and female adolescents. Adolescents spend a majority of time in school. So, perhaps it is less surprising that this context is most salient in influencing adolescent development of health and lifestyle behaviors. There are multiple mechanisms through which school disadvantage could influence obesity status in adolescence. For example, because the poor are more likely to be overweight, poor schools might have larger numbers of overweight and obese students who influence other adolescents' ideas of normal weight, as well as influence their health behavior. In addition to poor schools having a social environment with negative health role modeling, poor schools may not have healthy school lunch programs or physical education courses and facilities that might increase healthy eating and physical activity and reduce or prevent obesity. I do not measure these aspects of the school, but this would be an important area for future research.

These findings also highlight the lasting effects of adolescent social context on obesity in later life. The lifestyle behaviors and habits developed in adolescence carry into adulthood. Because of the enduring relationship between adolescent social disadvantage and obesity in adulthood, it is important to better understand what elements of the school social context serve to influence physical activity and eating behaviors that determine weight gain.

In addition, for females, family level disadvantage and peer education remain important influences on young adult obesity. Understanding the mechanisms that influence obesity risk at these levels of social context will also be important in reducing levels of obesity among females.

There are three important limitations to this research, which involve the measurement of neighborhood, selection bias and my focus on structural poverty.

Census block groups are imperfect operationalizations of the concept of local community (Sampson et al. 2002; Robert 1999). Some researchers argue that neighborhoods are better defined by the social networks of neighborhood interactions or the logic of street patterns, as well as other physical and social characteristics of neighborhoods collected through direct observation (Burton, Price-Spratlen and Spencer 1997; Coulton et al. 2001; Chaskin 1995, 1997; Grannis 1998, Sampson and Raudenbush 1999). Although Add Health does not contain the data to support these definitions of neighborhood, the school-based sampling design allows for accurate descriptions of an adolescent's school and peer group context (refer to the Data section for a more detailed description). Schools also help to serve as an adequate proxy of the neighborhood environments where respondents reside. In addition, although these administratively defined units (i.e. block groups) serve as imperfect proxies of neighborhoods, they are generally consistent with the idea of nested ecological structures (Brooks-Gunn et al. 1997a; Sampson et al. 2002).

As discussed earlier, there is also the possibility of selection bias at multiple levels of social context. Selection bias is problematic because I might be capturing differential selection into peer groups, schools and neighborhoods, rather than true contextual effects. I attempt to minimize selection bias by controlling for characteristics associated with selection

into schools and neighborhoods, and also associated with health (i.e., parent education, parent welfare/poverty, parent obesity, family structure and race/ethnicity). These controls also help to account for the aggregation effects of individual characteristics that are confounded with school-level measures (Manski 1993)

This analysis focused on the structural aspects of socioeconomic disadvantage at the family, peer, school and neighborhood level in relation to obesity at the individual level. A growing amount of research investigating the contextual effects of disadvantage on health has begun to study the more proximal mechanisms that link disadvantage to health (Pickett and Pearl 2001; Robert 1999). Although an important endeavor, the study of basic relationships between disadvantage at the aggregate level and health remain a research priority in the fields of both sociology and public health (Link and Phelan 1995). No research has examined the multiple levels of contextual disadvantage in one model. We need to first understand the multiple, independent and interactive effects of social context on obesity in adolescence and its enduring effects in adulthood before examining mechanisms. Following along the lines of mechanisms research, in Chapter 4, I investigate the intervening roles of parenting practices in the relationship between poverty and physical activity outcomes in adolescence and young adulthood.

References

- Adler, Nancy E., Thomas Boyce, Margaret A. Chesney, Sheldon Cohen, Susan Folkman, Robert L. Kahn, and Leonard Syme. 1994. "Socioeconomic Status and Health: The Challenge of the Gradient." *American Psychologist* 49:15-24.
- Anderson, Elijah. 1999. *The Code Of The Street: Decency, Violence, And The Moral Life Of The Inner City*. New York, NY: W.W. Norton.
- Anderson, Elijah. 1990. *Streetwise: Race, Class, And Change In An Urban Community*. Chicago, IL: University of Chicago Press.
- Antonovsky, Aaron. 1967. "Social Class, Life Expectancy and Overall Mortality." *Milbank Memorial Fund Quarterly* 45:31-37.
- Ben-Shlomo, Yoav and Diana Kuh. 2002. "A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges and Interdisciplinary Perspectives." *International Journal of Epidemiology* 31: 285-293.
- Bishaw, Alemayehu. 2005. "Areas with Concentrated Poverty: 1999." Census 2000 Special Reports, U.S. Census Bureau. Retrieved October 18, 2007 (<http://www.census.gov/prod/2005pubs/censr-16.pdf>).
- Blackwell, Debra L., Mark D. Hayward and Eileen M. Crimmins. 2001. "Does Childhood Health Affect Chronic Morbidity in Later Life?" *Social Science and Medicine* 52:1269-1284.
- Boardman, Jason D., Jarron M. Saint Onge, Richard G. Rogers and Justin T. Denney. 2005. "Race Differentials in Obesity: The Impact of Place." *Journal of Health and Social Behavior* 46: 229-243.
- Borsari, Brian and Kate B. Carey. 2001. "Peer Influences on College Drinking: A Review of the Research." *Journal of Substance Abuse* 13(4): 391-424.
- Braveman, Paula A., Catherine Cubbin, Susan Egerter, Sekai Chideya, Kristen S. Marchi, Marilyn Metzler, Samuel Posner. 2005. "Socioeconomic Status in Health Research: One Size Does Not Fit All." *The Journal of the American Medical Association* 294(22): 2879-2888.
- Bronfenbrenner, Urie. 1979. *The Ecology of Human Development: Experiments by Nature and Design*. Cambridge, MA: Harvard University Press.
- Bronfenbrenner, Urie and Pamela A. Morris. 1998. "The Ecology of Developmental Processes." Pp.992-1028 In *Handbook of Child Psychology: Vol. 1: Theoretical Models of Human Development, 5th Edition*, edited by W. Damon and R. M. Lerner, New York, NY: Wiley.

Bronte-Tinkew, Jacinta, Brett Brown, Jennifer Carrano and Rebecca Shwalb. 2005. "Logic Models and Outcomes for Youth in the Transition to Adulthood: Report to the DC Children and Youth Investment Trust Corporation." Washington, D.C.: Child Trends, Inc., Retrieved September 21, 2006 (www.childtrends.org/Files/Transition_to_Adulthood_Report-FINAL.pdf).

Brooks-Gunn, Jeanne, Greg J. Duncan, Pamela K. Klebanov, Naomi Sealand. 1993. "Do Neighborhoods Affect Child and Adolescent Development?" *American Journal of Sociology* 7(2):55-71.

Brooks-Gunn, Jeanne and Greg J. Duncan. 1997. "The Effects of Poverty on Children." *The Future of Children. Children and Poverty* 99:353-395.

Brooks-Gunn, Jeanne, Greg J. Duncan and J. Lawrence Aber (Eds.). 1997a. *Neighborhood Poverty: Context and Consequences for Children Volume I*. New York, NY: Russell Sage Foundation.

Brooks-Gunn, Jeanne, Greg J. Duncan and J. Lawrence Aber (Eds.). 1997b. *Neighborhood Poverty: Policy Implications in Studying Neighborhoods Volume II*. New York, NY: Russell Sage Foundation.

Brown, B. Bradford. 1990. "Peer Groups and Peer Cultures." Pp. 171-96 in *At the Threshold: The Developing Adolescent*, edited by S.S. Feldman and G.R. Elliott. Cambridge, MA: Harvard University Press.

Burton, Linda. M., Townsend Price-Spratlen, and Margaret B. Spencer. 1997. On Ways of Thinking About and Measuring Neighborhoods: Implications for Studying Context and Development Among Minority Children. In *Neighborhood Poverty: Contexts and Consequences for Development*, edited by G. Duncan, J. Brooks-Gunn, and J. Laser. New York: Russell Sage Foundation.

Centers for Disease Control and Prevention (CDC). 2004. "Overweight Children and Adolescents: Recommendations to Screen, Assess and Manage." Atlanta, GA: Centers for Disease Control, Retrieved April 10, 2004 (www.cdc.gov/nccdphp/dnpa/growthcharts/training/modules/module3/text/module3print.pdf).

Chantala, Kim, C. M. Suchindran and Dan Blanchette. 2005. "Adjusting for Unequal Selection Probability in Multilevel Models: A Comparison of Software Packages." North American Stata Users' Group Meetings 2005 from Stata Users Group, Retrieved January 6, 2008 (<http://repec.org/nasug2005/Chantala.ppt>).

Chaskin, Robert J. 1995. *Defining Neighborhood History, Theory, and Practice*. Chapin Hall Report. Chicago, IL: Chapin Hall Center for Children at the University of Chicago.

Chaskin, Robert J. 1997. "Perspectives on Neighborhood and Community: A Review of the

Literature.” *Social Science Review* 71:521-547.

Christakis, Nicholas A. and James H. Fowler. 2007. “The Spread of Obesity in a Large Social Network Over 32 Years.” *The New England Journal of Medicine* 357:370-379.

Cohen, Deborah A , Brian K. Finch, Aimee Bower and Narayan Sastry. 2006. “Collective Efficacy and Obesity: The Potential Influence of Social Factors on Health.” *Social Science and Medicine* 62:769-778.

Cole, Tim J., Mary C. Bellizi, Katherine M. Flegal, William H. Dietz. 2000. “Establishing a Standard Definition for Child Overweight and Obesity Worldwide: International Survey.” *British Medical Journal* 320: 1–6.

Coulton, Claudia J., Jill Korbin, Tsui Chan and Marilyn Su. 2001. “Mapping Residents’ Perceptions of Neighborhood Boundaries: A Methodological Note.” *American Journal of Community Psychology* 29 371-383.

Crawford, Patricia B., Eva Obarzanek, George Schrepper, Phyllis Barrier, Sue Goldman, Margaret M. Frederick and Z. I. Sabry. 1995. “The Effects of Race, Household Income, and Parental education on Nutrient Intake of 9- and 10-Year-Old Girls: NHLBI Growth and Health Study.” *Annals of Epidemiology* 5(5):360-368.

Crimmins, Eileen M. and Yasuhiko Saito. 2001. “Trends in Healthy Life Expectancy in the United States, 1970-1990: Gender, Racial, and Educational Differences.” *Social Science and Medicine* 52(11):1629-1641.

Cummins, Steve and Sally McIntyre. 2006. “Food Environments and Obesity—Neighborhood of Nation?” *International Journal of Epidemiology* 35: 100-104.

Deshmukh-Taskar Priya, Theresa A. Nicklas, Su-Jau Yang and Gerald S. Berenson. 2007. “Does Food Group Consumption Vary by Differences in Socioeconomic, Demographic, and Lifestyle Factors in Young Adults? The Bogalusa Heart Study.” *Journal of the American Dietetic Association* 107:223-234.

Dietz, William H. 1998. “Health Consequences of Obesity in Youth: Childhood Predictors of Adult Disease.” *Pediatrics* 101:518-525.

Dietz, William H. and Steven L. Gortmaker. 2001. “Preventing Obesity in Children and Adolescents.” *Annual Review of Public Health* 22: 337-353.

Diez- Roux, Ana V. 2000. “Multilevel Analysis in Public Health Research.” *Annual Review of Public Health* 21: 171-192.

Douglas-Hall, Ayana and Michelle Chau. 2007. “Basic Facts about Low-Income Children Birth to Age 18.” National Center for Children in Poverty (NCCP). Retrieved October 18, 2007 (http://www.nccp.org/publications/pub_762.html)

Duncan, Greg J. and Stephen W. Raudenbush. 1999. "Assessing the Effects of Context in Studies of Child and Youth Development." *Educational Psychologist* 34(1):29-41.

Earls, Felton and Mary Carlson 2001. "The Social Ecology of Child Health and Well-Being." *Annual Review of Public Health* 22:143-166.

Elder, Glen H., Jr. 1998. "The Life Course as Developmental Theory." *Child Development* 69:1-12.

Elder, Glen H., Jr. 1997. "The Life Course and Human Development," Pp. 939-991 In *Handbook of Child Psychology, Volume 1: Theoretical Models of Human Development*, edited by R. M. Lerner and W. Damon. New York, NY: Wiley.

Elo, Irma and Samuel Preston. 1992. "The Effect of Early Life Conditions on Adult Mortality: A Review." *Population Index*. 58(2):186-212.

Entwisle, Barbara. 2007. "Putting People into Place." *Demography* 44(4):687-703.

Evans, William N., Wallace E. Oates and Robert M. Schwab. 1992. "Measuring Peer Group Effects: A Study of Teenage Behavior." *The Journal Political Economy* 100: 966-991.

Fass, Sarah and Cauthen, Nancy K. 2006. "Who are America's Poor Children? The Official Story." National Center for Children in Poverty (NCCP). Retrieved September 21, 2007 (http://www.nccp.org/publications/pub_684.html)

Frumkin, Howard. 2005. "Health, Equity, and the Built Environment." *Environmental Health Perspectives* 113(5): A290-A291.

Galbraith, Kenneth. 1969. *The Affluent Society*. Boston, MA: Houghton Mifflin.

Giordano, Peggy C. 2003. "Relationships in Adolescence." *Annual Review of Sociology* 29: 257-281.

Gephart, Martha A. 1997. "Neighborhood and Communities as Contexts for Development." Pp. 1-43. In *Neighborhood Poverty: Context and Consequences for Children Volume I*, edited by J. Brooks-Gunn, G. J. Duncan and J. L. Aber, New York, NY: Russell Sage Foundation.

Glass, Thomas A. and Matthew J. McAtee. 2006. "Behavioral Science at the Crossroads in Public Health: Extending Horizons, Envisioning the Future." *Social Science and Medicine* 62:1650-1671.

Grannis, Robert A. 1998. The Importance of Trivial Streets: Residential Streets and Residential Segregation. *American Journal of Sociology* 103(6):1530-1564.

Goodman, Elizabeth, Bin Huang, Terrance J. Wade and Robert S. Kahn. 2003. "A Multilevel Analysis of the Relation of Socioeconomic Status to Adolescent Depressive Symptoms: Does School Context Matter?" *The Journal of Pediatrics* 143: 451-456.

Gordon-Larsen, Penny, Robert G. McMurray and Barry M. Popkin. 2000. "Determinants of Adolescent Physical Activity and Inactivity Patterns." *Pediatrics* 105(6), Retrieved September 21, 2006 (<http://pediatrics.aappublications.org/cgi/content/abstract/105/6/e83>).

Guo, Guang and Hongxin Zhao. 2000. "Multilevel Modeling for Binary Data." *Annual Review of Sociology* 26: 441-462.

Halfon, Neal and Miles Hochstein. 2002. "Life Course Health Development: An Integrated Framework for Developing Health, Policy, and Research." *The Milbank Quarterly* 80(3): 433-479.

Harding, David J. 2007. "Cultural Context, Sexual Behavior, and Romantic Relationships in Disadvantaged Neighborhoods." *American Sociological Review* 72:341-364.

Harrington, Michael. 1997. *The Other America: Poverty in the United States*. New York, NY: Touchstone.

Harris, Kathleen Mullan, Francesca Florey, Joyce W. Tabor, Peter S. Bearman, Jo Jones, and J. Richard Udry. 2003. The National Longitudinal Study of Adolescent Health: Research Design. Retrieved September 21, 2006 (<http://www.cpc.unc.edu/projects/addhealth/design.html>.)

Hayward, Mark D. and Bridget K. Gorman. 2004. "The Long Arm of Childhood: The Influence of Early-Life Social Conditions on Men's Mortality." *Demography* 41:87-107.

House, James S., James M. Lepkowski, Ann M. Kinney, Richard P. Mero, Ronald C. Kessler, and A. Regula Herzog. 1994. "The Social Stratification of Aging and Health." *Journal of Health and Social Behavior* 35: 213-234.

House, James S., Ronald C. Kessler, A. Regula Herzog, Richard P. Mero, Ann M. Kinney, and Martha J. Breslow. 1990. "Age, Socioeconomic Status, and Health." *The Millbank Memorial Fund* 68:383-411.

Iceland, John and Rima Wilkes. 2006. "Does Socioeconomic Status Matter? Race, Class, and Residential Segregation." *Social Problems* 53: 248-273.

Institute of Medicine (IOM), Committee on Prevention of Obesity in Children and Youth. 2005. *Preventing Childhood Obesity: Health in the Balance*. Edited by J. P. Koplan, C. T. Liverman and V. I. Kraak. Washington, DC: National Academies Press.

Institute of Medicine (IOM). 2001. *Health and Behavior: The Interplay of Biological, Behavioral and Societal Influences*. Washington, DC: National Academies Press.

Janssen, Ian, William F. Boyce, Kelly Simpson and William Pickett. 2006. "Influence of Individual- and Area-Level Measures of Socioeconomic Status and Obesity, Unhealthy Eating, and Physical Inactivity in Canadian Adolescents." *The American Journal of Clinical Nutrition* 83: 139-145.

Jencks, Christopher and Susan E. Mayer. 1990. "The Social Consequences of Growing Up in a Poor Neighborhood." Pp. 111-186. In *Inner-City Poverty in the United States*, edited by L. E. Lynn Jr. and M. G. H. McGeary, Washington, DC: National Academy Press.

Leventhal, Tama and Jeanne Brooks-Gunn. 2003. "Children and Youth in Neighborhood Contexts." *Current Directions in Psychological Science* 12(1):27-31.

Kawachi, Ichiro and Lisa F. Berkman (Eds.). 2003. *Neighborhoods and Health*. New York, NY: Oxford University Press.

Kirby, James. B and Toshiko Kaneda. 2005. "Neighborhood Socioeconomic Disadvantage and Access to Health Care." *Journal of Health and Social Behavior* 46:15-31.

Kitagawa, Evelyn M. and Philip M. Hauser. 1973. *Differential Mortality in the United States: A Study of Socioeconomic Epidemiology*. Cambridge, MA: Harvard University Press.

La Greca, Annette M., Mitchell J. Prinstein and Michael D. Fetter. 2001. "Adolescent Peer Crowd Affiliation: Linkages with Health-Risk Behaviors and Close Friendships." *Journal of Pediatric Psychology* 26(3): 131-143.

Lareau, Annette. 2003. "Invisible Inequality: Social Class and Childrearing in Black Families and White Families." *American Sociological Review* 67(5): 747-776.

Lee, Hedwig, Kathleen Mullan Harris and Penny Gordon-Larsen. 2008. "Life Course Perspectives on the Links Between Poverty and Obesity During the Transition to Young Adulthood." *Population Research and Policy Review*. [Forthcoming]

Lee, Valerie E. and Anthony S. Bryk. 1989. "A Multilevel Model of the Social Distribution of Educational Achievement." *Sociology of Education* 62:172-192.

Link, Bruce G. and Jo Phelan. 1995. "Social Conditions as Fundamental Causes of Disease." *Journal of Health and Social Behavior* 35: 80-94.

Little, Ramon C., George A. Milliken, Walter W. Stroup and Russel D. Wolfinger. 1996. *SAS System for Mixed Models*. Cary, NC: SAS.

Lowry, Richard, Laura Kann, Janet L. Collins and Lloyd J. Kolbe. 1996. "The Effect of Socioeconomic Status on Chronic Disease Risk Behaviors Among U.S. Adolescents." *The Journal of the American Medical Association* 276(10): 792-797.

Manski, C. 1993. "Identification of Endogenous Social Effects: The Reflection Problem."

Review of Economic Studies 60:531-542.

Massey, Douglas S. 1996. "The Age of Extremes: Concentrated Affluence and Poverty in the Twenty First Century." *Demography* 33:395-412.

Massey, Douglas S. and Nancy A. Denton. 1993. *American Apartheid: Segregation and the Making of the Underclass*. Cambridge, MA: Harvard University Press.

McCulloch, Charles E. and Shayle R. Searle. 2001. *Generalized, Linear and Mixed Models*. Series in Probability in Statistics. New York, NY: Wiley.

Morenoff, Jeffrey D., Ana V. Diez Roux, Theresa Osypuk and Ben Hansen. 2006. "Residential Environments and Obesity: What Can We Learn about Policy Interventions from Observational Studies?" Bethesda, MD: National Poverty Center's "Health Effects of Non-Health Policy" Conference, Retrieved September 21, 2006 (<http://www.ssw.umich.edu/CASD/Morenoff%5B1%5D.pdf>).

National Center for Children in Poverty (NCCP). 2007. "Low-Income Children in the United States: National and State Trend Data, 1996-2006." Columbia University, Mailman School of Public Health, Retrieved October 18, 2007 (http://www.nccp.org/publications/pdf/text_761.pdf).

National Heart, Lung, and Blood Institute (NHLBI) Obesity Education Initiative. 1998. "NHLBI Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. Clinical Guidelines on the Identification, evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report." *Obesity Research* 6(Supplement): 51S-209S.

National Research Council (NRC) and Institute of Medicine (IOM), Committee on Evaluation of Children's Health. 2004. *Children's Health, the Nation's Wealth: Assessing and Improving Child Health*. Washington, DC: The National Academies Press.

Patrick, Heather and Theresa A. Nicklas. 2005. "A Review of Family and Social Determinants of Children's Eating Patterns and Diet Quality." *Journal of the American College of Nutrition* 24(2):83-92.

Pebley, Anne R., and Narayan Sastry. 2004. "Neighborhoods, Poverty and Children's Well-being: A Review," In *Social Inequality*, edited by Kathryn Neckerman, New York, NY: Russell Sage Foundation.

Perry, Cheryl P. 2000. "Paper Contribution E: Preadolescent and Adolescent Influences on Health." Pp. 217-253 In *Promoting Health: Intervention Strategies from Social and Behavioral Research*, edited by B.D. Smedley and S. L. Syme, Washington, DC: National Academies Press.

Pickett, K. E. and M. Pearl. 2001. "Multilevel Analyses of Neighborhood Socioeconomic

Context and Health Outcomes: A Critical Review.” *Journal of Epidemiology and Community Health* 55: 111-122.

Prinstein, Mitchell J., Julie Boergers and Anthony Spirito. "Adolescents' and Their Friends' Health-Risk Behavior: Factors That Alter or Add to Peer Influence." *Journal of Pediatric Psychology* 26(5): 287-298.

Rabe-Hesketh, S. and A. Skrondal. 2006. “Multilevel Modeling of complex Survey Data.” *Journal of the Royal Statistical Society* 160(4):805-827.

Rabe-Hesketh, S., A. Skrondal and A. Pickles. 2004. “GLLAMM Manual.” Berkeley, CA: University of California, Berkeley.

Raudenbush, Stephen W. and Anthony S. Bryk. 2002a. “A Hierarchical Model for Studying School Effects.” *Sociology of Education* 59: 1-17.

Raudenbush, Stephen W. and Anthony S. Bryk. 2002b. *Hierarchical Linear Models: Applications and Data Analysis Methods, 2nd Edition*. Thousand Oaks, CA: Sage Publications.

Richmond, Tracy K., Rodney A. Hayward, Sheila Gahagan, Allison E. Field and Michele Heisler. 2006. “Can School Income and Racial Composition Explain the Ethnic Disparity in Adolescent Physical Activity Participation?” *Pediatrics* 117(6): 2158-2166.

Robert, Stephanie A. 1999. "Socioeconomic Position and Health: The Independent Contribution of Community Socioeconomic Context." *Annual Review of Sociology* 25: 489-516.

Robert, Stephanie A. and Eric N. Reither. “A Multilevel Analysis of Race, Community Disadvantage, and Body Mass Index among Adults in the US.” *Social Science and Medicine* 59: 2421-2434.

Ross, Catherine E. and Chia-ling Wu. 1995. “The Links between Education and Health.” *American Sociological Review* 60:719-745.

Sallis, James F., Robert B. Cervero, William Ascher, Karla A. Henderson, M. Katherine Kraft and Jacqueline Kerr. 2006. “An Ecological Approach to Creating Active Living Communities.” *Annual Review of Public Health* 27: 297-322.

Sallis, James F., Judith J. Prochaska and Wendell C. Taylor. 2000. "A Review of the Correlates of Physical Activity of Children and Adolescents." *Medicine and Science in Sports and Exercise* 32(5): 963-975.

Sampson, Robert J., Jeffrey D. Morenoff and Thomas Gannon-Rowley. 2002. “Assessing 'Neighborhood Effects': Social Processes and New Directions in Research.” *Annual Review of Sociology* 28: 443-478.

- Sampson, Robert J., Stephen W. Raudenbush, Felton Earls. 1997. "Neighborhoods and Violent Crime: A Multilevel Study of Collective efficacy." *Science* 277:918-924.
- Sampson, Robert J. and Stephen W. Raudenbush. 1999. "Systematic Social Observation of Public Spaces: A New Look at Disorder in Urban Neighborhoods." *American Journal of Sociology*. 105(3): 603-651.
- Saporito, Salvatore and Deenesh Sohoni. 2007. "Mapping Educational Inequality: Concentrations of Poverty among Poor and Minority Students in Public Schools." *Social Forces* 85(3):1227-1253.
- Sausenthaler, Stephanie, Iris Kompauer, Andreas Mielck, Michael Borte, Olf Herbarth, Beate Schaaf, Andrea von Berg and Joachim Heinrich, for the LISA Study Group. 2007. "Impact of Parent Education and Income Inequality on Children's Food Intake." *Public Health Nutrition* 10(1):24-33.
- Serdula, Mary K., Donna Ivery, Ralph J. Coates, David S. Freedman, David F. Williamson and Tim Byers. 1993. "Do Obese Children Become Obese Adults? A Review of the Literature." *Preventive Medicine* 22: 167-177.
- Shavers, Vickie L. 2007. "Measurement of Socioeconomic Status in Health Disparities Research." *Journal of the National Medical Association* 99(9): 1013-1023.
- Snijders, Tom and Roel J. Bosker. 1999. *Multilevel Analysis: An Introduction to Basic and Advanced Multilevel Modeling*. Thousand Oaks, CA: Sage Publications.
- Stafford Mai and Marmot, Michael. 2003. "Neighbourhood Deprivation and Health: Does it Affect Us All Equally?" *International Journal of Epidemiology* 32: 357-366.
- Steffensmeier, Darrell and Dana Haynie. 2002. "Gender, Structural Disadvantage, and Urban Crime: Do Macrosocial Variables also explain Offending Rates?" *Criminology* 38(2):403-438.
- Stenhammar, Christina, Anna Sarkadi and Birgitta Edlund. 2007. "The Role of Parent's Educational background in Health Lifestyle Practices and Eating Habits of their 6-year-old Children." *Public Health Nutrition* 10:1305-1313.
- Taylor, Shelley E. and Rena L. Repetti. 1997. "Health Psychology: What is an Unhealthy Environment and How Does It Get Under the Skin?" *Annual Review of Psychology* 48:411-447.
- Tienda, Marta. 1991. "Poor People, Poor Places: Deciphering Neighborhood Effects on Poverty Outcomes." Pp. 244-262 in *Macro-micro Linkages in Sociology*, edited by J. Huber. Newbury Park, CA: Sage.
- U.S. Census Bureau, Geography Division. 1994. "Geographic Areas Reference Manual."

Geography Division, Bureau of the Census, Washington, DC. Retrieved October 18, 2008.(<http://www.census.gov/geo/www/garm.html>).

Unites States Department of Health and Human Services (HHS). 2007. "The 2007 HHS Poverty Guidelines." Retrieved September 21, 2007 (<http://aspe.hhs.gov/poverty/07poverty.shtml>).

Vargas, Clemencia M., Deborah D. Ingram, and Richard F. Gillum. 2000. "Incidence of Hypertensionand Educational Attainment: The NHANES-I Epidemiologic Followup Study." *American Journal of Epidemiology* 152(3): 272-278.

Wickrama, K. A. Thulitha, K.A.S. Wickrama, and Chalandra M. Bryant. 2006. "Community Influence on Adolescent Obesity: Race/Ethnic Differences." *Journal of Youth and Adolescence* 35(4): 641-651.

Wilson, William J. 1987. *The Truly Disadvantaged: The Inner City, the Underclass, and Public Policy*. Chicago: University of Chicago Press.

World Health Organization (WHO). 2000. "Obesity: Preventing and Managing the Global Epidemic: Report of a WHO Consultation" Geneva, Switzerland: World Health Organization (WHO Technical Report Series, No. 894), Retrieved September 21, 2006 (http://whqlibdoc.who.int/trs/WHO_TRS_894.pdf).

Zakarian, Joy. M., Melbourne F. Hovell, C. Richard Hofstetter, James F. Sallis and Kristen J. Keating. 1994. "Correlates of Vigorous Exercise in a Predominantly Low SES and Minority High School Population." *Preventive Medicine* 23: 314-321.

Figure 3.1. Conceptual Model

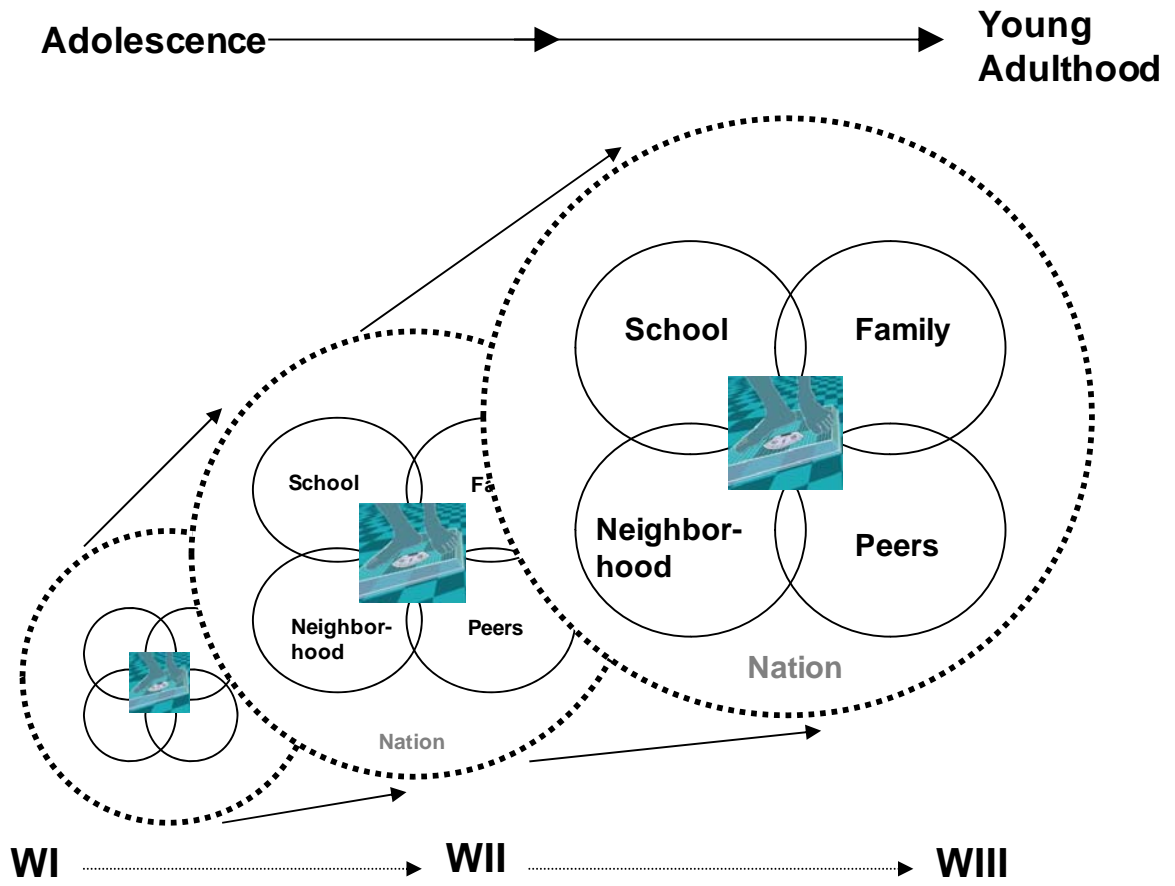


Table 3.1. Variable Descriptions, Means and Standard Deviations by Sex

Variable	Description	Female		Male	
		Mean	SE	Mean	SE
Outcomes					
Obesity					
Obese at Wave II	Obese at Wave II	0.10	0.007	0.12	0.009
Obese at Wave III	Obese at Wave III	0.23	0.012	0.22	0.010
Individual Level Measures					
Race/Ethnicity					
Non-Hispanic White (Reference Category)	Respondent reported white	0.70	0.031	0.69	0.032
Non-Hispanic Black	Respondent reported black	0.16	0.024	0.14	0.022
Hispanic	Respondent reported Hispanic	0.10	0.015	0.11	0.016
Asian	Respondent reported Asian	0.04	0.008	0.05	0.011
Other	Respondent reported other race	0.01	0.002	0.01	0.003
Age	Self-reported age at WI (Range: 12-20)	14.85	0.118	15.05	0.130
Parent Obese	Report of mother or father being obese	0.21	0.009	0.22	0.010
Missing Report of Parent Obese	Missing report of mother or father being obese	0.09	0.009	0.08	0.008
Family Level Measures					
Welfare Status and/or Poverty Status	Welfare receipt prior to the age and/or 18 of Family Income at WI < \$16,000/year	0.28	0.015	0.27	0.017
Parent Education High School or Less	Highest educated parent completed a high school degree/GED or less	0.64	0.018	0.61	0.017
Family Structure					
2 Biological Parents (Reference Category)	2 Biological/Adoptive Parents	0.60	0.015	0.61	0.016
Step family	Step family	0.16	0.008	0.16	0.007
Single mother	Single mother	0.19	0.011	0.17	0.012
Single father	Single father	0.02	0.004	0.03	0.003
Other family structure	Other family structure	0.03	0.004	0.03	0.005
Peer Level Measures					
Parent Education Less than High School	Proportion of peers with highest educated parent who completed a high school degree/GED or less (Range: 0-100%)	0.51	0.013	0.50	0.015
Single Mother Family Structure	Proportion of peers who live with their mother only (Range: 0-100%)	0.18	0.008	0.17	0.008
No Friendship Information	Respondent either reported no school friends or information on reported friends was not available (Range: 0-100%)	0.09	0.009	0.16	0.013
School Level Measures					
Parent Education Less than High School	Proportion of school students with highest educated parent who completed a high school degree/GED or less (Range: 9-77%)	0.51	0.012	0.51	0.012
Single Mother Family Structure	Proportion of school students who live with their mother only (Range: 3-41%)	0.19	0.007	0.19	0.007
Neighborhood Level Risk					
Adult Education Less than High School	Proportion of individuals 25 years or older who completed less than high school/GED (Range: 0-84%)	0.27	0.010	0.27	0.009
Proportion of Female Headed Households	Proportion of female headed households with own children < 18 yr. old at WI	0.07	0.004	0.07	0.004
Racial Dispersion	Continuous tract level measure of dispersion in racial composition (Range: 0-93%)	0.22	0.019	0.22	0.018
Urban Block Group	Categorical measure to designate if respondent lives in an urbanized area	0.47	0.045	0.46	0.045
N		3,926		3,622	

Data are weighted.

Table 3.2. Associations Between Disadvantage Measures and Obesity Status at Waves II and III by Sex

	Female		Male					
	Obese Wave II	Obese Wave III	Obese Wave II	Obese Wave III				
<i>Family Level Measures</i>								
Welfare/Poverty Status								
No	0.09	**	0.20	***	0.11		0.21	*
Yes	0.13		0.30		0.14		0.25	
Parent Education High School or Less								
No	0.07	**	0.17	***	0.09	***	0.18	***
Yes	0.11		0.27		0.14		0.25	
Mother Only Family Structure								
No	0.09	*	0.22	**	0.12		0.22	
Yes	0.13		0.29		0.14		0.24	
<i>Peer Level Measures</i>								
Parent Education High School or Less								
Less than 65%	0.08	***	0.21	**	0.11	*	0.22	
65% or more	0.15		0.28		0.15		0.23	
Single Mother Family Structure								
Less than 28%	0.09		0.22	*	0.11	*	0.20	**
28% or more	0.12		0.27		0.15		0.27	
<i>School Level Measures</i>								
Parent Education High School or Less								
Less than 60%	0.08	***	0.21	*	0.11	**	0.20	*
60% or more	0.14		0.28		0.15		0.26	
Single Mother Family Structure								
Less than 23%	0.09	**	0.20	***	0.11	*	0.21	*
23% or more	0.14		0.31		0.15		0.26	
<i>Neighborhood Level Risk</i>								
Adult Education Less than High School								
Less than 38%	0.09	**	0.21	***	0.11	*	0.20	***
38% or more	0.14		0.31		0.16		0.28	
Proportion of Female Headed Households								
Less than 10%	0.09		0.22	*	0.11	**	0.21	**
10% or more	0.12		0.28		0.17		0.28	
N	3,926		3,926		3,622		3,622	

Data are weighted.

* significant at .05 level

** significant at .01 level

*** significant at .001 level

Table 3.3. Mean Contextual Family, Peer, School and Neighborhood Disadvantage by Race and Sex

	Family Parent Education		Peer Parent Education		School Parent Education		Neighborhood Parent Education	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Female (N= 3,926)								
White	0.60	0.021	0.51	0.016	0.52	0.014	0.24	0.010
Black	0.72	0.032	0.52	0.026	0.50	0.019	0.36	0.016
Asian	0.54	0.060	0.37	0.039	0.45	0.018	0.27	0.022
Other	0.65	0.081	0.49	0.048	0.52	0.025	0.27	0.047
Hispanic	0.82	0.029	0.55	0.018	0.49	0.017	0.36	0.026
Male (N= 3,622)								
White	0.59	0.021	0.50	0.019	0.52	0.015	0.24	0.010
Black	0.67	0.035	0.49	0.023	0.50	0.020	0.35	0.018
Asian	0.40	0.036	0.37	0.028	0.43	0.020	0.25	0.022
Other	0.79	0.069	0.54	0.049	0.50	0.028	0.34	0.035
Hispanic	0.73	0.033	0.50	0.023	0.50	0.014	0.33	0.023

Data are weighted.

Education measure is parent education of a high school degree or less for all but neighborhood, which is the percentage of the adults with less than a high school degree.

Table 3.4. Mean Contextual Peer, School and Neighborhood Disadvantage by Family Disadvantage and Sex

	Peer Parent Education		School Parent Education		Neighborhood Parent Education	
	Mean	SE	Mean	SE	Mean	SE
Female (N= 3,926)						
Parent Education						
Parent More than High School	0.41	0.019	0.46	0.017	0.21	0.009
Parent High School or Less	0.57	0.011	0.54	0.010	0.31	0.010
Poverty/Welfare						
No	0.49	0.014	0.50	0.013	0.25	0.009
Yes	0.56	0.015	0.54	0.011	0.35	0.012
Male (N= 3,622)						
Parent Education						
Parent More than High School	0.41	0.018	0.47	0.015	0.23	0.010
Parent High School or Less	0.55	0.014	0.54	0.011	0.30	0.010
Poverty/Welfare						
No	0.48	0.017	0.50	0.014	0.25	0.009
Yes	0.55	0.016	0.54	0.011	0.33	0.011

Data are weighted.

Note:

All differences in means are significant at the $p < .001$ level.

Education measure is parent education of a high school degree or less for all but neighborhood, which is the percentage of the adults with less than a high school degree.

Table 3.5. Multilevel Logistic Regression Results for Obese at Wave II and Wave III for Females (N=3,926)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Obese WII	Obese WIII	Obese WII	Obese WIII	Obese WII	Obese WIII	Obese WII	Obese WIII	Obese WII	Obese WIII
<i>Individual Level Measures</i>										
Race/Ethnicity										
Non-Hispanic Black			0.502** (0.145)	0.444*** (0.110)	0.606*** (0.139)	0.523*** (0.109)	0.494** (0.159)	0.456*** (0.119)	0.495** (0.159)	0.447*** (0.119)
Hispanic			0.088 (0.183)	0.271* (0.129)	0.221 (0.175)	0.337** (0.127)	0.076 (0.199)	0.251 (0.141)	0.053 (0.200)	0.215 (0.141)
Asian			-0.234 (0.294)	-0.622** (0.231)	-0.033 (0.291)	-0.527* (0.231)	-0.193 (0.304)	-0.640** (0.238)	-0.158 (0.305)	-0.624** (0.239)
Other			0.305 (0.417)	0.4939 (0.313)	0.404 (0.410)	0.492 (0.311)	0.269 (0.417)	0.405 (0.314)	0.287 (0.418)	0.424 (0.314)
Age at WI			0.052 (0.038)	0.046 (0.028)	0.032 (0.037)	0.032 (0.028)	0.031 (0.037)	0.035 (0.027)	0.027 (0.037)	0.031 (0.027)
Parent Obese			1.413*** (0.123)	1.155*** (0.094)	1.401*** (0.122)	1.145*** (0.094)	1.398*** (0.122)	1.143*** (0.094)	1.400*** (0.123)	1.149*** (0.094)
<i>Family Level Measures</i>										
Welfare Status and/or Poverty Status										
			0.245 (0.129)	0.372*** (0.094)	0.174 (0.1329)	0.333** (0.094)	0.156 (0.130)	0.321** (0.095)	0.146 (0.130)	0.302** (0.095)
Parent Education High School or Less										
			0.313* (0.127)	0.340** (0.092)	0.164 (0.129)	0.247** (0.094)	0.133 (0.131)	0.222* (0.095)	0.111 (0.131)	0.200* (0.095)
Family Structure										
Step family			-0.224 (0.171)	-0.018 (0.117)	-0.223 (0.1704)	-0.019 (0.117)	-0.214 (0.170)	-0.0095 (0.117)	-0.222 (0.171)	-0.017 (0.117)
Single mother			0.053 (0.146)	0.088 (0.108)	0.062 (0.146)	0.092 (0.108)	0.054 (0.146)	0.090 (0.108)	0.058 (0.146)	0.096 (0.108)
Single father			-0.276 (0.444)	-0.186 (0.304)	-0.314 (0.442)	-0.201 (0.303)	-0.312 (0.441)	-0.195 (0.301)	-0.308 (0.442)	-0.218 (0.303)
Other family structure			0.066 (0.271)	-0.017 (0.208)	0.080 (0.270)	-0.011 (0.207)	0.086 (0.270)	0.001 (0.207)	0.096 (0.270)	0.007 (0.207)
<i>Peer Level Measures</i>										
Parent Education High School or Less										
									0.037 (0.023)	0.033* (0.016)
<i>School Level Measures</i>										
Parent Education High School or Less										
					0.025*** (0.005)	0.017*** (0.004)	0.024*** (0.006)	0.015*** (0.004)	0.021** (0.006)	0.012** (0.004)
<i>Neighborhood Level Risk</i>										
Adult Education Less than High School										
							0.003 (0.004)	0.002 (0.003)	0.003 (0.004)	0.002 (0.003)
Proportion of Female Headed Households										
							-0.001 (0.009)	0.000 (0.007)	-0.001 (0.009)	-0.001 (0.007)
Racial Dispersion										
							0.036 (0.024)	0.038 (0.019)	0.036 (0.024)	0.038 (0.019)
Urban Block Group										
							-0.019 (0.128)	-0.119 (0.096)	-0.018 (0.129)	-0.133 (0.096)
Constant	-2.311*** (0.075)	-1.314*** (0.055)	-3.948*** (0.580)	-2.783*** (0.429)	-4.840*** (0.596)	-3.385*** (0.438)	-4.872*** (0.598)	-3.382*** (0.434)	-4.828*** (0.598)	-3.350*** (0.434)
Log Likelihood	-1254.842	-2049.771	-1170.159	-1930.419	-1159.424	-1921.826	-1157.823	-1919.063	-1156.425	-1914.424
<i>Variance Component</i>										
σ^2_{ϵ} (SE)	0.227 (0.089)	0.208 (0.056)	0.076 (0.065)	0.075 (0.042)	0.005 (0.034)	0.033 (0.031)	0.000 (0.000)	0.014 (0.032)	0.000 (0.000)	0.010 (0.032)
σ^2_{η} , rescaled	0.747	0.684	0.251	0.246	0.018	0.108	0.000	0.044	0.000	0.034
ICC	0.065	0.059	0.023	0.022	0.002	0.010	0.000	0.004	0.000	0.003
Proportionate reduction in level-2 variance (vs. Model 1)	-----	-----	66.4%	64.0%	97.6%	84.2%	100.0%	93.6%	100.0%	95.0%

* significant at .05 level
 ** significant at .01 level
 *** significant at .001 level

Note:
 Standard errors are in parentheses
 Omitted categories are: race/ethnicity: white; family structure: two biological parents.
 Equation includes missing data indicators; these are not shown above.

Table 3.6. Multilevel Logistic Regression Results for Obese at Wave II and Wave III for Males (N=3,622)

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Obese WII	Obese WIII	Obese WII	Obese WIII	Obese WII	Obese WIII	Obese WII	Obese WIII	Obese WII	Obese WIII
Individual Level Measures										
Race/Ethnicity										
Non-Hispanic Black			0.144 (0.154)	0.124 (0.122)	0.233 (0.156)	0.165 (0.122)	0.063 (0.168)	0.073 (0.133)	0.065 (0.169)	0.071 (0.134)
Hispanic			0.127 (0.167)	0.231 (0.1289)	0.240 (0.170)	0.279 (0.129)	0.050 (0.185)	0.168 (0.143)	0.051 (0.185)	0.168 (0.143)
Asian			-0.103 (0.2378)	-0.004 (0.175)	0.059 (0.245)	0.073 (0.179)	-0.152 (0.258)	-0.045 (0.189)	-0.155 (0.258)	-0.045 (0.190)
Other			-0.021 (0.504)	0.284 (0.371)	0.053 (0.504)	0.317 (0.370)	-0.142 (0.509)	0.210 (0.374)	-0.144 (0.509)	0.211 (0.374)
Age at WI			-0.033 (0.037)	0.081** (0.028)	-0.051 (0.037)	0.072* (0.029)	-0.054 (0.037)	0.071* (0.028)	-0.053 (0.037)	0.070* (0.09)
Parent Obese			1.199*** (0.118)	1.006*** (0.096)	1.185*** (0.118)	1.000*** (0.096)	1.181*** (0.118)	0.998*** (0.096)	1.181*** (0.118)	0.999*** (0.096)
Family Level Measures										
Welfare Status and/or Poverty Status										
			0.099 (0.126)	0.016 (0.099)	0.052 (0.127)	-0.008 (0.100)	0.035 (0.128)	-0.018 (0.101)	0.035 (0.128)	-0.018 (0.101)
Parent Education High School or Less										
			0.203 (0.119)	0.208* (0.091)	0.106 (0.121)	0.158 (0.093)	0.080 (0.121)	0.140 (0.093)	0.082 (0.122)	0.141 (0.094)
Family Structure										
Step family			0.048 (0.156)	0.074 (0.118)	0.040 (0.156)	0.070 (0.118)	0.035 (0.156)	0.070 (0.118)	0.036 (0.156)	0.070 (0.118)
Single mother			0.232 (0.147)	0.041 (0.119)	0.240 (0.147)	0.044 (0.119)	0.220 (0.147)	0.037 (0.119)	0.220 (0.147)	0.037 (0.119)
Single father			-0.006 (0.333)	0.115 (0.245)	0.008 (0.334)	0.122 (0.245)	-0.009 (0.334)	0.117 (0.245)	-0.010 (0.334)	0.118 (0.245)
Other family structure			0.098 (0.328)	0.289 (0.236)	0.095 (0.327)	0.286 (0.236)	0.057 (0.328)	0.271 (0.235)	0.055 (0.328)	0.270 (0.236)
Peer Level Measures										
Parent Education Less than High School										
									-0.003 (0.023)	-0.002 (0.018)
School Level Measures										
Parent Education Less than High School										
					0.021*** (0.006)	0.010* (0.004)	0.021** (0.006)	0.009* (0.004)	0.021** (0.006)	0.009* (0.005)
Neighborhood Level Risk										
Adult Education Less than High School										
							0.003 (0.004)	0.003 (0.003)	0.003 (0.004)	0.003 (0.003)
Proportion of Female Headed Households										
							0.005 (0.009)	0.001 (0.007)	0.005 (0.009)	0.001 (0.007)
Racial Dispersion										
							0.055* (0.027)	0.032 (0.022)	0.055* (0.027)	0.032 (0.022)
Urban Block Group										
							0.102 (0.136)	0.020 (0.104)	0.104 (0.136)	0.019 (0.104)
Constant	-2.138*** (0.073)	-1.344*** (0.053)	-2.253*** (0.568)	-3.108*** (0.441)	-2.997*** (0.611)	-3.445*** (0.466)	-3.161*** (0.599)	-3.511*** (0.462)	-3.169*** (0.601)	-3.508*** (0.463)
Log Likelihood	-1285.348	-1882.416	-1227.556	-1815.701	-1220.646	-1812.7822	-1217.245	-1810.869	-1217.225	-1810.841
Variance Component										
σ^2_{ϵ} (SE)	0.150 (0.056)	0.108 (0.040)	0.132 (0.060)	0.067 (0.034)	0.101 (0.063)	0.058 (0.034)	0.059 (0.048)	0.039 (0.028)	0.060 (0.049)	0.039 (0.028)
σ^2_{η} , rescaled	0.493	0.341	0.433	0.221	0.332	0.189	0.196	0.129	0.198	0.129
ICC	0.044	0.031	0.038	0.020	0.030	0.017	0.018	0.012	0.018	0.012
Proportionate reduction in level-2 variance (vs. Model 1)	-----	-----	12.2%	35.2%	32.7%	44.6%	60.2%	62.2%	60.2%	62.2%

* significant at .05 level
 ** significant at .01 level
 *** significant at .001 level

Note:
 Standard errors are in parentheses
 Omitted categories are: race/ethnicity: white; family structure: two biological parents.
 Equation includes missing data indicators; these are not shown above.

THE INFLUENCE OF PARENTING PRACTICES AND BEHAVIORS IN THE RELATIONSHIP BETWEEN SOCIAL DISADVANTAGE AND PHYSICAL ACTIVITY IN THE TRANSITION TO YOUNG ADULTHOOD

The transition to adulthood is an important period in the life course marked by demographic milestones in education, employment, residence, fertility and union formation (Rindfuss 1991). The ability to make successful transitions from youth to adulthood requires parental and institutional support, as well as good physical health. Young people with physical disabilities, chronic illness, or mental illness face difficulties in accomplishing the tasks of the transition to adulthood, such as achieving financial and residential independence. Socially disadvantaged young people are especially at risk of facing difficulties in the transition to adulthood, because, in addition to other difficulties associated with low socioeconomic status (SES), they also have more health problems. (Osgood et al. 2005a; 2005b). In recent decades, obesity has increased in the adolescent population (Ogden et al. 2002; 2006), serving as another barrier to successful transitions to adulthood. Obese adolescents complete fewer years of education, are less likely to marry, and have a lower household income as adults, independent of their family's socioeconomic status (Gortmaker et al. 1993; Sobal and Stunkard 1989). Obesity is closely related to physical activity (Kumanyika et al. 2008). Better understanding the social influences on physical activity may serve to explain the role of health in the transition to adulthood, especially for disadvantaged populations.

Physical activity is a critical part of the energy balance equation. Low levels of physical activity contribute to overweight and obesity and the risk for cardiovascular disease, Type II diabetes, as well as a number of other chronic conditions (US DHHS 1996). Regular physical

activity declines dramatically during adolescence, especially among adolescent girls (Kimm et al. 2002; Nader 2008). 38% of adolescent girls and 24% of adolescent boys do not meet national recommendations for moderate to vigorous physical activity (US DHHS 1996; 2000). These low levels of physical activity among many adolescents have become a public health concern, especially as it relates to overweight and obesity (Hallal et al. 2006; Ogden, Carroll and Flegal 2003). Overweight and obesity among youth are increasing at an alarming rate in the United States, and have a direct link to youth population levels of physical activity (Kumanyika et al. 2008). There is also evidence that physical activity trajectories track¹ significantly from adolescence to young adulthood, having implications for health in adulthood (Raitakari et al. 1994, Telama et al. 2005). Therefore, understanding the determinants of physical activity in adolescence may be of critical importance in the prevention of obesity and its related chronic conditions in later in life.

Heightening public health concern is the disproportionate levels of overweight and obesity and lower levels of physical activity among racial/ethnic minorities and individuals of low socioeconomic status (SES) (Harris, King and Gordon-Larsen 2005; Harris et al. 2006; Gordon-Larsen, McMurray and Popkin 2000; Kimm et al. 2002; Van Der Horst et al. 2007). It is particularly important to understand the influences of adolescent physical activity for racial/ethnic minorities and individuals of low socioeconomic status to better understand and reduce racial/ethnic and socioeconomic disparities in physical activity and obesity in adolescence and adulthood.

Family context plays a pivotal role in influencing developmental trajectories and health behaviors and outcomes in adolescence and in the transition to young adulthood (Baumrind

¹ Tracking means to maintain a person's rank order over time. The tracking of physical activity trajectories means that one's relative level of physical activity tends to last (or track) over time (Malina 1996).

1991; Bronte-Tinkew 2005; Collins et al. 2000; Harris, Furstenberg and Marmer 1998; Harris and Marmer 1996; NRC 2002; Riesch, Anderson and Krueger 2006; Steinberg 2001). Parenting practices and behaviors are theoretically important predictors of child outcomes and research has consistently shown that parents serve an important role in influencing child and adolescent physical activity and other health behaviors related to obesity in childhood, adolescence and the transition to adulthood (Adkins et al. 2004; Birch and Fisher 1998; Crossman, Sullivan and Benin 2006; Davison and Birch 2001; Davison, Francis and Birch 2005; Hart, Bishop and Truby 2003; Mattocks 2007; Moore et al. 1991; Rhee et al. 2006; Robinson et al. 2001; Sallis, Prochaska and Taylor 2000; Van Der Horst et al. 2007).

Parenting behavior may serve as a particularly important determinant of adolescent physical activity to study in poor families, because there is a well-established negative link between socioeconomic disadvantage and physical activity outcomes for adolescents and adults (Brownson, Tegan and Luke 2005; Gordon-Larsen, Nelson and Popkin 2004; Gordon-Larsen et al. 2000), and because disadvantage can operate via parenting styles and practices (Aber et al. 1997; Brooks-Gunn and Duncan 1997; Conger and Donnellan 2007; Crosnoe, Mistry and Elder 2002; Elder et al. 1995; Grant et al. 2003; Harris and Guo 2000; Lareau 2002; 2003; McLeod and Shanahan 1993; McLoyd 1998; Smetana, Campione-Barr and Metzger 2006; Mistry et al. 2002). Parenting may be an important pathway through which SES influences physical activity in adolescence and the transition to adulthood. Family disadvantage affects parenting behaviors in multiple ways. Disadvantaged parents may not have resources, such as adequate health information, time to engage with their children in physical activity or monitor their children's inactivity, or money for children to be able to engage in organized activities. This lack of resources, in addition to economic insecurity, may lead to parenting practices that discourage

physical activity and encourage inactivity.

Although a growing amount of empirical research supports the idea that parenting practices mediate the relationship between poverty and various child outcomes (for reviews see Aber et al. 1997; Brooks-Gunn and Duncan 1997; Conger and Donnellan 2007; Hoff, Laursen and Tardiff 2002; McLoyd 1998), these relationships have not been investigated for physical activity outcomes. In addition, research has not investigated the longer term effects of parenting in adolescence on physical activity in young adulthood. Parental influences on adolescent health behavior are crucial, because they set up health trajectories that determine health patterns in later adulthood. Low levels of physical activity in adolescence track into young adulthood. Better understanding how disadvantage influences parenting in ways that affect both adolescent physical activity and subsequent young adult physical activity will provide important information on how health disparities are created and track over time.

Using nationally representative data from the National Longitudinal Study of Adolescent Health (Add Health), this chapter will investigate the mediating role of parenting practices in the relationship between social disadvantage and physical activity patterns in the transition from adolescence into young adulthood.

The specific aims of this chapter are to:

- (1) Create measures of parenting styles and behaviors.
- (2) Determine how parenting styles/behaviors differ by race/ethnicity, SES and sex.
- (3) Investigate the relationships between different parenting practices and styles and physical activity patterns.
- (4) Within a longitudinal framework, determine if parenting style/practices play a role in mediating the relationship between SES and physical activity patterns from adolescence and into

young adulthood.

This analysis will determine whether and which parenting practices play a role in mediating the relationship between poverty in adolescence on physical activity patterns over time. This research will measure multiple dimensions of parenting practices and behaviors that represent various pathways through which parenting may operate to mediate the relationship between poverty and physical activity outcomes in adolescence and the transition to young adulthood. As a result, this paper will be able to decipher what elements of parenting practices are most salient in linking disadvantage in adolescence to physical activity patterns in the transition from adolescence to young adulthood.

Previous Research on Parenting and Physical Activity

Parents play a central role in influencing adolescents' physical activity (Lindsay et al. 2006; Sallis et al. 2000). Their behaviors, attitudes, and beliefs substantially affect many of their children's health behaviors, including physical activity (Gustafson and Rhodes 2006; Lindsay et al. 2006; Pugliese and Tinsley 2007; Sallis et al. 2006). Parents help to determine their children's access and opportunities for physical activity, they support and promote (or discourage) physical activity, and they serve as role models for physical activity (Davison et al. 2005). Parents also help to create intimate and cohesive family contexts that promote physical activity (Kuo et al. 2007; Ornelas, Perreira and Ayala 2007).

Previous research indicates that children and adolescents engage in higher amounts of physical activity when supported by their parents, via the encouragement parents provide their children to be physically active, the time parents spend with their children engaging in physical activity, the provision of transportation to physical activities by parents, and the time parents

spend watching their children engage in physical activities and monitoring other activities related to physical activity, such as TV viewing (Adkins et al. 2004; Bauer et al. 2008; Davison, Cutting and Birch 2003; Gustafson and Rhodes 2006; Heitzler et al. 2006; King, Tergerson and Wilson 2008; McGuire et al. 2002; Neumark-Sztainer et al. 2003; Norman et al. 2005; Prochaska, Rodgers and Sallis 2002; Pugliese and Tinsley 2007; Sallis et al. 1992; 1999; 2000; Trost et al. 2003; Van Der Horst et al. 2007).

As socializing agents, parents serve as role models that influence the physical activity behaviors of their children (Anderssen, Wold and Torsheim 2006). Exposure to physically active role models provides support for remaining active and helps to establish positive social norms for exercise (Garcia et al. 1995). However, evidence for this relationship is complex and sometimes inconsistent (Sallis and Owen 1999; Trost et al. 2003). For example, although many (but not all) studies report a positive correlation between parental and child physical activity levels, when other forms of parental influence are considered along with parental physical activity, the importance of parental role modeling is reduced. And, other measures of parental behavior, such as parental encouragement, emerge as more important predictors of child physical activity (Brustad 1996; Dempsey, Kimiecik and Horn 1993). In addition to these measures, research has also found that family intimacy and cohesion, such as parental communication, monitoring and warmth and engagement in social and recreational activities are also related to physical activity (Kuo et al. 2007; Ornelas et al. 2007). Parental communication and monitoring serve to channel children into health promoting activities, such as physical activity. Parental warmth and engagement increase levels of perceived confidence in their children, which is a psychosocial correlate of activity behavior (Kimiecik, Horn and Shurin 1996).

In summary, research indicates many possible pathways through which parenting can

promote physical activity. In addition to role modeling and active support of physical activity, parental practices and behaviors that facilitate parent-child interaction and cohesion also have an important influence on physical activity. Racial/Ethnic minority and disadvantaged parents may face unique social circumstances that affect parenting practices and behaviors, which may have important implications on adolescent physical activity outcomes. These issues are addressed below.

The Influence of Disadvantage and Race/Ethnicity on Parenting Practices

Parent-child interactions are an important aspect of a child's social environment and have profound direct and indirect influences on children's development and health behaviors well into adolescence (Baumrind 1991; Birch and Fisher 1998; Collins et al. 2000; Elder 1999; Harris et al. 1998; Harris and Marmer 1996; NRC 2004). Parenting styles and practices differ by race/ethnicity (García Coll and Pachter 2002; Steinberg et al. 1992; Wu et al. 2003) and SES (Burton and Jarrett 2000; Elder et al. 1995; Hoff et al. 2002; Lareau 2002; Pinderhughes et al. 2001). The effectiveness of parenting styles and behaviors on child outcomes may also differ across cultural and socioeconomic groups (Baumrind 1972; García Coll and Pachter 2002; Darling and Steinberg 1993).

There are a number of reasons why parenting practices are influenced by socioeconomic status and race/ethnicity. Poor adults encounter a larger number of chronic conditions and negative life events than nonpoor adults. Work and income instability, inability to pay bills, food insecurity, poor housing, and lack of other basic needs characterize everyday life for many poor families (McLoyd 1998). Ethnic minority parents, especially African American parents, are disproportionately impoverished because they are overrepresented among the low paid and

unemployed and in single-parent homes (Iceland 2003). In addition, poor blacks enter poverty with fewer economic resources (McLoyd 1990) and are more likely than poor whites to live in isolated urban ghettos (Wilson 1987), which increases the severity of economic deprivation. These disadvantaged environments lead to stressful exposures that have serious implications for parenting practices.

The Family Process Model

The family process model (Conger et al. 2002; Conger and Conger 2002; Conger and Donnellan 2007; Elder 1999; McLoyd 1990, 1998) provides an important framework to understand the role disadvantage plays in influencing how parents interact with their children. The family process model is a framework originating from the child and adolescent development and social psychology literature, which argues that some economic disadvantage is filtered through family dynamics. More specifically, the model provides for a specific pathway through which economic hardships of daily life (e.g. low income and unstable work) result in felt economic pressures (e.g. unmet material needs; inability to pay bills), which lead to emotional distress and lack of confidence and self-efficacy, adversely affecting parenting. Because parents become consumed by their economic problems, they become less involved in their children's daily activities, they demonstrate less affection toward their children, and are more harsh, irritable, and inconsistent in their disciplinary practices (Conger and Donnellan 2007). Previous research provides evidence that negative life events and poverty heighten inconsistent, punitive and harsh parenting and less parental investment and affection (Brooks-Gunn and Duncan 1997; Grant et al. 2003; Elder et al. 1995; McLoyd 1998; Pinderhughes et al. 2000) and that this relationship is similar in both white and black families (Conger et al. 2002). However,

associations between socioeconomic status and poor monitoring and supervision of children have been less consistent (Hoff et al. 2002).

Ethnic and Minority Parenting

Ethnic minority parenting has been an understudied area in the parenting literature. However, there are some unique conditions under which ethnic minority parents must operate. As discussed above, ethnic minority parents are not only disproportionately poor and segregated, but their everyday lives are also influenced by their cultural orientations and by racism. Because of this, it is difficult to disentangle what aspects of minority parenting are influenced by social class (many ethnic minority families are poor), cultural orientation (parents may have different childrearing customs given their different cultural heritage from the majority), or minority status (the impact of being in the non-majority group due to racism and other factors) (García Coll and Pachter 2002; McLoyd 1990; 1998).

Despite these complexities, cultural traditions unique to each racial/ethnic group do influence parenting practices, beliefs and attitudes. For example, African Americans and Hispanics have different conceptions of a healthy body weight, and may not encourage their children who are overweight by medical standards to become more physically active because they are not overweight by their cultural standards (Harris and Koehler 1993; Kumanyika and Grier 2006). In addition, cultural attitudes that predispose children to sedentary leisure time activities, such as television viewing, may also discourage physical activity (Airhihenbuwa 1995; Beech et al. 2004). These more nuanced aspects of minority parenting are difficult to capture in large-scale surveys but have important implications for child outcomes (García Coll and Patcher 2002). Minority parenting, like all parenting, must be studied in the context in which it occurs

(García Coll and Patcher 2002; Kaufman and Karpati 2007). For the purposes of this paper, the context of most relevance is social disadvantage.

It is important to note that not all aspects of family life and parenting are influenced by socioeconomic status (García Coll and Patcher 2002; Lareau 2002). All parents share universal goals of parenting based on their need to enable their children to successfully transition from complete dependency in childhood to relative self-sufficiency as an adult. These goals include providing an environment that enables successful development, ensuring their children's physical safety and modeling and teaching normative social values (LeVine 1977). How these goals are accomplished (or fail to be accomplished) are influenced by economic resources and cultural orientation, as well as other factors.

This discussion of socioeconomic disadvantage, race/ethnicity, and parenting practices highlights the need for research to determine how economic disadvantage is related to physical activity through the influence of parenting practices, while controlling for the effects of race/ethnicity.

The Mediating Role of Parental Practices

Parents can influence their children's physical activity in several ways. They can encourage and support their children's physical activity through parent-child communication and emotional support in ways that encourage competence. They can play an instrumental role to facilitate physical activity such as providing transportation and access to organized sports. Parents can also serve as healthy physically active role models by engaging in physical activity with their children, such as walking or playing a sport and involvement in physical activities such as coaching and watching their children at sports events. In addition, parents can also

monitor their children's activity and inactivity (Anderson and Butcher 2006; Davison and Birch 2001; Gustafson and Rhodes 2006; Sallis et al. 2000; Lindsay et al. 2006; Pugliese and Tinsely 2007; Van Der Horst et al. 2007). Socioeconomic status is related to all of these aspects of parenting with implications for the health and well-being of children.

The Family Process Model

As discussed earlier, the family process model has been used to link negative parenting practices due to economic disadvantage to adverse child outcomes in education, mental and physical health. It has also been used to examine whether behaviors and attitudes of parents link disadvantage in adolescence to outcomes in young adulthood (e.g., Crosnoe et al. 2002). This research has found that the negative parenting practices of poor parents sometimes have detrimental effects on children's development, behavior, educational success and mental and physical health in childhood and adulthood.

Although family process models have been used extensively in social psychology and developmental research, they have not been used to examine health behaviors related to obesity such as physical activity. However, certain parenting practices due to lack of economic resources and stress may affect physical activity outcomes for adolescents that endure into young adulthood. If disadvantaged parents' ability to control and monitor their children is reduced, they might not be able to ensure that their children are engaging in physical activity, rather than watching TV or partaking in other sedentary behaviors, which are unhealthy habits that can carry into adulthood (Gordon-Larsen et al. 2004; Pate et al. 1999; Thompson, Humbert and Mirwald 2003). Low parent-child communication due to disadvantage may diminish parents' ability to facilitate children's physical activity and feelings of competence and self-efficacy. For example,

through communication a parent can encourage his/her child to try out for the team or ask to play a game of soccer with the neighborhood kids. A child of a parent who does not communicate or share closeness with his/her child might never build the confidence to engage in such activities.

Socialization Theory

Socialization theory provides another framework for the ways in which parenting processes are linked to physical activity outcomes. The socialization perspective views the family as the central source of socialization in the lives of children (Maccoby 1992). Parents serve as socializing agents and role models and their presence, parenting skills and relations with their children have an important influence on child outcomes (McLanahan, Astone and Marks 1991). In early childhood, teaching children socially-appropriate values and behavior is the main task of socialization. In later childhood as children move into adolescence, children are provided with more autonomy and are allowed to play a larger role in decision-making. Parents help their teens to engage in proper decisions and behaviors by using parenting elements such as monitoring, supervision and control. In addition socialization works via communicating and reasoning with adolescents as well as setting rules. The transmission of values from parents to children is heavily influenced by stable, nurturing and accepting parent-child relations (McLanahan et al. 1991), especially in adolescence when peer influences become stronger.

Previous research has supported the contention that authoritative parenting styles, characterized as a combination of attentiveness and affection with clear rules for correct behavior, are the most useful in supporting positive adolescent development, especially in regards to emotional adjustment (Baumrind 1972, Smetana et al. 2006). Similar to research using the family process model, research based on socialization theory finds that parenting styles differ by socioeconomic status and race/ethnicity. Poor parents and ethnic minority parents are

more likely to use an authoritarian parenting style, characterized by high levels of supervision, strictness and control and low levels of affection, than non-poor and white parents (Grant et al. 2003; Spencer 1990; Vega 1990). Other researchers argue that these typologies fail to take into account how the effectiveness of parenting styles might differ by socioeconomic status and race/ethnicity, especially when they are based on samples of white middle class families (Bradley 1998; Weiss 2002). In fact, some researchers argue that highly restrictive authoritarian parenting styles may be a necessary parenting strategy in poor, unsafe high-risk neighborhoods where strict monitoring is necessary to protect children from an adverse social environment (Elder et al. 1995; Furstenberg et al. 1999).

It should be noted that socialization theory and the family process model differ in how parenting processes are conceptualized to mediate the relationship between disadvantage and adolescent outcomes. The family process model contends that economic strains and stress due to economic hardship inevitably lead to diminished parenting skills and ineffective parenting practices that negatively affect children. Socialization theory argues that poor parents deliberately engage in certain practices that they feel will help to protect their children from and overcome the risks in their environment and hopefully lead to positive development. In regards to physical activity outcomes, highly restrictive parenting, even as a strategy to protect children from neighborhood risk, may be more detrimental to adolescents if it prevents or reduces their ability to engage in physical activity.

In addition, because socialization theory identifies parents as role models that expose and teach their children norms of behaviors, parents who are inactive and/or obese may serve to reduce or impede physical activity in their children. Role models shape the perceptions of adolescents and young adults regarding current and future lifestyles (Maccoby 1992). As

models, they support and reinforce the acquisition and maintenance of exercise behaviors or lack thereof (Lindsay et al. 2006). If a child lives with parents who fail to exercise and engage in sedentary behavior, youth are likely to mimic the lifestyles they see and retain these habits into adulthood (Fogelholm et al. 1999; McGuire et al. 2002). Because poverty is associated with an increased risk of obesity in all age groups, poor parents are also more likely to be obese and inactive, and, therefore, model unhealthy behaviors.

Social Capital

Another avenue of socialization important to parent-child relations is represented by the theoretical concept of social capital. Coleman's (1988) concept of social capital captures the resources embedded in the context of social relationships and ties that facilitate action. An important context of social relations, with strong implications for adolescent outcomes, is the family context. Social capital in the family is dependent upon parent-child relations. The quality and quantity of relations between adolescents and their parent facilitate healthy behaviors and can be seen as a social capital resource. A parent's time, attention and affection are important dimensions of parent-child relationships that will benefit children (Bourdieu 1985). Parents would be ineffective in encouraging positive healthy behaviors such as physical activity if they did not share close emotional bonds with their children. Opportunities to engage in physical activities as a family or discuss the importance of staying active would not occur without shared activities and time together. Within the family context, these social capital resources facilitate healthy behavior in children by serving as a source of family support and social control. As discussed earlier, poverty diminishes a parent's ability to spend time with children, share closeness or communicate.

Summary

The family process model and theories of parenting styles, role-modeling and social capital all highlight the important ways that that parents' behavior and practices can mediate the relationship between disadvantage and physical activity outcomes. Poverty not only limits the material resources parents can provide their children to engage in healthy behavior but it affects parents' ability to communicate, discipline and monitor their children, advocate for them and model healthy lifestyles. Together these theoretical frameworks will serve to guide the conceptualization of how the behaviors and practices of parents link disadvantage in adolescence to physical activity outcomes in adolescence and in the transition into young adulthood.

Conceptual Model

The conceptual model (see Figure 4.1) provides the longitudinal relationship between poverty status and change in physical activity from adolescence into young adulthood. In this conceptual model, parenting practices and styles during adolescence mediate the relationship between disadvantage and change in physical activity from adolescence into young adulthood. Adolescents either increase or decrease their levels of physical activity or maintain medium/high or low levels of physical activity into adulthood. Poverty is conceptualized as having enduring effects on physical activity through parenting practices. Data from all three waves in Add Health are utilized, where poverty status is measured in childhood and adolescence, parenting practices and behaviors are measured in adolescence (Wave I), and physical activity trajectories are constructed from data in adolescence (Wave II) and young adulthood (Wave III). All controls are measured at Wave I, the first data point in adolescence.

Data

Data come from the National Longitudinal Study of Adolescent Health (Add Health), which is an ongoing nationally representative, school-based study of adolescents in grades 7 to 12 that began in 1994. It was designed to explore the causes of health-related behaviors, with an emphasis on the influence of social context. In 1994 Add Health administered an In-School Questionnaire to every student attending school from a nationally representative sample of schools. A sample of 80 high schools and 52 middle schools from the U.S. was selected using a stratified cluster design. A subsample of individuals in these schools participated in the In-Home Interview in 1995 (Wave I), given an average of eight months after the In-School Survey, and again in 1996 (Wave II). In Wave III (2001-02), Wave I respondents were re-interviewed.

A parent, generally the mother, was also interviewed in Wave I. In-home adolescent questionnaires were administered by computer-assisted personal-interview (CAPI), as well as computer-assisted self-interview (CASI) for more sensitive questions. Ultimately, 20,745 in-home interviews were completed in Wave I; 17,713 parents answered child specific questions and 17,669 answered parent specific questions (more than one child was interviewed in some households). 14,738 in-home interviews were completed in Wave II (the seniors in Wave I were not followed). In Wave III 15,197 eligible original Wave I respondents completed the survey. In Wave I (WI), the age of participants ranged from 12 to 19 years, in Wave II (WII) from 13 to 20 years and Wave III (WIII) from 18 to 26 years.

Over 70% of the schools originally selected for the survey participated. Of the adolescents sub-sampled for the in-home questionnaires, 78.9% participated in WI. Parent interviews are available for 85% of these respondents. Of those eligible for participation in WII, 88.2% completed in-home interviews. Of those eligible for participation in WII, 77.4%

completed in-home interviews.

The fact that the data set is longitudinal and nationally representative, with extensive measures of socioeconomic status, health, family structure, composition and social interaction, race and ethnicity among other factors, makes it an ideal data set to investigate the relationship between disadvantage and physical activity in adolescence. Harris and colleagues (2003) provide a more detailed description on the Add Health Study.

This study uses data from the Wave I In -Home and Parent Questionnaires as well as the follow-up Wave II and III surveys. This analysis is therefore limited to adolescents who participated in all three waves of the study and have completed Parent Questionnaires. Exclusions included seriously disabled respondents and pregnant females. After applying these data constraints and deleting the cases with missing data on covariates, the final study sample contains 8,109 (4,195 females and 3,914 males) respondents.²

Measures

Table 4.1 provides means and standard errors of all measures used in this analysis for the total sample and by sex.

Control Measures

Race/Ethnicity: Add Health allows for rich detail in measures of race and ethnicity.

Race/ethnicity is self-reported at Wave I and is classified into five race and ethnic groups: non-Hispanic white (reference group), non-Hispanic black, Hispanic, Asian, or other racial/ethnic group. I control for race/ethnicity because of its possible confounding effects with

² Seniors at Wave I were not followed up in Wave II as part of the design of Add Health and thus the analysis sample is younger than at Wave I. Missing from the analysis sample are respondents who did not have a completed parent questionnaire (therefore, there are fewer immigrants in the sample).

socioeconomic status.

Sex: This measure is constructed from responses in the Wave I In-Home Questionnaire. This measure was crosschecked with WII and WIII responses. Research consistently shows that females exhibit lower levels of physical activity than males and that parental support differs by sex (Gustafson and Rhodes 2006).

Age: Age is a continuous measure of self-reported age at WI. Age ranges from 11 to 21 years of age. Levels of physical activity decrease with age (Davison and Birch 2001; Harris et al. 2006; Sallis et al. 2000).

Maternal Work Status: Maternal work status is measured by whether the mother worked full time using both WI respondent and parent reports. Respondents were asked to report if their mother worked for pay and approximately how many hours a week. Mothers who worked 35 hours or more per work were coded as working full time. For those adolescent reports that were missing on mother's work status, the parent reports of work status from the parent questionnaire was used. The parent and adolescent reports of mother's work status was also cross-checked for validity for cases with data from both interviews (giving preference to the parent report if inconsistent). Consistent with Census data, 58% of the adolescents at Wave I had a mother who worked full-time.

Maternal work status serves as a proxy for presence in the home. Mothers who work, especially if they work full-time, are less able to monitor their children due to a decrease in maternal presence and availability during the day (Jacobson and Crockett 2000). Because it is mainly the mother who supervises children's activities during the day, since a majority of fathers (in two parent households) work full time, monitoring is more affected by the mother's work status (Seltzer 1994). Poor working mothers, in particular, are often in difficult and unstable

work situations that often increase stress and decrease maternal availability, which reduce parental monitoring and involvement, weakens mother-youth communication and thus increases youth risk (Morris et al. 2001). Poor working mothers may not be able to take an active role in the decision making for their child's choice of physical activities because of time constraints due to workload and/or the stress associated with being in poverty.

Family Size: Family size is measured by using the respondent's reported count of number of siblings from Wave I Household Roster (thus, only siblings present in the household at Wave I). Family size can influence the ability of parents to monitor their children and the time they can devote to each of their children's activities. The mean number of siblings at WI was 1.58.

Extended Family: Extended family is a categorical measure indicating if the respondent was living with an extended family member at WI. It is measured by using the respondent reports of living with a grandparent, great grandparent, aunt, uncle, cousin or other relative from the WI Household Roster. The presence of extended family may influence the ability of parents to monitor their children and the time they devote to each of their children's activities. Extended family either help in parenting the children in the household or serve as an additional caretaking responsibility to the parent. 10% of the sample lived with extended family at WI.

Parent Obese: Using self-reports from the Parent In-Home Questionnaire, a respondent was coded as having an obese parent if either their biological mother and/or biological father were reported as being obese. Parental obesity is used as a control to account for the genetic predisposition for engaging in low levels of physical activity under the assumption that obesity is associated with less physical activity (Lauderdale et al. 1997). In addition, it serves as a proxy for parental role-modeling of low levels of physical activity, or some combination of genetic propensity and environmental risk. 23% of the sample has an obese parent.

Socioeconomic Disadvantage

Socioeconomic disadvantage is measured in two ways using welfare/poverty status and parent education. Family structure is used as a control. Although welfare/poverty status provides a more direct measure of deprivation than education, education is a socioeconomic indicator that is particularly likely to capture aspects of behavior and lifestyle (Shavers 2007). People with higher education levels have a greater awareness of health issues and are better able to make healthy choices and facilitate opportunities for physical activity. Parental education may also affect parental monitoring, with more educated parents doing a better job at monitoring their children (Lareau 2002), especially behaviors that directly relate to physical activity. This is especially relevant during adolescence when future lifestyle and health-related behaviors are often established.

Welfare/Poverty Status: Welfare/Poverty status is a dichotomous indicator of any welfare receipt before the age of eighteen or family income less than poverty level. This measure is constructed from data on the family's receipt of public assistance or welfare from WI and WII during adolescence in combination with a retrospective report at WIII on the receipt of welfare and public assistance prior to the age of eighteen. Using data from the WI Parent Questionnaire on reported annual income from 1994, family income is categorized as below poverty level if income was less than \$16,000 (roughly the poverty level for a family of four in 1994). I chose a welfare- and income-based measure of poverty over an only income-based measure due to the large proportion of missing data on income ($\approx 20\%$). About 28% percent of the total sample received welfare prior to the age of eighteen and/or was living below poverty at WI.

Parental Education High School Diploma (or Equivalent) or Less: Using data from the Wave I Parent Questionnaire, parents' education is measured as the higher of either mother's or

father's education. A respondent is considered disadvantaged if their highest educated parent has high school degree or equivalent (i.e., GED) or less. I substituted missing parental reports of education with adolescent reports of their parents' education. About 62% of the sample has a parent with a high school degree or less.³

Family Structure: There is rich detail on family of origin living arrangements. Adolescents are classified as those who live with two biological or adoptive parents (reference category), a stepfamily (biological mother and step father or a biological father and step mother), single mother, single father, and surrogate or foster parents (including grandparents, aunts and uncles, other adult relatives, or nonrelative adults). I control for family structure because of its possible confounding effects with socioeconomic status. In addition, the presence of only one parent in a household reflects a lower ability to provide monitoring of children's activities and less time and parental presence to serve as role-models and socialize children in a way that can form valuable parent-child relations (Amato 1993; McLanahan 1997).

Parenting Practices

Two dimensions of parenting practices will be measured for this analysis: parental control (supervision and monitoring) and parent-child relations (parental-child activity sharing, shared decision making, share meals, parental-child communication, and parental closeness and warmth). Each dimension is represented by multiple measures. These measures draw from the previous work of Harris and Ryan (1999) and Ryan (2001) and reflect socialization and social capital theories of parenting, as well as instrumental measures of parenting that are influenced by

Although a majority of the sample has a parent with a high school degree or less (62%), this subgroup is still a population at risk of lower levels of physical activity, compared to individuals who have attained more than a high school degree. Sensitivity analysis was performed using a measure of highest parent education as less than high school, similar results were found and measures using high school or less were stronger and more consistent.

poverty. I will measure multiple dimensions of parenting because separate domains have important independent effects in influencing adolescent development and well being (Harris and Ryan 1999). All measures come from respondent reports at WI.

Parental Control (Supervision and Monitoring): Parental control has two components: monitoring and supervision. Parental monitoring is measured using a validated additive scale (Harris and Ryan 1999). The scale (ranging from 0 to 7) is based upon seven items that ask if a respondent is permitted to make his/her own decisions about amount of TV watched, television shows viewed, what to eat, weeknight bedtime, weekend curfew, friends and clothes. The index is reverse coded such that high measures indicate high control by the parent. The supervision measure represents the amount of parental presence in the home. Parental supervision is measured using an additive index (ranging from 0 to 3) that is based upon three questions that ask the respondent if a parent is present most or all of the time when he/she leaves for school in the morning, returns from school in the afternoon, and goes to sleep at night.

Parent-Child Relations (Shared Activities, Shared Decision-Making, Shared Meals, Parent-Child Communication and Closeness and Warmth): Parent-child relations are represented by five separate measures: shared activities, shared decision-making, shared meals, communication, and closeness and warmth. Shared activities is a count (ranging from 0 to 5) of the activities a parent and child did together in the previous four weeks including: attended sports events, gone shopping, attended religious or church events, gone to the movies and worked on a school project together. Shared decision-making refers to a Likert scale measure of the extent to which the parent and child make decisions together regarding their child's life, ranging from 1, "always" to 5, "never." The scale is reverse coded such that high measures indicate a higher frequency of shared decision-making. This is the only parent-reported measure used in this

analysis. Shared dinners represents the frequency with which the adolescent and parent eat dinner together during the week (ranging from 0 to 7). Parent-child communication is created by summing respondent reports of engaging in the following discussions with parents within the past 4 weeks: talking about school work or grades and talking about other things the respondent doing at school (ranging from 0 to 2). The closeness indicator (ranging from 1 to 5) is a summary measure based upon the adolescent's mean response to four items: level of closeness, satisfaction with relationship, if parent is warm and loving, and satisfaction with communication. These questions were asked separately for resident mothers and resident fathers. For respondents living in a two-parent- household the average of responses for both parents is used. The available response is used when the teen only lives with one parent. Questions about shared activities and parent-child communication were also asked for nonresident mothers and fathers. For individuals who reported activities with their nonresident parents these responses were averaged with the responses for resident parents.

Because some of the items used to construct these parenting measures have not been directly linked to physical activity outcomes in the literature, bivariate relationships between these items and physical activity will initially be explored.

Physical Activity Trajectories

Physical activity is measured by a standard physical activity behavior recall in Add Health that is similar, although not identical, to other self-report questionnaires that have been used and validated in other large-scale epidemiological studies (Andersen et al. 1998; Sallis et al. 1993). In WII and WIII, a series of questions ask about participation in moderate to vigorous physical activity (MVPA), including skating and cycling, exercise and active sports (5-8 metabolic equivalents or METs), in units of times per week. One MET represents the energy

expenditure associated with quiet sitting. Each question represented a group of similar/related MVPA activities. Respondents indicated the number of times in which they engaged in moderate to vigorous physical activity for each group of activities according to the following four categories: 0; 1-2; 3-4; and 5+ times per week. After converting the categorical frequency response into a metric frequency, the number of times that the respondent engaged in moderate to vigorous physical activity is then summed across the three groups of activities listed at WII.⁴

The Wave III questionnaires added questions applicable to young adults (e.g., weightlifting, walking for exercise) in addition to the activities from the WII questionnaire, resulting in seven groups of activities.⁵ A standardized sum of MVPA was created to avoid reporting an artificial increase in reported activities levels in young adults resulting from the addition of these activities. The total sum of MVPA reported in WIII was standardized to be equivalent to that of WII by dividing by the total number of groups of activities at WIII [7] and then multiplying by the total number of groups of activities in WII [3], similar to the approach used by Gordon-Larsen and colleagues (1999, 2004).

Overall activity frequency was summed to determine total weekly MVPA at each wave. These sums were used to determine whether individuals met national recommendations for physical activity (Ainsworth et al. 1993) at each wave (five or more weekly bouts of MVPA). At WII, when respondents were adolescents, 40% of females and 25% of males did not meet national recommendations for physical activity. At WIII, when adolescents were young adults, these numbers increased to 89% and 79% respectively. The dependent variable contains four

⁴ In order to sum the frequencies of engaging in the 3 different groups of activities which are reported as categories of frequency, I assigned the midpoint of the range of frequency for each response category as follows: the response category 1-2 was assigned 1.5; response category 3-4 was assigned 3.5; and response category 5 or more was assigned 6.

⁵ At WIII response options were true frequency counts of activities ranging from 0 to 6, and 7 or more. For the response category 7 or more, I assigned the value of 7.

trajectories that capture change and continuity in meeting national recommendations for physical activity from WII to WIII: *Become Physically Active* (WII < 5 weekly bouts of MVPA; WIII ≥ 5 weekly bouts of MVPA) *Stay Physically Active* (WIII ≥ 5 weekly bouts of MVPA); *Become Not Physically Active* (WII ≥ 5 weekly bouts of MVPA; WIII < 5 weekly bouts of MVPA); *Stay Not Physically Active* (WII < 5 weekly bouts of MVPA; WIII < 5 weekly bouts of MVPA). The percentage of individuals who become physically active is very small (3%). Over 50% of males and females become **not active** in young adulthood.

Analytical Approach

All analyses will use STATA survey procedures with sampling weights to adjust for the clustered sample design and unequal probability of selection to ensure the results are nationally representative. Analysis will be guided by the four research aims of this chapter. To explore variation in parenting practices, I will calculate mean levels of parental control and parent child relations by race/ethnicity, sex and SES subgroups.

Bivariate logit models will then be used to determine the relationship between parenting styles/practices and the risk of not being physically active in adolescence and young adulthood (i.e., WII and WIII activity). Exploratory analysis continues by using bivariate multinomial logistic regression models to test the relationship between parenting practices and the risk of becoming or staying not active from adolescence into young adulthood.

Multinomial logistic regression is then employed to examine the relationships displayed in the conceptual model (Figure 4.1). Multinomial regression is appropriate when the dependent variable is an unordered nominal variable with n categories. The procedure estimates the log of the ratio of the probability of being in the nth category relative to a base category (stay active),

where the effects of independent variables are measured by the relative risk or odds (Long 1997). I focus on individuals who stay or become not active, because these groups are most at risk for becoming obese as well as developing other adverse health outcomes⁶.

The fundamental model takes the form:

$$\log\left[\frac{P_{ij}}{P_{iJ}}\right] = \beta_j x_i$$

The model is generalized to J categories, with the running index $j=1 \dots, J$. Where P_{ij} is the probability that individual i falls into category j and where x_i is a column vector of variables describing individual i and β_j is a row vector of coefficients for category j . Note that each category is compared with the highest category J .

Multivariate analysis begins with a baseline model of the relationship between family poverty (welfare/poverty status) and physical activity trajectories. Model 2 enters parent education. Model 3 enters race/ethnicity to establish the relationship between family disadvantage and obesity independent of race/ethnicity. Model 4 enters parenting measures to determine if parenting practices mediate the impact of poverty. Before analyzing a model with all the parenting variables included, I will enter them individually in order to examine their separate effects on the poverty coefficient. This approach will be used to more accurately determine which parenting elements act as mediators of poverty (Baron and Kenny 1986; Judd and Kenny 1981). Model 5 enters control measures. To the extent that parenting practices mediate the effects of poverty, we add to our understanding of the ways in which social disadvantage operates via parenting behaviors to influence physical activity trajectories from adolescence into young adulthood. In a 6th model, I estimated interaction effects between parenting variables that remain significant in Model 5 and sex. However, no interactions were

⁶ “Become active” is included as a category in the analysis, but results are not shown for this outcome.

significant and the results are not shown (but are available upon request).

Results

Parenting Differences

Tables 4.2 to 4.4 provide means of the parenting measures by race/ethnicity (Table 4.2), sex (Table 4.3), welfare/poverty status and parental education (Table 4.4). Below, I will focus most of the discussion of results on the differences in means that were found to be statistically significant.

Parenting Differences by Race/Ethnicity

Mean levels of parental monitoring did not differ greatly by race/ethnicity (See Table 4.2).⁷ Indeed, the only statistically significant differences were between whites and Asians, and white and Hispanics. Both Hispanics (2.28) and Asians (2.23) had a higher mean level of monitoring compared to whites (1.97). Blacks (2.32) experienced lower levels of parental supervision compared to whites (2.42) and Hispanics (2.47).

Mean level of activities in which a respondent and parent engaged was higher for whites (1.56) compared to blacks (1.43) and Hispanics (1.38). There were no statistically significant differences in shared decision making by race. African Americans (4.00) had the lowest mean level of shared dinner meals with parents compared to all other racial/ethnic groups. Whites had the highest mean level of shares meals (5.33). Mean levels of communication with parents about school were highest for Asians (1.25) compared to all other racial/ethnic groups. There was no variation in closeness by race.

⁷ Although the “other” race category has a higher mean, it is difficult to interpret due to the small sample size and it is not statistically significant from any of the other races.

In summary, there was not substantial variation in parenting by race/ethnicity. In terms of parental control, racial/ethnic minority parents engaged in higher levels of monitoring compared to whites. However, African American adolescents experienced lower levels of supervision compared to whites and Hispanics. In terms of parent-child relations, there were no statistically significant racial/ethnic differences in shared-decision making and closeness with parents. Whites engaged in higher levels of activities and ate dinner more frequently with their parents compared to most of the other racial/ethnic groups, but Asians engaged in the highest level of communication about school with parents compared to everyone else.

Parenting Differences by Sex

Mean levels of parental monitoring or supervision did not differ by sex (See Table 4.3). In addition, there were no statistically significant differences by sex in levels of shared decision making, activities, eating dinner with parents or communication. However, males (4.37) had higher levels of closeness with parents compared to females (4.20) and females (3.99) experienced higher levels of shared decision making with parents compared to males (3.93).

Parenting Differences by Welfare/Poverty Status and Parent Education

Welfare/Poverty Status

There were no statistically significant differences by welfare/poverty status in levels of parental monitoring, supervision and shared decision making (See Table 4.4). The poor engaged in lower mean levels of activities (1.30) and eating dinner (4.66) with parents compared to the non-poor (activities: 1.61 and dinners: 5.19). There were lower levels of parent-child communication about school among the poor (1.03) compared to the non-poor (1.13). The poor

(4.25) also had slightly lower levels of closeness with parents compared with the non-poor (4.30).

In summary, although poverty status was not related to the parental control measures of monitoring or supervision and was not related to shared decision making, it was related to a number of parent-child relation measures, with poor parents engaging in less activities, meal-sharing and communication with their children and having less close to their children compared to non-poor parents.

Parent Education

Similar to poverty status, there were no statistically significant differences by parental education in levels of monitoring and supervision (See Table 4.4). There were differences in parent-child relations. Respondents with parents whose education was a high school degree or less (1.39) had a lower mean level of shared activities compared to respondents with parents who earned more than a high school degree (1.71). Respondents with low parent education (LPE) (3.93) had slightly lower levels of shared decision making compared to non-LPE (4.00) respondents. LPE (4.96) respondents ate dinner with their parents less than non-LPE (5.15) respondents. LPE (1.05) respondents had lower levels of parent communication compared to non-LPE (1.19) respondents. Levels of parental closeness did not differ by parent education.

In summary, parent education was not related to parental control or closeness. However, LPE respondents engaged in lower levels of shared activities, shared decision making, shared dinners and communication compared to non-LPE respondents.

Bivariate Associations between Disadvantage and Parenting with Physical Activity

Table 4.5 provides the bivariate relationships between disadvantage measures, parenting measures and static and longitudinal measures of physical activity at WII and WIII. An odds ratio that is less than one for the “not active” category means there is a reduction in the likelihood of not being active or improved activity levels. An odds ratio of greater than one for the “not active” category indicates an increase in the likelihood of not being active and a reduction in levels of activity. Measures of change in physical activity can be interpreted in a similar fashion, using the “stay active” category as the reference category.

There was a negative relationship between welfare/poverty status and physical activity at WII and WIII. There was also a negative relationship between LPE and physical activity at both waves. Poor adolescents were 17% more likely to not be active at WII and 21% more likely to not be active at WIII than non-poor adolescents. LPE adolescents were 30% more likely to not be active at WII and 28% more likely to not be active at WIII than non-LPE adolescents. Poverty and parent education were also related to longitudinal measures of physical activity. Poor adolescents were 22% more likely to become not active from WII to WIII and 38% more likely to stay not active from WII to WIII (relative to staying active) compared to non-poor adolescents. LPE adolescents were 27% more likely to become not active from WII to WIII and 58% more likely to stay not active from WII to WIII compare to non-LPE adolescents.

Parenting measures were also related to physical activity outcomes. When comparing bivariate associations, parent-child relationship measures were more often associated with physical activity than parental control measures. Among parental control measures, monitoring was associated with physical activity at WII and staying not active from WII to WIII, with higher amounts of monitoring reducing the likelihood of not being active at WII and staying not active from WII to WIII. For example, a one-unit increase in parental monitoring was associated with a

14% decrease in the likelihood of being not active at WII, and a 15% decrease in the likelihood of staying not active between WII and WIII. Supervision reduced the likelihood of staying active from WII to WIII. Among the parent-child relations measures, shared activities, shared dinners, parent-child communication and closeness were related to physical activity at both waves. Higher levels of activities, meals, parent-child communication and closeness reduced the likelihood of not being active at both waves. Shared decision making was only related to physical activity at WII. Parent-child activities, communication and closeness were also related to both longitudinal measures of physical activity. Shared decision making and meals were only related to staying not active.

These results reproduce previous research on the relationship between SES and physical activity and highlight the importance of parent behaviors on influencing physical activity in adolescence and young adulthood. They also establish the potential for parenting behaviors to mediate family disadvantage effects on physical activity. These results also support previous research that show that education is more likely to capture aspects of behavior and lifestyle, such as physical activity, than income-based measures of socioeconomic status (Shavers 2007).

Longitudinal Multinomial Logistic Regression Models

Table 4.6 presents the multinomial logistic regression results for the main analysis. Model 1, which includes a measure of living below poverty or receiving welfare before the age of 18, replicates the descriptive results. Poor adolescents were 22% more likely to become not active from WII to WIII and 38% more likely to stay not active from WII to WIII (relative to staying active) compared to non-poor adolescents. Once parental education was included in Model 2, the effects of poverty became insignificant. LPE adolescents were 23% more likely to

become not active and 51% more likely to stay not active from WII to WIII. These results suggest that the negative influence of income on physical activity works through parental education. This also supports previous research that education has an independent effect on health, after controlling for income (Ross and Wu 1995).

Model 3 included measures of race/ethnicity. No measures of race/ethnicity were significantly related to physical activity when, poverty and education were included in the model. The effect of parent education was also not reduced with the inclusion of the race/ethnicity measures. Once parenting measures were included in Model 4, the effect of parental education on becoming not active became insignificant and the effect of parent education on staying not active reduced from 1.52 to 1.34. Among the parental control measures, parental monitoring was negatively associated with staying not active from WII to WIII. Shared activities and closeness and warmth were negatively related to both staying not active and becoming not active from WII to WIII. These results provide evidence that these parenting measures completely mediated the relationship between LPE and becoming not active from WII to WIII and partly mediated the relationship between LPE and staying not active from WII to WIII.

Following the rules of mediation outlined by Judd and Kenny (1981) and Baron and Kenny (1986)⁸, shared activities completely mediated the relationship between LPE and becoming not active from WII to WIII, and shared activities, dinners, decision-making and

⁸ Baron and Kenny (1986) and Judd and Kenny (1981) rules of mediation require that: (1) There must be a significant relationship between the independent variable (i.e., LPE) and the dependent variable (i.e., physical activity) (See Table 3); (2) the relationship between the independent variable (i.e., LPE) and the mediator (i.e., parenting measure) must be significant (See Table 2C); (3) the relationship between the mediator (i.e., parenting measure) and the dependent variable (i.e., physical activity) must be significant (See Table 3). It should be noted that for complete mediation, the independent variable (i.e., LPE) will no longer be significantly related to the dependent variable (i.e., physical activity) once the mediator is controlled for.

communication⁹ partly mediated the relationship between LPE and staying not active from WII to WIII. Although closeness and parental monitoring were significant in Model 3, they cannot be mediators because there was no significant relationship between parental education and these parenting measures at the bivariate level (See Table 4.4).

Model 5 includes controls for sex, age, family structure, family size, extended family, parental obesity and having a full-time working mother. Females were more likely to become and stay not active from WII to WIII compared to males. In addition, as age increased, the likelihood of staying not active also increased. There were no other statistically significant relationships between the control measures and physical activity. The relationship between LPE and staying not active remained significant and the coefficient did not reduce in size. Parental activities and closeness also remained significantly related to both staying and becoming not active. The relationship between parental monitoring and physical activity became insignificant, suggesting that parental monitoring is related to these control variables. Model 6 includes interaction terms between parent activities and sex, parental closeness and sex and parental monitoring and sex (results not shown). These results were not significant, suggesting there was no differential effect of parenting on physical activity by sex.

Discussion and Conclusion

This paper investigated the mediating effects of parenting in the relationship between poverty and physical activity outcomes in the transition to adulthood, using a nationally representative, ethnically and socioeconomically diverse sample. This paper also created multiple measures of parental control and parent-child interactions and examined how these

⁹ Although shared dinners, communication and decision-making were not significant in Model 4, they were significantly related to physical activity at the bivariate level (See Table 3) and also with the inclusion of poverty and race/ethnicity (results not shown).

parenting measures differed by race/ethnicity, sex and SES. Although there was interesting variation in parenting by race/ethnicity and sex, the most robust and consistent differences were by SES. Higher SES parents engaged in higher levels of activities, meal sharing, shared decision-making, communication and closeness, compared to lower SES parents. Level of parental control, measured by monitoring and supervision, did not vary by SES.

Of the two SES measures used in the analysis, parental education was the only SES measure significantly related to physical activity, when both welfare/poverty status and parental education were included in the model. This finding supports previous research that finds independent effects of education on health, after controlling for income (Ross and Wu 1995). Although both income and education are important components of SES, with independent effects on health, research has not been able to completely disentangle effects of income versus education (House et al. 1994). It is argued that the effects of income and education may work via different causal pathways to affect health. For example, in relation to physical activity, higher parental incomes can provide better access to recreational activities, such as sports, better schools with better quality exercise facilities and physical education programs, and better neighborhoods with access to parks and playground that facilitate physical activity. In contrast, better-educated parents have the knowledge and life skills to gain more ready access to resources and information that can promote their child's physical activity (Adler and Newman 2002). For example, higher educated parents might be more aware of the American Heart Associations' recommendations for daily moderate to vigorous physical activity for children and adolescents and the multiple ways that they can promote physical activity (AHA 2009) than non-educated parents of similar income levels. Research also finds that behavioral factors, such as lack of exercise, overeating, drinking and smoking, explain a large proportion of the relationship

between education and physical health (Mirowsky and Ross 2003; House et al. 1994). More theoretical and empirical work will be needed to better disentangle education and income effects on health and health behavior.

This paper also found that although measures of parental control did not mediate the relationship between LPE and physical activity, multiple measures of parent-child relations did mediate the relationship between LPE and physical activity. Shared activities completely mediated the relationship between LPE and becoming not active from WII to WIII, and shared activities, dinners, decision-making and communication partly mediated the relationship between LPE and staying not active from WII to WIII. Parental closeness and parental monitoring, were also related to staying not physically active, but did not serve to mediate the relationship between LPE and staying not physical activity. These results highlight the importance of parent-child interactions in influencing physical activity outcomes. In addition to instrumental support that parents provide, such as providing transportation to recreational activities, spending time with children in activities unrelated to physical activity, such as talking about school, eating, shopping or making decisions, also help to positively influence child physical activity. High levels of parent-child interaction increase children's feelings of self-efficacy and competence (Lareau 2002; 2003) that can translate into increased levels of physical activity. Disadvantaged parents engage in these activities less than more advantaged parents and engage in similar levels of monitoring and supervision as advantaged parents.

The results support both the family process model (Conger et al. 2002; McLoyd 1990, 1998) and socialization theory (Maccoby 1992). Lower levels of parent-child interaction among LPE parent supports the family process model, which argues that poor parents have a diminished ability to facilitate communication and engage in activities with children due to stress. However,

the similar levels of supervision and monitoring of LPE parents compared to non-LPE parents, does not support the family process model, which would argue that LPE parents have less ability to monitor and supervise their children. The similar levels of monitoring and supervision among low SES and higher SES parents does support socialization theory, which would argue that low SES parents might engage in levels of monitoring and supervision similar to or more than high SES parents, to protect their children from the risks in their environment and hopefully lead to positive development. As discussed earlier, although these parenting strategies of monitoring and supervision might protect children from other health risk behaviors, such as teen pregnancy or drug use, it does not facilitate physical activity. These results also support the importance of social capital within the family in the form of strong parent-child relations that help facilitate positive health behaviors of children. Both the mediating and independent effects of parent-child interactions on physical activity support social capital theory (Bourdieu 1985).

Other important demographic measures and measures of disadvantage, such as race/ethnicity, family structure, family size and presence of extended family members were not related to physical activity when education and income were also included in the model. In addition, parental obesity, which was used as a proxy for role-modeling of physical activity (and genetic predisposition for physical activity), was not significant in the full models. Previous research has found mixed evidence on the effects of parental role modeling of physical activity on their child's activity (Sallis and Owen 1999; Trost et al. 2003). The Add Health data do not contain sufficient information on parental behaviors to fully determine the existence of role-modeling effects. The significant relationship between female gender and physical activity highlight important and known gender differences in physical activity. Females exhibit lower levels of physical activity than males and parental support differs by sex (Gustafson and Rhodes

2006). However, the lack of significant interactions between sex and parenting measures used in this analysis indicate that the effect of parenting on physical activity does not differ by sex.

Despite its contributions to the literature, this paper also has a number of limitations. Little is known about the causal processes that link economic hardship to child outcomes, especially outcomes that persist into adulthood. Parenting practices cover only one of several important pathways between poverty in childhood and outcomes in childhood and later life (Brooks-Gunn and Duncan 1997). Multiple pathways exist that should be further explored to better understand socioeconomic disparities in physical activity, such as elements of the school, peer and neighborhood context (e.g., school opportunities for physical activity, friends' activity levels, neighborhood safety and physical environment).

An important concern that merits attention is endogeneity bias (also called reverse causality). Endogeneity bias occurs when important predictor variables are the result, rather than the cause, of the outcome of interest (Duncan, Magnuson and Ludwig 1999). In this proposed analysis, claims regarding causal direction cannot be made with certainty because characteristics of children may elicit certain modes of behavior from parents, rather than parenting practices influencing child outcomes. Because I am measuring parenting practices at WI and physical activity outcomes at WII and WIII, concerns about directionality are somewhat reduced.

A selection issue of particular concern is that parental practices could also be influenced by child overweight status at WI. I split the sample into those who were obese at WI and those who were not, and reran all models. There were not many differences in the coefficients. However, descriptively, mean levels of parental control and parent child-interactions did differ slightly by obesity status at WI. There were no particular patterns of differences in these relationships. I decided against controlling for obesity at WI or removing obese individuals from

the sample, because it would introduce additional biases to the sample.

In addition to unmeasured characteristics of children, social selection bias may operate if parents hold unmeasured characteristics related to their overall level of functioning and competence that could affect both their parenting and their income-related activities. This would mean that the association between parental SES, parenting, and child and adolescent outcomes is spurious because it is caused by a third variable. In this case both children's development and SES emanate from unobserved parental characteristics. The research consensus is that differences in child rearing by SES are partly due to the circumstances in which parents live and unmeasured characteristics of parents that affect their SES and parenting practices simultaneously (Conger and Donnelan 2007; Hoff et al. 2002). While, again, I cannot make claims about causal impacts, I am mainly interested in whether parenting behaviors mediate some of the effects of poverty. There is also a selection issue closely tied to the social contexts in which the parents and their children live. Physical activity and parenting practices are influenced by multiple levels of the social environment, beyond the family. Parents choose to live in certain neighborhoods and enroll their kids in certain schools that facilitate or impede physical activity. Bias is associated with unobserved characteristics of parents that affect their choices. However, the many family SES and parenting behaviors I include in this analysis represent a large set of parenting characteristics that likely capture this selection. Most analysis do not include the many and diverse parenting measures.

What happens during the transition to adulthood has a great impact on young people's futures. Better understanding the social influences of physical activity may serve to explain the role of health in influencing successful transitions into adulthood, especially for vulnerable and disadvantaged populations. Family context plays a pivotal role in the developmental trajectories

and health behaviors and outcomes in adolescence and in the transition to young adulthood. This paper has shown that parenting is an important pathway through which SES influences physical activity in youth that endure into adulthood. Parenting behavior is one of the most important modifiable factors for youth physical activity. Parental communication and engagement can serve to channel their children into engaging in health promoting activities, such as physical activity and can increase levels of perceived confidence in their children, which is a psychosocial correlate of activity behavior (Kimićek, Horn and Shurin 1996). Programs that can provide support to disadvantaged parents to engage in activities with their children and support and encourage shared dinner eating and decision-making may serve as useful family level interventions that can help to decrease socioeconomic disparities in physical activity. Future research should continue to explore other parenting measures that may also influence physical activity, such as extensive reasoning and enrollment in organized leisure activities.

References

- Aber, J. Lawrence, Neil G. Bennett, Dalton C. Conley and Jiali Li. 1997. "The Effects of Poverty on Child Health and Development." *Annual Review of Public Health* 18: 463-483.
- Adkins, Sarah, Nancy E. Sherwood, Mary Story and Marsha Davis. 2004. "Physical Activity among African-American Girls: The Role of Parents and the Home Environment." *Obesity Research* 12(Supplement): 38S-45S.
- Adler, Nancy E. and Katherine Newman. 2002. "Socioeconomic Disparities In Health: Pathways And Policies: Inequality in Education, Income, and Occupation Exacerbates the Gaps between the Health "Haves" and "Have-Nots." *Health Affairs* 21(2):60-76.
- Ainsworth B. E., W. L. Haskell, A. S. Leon, D. R. Jacobs Jr, H. J. Montoye, J. F. Sallis, R. S. Paffenbarger Jr. 1993. "Compendium of Physical Activities: Classification of Energy Costs of Human Physical Activities." *Medicine and Science in Sports and Exercise* 25:71-80.
- Airhihenbuwa, Collins O. 1995. *Health and Culture. Beyond the Western Paradigm*. Thousand Oaks, CA: Sage.
- Amato, Paul. 1993. "Children's Adjustment to Divorce: Theories, Hypotheses and Empirical Support." *Journal of Marriage and the Family* 55:23-38.
- American Heart Association (AHA). 2009. "Exercise (Physical Activity) and Children." Retrieved January 6, 2009 (<http://www.americanheart.org/presenter.jhtml?identifier=4596>).
- Andersen, Ross E., Carlos J. Crespo, Susan J. Bartlett, Lawrence J. Cheskin and Michael Pratt. 1998. "Relationship of Physical Activity and Television Watching With Body Weight and Level of Fatness Among Children." *The Journal of the American Medical Association* 279:938-942.
- Anderson, Patricia M. and Kristin F. Butcher. 2006. "Childhood Obesity: Trends and Potential Causes." *The Future of Children* 16(1):19-45.
- Anderssen, Norman, Bente Wold and Torbjorn Torsheim. 2006. "Are Parental Health Habits Transmitted to Their Children? An Eight Year Longitudinal Study of Physical Activity in Adolescents and Their Parents." *Journal of Adolescence* 29(4):513-524.
- Baron, Reuben M. and David A. Kenny. 1986. "The Moderator-Mediator Variable Distinction in Social Psychological Research: Conceptual, Strategic, and Statistical Considerations." *Journal of Personality and Social Psychology* 51(6):1173-1182.
- Bauer, Katherine W., Melissa C. Nelson, Kerri N. Boutelle and Dianne Neumark-Sztainer. 2008. "Parental Influences on Adolescents' Physical Activity and Sedentary Behavior: Longitudinal Findings from Project EAT-II." *The International Journal of Behavioral Nutrition and Physical Activity* Published online 2008 February 26. doi: 10.1186/1479-5868-5-12.

- Baumrind, Diana. 1991. "The Influence of Parenting Style on Adolescent Competence and Substance Use." *Journal of Early Adolescence* 11(1):56-95.
- Baumrind, Diana. 1972. "An Exploratory Study of Socialization Effects on Black Children: Some Black-White Comparisons." *Child Development* 43(1):261-267.
- Beech, Bettina M. Shiriki K. Kumanyika, Tom Baranowski, Marsha Davis, Thomas N. Robinson, Nancy E. Sherwood, Wendell C. Taylor, George Relyea, Ainong Zhou, Charlotte Pratt, Ayisha Owens and Nikko S. Thompson. 2004. "Parental Cultural Perspectives in Relation to Weight-Related Behaviors and Concerns of African-American Girls." *Obesity Research* 12:7S-19S.
- Birch, Leann L. and Jennifer O. Fisher. 1998. "Development of Eating Behaviors Among Children and Adolescents." *Pediatrics* 101: 539-549.
- Bourdieu, Pierre. 1985. "The Forms of Capital." Pp. 241-258 in J. G. Richardson, Ed., *Handbook of Theory and Research for the Sociology of Education*. New York: Greenwood.
- Bradley, Carla R. 1998. "Child Rearing in African American Families: A Study of the Disciplinary Practices of African American Parents." *Journal of Multicultural Counseling and Development* 26:273-281.
- Bronte-Tinkew, Jacinta, Brett Brown, Jennifer Carrano and Rebecca Shwalb. 2005. "Logic Models and Outcomes for Youth in the Transition to Adulthood: Report to the DC Children and Youth Investment Trust Corporation." Washington, D.C.: Child Trends, Inc., Retrieved September 21, 2006 (www.childtrends.org/Files/Transition_to_Adulthood_Report-FINAL.pdf).
- Brooks-Gunn, Jeanne and Greg J. Duncan. 1997. "The Effects of Poverty on Children." *The Future of Children. Children and Poverty* 99:353-395.
- Brownson, Ross C., Tegan K. Boehmer and Douglas A. Luke. 2005. "Declining Rates of Physical Activity in the United States: What Are the Contributors?" *Annual Review of Public Health* 26: 421-443.
- Brustad, Robert J. 1996. "Attraction to Physical Activity in Urban Schoolchildren: Parental Socialization and Gender Influences." *Research Quarterly for Exercise and Sport* 67(3):316-323.
- Burton, Linda and Robin L. Jarrett. 2000. "In the Mix, Yet on the Margins: The Place of Families in Urban Neighborhood and Child Development Research." *Journal of Marriage and the Family* 62:1114-1135.
- Centers for Disease Control (CDC) (2005). Youth Risk Behavior Surveillance-United States, 2005. *MMWR* 55(SS-5).
- Coleman, James. 1988. "Social Capital in the Creation of Human Capital." *American Journal of Sociology* 94:s95-s120.

Collins, W. Andrew, Eleanor E. Maccoby, Laurence Steinberg, E. Mavis Hetherington and Marc H. Bornstein, Marc H. 2000. "Contemporary Research on Parenting: The Case for Nature and Nurture." *American Psychologist* 5:218-232.

Conger, Rand D. and Katherine J. Conger. 2002. "Resilience in Midwestern Families: Selected Findings from the First Decade of a Prospective, Longitudinal Study." *Journal of Marriage and the Family* 64:361-373.

Conger, Rand D., Lora Ebert Wallace, Yumei Sun, Ronald L. Simons, Vonnie C. McLoyd and Gene H. Brody. 2002. "Economic Pressure in African American Families: A Replication and Extension of the Family Stress Model." *Developmental Psychology* 38(2): 179-193.

Conger, Rand D. and M. Brent Donnellan. 2007. "An Interactionist Perspective on the Socioeconomic Context of Human Development." *Annual Review of Psychology* 58:175-199.

Crosnoe, Robert, Rashmita S. Mistry and Glen H. Elder Jr. 2002. "Economic Disadvantage, Family Dynamics, and Adolescent Enrollment in Higher Education." *Journal of Marriage and the Family* 64:690-702.

Crossman, Ashley, Deborah Anne Sullivan and Mary Benina. 2006. "The Family Environment and American Adolescents' Risk of Obesity as Young Adults." *Social Science and Medicine* 63(9):2255-2267.

Darling, Nancy and Laurence Steinberg. 1993. "Parenting Style as Context: An Integrative Model." *Psychological Bulletin* 113(3):487-496.

Davison, Kirsten. K. and Leann L. Birch. 2001. "Childhood Overweight: A Contextual Model and Recommendations for Future Research." *Obesity Reviews* 2(3):159-171.

Davison, Kirsten. K., Tanja M. Cutting and Leann L. Birch. 2003. "Parents' Activity-Related Parenting Practices Predict Girls' Physical Activity." *Medicine and Science in Sports and Exercise* 35(9):1589-1595.

Davison, Kirsten. K., Lori A. Francis and Leann L. Birch. 2005. "Reexamining Obesigenic Families: Parents' Obesity-Related Behaviors Predict Girls' Change in BMI." *Obesity Research* 13(11):1980-1990.

Dempsey, Jill M., Jay C. Kimiecik and Thelma S. Horn. 1993. "Parental Influence on Children's Moderate to Vigorous Physical Activity Participation: An Expectancy-Value Approach." *Pediatric Exercise Science* 5(2):151-167.

Duncan, Greg J., Katherine A. Magnuson and Jens Ludwig. 2004. "The Endogeneity Problem in Developmental Studies." *Research in Human Development* 1:59-80.

Elder, Glen H. Jr. 1999. *Children of the Great Depression: Social Change in Life Experience: 25th Anniversary Edition*. Oxford: Westview Press.

- Elder, Glen H. Jr., Jacquelynne S. Eccles, Monika Ardelt and Sarah Lord. 1995. "Inner-City Parents Under Economic Pressure: Perspectives on the Strategies of Parenting." *Journal of Marriage and the Family* 57(3):771-784.
- Furstenberg, Frank F. Jr., Thomas D. Cook, Jacquelynne Eccles, Glen H. Elder Jr., and Arnold Sameroff. 1999. *Managing to Make It: Urban Families and Adolescent Success*. Chicago, IL: The University of Chicago Press.
- Fogelholm, M., O. Nuutinen, M. Pasanen, E. Myohanen and T. Saatela. 1999. "Parent-Child Relationship of Physical Activity Patterns and Obesity." *International Journal of Obesity* 23: 1262-1268.
- Garcia, Anne W., Mary Ann Norton Broda, Marilyn Frenn, Cynthia Coviak, Nola J. Pender and David L. Ronis. 1995. "Gender and Developmental Differences in Exercise Beliefs among Youth and Prediction of Their Exercise Behavior." *Journal of School Health* 65: 213-219.
- García Coll, C, and L. M. Pachter. 2002. "Ethnic and Minority Parenting." In Bornstein, MH (Ed.) *Handbook of Parenting: Social Conditioning and Applied Parenting*, 4, (2nd ed) 1-20). Mahwah, NJ: Lawrence Erlbaum
- Gordon-Larsen, Penny, Robert G. McMurray and Barry M. Popkin. 2000. "Determinants of Adolescent Physical Activity and Inactivity Patterns." *Pediatrics* 105(6), Retrieved September 21, 2006 (<http://pediatrics.aappublications.org/cgi/content/abstract/105/6/e83>).
- Gordon-Larsen, Penny, Robert G. McMurray and Barry M. Popkin. 1999. "Adolescent Physical Activity and Inactivity Vary by Ethnicity: The National Longitudinal Study of Adolescent Health." *Journal of Pediatrics* 135(3):301-6.
- Gordon-Larsen, Penny, Melissa C. Nelson and Barry M. Popkin. 2004. "Longitudinal Physical Activity and Sedentary Behavior Trends: Adolescence to Adulthood." *American Journal of Preventative Medicine* 27(4): 277-283.
- Gortmaker, S. L., Must, A., Perrin, J. M., Sobol, A. M. & Dietz, W. H. 1993. "Social and Economic Consequences of Overweight in Adolescence and Young Adulthood." *New England Journal of Medicine* 329:1008-1012.
- Grant, Kathryn E., Bruce E. Compas, Alice F. Stuhlmacher, Audrey E. Thurm, Susan D. McMahon and Jane A. Halpert. 2003. "Stressors and Child and Adolescent Psychopathology: Moving From Markers to Mechanisms of Risk." *Psychological Bulletin* 129(3): 447-466.
- Gustafson, Sabrina L. and Ryan E. Rhodes. 2006. "Parental Correlates of Physical Activity in Children and Early Adolescents." *Sports Medicine* 36(1):79-97.
- Hallal, Pedro C., Cesar G. Victora, Mario R. Azevedo and Jonathan C.K. Wells. 2006. "Adolescent Physical Activity and Health: A Systematic Review." *Sports Medicine* 36(12):1019-

1030.

Harris, Kathleen Mullan, Francesca Florey, Joyce W. Tabor, Peter S. Bearman, Jo Jones, and J. Richard Udry. 2003. The National Longitudinal Study of Adolescent Health: Research Design. Retrieved September 21, 2006 (<http://www.cpc.unc.edu/projects/addhealth/design.html>).

Harris, Kathleen Mullan, Frank F. Furstenberg, Jr., and Jeremy K. Marmer. 1998. "Paternal Involvement with Adolescents in Intact Families: The Influence of Fathers over the Life Course." *Demography* 35: 201-216.

Harris, Kathleen Mullan, Penny Gordon-Larsen, Kim Chantala and J. Richard Udry. 2006. "Longitudinal Trends in Race/Ethnic Disparities in Leading Health Indicators from Adolescence to Young Adulthood." *Archives of Pediatrics and Adolescent Medicine* 160: 74-81.

Harris, Kathleen Mullan and Guang Guo. 2000. "The Mechanisms Mediating the Effects of Poverty on Children's Intellectual Development." *Demography* 37(4): 431-447.

Harris, Kathleen Mullan, Rosalind Berkowitz King, and Penny Gordon-Larsen. 2005. "Healthy Habits Among Adolescents: Sleep, Exercise, Diet, and Body Image." Pp.111-132 in Kristin Anderson Moore and Laura H. Lippman, eds., *What Do Children Need to Flourish? Conceptualizing and Measuring Indicators of Positive Development*. New York: Springer.

Harris, Kathleen Mullan and Jeremy K. Marmer. 1996. "Poverty, Paternal Involvement, and Adolescent Well-Being." *Journal of Family Issues* 17: 614-640.

Harris, Kathleen Mullan and Suzanne Ryan. 1999. "Parental and Neighborhood Influences on Adolescent Risk Behavior." Presented at the Conference on Parenting and the Child's World: Multiple Influences on Intellectual and Social-Emotional Development, sponsored by the National Institute of Child Health and Human Development and Robert Wood Johnson Foundation: NIH Campus, Bethesda, MD.

Harris M. B. and K. M. Koehler. 1993. "Eating and Exercise Behaviors and Attitudes of Southwestern Anglos and Hispanics." *Health Psychology* 7:165-174.

Hart, K., J. Bishop, and H. Truby. 2003. "Abstracts of Scientific Presentation from the British Dietetic Association, Annual Conference, Nottingham, 07-14 June 2003 Changing Children's Diets: Developing Methods and Messages." *Journal of Human Nutrition & Dietetics* 16(50):365-370.

Heitzler, Carrie D., Sarah Levin Martin, Jennifer Duke and Marian Huhman. 2006. "Correlates of Physical Activity in a National Sample of Children Aged 9-13 Years." *Preventive Medicine* 42(4):254-260.

Hoff, Erica, Brett Laursen and Twila Tardif. 2002. "Socioeconomic Status and Parenting." Pp. 231-252 in *Handbook of Parenting, 2nd Edition*, edited by M.H. Bornstein. Mahwah, NJ: Lawrence Erlbaum Associates.

- House, James, James Lepkowski, Ann Kinney, Richard Mero, Ronald Kessler, and A. Regula Herzon. 1994. "The Social Stratification of Aging and Health." *Journal of Health and Social Behavior* 35(3):213-234.
- Iceland, John. 2003. *Poverty in America: A Handbook*. Berkeley, CA: University of California Press.
- Jacobson, Kristen C. and Lisa J. Crockett. 2000. "Parental Monitoring and Adolescent Adjustment: An Ecological Perspective." *Journal of Research on Adolescence* 10(1):65-97.
- Judd, Charles M. and David A. Kenny. 1981. "Process Analysis: Estimating Mediation in Treatment Evaluations." *Evaluation Review* 5(5):602-619.
- Kaufman, Leslie, and Adam Karpati. 2007. "Understanding the Sociocultural Roots of Childhood Obesity: Food Practices among Latino Families of Bushwick, Brooklyn." *Social Science and Medicine* 64:2177-2188.
- Kimiecik, Jay C., Thelma S. Horn and Chris S. Shurin. 1996. "Relationships among Children's Beliefs, Perceptions of their Parents' Beliefs, and their Moderate-to-Vigorous Physical Activity." *Research Quarterly for Exercise and Sport* 67:324-336.
- Kimm, Sue Y.S., Nancy W. Glynn, Andrea M. Kriska, Bruce A. Barton, Shari S. Kronsberg, Stephen R. Daniels, Patricia B. Crawford, Zak I. Sabry, and Kiang Liu. 2002. "Decline in Physical Activity in Black Girls and White Girls during Adolescence." *New England Journal of Medicine* 347(10):709-715.
- King, Keith A., Jenneifer L. Tergerson and Bradley R. Wislon. 2008. "Effect of Social Support on Adolescents' Perceptions of and Engagement in Physical Activity." *Journal of Physical Activity and Health* 5:374-384.
- Kumanyika, Shiriki and Sonya Grier. 2006. "Targeting Interventions for Ethnic Minority and Low-Income Populations." *The Future of Children* 16(1):187-207.
- Kumanyika, Shiriki K., Eva Obarzanek, Nicolas Stettler, Ronny Bell, Alison E. Field, Stephen P. Fortmann, Barry A. Franklin, Matthew W. Gillman, Cora E. Lewis, Walker Carlos Poston, June Stevens and Yuling Hong. 2008. "Population-Based Prevention of Obesity: The Need for Comprehensive Promotion of Healthful Eating, Physical Activity, and Energy Balance: A Scientific Statement from the American Heart Association Council on Epidemiology and Prevention, Interdisciplinary Committee for Prevention (Formerly the Expert Panel on Population and Prevention Science)." *Circulation* 118:428-464.
- Kuo, JoAnn, Carolyn C. Voorhees, Jennifer A. Haythornthwaite and Deborah Rohm Young. 2007. "Associations between Family Support, Family Intimacy, and Neighborhood Violence and Physical Activity in Urban Adolescent Girls." *American Journal of Public Health* 97(1):101-103.

Lareau, Annette. 2003. *Unequal Childhoods: Class, Race, and Family Life*. Berkeley, CA: University of California Press.

Lareau, Annette. 2002. "Invisible Inequality: Social Class and Childrearing in Black Families and White Families." *American Sociological Review* 67(5): 747-776.

Lauderdale, Diane S., Richard Fabsitz, Joanne M. Meyer, Phyliss Sholinsky, Viswanathan Ramakrishnan and Jack Goldberg. 1997. *Medicine & Science in Sports & Exercise* 29(8):1062-1068.

LeVine, R. A. 1977. "Childrearing as Cultural Adaptation." In P.H. Leiderman, S.R. Tulkin and A. H. Rosenfeld (Eds.), *Culture and Infancy* (pp.15-27). London: Academic Press.

Lindsay, Ana C., Katarina M. Sussner, Juhee Kim and Steven Gortmaker. 2006. "The Role of Parents in Preventing Childhood Obesity." *The Future of Children* 16(1):169-186.

Long, J. Scott. 1997. *Regression Models for Categorical and Limited Dependent Variables*. Thousand Oaks, CA: Sage Publications.

Maccoby, Eleanor E. 1992. "The Role of Parents in the Socialization of Children: A Historical Overview." *Developmental Psychology* 28:1006-1017.

Maia, José A. R., Maritine Thomis and Gaston Beunen. 2002. "Genetic Factors in Physical Activity Levels: A Twin Study." *American Journal of Preventive Medicine* 23(2):87-91.

Malina, Robert M. 1996. "Tracking of Physical Activity and Physical Fitness Across the Lifespan". *Research Quarterly for Exercise and Sports* 67(Suppl. 3):S48-S57.

Mattocks, Calum, Andy Ness, Kevin Deere, Kate Tilling, Sam Leary, Steven N. Blair and Chris Riddoch. 2007. "Early Life Determinants of Physical Activity in 11 and 12 Year Olds: Cohort Study." *British Medical Journal*. Retrieved March 16, 2004 (doi:10.1136/bmj.39385.443565.BE).

McGuire, Maureen T., Peter J. Hannan, Dianne Neumark-Sztainer, Nicole H. Falkner Cossrow and Mary Story. 2002. "Parental Correlates of Physical Activity in a Racially/Ethnically Diverse Adolescent Sample." *Journal of Adolescent Health* 30(4): 253-261

McLanahan, Sara S. 1997. "Parent Absence or Poverty: Which Matters More?" in *Consequences of Growing Up Poor*, edited by Greg J. Duncan and Jeanne Brooks-Gunn. New York: Russell Sage Foundation.

McLanahan, Sara S., Nan Marie Astone, and Nadine F. Marks. 1991. "The Role of Mother-Only Families in Reproducing Poverty." in *Children in Poverty: Child Development and Public Policy*, edited by Aletha C. Huston, Cambridge, Australia: Cambridge University Press.

McLeod, Jane D. and Michael J. Shanahan. 1993. "Poverty, Parenting, and Children's Mental

Health.” *American Sociological Review* 58(3): 351-366.

McLoyd, Vonnie C. 1998. “Socioeconomic Disadvantage and Child Development.” *American Psychologist* 53(2): 185-204.

McLoyd, Vonnie C. 1990. “The Impact of Economic Hardship on Black Families and Children: Psychological Distress, Parenting, and Socioemotional Development.” *Child Development* 61: 311-346.

Mirowsky, John and Catherine E. Ross. 2003. *Education, Social Status, and Health*. Aldine de Gruyter: New York.

Mistry, Rashmita S., Elizabeth A. Vanderwater, Aletha C. Huston and Vonnie C. McLoyd. 2002. “Economic Well-Being and Children’s Social Adjustment: The Role of Family Process in an Ethnically Diverse Low-Income Sample.” *Child Development* 73(3):935-951.

Moore, L. L., D. A. Lombardi, M. J. White, J. L. Campbell, A. F. Olshan and R.C. Ellison. 1991. “Influence of Parents Physical Activity Levels on Activity Levels of Young Children.” *Pediatrics* 118(2): 215-219.

Morris, Pamela A., Aletha C. Huston, Greg J. Duncan, Danielle A. Crosby and Johannes M. Bos. 2001. *How Welfare and Work Policies Affect Children: A Synthesis of Research*. New York: Manpower Demonstration Research Corporation. Retrieved September 21, 2004 (<http://www.mdrc.org/publications/100/full.pdf>).

Nader, Philip R., Robert H. Bradley, Renate M. Houts, Susan L. McRitchie and Marion O’Brien. 2008. “Moderate-to-Vigorous Physical Activity from Ages 9 to 15 Years.” *The Journal of the American Medical Association* 300(3):295-305.

National Research Council (NRC) and Institute of Medicine (IOM), Committee on Evaluation of Children's Health. 2004. *Children's Health, the Nation's Wealth: Assessing and Improving Child Health*. Washington, DC: The National Academies Press.

National Research Council (NRC) and Institute of Medicine (IOM). 2002. *Community Programs to Promote Youth Development*. Edited by J. Eccles and J. A. Gootman. Washington, DC: National Academies Press.

Neumark-Sztainer, Dianne, Mary Story, Peter J. Hannan, Terri Tharp and Jeanna Rex. 2003. “Factors Associated With Changes in Physical Activity: A Cohort Study of Inactive Adolescent Girls.” *Archives of Pediatrics and Adolescent Medicine* 157(8):803-810.

Norman, Gregory J., Béatrice A. Schmid, James F. Sallis, Karen J. Calfas and Kevin Patrick. 2005. “Psychosocial and Environmental Correlates of Adolescent Sedentary Behaviors.” *Pediatrics* 116:908-916.

Ogden, Cynthia L., Margaret D. Carroll and Katherine M. Flegal. 2003. “Epidemiologic Trends

in Overweight and Obesity.” *Endocrinology and Metabolism Clinics of North America* 32(4):741-760.

Ogden, Cynthia L., Katherine M. Flegal, Margaret D. Carroll and Clifford L. Johnson. 2002. “Prevalence and Trends in Overweight Among U.S. Children and Adolescents, 1999-2000.” *Journal of the American Medical Association* 288(14): 1728-32.

Ogden, C. L., Carroll, M. D., Curtin, L. R. C., McDowell, M. A., Tabak C. J. and Flegal, K. M. 2006. “Prevalence of Overweight and Obesity in the United States, 1999-2004.” *Journal of the American Medical Association* 295:1549-1555.

Ornelas, India J., Krista M. Perreira and Guadalupe X. Ayala. 2007. “Parental Influences on Adolescent Physical Activity: A Longitudinal Study.” *International Journal of Behavioral Nutrition and Physical Activity* Published online 2007 February 2. doi: 10.1186/1479-5868-4-3.

Osgood, D. Wayne, E. Michael Foster, Constance Flanagan and Gretchen R. Ruth. 2005a. *On Your Own Without a Net: The Transition to Adulthood for Vulnerable Populations*. Chicago: University of Chicago Press.

Osgood, D. Wayne, Gretchen R. Ruth, Jacquelynne S. Eccles, Janis E. Jacobs, and Bonnie L. Barber. 2005b. “Six Paths through the Transition to Adulthood, Their Predictors and Consequences.” In *On the Frontier of Adulthood: Theory, Research, and Public Policy*, Richard A Settersten, Jr, Frank F. Furstenberg, Jr., and Rubén G. Rumbaut, Eds, p. 320-355. Chicago, IL: University of Chicago Press.

Pate, Russell R., Stewart G. Trost, Marsha Dowda, Alise E. Ott, Dianne S. Ward, Ruth Saunders, and Gwen Felton. 1999. “Tracking of Physical Activity, Physical Inactivity, and Health-Related Physical Fitness in Rural Youth.” *Pediatric Exercise Science* 11(4):364-376.

Pinderhughes, Ellen E., Robert Nix, E. Michael Foster and Damon Jones. 2001. “Parenting in Context; Impact of Neighborhood Poverty, Residential Stability, Public Services, Social Networks, and Danger and Parental Behaviors.” *Journal of Marriage and the Family* 63(4):941-953.

Pinderhughes, Ellen E., Kenneth A. Dodge, John E. Bates, Gregory S. Pettit and Arnaldo Zelli. 2000. “Discipline Responses: Influences of Parents’ Socioeconomic Status, Ethnicity, Beliefs about Parenting, Stress, and Cognitive-Emotional Processes.” *Journal of Family Psychology* 14(3): 380-400.

Prochaska, Judith J., Miki W. Rodgers and James F. Sallis. 2002. “Association of Parent and Peer Support with Adolescent Physical Activity.” *Research Quarterly for Exercise and Sport*. 73(2):206-210.

Pugliese, John and Barbara Tinsley. 2007. “Parent Socialization of Child and Adolescent Physical Activity: A Meta-Analysis.” *Journal of Family Psychology* 21(3):331-343.

Raftery, A.E. 1996. “Bayesian Model Selection in Social Research.” Pp. 111-163 in *Sociological Methodology, Volume 25*, edited by P. V. Marsden. Oxford: Basil Blackwell.

Raitakari, Olli T., Kimmo V K. Porkka, Simo Taimela, Risto Telama, Leena Räsänen and Jorma S. Viikari. 1994. "Effects of Persistent Physical Activity and Inactivity on Coronary Risk Factors in Children and Young Adults The Cardiovascular Risk in Young Finns Study." *American Journal of Epidemiology* 140: 195-205.

Rhee, Kyung E., Julie C. Lumeng, Danielle P. Appugliese, Niko Kaciroti and Robert H. Bradley. 2006. "Parenting Styles and Overweight Status in First Grade." *Pediatrics* 117:2047-2054.

Riesch, Susan K., Lori S. Anderson and Heather A. Krueger. 2006. "Parent-Child Communication Processes: Preventing Children's Health-Risk Behavior." *Journal for Specialists in Pediatric Nursing* 11(1): 41-56.

Rindfuss, Ronald R. 1991. "The Young Adult Years: Diversity, Structural Change and Fertility. Population Association of America Presidential Address." *Demography* 28:493-512.

Robinson, Thomas N., Michaela Kiernan, Donna M. Matheson and K. Farish Haydel. 2001. "Is Parental Control over Children's Eating Associated with Childhood Obesity? Results from a Population-Based Sample of Third Graders." *Obesity Research* 9(5): 306-312.

Ross, Catherine and Chia-ling Wu. 1995. "The Links between Education and Health." *American Sociological Review* 60(5):719-745.

Ryan, Suzanne. 2001. *Poverty, Social Context, and Adolescent Emotional Health*. Doctoral dissertation, University of North Carolina at Chapel Hill.

Sallis, James F., John E. Alcaraz, Thomas L. McKenzie and Melbourne F. Hovell. 1999. "Predictors of Change in Children's Physical Activity Over 20 Months: Variations by Gender and Level of Adiposity." *American Journal of Preventive Medicine* 16(3): 222-229.

Sallis, James F., John E. Alcaraz, Thomas L. McKenzie, Melbourne F. Hovell, Bohdan Kolody and Philip R. Nader. 1992. "Parental Behavior in Relation to Physical Activity and Fitness in 9-Year-Old Children." *American Journal of Diseases of Children* 146(11): 1383-1388.

Sallis, J. F., M. J. Buono, J. J. Roby, F. G. Micale and J. A. Nelson. 1993. "Seven-day Recall and Other Physical Activity Self-Reports in Children and Adolescents." *Medicine and Science in Sports and Exercise* 25: 99-108.

Sallis, James F., Robert B. Cervero, William Ascher, Karla A. Henderson, M. Katherine Kraft and Jacqueline Kerr. 2006. "An Ecological Approach to Creating Active Living Communities." *Annual Review of Public Health* 27: 297-322.

Sallis, James F. and Neville Owen. 1999. *Physical Activity and Behavioral Medicine*. Sage, Thousand Oaks, CA.

Sallis, James F., Judith J. Prochaska and Wendell C. Taylor. 2000. "A Review of the Correlates

of Physical Activity of Children and Adolescents." *Medicine and Science in Sports and Exercise* 32(5): 963-975.

Seltzer, Judith. 1994. "Consequences of Marital Dissolution for Children." *Annual review of Sociology* 20:235-266.

Shavers, Vickie L. 2007. "Measurement of Socioeconomic Status in Health Disparities Research." *The Journal of the National Medical Association* 99(9):1013-1023.

Smetana, Judith G., Nicole Campione-Barr and Aaron Metzger. 2006. "Adolescent Development in Interpersonal and Societal Contexts." *Annual Review of Psychology* 57:255-284.

Sobal, J. and Stunkard, A. J. 1989. "Socioeconomic Status and Obesity: A Review of the Literature." *Psychological Bulletin*, 105, 260-275.

Spencer, Margaret B. 1990. "Development of Minority Children: An Introduction." *Child Development* 61:267-269.

Steinberg, Laurence. 2001. "We Know Some Things: Parent-Adolescent Relationships in Retrospect and Prospect." *Journal of Research on Adolescence* 11(1):1-19.

Steinberg, Laurence; Sanford M. Dornbusch and B. Bradford Brown. 1992. "Ethnic Differences in Adolescent Achievement: An Ecological Perspective." *American Psychologist* 47(6):723-729.

Strauss, Richard S., Daria Rodzilsky, Gail Burack and Michelle Colin. 2001. "Psychosocial Correlates of Physical Activity in Healthy Children." *Archives of Pediatrics & Adolescent Medicine* 155(8):897-902.

Telama, Risto, Xiaolin Yang, Jorma Viikari, Ilkka Välimäki, Olli Wanneand Olli Raitakari. 2005. "Physical Activity from Childhood to Adulthood: A 21-year Tracking Study." *American Journal of Preventive Medicine* 28(3):267-273.

Thompson, Angela M., M. Louise Humbert, and Robert L. Mirwald. 2003. "A Longitudinal Study of the Impact of Childhood and Adolescent Physical Activity Experiences on Adult Physical Activity Perceptions and Behaviors." *Qualitative Health Research* 13:358-377.

Trost, Stewart G., James F. Sallis, Russell R. Pate, Patty S. Freedson, Wendell C. Taylor and Marsha Dowda. 2003. "Evaluating a Model of Parental Influence on Youth Physical Activity." 25: 277-282.

U.S. Department of Health and Human Services (US DHHS) (1996). *Physical Activity and Health: A Report of the Surgeon General*. Atlanta, GA: U.S. DHHS, CDC, National Center for Chronic Disease Prevention and Health Promotion. Retrieved October 18, 2007. (<http://www.cdc.gov/nccdphp/sgr/contents.htm>).

U.S. Department of Health and Human Services (US DHHS) (2000). *Healthy People 2010*.

Washington, DC: US Department of Health and Human Services. Retrieved January 14, 2007. (<http://www.healthypeople.gov/>).

Van Der Horst, Kalzine, Marijke J. Chin A. Paw, Jos W. R. Twisk and Willem Van Mechelen. 2007. "A Brief Review on Correlates of Physical Activity and Sedentariness in Youth." *Medicine & Science in Sports & Exercise*. 39(8):1241-1250.

Vega, William A. 1990. "Hispanic Families in the 1980s: A Decade of Research." *Journal of Marriage and the Family* 52:1015-1024.

Weiss, Robert. 2002. "Parenting Dimensionality and Typology in a Disadvantaged, African American Sample: A Cultural Variance Perspective." *Journal of Black Psychology* 28:142-173.

Wilson, William J. 1987. *The Truly Disadvantaged: The Inner City, the Underclass, and Public Policy*. Chicago: University of Chicago Press.

Wu Ying, Bonita F. Stanton, Jennifer Galbraith, Linda Kaljee, Lesley Cottrell, Xiaoming Li, Carole V. Harris, Dawn D'Alessandri and James M. Burns. 2003. "Sustaining and Broadening Intervention Impact: A Longitudinal Randomized Trial of 3 Adolescent Risk Reduction Approaches." *Pediatrics* 111(1): 32-38.

Figure 4.1. Conceptual Model

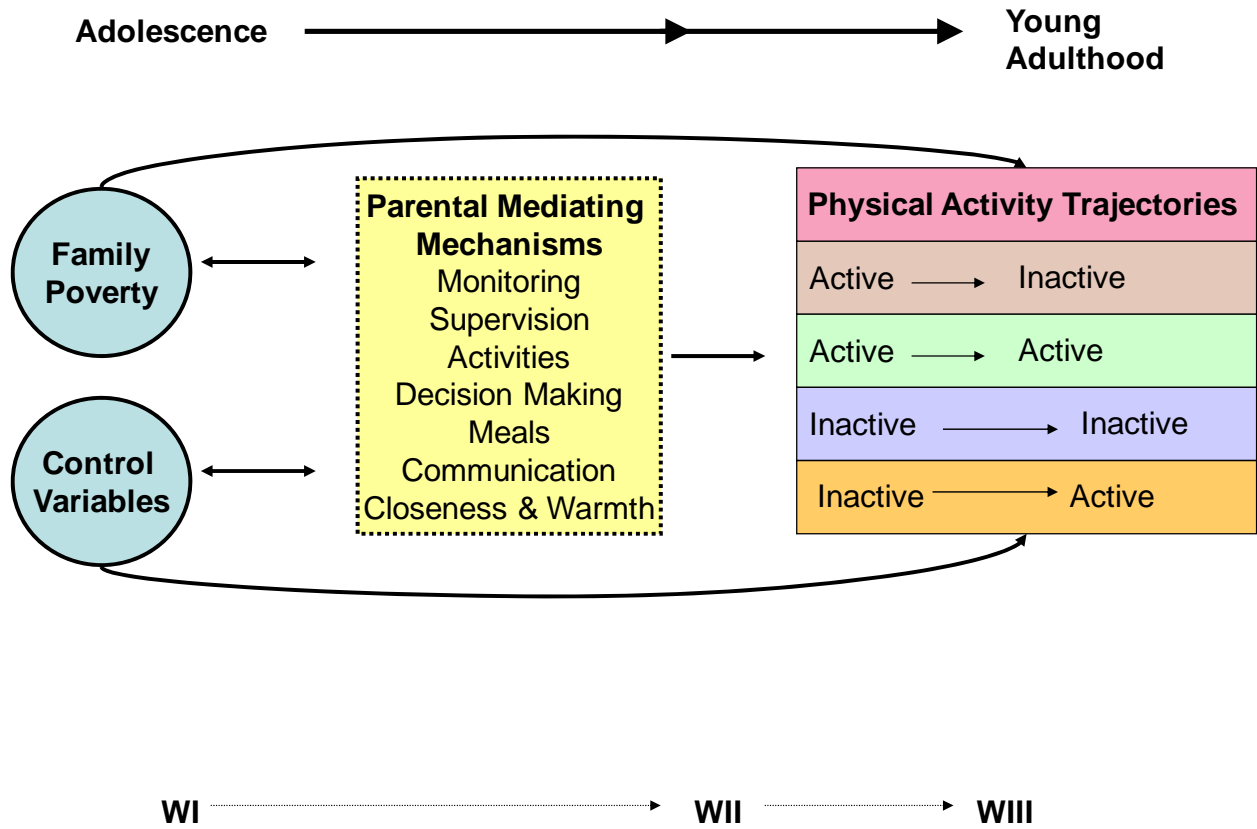


Table 4.1. Variable Descriptions, Means and Standard Deviations by Sex

Variable	Description	Total		Female		Male	
		Mean	SE	Mean	SE	Mean	SE
Outcomes							
Change in Physical Activity							
Become Not Active	Active at WII but not active at WIII	0.54	0.010	0.52	0.015	0.57	0.010
Stay Not Active	Not active at WII & not active at WIII	0.29	0.010	0.37	0.014	0.22	0.010
Become Active	Not Active at WII but active at WIII	0.03	0.003	0.02	0.004	0.03	0.004
Stay Active	Not active at WII & not active at WIII	0.13	0.005	0.09	0.006	0.18	0.008
Static Measures of Physical Activity							
Not Active at Wave II	Less than 5 bouts of Moderate to Vigorous Physical Activity at WII	0.32	0.010	0.40	0.015	0.25	0.011
Not Active at Wave III	Less than 5 bouts of Moderate to Vigorous Physical Activity at WIII	0.84	0.007	0.89	0.008	0.79	0.009
Disadvantage Measures							
Welfare/Poverty Status	Welfare receipt prior to the age of 18 and/or Family Income less than \$16,000/year	0.28	0.016	0.28	0.017	0.29	0.018
Parent Education High School or Less	Highest educated parent completed a high school degree (or equivalent) or less	0.62	0.019	0.63	0.020	0.61	0.020
Control Measures							
Ethnic Minority Status							
Non-Hispanic White (Reference Category)	Respondent reported white	0.72	0.028	0.72	0.029	0.71	0.028
Non-Hispanic Black	Respondent reported black	0.13	0.019	0.14	0.020	0.13	0.020
Hispanic	Respondent reported Hispanic	0.12	0.017	0.11	0.017	0.12	0.018
Asian	Respondent reported Asian	0.03	0.007	0.03	0.007	0.03	0.007
Other	Respondent reported other race	0.003	0.001	0.003	0.001	0.00	0.001
Female	Respondent reported female	0.49	0.008	-----	-----	-----	-----
Age	Self-reported age at WI (Range: 11-21)	14.94	0.115	14.84	0.115	15.04	0.121
Parent Obese	Report of mother or father being obese	0.23	0.007	0.23	0.008	0.24	0.010
Family Structure							
2 Biological Parents (Reference Category)	2 Biological/Adoptive Parents	0.63	0.012	0.62	0.014	0.63	0.015
Step family	Step family	0.16	0.006	0.17	0.009	0.16	0.007
Single mother	Single mother	0.20	0.011	0.20	0.011	0.20	0.013
Single father	Single father	0.00	0.001	0.00	0.001	0.00	0.001
Other family structure	Other family structure	0.01	0.001	0.01	0.002	0.01	0.001
Full-time Working Mother	Mother works full time	0.58	0.011	0.57	0.013	0.58	0.014
Family Size	Number of Siblings (Range: 0-12)	1.48	0.031	1.49	0.042	1.46	0.030
Extended Family	Respondent reported living with an extended family member at WI	0.10	0.007	0.10	0.008	0.09	0.008
Mediating Parenting Measures							
Parental Control							
Monitoring	Seven item additive scale of parental monitoring of amount and type of TV watched, eating, curfew, bedtime, friends and clothes (Range: 0-7)	2.03	0.053	2.02	0.052	2.04	0.061
Supervision	Three item additive index of parental presence when respondent leaves and comes home from school and goes to bed at night (Range: 0-3)	2.41	0.014	2.40	0.017	2.41	0.018
Parent Child Relations							
Shared Activities	A count of activities of the activities a parent and child did together in the previous four weeks (5 Items) (Range: 0-5)	1.52	0.029	1.49	0.032	1.55	0.035
Shared Decision-Making	Parent report of frequency in which parent and child make decisions together regarding the child's life (Range: 1-5)	3.96	0.017	3.99	0.022	3.93	0.021
Shared Meals	The frequency with which the respondent and parent eat dinner during the week (Range: 0-7)	5.04	0.063	5.00	0.075	5.06	0.068
Parent-Child Communication	Sum of two types of discussions with parents in the previous four weeks including school work or grades, or other things at school (Range: 0-2)	1.10	0.014	1.12	0.018	1.08	0.019
Closeness and Warmth	Summary measure based on mean response to four items: level of closeness, satisfaction with relationship, if parent is warm and loving and satisfaction with communication (Range: 1-5)	4.29	0.016	4.20	0.019	4.37	0.020
N		8,109		4,195		3,914	

Data are weighted.

Table 4.2. Mean Parenting Measures by Race/Ethnicity (N=8,109)

	Monitoring		Supervision		Activities		Decision		Meals		Communicate		Closeness	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
White	1.97	0.061	2.42	0.016	1.56	0.033	3.95	0.019	5.27	0.074	1.10	0.018	4.28	0.019
Black	2.11	0.080	2.32	0.038	1.43	0.05	3.93	0.037	4.00	0.088	1.12	0.035	4.34	0.036
Asian	2.23	0.128	2.33	0.06	1.55	0.088	4.03	0.069	4.73	0.215	1.25	0.041	4.23	0.051
Other	2.43	0.455	2.47	0.157	1.49	0.336	4.10	0.111	5.46	0.41	0.84	0.173	4.21	0.229
Hispanic	2.28	0.107	2.47	0.033	1.38	0.052	4.02	0.059	4.88	0.116	1.07	0.031	4.28	0.031

Table 4.3. Mean Parenting Measures by Sex

	Monitoring		Supervision		Activities		Decision		Meals		Communicate		Closeness	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Male (N=3,914)	2.04	0.061	2.41	0.018	1.55	0.035	3.93	0.021	5.06	0.068	1.08	0.019	4.37	0.020
Female (N=4,195)	2.02	0.052	2.40	0.017	1.49	0.032	3.99	0.022	5.00	0.075	1.12	0.018	4.20	0.019

Table 4.4. Mean Parenting Measures by Socioeconomic Status (Poverty/Welfare and Parent Education High School or Less)

	Monitoring		Supervision		Activities		Decision		Meals		Communicate		Closeness	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Non Poor	2.01	0.055	2.40	0.014	1.61	0.03	3.97	0.016	5.19	0.07	1.13	0.017	4.30	0.018
Poor	2.09	0.069	2.43	0.025	1.30	0.037	3.93	0.031	4.66	0.081	1.03	0.022	4.25	0.023
More than HS	1.97	0.059	2.38	0.019	1.73	0.038	4.00	0.018	5.15	0.077	1.19	0.022	4.31	0.02
HS or Less	2.07	0.060	2.42	0.016	1.39	0.029	3.93	0.023	4.96	0.07	1.05	0.018	4.28	0.018

Table 4.5. Associations Between Disadvantage Measures, Parenting Measures and Physical Inactivity at Waves II and III (Bivariate Odds Ratios)

	Static Measures of Activity		Change in Activity	
	Not Active WII	Not Active WIII	Become Not Active WII	Stay Not Active WIII
<i>Disadvantage Measures</i>				
Welfare/Poverty Status	1.17* (0.087)	1.21* (0.097)	1.22* (0.112)	1.38** (0.164)
Parent Education High School or Less	1.30*** (0.085)	1.28** (0.109)	1.27** (0.114)	1.58*** (0.158)
<i>Parental Control</i>				
Monitoring	0.86*** (0.019)	0.96 (0.023)	1.00 (0.026)	0.85*** (0.028)
Supervision	0.93 (0.047)	0.92 (0.050)	0.90 (0.054)	0.86* (0.059)
<i>Parent Child Relations</i>				
Shared Activities	0.66*** (0.021)	0.74*** (0.025)	0.77*** (0.028)	0.52*** (0.022)
Shared Decision-Making	0.91** (0.030)	0.92 (0.044)	0.93 (0.051)	0.85** (0.049)
Shared Meals	0.90*** (0.012)	0.97* (0.016)	0.99 (0.020)	0.89*** (0.019)
Parent-Child Communication	0.91* (0.034)	0.88* (0.048)	0.88* (0.052)	0.82** (0.049)
Closeness and Warmth	0.68*** (0.033)	0.72*** (0.054)	0.73** (0.068)	0.52*** (0.046)
N	8,109			

Data are weighted.

Standard errors are in parentheses

* significant at .05 level

** significant at .01 level

*** significant at .001 level

Base category for change in physical activity is Stay Active.

Results for Become Active category are not shown.

Table 4.6. Multinomial Logistic Regression Models for Change in Physical Activity (Odd Ratio) (N=8,109)

Variable	Model 1		Model 2		Model 3		Model 4		Model 5											
	Become Not Active	Stay Not Active	Become Not Active	Stay Not Active	Become Not Active	Stay Not Active	Become Not Active	Stay Not Active	Become Not Active	Stay Not Active										
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE										
<i>Disadvantage Measures</i>																				
Welfare/Poverty Status	1.22*	0.112	1.38**	0.164	1.14	0.113	1.22	0.149	1.16	0.119	1.21	0.151	1.11	0.119	1.07	0.142	1.08	0.120	1.11	0.159
Parent Education High School or Less					1.23*	0.117	1.51**	0.151	1.24*	0.116	1.52**	0.153	1.15	0.109	1.34**	0.136	1.14	0.107	1.32**	0.134
<i>Ethnic Minority Status</i>																				
Non-Hispanic Black									0.93	0.119	1.11	0.182	0.96	0.123	1.11	0.176	0.91	0.120	1.07	0.169
Hispanic									0.86	0.139	0.90	0.152	0.85	0.139	0.89	0.153	0.87	0.141	0.91	0.179
Asian									0.81	0.206	0.98	0.257	0.77	0.197	0.93	0.234	0.81	0.211	0.95	0.268
Other									0.38	0.243	0.40	0.293	0.36	0.215	0.37	0.256	0.37	0.231	0.34	0.252
<i>Parental Control</i>																				
Monitoring													1.02	0.027	0.90**	0.029	1.01	0.028	0.96	0.031
Supervision													0.92	0.057	0.97	0.072	0.97	0.071	1.02	0.088
<i>Parent Child Relations</i>																				
Shared Activities													0.79***	0.035	0.59***	0.028	0.79***	0.036	0.61***	0.031
Shared Decision-Making													1.00	0.056	1.01	0.060	0.98	0.055	0.99	0.063
Shared Meals													1.03	0.023	0.98	0.023	1.03	0.024	1.00	0.023
Parent-Child Communication													0.99	0.058	1.04	0.066	0.97	0.058	0.97	0.063
Closeness and Warmth													0.80*	0.077	0.66***	0.061	0.83*	0.076	0.76**	0.070
<i>Control Measures</i>																				
Female																	1.79***	0.150	3.84***	0.391
Age																	0.97	0.036	1.26***	0.049
Parent Obese																	0.97	0.105	1.15	0.133
<i>Family Structure</i>																				
Step family																	1.05	0.124	0.89	0.133
Single mother																	1.19	0.150	1.06	0.162
Single father																	0.70	0.722	2.05	2.478
Other family structure																	1.33	1.033	0.73	0.627
Full-time Working Mother																	1.14	0.139	1.24	0.157
Family Size																	0.99	0.039	0.99	0.044
Extended Family																	1.04	0.168	0.95	0.156
Pseudo Log Likelihood	-15294195		-15267156		-15250922		-14835002		-14305800											
Pseudo R Squared	0.0009		0.0027		0.0037		0.0309		0.0655											

Data are weighted.

* significant at .05 level

** significant at .01 level

*** significant at .001 level

Base category for change in physical activity is Stay Active.

Results for Become Active category are not shown.

Omitted Categories: Non-Hispanic White, Two Biological Parents, Missing Parent Obese

CHAPTER 5: CONCLUSION

This dissertation research investigated the relationship between social disadvantage and obesity outcomes using the National Longitudinal Study of Adolescent Health (Add Health). Using a dynamic longitudinal model, I examined the impact of social disadvantage in adolescence on patterns of obesity and physical activity across the transition to young adulthood, a period when adult lifestyle behaviors are solidified and major racial/ethnic and socioeconomic disparities become apparent. The highest rates of obesity occur among the most disadvantaged population groups, racial and ethnic minorities, and those with the highest poverty rates and the least education. Striking disparities in the burden of illness and death experienced by racial and ethnic minorities exist despite improvements in the overall health of the nation. Obesity is a particularly serious public health concern because it increasingly affects the youngest and most vulnerable population groups, children and adolescents. Obesity in childhood tracks into adulthood, increasing adult health problems and compounding social and economic inequality.

I used a life course framework and the ecological model of human development to conceptualize the mechanisms of social disadvantage that link race/ethnicity and low SES to obesity and physical activity. I used data from three waves of Add Health to track obesity and physical activity trajectories from adolescence (WII: ages 13-19 y.) into adulthood (WIII: ages 18-26 y.) and to measure factors at the individual, family, peer, school and neighborhood levels in adolescence (WI and WII) that shape these trajectories.

My three specific aims investigated the role of social disadvantage in the social contexts of young people's lives and determined whether they served as mechanisms through which race/ethnic and SES disparities operate. These mechanisms include: (1) cumulative risk (CR) indices, which refers to the number of risk factors that exists in a child's social environment with the assumption that disadvantage is related to the accumulation of risk factors rather than a singular exposure; (2) Multilevel socioeconomic disadvantage—disadvantage present in the peer, school, and neighborhood context (e.g., proportion of peers/school/neighborhood with parents who have a high school diploma or less) and (3) parenting styles and practices.

Each specific aim was addressed in a series of three papers. In the first paper, I found that risk does operate in a cumulative manner, although this relationship differs by sex and level of social context. Cumulative risk also mediated the relationship between both race and obesity and poverty and obesity in transition to adulthood. In the second paper, I found school level social disadvantage to be the most salient context influencing obesity. In the third paper I found that multiple measures of parenting mediate or directly influence the relationship between social disadvantage and physical activity, including shared activities, dinners, decision-making, communication and closeness.

Several themes can be drawn from the findings in this research. The first theme is that African Americans and Hispanics, especially poor minorities, continue to be disadvantaged in the United States. Add Health provides a representative sample of adolescents in middle school and high school in the mid-nineties. This recent cohort of individuals represents a good picture of the social contexts and health profiles of young Americans. This research found that African Americans and Hispanics continue to be

disadvantaged along multiple dimensions of social context. Most striking is that *non-poor* African Americans face *higher* levels of risk for obesity both at the individual/family-level and neighborhood level than *poor* whites. African-American and Hispanic youth also live in more disadvantaged neighborhoods, attend more disadvantaged schools and have more disadvantaged peer groups. These findings highlight numerous aspects of the social environment that contribute to racial and socioeconomic disparities in obesity.

A second theme that can be drawn from this research is that social context matters. In Paper 1, I found that the relationship between cumulative risk at the individual/family level and obesity outcomes in the transition to adulthood and cumulative risk at the neighborhood level and obesity outcomes in the transition to adulthood differed. Among females, individual/family risk completely mediated the relationship between welfare/poverty and change and continuity in obesity status from adolescence into adulthood. However, when neighborhood risk was added to the female model, the relationship between being black and obesity was completely explained. Thus, among females, individual and family-level factors explain the disparities in obesity by poverty, but neighborhood differences explain the obesity disparities by race. The magnitude of the relationship between cumulative risk and obesity also varied according to context (i.e., the measure of cumulative risk used) with stronger relationships found for individual/family risk versus neighborhood risk. I also found in Paper 2 that school level social disadvantage was most salient in influencing obesity for both males and females. This research highlights the importance of measuring multiple levels of social context in which an individual operates.

The third theme that can be drawn from this research is that a longitudinal perspective provides insights not gained from point-in-time designs. This research highlighted the

enduring influence of social environment in adolescence on outcomes in young adulthood. Examining the role of context within a longitudinal framework helps to show important long term effects of social context on health. In all chapters I found evidence of longitudinal relationships. Risk at both the individual/family and neighborhood level in adolescence influenced both incidence and continuity of obesity in the transition to adulthood. School level social disadvantage also influenced obesity in young adulthood. Parent-child relationship quality was strongly associated with both incidence and continuity of low levels of physical activity in the transition to adulthood.

The fourth theme that can be drawn from this research is that gender matters. Paper 1 and Paper 2 analyses found that the relationship between social disadvantage and obesity differed by sex. Risk factors for obesity differed by sex and the influence of social context on obesity differed by sex. Although gender was not a focus of this analysis, these results highlight the need for a better understanding of why relationships differ by sex. Future research should work to better explain these differences. In addition, understanding differences by both race/ethnicity and sex is another important area for future research.

Finally, an overall theme that can be drawn from this research is that social disadvantage is complex and the mechanisms that link disadvantage to health are complex. Social disadvantage and the mechanisms that link social disadvantage to health should be measured at multiple levels and over time. My dissertation was an attempt to measure disadvantage in multiple ways using different theoretical models and analytical frameworks. I found that all levels of social context matter and that relationships between disadvantage and health differ depending on the way that disadvantage is conceptualized. This supports theories that social disadvantage is a fluid and multidimensional concept. All measures of

social disadvantage used in this analysis add to our understanding of obesity and physical activity disparities. More research needs to be done to find ways to better capture the social context of disadvantage in ways that can integrate multiple theoretical perspectives.

This dissertation contributes to social science research on racial and ethnic and socioeconomic health disparities by utilizing dynamic, interconnected and multilevel conceptualizations of the environment to study obesity and physical activity outcomes. By identifying alterable mechanisms operating in the social environments of disadvantaged populations that produce obesity and physical activity disparities, this research can inform policies and interventions aimed at eliminating them.

In future research, I would like to continue to pursue questions at the intersections of social inequality and health, especially those that have arisen in the process of writing this dissertation. The coming release of Wave IV of Add Health will provide an unparalleled opportunity to study the long-term effects of child and adolescent social context and health status on a number of outcomes in adulthood and I plan to use these new data in my future research.

Studying obesity among adolescents and young adults has led me to think that obesity is not only influenced by socioeconomic factors in adolescence, but also serves as a marker of social stratification in adulthood by its subsequent impact on measures of social status, such as marriage, educational, employment and psychosocial outcomes. In particular, I want to examine the cumulative effects of obesity over time on quality of life (measured using social, economic and health factors) and how this relationship differs by race/ethnicity, sex

and neighborhood context. I plan to study these relationships using Wave IV of Add Health when respondents complete the transition into adulthood at ages 24-32.

I have consistently found large gender and racial/ethnic differences in the relationship between social disadvantage and obesity. Because much of the research on obesity is descriptive, the reasons for these differences remain largely unexplained. My training in social demography has taught me that description is an essential starting point before moving to test the social factors that can help to explain disease patterns and health inequalities. My dissertation and collaborative research is a blend of this descriptive demographic approach and use of sociological theory to explain health disparities using large nationally representative data sets. However, it will be important to move into using different research methods and datasets to answer more nuanced questions about health disparities, such as explaining gender and race/differences in obesity. This includes the collection of ethnographic information to better understand the influence of poverty on food choice, eating and physical activity patterns for different demographic subgroups and the use of smaller data sets that provide rich descriptions of the neighborhood environment and family context in which individuals operate. It also includes the utilization of data sets that contain geocoded information on the built environment. I can investigate not only how built environments differ by neighborhood socioeconomic status, but also how particular aspects of the built environment differentially affect the physical activity and food choices of males and females and different racial/ethnic groups.

My research has also found strong linkages between parental obesity and child obesity, after controlling for a number of relevant socioeconomic factors. Sociological explanations would link this strong relationship to role-modeling and parental behavior,

something I have tried to capture in my dissertation. However, a biological role is important to consider when examining the parent-child obesity link. For example, does parenting mitigate or compound obesity risk for individuals with certain genetic profiles? Do specific aspects of socially disadvantaged environments serve to moderate genetic propensities toward obesity? How do biological data fit into a larger framework in understanding the relationship between social context and obesity?

Being able to incorporate information from ethnographic research, the built environment and biological data in a way that complements and broadens sociological understanding of the way that social context influences health, will help move my field forward and I look forward to contributing to the literature in this way. In addition, I will be able to apply my research in ways that will improve population health, especially for the most vulnerable and disadvantaged populations in this country.